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• Supportive care is based on recognition of patients needs
• Focused monitoring and patient evaluation leads to individualized care.
• Appropriate patient evaluation provides for the recognition of anesthetic risks and anesthetic concerns for that specific patient and procedure. “Problem-based” anesthetic management is the framework for individualized patient care.
• What “anesthetic concerns” have you identified for this patient? “100 things are missed due to not looking for every 1 thing missed due to not knowing”.
• Preanesthetic physical examination and laboratory analyses - individualized
  o “minimum data base” based on risk
  o diagnostic imaging
  o radiographs, contrast studies, CT, MRI,
  o ultrasonography, scintigraphy, etc.
  o other directed testing

ASA physical status categories
0. American Society of Anesthesiologists (ASA)
1. ASA I - excellent anesthetic risk
2. ASA II - good anesthetic risk
3. ASA III - fair anesthetic risk
4. ASA IV - poor anesthetic risk
5. ASA V - guarded anesthetic risk additional “Emergency” designation (x)-E

Ventilatory complications
• Airway obstruction
• Inadequate Delivery of Oxygen
• Hypoventilation
• Inadequate Ventilation, Apnea
• Hyperventilation: Tachypnea or panting
• Irregular patterns of ventilation
• All anesthetics are respiratory depressants!
• Anesthetic overdose: Relative or Absolute
• Direct depression of central respiratory centers
• Secondary to circulatory depression
• Specific drug actions

Hypoventilation requires patient support
Endotracheal intubation, ventilatory support by IPPV manual or mechanical ventilation based on patient monitoring, evaluate and address the underlying problem.

Hyperventilation and/or panting are less common, but may reflect hyperthermia, pain, or occur as a side-effect of specific drugs. Control of body temperature, management of pain, and control of ventilation may be necessary.

Support for circulatory compromise
Hypotension is a common problem due to hemorrhage and/or vasodilatation. Circulatory support is largely based on fluid therapy, including crystalloids, and colloids. A variety of fluids are now available, with blood substitutes serving and important need. Vaso-active agents help support blood pressure, cardiac function, and tissue perfusion.

Temperature support
Hypothermia is an almost universal problem in small animal anesthesia and many areas of critical care. Risks of thermal support have been great with older heating strategies. The forced warm air systems offer a new method and much better means of providing thermal support.
• Metabolic and endocrine support is needed by some at-risk patients due to immaturity or disease.
• Inadequate glycogen storage or availability
• Adrenal insufficiency
• Excessive physiological demands

**Delayed recovery from anesthesia**
0. Anesthetic overdose
1. Inadequate elimination or metabolism
2. MDR-1 Mutations, Heterozygous or homozygous, Genetic testing is available
3. Hypothermia
4. Debititation
5. Neurological deterioration

**Management of delayed recovery**
0. Physiological support
1. “SOP” - monitor, evaluate, diagnose, treat
2. Facilitate elimination or metabolism
3. Reversal of anesthetic drugs only when appropriate
Clinical Pain: How to Prevent or Manage it
Ralph Harvey, DVM, MS, DACVAA
University of Tennessee
Knoxville, TN

Pre-emptive analgesia
Balanced analgesia

Dose to effect

Clinical uses of opioids

- Antitussive: cough suppressant action of opioids
- Pre-anesthetic medication, analgesia and sedation
- Induction of anesthesia: sole drug for some species (humans), valuable adjuvant for veterinary patients
- Intraoperative analgesia: reduced anesthetic requirement, decrease complications and improve outcome
- Postoperative pain relief: improved outcome and quicker recovery, reasonable post-op comfort for all
- Critical care adjuvant: basic analgesia, facilitates mechanical ventilation, tolerance of tracheal tube, and other supportive measures. Providing good analgesia improves clinical outcome for critically injured patients.

Opioid analgesics and recommended doses

**Morphine**
- Dog: 0.25 - 0.5 mg/lb (0.5 - 1.0 mg/kg) IM, SC
- Cat: 0.025 - 0.05 mg/lb (0.05 - 0.1 mg/kg) IM, SC (Very useful at 1/10th the dog dose)
- Duration: 4-6 hours
- Sedation accompanying analgesia
- Vomiting, diarrhea, and bradycardia may occur
- Hypotension and bronchoconstriction (histamine release, especially with IV use) may occur

**Meperidine (demerol)**
- Dog: 1-5 mg/lb (2-10 mg/kg) IM, SC
- Cat: (usually effective, avoid high doses)
- Duration: 0.5-1 hour
- Mild sedation
- Rarely histamine release and hypotension
- Cardiac and gastrointestinal effects are relatively minimal

**Hydromorphone**
- Dog: 0.025-0.05 mg/lb (0.05-0.1 mg/kg) IM, SC, IV
- Cat: 0.01-0.025 mg/lb (0.025-0.05 mg/kg) IM, SC, IV
- Duration: 4 hours
- Panting, vomiting, diarrhea, bradycardia may occur
- Dose-dependent sedation or excitement

**Fentanyl**
- Dog and Cats: 2-10 micrograms/kg/hr as a constant rate infusion after IV loading dose of 2-10 micrograms/kg
- Rapid onset and short duration
- Procedural uses and as a CRI for sustained and titratable analgesia in critical care
- May be combined with lidocaine CRI
- Recuvyra TransDermal fentanyl for dogs, sustained fentanyl analgesia

**Mixed-acting agonist-antagonist opioid analgesics**

**Pentazocine (talwin)**
- Dog: 1-2 mg/lb (2-3 mg/kg) IM
- Cat: same as dog dose
- Duration: 4-6 hours
- Only mild analgesia
- Minimal systemic effects
- Narcotic reversal, ceiling effect
Butorphanol (torbutrol, torbugesic, stadol)
- Dog: 0.1-0.2 mg/lb (0.2-0.5 mg/kg) IV, IM, SC
- Duration: 1-2 hours
- Antitussive effect (often desirable)
- Minimal systemic effects
- Narcotic reversal (partial reversal)
- "Ceiling effect" – very limited analgesia, useful for mild sedation
- Cat: (same as for dog, except for a longer duration of action 2-4 hours)

Nalbuphine (nubain)
- Dog: 0.2-1.0 mg/lb (0.5-2.0 mg/kg) IM, IV
- Limited effectiveness - “ceiling effect”
- Minimal sedation
- Minimal systemic effects
- Narcotic reversal, ceiling effect

Partial agonist opioid analgesics
Buprenorphine (Buprenex, Temgesic, Simbadol, Buprenorphine-SR)
- Dog: 0.005-0.01 mg/lb (0.01-0.02 mg/kg) IM, SC
- Cat: 0.005-0.01 mg/lb (0.01-0.02 mg/kg) IM, SC, and oral transmucosal with excellent uptake
- Duration:
  - Buprenorphine: 6-8 hours (slow onset, even with IV injection, 30-40 minutes)
  - Buprenorphine-SR: (claimed 72 hours, not FDA approved)
  - Simbadol: FDA approved, 24 hours duration in cats, euphoric behavior and mydriasis
- Complications: Minimal systemic effects, some “ceiling effect”, but rather potent analgesia.

Non-opioid analgesics
Tramadol (ultram, ultracet, etc.)
- Mild opioid, non-NSAID analgesic with mu-opioid binding activity and interference with both serotonin storage and norepinephrine re-uptake. Analgesic action exceeds mu receptor binding characteristics. First metabolite has greater mu-binding than parent compound. Not a DEA controlled substance. Tramadol may be effective where a weak opioid such as codeine would be chosen. Tramadol dose in Dogs: 1-4 mg/kg PO q 8-12 h for 5-7 days. 50mg tablets available in the US. Call Besse medical 1-800-477-7119.

Ketamine (ketaset, ketalar)
- Very low doses (actually sub-anesthetic, “sub-psychogenic” doses) potentiate opioid analgesics.
- The use of these very low doses, in balanced analgesic combinations, is a relatively new strategy. Dog or Cat doses: 0.01-0.5 mg/lb (0.02-1 mg/kg) IM, SC, PO, combined with opioids, etc.
- "Cataleptic" effects are minimal or absent at these low doses. Duration of 4-6 hours.
- CRI: loading dose 0.5-2.0 mg/kg, CRI at 2-10 micrograms/kg/min

Dex-Medetomidine (DexDomitor, DexDomitor 0.1, precedex in humans)
- Procedural analgesia, marked sedation. Standard dose is scaled to BSA in dogs (see box label).
- We use “lower dose” DexDomitor method: DexDomitor (5-10 microgram/lb, 2.5 to 5 microgram/kg) IM or IV, combined with Torbugesic (0.1-0.2 mg/lb, 0.2-0.4 mg/kg), or other opioids, IM or IV.
- Reversal with Antisedan (by IM injection only) leaves the mild Torbugesic effect intact.
- Recent evidence and extensive clinical experience in humans and veterinary patients supports expanded extra-label use, including post-operative management of delirium.
- Shorter duration and less need for reversal of DexDomitor relative to Domitor may be due to actions of Levo-Domitor in the previous formulation.

Xylazine (rompun, anased)
- Lacks specificity. A poor substitute in dogs and cats for dexmedetomidine.

Lidocaine CRI
- Pro kinetic, reduced dose of other analgesics, possible anti-inflammatory action
- Loading dose 1-2 mg/kg, CRI at 25-100 micrograms/kg/min
- Easy set-up method: 68 cc of 2% lidocaine added to liter bag of IV fluid (or 34 cc lidocaine added to a 500 cc bag) administered at 1cc/pound/hour will provide 50 micrograms/kg/min
- Adjust rate between 0.5 and 2.0 cc/lb/hr to give lidocaine at 25 to 100 mcg/kg/min
- Reduce or discontinue if clinical signs of intolerance or overdose toxicity occur: nausea, CNS stimulation (twitching or seizures)

**Non-steroidal anti-inflammatory drugs (NSAID’s) and related analgesics**

Perioperative risks of renal damage may be reduced by the appropriate administration of IV fluids before and during anesthesia to maintain blood pressure and renal blood flow. GI irritation may be subject to great individual variation. Patients should be monitored for development of intolerance and side effects as well as for changes in efficacy. At this time, any chronic use of NSAIDs in cats in the US is an “extra-label” use. See 2015 AAHA Pain Management Guidelines. Some of these NSAID’s are listed for historical reference only!

**Meclofenamic Acid (arquel) (meclomen)**
- Dog: 0.5 mg/lb (1.1 mg/kg) PO q 24 h Short-term use only
- Recommended for medical management of orthopedic conditions, e.g. hip dysplasia
- Complications reported with chronic administration: vomiting, tarry stools, mucosal erosions, leukocytosis, and decreased hemoglobin

**Ketoprofen tablets and injectable**
- Dog or Cat: 0.5-1 mg/lb (1-2 mg/kg) q 24 hours, IV (dogs only), IM, SC, PO initially, then reduce this dose by 1/2 for subsequent doses
- Mild to moderate pain
- Recommended for not more than five days in dogs. Use may be limited to one day in cats.
- Complications may include GI irritation, ulcers, and renal damage.

**Carprofen (rimadyl)**
- Dog: 1mg/lb q12h (or 2 mg/lb q24h) per os. Cat: 1 mg/lb q12-24h, 1-2 doses only
- Most popular NSAID in dogs
- Relatively specific for COX-2 (anti-inflammatory) effects.
- Oral formulation available in 25, 75, and 100mg caplets or chewable tablets.
- Injectable carprofen 2mg/lb (4 mg/kg)

**Etodolac (etogesic)**
- Dog: 4.5-6.8mg/lb (10-15 mg/kg) q24h per os.
- Newer NSAID with reduced GI toxicity.
- Relatively specific for COX-2 (anti-inflammatory) effects.
- Available in 150 and 300mg scored tablets.

**Deracoxib (deramaxx)**
- Dog: 1-2 mg/kg/day as single daily dose for OA; or at 3-4 mg/kg/day as a single daily dose not to exceed 7 days, for post operative inflammation and pain
- “Cox-2 specific” action
- Flavored tablets.

**Tepoxalin (zubrin)**
- Dog: 10-20 mg/kg on first day, then 10 mg/kg daily for OA or postoperative pain
- Rapidly disintegrating tablets (Redi-tabs)
- “Dual-pathway” inhibition of Cox/Lox. A different approach to the Cox-1/Cox-2 dogma on side effects of NSAID’s

**Meloxicam (metacam)**
- Dog: oral (flavored) liquid, 0.2 mg/kg on day one, then 0.1 mg/kg daily on food
- “Out-sells Rimadyl in Canada at 5:1” Work down to lowest effective dose.
- Extensive clinical experience in Canada and EU, including the injectable formulation
- Cats: 0.3 mg/kg (0.14 mg/lb), injectable by subcutaneous route, single dose
- Oral meloxicam for cats (extra-label): Recommended often for cats at one drop q24-48 hrs after loading dose of two drops. Chronic use is controversial but recommended by some experts. (Little research data as yet. Risk of renal toxicity.) 0.1mg/kg as single dose Day 1; 0.05mg/kg as single dose Days 2,3,4; 0.025mg/kg as single dose on Days 5,6,7 and thereafter q48h

**Firocoxib (previcox)**
- Dog: Chewable tablets, 5 mg/kg (2.27 mg per pound) every 24 hours orally, with or without food. Available as 57 mg and 227 mg tablets. Selective inhibition of cyclooxygenase-2 (COX-2).

**Robenacoxib (onsior)**
- Very high margin of safety. Chemically modified diclofenac. Highly palatable in cats.
- Oral tablets (6mg) and injectable liquid (20 mg/ml, 2 mg/kg by subcutaneous injection)

Other NSAIDs
Many available. Toxic side effects can be prominent. Many reports warn of G.I. toxicity. Considerable patient-to-patient variation in susceptibility to toxic effects. Anti-inflammatory and analgesic effects are also quite individual (as in human patients). G.I. protective agents (e.g. misoprostol) can be beneficial.

Misoprostol for GI protection
Misoprostol (Cytotec) can be given to dogs or cats at 2-5 mcg/kg orally (once a day in cats, twice daily in dogs). It is used to increase tolerance to NSAID’s by reducing G.I. ulceration. Toxicity/side effects are anorexia and hepatopathy. Liver function testing (or liver enzyme analysis) should be performed monthly with misoprostol use (especially if misoprostol and NSAID’s are administered concurrently).

Acetaminophen (Tylenol, etc.)
- Analgesic and antipyretic for dogs, but not anti-inflammatory.
- Dog: 4.5-6.8mg/lb (10-15 mg/kg) q8h per os.
- Lacks GI toxicity. Can be most useful for dogs in opioid combinations, particularly for cancer pain, but also popular for postoperative use as Tylenol #3 or #4.
- **VERY TOXIC FOR CATS!**

Acetaminophen toxicosis in cats
- Clinical Signs: cyanosis, dyspnea, facial edema, depression, and emesis
- Etiology: glucuronyl transferase deficiency (relative to other species)
- Therapy: Avoid stress!! If recent ingestion - induce vomiting Activated charcoal (2gms/kg) along with a saline cathartic, e.g. sodium sulfate (0.5 mg/kg) as a 20% slurry. Oxygen ASAP if cyanotic, but without adding further stress. Acetylcysteine (Mucomyst, Mucosol) 140 mg/kg PO or IV, and repeat at 70 mg/kg q6h for 36 hrs (seven treatments). Ascorbic acid 30mg/kg orally or parenterally with the acetylcysteine. Fluid therapy - lactated Ringer's solution. Continued intensive care and physiological support.
  - (See: Cullison, R.F., Comp. Cont. Ed. 6(4): 315-320, April 1984.)

Epidural analgesia and anesthesia:
Spinal or epidural opioid analgesia has been well described and proven effective in veterinary medicine. Epidural morphine analgesia is widely used in referral and academic centers and is increasingly popular as a method for providing long-lasting profound analgesia. The technique is rather simple, easily accomplished with basic clinical skill, and can be very cost-effective for providing substantial analgesia.

A relatively very small dose of morphine (0.1 mg/kg) is administered by epidural injection after induction of general anesthesia but prior to the surgical procedure. Effective pain relief persists into the following day (12 - 24 hrs) and is accompanied by a blunting of deleterious post-operative increases in stress hormones and the metabolic response to surgery. Combinations of morphine and bupivacaine provide the greatest analgesic effect.

Anatomical landmarks for lumbosacral epidural injection
- Iliac crests, dorsal midline, and lumbar vertebral spinous processes
- For epidural injection, patients are under anesthesia and positioned in either sternal or lateral recumbency based on personal preference and best recognition of anatomic landmarks.

Dose recommendations
- Morphine* 0.1 mg/kg
- Diluted to total volume of 0.2 ml/kg
- (Maximum of 6 ml for dogs and 1.5 ml for cats) using:
  - Saline
  - (or) Lidocaine (2.0%)
  - (or) Bupivacaine (0.5 %)

Duration of analgesia: 12-24 hrs.

Contraindications
- Local infection
- Neurological dysfunction
- Marked obesity (increased difficulty)
- Hypovolemia / Hypotension (avoid the local anesthetics)
Preservative-free morphine (e.g. Duramorph), designed for epidural use, is the best-recommended product. With the preservative-free morphine preparation, cost is increased. We currently do use the preservative-free morphine, usually in combination with either saline or 0.5% bupivacaine. The Duramorph preparation is at a concentration of 1.0 mg/ml. We administer 1 cc Duramorph per 10 kg body weight (0.1 cc/kg) mixed with either saline or bupivacaine, also at 1 cc per 10 kg, for a total volume of 2 cc per 10 kg, with a maximum total volume of 6 cc. The 30-pound dog in this illustration would receive 1.4 cc of Duramorph mixed with 1.4 cc of either saline or bupivacaine.

Combinations of opioids, alpha-2 agonists, and local anesthetics are becoming popular (once again) for epidural regional analgesia/anesthesia in various large animals including horses, cattle, and llamas.

Fentanyl patches for sustained analgesia

Trans-dermal uptake of sustained-release fentanyl from a patch applied to the skin can provide long-term analgesia. This relatively new technology has now moved from the management of cancer pain in human patients to veterinary care where it is useful for sustained analgesia in animals with significant trauma, e.g. multiple fractures after vehicular trauma, as a portion of the management of post-operative pain, and in some cancer patients. Fentanyl patches can be very useful in cats as well as dogs.

The fentanyl patches sold as Duragesic from Janssen. They are available in different rates of drug delivery: 25, 50, 75, and 100 microgram/hour. Fifty mcg patches have been reported effective for small and medium size dogs. The 25mcg patches have been used extensively in cats. The behavioral effect of dysphoria and dementia may be unacceptable in some animals and may require tranquilization or removal of the patch. Uncovering only half of the barrier layer before application has been used in an attempt to reduce the dose, and minimize this problem, particularly in smaller dogs and in smaller cats, but is unreliable.

Patches are applied to clipped skin in an area that the animal cannot reach, such as the dorsal neck or the interscapular area of the dorsal midline. Uptake is somewhat variable among patients and clinical efficacy may be related, in part, to differences in uptake of fentanyl. Onset of analgesia is probably several hours after application of the patch. Hence, for operative or traumatic pain, some other drug should be used initially to provide analgesia. It is very important that the patient is prevented from damaging or ingesting these patches or the contents. Be aware that application of a heating pad, as during surgery, can greatly increase uptake of the drug with significant overdose possible. Duration of effectiveness is roughly four days.

Fentanyl can be a highly abused opioid and there have been reports of clients diverting fentanyl patches from their animals for drug abuse/diversion purposes. Some clinicians find the fentanyl patches a very useful part of managing cancer pain in outpatients, including the terminally ill. It is important to emphasize the potential dangers and the importance of protecting other pets and children from ingestion or other possible exposures. Expended (used) patches still contain fentanyl, and should be handled with care. Duragesic Patches are approved for use in human patients only. All veterinary use is strictly off-label.

Analgesic CRI (constant rate infusion)

CRI options for analgesia

- Lidocaine
- Low-dose ketamine
- Fentanyl
- Morphine
- Combinations of analgesics (e.g. lidocaine plus fentanyl cri)

Adjunctive analgesics

- Tramadol 4-6 mg/kg q 8 hours. Interesting mechanisms of action. Challenging validation for the management of suffering aspects of pain.
- Gabapentin: 3-10 mg/kg q 6-12 hours. Endorsed in 2015 Updated AAHA Pain Management Guidelines.
- Amantadine 3-5 mg/kg q 24 hours.
- Other adjunctive medications.
Favorite Techniques:
Yours and Mine, for Restraint, Sedation and Brief Anesthesia
Ralph Harvey, DVM, MS, DACVAA
University of Tennessee
Knoxville, TN

Obvious (but important) principles
- Appropriate physical exam and other evaluation as indicated
- Customize protocol for each patient's unique situation
- Pharmaceuticals do not take the place of clinical finesse
- Monitor for physiological effects and respond as needed
- Determine adequacy of recovery / fitness for return to ward/owners
- Counsel owners that recovering outpatient animals should be allowed to recover quietly and can not be trusted to respond typically for +/-24 hrs.
- Consult me or another anesthesiologist at any time for specific suggestions.
- Nobody likes an adventurous anesthetist!

There are many other useful options and an infinite variety of clinical situations may occur.

A few of our favorite methods
1. Acepromazine - reliable tranquilization, not recommended for vicious or potentially dangerous animals, side effects are primarily hypotension but rarely some seizure-prone patients may develop seizures, contraindicated in shock or other hypovolemic conditions and in patients with liver disease, duration of effect 2 - 4 hrs, which is too long for most purposes. Very rarely do we use the higher doses.
   a. Ace at 0.01 to 0.05 mg/kg (0.02 to 0.1 mg/lb), max.3 mg total dose, SC, IM, IV
2. Acepromazine & Torbugesic (or other opioid) - substantial and reliable effect; not as often recommended for older/compromised patients; fairly prolonged effect, particularly if hepatic function is impaired; may not be the best choice for out-patients since return to "street fitness" often requires 4-6 hrs.
   a. Ace at 0.01 to 0.05 mg/kg, max.1.0 mg, SC, IM, or IV
   b. Torbugesic at 0.2 to 0.4 mg/kg, SC, IM, or IV
   c. Optional anticholinergics, to avoid or treat bradycardia:
      i. Glycopyrrolate at 0.005 to 0.015 mg/kg, SC, or IM
      ii. Atropine at 0.02 to 0.05 mg/kg, SC, or IM
3. Valium (diazepam) & Torbugesic - less "heavy handed" than Ace & Torbugesic, but also a less substantial and less reliable effect; one of our favorite pre-anesthetic choices for the debilitated generic or geriatric/high risk patient; expect peak effects to last for about 30 min with return to "street- fitness" within 1-2 hrs. Midazolam (Versed) is currently taking the place of diazepam. One-half the listed dose of Valium or Versed is often adequate. Excitement and disorientation may occur and patient should not be stimulated nor trusted to remain sedate.
   a. Torbugesic at 0.2 to 0.4 mg/kg SC, IM, or IV. Followed by:
   b. Valium at 0.25 to 0.4 mg/kg IV (IV route strongly preferred)
   c. or Versed at 0.1 to 0.5 mg/kg SC, IM, or IV
      i. Optional anticholinergic (infrequently needed):
         1. Glycopyrrolate at 0.005 to 0.015 mg/kg, SC, or IM
         2. Atropine at 0.02 to 0.05 mg/kg, SC, or IM
4. Telazol - remarkable physiologic stability, but beware of respiratory depression and potential to initiate seizures; avoid in patients with respiratory compromise and in those with hypertrophic cardiomyopathy or seizure history or certain intracranial or intraocular disorders (increases IOP and ICP). Expect fairly full recovery within 2 hours, but some residual effects are unfortunately too common.
   a. Telazol at 1-3 mg/lb (2-6 mg/kg) SC or IM
      i. Begin with lower doses for restraint
   b. Torbugesic at 0.1-0.2 mg/lb (0.2-0.4 mg/kg) mixed with Telazol
      i. May be given carefully by IV route - increases side-effects
   c. Optional anticholinergic to reduce salivation:
      i. Glycopyrrolate at 0.005 mg/lb (0.01 mg/kg) SC or IM
5. Dex-Medetomidine (DexDomitor, DexDomitor 0.1)) – powerful sedative/hypnotic, similar to medetomidine (Domitor). Recommended for young, healthy, exercise-tolerant dogs. Current evidence and clinical experience both support expanded extra-label use in broader categories of patients, both human and veterinary. Very low doses quite useful to prevent or manage post-operative delirium. Patient monitoring is important. Availability of specific antagonist (Antisedan) contributes to safety and utility. Useful for examinations and brief procedures. Profound bradycardia and hypertension may occur. Tissue perfusion is decreased. Pulse oximeters may fail to detect signal. Use of atropine or other anticholinergics is controversial. We avoid the anticholinergics. Standard dosing in dogs is scaled to body surface area (see insert or dosing guides).

   a. We use a low dose Dexdomitor method (typically 0.0005 to 0.0025 mg/kg), combined with Torbugesic (0.2-0.4 mg/kg) or other opioids. These low doses of Dexdomitor, when combined with an opioid, are very effective. Reversal with Antisedan (by IM injection only) leaves the mild Torbugesic effect intact. Reversal is less often needed with Dexdomitor than with Domitor. Differences in duration of effect and in sedation is interestingly related to the presence of "levo-domitor" in the earlier (Domitor) formulation.
When to treat pain
Newly available analgesics and novel methods for the use of standard medications have greatly expanded options for safe and effective relief of pain in veterinary patients. Analgesic therapy should be considered an integral part of our care when there is a reasonable possibility that pain might result from a medical procedure or condition. The best results are obtained when the analgesics are given before surgery. The key concept is “pre-emptive analgesia”. Recognizing pain in animals requires consideration of overt signs and subtle behavioral changes. As in people, individual analgesic requirements and responses vary with the animal and the peculiarities of each situation through recovery from surgery or critical illness. Therefore, always “dose to effect.”

Pre-emptive analgesia
The best results are obtained when the analgesics are given before surgery. New routes and methods of drug administration are being developed and validated. These include patient-controlled analgesia (PCA) for humans, trans-dermal opioids (patches), controlled release gels, and neuroaxial (epidural and spinal) analgesics.

Multi-modal analgesia
Combination of pain-management methods works much in the same way that we can use anesthetic agents in various combinations for the best patient care. For “balanced analgesia” this may be represented in using some opioid as a pre-anesthetic and post-operative analgesic, along with use of a local anesthetic block. Or perhaps a pre-operative opioid, a local anesthetic infusion both during and after surgery, and an NSAID post operatively. Multi-modal or balanced analgesia has been shown to greatly improve analgesia with fewer side effects than might result from a more massive dose of any single analgesic medication.

Local anesthetic regional analgesia techniques
Techniques for use of local and regional anesthetics in small animal patients are easily learned and applied to substantially reduce the doses of other anesthetics and analgesics needed. These techniques are very cost-effective and greatly improve patient care. In combinations with other strategies (e.g. opioids, NSAID's, dissociative anesthetics) for preventing and relieving clinical pain, these anesthetic/analgesic procedures contribute to “balanced” analgesia or “multi-modal” analgesia.

To avoid toxic effects, the total volume of bupivacaine (0.5%) or lidocaine (2%) should always be less than 0.4 ml/kg or 0.2 ml/lb. Signs of an overdose include nausea, twitching or possibly seizures. At higher doses, cardiac depression can occur, particularly with overdoses or accidental IV injections of bupivacaine. To minimize the risk of accidental IV injections, always aspirate before injection.

Epidural injection
Landmarks are the Iliac crest, dorsal midline, and dorsal lumbar vertebral spinous processes.

Drugs used are typically preservative-free morphine (e.g. Duramorph), designed for epidural use, as the best-recommended product. With the preservative-free morphine preparation, cost is substantially greater than with parenteral morphine. We currently do use the parenteral morphine, usually in combination with either saline or 0.25% bupivacaine. The 0.1 mg/ kg body weight mixed with either saline or bupivacaine, at 0.5 mg/kg.

Brachial plexus nerve block
Produces anesthesia / analgesia distal to the and including the elbow, using bupivacaine at 0.2 ml/kg (0.1 ml/lb) with a 22 ga. 1.5-3.5 inch needle inserted between the shoulder joint and ribs, parallel to vertebrae. Aspirate, inject 0.2cc, withdraw slightly, repeatedly to distribute the bupivacaine. Keys to success include efforts to distribute drug, aspirate to avoid IV injection and toxicity, and minimize volume at each injection site to avoid nerve damage.

Maxillary nerve block
The field blocked includes the maxilla, upper teeth, lip, and nose of the injected side.

Insert needle toward the pterygopalatine fossa from the transition of the hard palate and soft palate or from caudal to the last maxillary tooth, or from below the ventral margin of zygomatic arch, 0.5 cm lateral to the lateral canthus of the eye. Aspirate, to rule out vascular injection, and deposit drug. Dose: 0.1-1.0 ml bupivacaine or Septocaine (preferred).
Mandibular nerve block
The mandibular nerve block is very easy to perform and very inexpensive. The mandibular foramen is located on the medial aspect of the mandible, at the transition from the vertical portion to the horizontal portion of the mandible. We slide the needle, usually a 22ga., 1.5 inch needle, along the interior aspect (medial or oral surface) of the mandible to a site about 1/2 way across that surface (easy to see the foramen on a skull or in a textbook figure). The block can be performed either from inside or outside the oral cavity. Obviously, this nerve block should be performed AFTER induction of anesthesia, but should be done BEFORE surgery is begun.

Mandibular nerve block
Blocking the Inferior Alveolar Branch blocks the mandible, lower teeth, and lip. Insert the needle at the lower angle of the jaw, rostral to the angular process, and advance dorsally to the mid-portion on the medial aspect of the mandible. Aspirate, and then deposit the drug at the surface of the bone. The usual dose range is 0.1-1.0 ml bupivacaine or the dental anesthetic Septocaine (preferred) at lower volume. Doses have not yet been adequately defined.

Declaw analgesia fore limb blocks
Distal Fore Limb Blocks (declaw analgesia blocks) are performed at a level just proximal to the carpus to block the Superficial Radial Nerve (dorsomedial carpus), Ulnar Nerve (branches), lateral carpus, Median Nerve, Ulnar Nerve (branch) on the palmar carpus adjacent to the accessory carpal pad. Deposit 0.1-0.3 ml Bupivacaine 0.5% at each site.

Note: Never use locals containing epinephrine (e.g. Septocaine or lidocaine with epinephrine) for any extremities or tissues with terminal arterial supply (nothing involving the feet, ears or tail!)

Intra-articular stifle block
For the Intra-articular Stifle Block, a 1" 22g needle is used. With the patient in lateral recumbency, with the affected limb uppermost, flex the stifle and apply digital pressure to the medial side of the straight patellar ligament. Insert the needle on the opposite side of the straight patellar ligament midway between the patella and the tibial tuberosity and direct it obliquely and distally toward the intercondylar space of the tibia. Note: possible chondrotoxicity of bupivacaine suggests that this use be reserved for special circumstances and is not routine in our hospital.

Distention of the joint is noted during injection with the long-lasting local anesthetic Bupivacaine 0.5%. This provides 3-6+ hours of duration with a dose 0.2 ml/kg (0.1 ml/lb). Injection is made both pre-op and post-op for best effect.

Wound diffusion or soaker catheters
This is a powerful, effective, easy and cheap way to deliver local anesthetics right to the site of nociceptive transduction and primary afferent transmission. Catheters are commercially available or can be easily fabricated. Elastomeric pumps, syringes and a variety of other devices can be applied to control delivery of local anesthetic to the wound.

Lidocaine intravenous CRI (constant rate infusion) for dogs
Provides an analgesic contribution and reduction in anesthetic requirements. The reduced inhalant anesthetic requirement improves blood pressures and is prokinetic. There is also possible anti-inflammatory contribution. This is a very cost-effective analgesic contribution to opioid analgesics.

Lidocaine loading dose of 1-2 mg/kg may be administered by slow IV injection over three minutes, but is usually not necessary as onset is rapid. Constant Rate Infusion is provided at 20-100 micrograms/kg/minute (0.05-0.1 mg/kg/min) by syringe pump or by controlled drip.

Easy set-up method is as follows: 68 cc of 2% lidocaine is added to a liter bag of IV fluid, administered at 1cc/pound/hour will provide 50 micrograms/kg/min. Adjust between 0.5 and 2.0 cc/pound/hr. Reduce or discontinue if clinical signs of intolerance or overdose occur: nausea, CNS stimulation (twitching or seizures).

Other CRI options for analgesia include: low-dose ketamine, fentanyl, or morphine. A combination of analgesics, one of our favorites, is lidocaine and fentanyl (1:1 mixture) adjusting the rate as needed. Begin at 50 mcg/kg/min for lidocaine and 7.5 mcg/kg/min fentanyl which is achieved at an infusion rate of 0.3 ml/kg/hr of the 1:1 mixture of lidocaine and fentanyl. Adjust the infusion rate as needed to achieve and balance the desired analgesia and sedation.
Implementing the AAHA Guidelines for Dogs and Cats in Your Practice
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Improving the standards of anesthesia and pain management across our entire profession was a goal in developing the AAHA Guidelines for anesthesia and pain management. There are many areas to consider with considerable potential for practical value-added improvement. Anesthetic care of veterinary patients has not gotten easier over the years. Really, any part of medical care become “easier”? It is clear that anesthesia has become better and safer in that we are now able to provide successful anesthetic management for patients who would not have had a reasonable chance a few years ago. In many cases, these are even managed as outpatients, quickly returned to their owners in full recovery. Our choice of anesthetic drugs has greatly expanded, and safer anesthetic agents are indeed responsible for much of the improvement noted. The use of more sophisticated monitoring and better physiologic support has become widespread, with continued rapid growth apparent in this area. In spite of increased owner expectations and the fact that veterinarians now have sicker patients presenting with concurrent diseases, injuries, or debilitation, we can increasingly manage our patients successfully with the improvements in anesthesia and related peri-operative care.

Better training and ongoing training
This collection of proceedings on veterinary anesthesia helps to provide an update on current and developing methods. Continuing education seminars and numerous other contemporary publications attempt to further these same goals. The education of veterinarians and veterinary technicians now includes rather extensive attention to anesthesia and related topics. Veterinarians with advanced training in anesthesia and board certification by the American College of Veterinary Anesthesia and Analgesia are now involved in the training of new veterinary students at almost all North American colleges of veterinary medicine. Through the North American Veterinary Technician Association, licensed veterinary technicians with advanced knowledge, training, and skills in veterinary anesthesia may now pursue Veterinary Technician Specialist certification.

Monitoring and attention to detail
In addition to veterinarians, well-trained technicians continuously evaluate the patient throughout anesthesia. Awareness of the ever-changing condition of the anesthetized patient is a shared responsibility that can only be shared effectively and safely when the medical team works together. We intend to remain aware of even subtle changes in patient status under anesthesia. We must always recognize that challenges to the welfare of our patients come not only from their underlying illness or injury, but also as undesired effects that even the best anesthetic care may present.

Modern monitoring equipment is increasingly available at reasonable cost for veterinary use. We no longer need to rely upon out of date, poorly serviced, unsafe, and inappropriate equipment that has been discarded from human patient use. Fortunately, however, there is good quality equipment still available from the human patient market. Increasingly, that equipment now can be found with good warranty protection, recent service records, and, importantly, with design and function capabilities well suited to veterinary patient needs. There is also good quality equipment available specifically for the veterinary patient. Medical equipment sold exclusively for veterinary use does not receive the degree of oversight and approval required for human-use equipment. In spite of this, there is very good veterinary-specific medical equipment. The demands of veterinarians, and of the animal owners, for improved anesthetic delivery, monitoring, and support has fueled the growth of this industry.

No longer is the application of relatively advanced monitoring equipment and anesthesia machines limited to academic institutions or referral practices with heavy surgical caseloads. Monitoring of electrocardiogram, temperature, blood pressure, and pulse oximetry are rapidly becoming more routine, even in general veterinary practices. Airway monitoring of carbon dioxide and anesthetic gases in the breathing circuit is also becoming more popular. Proper use of these technologies requires a good working knowledge of the normal values, the significance of deviations, and an understanding of appropriate management options.

New options in anesthetics
Through the use of a good variety of injectable and inhalant anesthetics, great anesthetic safety and convenience is possible for our patients. Remarkable improvements for rapid and smooth recovery have developed in “out-patient” anesthesia. The recent popularity of several injectable anesthetics, most popularly propofol, and our new injectable anesthetic, alfaxalone have greatly improved our options. Propofol shortages resulted from the removal of defective generic products, but we all managed that temporary supply and demand issue. Isoflurane has been the strongly predominant inhalant anesthetic for several years. The more newly available inhalant, sevoflurane, can be used to provide for a remarkably rapid yet smooth induction and recovery from anesthesia, and can provide for a rapid change in the level of anesthesia as needed. Appropriate use of these new agents requires skill and knowledge and will be addressed more fully. All anesthetics have a limited therapeutic index, or margin of safety. All can depress vital functions, and
inappropriate use can result in loss of life. It is useful to remember the old guideline: “There are no safe anesthetics, just safe anesthetists.”

While we enjoy a wealth of new options and opportunities in veterinary anesthesia, we must make changes in our anesthetic strategies carefully, recognizing that experience is necessary to identify any abnormal responses from those that should be expected. Careful and conservative use of any new anesthetic or technique is crucial. “Nobody likes an adventurous anesthetist!”

**Individualized anesthetic care**

Much more important than the choice of which specific anesthetic drugs or equipment we use, however, is the manner in which we select them and the skill and care with which they are used in our patients. Best use of various options requires an individualized approach to anesthetic management. In treating infectious diseases, veterinarians wouldn’t choose the same antibiotic for every patient or condition encountered. Similarly, the best choice among options in anesthetic care of individual needs and individual risks vary widely among veterinary patients. We recognize breed sensitivities and relative contraindications in choice of anesthetics. For many years, breed associations have provided warnings based on anecdotal reports. With continued research, some of these have been or will be substantiated. Others perhaps will be refuted. In the absence of clarifying data, caution dictates selection and use of the best anesthetics from among the many choices available. Patient differences that are important in anesthetic care are obviously not only those that relate to species, breed and age differences. As a simple example, patients undergoing elective surgery or those traumatically injured both need analgesic therapy. Opioid analgesics, for instance, have varying efficacy and duration of action. The range of choices allows for brief, mild analgesia such as for an outpatient neuter, all the way to profound analgesia for the care of a substantially traumatized animal.

**Preanesthetic evaluation and screening**

Better anesthetic care also includes more thorough preanesthetic evaluation, which can fit nicely into a comprehensive approach of well-patient care and the work-up of the non-elective patient. Preanesthetic evaluations should be tailored to the needs of the patient. For example, the preanesthetic evaluation of a diabetic patient would include blood glucose determination(s) to help guide physiologic support as a part of the anesthetic care. Basic physical findings may lead to more extensive evaluations. For example, if a heart murmur is detected in a young cat, an echocardiogram may be performed to rule out cardiomyopathy before subjecting the animal to the stresses of anesthesia. Not all patients need the same level or intensity of preanesthetic evaluation or screening. Matching the process to the patient becomes cost-effective for the pet owner as well as for the practice owner.

**Geriatric patient care**

It is fortunate that the improved role of pets in our society has in various ways kept animals as a part of the family for more years. With an aging pet population, and with keen interest in keeping pets as very functional members of the family group, we have the opportunity to care for many more geriatric patients. These much-loved older pets often receive more extensive preanesthetic evaluation, which helps us identify marginal reserve function and any subclinical organ disease or dysfunction. Geriatric patients have dramatically reduced requirement for many anesthetics, and could be overdosed at standard recommended drug doses. Armed with this information, the veterinarian can individualize anesthetic care to minimize the risks of complications. Typical of this patient type would be the older dog presented for routine dental care. Through our improved care, we can extend not only the lifespan, but also the “healthspan” of these animals.

**Outpatient anesthesia**

As human patients, we expect to have most minimally invasive medical procedures, and even many substantial surgeries, conducted on an outpatient or same-day basis. Reduced hospital costs are not the only concern driving this change in human patient care. Everyone is happier and can return to daily routines more quickly when we are able to shorten hospital stays. This all applies to veterinary medicine as well. Better anesthetic care is a major component of this change. Clients personally experience it in their own medical care from the perspective of patients, and now they expect it in the veterinary care we deliver for their pets. Reliable, fast, and smooth recovery from anesthesia is a wonderful feature of many of the more modern anesthetic methods. While every patient differs, we’ve come to expect more and more of our patients to bounce back quickly.

**Prior preparation prevents problems**

Readiness includes anticipation of contingencies and willingness to consider, and perhaps move along to, what we have in mind as the “Plan B” for that patient. This is recognition of whatever else might be likely to happen for this animal other than the expected course of events. Those who are ready for these contingencies can intercept developing problems before they reach the “crisis” stage. This requires attentiveness to warning signs. Good anesthetic monitoring and appropriate responses to changing patient status are much more successful strategies for patient care than would be any level of expertise in crisis management.
**Pain management**

Our clients expect optimal control of animal pain. Clients expect the best in anesthetic survival and in relief of pain. Their most basic expectations are that their pet will survive and that it will not hurt. We do have the tools available to effectively manage procedural, traumatic, and peri-operative pain. We also have increasingly fine methods for very effectively managing the more chronic pains of degenerative joint disease and cancer. The three principles of effective pain management are: (1) pre-emptive analgesia, (2) balanced analgesia, and (3) willingness to dose-to-effect. Application of these principles can help us devise very effective pain management for every patient. Smart use of analgesic strategies offers tremendous benefit through relief of unnecessary pain and suffering. Improvements in the areas of the recognition and management of animal pain have been arguably greater than in any other aspect of veterinary anesthesia. Benefits include improved patient comfort, but also reduced anesthetic requirements, shortened hospital stays, improved immune function, and reduced morbidity and mortality. Good quality pain relief is also very cost-effective.

**Supportive care**

As an example of basic physiological support, the provision of fluid therapy and appropriate patient warming devices is increasingly commonplace in veterinary anesthetic care. Fluid therapy is an appropriate measure to compensate for the vasodilatation and hypotension that can commonly occur with the best of anesthetic techniques. We also recognize, through the increased use of blood pressure monitoring, that many of our patients can become hypotensive. Our older patients may be particularly susceptible to deleterious consequences of inadequate tissue perfusion. Patient warming devices that gently circulate warm air or warm water have replaced dangerous electric heating pads and bags or bottles of warm (or hot) water. All too often, electric heating pads and hot water bags and bottles have either burned animals or failed to properly prevent hypothermia. With individualized patient management, which includes physiologic support, those animals with particular needs or susceptibilities are better prepared for the rigors of anesthesia and surgery.

**Summary**

There seems to be little upper limit to the sophistication of medical care demanded by the pet owning public. Improvements in all areas of veterinary medicine are being rapidly embraced. The standard of care is indeed moving forward in all of our profession, and that was the larger goal in establishing the AAHA Guidelines for Anesthesia and for Pain Management. As tools to facilitate incremental improvements, they aid the profession and each of us. Full implementation of the AAHA Guidelines is very practical for many of our hospitals. Our best clients assume that the veterinary anesthetic care and pain management their animals receive are already at a very high level of sophistication, perhaps even comparable to that afforded human patients. Our obligation to do the best we can for our patients and for our clients requires that we move forward and maintain very high standards in providing anesthesia and analgesia.

**References**

1. 2011 AAHA Anesthesia Guidelines for Dogs and Cats, and supporting materials, [link]
2. 2007 AAHA/AAFP Pain Management Guidelines for Dogs and Cats, Pain Management Resources, [link]
3. Pain management Resources, [link]
New Drugs and Clinical Techniques: What’s Working for your Hospital
Ralph Harvey, DVM, MS, DACVAA
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Non-steroidal anti-inflammatory drug therapy in cats
- Perioperative NSAID for inflammatory pain
- Multi-Modal or Balanced Analgesia
- Patient Selection

Changing paradigm for NSAIDs
- Feline ignored for too long
- Incidence of DJD is high in older cats
- Behavioral and Post Mortem evidence is clear

Extra-label use in United States
- Use with Caution in DJD / Chronic use
- Dose to Lean Body Weight
- Regular laboratory testing for chronic therapy
- Regular Consultation / Adjust Therapy Plan

Robenacoxib (onsior)
- Tablets approved (US) three day course of periop therapy
- Rapid onset, short plasma T1/2, pKa 4.7, Freed from albumin at low pH - sites of injury or inflammation
- Limited toxicity
- Dose 1 mg/kg (1-2.4 mg/kg) up to 3 days approved in US
  - avoid combinations with other NSAIDS, avoid with renal disease
  - injectable form approved in other markets – awaiting changes
- Paradigm shift in pre-op use: Oral! and NSAID!
- Highly palatable (Compliance improved)

Alfaxalone Alfaxan from jurox
- Injectable Steroidal Anesthetic
- Analogue of Progesterone
- Approved for IM as well as IV administration
- Solubility Issues Resolved via Cyclodextrin Cage

Comparative studies: Alfaxan / propofol infusions
- Favorable Therapeutic Index
- Pharmacodynamics Similar to Propofol

Natural neuroactive steroids
- Synthesized by the brain and nervous system
- Regulation of myelinization
- Neuroprotection
- Growth of axons and dendrites
- Modulate expression of particular subunits of GABA_A

Alfaxalone
- Alfaxalone (3α-hydroxy-5α – pregnane -11, 20 – dione) is an ultra-short acting anaesthetic a neuroactive steroid producing anaesthetic effects through activity at the GABA-A receptor
- Similar molecular structure to progesterone, however, at clinical concentrations alfaxalone does not bind to sex hormone, glucocorticoid or mineralocorticoid receptors
Saffan® (vet product) and Althesin® (human product)
- Alfaxalone (9 mg/mL) plus Alphadalone (3 mg/mL)
- A different neurosteroid that improved solubility but has little anaesthetic potency
  - Cremophor®-EL
    - Castor oil derivative to allow solubility
    - Caused histamine release

Alfaxalone - many beneficial properties as an anaesthetic
- It is potent. It has a wide safety margin. It is rapidly metabolised.
- Work on cyclodextrins started to highlight a new way to solubilize lipophilic compounds in the mid 1990s
- In Alfaxan, the alfaxalone molecule is solubilised in cyclodextrin. Cyclodextrins are round complex sugars derived from starch
- Lipophilic (fat soluble) drugs like alfaxalone can be hidden inside to allow the complex to be dissolved in water

Pharmacokinetics
- Alfaxalone is rapidly and consistently cleared, as the metabolic pathways are the same as those used for metabolism of endogenous steroids
- Cats and dogs do produce different metabolites, however accumulation of drug does not appear to occur with repeated dosing to maintain clinical anaesthesia in either species

Pharmacodynamics
- Duration of anaesthesia after full induction dose
- Unpremedicated Dogs (~10 min)
- Unpremedicated Cats (~25 min)
- Cardiovascular and respiratory function is well maintained
- Rapid and smooth induction
- Appropriate muscle relaxation
- Short recovery times even when used as CRI
- Not cumulative in tissues after repeated doses
- Dose-dependent cardiopulmonary depression

Usage tips
- Best administered slowly IV over 60 seconds
- Decreases the risk of apnea and allows the patient to choose the dose required for induction
- Not analgesic
- Ensure to address pain
- Rapidly cleared
- Can result in very rapid / uncontrolled recoveries if not well premedicated
- Minimize stimulation during recovery. Keep the recovery area quiet

Alfaxalone – propofol similarities
- Smooth and rapid induction and recovery
- Apnea as most prominent effect at higher doses
- Short duration of action
- Good muscle relaxation
- Wider margin of safety
- IM Injection Option

Buprenorphine – optimized options
- Simbadol – 24 hours FDA Approved for Cats
- Unique Profile in Cats
- Unique Physiology, Pharmacology of Higher Dose
- Excess Substrate Delays Elimination of Active Drug
- Once Daily Surgical Pain Control
Buprenorphine-SR
- Aqueous polymer vehicle compounded product, lack of evidence, concerns limit use

Recuvrya – extended release transdermal fentanyl
Exposure concerns
- Precautions
  - Application
  - Supervision
  - Protection
- Analgesia – long duration, for dogs only
- Mild sedation or euphoria
  - Infrequent dysphoria
- Reduced food intake

Cerenia® (maropitant citrate)
- An antiemetic therapy for the prevention and treatment of acute vomiting in dogs, and for the prevention of vomiting due to motion sickness in dogs. Oral, or by SC (stings), or IV injection.
- Greatly improves return to spontaneous feeding following opioids!
- Value added for many clients.
Sedation and Restraint for the Fractious Patient - Safety and Convenience for All
Ralph Harvey, DVM, MS, DACVAA
University of Tennessee
Knoxville, TN

Obvious (but important) principles
- Appropriate physical exam and other evaluation as indicated
- Customize protocol for each patient's unique situation
- Pharmaceuticals do not take the place of clinical finesse
- Monitor for physiological effects and respond as needed
- Determine adequacy of recovery / fitness for return to ward/owners
- Counsel owners that recovering outpatient animals should be allowed to recover quietly and can not be trusted to respond typically for +/-24 hrs.
- Consult me or another anesthesiologist at any time for specific suggestions.
- Nobody likes an adventurous anesthetist!

There are many other useful options and an infinite variety of clinical situations may occur.

A few of our favorite methods
1) Acepromazine - reliable tranquilization, not recommended for vicious or potentially dangerous animals, side effects are primarily hypotension but rarely some seizure-prone patients may develop seizures, contraindicated in shock or other hypovolemic conditions and in patients with liver disease, duration of effect 2 - 4 hrs, which is too long for most purposes. Very rarely do we use the higher doses.
   Ace at 0.01 to 0.05 mg/kg (0.02 to 0.1 mg/lb), max.3 mg total dose, SC, IM, IV
2) Acepromazine & Torbugesic (or other opioid) - substantial and reliable effect; not as often recommended for older/compromised patients; fairly prolonged effect, particularly if hepatic function is impaired; may not be the best choice for outpatients since return to "street fitness" often requires 4-6 hrs.
   - Ace at 0.01 to 0.05 mg/kg, max.1.0 mg, SC, IM, or IV
   - Torbugesic at 0.2 to 0.4 mg/kg, SC, IM, or IV
   - Optional anticholinergics, to avoid or treat bradycardia:
     - Glycopyrrolate at 0.005 to 0.015 mg/kg, SC, or IM
     - Atropine at 0.02 to 0.05 mg/kg, SC, or IM
3) Valium (diazepam) & Torbugesic - less "heavy handed" than Ace & Torbugesic, but also a less substantial and less reliable effect; one of our favorite pre-anesthetic choices for the debilitated generic or geriatric/high risk patient; expect peak effects to last for about 30 min with return to "street fitness" within 1-2 hrs. Midazolam (Versed) is currently taking the place of diazepam. One-half the listed dose of Valium or Versed is often adequate. Excitement and disorientation may occur and patient should not be stimulated nor trusted to remain sedate.
   - Torbugesic at 0.2 to 0.4 mg/kg SC, IM, or IV. Followed by:
   - Valium at 0.25 to 0.4 mg/kg IV (IV route strongly preferred)
   - or Versed at 0.1 to 0.5 mg/kg SC, IM, or IV
     - Optional anticholinergic (infrequently needed):
       - Glycopyrrolate at 0.005 to 0.015 mg/kg, SC, or IM
       - Atropine at 0.02 to 0.05 mg/kg, SC, or IM
4) Telazol - remarkable physiologic stability, but beware of respiratory depression and potential to initiate seizures; avoid in patients with respiratory compromise and in those with hypertrophic cardiomyopathy or seizure history or certain intracranial or intraocular disorders (increases IOP and ICP). Expect fairly full recovery within 2 hours, but some residual effects are unfortunately too common.
   - Telazol at 1-3 mg/lb (2-6 mg/kg) SC or IM
     - Begin with lower doses for restraint
   - Torbugesic at 0.1-0.2 mg/lb (0.2-0.4 mg/kg) mixed with Telazol
     - May be given carefully by IV route - increases side-effects
   - Optional anticholinergic to reduce salivation:
     - Glycopyrrolate at 0.005 mg/lb (0.01 mg/kg) SC or IM
5) Dex-Medetomidine (DexDomitor, DexDomitor 0.1) – powerful sedative/hypnotic, similar to medetomidine (Domitor). Recommended for young, healthy, exercise-tolerant dogs. Current evidence and clinical experience both support expanded extra-label use in broader categories of patients, both human and veterinary. Very low doses quite useful to prevent or manage post-operative delirium. Patient monitoring is important. Availability of specific antagonist (Antisedan) contributes to safety and utility. Useful for examinations and brief procedures. Profound bradycardia and hypertension may occur. Tissue perfusion is decreased. Pulse oximeters may fail to detect signal. Use of atropine or other anticholinergics is controversial. We avoid the anticholinergics. Standard dosing in dogs is scaled to body surface area (see insert or dosing guides).

We use a low dose Dexdomitor method (typically 0.0005 to 0.0025 mg/kg), combined with Torbugesic (0.2-0.4 mg/kg) or other opioids. These low doses of Dexdomitor, when combined with an opioid, are very effective. Reversal with Antisedan (by IM injection only) leaves the mild Torbugesic effect intact. Reversal is less often needed with Dexdomitor than with Domitor. Differences in duration of effect and in sedation is interestingly related to the presence of "levo-domitor" in the earlier (Domitor) formulation.
Avian 911:
Managing the Emergent Sick Bird
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The avian patient represents a unique challenge for the practitioner and critical care measures are often needed during emergency visits. Critical care is an integral part of emergency management of disease in zoological species. A high mortality rate is inherent in exotic animal emergency medicine. The nature of these species and the fact that they are fairly recently domesticated dictates that they hide their illness until they are decompensated. Owners need to be aware that their pet is in serious condition but by providing critical care, you provide them with the chance for their pet’s recovery.

Critical care of avians
The statement that by the time the animal is showing signs of illness it is in critical condition was never truer than it is for avian patients. It is very important that owners be made aware that their pet is very sick and that it may decompensate at any time during your exam or treatment. It may only be possible to do one procedure at a time, giving the bird time to recover in between. If at any point during handling the bird is open mouthed breathing, stops struggling, seems dazed, develops a weak grip, or shuts its eyes longer than momentarily, put it down to recover. Oxygen cages or flow-by oxygen may help. Anesthetic restraint may be administered to extremely stressed or fractious patients. Monitor respiratory rate, effort, and heart rate during the entire anesthetic event. Keep the anesthetic duration to a minimum time. Birds can be intubated using an appropriately sized endotracheal tube, 2.5-4.0 I.D., by placing the tube in the glottal opening at the base of the tongue. Do not inflate the cuff as this may result in tracheal necrosis as the cartilaginous rings do not expand.

Hospitalization and caging considerations
Supplemental heat is very important as sick birds quickly lose the ability to thermoregulate. An incubator is best means of providing supplemental heat and should be set at approximately 90-95 degrees F. An appropriately sized perch should also be provided if the animal is strong enough to perch. Food and water should be made available at all times. Have the owner bring the animal’s food as birds tend not to eat what they are unfamiliar with. If the bird’s regular food is unavailable, providing a seed mixture may encourage self-feeding. If the bird is not eating on its own, supplemental feeding is imperative. Several supplemental feeding diets are available and will provide complete nutrition to ill birds. The gruel must be administered by gavage tube directly into the bird’s crop and should be warm (101-104°F). A metal ball-tip feeding tube is ideal. Make sure to palpate the tube in the crop to avoid infusing gruel into the trachea. The crop capacity varies with the size of the bird. In general, the average gavage feeding amount equals 2.5% of the bird’s body weight (0.025 x BW in grams = milliliters of formula per feeding). Feeding interval is generally 6 hours but may be longer depending on crop emptying. Make sure to watch the oral cavity as the food is administered to check for over-filling.

Fluid therapy
IV catheterization of birds is possible using the jugular vein, the medial metatarsal veins, or the basilic veins. However, IV catheters placed in psittacine birds (parrots) are often removed prematurely by the patient. An IO catheter is placed in the distal ulna or the proximal tibia in birds. This should be performed in a sterile manner. The catheter should be taped and bandaged in place using a figure 8 wing wrap. A 3 cc syringe case can be fashioned into a catheter guard as psittacine birds will often chew on the catheter. Fluids can also be provided by SQ or IO routes. SQ fluids are administered in either skin fold located where a leg meets the body or in the wing skin folds where the wings meet the body. Fluids given should be warmed prior to administration. Bolus fluids may be given IV or IO at a rate of 10-25 ml/kg over 5-10 minutes. LRS or NaCl 0.9% are most commonly given and may be spiked with 50% dextrose to create a 5% dextrose solution (add 1 ml of 50% dextrose to 10 ml fluids). Fluids with a dextrose concentration of >2.5% should not be given by SQ route. Fluids administered for more than a day should be supplemented with 0.1-0.2 mEq potassium per day. Maintenance fluid rates for birds are 40-69 mls/kg/day. Shock doses rise to 10 ml/kg/hr for 2 hours, then 5-8 ml/kg/hr until the patient is stable. If colloidal fluid therapy is indicated, Hetastarch at a rate of 10-15 ml/kg IV or IO per 8 hrs may be used.

Basics of hospitalization care
Supplemental heat is critical for patients that have lost or do not have the ability to thermo-regulate their own body temperature. This is especially true of avian species. Providing an external heat source reduces the physiological stress on the animal during hospitalization care. Pediatric or veterinary incubators are extremely useful in caring for critical patients. Incubators come in a variety of sizes and may incorporate fluid pumps and oxygen delivery systems. Many times, used pediatric incubators that are in good working order may be obtained from human hospital surplus. Radiant heating lamps may be attached to cage doors or placed over aquaria to provide ambient heating when an incubator is unavailable. Do not allow the patient or potentially flammable materials to be
in direct contact with the lamp as burns are possible. Heating pads and heating discs are good alternatives for providing supplemental heat for patients that are ambulatory and do not need constant thermal regulation.

Nutritional support must be provided to hospitalized patients, especially those with high metabolic rates where anorexia results in cachexia rapidly. The caloric requirements of the patients must be calculated and those requirements met on a daily basis through self-feeding or supplemental feeding. Metabolic rates are measured in kilocalories per day (kcal/day). The basal metabolic rate (BMR) takes into account a species coefficient (K) based on the animal’s metabolic rate. Once the BMR is calculated, the maintenance energy requirement (MER), the metabolic rate based on activity, can be determined by multiplying the BMR by the activity level of the patient (1-2, 1.5 for convalescing animals, 2 for growth).

- BMR kcal/day = K x Weight kg \(^{0.75}\)
- MER kcal/day = 1.5 x BMR
- K values
  - passerines = 129
  - non-passerines = 78

Once the number of kilocalories needed for MER are determined, the amount of food needed to meet that requirement can be calculated. The best way to determine if a patient is receiving appropriate nutrition is to weigh the patient daily on an accurate gram scale. Supplemental feeding can then be adjusted as appropriate.

CPR and euthanasia
Zoological species presenting in critical condition have a high mortality rate due to the nature of their illnesses, the advanced nature of the emergency when clinical signs are first observed, and the rapidity with which their condition declines. Respiratory resuscitation, if performed when respiratory arrest is first observed, is often successful and should be the first component of cardiopulmonary resuscitation (CPR). If at all possible, the patient should be immediately intubated and ventilated. If intubation is not possible, then artificial respiration may be performed by mouth to face respiration. Most zoological species maintained as pets are small enough that the handler’s mouth will cover the patient’s nose and mouth. Consideration must be given to the zoonotic potential of the patient before this technique is used. Small puffs of air are generally sufficient to inflate the patient’s lungs. Patients that are showing decreased respiration but that are not in full arrest will benefit from oxygen therapy delivered by mask. Air sac cannulation of birds is an option for providing artificial ventilation or oxygen therapy when intubation is ineffective or unavailable. With the bird in lateral recumbency and the leg pulled cranially, a small incision is placed just caudal to the thigh and the body wall is punctured. A breathing tube is then introduced into the body cavity and air sac. IV or IO emergency drugs are administered similarly to their use in domestic pets. If vascular access is unavailable, then drugs may be administered via endotracheal tube directly into the lungs. If cardiac arrest develops, cardiac compressions must be given at a rapid rate. Compression of the body wall over the location of the heart will compress the heart and circulate blood. Compressions in a bird should be done with the bird in dorsal recumbency while the keel is compressed towards the spine.

Euthanasia is the preferred or elected treatment for some zoological species presented for critical care. If vascular access is available by IV or IO routes, euthanasia solution may be administered routinely. Intracoelomic injections in birds are contraindicated due to the air sac system. Whenever possible, the patient is placed under general anesthesia prior to euthanasia to minimize stress and discomfort.

References
Providing care to avian patients requires skill in performing several basic procedures to aid in the evaluation of the bird as well as in acquiring diagnostic samples and implementing therapy. Many of these skills are similar to those performed on domestic animals but have been modified to accommodate the unique anatomy of birds.

**Wing trim**
Trimming of the flight feathers of the wing is an effective means of controlling a bird’s ability to fly which is useful in training the bird to be a healthy companion and also in preventing injury that may occur during flight. When performed correctly, a wing trim will prevent the bird from gaining lift during flight while allowing the bird to glide gently over a few feet to a safe surface. However, wing trims done incorrectly may actually predispose the bird to a myriad of problems including uncontrolled flight with the potential for impact injuries, feather damaging behavior, and negative behavior during subsequent grooming procedures. Wing trims should not be performed on birds that require flight to avoid dangers inherent in their environment such as domestic pets with which they are in direct contact. It is important to consider the species and body type of the bird prior to performing a wing trim as this will impact the number of feathers to be trimmed. Larger bodied birds, such as Amazon parrots and African grey parrots, generally need fewer feathers (4-5) trimmed to control flight compared to light-bodied, agile birds such as cockatiels and budgerigars which require the trimming of more feathers (6-7) to control flight. It is better to be conservative when choosing the number of feathers to be trimmed until the bird can be assessed to determine if more feathers need to be trimmed. Trimming the feathers of both wings will prevent balance issues when flight is attempted by the bird. Ideally, young birds will be allowed to develop flight skills prior to receiving their first wing trim. Cutting instruments such as suture removing scissors or regular utility scissors are useful when trimming feathers of small birds however wire cutters may be optimal when trimming the primary feathers of large birds.

**Nail trim**
Nail trims are routinely performed to provide for handler comfort and also to keep the nails an appropriate length to avoid breakage of the nail or snagging on cage materials. Trimming nails, therefore, is important for bird and owner. The frequency of trimming depends on diet, activity level, and perch sizes and materials. Larger perch diameters favor natural wear of the nails. Sandpaper perch covers should not be used! A pair of suture removal scissors or a commercially available nail trimmer for cats and small mammals is sufficient to trim the nails of small birds (cockatiels and smaller). A power rotary tool is commonly used to perform nail trims on larger birds. alternatively, a handheld file, such as an emery board, may be used if the bird is trained to tolerate unrestrained grooming. Performing a nail trim is usually a two-person job with one person restraining the bird and the other performing the pedicure. Styptics should be available in the event of bleeding which will occur if the nail is cut too short. Styptics will not work if blood is flowing freely so the flow must be stopped by pinching the toe on the sides with fingers. The blood can then be cleaned off and the styptic applied to the surface of the cut nail. The grinding tools take off the nail more gradually and produce some heat so that the nail is cauterized as it is worn away. Professional help should be sought if the owner is not absolutely sure of how to perform these procedures. The owner should be informed that the bird might have a poor grip on perches for a few days.

**Beak trim**
The beak of normal birds should not require trimming. While some mild flaking of outer layers of the beak may be normal, excessive thickening or overgrowth of the beak may be an indication of a problem. It is advisable require an exam before performing a beak trim on a new patient. This is partly to determine the cause of the overgrown beak and correct it, and partly to assess the bird’s ability to tolerate the stress of the beak trim. The quality and texture of the beak keratin, and the length and the occlusion of the rhinotheca and gnaotheca are important parameters to evaluate. The clinician should refer to normal beaks for each species to determine the normal shape. When trimming is required or when an owner wants the beak smoothed for cosmetic purposes, a hobby drill with a grinding stone tip is used to shape the beak. Care should be taken not to take too much off since severe bleeding can occur. Also, many parrots will try to feel the instrument with their tongue, causing trauma to the tongue. Blunting the beak for biting birds is ineffective; it just changes the handler's injury from a puncture wound to a crushing wound, which is often worse.

**Phlebotomy**
Hematology and clinical chemistries are often the initial step in the work up. Whenever possible, collection of blood should be the first procedure of the workup. The prolonged stress of handling can alter some of the clinical values. Blood can be obtained either by venipuncture of the jugular, basilic or medial metatarsal vein, or by clipping a nail in the quick and collecting the blood in capillary
need for a figure 8 bandage. Figure 8 wraps should be removed as soon as no longer needed or at a minimum of every 1-2 weeks in tape applied around the carpus and a second strip encircling the elbow and distal wing may provide sufficient support, foregoing the A tape splint is useful for stabilizing fractures, luxations, and injuries of the pelvic limb, distal to the stifle, of small birds <250 grams. Tape splint order for the wing to undergo controlled range of motion exercises. In order for the tape splint to provide rigid support, the tape must be in direct contact with the skin. All the feathers will need to be tube placement into the crop prior to introduction of the food. The diet to be fed should be warmed to an appropriate temperature (101-104°F) and should be mixed well to avoid hot spots within the gruel. As the tube is withdrawn from the esophagus, keep the bird’s neck extended to minimize the risk of regurgitation. If some of the gruel is regurgitated into the oral cavity, release the bird and allow it to clear the food from its mouth by itself.

Gavage tube placement for feeding or crop wash
A crop wash or aspirate is indicated when the crop content is needed for evaluation such as in birds that are regurgitating, have delayed crop emptying, or other crop disorders. If the crop is full and the content is not too viscous to be aspirated, no solution need be infused into the crop prior to withdrawing the sample. Sterile saline is used if the crop is empty and an isotonic solution is needed to flush the crop chamber. Cytology and direct wet mount exam of the sample will reveal potential abnormalities such as pathologic bacteria, yeast, and protozoal populations.

Nutritional support is critically important in ill avian patients, especially when the patient is anorexic. Several commercially available assisted feeding diets are available. Tube feeding is performed in the same manner as performing a crop wash with the exception that a gruel diet is infused into the crop during tube feeding and is not aspirated back out. It is also important to ensure the tube placement into the crop prior to introduction of the food. The diet to be fed should be warmed to an appropriate temperature (101-104°F) and should be mixed well to avoid hot spots within the gruel. As the tube is withdrawn from the esophagus, keep the bird’s neck extended to minimize the risk of regurgitation. If some of the gruel is regurgitated into the oral cavity, release the bird and allow it to clear the food from its mouth by itself.

Catheterization
Vascular access in avian patients is achieved through placement of intravenous (IV) or intraosseous (IO) catheters. In most practices, IO catheters are utilized as they are relatively well tolerated by the patients and are straightforward to place. However, IO catheters are also potentially painful and traumatic as the needle placement requires penetration of the bone. IV catheters are especially useful in birds that are unlikely to traumatize the catheter with their beaks (waterfowl, pigeons/doves) or those undergoing surgical procedures that require fluid support intra-operatively. Common sites for IV catheter placement include the medial metatarsal veins, the basilic veins, and the right jugular vein.

Intraosseous catheter placement is commonly performed in avian patients that require aggressive fluid therapy or intravenous administration of medications when an IV catheter is not possible or practical. IO catheters are fairly well tolerated in birds and do not result in long-term detrimental effects if placed in an aseptic manner with minimal trauma to the surrounding tissue. Sites for IO catheter placement include the distal and proximal ulna, and the proximal tibiotarsus. Care must be taken to avoid placing the catheter in a manner that would interfere with the articular surfaces of the carpus, elbow or stifle respectively. Because of the insertion of the needle into bone during catheter placement, a spinal needle is best suited for an IO catheter is the needle’s stylet prevents the formation of a bone core that can occlude the catheter. An appropriately sized hypodermic needle may also be used. If a bone core prevents the catheter from being patent, the needle is removed and a replacement needle of the same size is placed following the original needle’s path. The needle size chosen is based upon the diameter of the host bone and should approximate 50% of the bone diameter. IO catheter placement causes temporary discomfort for the bird. Lidocaine injected subcutaneously and around the periosteum of the host bone can help reduce the associated pain. Due to the confined space of the medullary canal of the bone, administration of medication or fluids through the IO catheter will meet more flow resistance than that experienced with IV catheters.

Figure 8 wing wrap
A figure 8 wing wrap is used to provide external coaptation of fractures of the radius, ulna, major and minor metacarpals and other bones distal to carpus; stabilize elbow or carpal luxations; secure distal ulnar IO catheter in place; protect soft tissue wounds on the wing distal to the elbow. A figure 8 wing wrap should not be used to stabilize a humeral fracture unless used in conjunction with a body wrap to secure the shoulder as well. Place the bandage as far under the axilla (armpit) as possible while incorporating the secondary and tertiary covert feathers on the dorsum into the bandage to prevent slippage off the elbow. Figure 8 bandages that are too tight will place stress on the joints of the wing potentially leading to joint injury. The wing should be held in a normal position in the completed wrap. Figure 8 bandages that are too bulky will cause balance disturbances for small patients. In these cases, bandage tape applied around the carpus and a second strip encircling the elbow and distal wing may provide sufficient support, foregoing the need for a figure 8 bandage. Figure 8 wraps should be removed as soon as no longer needed or at a minimum of every 1-2 weeks in order for the wing to undergo controlled range of motion exercises.

Tape splint
A tape splint is useful for stabilizing fractures, luxations, and injuries of the pelvic limb, distal to the stifle, of small birds <250 grams. In order for the tape splint to provide rigid support, the tape must be in direct contact with the skin. All the feathers will need to be
plucked from the area to be bandaged. Depending on which part of the leg needs stabilization, the technique varies as described below. The tape must be applied in a spiral manner to avoid a tourniquet effect on the limb if swelling occurs. Tape splints applied to limbs that are swollen may become loose once the swelling resolves and will need to be reapplied or snugged down. Radiographic evaluation of bones can be performed through the tape splint negating the need to remove the splint for imaging. In general, a tape splint is left on for 4 weeks. Removal of the tape splint may cause discomfort and stress to the patient as often feather regrowth has occurred. Anesthesia or sedation of the patient may be necessary to facilitate removal which is performed by cutting the tape longitudinally and peeling it off the skin.

**Tracheal intubation**

Inhalant gas anesthetic agents are most commonly used for anesthetizing avian patients and induction may be performed using a mask or chamber. However, for all but the briefest anesthetic episodes, tracheal intubation should be performed once induction is complete in order to protect the bird’s airway and to provide for a means of assisted ventilation while the bird is under anesthesia. The glottis is positioned at the base of the tongue and is fairly easily visualized in non-psittacine birds. Parrots possess a large muscular tongue that, with the limited oral access due to the hooked beak, prevents easy visualization of the glottal opening. In birds where the glottal opening is hard to see, the beak may be held open with gauze strips and a light source (transilluminator) can be used to push the tongue ventrally while illuminating the back of the throat. The tongue may also be grasped with atraumatic forceps and extended to elevate the glottal area to facilitate visualization. The avian trachea has solid cartilaginous rings that do not stretch to accommodate endotracheal tubes that are larger in diameter than the trachea. Additionally, the tracheal lumen narrows as the trachea extends from the glottis into the neck. A non-cuffed, small diameter (2.0-4.5 I.D.) endotracheal tube is used when intubating birds. In some cases, a red-rubber catheter or IV catheter that has been modified into an endotracheal tube may be needed. If a cuffed endotracheal tube is used, the cuff should be deflated as much as possible and should not be inflated once the tube is in place. Forcing an endotracheal tube into the trachea when the tube is too large or inflating the cuff of a tube may result in tracheal stricture and subsequent respiratory difficulty.

**Air sac cannulization**

When a bird is presented with severe upper airway obstruction caused by foreign bodies, exudate, or other problems, an airway must rapidly be established. Also, some small birds undergoing surgery of the oral cavity may require access to the respiratory tract that bypasses the upper airways. In these situations, birds can be ventilated through their air sacs. These should be maintained only short term since the air is bypassing the normal defense mechanisms of the upper airways. However, this technique allows adequate time to properly treat the upper airway problems.

**Nasal flush**

A nasal flush may be performed as a diagnostic tool, allowing for cytologic evaluation or culture of the fluid collected from the wash. Conversely, a nasal flush may be performed as a therapeutic technique to clear the nares of debris or to administer medications into the nares and infraorbital sinuses. Minimal pressure should be used when performing a nasal flush and the volume infused corresponds to the size of the patient. Recommended volumes for small birds (budgie, cockatiel, lovebird) are 1-3 ml, for medium-size birds (Amazon parrot, AGP) are 4-10 ml, and for large birds (macaws, cockatoos) are 10-15 ml.

**Sinus wash**

A sinus wash, or flush, is used as a means to acquire diagnostic samples or to provide treatment to birds suffering from sinusitis of the infraorbital sinuses. Samples collected may be used for cytologic evaluation and microbial culture. The wash may assist in the removal of sinus exudate and may be used to infuse medication. Due to the proximity of the needle’s insertion to the eye, general anesthesia or sedation may be needed to prevent the bird from struggling and inadvertently causing trauma to the surrounding tissue. Some bleeding may result from a sinus wash in birds with significantly inflamed tissues. Only non-irritating solutions should be used to perform a sinus wash. Aminoglycoside antibiotics (using a 1 mg amikacin or gentamicin/1 ml sterile saline solution) are often used to treat bacterial sinusitis via a sinus wash. The infraorbital sinus is more easily accessed if the bird’s mouth is open. An oral speculum may be used if the sinus wash is being performed on an awake bird.

**References**

Bird Basics: Procedure and Technique Manual, University of Illinois College of Veterinary Medicine, Zoological Medicine, 2015.
Modern medicine offers many options for the diagnosis and treatment of disease conditions in pet birds. Proper sampling and sample handling will ensure optimal results. Additionally, a basic knowledge of diagnostic testing and normal parameters will allow for the prompt detection of anomalies, abnormalities, and disease states. The hallmarks of veterinary practice, a thorough physical exam, diagnostic testing, and the administration of quality care, apply to the care of avian species kept as pets. Many times, diagnostic testing is even more critical in these species than in domestic animal care because of their tendency to hide illness or injury until a critical stage. A thorough history and physical exam may provide clues as to the underlying problems but advanced diagnostic testing may be needed to obtain a definitive diagnosis and to develop a treatment plan. A basic understanding of diagnostic sample acquisition from avian species and test interpretation will allow the veterinarian to provide care for these species.

General principles of the avian exam
Prior to handling the bird, obtain a complete history including origin of bird, exposure to cage mates or other birds, diet and water consumption, cage environment, reproductive history, health history, appetite, activity, and information regarding the presenting complaint. Be sure to observe the bird in the cage. Look for evidence of illness, respiratory character, ambulation and ability to perch, and interest in the environment. A bird that is fluffed, or sleeping during an exam may be in critical condition. If at all possible, owners should be instructed to present the bird’s normal cage and not to clean the cage prior to the visit. Examine the bird’s droppings. Make sure to check the feces, urine, and urates individually. Feces may be either brown or green but should be formed. Urates are generally chalky white and green urates may indicate hepatic disease. Many owners mistake polyuria for diarrhea. Check for urine staining from previous droppings in the cage to evaluate the normal amount of urine being produced. Many birds will become polyuric when stressed so droppings produced at the time of exam may be an inaccurate example. Be prepared for your exam and diagnostic sampling prior to handling the bird. This will minimize the duration of restraint and stress for the bird.

The physical exam should begin with the head including eyes, ears, nares (nostrils), and oral cavity. A pair of forceps can be used to open the mouths of smaller birds, while an avian oral speculum can be used for larger birds. The crop should be palpated for contents or thickening of the wall. The pectoral musculature should also be assessed to determine body condition (1-5 scale). Ideally, the muscles should curve gently away from the keel. The coelom should be palpated for obvious mass effects. The vent (opening to the cloaca) should be visualized to check for pasting of fecal material or other problems. The extremities should be checked for wounds or asymmetry. Hydration and perfusion may be assessed by viewing the basilic vein which runs across the ventral aspect of each elbow, looking at eye turgor, and assessing the oral mucosa. The plumage should be checked for missing or damaged feathers and the integument checked for abnormalities. Auscultation should be performed to assess heart, lungs, tracheal and air sac health. An accurate weight must be obtained.

Hematology
A complete blood count (CBC) and plasma chemistry are essential when evaluating a bird’s health status. Both of these tests can be performed on a sample collected in a lithium heparin (green top) collection tube which preserves avian cells well and allows for greater plasma volume recovery. EDTA (purple top) collection tubes may also be used but are less optimal because of sample erythrocyte hemolysis in some species. Microtainers (BD manufacturer) are useful when working with small collection volumes to prevent sample dilution. A CBC should include measurement of the total protein (TP), packed cell volume (PCV), and evaluation of the blood cells including thrombocytes, erythrocytes, and leukocytes. Avian erythrocytes and thrombocytes are nucleated which necessitates manual techniques for cell counting. After 24 hours, samples maintained in all anticoagulant types (EDTA, Heparin and Citrate) decrease in sample quality. Therefore blood smears should be made before placing the sample in the collection tubes or very shortly thereafter.

Several sites are available on the avian patient for venipuncture; jugular, basilic (ulnar) and medial metatarsal veins. The jugular vein is easily visualized on the right side of the neck in an apteryia and is less prone to the development of a post venipuncture hematoma. Restraining for venipuncture of the right jugular vein can be achieved using one or two people. If the phlebotomist is going to restrain, the method used for small birds, then the head is restrained between the index and middle fingers of the left hand, with the bird in left lateral recumbency. The feathers are wetted and parted over the naturally bare area of skin over the right neck to expose the vein. The thumb pushes the right wing caudally and holds off the jugular vein at the thoracic inlet. A syringe with a 25-27 gauge needle is inserted either caudally or cranially into the vessel.

If the patient is a larger bird, two people will be required in order to successfully draw blood. The assistant will restrain the bird’s head with their left hand, while holding it in left lateral recumbency. Their right hand will hold the feet and tip of the right wing. The
phlebotomist will hold off the jugular vein with their left hand and, after wetting the feathers to expose the vein, will enter the vein using a 25-27 needle and syringe as described above.

The total blood volume of a bird is approximately 10% of its body weight (6-12 ml/100g BW). Normal healthy birds can lose 1% of their total blood volume without problems. Therefore, a 30 g budgerigar has an estimated total blood volume of 3 ml of which 0.3 ml could be collected safely for hematology. Pressure should be applied to the site after withdrawing the needle for approximately one minute to prevent hematoma formation. Blood should be collected for a CBC and chemistry profile. If the clinic is not equipped to perform these tests, a PCV, TP, blood glucose, and blood smear should be prepared. An estimated white blood cell count (WBC), cell differential, and evaluation of cell morphology can be completed from a well prepared blood smear. Reference ranges for a variety of species are published.

**Complete blood count**

Performing an avian CBC requires a time-intensive process due to the presence of a nucleus in all blood cell types, including thrombocytes and erythrocytes. Some in-house labs and several commercial labs are able to provide this service but access to results may be delayed. In cases where an immediate assessment of the leukogram is necessary for diagnosis and treatment planning, an estimated leukogram count is useful. An estimated total WBC count may be obtained by averaging the leukocytes observed in ten high power fields and multiplying the average by 2000.

**Erythrocytes**

The PCV and TP values are obtained routinely. Caged birds generally have a PCV of 35-55%. A PCV of <35% indicates anemia and a PCV of >55% indicates dehydration or polycythemia. Normal birds show a 1-5% erythrocyte polychromasia, but this increases with regenerative anemia. Reticulocytes represent <10% of total RBC counts in most normal adult birds. Immature red blood cells (RBCs) are rounder and show polychromatophilic cytoplasm that is more basophilic in color. Polychromatic cells are reported either using a 1-4 number code with 1 being normal or as slight, moderate, or maximum. Anisocytosis (variable cell size) occurs normally in peripheral avian RBCs. RBCs may vary from round to elongated or irregular in shape. The nucleus can vary in its location in the cell and can have indentations, constrictions or protrusions.

**Leukocytes**

- Smudge cells commonly seen in avian blood films are artifacts of slide preparation. A large number of smudge cells affects the heterophil, eosinophil and basophil counts as they generally represent granulocytes.
- Heterophils are the most common granulocyte in avian blood. These are round cells with colorless cytoplasm containing eosinophilic rod-shaped granules. The nucleus is lobed, containing course clumped chromatin that stains purple, but is often partially hidden by the cytoplasmic granules. Heterophils play a role similar to the neutrophil in mammals and will demonstrate toxic changes in the face of inflammation. Toxicity is scored from +1-4 based on the following criteria; basophilic cytoplasmic granules, nuclear hyper segmentation (>3 lobes), cytoplasmic vacuolization and basophilic cytoplasmic color. Band heterophils can be seen and demonstrate a lack of nuclear segmentation.
- Lymphocytes are very common in avian species, often representing half of the total WBC count. They occur in 3 sizes (small, medium, large) with most lymphocytes in peripheral blood being small or medium. These are typically round cells with a round centrally located nucleus. The nucleus occasionally is indented slightly. The nuclear chromatin is densely clumped or reticulated and there is a high nuclear to cytoplasmic ratio. Reactive lymphocytes occur frequently in sick avian patients and are identified by their darker blue cytoplasm nuclear changes and prominent nucleoli, scalloping of cytoplasm and cytoplasmic vacuoles.
- Eosinophils are typically round with uniform clear, pale blue cytoplasm that contains round eosinophilic granules. These cells are typically less numerous than heterophils in the peripheral blood. Cursory examination of the smear should allow the viewer to distinguish between the two cell types. The nucleus is lobed and stains blue. Toxic changes generally do not occur.
- Basophils are round cells with round centrally located nucleus. The light blue staining nucleus is often hidden by deeply basophilic cytoplasmic granules. Toxic changes don’t normally occur.
- Monocytes are large cells with irregular shape. They contain round to bi-lobed nuclei with lacelike reticular chromatin. The chromatin can be clumped but will be small in number. Vacuoles may be visualized in the cytoplasm.
- Thrombocytes are the equivalent to the mammalian platelet and are nucleated. Thrombocytes tend to clump together. They are oval cells like the RBCs but are smaller and rounder than mature RBCs. The cytoplasm is clear and scant. The average number seen on an avian blood smear is 5 per oil field and are reported as adequate, increased, or decreased.

**Plasma biochemistry**

Avian plasma chemistry evaluation should include glucose, total protein (TP), albumin, aspartate aminotransferase (AST), creatinine kinase (CK), uric acid (UA), amylase, and electrolytes including calcium and phosphorus. Alanine aminotransferase (ALT), gamma glutamyl transferase (GGT), and alkaline phosphatase (ALP) are non-specific for liver in avian patients and offer little valuable
Cytological evaluation is very useful in avian medicine and is considered a standard part of healthy and ill bird examinations. Avian cytology focuses on evaluating active infection vs. food contamination, the presence and type of any cells (inflammatory, squamous epithelial), and the type of bacteria (gram-positive rods and cocci, gram-negative rods), the presence of morphologically distinct bacteria (Clostridium, spiral-type bacteria), the presence of yeast organisms (Candida spp., avian gastric yeast) and whether or not they are budding (indicative of active infection vs. food contamination), the presence and type of any cells (inflammatory, squamous epithelial). All these parameters offer clues as to the health of the patient and characterize the nature of any problems noted on physical exam. Cytology of tissue samples may be stained with a variety of stains to help detect the presence of specific etiological causes. Sample acquisition for cytological evaluation can be obtained through abdominocentesis, endoscopic biopsies, bone marrow aspirates, choanal and cloacal (or fecal) swabs, nasal or sinus flushes, joint aspirates, tracheal washes, and crop washes. Skin scrapes and skin impression smears are also evaluated via cytology and are useful diagnostic tools for dermatology of avian species.

Fecal wet mounts are useful for detecting motile bacteria (Campylobacter) or protozoal parasitism (Hexamita, Giardia). Avian gastric yeast is also best detected in this way. Fecal flotation for gastrointestinal parasitism detection is indicated for new birds, avairy birds, or outdoor birds.

**Cytology**

Cytological evaluation is very useful in avian medicine and is considered a standard part of healthy and ill bird examinations. Avian patients have predominately a gram positive bacterial population as their normal flora. Gram stain cytology can be performed on choanal swabs, fecal or cloacal swabs, crop wash fluid, vomitus or any other material when a gram negative bacterial infection is suspected. These same samples can and should be submitted for bacterial culture and antimicrobial sensitivity testing. When evaluating a gram stain several parameters should be noted; the amount of bacteria present (low to high), the percentage of different bacteria (gram-positive rods and cocci, gram-negative rods), the presence of morphologically distinct bacteria (Clostridium, spiral-type bacteria), the presence of yeast organisms (Candida spp., avian gastric yeast) and whether or not they are budding (indicative of active infection vs. food contamination), the presence and type of any cells (inflammatory, squamous epithelial). All these parameters offer clues as to the health of the patient and characterize the nature of any problems noted on physical exam. Cytology of tissues or samples may be stained with a variety of stains to help detect the presence of specific etiological causes. Sample acquisition for cytological evaluation can be obtained through abdominocentesis, endoscopic biopsies, bone marrow aspirates, choanal and cloacal (or fecal) swabs, nasal or sinus flushes, joint aspirates, tracheal washes, and crop washes. Skin scrapes and skin impression smears are also evaluated via cytology and are useful diagnostic tools for dermatology of avian species.

**Urine evaluation**

Avian dropping contain three portions; urine, urates, and feces. It is impossible to perform cystocentesis on a bird as they do not have urinary bladders. Urine can be evaluated however if collected from the droppings in a timely manner. If urine is needed for evaluation, plastic wrap or wax paper should be placed under the bird in the cage. As droppings occur, a syringe may be used to separate the clear liquid portion of the dropping which is the urine. The presence of bacteria in these samples should be expected as a result of some mixing with fecal material. Specific gravity in avian urine is usually between 1.005-1.020 g/µl and pH is 6.5-8.0. Very little protein should be in the urine as protein is excreted as urates in the droppings. Healthy birds will have little to no glucose in the urine.

**Cytological evaluation**

- **Glucose:** Birds have normally high glucose levels (300-500 mg/dL).
- **Total Protein/Albumin:** The TP is comprised of albumin and globulins and is measured in serum once fibrinogen has been removed in the blood clot. If plasma is used, total solids are measured and include fibrinogen as well as albumin and globulins. Albumin generally accounts for 1.0-2.5 g/dL of the TP (2.5-5 g/dL) with the remainder being globulins. Low TP may indicate malnutrition, acute hemorrhage or chronic intestinal parasitism. High TP may reflect dehydration, acute inflammation, chronic disease, or infection with antigen stimulation. Protein electrophoresis is useful to evaluate avian protein levels. Increases in alpha, beta, or gamma globulins indicate active inflammatory responses and can give the practitioner an idea of the chronicity of the inflammation.
- **AST (aspartate aminotransferase):** This enzyme is found in liver, skeletal muscle, heart, brain and kidney. Elevations may also occur with muscle catabolism. Pre-renal azotemia has little effect on UA level in contrast to renal and post-renal conditions. UA is a more sensitive parameter in birds to evaluate kidneys whereas elevations in phosphorus are the hallmark of renal disease in reptiles. Generally <400 U/L.
- **CK (creatine kinase):** CK is found in skeletal muscles, heart muscle, and brain. CK levels are used in birds to differentiate between liver and muscle cell damage. Generally <500 U/L.
- **Uric Acid:** Uric acid is useful in avian serum and should be submitted for renal health although it may be naturally elevated in carnivorous species, especially post-prandial. Elevations may also occur with muscle catabolism. Pre-renal azotemia has little effect on UA level in contrast to renal and post-renal conditions. UA is a more sensitive parameter in birds to evaluate kidneys whereas elevations in phosphorus are the hallmark of renal disease in reptiles. Generally <10 mg/dL, raptors < 20 mg/dL.
- **Amylase/Lipase:** Pancreatic enzymes. Amylase and lipase may be elevated with pancreatitis but is inconsistent in mammals. Amylase may be more sensitive in birds to detect pancreatitis with normal levels generally being <900 IU/L. Waterfowl have much higher amylase levels normally. Amylase can also be elevated with renal disease.
- **Calcium:** Species variation is very slight. Most species maintain a range of 8-12 mg/dL. Egg laying females can have very high calcium but most of this is highly protein bound. Total calcium is comprised of bound and unbound (free) calcium.
- **Phosphorus:** Most species maintain a range of 3-6 mg/dL. Elevations are seen most commonly in renal disease.
presence of hematuria may indicate primary kidney disease or cloacal lesions. Ketones, bilirubin, and urobilinogen are not expected. Amorphous urate crystals are a common finding in avian urine. Hemoglobin-like, granular, cellular and hyaline casts have been seen in avian urine. Casts are highly significant and are a clear indication of renal disease.

**Imaging**

Radiography is extremely useful for evaluating avian patients for orthopedic disease, respiratory disease, gastrointestinal disease, and urogenital disease. Barium or other contrast media may be used for ancillary studies. Two views are required to adequately evaluate a radiographic study of a bird, lateral and ventro-dorsal. General anesthesia may be necessary for optimal positioning. Radiographic lesions include changes in size, position, and opacity of structures.

- **Skeletal System** – The avian spine has a high degree of fusion and there is a general reduction of bones distally in the appendages. The most common lesions of the skeletal system are fractures. Fractures should be evaluated in the same way that those of mammals are evaluated. Two views are needed to fully evaluate the skeletal system.

- **Respiratory System** – The lungs do not inflate and they are fixed to the dorsum of the cranial coelomic cavity. The alveolar walls have a honeycomb appearance and are best evaluated on the lateral (L) view. On dorsoventral (DV) views, the lungs should appear as uniform soft tissue opacities in the cranial coelom bilateral to the heart. The air sacs should have an air opacity and the air sac membranes should not be visible. Air sac membrane thickening will appear as fine white linear opacities at the air sac margins. These are best noted on the DV view. The caudal thoracic air sac can be visualized caudal to the lungs on the L view.

- **Circulatory System** – The heart is visible ventrally resting against the sternum (keel) on the L view. The aorta and pulmonary artery are visible as they exit the base of the heart. The aorta can sometimes be followed as a soft tissue opacity caudally. The heart is centered in the cranial coelom on the DV view and forms an hourglass silhouette with the hepatic outline. Several bright soft tissue opacities are often visualized just cranial and adjacent to the heart, which represent large blood vessels.

- **Gastrointestinal System** – On both views, the liver is caudal to the heart. The hourglass silhouette that the liver makes with the heart can be used to assess hepatic size. Paired lines drawn from the shoulders to hips on each side should roughly encompass the width of the liver. A liver that extends beyond these borders may display hepatomegaly. On the L view, the liver is ventral and adjacent to the body wall. The crop is often visualized as a distension cranial to the thoracic inlet on both views. Food material may be seen as a mottled opacity. The esophagus can be traced on the L view from the crop, just dorsal to the trachea, to the level of the liver where it distends slightly as it becomes the proventriculus. Juvenile birds often have large proventriculi. The proventriculus narrows slightly at the caudal end, the isthmus, and enters the ventriculus which is visible in the mid-coelom caudal to the liver. Many times this organ is identifiable because it contains radio-opaque material such as grit, seed material, or other digesta. The ventriculus is displaced caudally in the case of hepatomegaly. The spleen of psittacine birds is spherical and may be visible just dorsal to the isthmus on the L view if splenomegaly is present. The sections of intestines are indistinguishable from each other until the colon is seen entering the cloaca caudally. The DV view is less desirable for evaluating the structures of the GI tract except for the ventriculus which often is readily identifiable. The tract is predominantly found on the left side and is superimposed over the liver, spleen, and reproductive tract.

- **Urogenital Systems** – The kidneys sit in the renal fossa along the dorsal body wall of the caudal coelom. The three lobes are often indistinguishable and the kidneys are superimposed on L view. Nephromegaly involving the cranial pole can be identified. Increased opacity of the kidneys may represent increased uric acid content. Female birds have one ovary and oviduct located on the left side. Developing ova in the ovary may appear as spherical opacities dorsal and caudal to the isthmus. This may displace the ventriculus ventrally. A male bird has paired testes which attach cranial and medial to the kidneys, just ventral to the dorsal spine.

**Endoscopy**

Endoscopic evaluation has become readily available for avian practitioners. The bird’s body is perfectly designed to fully utilize this tool for visualization and sample collection of many internal structures. The endoscope is inserted into the body and the air sac system eliminates the need to infuse air. Biopsies can be collected routinely. Care should be taken to avoid infusing fluids into the body cavity as this may cause serious complications. The practitioner should be familiar with avian anatomy as some organs are only visible from one side and a right or left approach will been to be chosen.

**References**


While house soiling is one of the most common behavioral complaints and reasons for relinquishment, abandonment, and euthanasia in cats, aggression is a close second. Cats are commonly aggressive to other cats in their homes, and they may also be aggressive to people in a number of contexts. Stranger-directed aggression is likely an under-reported type of aggression in cats. This behavior is important because aggression to strangers can negatively impact the human-animal bond, cause significant injuries to people and other animals, and create an unsafe situation for medical personnel and pet-sitters. Cats that are aggressive to strangers are likely to be aggressive during veterinary treatment.

Aggression to strangers can develop as a cat enters social maturity, but it can begin in kittenhood as well. Fear and territorial motivations are common. Cats over age 3 that develop aggression to strangers while in an otherwise stable home environment, must be medically evaluated for a number of diseases that can increase irritability (hyperthyroidism, hypertension, osteoarthritis, neoplasia, infectious agents, etc.). These behaviors can be prevented in some cats with early positive socialization and positive experiences with strangers throughout the cat’s life stages. It is important to note that “positive experiences” are defined by the individual cat. Owners and veterinarians who understand feline body language will have the best success at ensuring the cat’s exposures to strangers set the cat up for immediate and future success.

As with most behavioral disorders, multi-modal treatment is recommended and should include avoidance/safe management, environmental enrichment, and desensitization and counter-conditioning to triggers. In some cases, medication may be warranted either during trigger times, daily, or both.

Safe management is critical to prevent the cat from practicing and being reinforced by the behaviors as well as to prevent injuries to strangers. The easiest way to manage cats who are aggressive to strangers is to keep them from having access to strangers completely. This can generally be done by creating a safe zone (such as a bathroom, guest room, multi-level cat cage, or large exercise pen +/- top) for the cat and putting the cat there BEFORE guests arrive and only allowing the cat out of the safe zone after the strangers leave. If the cat will be confined in the same area as the guests (like in a cat cage), it is generally best to cover the confinement zone so that cat can’t see the visitors. It is also important to instruct visitors to completely ignore the cat (no petting, no talking, and no looking). Most cats can be taught to go to their safe zones when the doorbell rings within a few days to weeks if owners take a few minutes per day to work on the behavior with a rational behavior modification plan.

Unfortunately, management alone doesn’t help teach the animal new ways to respond. This means if emergency personnel, pet-sitters, baby-sitters, house keepers, etc. ever need to enter the home the cat must be confined over the long-term for safety. Unfortunately, in an emergency, many families will forget to confine the cat. Families may also feel trapped at home when they can’t find a pet-sitter.

Owners should be counseled not to handle cats that are in the midst of aggressive events because they may be severely injured. A thick blanket can be tossed on a cat in the midst of an aggressive event as can a glass of water or an upside down box. Shaken seltzer sprayed at the cat can be especially effective for stopping events in progress. There are emergency management techniques, not interventions for everyday use. Emergency items can be placed in potential trigger areas for easy access.

Because cats are acutely sensitive to being removed from their home environments, it is generally best for them to have in-home pet sitters when owners must leave. In order to make this safe, owners should experiment with confinement methods and potentially medications to reduce fear and aggression. If possible, the owners should get the help of a pet sitter who will be available over the long term. Then with the help of a rational behavior modification plan, the cat can be taught to tolerate or even “like” the pet sitter. If the cat is truly better when boarded away from home, this should be pursued instead.

Once owners know the cat’s warning signs and specific triggers (door bell, petting, moving from standing to sitting, loud voices, smell of other cats on the stranger, etc.) treatment can focus on desensitization and counterconditioning. As treatment progresses, some cats may graduate from strict confinement to wearing a leash and harness during exposures and eventually may be able to be “at large” with strangers who can follow instructions (generally to ignore the cat strictly).

Environmental enrichment is a critical component of keeping cats behaviorally and medically healthy. Cats should eat as many meals as possible from puzzle toys, have appropriate play sessions with owners for at least 7 minutes per day, have multiple scratching posts, and be provided with multiple, soft elevated areas for resting and hiding.

Medications such as SSRIs, benzodiazepines, trazodone, and gabapentin can be helpful for cats with stranger-directed aggression. SSRIs are best given daily, but the other meds could be used at trigger times only even for several days at a stretch if needed.

Supplements, diet change, and pheromone therapy can also be helpful for some of these patients and can be implemented concurrent with medication protocols.
Patients with stranger-directed aggression can improve significantly with treatment. But treatment for this problem is not inherently obvious to most clients. They generally need guidance from a veterinarian skilled in applied behavior analysis and knowledgeable about normal feline behavior as well as psychoactive medication use. Thankfully, veterinarians are very capable of learning and implementing appropriate treatment and keeping their cat patients out of the “dog house.”
House soiling can damage the human-animal bond and leave the animal vulnerable to re-homing, abuse, abandonment, neglect, and euthanasia. Therefore, veterinarians must understand the basics of coaching owners on house training.

House soiling is a normal behavior of dogs. Unlike cats, they are not naturally predisposed to use a bathroom facility humans readily provide without any actual training. Thankfully, most dogs are relatively easy to house train if owners follow instructions. Many young patients can be house trained in as little a few days to a few weeks. A variety of techniques are recommended for housetraining. However, many are irrationally punitive, some are outright abusive, and many are ineffective. Science-based strategies for house soiling are well-known.

Many young patients can improve in their house training in as little as a few days with an appropriate plan. Patients who have been living in soiled environments, have been urinating/defecating on common substrates found indoors, or who have unknown training backgrounds can be harder to house train. The majority of shelter dogs can be house trained in 4 weeks if the owners are provided with 5 minutes of counseling at the time of adoption.¹

Medical co-morbidities, especially those causing PU/PD, urinary or fecal incontinence, pain during positioning for elimination behaviors, or diarrhea can cause previously house trained dogs to begin house soiling or complicate attempts at house training.

In addition, dogs may urinate or defecate in the home for reasons unrelated to voiding the urinary bladder or bowels. For instance, dogs urine mark and even fecal mark in response to a variety of internal and external triggers. Co-morbid anxiety and panic disorders can make it virtually impossible for even house trained animals to control their urination and bowel movements.

It is critical medical and/or behavioral co-morbidities be assessed and treated in order to see improvements in house soiling behaviors. Physical examination, stool sample, CBC, chemistry, urinalysis, urine culture and sensitivity, abdominal radiography including the entire urinary tract and gastrointestinal tract may be needed. Contrast radiography may also be helpful if abnormal anatomy is suspected. Abdominal ultrasonography, diet trials, anal gland expression, and empirical treatment for parasites may also need to be considered. These diagnostics are especially important when previously house trained animals begin to house soil. However, even puppies may need medical diagnostics if they are not improving quickly with rational behavior modification.

Behavioral co-morbidities such as fear, panic, anxiety, cognitive dysfunction, submissive urination, excitement urination, and urine or fecal marking can often be assessed with verbal history AND a video of the dog in the problem context (whenever possible and safe).

Uncomplicated house soiling behaviors such as those often exhibited by puppies, have a good prognosis. Treatment includes management to prevent accidents, improved access to appropriate elimination areas, and reinforcement of urination and defecation in the desired location.

Puppies and dogs with unknown house training capabilities should either be confined in a safe zone or attached to an adult with a leash whenever they are not in the elimination area. Successful confinement zones are secure and comforting for the animal. They include toys, soft resting areas, and flooring that is easily cleaned.

Crating when unsupervised is a traditional and effective management strategy for puppies and dogs who can tolerate this type of confinement. Crates should be large enough for the animal to stand comfortably and to turn around. Soft resting substrates, such as beds, can be provided to cover the crate floor. If the puppy or dog urinates or defecates in this area AND the animal was not confined so long that elimination was inevitable, the size of the confinement area and the animal’s level of distress during confinement must be assessed. Animals that are fearful of confinement must be taught to be comfortable with confinement using positive reinforcement training. In the meantime, appropriate use of trigger time medications, pheromones and supplements (although not proven effective for this purpose) should be considered.

Animals who do not appear to have an inclination towards keeping their “dens” clean or whose owners cannot commit to providing time in an appropriate elimination area every 2 hours (at the start) must be confined in larger areas. These areas need to provide distance between an “appropriate” elimination area and a resting area. When providing and indoor substrate and location for elimination, it is ideal to provide the same substrate as outdoors or a substrate that is as close as possible. This decreases confusion for the dog as he/she learns about preferred areas for elimination.

If the animal is forced to eliminate on common household surfaces or to eliminate on himself or his bed, it is likely that house training will be prolonged. In addition, it is unethical to force an animal to rest in his/her excrement.

A schedule is critical to house training success. Dogs most often need to urinate and/or defecate after sleep, play, eating/drinking. A general rule of thumb at the beginning of house training is to take the animal to the preferred toileting area at least every 2 hours, after play, sleep, eating or drinking, and immediately before bed and after waking in the morning. He/she should also be taken to the

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toileting area at any other time pre-elimination signals are given (such as circling, increased distraction from play, sniffing the ground, going to the door, whining, increased pacing/restlessness). The animal should be taken directly to the preferred toileting area (on leash or in arms if needed) by an adult. Once there, the animal should be allowed to sniff a several foot radius in the toileting area. Immediately (within 1-2 seconds) after the animal urinates or defecates, the animal should be given a powerful reinforcer (for many dogs this is a special food, but it could also be a short burst of play or access to the rest of the environment for those dogs who are not food motivated). It is best to make sure the animal urinates/defecates before you instigate play or take the dog on true walk. If the animal spends 10-15 min in the elimination area without eliminating, he/she can be taken indoors and either confined or strictly observed via leashing to an adult (sometimes called “umbilical cord training”). When the first possible pre-elimination behavior is exhibited the animal can be taken back to the toileting area and the process can be repeated. It is important to note, if and owner takes a dog outside to eliminate and then plays or takes the dog on a walk before the animal eliminates, he/she may accidentally teach the dog to ask to go out for play or walks rather than elimination. For those dogs who truly enjoy being outside, it is important to remember to allow them to stay outside for at least 5-10 minutes after they urinate or defecate. IF an owner takes them inside immediately after elimination, these dogs will often learn to hold urine or feces as long as possible so they can stay outside. This can be a problem in inclement weather or when an owner is in a hurry.

Punishment is contraindicated for animals who house soil. The typical “rub his nose in it” strategy is unnecessary and can cause dogs to fear handling as well as urinating/defecating in front of their owners. It is important dogs feel comfortable eliminating in the presence of people for medical and management reasons. Need a urine sample? Good luck with that if you’ve been punishing the dog for urinating in your presence. Need to take your dog out to eliminate on leash? You may have a struggle if you’ve accidentally taught your dog to fear urinating in front of you. In addition, dogs who are punished for house soiling often learn to urinate or defecate when the owner isn’t watching or in more hidden areas of the home. This makes house training more difficult in the end. This is why the old adage “hit the dog with a newspaper” has become “hit yourself with a newspaper.” It is the owner’s responsibility to provide the dog with adequate opportunity to urinate and defecate in preferred areas. If the owner has broken this contract by allowing the dog access to “unsafe” areas when he/she isn’t supervised or if the owner has not taken the dog out frequently enough to keep the bladder and bowels empty, there is only a person, not a dog, to blame.

References
Redirected aggression is agonistic behavior directed towards an available target (victim) who is not the primary trigger. This often happens because the primary target is inaccessible to the aggressor.

### Clinical signs

**Cats**
- Growling, lip licking, tail thrashing, hissing, swatting, striking, screaming, yowling, piloerection, scratching, and/or biting

**Dogs**
- Barking, growling, snarling, snapping, and/or biting (not in play)

Triggers are often unfamiliar animals, scents, or sounds. Available targets are generally owners and familiar/household animals. These events are more common in cats who have a history of noise or other fears.  

### Prevention

Proactive socialization during the puppy and kitten vaccination series and afterwards is strongly recommended to help prevent animals from becoming pathologically frightened by common environmental triggers resulting in redirected aggressive events.

### Injuries

Victims can be severely injured. Affected animals are at high risk of euthanasia, abuse, abandonment, neglect, re-homing, or surrender to a shelter.

### Duration

Variable. Onset is often sudden. In dogs, events often stop quickly after being triggered (within 60 seconds) and behavioral arousal may or may not stay elevated for hours or days depending on the individual dog’s behavioral sensitivity and co-morbidities. In cats, events are often acute to the eyes of owners. However, history taking often, but not always, reveals an identifiable trigger and escalation pattern before the aggressive outburst. Once triggered, cats can stay behaviorally aroused and even potentially dangerous for several days.

Emergency stabilization and includes low-stress removal of the animal from the inciting trigger and the available target of the aggression until controlled introductions can be safely initiated. Cats exhibiting this behavior often benefit from a “cool down” period in a safe zone such as a bathroom, guest room, basement, or covered multilevel cat cage. This space should be quiet, have food, water, low lighting, toys, Feliway and/or Feliway Multicat, the cat’s preferred litterbox and scratching materials. In susceptible cats, safe zones are often kept ready for use and may be utilized on a daily basis as each cat’s individual core living area. For dogs, separating the dog and the target by baby gates or by rotating living areas may be sufficient.

Safe introductions require dogs who have bitten or threatened to bite be happy wearing leashes, body/head harnesses, and basket muzzles (see www.muzzleupproject.com for information). Cats need to be taught to stay happily in multi-level cat cages and/or to wear body harnesses and leashes. Animals can be taught to enjoy placement and wearing of these tools through positive reinforcement training.

Behavior modification includes desensitization and counterconditioning to the target, environmental enrichment, assessment/improvement of the species-specific basic needs plan, desensitization and counter-conditioning to the trigger (if identifiable), relaxation work, positive reinforcement training for specific cued behaviors (Watch Me, Go To Room/Mat, U-turn, etc.) and treatment of any co-morbid behavioral pathologies.

Medications can be very helpful, especially in cases where the behavior is frequent, injurious, and/or complicated by medical or behavioral co-morbidities. SSRIs and TCA’s are frequently prescribed. Trigger time medications such as clonidine, trazodone, and gabapentin may also be useful. Benzodiazepines are controversial for use in cases of aggression, however, in the author’s experience they can be quite helpful, especially early in treatment for cats with this disorder.

Animals with re-directed aggression can improve with appropriate, efficient, and pro-active treatment. Without treatment, redirected aggression can cause severe injuries, cause family relationships to decompensate significantly, and lead to the patient’s death. In order to help affected families, a behavior consult should be performed to create a multi-modal action plan with structured follow-up.

### References

Why should a veterinarian know the basics of treating unruly behaviors in dogs?
Some dogs have serious behavioral problems that pose safety and welfare issues for their families. But even very common, easily corrected unruly behaviors (jumping up, pulling on leash, barking, etc.) can result in dissolution of the human-animal bond and lead to euthanasia or surrender. A 1998 study by Dodman and Patronek estimated that approximately 224,000 behavioral euthanasias were performed and that many veterinarians didn’t feel comfortable performing behavioral euthanasias. However, it also showed that many veterinarians don’t ask about behavioral issues routinely and are uncomfortable handling behavioral them.

An unruly behavior is a normal dog behavior that is not preferred by most human households. These behaviors are encouraged by the inadvertent application of learning theory and lack of exercise, social structure, and enrichment. Unruly behaviors are a common source of discontent amongst owners, but they can also be a great opportunity to help bond a client to your practice and improve the quality of life for your patients. Inefficient attempts at controlling unruly behaviors can leave owners frustrated and their dogs vulnerable to inappropriate attempts at correcting these behaviors. Many trainers market themselves as capable of improving unruly behaviors. Certainly, they should be able to. However, because animal training is a completely unregulated industry, it is vital that owners start with easy activities so that the dogs don’t get too frustrated to engage. Then they can continue to target games to the skill level of their dogs, gradually increasing the level of difficulty. Social structure can be improved using a program in which owners are required to intensify.

Knowing how dogs learn
Dogs learn according to the same rules as other species. The most frequently recommended learning schemes use negative punishment, positive reinforcement, and negative reinforcement. Positive punishment is rarely considered a reasonable or ethical first-line treatment for behavioral problems. Negative punishment is the removal of something the dog wants in order to decrease the likelihood that the target behavior will occur in the future. For example, an owner turns away or leaves the room when a dog jumps up in order to decrease future jump up events. Positive reinforcement is the addition of something the dog wants so as to increase the
likelyhood that the target behavior will occur in the future. For example, if an owner notices that his/her dog is resting quietly during
dinnertime, he/she may toss a small piece of food to the dog in order to increase the likelihood that that the resting behavior will occur
during future dinners. Negative reinforcement is the removal of something the dog finds aversive as soon as the target behavior
occurs. This removal increases the likelihood that the target behavior will occur in the future. For instance, a properly handled head
halter will apply pressure over the nose when the dog is pulling. This pressure over the nose will immediately release when the leash
is loose, thereby increasing the likelihood of loose-leash walking in the future. Positive punishment is the application of an aversive
stimulus in order to decrease the likelihood that the targeted behavior will occur in the future. For instance, if a dog jumps up to greet,
and the owner wants to decrease this behavior using positive punishment, the owner might pinch the dog’s toes when the dog jumps
up.

Common unruly behaviors

Attention-seeking behaviors
Many so-called “unruly” behaviors are really attention-seeking behaviors (pawing, licking, barking, nudging, mounting,
destructiveness in the owner’s presence, theft, etc.). They often develop from normal dog behaviors when people accidentally
reinforce them, usually intermittently. The history of intermittent reinforcement can make these behaviors quite resistant to extinction
over time. Attention-seeking behaviors are almost always treated by removing attention immediately and consistently every time the
dog performs the behavior (for instance, leaving the room if the dog steals an item, leaving the room if the dog jumps up, etc.). In
addition, the owner is encouraged to proactively pay attention to the dog when it is performing acceptable activities (resting, playing
with his/her own toys, greeting with all four feet on the floor, etc.).

Mounting
Mounting behaviors can be part of normal play. Other causes for mounting include attention-seeking, social jockeying, and sexual
motivations. Mounting can be a displacement behavior in some dogs. Treatment includes assessing whether the behavior is
problematic or not (for instance, some mounting between dogs is normal and may not be a problem). Use of a previously learned cue
(such as touch or coming when called) to move the dog away from situations that often stimulate mounting can be very helpful.
Controlled behavior modification sessions can also lead to improvements. For instance, a dog that mounts visitors could be taught to
play fetch with them or chew on a special toy while they are present. Attention-seeking mounting can be decreased by completely
ignoring the dog or moving the dog to a time-out for a few minutes and making sure to reward at least 10 appropriate behaviors each
hour. Sexual motivations for mounting can be improved by neutering in many cases.

Jumping up
Jumping up is a normal behavior. It is encouraged by normal human behaviors such as patting the chest, petting the dog when he/she
jumps up, and hugging the dog. Since many dogs actively want to investigate or lick faces of other dogs and humans, they may jump
up to do so unless people lean down. Many people inappropriately use punishment while intermittently and accidentally rewarding
this behavior. For instance, a person may knee a dog in the chest when he/she jumps up and the person is wearing work clothes, but
encourage the dog to jump up at other times. Jumping up is treated simply by turning away from the dog or leaving the room when
he/she jumps up and turning around/returning and paying attention to the dog as soon as all four feet are on the floor. More structured
training can also be helpful (such as teaching the dog to sit for all interactions including greetings).

Pulling on leash
Teaching a dog to walk on a loose leash is very challenging for most families. It requires a significant amount of patience and impulse
control for both the dog and the walker. It is very important that handlers remember that, in general, dogs walk much faster than
people. Walking at a human pace can be quite frustrating and unnatural for them. Like any unnatural behavior pattern, walking on a
loose leash takes more time to learn and requires a higher rate of reinforcement than easier, more natural behaviors.

Many devices are purported to be useful for stopping pulling. While a skilled or very patient handler can teach a dog to walk on a
loose leash with a regular leash and buckle collar, most people reach for some type of walking tool designed to improve control.
Choke, prong, and electric collars are almost uniformly inappropriate for this type of work, since the pain they cause can make dogs
fearful of stimuli that are associated with the discomfort. Head halters, front-attaching body harnesses, and body harnesses that
tighten somewhat around the barrel of the chest can all be helpful aids while working on loose leash walking. There are multiple
methods for teaching loose leash walking. In general, they focus on various ways to reinforce walking beside the owner (such as high-
frequency, small treats given beside the owner’s leg or clicking and treating every time the dog is within the range that the handler
finds acceptable). When the dog pulls, many methods encourage the handler to stop the walk completely or turn in the opposite
direction until the dog comes back to the handler.

Mouthing
Mouthing is a common behavior of puppies that can extend into adulthood if the owner doesn’t respond appropriately to it. Many
different methods of discouraging mouthing behavior are reasonable and some can be used in combination. A common method for
teaching dogs not to mouth people or to attenuate their bite pressure is to end all games consistently when mouthing of a person
occurs. One way to achieve this is to keep the dog in a confinement zone even during play (such as one room or an exercise pen). If
the dog mouths, the person immediately gets up and leaves the dog for at least several seconds (or until the dog is doing a behavior that the owner wants to reward by returning). Another option is to take the dog to a previously determined time-out spot and ignore him/her until the dog is performing a behavior that the owner wants to reward by returning. Some people may benefit from using taste aversion substances on their hands or clothes as they initiate this work, especially if the dog is large or bites are painful. Used alone, taste aversion substances are unlikely to solve the problem in very mouthy dogs. Dogs that are very mouthy can also benefit from being taught to carry items in their mouth during times when mouthing is a problem. Creating other targets for mouthing behavior can be helpful (for instance, controlled tug games). Muzzles can be utilized when trained appropriately for severe, intractable, or unpredictable situations.

**Destructive behavior**

Destructive behavior is often a consequence of normal exploratory behavior, especially in juvenile and adolescent dogs. This behavior can be attenuated by providing adequate stimulation in the form of rotating food-dispensing puzzles and other toys/games in combination with trained confinement and appropriate exercise. Destruction of toys is a normal part of dog behavior. Destruction of stolen items can occur merely due to their novelty. Targeting of stolen items can be a learned behavior in some dogs; an intelligent dog quickly discovers that grabbing eyeglasses off of the coffee table increases owner interaction much more quickly than playing with that same old rope toy.

**Vocalizing when confined**

It isn’t natural for dogs to be confined for long periods. In general, teaching a dog to be crated should be a gradual process during which the dog learns that very special things happen in the crate, that there is no need to panic in the crate, and that vocalizing, scratching, and so on are ineffective at getting the dog out of the crate. Beginning crate training often starts with teaching the dog to go into and out of the crate on cue. Gradually the door can be closed behind the dog for longer and longer periods with the owner either leaving a long-lasting food-dispensing toy inside the crate or making a commitment to reinforcing the dog by hand intermittently while the dog is in the crate.

**Excessive barking**

In general, barking is a normal behavior that is frequently accidentally reinforced by humans. There are many motivations for barking including attention-seeking, play-related, fear/threat-aversion, behavioral arousal, cognitive problems, distress or anxiety, etc. Excessive barking must be treated on a case-by-case basis, taking potential motivations into account. In general, attention-seeking barking should never be rewarded. Instead, the owner should focus on paying attention to the dog when he/she is quiet. For instance, a family whose dog barks for food while they are eating could use a combination of confinement and rewards for quiet behaviors (tossing treats, using an automatic food dispenser, or providing the dog with a long lasting food-dispensing toy).

**References**

Thunderstorm phobia is a behavioral disorder commonly seen in adult dogs. It causes significant amounts of suffering for veterinary patients and the families who love them, and without appropriate treatment it can worsen with time. This negatively impacts the patient’s overall welfare and increases the risk of the patient being abandoned, abused, neglected, rehomed, or euthanized. Undertreated thunderstorm phobia is a known, yet largely preventable, cause of death in dogs.

While some dogs will be triggered only as the thunder of a storm starts and recover as soon as it ends, many will experience suffering that expands outside of the weather event. Especially upon repeated exposure, certain patients will begin to experience the symptoms of thunderstorm phobia earlier and earlier in the process of the weather event, and they may also take longer and longer to recover. Occasionally, a patient may hide (for instance, under the bed) and resist removal attempts for several days. Common triggers include thunder, lightning, rain, wind, and changes in barometric pressure. However, many dog will also learn to associate flashing lights (not associated with storms), darkening skies/clouds, owner pre-storm routines, non-thunder noises (sirens, garage doors opening or closing, fluttering flags, etc.) with the possibility of an oncoming storm. These patients are exhibiting full blown anxiety disorders triggered by storms.

Symptoms include, but aren’t limited to: pacing, panting, hiding, destruction (often focused on “escape” such as going through windows, biting door frames, scratching at barriers used for confinement, etc.), anorexia, trembling/shaking, inability to move (catatonia). Many patients with thunderstorm phobia will crawl on owners and seem to be soliciting petting or holding. However, video assessments of these patients often reveals the patient is not comforted by physical contact. Traumatic injuries, especially during escape or destructive behaviors are common. They include, but aren’t limited to, broken claws (especially on the front feet), tooth damage/breakage (especially of the canines), high rise injuries from jumping out of windows, hit-by-car injuries due to bolting behaviors, lacerations of the paws, face, and legs, etc. It is worth noting for those patients for whom hiding and withdrawal is a primary clinical sign, this diagnosis is likely to be missed and it’s severity under-estimated.

In severe cases, the patient’s disorder may worsen to the point where a generalized anxiety disorder including generalized hypervigilance, excessive startle, persistent environmental scanning develops. These cases warrant immediate referral to a local veterinary behaviorist or a telemedicine consult between the primary care veterinarian and a veterinary behaviorist if there is no local option.

Definitive diagnosis is often easiest when video is available for review. However, if owners have witnessed behaviors consistent with the disorder and the behaviors are not present when storms are absent, thunderstorm phobia can be presumptively diagnosed. At the firs

Separation anxiety, noise phobia, and thunderstorm phobia are frequently co-morbid. In addition, many dogs with thunderstorm phobia have other fears, phobias, impulse control disorders, and/or anxieties. When the thunderstorm phobia is triggered it can worsen other behavioral pathologies significantly causing the patient to rapidly decompensate and subsequently increasing the mortality of the disorder.

Multimodal, immediate therapy is preferred. Proactive behavior modification for teaching the animal new coping strategies, event management protocols, anti-anxiety supplements, pheromones, tools, and psychoactive medications are often used together to promote remission of this anxiety and panic disorder.

Improving any behavioral disorder begins with resolving deficiencies in basic, species-specific total health management. Adequate water, food, shelter, play, exercise in non-threatening environments, social interactions with preferred associates (keep in mind, some dogs strongly prefer interactions with humans to interacting with other dogs), daily reward based training, and proactive treatment of any medical conditions must be addressed.

Once there is a plan in place for these basic needs, families need help keeping the animal feeling safe. The learner (AKA the dog) tells us what feels “safe” for him by his behavior. Is he willing to eat, play, and interact in his usual way with the environment and his family? If so, he is likely feeling safe. If even one of these changes negatively (decreased appetite for food in a normally food-motivated dog, for instance), then either the outside environment or the microenvironment (neurotransmitters and other physiological cascades) need to be supported to help the patient feel safe.

These dogs need a “safe” place to rest during storms or perceived weather events. Safe zones are defined by the patient. Ideally, a safe zone (AKA storm bunker) is a windowless room or a room with curtains/blinds. This room is on the inside of the home floorplan or in the basement when possible. Many dogs choose closets, bathrooms, or crates for their storm bunkers when given the freedom to choose. Classical music or white noise can help drown some outside noises.
All dogs with thunderstorm phobia deserve to have their suffering relieved through the pro-active and rational use of medications. Trigger time medications, such as clonidine, trazodone, and many different benzodiazepines can be exceptionally helpful for these patients. For patients who are triggering outside of specific weather events, daily SSRI or TCA support should be considered. Trigger time and daily medications are often combined in affected patients in order to improve speed of solid recovery. In one study, patients who were treated with clomipramine and alprazolam during a thunderstorm season maintained their improvements into the next thunderstorm season. This indicates that medication in these patients is not only a bandage for acute events, but also helps the brain pathology heal.

Behavior modification should be worked on at least 5 minutes per day (ONLY on non-storm days). It includes desensitization and counter-conditioning to each patient’s individual triggers and relaxation coaching that includes the storm bunker. For practitioners new to behavior or without the time to do in-depth behavioral counseling, a step in the right direction would be to recommend any of a few different noise desensitization and counterconditioning audio packages that include structured behavior modification plans. In addition, a relaxation and massage work can improve the patient’s baseline anxiety levels and increase his/her ability to tolerate trigger situations in the long term.

Specific anti-anxiety tools such as body wraps, shirts, capes, caps, goggles, head phones, and ear plugs are available to help ameliorate trigger intensity. Efficacy is patient dependent. However, one study showed significant improvements in patients wearing a body wrap during thunderstorms.

Pheromone therapy may be supportive in some patients. A variety of supplements can also be tried, but these should not be considered cornerstones of treatment for thunderstorm phobia. Supplements should be used in conjunction with psychoactive medications, behavior modification, and management for the best outcome. All supplements should be sourced from viable veterinary-specific providers who have researched the supplements they sell, assure consistency of product, or at least are sensitive to and reaching out for veterinary insight, criticism, and support. Your local board-certified veterinary behaviorist can guide you as to his/her anecdotal experience for situations where higher power evidence is lacking.

Symptoms of thunderstorm phobia can be significantly improved in most veterinary patients when treatment is immediate, proactive, and multi-modal. In addition, improvements can carry over into future thunderstorm seasons with certain protocols.

References
Aggression to owners is a common problem in many species of pets and it negatively impacts the human-animal bond. It can lead to relinquishment, abandonment, neglect, abuse, and euthanasia. Thankfully it can be improved significantly in many cases with science-based interventions. And many types of owner-directed aggression can be prevented with appropriate, reward-based training and timely socialization.

Aggression to owners can begin early in life, but it commonly develops as the animal hits social maturity. Common motivations include but aren’t limited to pain, fear, response to punishment, resource guarding of food or space, problems with physical handling, and social conflict.

Prognosis is dependent on a number of factors such as family composition and health, willingness to implement a science-based plan, severity of injuries, degree of warning, number of triggers, ability/willingness to avoid triggers, predictability, and co-morbid medical and behavioral disorders.

The best treatment is multimodal, as for most behavioral disorders. A combination of avoidance and management, environmental enrichment, desensitization and counter-conditioning, psychoactive medication, supplements, dietary change, and pheromone treatment should be considered.

Clients often require help creating a list of specific triggers. Once this list is developed, management plans should be developed for each trigger. For instance, if the dog is aggressive when people approach while he/she is eating, then the dog should only eat in a crate or in a private area where eating can be undisturbed. If the dog is aggressive when people pet him/her, then petting in the trigger area must be avoided.

Safety tools like baby gates, crates, leashes, head halters/body harnesses, basket muzzles, etc. should be implemented proactively and used in trigger situations. Patients often need to be trained using positive reinforcement to enjoy resting in crates and wearing basket muzzles (www.muzzleupproject.com is a great resource for owners working on this). At this time, cat muzzles for frequent wear are not generally available, so confinement and leash training is a cornerstone of safe management for those cats who might bite or scratch.

Avoidance and safe management are the minimal interventions for these behaviors. For some families they may seem sufficient. However, affected families are strongly recommended to implement environmental enrichment and behavioral therapy with a science-based trainer and/or a veterinary behaviorist if available. Unfortunately, avoidance can fail for a variety of reasons. One unlocked door can result in a bite after all.

Environmental enrichment is species specific and also should be individualized to each patient’s needs. In general, both dogs and cats need adequate, pleasant exercise, food puzzles rather than food in bowls, reward-based training, resting/hiding areas where they can remain undisturbed if they prefer, vertical and horizontal scratching areas for cats, and the ability to use as many of their safe, natural tendencies as possible.

Behavioral therapy can often be successful in only a few minutes per day. Specifics depend on the characteristics of the individual animal’s disorder. However, behavioral therapies should minimally be pleasant for the patient, and ideally they should be fun for both owners and patients. Positive punishment (yelling, hitting, kicking, alpha-rolling, scratching, grabbing, staring the animal down or handling the animal in any way that is designed to be threatening) is completely contraindicated because it can result in escalation of the behavior problem in the moment and in the future.¹

Medications, such as SSRIs or TCAs, are often considered and may be prescribed as long as there is a valid VCPR and the clients understand the off-label nature of all medications for aggression in cats and dogs. Supplements, diet change, and pheromones may also be helpful for some patients.

Most patients will improve with treatment within 4-8 weeks if families are following instructions. However, lifelong management and safety remain important for these patients.

References
One of the most complex issues that veterinarians face when managing behavioral problems in practice is when to prescribe psychoactive medications. Multiple levels of analysis and refined administration protocols are required in order to ensure rational use of these medications.

The first thing a veterinarian considering prescribing a trigger time medication should be thinking about is whether there is a valid veterinary client relationship. If none exists, then no medication can be prescribed legally. In addition, all frequently recommended trigger time medications are off-label for use in companion animals.

Extra-label drug use is rational when the patient and/or family is suffering from a behavioral problem, when the patient is a threat to himself or others. However, these medications, like all psychoactive medications, should be used only in combination with safety tools, aggression/anxiety management techniques, environmental management, and a rational, science-based behavior modification plan.

Choosing an appropriate medication for trigger time use is a complex process, but it is no different from medication decisions in other parts of veterinary medicine. The veterinarian must take into account multiple facts individual to the case (species, signalment, history, diagnosis, medication history, medical and behavioral co-morbidities) as well as the published data on efficacy and side effects for the specific medications being considered. A monitoring plan for treatment must be implemented as well as a safety plan. Potential side effects need to be discussed with family members. Route, cost, and duration of treatment must also be addressed.

Medications used only during trigger times need specific characteristics. They need to work quickly, last long enough to be helpful, have a side effect profile that doesn’t negatively impact the patient or the family members’ quality of life, and be affordable.

In addition, it’s helpful if these medications have a dose range that allows family members to titrate the patient’s most effective dose.

Commonly used trigger time medications include the benzodiazepines, trazodone, and clonidine

The benzodiazepines alter GABA (gamma-aminobutyric acid), the most widespread inhibitory neurotransmitter in the brain. This neurotransmitter moderates vigilance, anxiety, muscle tension, neuronal excitability, and memory (too much GABA can inhibit memory). Medications that increase GABA effects include diazepam, clonazepam, clorazepate, alprazolam, lorazepam, and oxazepam. These medications can be reversed with flumazenil. These medications are used off-label for control of anxiety1, phobias2, and historically urine marking3. They are controversial for cases where aggression is the primary complaint or a behavioral co-morbidity. Side effects include sedation, ataxia, increased appetite, muscle relaxation, paradoxical excitation/anxiety, idiopathic hepatic necrosis4, and impaired learning. Impaired learning is not a rational reason to exclude this category of medications from your tool box because anxiety, panic, and fear also impair learning. Dose decreases accommodate patients who have altered hepatic or renal metabolism, are taking other medications metabolized by CYT P450, are obese, or are elderly. With long term use, there is a chance of physical dependence and dose tolerance. Patients need to be weaned off benzodiazepines if they have been on these medications daily for a few weeks. Generally they are decreased by 25% weekly until the medication is discontinued completely. However, if they are truly being used as-needed for intermittent trigger times, weaning is unnecessary.

Dopamine blockers (most commonly acepromazine) are often used inappropriately for trigger times in patients with panic, phobia, anxiety. This medication is on-label for dogs, cats, horses for “control of intractable animals” and as an anti-emetic. However, it is not a true anxiolytic; rather, it is a conventional anti-psychotic. Acepromazine can be useful in combination with benzodiazepines and other trigger time medications when anxiolysis with more appropriate interventions has been insufficient to help calm the patient. Side effects (sedation, ataxia, aggression, hypotension/paradoxical tachycardia, and paradoxical excitability) can be prolonged and onset of best action can take several hours.

Trazodone is published for use in patients with anxiety disorders and for post-op calming of active patients.5,6 Trazodone is a serotonin antagonist/reuptake inhibitor. Veterinary studies report improvement in clinical signs around 60 to 90 min after administration in most patients. The medication is not controlled, readily available, and relatively inexpensive. Nausea is a side effect that can be prevented in many patients by starting at the low end of the dose range and titrating up as-needed. Other side effects, such as ataxia, sedation, pain, increased anxiety, agitation, or irritability can occur. The potential for priapism precludes this medication’s use in most intact, breeding males. This medication can be used safely, if carefully, with SSRIs, TCAs, clonidine, benzodiazepines, and even acepromazine.

Clonidine is published for use in canine patients with fear-related aggression, noise phobia, and separation anxiety.7 This alpha-2 agonist works by blocking NE release in the locus ceruleus and is effective in 60-90 min for many patients. It is not controlled, readily available, and relatively inexpensive. Side effects include sedation, ataxia, increased agitation, anxiety, and irritability, as well as...
as nausea. This medication can be used as a single agent or rationally with SSRIs, benzodiazepines, or trazodone if additional control of panic is required.

Gabapentin is used anecdotally in patients requiring trigger time meds who may also have neuropathic pain and/or do not respond to other interventions. It is also used for patients who have drug interaction issues precluding use of other more well-researched anti-anxiety medications. While it was once considered to work on GABA, it is now thought that it may function by altering glutamine. The dose range is wide and the short half-life requires re-dosing at least every 8 hours for most patients if control is required for an entire day or several days. This medication is relatively inexpensive, readily available, and not controlled. It can be used safely in combination with SSRIs, TCAs, benzodiazepines, clonidine, trazodone, and acepromazine.

There are a variety of medications that can be helpful for patients who are anxious, panicked, or phobic in specific situations. These medications can be used as monotherapies or combination therapies. They can be combined with other medications if needed. In addition, they can be used just as-needed or daily with additional bolus doses for trigger times. Situational anxiety, panic, and phobia can lead to death of patients through traumatic injuries as well as through abuse, abandonment, and euthanasia. Thankfully, most patients can improve quickly with treatment.

References
For a variety of reasons, cats get less veterinary care than dogs. Many of the specifics related to this difference are the result of the owner’s stress and the cat’s fear and distress on the way and while at the veterinary clinic. Indeed, some cats will be “off” for several hours after a veterinary visit and some may end up in fights with resident cats upon their return to the home. These fights can cause long-term damage in even previously healthy intercat and human relationships.

Thankfully, there are many ways we can encourage our clients to get their cats back in for the preventative and disease-related care they need. Clients and cats need to know they are coming into a safe, supportive environment. Without this, you are damaging the cat’s ability to receive thorough care, causing problems for your bottom line, and certainly contributing to unnecessary animal suffering.

Veterinarians take an oath to mitigate animal suffering. With the focus on low stress handling, it is going to get easier and easier to find veterinarians who are obviously and proactively working to make the veterinary experience more pleasant for cats. Wonderful, smart, dedicated, and sophisticated clients will vote with their pocket books. They already are.

First, it’s important to make sure your practice has a plan to support cats that are currently coming in. Improve the client and patient experience immediately. Commitment to the principles of low-stress handling and restraint is a bare minimum. If this is new for your practice, start with things clients notice. For instance, have staff inquire about what can be done to make the cat more comfortable in the practice at the time of scheduling. Ensure the cat is comfortable getting into and staying in his/her carrier. If not, email the client videos on teaching the cat to like the carrier. All clients should receive a short informational note about how to carry the cat once in a carrier (keep carrier covered, spray 20 minutes ahead of travel with pheromone spray, make sure there is a comfortable and clean soft blanket inside, keep carrier level to the ground and very stable rather than swinging or carrying it over the shoulder). Make medical record notes specific to each patient and accommodate where possible.

For any patients who are known to be very fearful or aggressive at the veterinary clinic or during travel schedule an appointment, possibly even without the cat if the VCPR is current, to discuss anti-anxiety medication trials and possible sedation or general anesthesia. These discussions are best had before the cat and the client are stressed.

A few days before the appointment or at appointment booking, remind clients to bring the cat’s favorite treats and toys as well as a favorite blanket. If you think you may need to draw blood, this is a good time to check and see if the owner is comfortable being present for procedures of this nature.

On the day of the appointment, have clients go directly into exam rooms if there is no cat-only area. If limited exam room space precludes this, have clients wait in their cars where it is generally quieter and less stressful. Call the client when you are ready for him/her to go into a room and provide an escort directly to the room.

If possible, have clinic rooms reserved for cats-only. These rooms should be free from pictures of cats or other animals since these can be frightening. Have an easy to sanitize basket or bed available on the exam table for the cat to cuddle in. Make it even better by keeping the table or potential resting areas warm with a low heating pad or other source of warmth. Open the carrier and allow the cat to wonder out rather than dumping or pulling the cat out. Take the carrier apart or open from the top and examine the cat in the carrier. Many cats do well when examined on the clinician’s lap. Offer tasty food and/or play during the exam to help distract the patient and help him/her associate veterinary handling with fun and yummy food. All handling done with the cat should be gentle and with his/her mental state in mind. And don’t wear that white coat. It’s startling and, often, a classically conditioned signal that bad things have arrived.

Of course, there are many other things you can do to have a cat friendly practice. But these are some great places to start. Once you feel ready, start reaching out to the cats your current clients already have who are not receiving adequate care. Find out if dog owning clients have cats that aren’t coming in, and create a system for following up to get them into the practice. If mail or email reminders to cats have gone unheeded, have the staff reach out more personally to get appointments scheduled.

For owners who haven’t responded to direct outreach by making an appointment, consider offering a webinar or, even better, and in-hospital, human-only, informational party where people can walk through the cat-friendly practice. This gives you a great chance to get clients specific information on carrier and travel training. You can also take this time to remind them of the importance of preventative care as well as signs of illness. This party can help clients feel less stressed at your hospital and bond them more strongly to the practice. One caveat is that this type of offering must be hosted by cat-loving staffers who are true “hospitalitarians.” Clients
know when they aren’t being genuinely welcomed, so interactions with ill-suited people can backfire. For really forward-thinking clinics, these parties can be offered frequently and address different topics. Remember- the more frequently clients have great experiences at your clinic, the less stressed they will be when they consider bringing their pet in, and this can result in more visits to the clinic.

Most of these are quick tips that require minimal investment, are easy to learn, and pay big dividends in client care. They can significantly improve the care you are able to provide your cat patients and get new cat patients in the door. And most importantly, when you build a relationship of trust and support with your cat clients they are going to bring their cats back before they get sick, allowing you to prevent a variety of illnesses and expenses. Your job will be more fun and profitable because when these client vote with their pocket books, they will be voting for you.
Behavior problems are both a health and welfare issue for the pet. They also weaken the pet owner bond, decrease the owner’s commitment to pet care, and a significant factor in relinquishment. Veterinary behavior counseling for pets with behavior problems is best be managed with a team approach of veterinarian, behavioral techniciand and trainers, support staff, and resource material. While each case needs to be managed individually a 5 step template can be used as a treatment strategy for each case.

1. Diagnosis
   Treatment must be tailored to the pet, the household, the owners and to the diagnosis. Behavioral problems and any change in behavior may be the first or only sign of a medical issue. Therefore screening for both behavioral and medical signs at each visit, and a workup that includes history, physical examination and diagnostic tests appropriate for the presenting signs is essential to health care. In addition to medical health issues, a problematic behavior might be normal but undesirable, or behaviorally abnormal in emotional or mental health.

2. Prognosis
   Prognosis is about whether the problem can be safely, effectively and humanely managed from the perspective of the pet and owner, the potential for further improvement, and owner expectations. Rehoming or euthanasia may be necessary if the pet’s welfare cannot be sufficiently addressed, the owners are unwilling or unable to implement effective safety strategies or they do not accept the limitations of what can realistically be achieved. For aggressive cases, the ESVCE position statement on risk management (esvce.org) describes the following steps; identify risk factors, determine who might be harmed and how, discuss precautions for each risk, record and implement, and update and review.

   Family and environmental factors including family makeup (age, impairments, other pets), schedule and the household itself are critical factors to assessing risk and determining prognosis. Unpredictability, dogs that bite in response to benign stimuli, dogs greater than 18 kg and dogs aggressive to family members especially children are risk factors for rehoming or euthanasia.\(^1\) Source, age of acquisition, age of onset, breed, early environment (prenatal, neonatal, maternal), medical health and ongoing learning all influence the development of aggression and whether it can be successfully managed and improved.

   Before beginning to counsel on management and treatment, it is necessary to determine the severity of the problem for pet and owner, whether a change in training techniques, control devices, or drugs may be useful and whether further improvement is practical.

3. Management
   Behavioral management to avoid stimuli and prevent recurrence is the first step in treatment a) to insure safety b) for the health and welfare issue of the pet, and c) to prevent further learning / conditioning. In addition review the pet’s behavioral needs and insure that there is adequate enrichment and that needs are being adequately met. Unpleasant outcomes including how the stimulus, owner and pet responds, will condition further fear and anxiety. On the other hand, each time the stimulus retreats from the threat or the pet is removed from the situation the behavior is negatively reinforced. Prevention and avoidance allow for a cooling off period in which the behaviors are not repeated, the physiologic and psychologic effects of fear, anxiety and stress are avoided, the pet’s environmental needs can be effectively addressed and reward based (foundation) training implemented in an environment in which the pet can succeed. If drug therapy is indicated it can be administered to effect before any further exposure training is implemented.

   Owners must learn to read and recognize communication signaling of their pet, including facial expressions, body postures and actions to a) identify signs of relaxation (to be able to train and shape increasingly more calm responses) and b) identify the threshold / limit at which signs are first seen so that the pet can be kept beyond the threshold at which problems begin to arise. See resource list below

   Each situation in which the problem might arise and all stimuli that might trigger the behavior must be prevented by keeping the pet away from locations and situations in which problems might arise, avoiding actions and interactions that might trigger the behavior, and identifying and avoiding or staying below the threshold at which the pet might react (i.e. distance, stimulus, environment). Consider whether products are needed that can aid in achieving desirable outcomes and preventing undesirable behavior including a leash and head halter, or a leash and front attachment / no pull harness. A basket muzzle should be used in any situation where safety cannot be insured or problems cannot otherwise be prevented (see muzzleupproject.com). Positively condition the pet to each new management product.
i) Fear and aggression on walks: Dogs that are fearful or aggressive when on walks should be walked at times or in places where
fear evoking stimuli can be avoided, or kept on leash (perhaps with head halter or no pull harness) at a distance, location or with
stimuli where the dog can be effectively calmed. Taking the dog to an area where stimuli can be effectively prevented or avoiding
walks entirely (and providing alternative form of enrichment) may be the only effective way to prevent exposure.

ii) Fear and aggression to visitors: For dogs and cats that are fearful or aggressive toward visitors to the home, the pet should be
confined to a safe haven where it is comfortable and secure when visitors arrive (crate, room, barricade). Providing favoured treats,
food manipulation toys or chews, or the use of white noise, classical music, pheromones, and lavender can help to keep the pet calm in
its safe haven. The pet can be allowed controlled exposure at a safe distance if it can be calmed and controlled such as with leash and
head, body control device.

iii) For fear and reactivity to visual and auditory stimuli, exposure might be minimized by preventing or muting exposure such as
by housing the pet away from, or blocking access to doors and windows, keeping the pet on leash to prevent access, or using window
coverings or wax paper to block the view. Providing highly enriching and enjoyable activities can keep the pet focused away from
stimuli and occupied in desirable behaviors. Visual stimuli can also be reduced with eye covers such as thundercap or doggles.
Audible stimuli can be muted or exposure prevented by disconnecting the door bell, housing the pet away from stimuli, sound baffling
of rooms or crates, ear covers such as mutt muffs, or with white noise, a fan or music. Some studies have demonstrated that classical
music, music designed for pets or even a talking book may also be calming.

iv) If specific actions, interactions or situations trigger the response, these need to be identified so that strategies can be
implemented for avoidance. The more specific the trigger the more specific the avoidance can be. For example dogs that are
aggressive when in possession of favoured resources or when resting, should be housed in a safe haven where they can be avoided
during eating, chewing, playing with toys or resting. If there is fear or aggression between dogs or cats in the home, they may need to
be physically separated into separate areas of the home or on leash. However if the interactions that trigger the response (e.g. food,
resting areas, locations, homecomings, owner attention) are identified, the pets would only need to be kept separated prior to and
during any of these interactions. Conflict over space and resources might also be addressed by providing sufficient opportunities and
outlets (abundance of riches) for the pets to engage in their normal species typical behaviors separately and independently. For cats
this would include climbs, perches, litter boxes / litter stations, feeding stations, water bowls, and play sessions with the owners.

4. Modifying behavior

Behavior can be modified by teaching (rewarding and shaping) behaviors you want, and preventing or ignoring behaviors you don’t
want. Punishment should be avoided as it does not teach the pet what is desirable. In fact, even if it is effective at decreasing
undesirable behaviors it does so by causing fear of repeating the behavior leading to avoidance (flight), freezing or aggression (fight),
and may condition further negative emotions to toward the stimulus or cause fear, anxiety or conflict in its relationship with the
punisher.

Rewards should be given repeatedly and immediately after those behaviors you want the pet to learn (repeat). Rewards given any
other time will slow learning, increase uncertainty or inadvertently reward undesirable behavior (see structured interaction training).
Therefore whenever the owner has anything of value, whenever the pet wants anything from the owner, and whenever the owner
observes the pet engaging in desirable (target) behaviors the pet should be immediately rewarded. The three parts to the command –
response – reward sequence are a) getting the desired behaviors, b) rewarding (marking and capturing the behavior, and c) putting the
behavior on cue. Training under the guidance of a force free / reward based trainer or behavioral technician, can help owners to
achieve these goals.

Foundation behaviors should first be taught in environments with minimal distractions to insure success, using lure reward, target
training, prompting (e.g. with leash and head halter) or simply by close observation to reinforce the desired outcome. With repetition,
immediate timing and highly motivating rewards, the behavior can be gradually shaped to achieve the desired training goal (including
immediacy, duration and relaxation) before moving on to gradually more distracting environments. Working together with a second
calm and well trained dog with whom the dog is socially familiar can help to facilitate training and reduce arousal for some dogs.
Clicker training can help to insure effective timing of rewards as can products such as treat and train or pet tutor that can deliver
rewards remotely.

a) Structured interaction training – say please by sitting (i.e. what to do when you have something that the pet wants): Everything
the pet wants is a potential reward; therefore being consistent in always having the pet sit or lie down calmly before a reward is given,
teaches the pet how to ask for what it wants. In fact, a lack of rule structure and inconsistent responses may lead to inadvertent
reinforcement of undesirable behaviors or cause increasing uncertainty and anxiety, whenever the owner has something the pet values.
Consistency is needed to teach the pet how to get what it wants (by sitting calmly) and gradually shaping increasingly calmer and
more relaxed behaviors before the reward is given.

b) Reward desirable behaviors and ignore or prevent undesirable. For some problems a leash left attached can help to stop or
prevent undesirable and guide into desirable. Reward training includes a) giving rewards immediately each time you observe a

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behavior you want the pet to learn or repeat (e.g. consider clicker training) b) training the pet how to behave each time you have something it wants (e.g. affection, toy, walk) and c) training new and desirable behaviors on cue using valued rewards (training sessions). Keep the rewards out of sight (in your hand, in a treat pouch or pocket) but give consistently, frequently and immediately so that the pet learns to respond to the cues (not to seeing the reward).

c) Use reward training to teach behaviors that will serve as a foundation for further improvement (exposure) i.e. to replace the current response with a behavior that is desirable (differential reinforcement of an alternative behavior, response substitution). If a management product is to be used during exposure (basket muzzle, leash and head halter) then the pet should be positively conditioned and trained with the product in the absence of fear evoking situations and stimuli.

- Foundation basics – watch, sit / focus, touch or target
- Foundations for problems that arise in the home – mat, crate or room, leave it
- Foundations for problems that arise on walks - loose leash walk, let’s go (turn away)
- Depending on the household and problem additional foundations might include drop it / give, chin rest, find it, recall / come
- Cues can also be associated with high value items (name the item or interaction) to direct the pet to engage with these favoured items e.g. toys, food, treats, chews

d) Exposure training (set up to succeed)

i) Only when the pet will reliably display the foundation behaviors (immediate, relaxed, and of sufficient duration) in a variety of environments with increasing levels of distraction, would it be possible to proceed to setting up controlled levels of exposure to stimuli to train and reward desirable outcomes (response substitution, Differential reinforcement of an alternative behavior), and make positive associations with the stimuli (counterconditioning). Exposure should be controlled (set up) for initial exposure training to insure that successful outcomes can be safely and effectively achieved.

ii) Identify the threshold at or below which the fearful, anxious or aggressive response would be triggered by selecting the environment / location, stimuli, distance or volume, at which success can be achieved. Continually monitor the pet’s body language for any signs of emerging fear, anxiety or stress to be able to immediately stop, return to a calm baseline and reward.

iii) To be successful, it will be necessary to develop gradients of rewards and stimulus intensity to be able to start with a strong enough reward and mild / muted enough stimulus (stimulus, location / environment, level of arousal, intensity, distance) to make positive associations as soon as the pet orients to the stimulus (counterconditioning) or to cue the pet to a desirable outcome (focused, calm) and reward.

5. Medication

Psychotropic drugs and natural products can be used for reducing the signs associated with phobic, panic or chronic anxiety and to improve trainability in situations where the pet is anxious, fearful, or aroused. Drugs may also have a dramatic effect when there is brain pathology, as with compulsive disorders, impulse dyscontrol, reactivity or cognitive dysfunction syndrome. However, drugs do not change the relationship with the stimulus so that concurrent behavior modification will also be needed to desensitize, countercondition and train desirable responses. While some practitioners and owners may be reluctant or averse to using drugs until the pet’s response to environmental management and behavior modification has been addressed, medication should be dispensed as soon as it is determined that it is indicated or necessary, to address the pet’s welfare, facilitate training and behavior modification, prevent further progress and address the underlying behavioral pathology / mental health of the pet. In addition since drugs such as selective serotonin reuptake inhibitors (SSRI’s) and tricyclic antidepressants (TCA’s) take 4 weeks or longer to achieve full therapeutic effect, starting drugs these early in the course of treatment will still require sufficient lead time to assess effect.

Reading body language

Canine
- Learn to speak dog and teach your kids: doggonesafe.com
- Zoom Room Guide to Body Language https://www.youtube.com/watch?v=00_9JPltXHI

Feline
- CatChannel - http://www.youtube.com/watch?v=rhLULk9Xr1E
- http://www.humanesociety.org/animals/cats/tips/cat_communication.html
Choosing a trainer
- https://pawsforpraise.wordpress.com/2013/07/21/finding-the-right-dog-trainer-harder-than-you-think
- Karenpyroracademy.com
- www.petprofessionalguild.com – Positive reinforcement trainers
- https://academyfordogtrainers.com
- http://www.ccpdt.org – Certified professional pet dog trainers
- https://positively.com/ - Victoria Stillwell professional dog training

References
Is it behavioural or is it medical?
Behavioural signs can be the first or only indication of an underlying medical issue. Therefore the first step is to identify all presenting signs and do a comprehensive assessment to determine if there is an underlying medical cause. Conversely the presence of a behavioral inciting factor or a known breed predisposition in the absence of abnormal medical findings should point to a behavioral cause. In addition, stress may cause or contribute to dermatologic, gastrointestinal, and urinary tract disorders.

Abnormal repetitive behaviors
Repetitive behaviours in companion animals have been termed compulsive, obsessive-compulsive, and stereotypies, or may arise from other behaviour pathology including hyperactivity disorders and dissociative syndrome. One recent paper identified a link between tail chasing in Bull Terriers and autism in humans. Since these behaviours likely represent a heterogeneous group of conditions with differing pathologies, abnormal repetitive behaviour (ARB) might be used to describe the clinical presentation until a diagnosis can be made.

When presented with an ARB, the first step is to identify all presenting signs and to determine whether these signs are due to an underlying medical cause. The diagnostic dilemma is further complicated by the fact that the behaviour (e.g. self-trauma) can lead to medical complications including pain, inflammation, and infection. Stress may also contribute to the medical signs. Even if a medical problem is identified and treated, behavioural signs may persist due to alterations in neurotransmitters and receptors, further learning and owner responses that stress, frustrate or reinforce the pet.

Compulsive disorders
Compulsive disorders are abnormal, repetitive, variable in form and fixated on a goal. They may be sufficiently exaggerated, intense, or sustained that they cannot be easily disrupted or switched to another behavior. In addition, there may be a lack of control in initiating or terminating the behaviour. Compulsive disorders such as self-trauma, circling or tail chasing might initially arise as displacement behaviors arising from conflict or frustration. Conflict is when the pet has competing motivations or uncertainty, while frustration is when the pet is motivated to achieve a goal but physically or behaviorally prevented. Displacement behaviors might resolve if the inciting factors are avoided and the conflict or frustration addressed. In fact, dogs displaying repetitive behaviors at times of high arousal are distinct from those displaying stereotypic behaviors in which arousal is low.

In compulsive disorders signs begin to arise outside the original context and begin to impact on normal daily functions. They are derived from normal behaviors such as grooming, predation, or ingestion. There may be a genetic disposition with breed predilections such as tail chasing in German Shepherds, spinning in Bull Terriers, wool sucking in Oriental cats and flank and blanket sucking in Dobermans, for which a genetic locus has been identified.

Abnormal serotonin transmission has been identified as a primary mechanism by which compulsive disorders are induced. Brain areas of interest include the prefrontal cortex and amygdalae. As in humans with obsessive compulsive disorders, drugs that inhibit serotonin reuptake (e.g. clomipramine, fluoxetine) are most effective. However, multiple neurotransmitters have been implicated including alterations in dopaminergic and glutamatergic pathways or opioid receptors.

Stereotypies
Stereotypies are defined as repetitive behaviors that are unvaried in sequence and have no obvious purpose or function. They have been commonly reported in farm, zoo, and laboratory animals and arise in situations of conflict or frustration related to confinement, husbandry, or deficits in housing. They may arise when the environment lacks sufficient outlets for the animals full behavior repertoire, with maternal deprivation, and due to neurologic disorders. Stereotypic behaviors, at least in their early stages, may provide a mechanism for the pet to cope. For example, non-nutritive suckling in calves may aid in digestive processes. Stereotypies might be induced by dopaminergic stimulation.

Differential diagnosis
Even if no medical causes are found, a therapeutic response trial might still be required to differentiate medical (e.g. seizure, dermatologic, gastrointestinal) from behavioral. Focal seizures might be the cause of neurologic signs; self-traumatic disorders might be due to hypersensitivity reactions; and ingestive behaviors might be caused by gastrointestinal diseases. Feline hyperaesthesia can have a dermatologic, neuromuscular, or behavioral cause.
Neurologic or behavioral
Some of the most difficult cases to diagnose are signs that might be attributed to neurological disorders such as circling, air snapping, fixed staring, tail chasing, spinning, pouncing, star gazing, head shaking and checking. Behavioral signs may be associated with virtually any disease that affects the CNS or its circulation.

Careful review of all clinical signs, video of the problem, physical and neurological exam, and diagnostic tests including blood, urine, imaging, or EEG may need to be considered if a neurologic cause is suspected. With neurological deficits, altered mental status, increased sleep, circling, head pressing, seizures, emesis, or altered eating and drinking, a neurologic diagnosis can be made. However, behavior signs may arise in the absence of neurologic signs. Changes in behavior, personality, or mood, decreased responsiveness to stimuli, or loss of previously learned behavior including housetraining indicate forebrain disease. Altered responsiveness to stimuli can also arise from sensory or motor dysfunction.

Epilepsy is a rule-out when pets are presented with focal motor or sensory signs or altered mood or temperament. Seizures may be generalized with convulsions and loss of consciousness but focal seizures may present with motor or sensory signs such as fly biting, chewing, swallowing, star gazing, tailing chasing and aggression. In one study of dogs with fly snapping or “fly catching syndrome” (10 of which were CKCS) and all of which had initial neurological assessment including some or all of MRI, CSF, EEG and BAER testing, 34% of 11 dogs improved with phenobarbital (2-3 mg/kg bid) and 100% of 11 dogs with fluoxetine at 1 mg/kg bid (as defined by 50% or greater response). Two of these dogs were CKCS with Chiari malformation. Concurrent signs in some of the dogs included licking in the air; face, neck and ear scratching; tail chasing; and hind limb biting. Behavior causes must also be differentiated from other epidemic events including tremors, narcolepsy, and syncope.

Unlike seizures repetitive behaviors can generally be interrupted or the situations in which they arise predicted or reproduced. Some pets are so intent on repeating the behavior that they will move away and repeat the behavior in the owner’s absence. Oral behaviors including air snapping, licking, sucking, pica, smacking lips, and gulping can also be a result of a gastrointestinal disorder.

If diagnostic testing is unable to yield a definitive diagnosis, a therapeutic trial may be the next option. For focal seizures, an improvement might be expected with phenobarbital, potassium bromide, or levetiracetam while gabapentin, carbamazepine or clonazepam might be added for refractory cases. When neuropathic pain is a consideration a trial with gabapentin, pregabaline, carbamazepine or amitriptyline might be warranted. A trial with drugs or dietary management might be needed to rule out a gastrointestinal cause. For compulsive disorders, clomipramine or fluoxetine might be the first choice.

Dermatologic or behavioral
To diagnose behavioral self-trauma (e.g. biting, chewing, licking, and excessive barbering) medical causes must first be excluded including pain or pruritus (e.g. neuropathies, hypersensitivity reactions), infections, tumours, endocrinopathies or systemic diseases (e.g. hepatocutaneous syndrome). The diagnostic work up should include examination, blood and urine testing (including viral or endocrine if indicated), and dermatologic testing including trichogram, fungal culture, skin scraping, cytology and possible biopsy. The presence of primary lesions and the sites of the alopecia or self-trauma might suggest other potential diagnoses.

For cats with self-trauma, if diagnostic tests are negative, parasitic hypersensitivity, food hypersensitivity and atopy must still be ruled out. Therefore a therapeutic trial with a parasiticide and a novel or hydrolysed protein diet for at least 8 weeks might be instituted, followed by a steroid response trial to rule out atopic pruritus. Using this protocol in 21 cases presented for psychogenic alopecia, 76% had a medical aetiology (primarily adverse food reactions, atopy or both), 10% were compulsive and 14% were combined medical and behavioral. Although biopsies might indicate an inflammatory response, some cats with histologically normal skin had a medical cause.

For acral lick dermatitis, when diagnostic tests do not identify the cause, therapeutic trials with antibiotics, pain medication, anti-inflammatory drugs, a food trial or parasiticides should be implemented. However, antibiotic selection is a challenge since cases may be multi-drug resistant and deep cultures may not be consistent with superficial cultures. Owner supervision and preventive mechanisms such as bandaging or E-collars may also be necessary to allow the lesions to heal.

Tail mutilation and hyperaesthesia in cats might be seen in situations of high arousal but can be due to focal seizures, spinal disease, neuropathies, FeLV induced myelopathy, neuropathic pain or compulsive disorders. While rippling skin may be the primary sign, self-directed aggression, redirected aggression, vocalization, excessive licking, running and soiling may also be signs. Therapeutic response trials might include an SSRI or clomipramine for compulsive disorders, clonazepam for seizure control and reducing arousal and anxiety, gabapentin to help calm, control seizures and treat neuropathic pain, or drugs for pain management.

Treatment of abnormal repetitive behaviors
Behavioral management combined with drug therapy is required for the successful control of most behavioral repetitive disorders. Potential sources of stress should be identified and addressed. Lack of predictability in the daily routine, changes in the pet’s schedule or home environment, unpredictable consequences, lack of sufficient outlets for normal behaviors, and sources of anxiety should all be addressed. The pet should be enhanced enrichment to provide outlets for its behavioral needs (e.g. explore, hunt, scavenge, perch,
scratch), a regular and stimulating daily routine including social play, reward training, and object play to keep the pet positively engaged. Training should reward behaviors that are desirable and prevent or ignore behaviors that are undesirable, and no punishment of undesirable. Interactions with the pet should be structured by teaching sit (say please) before getting anything of value (e.g. treats, toys, play). Predictable interactions give the pet control.

For repetitive behaviors, triggers should be identified so that recurrence of the behavior can be prevented and underlying conflict and frustration resolved. When not actively engaged with the owners (or at rest or sleep) the owners should engage the dog in constructive activities (e.g. food toys, chews). In situations where problems arise, the pet should be taught to engage in alternative desirable behaviors (e.g. sit at greeting). At the onset of any undesirable behavior, the owner should immediately cue the pet into a desirable behavior (e.g. sit, down, come, go to your mat), or leave a leash attached (to a head halter or body harness) to be able to physically prompt the pet into an acceptable outcome.

Medication

Most compulsive disorders should improve substantially with SSRI’s such as fluoxetine, sertraline, paroxetine or citalopram or with clomipramine to enhance serotonin transmission (compulsive) and possibly inhibit dopamine activity (stereotypy).12-15 After 4-6 weeks, if there is insufficient response higher doses may be needed. Tricyclic antidepressants (TCA) other than clomipramine are not as effective since they are less selective for serotonin reuptake inhibition.

Since altered glutamatergic neurotransmission may be a factor, blocking glutamate sensitive NMDA (N-methyl-D-aspartic acid) memantine or dextromethorphan might be an effective adjunctive therapy.5,6 However, due to its short half-life, rapid clearance and variable absorption in dogs, dextromethorphan may not be a reliable treatment. Memantine may be effective alone or combined with fluoxetine.16 Huperzine A, an extract of club moss also acts as NMDA receptor antagonist, as well as cholinesterase inhibitor and may have anticonvulsant properties. It may therefore have a consideration as an adjunctive therapy for abnormal repetitive disorders. A dose of .05-0.1 microgram/kg 2 to 3 times daily has been reported to be used in dogs with partial seizures.17

Opioid antagonists such as naloxone and naltrexone have also demonstrated success in reducing stereotypes such as self-traumatic disorders.4 For stereotypic behavior, especially self-licking, selegiline may be an effective therapeutic agent with a response of greater than 80% when the response is still regulated by external factors, perhaps due to external reinforcement.2

Drugs such as benzodiazepines, trazodone, or clonidine might be added adjunctively prior to anxiety evoking situations. Natural products might also be used adjunctively to help the pet to calm including pheromones, l-theanine, alpha-casozepine, tryptophan combined with a reduced protein diet, a diet combining alpha-casozepine and tryptophan, melatonin, aromatherapy or perhaps classical music.

References

7. Hartgraves SL, Randall PK. Dopamine agonist-induced stereotypic grooming and self-mutilation following striatal dopamine depletion. Psychopharm 1986; 90; 358-63

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Cognitive dysfunction syndrome (CDS) is a neurodegenerative disorder of senior dogs and cats which is characterized by a gradual cognitive decline and increasing brain pathology. The diagnosis is based on clinical signs described by the acronym DISHA; Disorientation; altered social Interactions c) altered Sleep-wake cycles d) Housesoiling and e) Altered activity levels which may be accompanied by an increase in signs of Anxiety.1,2.

While a decline in learning and memory may be the most important indicator of cognitive decline, the average pet may appear minimally challenged. Therefore the development and validation of tests in the laboratory (e.g. spatial memory, attention, reversal) for assessing age related decline in different cognitive domains has been instrumental in identifying learning and memory deficits and providing a methodology by which products might be assessed.1,3-6 These and other studies have demonstrated memory impairment in dog and cats as early as 6 years of age.5

Prevalence
Not all dogs and cats develop CDS. Prevalence ranges as high as 74% have been reported.7 In one recent study by Salvin et al., prevalence of CDS in dogs ranged from 5% in dogs 10-12 to 41% in dogs over 14 with an overall prevalence of 14.2%.8 In a cat study, 35% had signs consistent with CDS; 28% of 95 cats aged 11 to 15 and 50% of 46 cats over 15.9 In addition, both prevalence and severity increase with age with dogs with 40% of dogs with no impairment progressing to mild impairment and 24% of mild impairment progressing to moderate over 6 months.7

Behavior signs in senior pets
a) Owner reported signs
The most commonly reported signs in senior pets at behavior referral practices reflect those that are sufficiently problematic to the pet or the owner to seek help. While CDS may be an underlying factor, other neurologic diseases, sensory decline, endocrine and metabolic disorders, musculoskeletal disease and other causes of pain must be ruled out. In a Spanish study by Marrioti et al of 270 dogs over 7 years of age presented for behavior problems, 32% displayed aggression to family members, 16% aggression to family dogs, 9% barking, 8% separation anxiety, 6% disorientation and aggression towards unfamiliar people, 5% housesoiling, 4% destructive and compulsive disorders and 3% noise fears. Of 83 cats referred for behavioral consultations most cats presented with marking or soiling (73%), followed by aggression (16%), vocalization (6%) and restlessness (6%).1

b) Cognitive decline and dysfunction
As signs of cognitive decline may initially be subtle and pet owners unaware that treatment options are available, many cases go unreported until the signs become problematic for the owners, or a welfare issue for the pet. However, early detection allows for early intervention so that further decline might be slowed and behavioral signs improved. Yet most cases go unreported.8 Therefore veterinarians must be proactive in questioning owners for signs. In dogs 8 years and older, activity and play levels, response to commands, and fears and phobias deteriorated most, although medical causes were a possible cause.11 In another study of dogs over 8, the most common signs of CDS were sleeping more during the day and restless at night (57%), altered social interactions (51%), disorientation (49%) and anxiety (46%).2 For dogs with mild cognitive dysfunction, the predominant sign was daytime sleep (70%) with anxiety in 11% of dogs while anxiety in the non-cognitive dysfunction dogs was 4%. The most commonly reported sign in cats 10-13 was altered social interactions while for cats 15 and over, alterations in activity including aimless activity and vocalization were most common.1,9

Diagnosis of CDs
When signs of CDS are identified, a diagnostic workup is necessary to rule out medical, physical and motor dysfunction as a cause of the signs. Next to neurological disease, sensory decline, endocrine and metabolic disorders and musculoskeletal disease are the primary rule-outs. Questionnaires can be particularly useful for identifying signs but there is minimal validation and physical conditions can cause many of these signs. 7,11-13 However, by screening each pet with a broad based questionnaire for signs that have appeared or worsened since 8 years of age (or compared to 6 months previously), pets will be identified for further assessment as to whether the signs are physical or cognitive. Behavioral changes correlated with brain aging include activity which may initially decline but progress to increased activity, restlessness and aimless behavior.14
Environmental management and cognitive enrichment

When cognition is impaired, diet, drugs or supplements might be useful in improving signs and slowing the progress of CDS. Canine studies have demonstrated that mental stimulation in the form of training, play, exercise and manipulation toys can help to maintain quality of life as well as cognitive function.\(^{19}\)

Medical and nutritional therapy

Selegiline is a monoamine oxidase B inhibitor which has demonstrated efficacy in improving cognitive signs. It has been shown to increase 2-phenylethylamine in the dog brain, a neuromodulator that enhances dopamine and other catecholamines in the cortex and hippocampus. It may also contribute to a decrease in free radical load through decreased production and increased clearance. Dose is 0.5 – 1.0 mg/kg daily.

Since the elderly are particularly susceptible to the effects of anticholinergic drugs, it is prudent to avoid drugs with anticholinergic effects. In fact, drugs or natural products that enhance cholinergic transmission might have potential benefits for improving signs of CDS in dogs and cats. Propentofylline, a xanthine derivative is licensed for lethargy and depressed demeanour in old dogs in some countries but not in North America. It may increase blood flow and inhibit platelet aggregation and thrombus formation. Other treatment strategies include nicergoline an alpha 1 and alpha 2 agonist and the NMDA antagonist memantine. No drugs are approved for cats; however, selegiline and propentofylline may be useful.

A primary therapeutic strategy for cognitive dysfunction in dogs, cats and humans is to reduce the risk factors that contribute to cognitive decline. It is likely that an integrative approach is required. In dogs, a senior diet (Canine b/d, Hills Pet Nutrition) has been shown to improve signs and slow the progress of cognitive decline. It is supplemented with fatty acids, antioxidants (vitamins C and E, beta carotene, selenium, flavonoids, carotenoids), and dl-alpha-lipoic diet and l-carnitine to enhance mitochondrial function.\(^{5,19}\)

The combined effect of the diet plus an enriched environment provided the greatest improvement.\(^{19}\) However, while enrichment resulted in improvement in cognitive function, the dietary therapy resulted in a reduction in reactive oxygen species and in beta-amyloid accumulation.\(^{5}\)

A Purina Veterinary Diet (Essential Care Senior, Pro Plan Bright Minds), supplemented with botanic oils containing medium chain triglycerides provides ketone bodies as an alternate source of energy for aging neurons, has also been shown to significantly improve CDS in dogs.\(^{20}\) For cats, a diet (not commercially available) developed by Nestle Purina supplemented with antioxidants (Vitamins E and C and Selenium), arginine, B vitamins and fish improved learning and memory tasks compared to a control diet in cats 5.5-8.7 years.\(^{3}\)

Senilife® (CEVA Animal Health), has demonstrated efficacy in improving cognition in both a laboratory model and clinical studies in dogs. It contains phosphatidylserine, a membrane phospholipid as well as Gingko biloba, vitamins E and B6 and resveratrol. Another product with phosphatidylserine, omega-3 fatty acids, vitamins E and C, l-carnitine, alpha-lipoic acid, coenzyme Q and selenium is available in the UK.\(^{21}\) There is also a cat product with no alpha-lipoic acid.

S-adenosyl-l-methionine (Novifit®, Virbac) may help to maintain cell membrane fluidity and receptor function, regulate neurotransmitter levels and increase glutathione production. Improvement has been demonstrated in dogs in a placebo controlled trial and in laboratory studies in dogs and cats.\(^{4,22}\)

Apoaequorin (Neutricks™) is a protein found in jellyfish that in laboratory trials improved learning and attention in dogs. It is a calcium buffering protein that may provide neuroprotection against aging.\(^{23}\)

Behavior modification and adjunctive medications

Together with diets, supplements and drugs for the treatment for CDS, psychotropic medications may be required to manage underlying stress and address those signs such as night waking, agitation and anxiety that continue to be problematic for the owner and pet. Clinical signs may persist even if CDS and underlying anxiety and stress have been effectively addressed. Therefore concurrent environmental management and behavior modification are also likely to be needed.

Since anticholinergic drugs should be avoided, SSRI’s or buspirone might be preferred options for ongoing use. Trazodone might also be considered either alone or in combination with an ongoing SSRI or buspirone. However, drugs that increase serotonin, should
not be used concurrently with selegiline. While benzodiazepines could contribute to further cognitive deficits, they may be useful in managing signs of anxiety and sleep disturbances. Lorazepam, clonazepam, and oxazepam are preferred since they have no active intermediate metabolites. Adjunctive use of propranolol or clonidine may reduce some of the noradrenergic effects contributing to the signs of anxiety. Gabapentin might reduce reactivity and neuropathic pain. Natural products might also aid in the control of anxiety.

Selected references

The stress response
The stress response is caused by physical and emotional factors that trigger behavioral, psychological, endocrine and immune effects. In the acute response, the autonomic nervous system, HPA axis, and cardiovascular, metabolic and immune systems work together to manage stress and return the pet to homeostasis.

A principle component of the stress response is the release of corticotrophic releasing factor from the hypothalamus which stimulates the release of ACTH leading to an increase in cortisol. Cortisol stimulates gluconeogenesis, a reduction in inflammation, suppression of immune function, and decreased gastrointestinal motility. Behaviorally cortisol is associated with a passive strategy, loss of control, and may also increase sensitivity to aversive events.\textsuperscript{1,2} In one study, dogs on glucocorticoids were significantly less playful, more nervous/restless, more fearful/less confident, more food aggressive, more prone to bark, startle, and react aggressively when disturbed and avoid people or unusual situations.\textsuperscript{3}

The second component is the sympathetic-adrenal-medullary (SAM) system which releases noradrenaline and adrenaline to handle the threat (fight or flight) resulting in an increase in cardiovascular tone with increased blood pressure, heart rate, and respiratory rate, contraction of the spleen leading to an increase in PCV, anti-diuresis, and increased blood flow to skeletal muscles. Noradrenaline also has behavioral effects including arousal, alertness, sensitization and fear conditioning.\textsuperscript{1}

A third component is the lactotrophic axis, prolactin and oxytocin. Prolactin which is suppressed by dopamine, decreases in response to acute stress.\textsuperscript{4} B-endorphin and vasopressin are also associated with stress. In one study cortisol, progesterone, beta-endorphin, vasopressin, heart rate and haematocrit were all increased during and after gunshots in the fearful dogs compared to the fearless dogs.\textsuperscript{1}

While the stress response is an adaptive mechanism, recurrent and chronic stress may lead to dysregulation and an inability to return to homeostasis, as a result of ongoing stimulation of the HPA axis, catecholamine system and other mediators such as excitatory amino acids and brain derived neurotropic factor (BDNF). The ability to adapt to repeated stress varies with early experience, previous experience, genetics, physical health, environment, diet, enrichment and the stressor itself. Stressed animals may have higher levels of plasma 5HT and dopamine compared to controls.\textsuperscript{5} Increases in dopamine may enhance aggressive behavior and lead to an increase in grooming and stereotypic behaviors. In one canine study, generalized anxiety with autonomic signs, fear aggression, displacement behaviors and stereotypies was associated with an increase in prolactin, but not in dogs with acute fears and mild phobias.\textsuperscript{6} As dopamine has a suppressive effect on prolactin, dogs with high prolactin were improved with selegiline. On the other hand dogs with lower prolactin levels were improved with fluoxetine. Elevated cortisol levels in aggressive dogs compared to non-aggressive dogs, may indicate a relationship between aggression and stress.\textsuperscript{7}

Stress and physical health
In humans there may be a correlation between stress and poor immune function, cardiovascular disease, skin disease, asthma, gastrointestinal disorders, and cellular aging. Similarly in pets, stress may alter immune function, reduce reproductive success and contribute to gastrointestinal, dermatologic, respiratory, urologic, reproductive and cardiac conditions, behavioral disorders and a shortened lifespan.\textsuperscript{8} In recent studies of environmental stressors in colony cats, sickness behaviors associated with the gastrointestinal tract and behavior problems including avoidance behaviors and soiling in both cats with FIC and control cats.\textsuperscript{9,10} In addition, grooming may increase following acute stressors in cats.\textsuperscript{11} Feline respiratory disease and weight loss has also been demonstrated to be associated with increased environmental stress in shelter cats.\textsuperscript{12} When comparing shelter housing, cats provided with enriched housing when first admitted to the shelter including an added shelf with a towel draped to allow for hiding, plus additional toys and consistent handling and social interactions, had lower stress scores, were adopted sooner, and had less illness (26% to 12%).\textsuperscript{13}

Cats with feline interstitial cystitis (FIC) have altered bladder permeability during stress when compared to cats in an enriched environment.\textsuperscript{14} An increase in plasma noradrenaline has been demonstrated in cats with interstitial cystitis. Cats with FIC that received MEMO (multimodal environmental modification) had a significant reduction in FIC, respiratory disease, fearfulness, and nervousness and less inflammatory bowel disease and aggression.\textsuperscript{15} In one study there were less bouts of FIC when a Feliway™ diffuser was installed. Behavioral risk factors for FIC might include moving house, movement blocked by other cats, living with dogs, or living with another cat with which there is conflict.\textsuperscript{16,17} In a recent study comparing urine spraying and cats that were not using the litter box, both the behavioral normal and problem cats in the spraying household had elevated glucocorticoids but not the cats in the household where there was a failure to use the litter tray.\textsuperscript{18}
Stress and anxiety can alter bacterial flora, inhibit gastric emptying, increase colonic activity, and increase intestinal permeability leading to irritable bowel syndrome, inflammatory bowel disease, gastrointestinal reflux, and heartburn. In pets, acute fear and anxiety can lead to a decrease in appetite or anorexia, diarrhoea, gastrointestinal reflux, and heartburn. In pets, acute fear and anxiety can lead to a decrease in appetite or anorexia, diarrhoea, gastrointestinal reflux, and heartburn.

Stress can affect reproductive health by decreasing sperm quality, inhibiting sexual interest, erection and ejaculation in males and inhibiting ovulation and decreasing fertility in females.

Although the immune response associated with stress is intended to enhance defence mechanisms, in some individuals stress may contribute to inflammatory dermatoses, including atopic dermatitis, psoriasis, and urticaria. In humans with atopic disease stress may lead to increased levels of IgE and eosinophils and an over-reactive sympathetic adreno-medullary system, and increased epidermal permeability. A similar response in pets might exacerbate atopic disease. In one study of dogs with recurrent pyoderma, psychogenic factors were identified. In addition, dogs with non-social fear and separation anxiety had increased severity and frequency of skin disorders.

With increasing age there is an increase in tissue hypoxia, alteration in cell membranes, increased production and decreased clearance of free radicals, a decline in organ, sensory, immune and mental function. These changes may reduce the pet’s ability to respond to stressful events, resulting in increased susceptibility to tumours, disease, and behavior problems.

Stress and behavioral health

Chronic anxiety and stress may lead to behavioral disorders in humans including panic, post-traumatic stress, generalized anxiety, obsessive-compulsive, and impulse control disorders as well as anxiety, social, and other phobias which may all have animal correlates. When pets are in conflict (competing motivations), frustrated (where the pet is unable to achieve its goals) or when the behavioral needs of the pet are not adequately addressed, displacement behaviors such as self-trauma, spinning, tail chasing or hyperesthesia might be exhibited. These signs are more likely to arise in pets that are anxious or reactive. Behaviors that arise in response to a specific stimulus might be resolved if inciting factors can be avoided, owner responses are consistent and predictable and alternative acceptable behaviors are rewarded. Pet owners may further aggravate the problem if they reinforce, punish or are inconsistent.

Repetitive behaviors in companion animals have been referred to as compulsive, obsessive-compulsive, and stereotypies. Hallucinatory stereotypies such as snapping at the air, looking upward and circling have been described in the French literature as a sign of dissociative syndrome. Repetitive behaviors may also arise in hyperactivity disorders. Since these repetitive behaviors may be a heterogeneous group of conditions with different pathogenesis, the term abnormal repetitive behavior (ARB) might better describe the clinical presentation until a clear diagnosis can be made. In addition the presentation and pathology may change over the course of the disease (chronicity). Although genetics likely plays a significant role, stress (including maternal deprivation, unpredictability, conflict, goal frustration or the lack of outlets to engage in normal species typical behaviors) appears to be a primary contributing factor. (See notes on repetitive disorders)

Stress prevention and management

Preventing, minimizing, managing and resolving stress is essential for maintaining both behavioral and medical health and welfare. Prenatal, perinatal and post-natal stressors can have a profound effect on learning, memory, reactivity, and fear conditioning. While mild prenatal stress, early handling, and good maternal care produce more resilient offspring with lower HPA axis activation in response to stress and improved task learning, prenatal stress, maternal malnourishment or maternal deprivation, cause delays in brain and physical development, hyper-reactivity, increased fear and aggression, decreased learning ability and a sensitizing effect.

The primary sensitive (socialization) period is a critical time for establishing healthy social relationships with animals and people and reducing environmental fear through exposure to a broad range of stimuli with each of the senses. Puppy classes beginning during the sensitive period (12 -14 weeks) and kitten kindy ideally beginning before the end of the sensitive period (7 to 9 weeks) can be a valuable and productive way to make positive associations with varied social and inanimate stimuli, improve training skills, and address emerging problems.

Animals kept in a restricted environment may not have adequate opportunity to engage in their full behavior repertoire. To effectively address the pet’s emotional and behavioral well-being and prevent conflict and frustration the focus should be on providing the pet with i) predictability and control ii) enrichment that provides outlets for all behavioral needs iii) a safe haven and iv) reward based learning.

- Allowing the pet control to choose activities in which it wants to engage and avoid those it does not, prevents the stress that might arise if the pet were forced into interactions or housing that are undesirable. Therefore it is essential to set up the environment to encourage the pet to make choices (where to climb and perch, chew, scratch, sleep; how to greet)
that are acceptable to the pet and owner. All rewards should be given predictably (i.e. structured interaction training, doggy please) where anything of value (i.e. affection, toys, food) is only given for sit. Predictability gives the pet control by learning what behaviors get rewards.

- Enrichment should provide the pet with opportunities to meet its behavioral needs and normal daily time budget. This should include social activities including reward training and play that is appropriate for the species and breed (e.g. retrieving, herding, tug games, predatory play), other interspecific or intraspecific social play, object play including chews and feeding toys and opportunities to explore, chew, scavenge, hunt, climb, perch, scratch and eliminate.

- To insure comfort, safety and control for the pet while preventing behaviors that are undesirable, the pet should have a safe haven for housing i.e. a conditioned place of safety outside of times when there are stressors. Use of the safe haven can be encouraged by leaving treats, toys or chews in the area, rewarding entry (e.g. clicker training) and placing on cue. The location should be one in which the pet feels comfortable sleeping or spending time on its own. When in its safe haven the pet should never have attention forced upon it, or be forced to leave the area.

- Learning should be a reward based process to increase behaviors that are desirable, NOT punish behaviors that are undesirable. Training that provides the pet with control can add to the pets enrichment. Consistency, timing and repetition is necessary for learning. Clicker training can be particularly effective for shaping and immediate timing.

- Medications might be considered as prevention or treatment for potentially stressful situations. Dog appealing pheromone might help ease the introduction of puppies into their new homes. Puppies enrolled in puppy socialization classes that wore pheromone collars were less fearful and more social than dogs with placebo collars, while puppies introduced into new homes adapted more quickly and were less stressed in novel situations. Feliway may also help to reduce stress and facilitate introduction of kittens into the new home or at times of stress and change. Other products that might calm include l-theanine, tryptophan in combination with a reduced protein diet, alpha-casozepine, a diet combining alpha-casozepine and tryptophan, and perhaps aromatherapy or music therapy. Before a known stressful event, drugs such as benzodiazepines, clonidine or trazodone might be used on an as needed basis, or clomipramine, fluoxetine or buspirone on an ongoing basis.

Resources

AAFP and ISFM feline environmental needs guidelines: http://bit.ly/14uWTCB.
Websites: indoorpet.osu.edu, catvets.com, icatcare.org, clickertraining.com

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Disaster Preparedness for Food-Animal Practitioners
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It is likely that food animal practitioners will find themselves involved in assisting with a disaster at some point in their professional career. Food animals can be involved in many types of disasters, from accidental (or non-intentional) to intentional. Non-intentional disasters can include weather related events such as tornadoes, hurricanes, floods and blizzards, or naturally occurring disease-related events such as emerging or foreign animal disease outbreaks. Non-intentional disasters can also be accidental, such as major disease outbreaks, vehicular wrecks or the release of chemicals following a fertilizer plant explosion. Thousands of accidents occur yearly on roads and highways involving livestock. Intentional disasters occur from the result of man-made purposeful acts such as arson, terrorist acts (including agroterrorism and bioterrorism), or intentional release of chemical hazards.

Weather-related disasters occur frequently in the United States. According to the National Oceanic and Atmospheric Administration (NOAA), ten billion-dollar weather related disasters impacted the United States in 2015. For the past 30 years, the majority of these events have occurred in the southern United States. This included winter storms, tornadoes, and flooding. Smaller scale events can have equally tremendous effects on the communities where they occurred. Winter Storm Goliath killed tens of thousands of livestock in January of 2016, and flooding across Texas and the midwest have already cost millions of dollars in losses. Heat can also be a major factor is disasters, especially with a large part of the US currently being under drought conditions. Large numbers of livestock are lost every year due to extreme heat conditions - the year 2012 was the warmest and second-most extreme year on record for the contiguous US, with every state reporting above-average temperatures.

According to the National Agriculture Statistics Service (NASS), there were close to 90 million cattle and calves, including over 9.3 million dairy cattle and 30.3 million beef cows, and 67.6 million hogs on inventory in the US in January 2016. It has been estimated that over 1 million hogs are in transport at any given day in the US, travelling to slaughter or to other production units. All livestock numbers, including sheep and goats, appear to be increasing over the past several years. With these numbers of livestock, and the number of weather and climate associated disasters reported in the US every year, veterinarians should be prepared for the next disaster. From a food animal veterinarian’s perspective, in addition to the direct and immediate impact a disaster can have on the people and animals in their community, a disaster can have devastating effects on the agricultural economy for years to come.

Disaster management has three phases: Disaster readiness, disaster response, and disaster recovery. Each play an important role in the overall successful mitigation of an event, but disaster readiness is the most important phase that will guide response and recovery activities. Disaster readiness begins with an awareness. Quite often we hear veterinarians state that they don’t know where to fit in to a disaster management plan. Food animal veterinarians should recognize that their skills extend beyond the traditional veterinary medical care that they are used to providing. Veterinarians have unique training in areas such as epidemiology, food safety, animal care/welfare, zoonotic diseases, environmental health, and foreign animal diseases. In addition, veterinarians are able to recognize the importance of the human-animal bond, and have the ability to deal with both human and animal issues in the event of a disaster. However, in addition to the standard and expected veterinary care, veterinarians can contribute leadership, organization, and communication skills to disaster management. Not only is it important for veterinarians to be aware of their role in a disaster, but first responders and emergency managers at the local, state, and federal levels need to be aware of the important roles veterinarians can play in disaster management. This involves becoming active in the community and surrounding areas.

Disaster readiness also includes preparedness. This may be one of the most important but most misunderstood areas of disaster management. From a practical standpoint, most food animal veterinarians don’t have the time to spend taking lengthy classes and workshops that teach them how to be involved in disaster management. However, more and more resources have become available to veterinarians through on-line learning modules, veterinary continuing educational programs, and locally-supported exercises that can help a veterinarian prepare for a potential role in a disaster situation. As more people realize the important of veterinarians in disasters that can affect animal populations, we see more and more opportunities being offered at local, state, and federal levels.

Disaster preparedness occurs at three levels: personal preparedness, business preparedness, and community preparedness. At minimum, veterinarians should have a disaster plan for themselves and for their families. You cannot effectively help others if your own house is not in order. Personal preparedness is also essential before considering volunteering to respond to an outside disaster. Veterinary responders should make sure their own families and animals will be cared for in their absence so that other first responders and resources aren’t taxed further by caring for their families. This includes making arrangements for transportation and evacuation as necessary, food/water/shelter, as well as gathering important documents and goods. Veterinarians should remain up-to-date on their vaccinations at all times, especially rabies and tetanus. When responding to larger outside events, they may also want to include hepatitis A and B, and influenza, and have a current TB negative test.

How about a disaster plan for your practice or clinic? Business continuity is the term used to describe planning and preparedness activities to ensure a business’s critical functions will continue despite serious incidents or disasters that might have otherwise
interrupted them. Veterinarians should have a practice disaster plan, and be aware of the local resources should a disaster arise. Having a communication plan is especially important in an ambulatory situation, where communication can be unreliable even on a good day. Patient records and other important information should be stored electronically and backed up daily to an off-site location. Identifying a “sister clinic” or another veterinarian to help care for patients and cover calls if a disaster occurs is a good way to ensure continuity of operations while you are dealing with an event. In some cases, employment agreements can also be made for your staff so that they can continue to support their own families. Most major pharmaceutical companies and distributors have emergency provisions to supply veterinarians with needed items. In addition to ensuring veterinary care, locate an area where patients can be housed in an emergency, such as a livestock arena or humane society. If you have potentially hazardous materials in your practice, such as chemicals or radiological materials, it’s a good idea to have the local fire or haz-mat officials in for a tour of your facilities.

Local responders will expect a food animal veterinarian to respond in the case of an event such as an overturned trailer that involves livestock. They will expect that the veterinarian will know how to handle and treat animals with the normal standard of care. Do you have the necessary equipment (helmets, straps, etc) to safely respond? Can you quickly and efficiently perform euthanasia if indicated? Most veterinarians have built valuable relationships with the local farm co-operative store and county extension livestock agents for livestock resources. Know who the county emergency manager is, and get to know the local fire chief and sheriff’s office.

In addition to preparing their families and businesses for a disaster, food animal veterinarians can help their clients prepare for disaster events. What types of hazards are your community exposed to? In the northern states, this could mean preparing for inclement weather such as snow and ice storms. The loss of electricity, often seen following ice storms, can be devastating to a livestock operation. Are contingency plans in place to milk cows following a long-term power outage? Farm operations can be encouraged to develop emergency plans, such as having back-up power or “sister farms” to send animals to should the need arise. Livestock should be kept current on their vaccinations to protect them if they needed to be moved quickly. Permanent animal identification should be used to prevent livestock from being lost or stolen, as can be the case when fence lines are destroyed. In feeder-type operations, producers should know how much feed is on hand should there be a disruption in the transportation chain. In the southern states, hazards can include tornadoes, hurricanes, or heat-related events. Identifying alternative water sources can be a major issue for livestock producers living in coastal areas.

Private food animal practitioners are busy by nature, and usually have long unpredictable work hours. Veterinarians who wish to expand their disaster work beyond the private practice and the local area have many opportunities to do so, with involvement ranging from local to international activities. All of these opportunities require some time commitment on behalf of the veterinary volunteers. Since all disasters happen locally, veterinarians are encouraged to first become involved with the local animal response teams. These can be found by contacting local or state veterinary associations, state animal response teams (SART), or the state veterinarian’s office. Most communities also have some form community emergency response teams (CERTs), which can be found by contacting the local/state Emergency Management Agency (EMA). Local emergency planning committees (LEPC) or the local emergency management agencies are usually comprised of various community members, in addition to first responders and emergency management directors. Opportunities are also available at the national level through groups such as the USDA National Animal Health Emergency Response Corps (NAHERC), which deals with livestock issues, or the USDA DHHS National Veterinary Response Team (NVRT) or American Veterinary Medical Foundation-sponsored Veterinary Medical Assistance Teams (VMAT), which deal mainly with companion animals, and other private, non-profit organizations.

In summary, food animal veterinarians will likely find themselves in a disaster situation at some point in their career. Disaster readiness, which includes awareness and preparedness, is the most important phase of disaster management that will guide response and recovery activities. Whether a local or large-scale response is needed, veterinarians are expected to have an awareness of disaster management in addition to being prepared to provide the standard of care to animals of which we are all trained. Recognizing these expectations, as well as recognizing our limitations, will be critical to the successful mitigation and recovery of any disastrous event.
Emerging infectious diseases of food animals are a major threat to both domestic and global animal health. Many livestock disease can also be zoonotic, posing additional threats to ourselves, our clients, and our communities. Controlling these diseases is crucial to protecting animal health and the agricultural economy, as well as promoting public health and food safety and security.

Agriculture is a $1.24 trillion dollar industry in the US, accounting for 13.4% of the US GDP. Over 22 million people in the US are employed in farm or farm-related jobs. Clearly, the introduction or re-introduction of an emerging or exotic food animal disease could be detrimental to all levels of a community.

Diseases of animals and livestock that have been eradicated from the United States or diseases that have never been detected in the United States are termed as foreign animal diseases (FAD’s). Due to international standards, inspection procedures, biosecurity, and diligence on the side of individuals such as veterinarians and producers, we have been fortunate in the US that foreign animal diseases have not been recently introduced. Emerging diseases, however, continue to plague livestock systems in the US.

An emerging disease is a newly a new or newly identified pathogen or syndrome, or one that has resulted in new manifestations of an infectious disease. A re-emerging disease is a previously identified or known pathogen that is increasing in incidence, expanding to new geographical areas, or affecting new populations or species.

This session will review foreign FAD’s that pose a continual threat to the US, such as foot-and-mouth disease (FMD) and Rift Valley Fever (RVF), and provide an update on emerging or re-emerging diseases currently encountered in US livestock populations.

**Bovine tuberculosis**

Bovine tuberculosis (TB) is caused by *Mycobacterium bovis*. TB is a chronic, progressive disease of cattle which generally manifests as emaciation, low-grade fever, inappetence, and weakness. Pulmonary involvement is common and affected animals may have a chronic cough, dyspnea, or tachypnea. Lymphadenopathy may also be present. Cattle are the primary hosts for M. bovis, although other mammal species can be infected. Vaccinations are not available for cattle, although vaccines for wildlife are currently being developed and tested. Tuberculosis is zoonotic and can be transmitted from infected animals to humans through inhalation or the ingestion of raw milk.

Bovine TB has been the target of cooperative federal-state-industry efforts since 1917 with the establishment of the US Bovine Tuberculosis Eradication Program. Since that time, the prevalence has been reduced from 5% to approximately less than 0.001%. The disease has been mostly eliminated from US cattle populations, with the exception of a few areas. In addition to sporadic outbreaks in the Yellowstone basin, sporadic outbreaks of bovine TB continue to occur, most recently reported in Texas and Michigan cattle herds (2016). Epidemiologically, most infections in domesticated cattle herds have been traced back to either wild deer/elk populations or contact with Mexican cattle.

**Influenza viruses**

Influenza C virus (IVC) is predominantly a human pathogen, causing respiratory disease in infants and children. Infected swine show no to mild clinical signs. Transmission between humans and pigs is suspected, but the mechanism and direction are not clear. There is no evidence that IVC can be spread through the consumption of meat from infected animals. To date, IVC has only been isolated from pigs in China (1981).

Influenza D virus (IVD) has been recently identified in cattle and pigs (Oklahoma, 2011). Cattle are thought to be the natural reservoir of IVD, with occasional spillover into swine. Infected swine show no to mild clinical signs. It is not known if IVD is zoonotic, but it has been shown to infect ferrets experimentally, which are used as human models.

Vaccinations for livestock against IVC or IVD are not available. While no animal to human transmission of these viruses has been reported, given the potential for viral assortment, veterinarians and farm workers with increased exposure to livestock and poultry are at increased risk for potential zoonotic infection. The use of personal protective equipment such as masks in persons with close contact with sick livestock or poultry will help reduce the risks in susceptible populations.

**Seneca Valley Virus**

Seneca Valley Virus is a small non-enveloped picornavirus known to cause idiopathic vesicular disease in swine. It was identified in 2002, and has caused sporadic outbreaks in the US swine population. The virus has also been found in healthy pigs, and antibodies have been discovered in swine, cattle, and wild mice in the US. It causes lesions on the limbs, coronary bands, oral, mucosa, snout, and nares which are clinically indistinguishable from foot-and-mouth disease. Fever and lameness can also be seen. The transmission and pathogenicity of SVV are unclear, although swine are believed to be the natural host. There are no vaccines
available for SVV. While SVV is not currently reportable in the US (although several states do have requirements), clinical signs should warrant an FAD investigation. There have been no reported cases of SVV in humans.

**Vesicular Stomatitis**

Vesicular Stomatitis (VSV), caused by one of four named vesiculoviruses, is a viral disease found in the western hemisphere, endemic in warmer regions of North, Central, and South America. It mainly affects horses and cattle, although swine, sheep, llamas and alpacas have also been infected. VSV is spread through insect vectors such as sand flies, black flies, and culicoides midges. Once infected, direct animal-to-animal transmission can occur, although livestock are not believed to maintain the virus long-term. VSV causes vesicles, erosions, and ulcers on the mouth, feet, and udder, and is a reportable disease in many states due to the fact that it cannot be distinguished clinically from foot-and-mouth disease in cattle. Humans can rarely become infected with an influenza-like illness after handling affected animals.

Outbreaks of VSV have been seasonal in nature. Although recent years have seen an increase in the number of VSV outbreaks in the western US, there are currently no VSV-affected premises under quarantine in the US as of March, 2016. Recent outbreaks in horses and cattle, have been reported in Arizona, Colorado, Nebraska, New Mexico, South Dakota, Texas, Utah and Wyoming.
Evidence-Based Decision Making for Food-Animal Practitioners

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Food animal practitioners make many rapid clinical decisions about the animals under their care every day. Clinical decision-making in food animal veterinary medicine requires an understanding of many different concepts, including critical assessment of resources, causation and association, diagnostic testing, disease control and prevention options, treatment evaluation, and economical factors.

Evidence-based medicine and epidemiology are closely linked. According to Sackett et al, evidence-based medicine (EBM) is “the conscious, explicit, and judicious use of current best evidence in making decisions about individual patients…” Similarly, clinical epidemiology has been defined as “The science of making predictions about the individual patients by counting clinical events in other patients (or populations), using strong scientific methods for studies of groups of patients to ensure that the predictions are accurate” (Clinical Epidemiology: The Essentials. Fletcher, Fletcher and Wagner).

In food animal practice, where decisions will almost always impact a population of animals beyond the individual, evidence-based medicine and clinical epidemiology are critical disciplines. Evidence-based decisions are dependent upon three factors: the resources (or research) available, the patients or herds that we are working with, and our own clinical experiences and judgements. Each set of circumstances and experiences we encounter are unique and require individualized attention to decisions that are made. Regardless of these individual factors, general steps for veterinarians to use when answering a clinical question are as follows:

Ask an answerable question
The clinical question should be specifically defined in terms of the animal or population being cared for. Especially in food animal practice, communication with owners is essential to ensure we are supporting the owner's objectives and goals. Are we treating an individual animal to survive, or are we trying to decrease overall herd morbidity or increase production? The question should also include intervention strategies, treatment comparisons (where applicable, and desired outcomes. For example, the owner may ask "Will the metaphylactic use of antibiotic X compared to no metaphylaxis use result in fewer pulls in stocker cattle?" You may want to further define this question as to specify a reduction in treatment costs, reduction of morbidity, etc.

Search for the best available evidence to answer the question
Many resources are available to the veterinary practitioner, ranging from expert opinion, textbooks, and conference proceedings to scientific studies and meta-analyses. In school, we relied heavily on textbooks and notes from professors and "experts." In the past ten years, students and practitioners alike have also learned to rely on internet sources for textbooks as well as other sources information. As we've gone further along in our DVM careers, we rely less on textbooks and more on CE meetings, specialists, refereed journals, and of course, the internet. There seems to be an information overload, and not enough time to learn it all. Modern technology gives us new diseases, new diagnostics, new treatments, and new hypotheses every time we turn around. Therefore critical interpretation of the resources is becoming more and more important in evidence-based decision making.

Bibliographic databases of veterinary resources are available that include such as CAB (CABI), PubMed (US National Library of Medicine) and Scopus (Elsevier). Many of these databases have free full-text online access or are available through various institutions, organizations, or employers. Google Scholar can be another good resource, although one has to filter through the results wisely. Depending on your question, information may be scarce, especially as is the case in the most recent treatment or diagnostic methods. Sometimes the best evidence is expert opinion.

When performing a literature search, use the key words described in your clinical question. You may need to include synonyms or related terms. Keep in mind that terms used in veterinary medicine may be different than those used in human medicine, and other languages/countries may also differ in terminology. For example, "metaphylaxis" is not a concept that is utilized in many other countries, nor is it a term used in human medicine. Key words such as "prophylaxis" or "treatment" may also be useful.

Critically appraise the available evidence
All of the evidence you find to support your decision-making process should be evaluated for their strengths and weaknesses. Many cite the evidence pyramid, a representation of the general strength of evidence of literature and other resources. As you move up the pyramid, the general strength and quality of evidence increases. However, each type of evidence is not appropriate in every clinical situation, and quality of the resource may vary. For example, a well-performed case-control study maybe more valuable to the veterinary practitioner than a severely biased randomized controlled trial.
While assessing the strength of evidence obtained, three questions should be asked when reviewing the literature: 1) Are the results valid? Identify potential sources of bias such as lack of randomization and blinding, allocation concealment, intention to treat, follow-up, and conflicts of interest. For example, most of the research studies performed on newer food animal antimicrobials were performed by drug company sponsors. While this does not discount the validity of the work, it does justify a more critical evaluation of the work. 2) What are the results? Do authors report size of the effect, relative versus absolute numbers, p-values and confidence intervals? 3) Will the results help me care for my clients? Does the literature address similar populations, important outcomes, and individual (herd) values?

Your understanding of epidemiology really comes in handy when evaluating scientific studies. First of all, it is important to remember that association does not mean causation. Determining causation relies on many factors such as descriptive epidemiology, adherence to philosophies such as Koch's postulates and Hill's criteria, judgement and critical thinking, and of course statistics. Measures of the strength of an association (for example, between treatment and recovery) include descriptive data (morbidity, mortality, case fatality rates, etc), odds ratio, relative risk, attributable risk, relative risk reduction, correlation, etc. The measures of significance of an association are determined by statistical analyses often portrayed as p-values and confidence intervals. Are these measures used properly in the article you are reviewing? While a lot of emphasis is placed upon statistical significance, from a clinical standpoint one has to also remember that statistical significance does not necessarily equal biological significance. Critical evaluation of the literature may be one of the most overlooked, yet most important part of the decision-making process. With all of the information available these days, the busy practitioner has to learn how to quickly and efficiently sort the wheat from the chaff.

**Apply evidence in a clinical setting**

From an epidemiological standpoint, this is where your professional knowledge of the animal or herd will play a large part in the process of applying and evaluating outcomes. After sifting through the evidence supporting your decision, you have to make choices that are practical, efficient, and economical all the while maintaining our dedication to animal care, animal welfare, and public health.

**Evaluate outcomes**

Clinical outcomes need to be assessed following your decision. Are herd records available? Are the owners or managers willing to collect and share the outcome data? Veterinary practitioners need to work with the owners to define criteria for treatment “success” and monitor animal/herd performance. From this information, changes can be planned, improvement maintained, protocols established, and standards defined. Combined with clinical applications, outcomes assessment should be viewed as a continual process.

With the vast amount of knowledge available in veterinary medicine, food animal practitioners are challenged to keep up with the advances when making clinical decisions. At the same time, it is not feasible or practical to expect the busy practitioner to spend time evaluating every clinical decision he/she makes through EBVM. EBVM is move away from the traditional approach of textbooks and notes to a more progressive use of clinical research. While resources in veterinary medicine are not as available and abundant than those in human medicine, evidence-based veterinary medicine is a growing field, quickly gaining momentum in academic as well as commercial and private settings. Learning and applying EBVM approaches in food animal practice can benefit not only the herd owner, but the public and consumer as well through the application of best management practices.
New Drug Laws for Food-Animal Practitioners
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We enjoy one of the safest and most affordable food supplies in the world thanks to years of hard work by many – farmers, ranchers, veterinarians, processors, packers, distributors, government agencies, and others. So that we can continue this privilege, it is the responsibility of veterinarians along with livestock producers to understand and follow the laws and be prepared to meet the new and changing standards set in the years to come.

The US Food and Drug Administration (FDA) is responsible for protecting public health by assuring the safety, efficacy, and security of human and animal drugs, biological products, and the food supply, among other things. All antibiotics available for use in livestock - OTC, prescription (Rx), or through a veterinary feed directive (VFD), are used under FDA guidance and regulation. Depending on the product label, OTC and Rx products can be administered through the feed, water, or by injection. While the first two categories have been around for many years, the VFD option is a fairly recent one in livestock production. The Animal Drug Availability Act (ADAA), passed by Congress in 1996, established this new regulatory category for distributing certain animal drugs in or on animal feed. Examples of new animal drugs approved with a VFD label are florfenicol use in fish and tilmicosin for use in swine and cattle.

So why do we need more regulation on using the medicated feed products? There has been a growing concern that the use of antibiotics in veterinary medicine, specifically those used in food-producing animals, are contributing to the increasing problem of antibiotic resistance in humans. A recently released report by the FDA (Dec. 2015) stated that the majority of antimicrobial drugs approved for use in food animal species in 2014 were marketed for use in the feed (74%) or water (22%). Furthermore, the report indicated that 97% of these drugs were dispensed OTC, or without veterinary supervision.

Given this concern, recent regulations propose phasing out the use of medically important antibiotics in food animals for production purposes and to ensure that licensed veterinarians oversee other uses of such drugs (Figure 1). In 2003, the FDA published the Guidance for Industry (GFI) #152, which designated antimicrobials used in food animal species according to their importance in human medicine. In 2010, GFI #209 established two voluntary principles: the use of medically important antibiotic drugs in food-producing animals should be limited to uses that are considered necessary for assuring animal health (as opposed to growth promotion); and the use of medically antibiotic drugs in food-producing animals include veterinary oversight or consultation. The #213, published in 2013, provided the whereby makers of medically important could voluntarily change the drug’s label to claims for growth promotion and establish indications for therapeutic use in food-producing animals. GFI #213 was released as a final ruling in June 2015, and will become effective January 1, 2017.

After Jan. 1, 2017 the use of medications in feed that are considered “medically important” in human medicine will be restricted to

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**Table 1: Drugs Transitioning from OTC to VFD Status after Jan. 1, 2017**

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Examples of proprietary drug names</th>
</tr>
</thead>
<tbody>
<tr>
<td>chlortetracycline</td>
<td>Aureomycin, CTC, CTC, Clarotet, Chlormax, Chlortetracycline, Pennchlor, Deracin</td>
</tr>
<tr>
<td>chlortetracycline/sulfamethazine</td>
<td>Aureo S, Aureomix S, Pennchlor S</td>
</tr>
<tr>
<td>chlortetracycline/sulfamethazine/penicillin</td>
<td>Aureomix 500, Pennchlor SP</td>
</tr>
<tr>
<td>hygromycin B</td>
<td>Hygromix</td>
</tr>
<tr>
<td>lincomycin</td>
<td>Lincomix</td>
</tr>
<tr>
<td>oxytetracycline (OTC)</td>
<td>TM, OXT, Oxytetracycline, Pennox, Terramycin</td>
</tr>
<tr>
<td>oxytetracycline/neomycin</td>
<td>Neo-Oxy, Neo-Terramycin procedures</td>
</tr>
<tr>
<td>penicillin</td>
<td>Penicillin, Penicillin G Procaine, GFI antibiotics</td>
</tr>
<tr>
<td>sulfadimethoxine/ormetoprim</td>
<td>Rofenaid, Romet</td>
</tr>
<tr>
<td>tylosin</td>
<td>Tylan, Tylosin, Tylovet phase out</td>
</tr>
<tr>
<td>tylosin/sulfamethazine</td>
<td>Tylan Sulfa G, Tylan Plus Sulfa G</td>
</tr>
<tr>
<td>virginiamycin</td>
<td>Statacin, Virginiamycin, V-max</td>
</tr>
</tbody>
</table>

*Additional drugs may be approved but are not currently marketed for livestock.
Adapted from FDA CVM. Most current list of VFD drugs can be found at: http://www.fda.gov/AnimalVeterinary/DevelopmentApprovalProcess/ucm482107.htm
treatment, control, or prevention of bacterial diseases of animals, under the oversight of a veterinarian. This removes the growth promotion, feed efficiency, and milk production uses from the labels of all currently approved medically important drugs used in feed. Furthermore, any listed "medically important" antibiotics currently available in the feed for animals will require a VFD (Table 1). In addition, medically important antimicrobials currently available to be used in the water will require a veterinary prescription.

Producers will be required to obtain a VFD from their veterinarian for medicated feeds such as CTC (chlortetracycline) for anaplasmosis control and medicated milk replacers. Ionophores, coccidiostats, and anthelmintics, as well as a few other antimicrobials will be exempt as they are considered “not medically important” in human medicine. The VFD’s must be filled through an approved feed mill or feed distributor. As long as the feed distributor (co-op, feed mill, etc.) has a “letter of intent” on file with the FDA, and an “acknowledgement letter” with the drug supplier or feed manufacturer, they will be able to keep a stock of medicated feeds on hand to distribute to customers with a valid VFD. A VFD can be written for multiple locations within a state owned by the same person as long as the feed is acquired from the same distributor.

Veterinarians will be responsible for all information contained on the VFD. The veterinarian must be licensed in the state where the animals reside. A VFD will have both an expiration period and a duration of use specified on it. The expiration period refers to how long the VFD is valid for. This will be determined by either the product label or by your veterinarian after evaluating the medical needs of your animal(s), and cannot exceed a period of 6 months. The duration of use refers to the amount of time an animal or group of animals should be fed the medicated product. The duration of use will be specified by the labelled directions for that particular drug. Additional details such as number and type of animals being treated, condition being treated, and level of antibiotic to be delivered to the animals will also be required on the VFD. Telephone orders will not be allowed, although electronic means may be used to deliver the initial VFD request. The producer, veterinarian, and feed distributor must keep records for 2 years. VFD manufacturers must keep product manufacturing records for 1 year.

Dispensing, prescribing, or authorizing a prescription or VFD product requires a valid veterinary-client-patient relationship (VCPR). It is illegal for a veterinarian to dispense or write a prescription or VFD for an animal/ herd they have not seen or are unfamiliar with. A VCPR is important for both veterinarians and livestock producers because it communicates a type of “agreement” between parties on the responsibility and care for the animals. Under the guidelines of the FDA Animal Medicinal Drug Use Clarification Act (AMDUCA), a VCPR exists when all of the following conditions are met (Title 21, Code of Federal Regulations, Part 530 or 21 CFR 530):

- A valid veterinarian-client-patient relationship is one in which:
  a. A veterinarian has assumed the responsibility for making medical judgments regarding the health of (an) animal(s) and the need for medical treatment, and the client (the owner of the animal or animals or other caretaker) has agreed to follow the instructions of the veterinarian;
  b. There is sufficient knowledge of the animal(s) by the veterinarian to initiate at least a general or preliminary diagnosis of the medical condition of the animal(s); and
  c. The practicing veterinarian is readily available for follow-up in case of adverse reactions or failure of the regimen of therapy. Such a relationship can exist only when the veterinarian has recently seen and is personally acquainted with the keeping and care of the animal(s) by virtue of examination of the animal(s), and/or by medically appropriate and timely visits to the premises where the animal(s) are kept.”

Depending on your state, the federal or state definition of a VCPR will apply when writing a VFD. Many state veterinary boards have defined the VCPR as part of their practice acts, requiring the existence of a VCPR prior to a veterinarian diagnosing or treating an animal, or prescribing or dispensing medications. If the FDA has determined that the state definition of VCPR is not sufficient for VFD purposes, then the federal definition will apply. A list of states and their VCPR status can be found on the FDA’s website. Organizations such as the American Association of Bovine Practitioners also provide guidelines for establishing and maintaining a VCPR in bovine practice, including maintaining written agreements with producers and maintaining relationships with consultants and other veterinarians. As written, the federal VCPR may be subject to various levels of interpretation.

Veterinarians should review their state’s practice act and be familiar with the VCPR as it applies to them and their responsibilities to dispense and prescribe medications. In addition, veterinarians can work with their clients to evaluate current and future medicated feed usage. Principles of judicious use of antimicrobials should be reviewed, and continued evaluation of herd health management practices will be essential. Focusing on disease prevention strategies such as calving management, fly control and low-stress preconditioning programs for calves, may reduce the need for antimicrobials. Evaluate your own medical record-keeping practices, as well as your clients’, since new rules will require additional recordkeeping and documentation. Finally, understand the VFD process so that future implementation of the new drug laws in your practice will be as smooth and painless as possible.
By definition, biosecurity refers to measures taken to prevent the introduction or re-introduction of diseases into susceptible populations. As food animal veterinarians, we are responsible for applying biosecurity principles to protecting both animal health and human health. Specific reasons for protecting animal health may include decreasing illness and discomfort, decreasing treatment costs, decreasing production losses, decreasing discarded meat, and decreasing livestock death. Ways to protect human health may include decreasing potential zoonotic pathogens (harmful organisms that can be transmitted from animals to humans) and ensuring a safe, wholesome human food supply.

- But what does this mean in the “real world”?
- How does it fit in your daily work routine?
- And whose job is it anyways?!

Biosecurity has many meanings, and biosecurity concepts can be applied at the national, state and local/premise levels. Emphasis is placed on local and individual premise biosecurity planning since those are areas most likely to be influenced by the private veterinary practitioner. An overview of biosecurity will be provided, followed by large animal examples and discussions.

National level biosecurity
With over 250,000 people entering the United States daily, there is a need to protect our agriculture industry from foreign animal diseases and invasive pests that may be intentionally or unintentionally introduced. One of the major components of national level biosecurity in animal populations is in inspection and quarantine activities. In addition to the USDA APHIS VS, many agencies are involved in agrosecurity, including the USDA Food Safety Inspection Service, USDA Plant Protection and Quarantine, US Department of Homeland Security, and the US Department of Justice. An awareness and recognition of potential agroterrorism and bioterrorism agents by not only these agencies, but by private veterinarians, also helps protect our national borders. Veterinarians will be the first line of defense in the recognition and control of potential foreign and emerging animal diseases. Given that many diseases of concern are endemic to other countries, the risk of disease introduction due to increased globalization and international travel is real.

State level biosecurity
State-level biosecurity measures protect a state’s agriculture industry through programs such as disease monitoring, control and eradication programs, and the issuance of health papers (CVI’s) and movement permits. Furthermore, notifiable disease reporting and syndromic surveillance activities can help protect a state's livestock population. Veterinarians are encouraged to report unusual animal illnesses or deaths to the state veterinarian, and report suspicious vehicles or persons to the local law enforcement agency.

Local/premise level biosecurity
Local level biosecurity refers to the premise level, which includes your client’s operation as well as your veterinary facility. The term biocontainment is used when referring to measures taken to prevent the spread of disease within a premise. For example, the practice of removing dairy calves at birth to reduce pathogen transmission from its dam is considered biocontainment. Biosecurity refers to measures taken to prevent the spread of disease between premises. An example of this would be the washing of boots and equipment between farm calls to reduce any infectious disease spread (and as persons who have multiple animal contacts on multiple farms, we could be considered "high-risk" individuals when it comes to biosecurity). These two words are often used interchangeably.

Biological risk management (BRM) refers to the overall process of awareness education regarding the risk of infectious diseases entering or spreading through an animal facility. BRM recognizes that disease on an operation cannot be entirely eliminated, but the risks associated with those diseases can be managed through effective control measures.

Controlling risk of disease, or BRM, can be done through several methods, including creating HAACCP plans or performing risk analyses. The risk analyses process is an easy, flexible yet methodical way to identify, control, and evaluate biosecurity measures. The three stages to a risk analysis are risk assessment, risk management, and risk communication.

1. Biosecurity risk assessment: The risk assessment involves the identification of disease hazards, assessment of disease exposures and potential consequences, and characterization of disease risks (usually high, medium and low). This can be done by performing a thorough herd history, and examining the components of the epidemiological triad of agent, host, and environment.

   a. Agent: What diseases do you need to protect the herd from? How are they transmitted? And what is the likelihood of them being encountered in a specific population? Agents can be characterized according to
probability and impact. For example, the probability of encountering FMD on a dairy operation may be low, but the impact would be huge.

b. Host: What populations of animals (or people) are you trying to protect? Who are considered "high risk" versus "low risk" for disease? Factors such as age and immune status should be considered.

c. Environment: What is the potential for disease exposure in the host population? What is the source of new additions? Do animals travel for breeding or exhibition purposes? Does the operation practice all-in-all-out management?

Economic assessments and cost-benefit options are very important. There are costs of diagnostic testing, costs of preventive measures such as vaccinations, and costs of treatment options, as well as benefits in terms of increased health and productivity. In most cases, it may not be cost-effective to implement all possible biosecurity measures. Remember, you can’t protect everyone from everything!

2. Biosecurity risk management: Risk management involves establishing herd health goals from the risk assessment, determining risk reduction strategies that are practical and effective, implementing needed improvements, and evaluating the strategies. The objectives and goals of the herd owner must be considered, as well as the level of aggressiveness desired (prevention, control, or elimination of disease). In other words, this is the action part of your biosecurity plan. How are you going to reduce the risks of disease on or to your client's farm, and how are you going to monitor your actions? It is important to realize that this is a changing process – biosecurity risk management procedures may cause you to modify your biosecurity risk assessments, and vice versa. Specific areas to consider include isolation and quarantine procedures, visitor and traffic control, and sanitation and disinfection measures.

3. Biosecurity risk communication: Risk communication is the third step to the biosecurity risk analysis process. It is important to document and share your plans. If your clients and their employees and visitors are unaware of the biosecurity plan, it is not likely to be followed. If you don’t share your plan, it’s no good. Specific practices to consider include fostering interaction with the farm management team, education of the team, development of standard operating procedures, evaluation of management practices and production records, and reassessment.

Biosecurity risk analyses can be agent specific (e.g. BVDV, Johnes), industry specific (e.g. heifer-rearing operations, sow units), or situation-specific (4H events). In reality, veterinary practitioners are rarely called out to farms or operations to perform biosecurity risk analyses unless they are involved in a disease control program such and BVD or Johnes's disease where an assessment is incorporated into the program guidelines. Most times veterinarians are called out to investigate disease outbreaks or losses of production that may have resulted in biosecurity breaches. These outbreak investigations provide a good opportunity for the herd veterinarian to discuss biosecurity and biocontainment and perform a risk analysis.

Finally, how good are your biosecurity practices? As veterinarians, we have routines that we follow on a daily basis. We should periodically evaluate our own biosecurity practices, and ensure that our employees adhere to them. Everyone has a role in biosecurity. A risk analysis can be performed in your clinic to help identify areas of risk and potential mitigation options. By following national and state biosecurity practices, and providing a good example to our employees as well as our clients, we are demonstrating a commitment to health and productivity at all levels.
Food animal veterinarians are facing challenging times. Balancing animal care, public health, and business sustainability (for ourselves as well as our clients) affects every decision that we make. In addition to common diseases of production such as acidosis and lameness, concerns such as emerging diseases and new animal drug laws must be considered in our herd health plans. We find ourselves having to work within the reality of the present agricultural economy to help keep our clients in business and ensure a sustainable food supply beyond the farm gate. As food animal production is becoming more complex, we find ourselves shifting from individual animal medicine to herd health and production management.

As busy practitioners, finding time to really talk to clients can be difficult. But before a veterinarian can truly evaluate herd health and production, they have to understand the goals of the operation. Is the business the primary source of income, a secondary source of income, or a hobby? The business plan is a good place to start the discussion; if one is not in place, the veterinarian can help assemble the team to assist the owner in creating one. Extension specialists, nutritionists, bankers and family members may all be included. It’s important that everyone is on the same page as to the goals of the operation. For a cow-calf operation, for example, an understanding of the marketing options is necessary to create herd health protocols.

In addition to understanding the goals of the operation, we must have a good understanding of the operation's production calendar. Research has shown that in order to be productive, beef and dairy cows should calve at 2 years of age, and have a calf every year. Thus herd management is a constant cycle centered around two main overlapping seasons - breeding season and calving season. All of the other herd health management decisions are made around these two times, which often times get made according to other agricultural commodities or off-farm employment obligations.

While it may seem simplistic, setting up a herd health calendar with your clients can help to maintain their production goals. This will also help us as veterinarians to make more meaningful suggestions and contribute to the overall management of the herd. While there are no “cookbook” health programs, visualizing the calendar can keep management practices on time, promoting efficiency and production. The calendar can be as basic or complex as necessary to fit the needs of the client. At minimum, the calendar should include both the breeding season as well as the calving season (Fig. 1). More extensive calendars may include nutritional information such as pasture rotation and supplemental feeding. While the breeding and calving calendars overlap, we will discuss each of these separately in order to emphasize opportunities for veterinarians to be involved in herd health management.

**Breeding season**

- Producer should establish a definite 75- to 90-day calving season that will efficiently use available resources. If a bull is used, have definitive in/out dates.
- A Breeding Soundness Exam (BSE) should be performed on bulls prior to each breeding season. This includes a complete physical examination and semen evaluation.
- Ensure the right bull/cow ratio is used so the majority of the calves are born early in the calving season.
• Perform reproductive exam (BSE) on replacement heifers, including reproductive tract exam and pelvic measurements. First-calf heifers should be bred to calve 1 month before the main herd. Heifers should reach 65 percent of expected mature size by first breeding at 14 to 15 months of age.
• Pre-breed vaccinate cows against IBR, BVD, BRSV, PI3, leptospirosis, and campylobacteriosis not less than 30 days prior to start of breeding season. Discuss MLV versus killed vaccinations with producer.
• Treat for internal and external parasites.
• Check body condition score (BCS) and soundness of brood cows (for example, teeth, eyes, udders, reproductive tracts). Cull open, poor-producing, unsound cows.
• Observe bulls and cows during breeding season. Record breeding dates to detect any breeding problems.
• Pregnancy check cows. Revaccination, deworming mid-gestation or at time of pregnancy check may be indicated. Bleed cows for disease testing if indicated (Johnne's, brucellosis, etc.).
• Vaccination against scours (rotavirus, coronavirus, clostridial organisms, E. coli) should be given approximately 1 month prior to calving if utilized.
• Remove bulls from cow herd to maintain a tight calving season.

Calving season

• Producer should provide a clean, well-drained calving pasture convenient for frequent observation and with easy access to working facilities. Discuss the sand hills calving system or similar pasture utilization techniques to reduce the chance of calf scours.
• Recommend dehorning and castrating calves at birth, or as early as possible (unless seedstock).
• Calves should be ear-notched for BVD-PI at birth or at preconditioning.
• Develop a preconditioning program for calves, depending on management and marketing plans. Discuss MLV versus killed vaccinations with producer.
• Pre-wean vaccinations - IBR, BVD, BRSV, PI3, clostridial organisms
  o Prefer two sets of vaccinations prior to weaning for best immunity
  Optional: Mannheimia (Pasteurella), Histophilus (Haemophilus), brucellosis (heifers 4-12 mos of age)
  o Deworm/fly control
  o Dehorn, castrate if not done at birth. Discuss analgesia and anesthesia in older animals.
• Evaluate BCS of cows. Producer may consider creep feeding calves to improve BCS of cows.
• Wean calves. Advocate low-stress handling of cattle at all times.

We can also help our clients be more efficient and productive by providing assistance in evaluating herd records. Records will help us determine if breeding and calving goals are being met, and to set standards and goals for the next season. Many of our clients collect data; the problem is that they do not do anything with it. Our role as food animal veterinarians should be to assist the producers in using their records to make sound decisions based on production and economics.

Your involvement in the activities listed on the herd health calendar will depend on the experience and objectives of the producer. Communication between veterinarians and producers is essential to the veterinary-client patient relationship. By sitting down and discussing the herd health calendar with our clients, we will gain a more complete understanding of the operation and be more able to make decisions and provide recommendations.
We are seeing the US cattle herd rebuilding at astonishing rates, likely in part due to last year’s favorable cattle prices. According to the National Agriculture Statistics Service (NASS), there were close to 90 million cattle and calves, including over 9.3 million dairy cattle and 30.3 million beef cows on inventory in the US in January 2016. All livestock numbers, including sheep and goats, appear to be increasing over the past several years. More of our clients are retaining heifers and (hopefully) investing in the future of the herd. Reproductive diseases can put a quick halt to our clients’ progress. Sometimes it’s easy to forget about those diseases that show few clinical signs, are not easily detectable, and don’t have a reliable vaccine for. Two such parasitic disease that affect cows and bulls, respectively, are neosporosis and trichomoniasis.

**Neosporosis**

Neosporosis, caused by the protozoan parasite *Neospora caninum*, can occur as an endemic, epidemic, or sporadic disease in cattle herds. Clinical signs of bovine neosporosis include premature calves, abortion, and stillbirth. Cows of any age may abort between 3 and 9 months of gestation, however, most commonly between 5 to 7 months. Fetuses may abort, be resorbed, become mummified or autolysed, stillborn, or born alive with or without clinical signs. Persistent infection occurs in those animals born infected without clinical signs, and those calves can then transmit the parasite to their own offspring during gestation. Although the reproductive signs are most notable and well-documented, little is known about the effects of infection on the production of infected animals and their offspring.

*N. caninum* has been well documented as a cause of bovine reproductive disorders. The dog has been identified as the definitive host for the parasite, although other wild animals such as raccoons, deer, grey foxes and coyotes have been implicated in the sylvatic transmission of the parasite. Transmission of the parasite occurs only through the ingestion of infected oocysts or through vertical transmission from the dam to the fetus. Animal to animal transmission of the parasite has not been reported.

Worldwide, it is estimated that 2/3 of *N. caninum* infections are found in dairy cattle, while 1/3 are found in beef cattle. A recent systematic review estimated that annual losses on beef farms worldwide were $150 per operation and $5.00 per pregnant cows and heifers. Economic analysis of the effects of neosporosis include estimates of abortion losses, early fetal death, stillbirths, increased culling, reduced production, and reduced value of breeding stock. However, few epidemiological studies have been done to quantify the true economic significance of the disease on overall livestock production.

The serological status of animals in a herd at risk for infection must be determined prior to recommending prevention and control options. It has been shown that congenitally infected heifers in herds with endemic infections are more likely to abort than non-infected heifers. Following an outbreak of neosporosis in beef cattle, it was suggested that cows with serological evidence of previous exposure to *N. caninum* were less likely to abort or have premature calves. Bovine abortions caused by *N. caninum* may show epidemic as well as endemic patterns, emphasizing the need for monitoring and surveillance in herds which have experienced outbreaks. Thus, the economic effects following an initial outbreak may be seriously underestimated given the chronic nature of the disease.

Diagnosis of *N. caninum* can be challenging. Serology can be performed on cows and fetuses, and lesions may be detectable through immunohistology. PCR may also be used. Bulk milk testing has been used to estimate herd prevalence in dairies. Seropositive animals are more likely abort than seronegative animals, however, asymptomatic congenital infections are common, and identification of the parasite or evidence of infection does not necessarily equal causation. Caution must be used in interpretation of results since detectable concentrations of circulating antibodies to *N. caninum* may also fluctuate over time, changing the serologic status of individual animals over time. Oftentimes the diagnosis of neosporosis as cause of a reproductive failure requires the ruling out of many other factors, which can be expensive and time-consuming. An examination of the other risk factors and overall herd management will assist in making further diagnostic and clinical decisions.

While several North American studies have examined the effects of *N. caninum* infection in beef cattle production, the majority of results have been inconclusive or contradictory. Reduced performance of positive calves in the feedlot has been observed, implicating the possible role of subclinical infection on production in calves. A longitudinal study in Texas, based on a single serologic assay at entry, found that feedlot calves with antibodies to *N. caninum* showed significant reductions in postweaning weight gain, carcass weight, and economic return. Another study showed that short-term reductions in weight gain and feed efficiency were associated with antibodies to *N. caninum* in weaned beef steers. However, there were no significant effects of serological status on average daily feed intake. A study in Mississippi demonstrated that seropositive cows had calves with heavier weaning weights, and seropositive
calves had heavier weaning weights than their seronegative counterparts. Positive effects on production were also demonstrated in beef cattle studies in Canada.

There are currently no approved vaccines or treatments available against *N. caninum*. Toltrazuril and ponazuril have been shown to be experimentally effective against the tachyzoites in calves. Recent studies have also examined the use of monensin in cattle as a prophylactic measure; however, results are not conclusive. It is also important to remember that any off-label use of medicated feeds is strictly prohibited. Prevention of neosporosis thus must focus on biosecurity and environmental management, as well as a targeted test and cull strategy where indicated.

**Trichomoniasis**

Trichomoniasis is a true venereal disease caused by the protozoan organism *Tritrichomonas foetus* and spread solely through sexual contact. In cows, the organism lives in the vagina, cervix, uterus and oviducts. Infected cow herds experience infertility, pyometras, and early abortions. Breeding usually results in conception, but pregnancy losses occur during the first few weeks of gestation, resulting in repeat breeders and inconsistent calving distributions. Sometimes, cows have a white vaginal discharge following initial infection. From a veterinary standpoint, you will likely be approached by the concerned client who is seeing his cows return to heat or fail to calve.

The reservoir for this organism is the persistently infected bull, and the addition of infected bulls or cows into a herd is the most common source of introduction. The organism lives in the internal sheath and skin folds of the bull’s penis. As bulls age, the skin folds grow, giving the organism more places to grow and thrive. Therefore, older bulls (that is, bulls more than 4 years old) are more likely to be persistent or chronic carriers of the disease. In older bulls, it is a life-long infection and can be transmitted from one breeding season to another if left undetected. Younger bulls can become infected, but usually clear infection after a period of time. Trichomoniasis causes no clinical signs in the bull and does not affect sexual behavior or semen quality.

Identifying persistently infected bulls can be difficult, as the routine breeding soundness exam (BSE) will not detect any abnormalities. All newly introduced non-virgin bulls over 18 months of age should be tested for trichomoniasis. To diagnose infection, a preputial scraping or preputial wash is taken from the sheath of the bull’s penis, at the fornix area of the prepuce. A dry or wet scraping technique can be used. If pooling samples from bulls, take samples individually and have the lab perform the pooling. The sample is placed in culture media, which should be kept at the proper temperature at all times. Proper handling is essential - there are many factors known to contribute to the inaccuracy of diagnostic testing, such as: 1) method of specimen collection; 2) type of transport medium; 3) transport time and temperature; 4) and incubation conditions. The failure to identify organisms in a positive animal can be detrimental since the presence of an infected animal in a herd allows infection to propagate.

A culture or polymerase-chain-reaction (PCR) test is then performed on the sample. Three separate culture tests, each conducted not less than one week apart, or one PCR test should be performed. Individual states may have different requirements for trichomoniasis testing of bulls for interstate movement, although an attempt is under way to harmonize state requirements. A bull undergoing three separate culture tests must be negative on each test to be considered free of trichomoniasis. Cultures taken at 1-week intervals for 3 weeks will identify most infected bulls. Studies have shown that a single culture will miss about 10 to 20 percent of infected bulls. Culture and PCR both have reported sensitivity of 65-96% and specificity of 96-98%. Therefore, current testing recommendations suggest that you can be approximately 99 percent sure that your bull is negative after 3 negative tests.

Reasons for false negative test results include: failure to collect the organism from the prepuce, improper sample handling, degradation of DNA, presence of blood in the collected sample, and interference/cross reactivity with natural flora of the animal. False positives can also be economically harmful as these animals may be subject to unnecessary and often expensive repercussions such as retesting, quarantine and isolation, and removal from the herd. The presence of fecal trichomonads, as well as cross-contamination of samples, can lead to false positive results.

There are no approved treatments for infected bulls or cows. A vaccine is available for cows against *Tritrichomonas*, and studies suggest that the vaccine may decrease the duration of shedding and prevent some pregnancy losses. Given the lifelong nature of bull infections and lack of approved treatments, slaughtering infected bulls is recommended. Cows often clear the infection naturally with several months of sexual rest; however, immunity does not last long and they are susceptible to reinfection.

As a sexually transmitted disease, trichomoniasis can be controlled by good biosecurity and breeding management. Artificial insemination using semen from reputable bull studs is an effective way to prevent sexually transmitted diseases. Rare cases of disease transmission through contaminated semen or AI equipment have been reported. To prevent the spread of the disease when breeding cows naturally, recommend the use of virgin bulls that have never been pastured or housed with a bull that has bred a cow. If non-virgin bulls must be brought onto the farm, follow a rigorous testing and quarantine protocol.

To control trichomoniasis in an infected herd, identify positive animals and remove positive bulls. If one bull is found infected, then the rest of the herd is probably also infected. Quarantine and enforce sexual rest for females exposed to or suspected of carrying the disease. Quarantine alone will not protect the herd because there are few symptoms to help identify infected animals.
In summary, neosporosis and trichomoniasis continue to be significant causes of losses in cattle operations in the US. Neosporosis remains a leading cause of abortion in beef and dairy cattle for many reasons, including the inability to control canine exposure and the challenges in diagnosis. Trichomoniasis remains a costly venereal disease of cattle and the target of many state import regulations. Diagnosis may be challenging, and current methods for the detection of the protozoan organism *Tritrichomonas foetus* may be ineffective in preventing the introduction of infected bulls into non-infected herds.

An understanding of the epidemiology of the diseases will help you assist your clients in making decisions on control and prevention of the parasite. Consulting with your clients on overall good management practices can also help control reproductive diseases: maintaining a defined breeding season and performing pregnancy exams, culling open cows, not purchasing older cows or bulls, performing a breeding soundness exam (BSE) on all bulls before the breeding season, and not sharing or leasing bulls. With the increase of interstate trade and rebuilding of the US beef herd, it is important to keep these two diseases in mind when making herd health and biosecurity recommendations.
Veterinarians play a huge role in protecting consumer confidence in the food supply through participation in programs such as the Beef Quality Assurance (BQA), Dairy BQA (DBQA), Milk and Dairy Quality Assurance (DQA). According to the 2011 National Beef Quality Audit, food safety was the top quality challenge reported to the industry, which includes both fed and non-fed (cull or market) beef and dairy cattle. It is interesting to note that non-fed cattle, including cull dairy cattle, make up 15-20% of the US supply of beef. By uniting veterinarians, animal scientists, feed suppliers, animal health companies, packers, retailers and state and federal regulators with producers, the quality assurance programs act as catalysts to encourage use of the latest science and technology to meet expectations about beef and dairy quality and safety.

Quality Assurance guidelines are intended to provide consistency in good management practices, and include guidelines on feedstuffs, feed additives and medications, processing/treatment records, injectable animal health products, and care and husbandry practices. The programs also ask everyone involved with beef and dairy production to follow the FDA/USDA/EPA guidelines for product use and to use common sense, reasonable management skills, and accepted scientific knowledge to avoid product defects at the consumer level. Every management practice that you perform on or recommend for your client’s cattle may affect the wholesomeness and quality of the final meat or milk product. As veterinarians, it is our responsibility to ensure our clients also understand the importance of these practices.

Quality Assurance programs consist of training in three common areas based on good management practices (GMP’s) for livestock: targeted breeding, proper management and responsible culling.

Targeted breeding in the herd leads to customer satisfaction, concentrating on desired meat and milk products as the ultimate consumer endpoint. As veterinarians we can help our clients set production goals and develop breeding programs using known genetics and a defined plan to meet these goals. We can also help clients develop good reproductive health protocols to meet our breeding, conception, and production goals, which may include heifer reproductive tract exams and effective synchronization programs.

Proper management practices enhance beef and dairy quality and product value, and assist in preventing problems rather than fixing them. Adopting good management practices will help your clients improve on things that they may already be doing, for example, by providing processing, medication and record-keeping guidelines. As a veterinarian, you are the most qualified person to perform risk assessments and make health management recommendations on an operation. A big part of proper management is ensuring that animals are managed and handled humanely, given the needed food, water, and care to protect their health and well-being. This care should be extended into the transportation period, minimizing stress and providing safe holding facilities.

Whether you are performing procedures or simply providing guidance to your clients, make sure your clients understand how to properly store, handle, and administer biological products. They should understand how to read a feed or biological product label before using any product. Now is also a good time to discuss the need for a veterinary feed directive (VFD), since the regulations will be changing, or alternative disease prevention techniques. Discuss the need for pain control or anesthesia when dehorning or castrating animals.

Responsible culling improves herd productivity and efficiency by ensuring that your clients don’t keep their problem animals. This includes giving attention to quality control points such as body condition, reproductive management, and disease control. The monthly (dairy) or annual herd check (beef) is a great time to show your producers how to mouth cows to estimate age while discussing culling and nutrition. Examining additional physical traits such as udder, eyes, feet and legs may help make other retention decisions. Diagnostic testing is just another area where the veterinarian is invaluable – diagnosing an open cow through pregnancy testing can save your clients in feed and maintenance costs. Removing animals testing positive for diseases such as Johne’s and BVD can also save money in treatment and replacement costs in the long run. Cattle suffering from chronic or incurable injury or disease should be culled early. Management of downer animals is becoming an increasing concern, often exaggerated by hidden videos showing the inhumane treatment of cattle. Educating clients on how to handle and when to cull or euthanize downer cattle is an important component of quality assurance. Euthanasia should be only be performed by persons trained in the proper methods.

The overall goal of the beef quality assurance programs is to assure consumers that all cattle shipped from any beef production facility are healthy, wholesome and safe. Likewise, dairy quality assurance programs are based on the delivery of safe and wholesome dairy beef and dairy products. The animals should have been raised in a proper manner, following good management practices. We also need to assure consumers that animals have been raised humanely and under acceptable standards of care. Many of the QA herd health guidelines are commonplace in the daily activities of a food animal veterinarian: proper use of vaccinations to prevent disease, proper care of biologicals, and using good biosecurity protocols, to name a few. BQA is designed to enhance carcass quality by
preventing residues, pathogen contamination and carcass defects such as injection site blemishes and bruises. However, as veterinarians it is also our responsibility to ensure our clients also understand the importance of these same practices.

New BQA guidelines published last year have established recommendations for pain control and humane euthanasia in cattle, among other things. When was the last time you recommended pain control prior to castration? Or discussed humane euthanasia with your cattle clients? Another hot topic in the livestock industry is the new veterinary feed directive (VFD) regulation that will be implemented in December 2016. Now more than ever it is important that we are prepared to discuss the newest issues impacting quality assurance with our livestock producers.

Residue prevention is an important component of the DBQA and DQA programs. One important theme of all BQA programs is the importance of establishing a valid veterinary-client-patient relationship (VCPR). The veterinary-client-patient relationship (VCPR) is a term used to define the relationship between veterinarians and their clients. Many state veterinary boards have defined the VCPR as part of their practice acts, requiring the existence of a VCPR prior to a veterinarian diagnosing or treating an animal, or prescribing or dispensing medications. Maintaining a valid veterinary-client-patient relationship will help ensure that drug administration and withdrawal recommendations are followed.

So where does beef and dairy quality assurance fit into your food animal practice? Hopefully, the answer is everywhere. As veterinarians, it is our responsibility to promote consumer confidence in the food supply by ensuring beef and dairy products are wholesome and safe. Quality assurance programs aren't just for producers - veterinarians play a key role in the education of clients, as well as in performing good management practices. If you have never been certified through a beef or dairy quality assurance program, or if it has been awhile since you took a certification course, now is a good time to get updated on some really important cattle health and QA topics. As a veterinarian, you are encouraged to become a certified QA Trainer in your community and take advantage of opportunities that can positively impact the local livestock industry. Veterinarians can also play an integral part in farm audits in the beef and dairy industry. Through the national BQA program, audits are currently available for feedlots and stocker operations. Through the DQA programs, audits are available for milk and heifer rearing facilities. Remember, as food animal veterinarians, we are responsible for helping our clients deliver a safe and wholesome food supply. Veterinarians, seedstock, cow-calf and dairy producers, stocker operators, and feeders all must take responsibility for the production of a safe food product through proper animal care, handling, and management practices.
Advanced Diagnostic Techniques in Respiratory Disease

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While frequently the individual animal and/or herd is treated for respiratory disease based on history and physical examination findings alone, there are certain situations where advanced diagnostics are indicated. With the exception of thoracic radiography in adult bovines, all of these techniques could be performed in the field if the equipment is available (for example endoscopy and ultrasound). Advanced diagnostics may be employed to gain a sample for further diagnostics in cases of failure of treatment or vaccine breaks; to identify pathogens prior to design of a vaccine protocol; confirm presence of pleural fluid or a pneumothorax based on absence of lung sounds on auscultation and further evaluate the upper airway using endoscopy in cases of stertor.

This lecture will cover review of auscultation of the respiratory tract and describe how to perform each technique and the situations in which they are indicated.

Advanced clinical examination of the respiratory tract
Good auscultation of the respiratory tract requires a quiet background noise level. In tractable animals, abnormal lung sounds may be able to be detected by use of a rebreathing bag. This can be achieved by placing a rectal sleeve or feed sack over the nose of the bovine. The increased level of CO₂ will stimulate deeper respiratory effort. Increased loudness of breathing sounds can occur as a result of increased respiratory rates (pain, excitement, high environmental temperatures) or pathological conditions. Sound is more efficiently transmitted through dense material; therefore, increased lung sounds are often consistent with lung consolidation. Percussion of the thorax can help in identifying transition from aerated to non-aerated lung. The thorax should be systematically percussed either on each rib or each intercostal space. Normally there is decreased resonance from dorsal to ventral over the thorax. Dullness is also heard over the heart, and other solid organs (liver/spleen). Increased resonance may indicate pneumothorax, while decreased resonance may indicate pleural fluid or lung consolidation.

Thoracic ultrasound
The major advantage of ultrasonography is because it is a non-invasive technique. Ultrasound waves cannot penetrate through air, which causes reverberation artifacts, therefore when ultrasounding the ‘lungs’ we are only able to penetrate to the surface of aerated lung. In normal lungs, this results in the ability to assess the pleural surface only. The penetration depth in most cases is therefore approximately 5cm. At this depth a frequency of 5-7mHz will be sufficient. Therefore, ultrasound of the thorax can easily be performed with a rectal probe. The key to achieving good images using ultrasound is firstly to have good contact with the skin. In cattle this usually requires clipping the hair, washing with soap and drying and then applying a contact medium. Acoustic gel, isopropyl alcohol, obstetrical lube or mineral oil can be used. Acoustic gel is recommended to prevent issues with ultrasound warranties. To fully assess the thoracic cavity using ultrasound, the whole lung field should be scanned in a systematic manner. Most commonly this is done moving from the caudal to cranial rib spaces from dorsal to ventral. The sliding motion of the normal pleural surface should be observed. ‘Comet tails’ are a type of artifact created when a small number of highly reflective material is present. These may also be described as a ‘roughening’ of the pleural surface. Pulmonary lesions can only be identified with ultrasound when they involve the lung surface. Lesions may represent abscess, areas of necrosis or masses (primary neoplasia or metastasis). Drainage or aspiration of an abscess should only occur when there is no sliding of the pleura indicating the 2 pleural surfaces are adhered. This is to prevent leakage of purulent material into the pleural space. Pulmonary consolidation can appear as lung tissue that has a ‘hepatic’ appearance. This may be termed hepatized lung. Pleural fluid may have a range of echogenicities from hypoechoegenic (black) which is a common representation of a transudate to mixed or hyperechogenic (white) if purulent effusion is present. Pleural fluid is most commonly identified in the ventral thorax, and is represented by the displacement of the visceral pleural surface from the thoracic wall. Ultrasound also allows for safe way to obtain an aspirate. Cranioventrally, the heart should be identified to differentiate pleural fluid from pericardial fluid. Cattle have a complete mediastinum, therefore both sides should be evaluated to determine presence and quantity of pleural fluid. Both sides may require draining if large quantities are detected. A pneumothorax can be identified when there is an air interface observed dorsally but the sliding action of the pleura is not observed.

Thoracic radiographic
The width of a bovine thorax may be over 100cm. In order to achieve good x-ray penetration in large bovines, common settings used are 40mAs 136kvp for the dorsal thorax and 80mAs 136 kvp for the reticular area. A significant portion of the cranioventral lung, that is frequently the desired view site also sits beneath the thoracic limbs and triceps muscle. Therefore, to achieve a diagnostic radiograph, we must position the standing animal stretched out, or in smaller bovines, lay them in lateral recumbency with the thoracic limbs extended forward.

Trans-tracheal wash
A trans-tracheal wash (TTA) takes a sample from the trachea that may be representative of the whole lung. Therefore, a TTW is indicated where there may be focal lung disease. In order to perform this, the animal should be restrained in a chute. The head is then
extended dorsally exposing the ventral neck. This can be achieved by placing 2 halters on the animal and tying their heads up. In larger, more fractious animals, nose tongs or a standing stun sedation (butorphanol 0.025-0.05mg/kg, xylazine 0.05-0.1mg/kg and ketamine 0.1-0.2mg/kg SC or IM) may be employed. A large area of the ventral neck is clipped and aseptically prepared. The trachea is palpated, at the level of the mid neck, and stabilized with one hand. A small subcutaneous block with 2% lidocaine is administered. A cut down through the skin is performed in this location. Younger animals and those with thinner skin a cut down may not be required. TTW kits are available that consist of a needle with stylet and silicone tubing. Alternatively, a 14 gauge 2inch needle and a 3.5F 30cm urinary catheter can be used. The needle is introduced through the skin until the tracheal rings can be felt with the tip of the needle. The needle is then advanced between 2 rings into the trachea. Correct placement can be determined by air exiting the needle. The catheter is then advanced into the trachea. 60ml aliquots of 0.9% or PBS saline is rapidly infused through the catheter and then immediately aspirated. The sample can be collected into serum (red top) tubes for sterile culture, and into EDTA tubes for cytology. When testing for viruses, the laboratory to which the sample is going to be sent should be contacted prior to sample collection to ensure correct handling of the sample.

**Broncho-alveolar lavage (BAL)**

A BAL can be performed with via endoscopy, or blindly using a 1 or 2 tube system. A BAL is indicated where there may be diffuse lung disease, as control of which bronchus the tube ends up in is difficult. Where endoscopy is used, the endoscope should be prepared and soaked in a glutaralydehyde solution to ensure that it is clean and no contamination of any samples taken for culture will occur. The protocol used at Texas A&M for scope preparation is 12 minutes of soaking Once the endoscope is passed through the ventral meatus into the trachea and down to a bronchus, 20ml of warmed saline is infused and immediately aspirated to obtain the sample. Local lidocaine sprayed onto the nares can facilitate the passage of the scope. The sample can be used for cytology, culture or virus isolation. Viral transport medium may be used to improve results.

Using the blind technique an 8mm diameter 90cm flexible tube is passed into the trachea and into a bronchus. A 6mm diameter tube is then passed through the larger tube. The 2 tube technique decreases contamination of the sample. The wash is performed and sample collected as before.

**Endoscopy**

Endoscopy is very useful in cases of upper respiratory disease that may present with stridor, respiratory distress, decreased nasal airflow and also in cases of pharyngeal swelling or trauma. A 9.5 mm diameter 1 meter endoscope can be passed up the ventral meatus. The pharynx, larynx, arytenoid cartilages, epiglottis, and trachea can be examined. The nasal passages are most easily evaluated when the endoscope is slowly removed. In fractious animals, a standing stun sedation may be required. In smaller patients a pediatric 5mm diameter scope can be used.

**Nasopharyngeal swabs**

Long handled sterile swabs are required. Vigorous swabbing is needed to obtain a good mucosal sample. The diagnostic yield of a swab is less than for TTW or BAL samples.

**Serology**

Paired serology can be used to assess titers for IBR, PI3, BRSV and BVD. When choosing cattle to sample, the ideal candidates will be those that are currently febrile, unvaccinated and untreated. A second sample should be obtained from the same animals in 4-6 weeks. The blood should be centrifuged and the serum removed and frozen until all the samples are sent off together to prevent laboratory variation.

**Thoracocentesis**

Thoracocentesis is indicated when pleural fluid is identified by ultrasound, or suspected on auscultation and percussion of the thorax. When performing blind thoracocentesis, care must be taken not to accidentally stick the heart. Therefore, it is recommended to do under ultrasound guidance. Once the location has been selected, the hair is clipped and the area aseptically prepared. A local block through the skin and intercostal muscles is performed using lidocaine 2%. A 18 or 20G spinal needle (cm) or a 1.5 inch needle can be used to obtain an aspirate. If a large volume of fluid needs to be drained, a teat cannula can be used. After local anesthesia, a stab incision is made with a #15 blade. The teat cannula is inserted through the parietal pleura. Penetration of the pleura is painful and restraint of the animal in a chute is usually necessary. For continued drainage, a chest tube can be placed.

**Other testing**

Other tests that can be utilized in respiratory cases include arterial blood gas analysis, fecal Baermann testing, complete blood counts, lung biopsy and necropsy examination.

**References**

Approximately 6.3 million American youths are involved in 4H programs, and in 2013 there was nearly 580,000 FFA members. A proportion of these students are involved in animal agriculture and raising livestock projects. These are usually competitive projects whilst producing meat (or milk) that enters the food chain. Many of these livestock projects are raised by families with minimal background in livestock, and consequently the care and health of these projects can suffer. Due to the competitive nature, the unfortunate consequence is the occurrence of ‘cheating’ or performing unethical practices to win at all costs. In order to maximize fairness amongst all the competitors, many shows (both major and local county) perform drug testing.

Drug use in show animals is somewhat different from drug use in commercial production animals. Firstly, show animals undergo additional testing at the plant. Secondly, drug testing performed by drug/toxicology labs tests to a zero tolerance level. This means that no trace of the drug should be present in the urine. Even for labeled drugs that are used exactly as per the label, will have longer withdrawal times. Typically, doubling or tripling the withdrawal time is recommended to be safe. Each show may also have different requirements for different classes (e.g. heifers vs. market goats) and different regulations for labeled drugs (for example Palene (ractopamine) and Zilmax (zilpaterol)). For extra-label drugs, the withdrawal time should always be determined using Food Animal Drug Residue Avoidance Databank (FARAD) system. Any veterinarian working with show animals should always verify drug use regulations and date of the show.

As with all drug use in food animal species AMDUCA regulations for extra-label drug use should be followed closely, and no prohibited drugs administered. A list of prohibited drugs are available on the AMDUCA and FARAD websites. Commonly misused prohibited drugs include Baytril (enrofloxacin) use for anything other than respiratory disease in beef cattle (and dairy calves <20 months of age) and nitrofurazone.

There is minimal peer reviewed literature investigating causes of morbidity and mortality in show cattle. However, show cattle are most commonly managed similarly to feedlot cattle, in the way that they are kept in confinement, with historical or recent shipping, fed rapidly fermentable carbohydrate diets and kept in some degree of confinement with other cattle. Therefore, much comparison in terms of etiology, diagnosis and treatment can be made between the two groups of animals and extrapolations made from feedlot cattle to show cattle. Common presenting complaints of show cattle presenting to our clinic are respiratory disease, lameness and inapetance with or without the presence of chronic ruminal bloat.

**Respiratory disease**

The two most common causes of respiratory disease in show cattle on feed in our clinic’s experience are bronchopneumonia (shipping fever) and acute interstitial pneumonia (AIP). Cattle with bronchopneumonia present with the typical signs of pyrexia, inappetance, increased respiratory rate, and nasal/ocular discharge. Auscultation of the lungs confirms bronchopneumonia with crackles/wheezes or increased bronchovesicular sounds. Confirmation of bronchopneumonia can be done by ultrasound where pleural changes, pleural fluid or lung consolidation may be seen depending on duration of disease. Radiographs typically show alveolar patterns in the cranioventral lung fields. Treatment with antimicrobials and antiinflammatories typically results in a response. In our experience, show cattle typically have an unknown or incomplete respiratory vaccination history. It is our recommendation that show animals are vaccinated with modified live vaccines prior to the start of the show season. Ag/show barns should be managed as a group of cattle to prevent spread of disease, which is often challenging with multiple owners and variable levels of knowledge and even prescribing veterinarians managing animals within the same environment or shared airspace.

Atypical interstitial pneumonia is a poorly defined disease. Fog fever and acute bovine pulmonary emphysema and edema (ABPPE) are both terms that have been used to describe the same disease. Diagnosis of AIP is by post mortem examination. Acute respiratory distress syndrome (ARDS) is the preferred term for clinical diagnosis. On gross necropsy the lungs fail to collapse and are rubbery on palpation. They have diffuse alveolar and interstitial edema, emphysema of the interlobar septa and bullae. On histopathology, type II alveolar hyperplasia and hyaline membranes are noted. Chronically, bronchopneumonia may be involved also.

Clinically, cattle present with respiratory distress (elevated rate and effort), their head may be lowered, open mouth breathing may occur when they are stressed or moved, and excessive frothing/salivation. Elevated rectal temperature may be present. Typically these animals do not cough or have significant nasal discharge. Rupture of bullae may present with subcutaneous emphysema over the thoracic region. Differential diagnosis for animals presenting with ARDS syndrome include necrotic laryngitis, severe bronchopneumonia, tracheal edema, heart failure or anaphylaxis. In our experience in a referral hospital, most cattle present with the history of unresponsiveness to treatment with antimicrobials. Cattle are also commonly late in the feeding period.

Etiology of AIP is thought to be due to ingestion of D,L tryptophan which is converted to 3-methyl indole, a respiratory toxin. Classically this occurs when moving cattle from dry pasture to lush green pasture. Ingestion of the pneumotoxins perilla mint ketone
Dermatitis is diet and increasing forage. Aspirin and monensin have been suggested for use in feedlot cattle with AIP.

Lameness presentation in show cattle can vary from the obvious lameness that can be scored using one of the many scoring systems developed for cattle (ref from in practice) to the subtle. Show cattle of the ‘club calf’ variety have different conformation than commercial cattle, and their post legged nature can make identifying lameness due to pain vs. mechanical lameness due to degenerative joint disease difficult to evaluate. Show cattle may be kept tied and standing for long periods of time, which would be abnormal for cattle in nature. Common history includes lameness or stiffness when first rising. Video evidence of lameness at home can be of assistance in diagnosis and localizing lameness. One advantage of lameness examinations in show cattle is the ability to see them walk freely, on a halter, over different surfaces, at different speeds and gaits and turn tightly as would be performed in an equine lameness examination. Some cattle will also permit flexion tests of the limbs. Nerve blocks of the limbs similar to that performed in horses can be utilized. Often this requires restraint in a turn over chute but can be performed standing. The most common block performed would be a 4 point block of the distal limb to rule out foot pain. In our experience, the most common form of foot pain in show animals is laminitis (small ruminants may present more commonly for acute laminitis than cattle). Foot rot and digital dermatitis is also diagnosed in show cattle in our clinic. Effusion of the joints (carpus and tarsus most commonly) is also frequently seen. Ultrasonography, radiographs and arthrocentesis can be performed as in the horse to differentiate infectious arthritis vs. degenerative joint disease. A common pathological cause of joint effusion is osteochondrosis (OC). Joint effusion may also be seen secondary to poor conformation without radiographic evidence of OC.

Spastic paresis
Spastic paresis is caused most typically by repetitive contractions of the gastrocnemius muscle (other muscles can be involved in Belgian Blue and Romagnola double muscled cattle but the gastrocnemius is the most common). Affected cattle stand with the hock in extension, lifting the affected leg in extension behind them. Bilateral disease may cause the animal to stand with an arched back and weight shift. When lying the animal appears normal. This disease is also known as Elso heel, as the disease was first recognized in Holstein Freisian cattle descending from the bull Elso. The disease has been reported in many purebred cattle and also in composite show cattle breeding. The incidence of disease is unknown as many cases go unreported. The cause of the clinical signs is proposed to be due to an overactive stretch reflex. No definitive etiology of the disease has been determined. Depending on the severity of disease, cattle may present at weeks to months old, and the disease is progressive.

Diagnosis is by palpation of the gastrocnemius tendon and muscle which will be rock hard. Epidural injection of procaine has eliminated clinical signs when the gastrocnemius muscle was involved alone. Differential diagnosis is upwards fixation of the patellar (most typically seen in older cattle) and other conformational abnormalities of the hind limbs.

Treatment options are limited and there are significant ethical considerations to correcting a show calf with spastic paresis. Common surgical treatments performed include neurectomy of the tibial nerve and/or transection of the gastrocnemius tendon. Transection of the gastrocnemius can result in failure to bear weight on the limb. Some calves may make it to early slaughter, although euthanasia is recommended for severely affected animals on welfare grounds.

Osteochondrosis
Osteochondrosis is part of a syndrome incorporating both osteochondrosis dessicans (OCD), a disruption of endochondral ossification leading to the formation of flaps, and subchondral bone cysts where defective cartilage folds and forms a cyst. The subchondral bone cyst may precede OCD. Both have been reported in cattle. There are limited studies on the etiology, presentation and treatment of
osteochondrosis in cattle. Studies available suggest that older (18-24 month old) bulls are more affected than females. There is limited information on the most common presentation of OC to our clinic which is in young rapidly growing bulls and steers. The cause of OC in cattle is proposed to be similar to other species, that is, high energy diets fed to rapidly growing animals. A variety of breeds have been reported to show lesions (both beef and dairy). Typical clinical presentation of OC is mild to moderate lameness with effusion of the joint. Arthrocentesis has shown normal to mild non septic inflammation. Diagnosis is by radiology (or advanced imaging using CT/MRI). Multiple views are required to fully evaluate the affected joints. The most common localization is the stifle (lateral trochlear ridge with patella involvement, medial trochlear ridge, lateral condyle, tibial plateau) and the tarsus (distal intermediate, medial and lateral trochlear ridges, medial and lateral malleoli). Other joints affected include the shoulder, distal radius and phalanges. Multiple affected locations can occur in the same animal. The atlanto-occipital joint has been reported in feedlot cattle.

Treatment options have typically included rest, decreasing plane of nutrition, and anti-inflammatories or surgical debridement. Recommended antiinflammatories in show cattle include short term intravenous flunixin meglumine at the labeled dose and meloxicam (typical dosage 1mg/kg loading dose followed by 0.5mg/kg every other day). Phenylbutazone is not recommended due to prolonged withdrawal times and there is limited evidence to show effectiveness for aspirin. Other treatments include intra articular injections of antiinflammatories (triamcinolone, methyl-predisolone), hyaluronic acid (Legend), and antibiotics (only legal intra-articular antibiotic for cattle remaining is ampicillin). Adequan (polysulfated glycosaminoglycan) and oral supplementation with chondroitin/glucosamine have all been used. Treatment regimens for Adequan include 1mg/kg once a week for 3 weeks then decreasing to every 2 weeks for 3 doses. There is minimal evidence based support for any of these treatments for OCD lesions. Differential diagnosis includes septic arthritis, trauma (cruciate rupture) and osteoarthritis. History, arthrocentesis and radiology are necessary to differentiate. Prognosis is variable depending on chroncity of lesions.

Inappetance
Show cattle can present with varying degrees of inappetance late in the feeding period which may be associated with chronic intermittent bloat. The most common cause is acute, or chronic, rumen acidosis as a result of prolonged feeding of carbohydrate diets and changes to the ruminal wall as a result. In addition, show cattle may have forage further reduced close to the show to decrease ‘gut fill’. Rumen analysis (pH and protozoal motility) is important in the work up of inappetant show cattle. In acute rumen acidosis episodes, treatment may require rumen neutralization with alkalinizing salts (magnesium oxide or magnesium hydroxide) if pH is <5.5. There are 3 sizes of rumen protozoa, and the larger species die off faster in acidic environments. Probiotics and yeast containing pre-made powders may help to improve rumen activity, however in our experience transfaunate with healthy rumen fluid from a donor animal is most effective. Placement of a permanent rumen cannula in a donated animal is a relatively simple surgical procedure that can be performed in practice. Prevention and client education is key to preventing further incidences of bloat and inappetance. Forage is essential in the diet to promote mastication, salivation and bicarbonate production. Monensin, tylosin, virginiamycin buffers and fats are used in feedlot cattle diets to minimize control acidosis. Care must be taken to follow regulations and appropriate withdrawal times used in show cattle. Appropriate treatment and prevention for sequelae to sub acute rumen acidosis (laminitis, mycotic rumenitis, liver abscesses, polioencephalomalacia, Clostridial overgrowth) may be necessary.

References
Evidence based medicine (EBM) is the conscientious, explicit and judicious use of the best available evidence from research to apply to the care for either an individual patient, or as is more frequent in food animal medicine, the care of the herd (Rosenthal, 2004). In human medicine, EBM is further advanced with more systematic reviews, large clinical trials and meta-analyses that have been used to determine standards of care. In food animal medicine there is no formalized standard of care for specific diseases and the number of systematic reviews on which to base them are lacking. Therefore, it is necessary for the practitioner to have the tools available to them to evaluate the literature themselves in order to answer a clinical question. Texas practitioners, members of AABP, AAEP and AASRP (and other groups) are fortunate that they have the Texas A&M Medical Sciences Library (MSL) resources to them. The MSL has a ‘get it for me’ service in which practitioners can either request a specific paper, or submit a question and the librarians will perform a search of the literature using Medline (Pubmed) and CAB abstracts among others to gather relevant papers. It is however, necessary for the practitioner to have the skills to critically appraise the papers. While the principles of EBM may be new, as veterinarians we try to answer questions both posed by the producer and by the clinical case we are treating, on a daily basis. By using EBM, the practitioner can tailor their refreshment of the current literature to that which is clinically applicable to them.

For this lecture, 5 papers that were determined to be of interest to veterinary practitioners were chosen. A search of the PubMed database was performed in March 2016 using the search term [bovine OR cow OR cattle OR calf OR calves] in the title or abstract, published in 2015 which returned 10132 potential manuscripts. The top 15 journals based on impact factor from Thomas Reuters (formerly ISI) that published food animal articles were selected and a search for papers containing bovine OR cow OR cattle OR calf OR calves was performed (see box 1 for Journals). Veterinary Clinics North America Food Animal was excluded as it only contains review articles. It should be noted that PubMed does not search the journal Bovine Practitioner, and this Journal did not fall into the top 15 veterinary science journals based on impact factor. Animal Science was also not included in the top veterinary science journals. Article titles were scanned for relevance and any bovine papers with potential clinical significance had their abstract read. Articles were chosen based on their relevance to clinical practice while describing different types of study for comparison (e.g. randomized control trial, case study, longitudinal study).

The following articles were chosen:

- Bellino, C., Miniscalco, B., Bertone, L., Cagnasso, A., Ocegiena, E., Gianella, P., D’Angelo, A. Analysis of cerebrospinal fluid from cattle with central nervous system disorders after storage for 24 hours with autologous serum. BMC Veterinary Research. 2015. 11:201
- Jones, M.L., Washburn, K.E., Fajt, V.R., Rice, S., Coetzee, J.F. Synovial fluid pharmacokinetics of tulathromycin, gamithromycin and florfenicol after a single subcutaneous dose in cattle. BMC Veterinary Research. 2015. 11:26

Evidence based medicine has 4 major steps. The 5th step is a self-assessment of the process in order to develop EBM skills further.

1. Formulate an appropriate question for the clinical case using the PICO or PECOT principles. P is for patient or problem, I is for intervention or E for exposures, C is for control group to define the alternative, O is for the outcome and T is the time frame.
For example: a producer wants to know whether Zactran (gamithromycin) antimicrobial administered to stocker calves for treatment of pneumonia is better than Micotil (tilmicosin). In order to formulate a clinical question the veterinarian must determine what ‘better’ is to the producer, for example increased growth rates, decreased number of treatments or increased economic returns. For this scenario P is a stocker calf, I is Zactran, C is Micotil and O is increased growth rates.

The question can however focus on any aspect of a case: clinical findings, etiology, differential diagnosis, diagnostic tests, prognosis, therapy, and prevention.

2. The second step is to search the literature to identify a list of publications. A search for this clinical question will involve ‘calf OR calves OR bovine OR cattle AND Zactran OR gamithromycin AND Micotil OR tilmicosin’. If you are using the MSL service, this part is performed by the librarian. If you are performing the search yourself, in order to generate a comprehensive search, remember to include different spellings or different meanings for the same word (e.g. calf vs. cow vs. bovine vs. heifer vs. steer etc).

3. The third step is the most challenging and involves critical appraisal of the literature to determine firstly whether it answers the question and secondly whether the results are applicable, reliable and useful. It must be remembered even if a study is published in a peer reviewed journal the study itself is not necessarily of good quality. Journal quality is determined by the impact factor and is determined, among other factors, from the number of times an article is cited from that journal. In comparison to human journals, veterinary journals have lower impact factors. For example Nature’s impact factor was 36.28 in 2012, JAVMA was 1.72. Therefore, veterinary journals should only be compared amongst themselves.

   There are several ways to evaluate the literature. One can use a scoring sheet or a series of questions should be asked in order to evaluate papers (Table 1).

   The level of evidence that the papers demonstrate should be evaluated according to the pyramid in Figure 1. The higher up the pyramid, the stronger the evidence. Recent papers will carry more weight when it comes to evaluation, as will the relevancy of the paper to the question. Not all types of studies are applicable for every type of question. For example, a question involving treatment outcomes, as above, a randomized clinical trial is most appropriate; for a frequency question (e.g. I am seeing a higher number of foot rot cases in a beef herd, is this higher than is to be expected) a cross sectional study or a prevalence study is appropriate; and for a prognosis question, (e.g. I am treating a goat for Staphylococcus aureus mastitis, what is the likelihood that this will reoccur in future lactations) a prospective cohort study will best answer the question. Table 2 describes the different types of studies and what they are useful for.

   When evaluating papers, the title will give initial indication if the paper is relevant, however ideally more than the title should be read. Most of the information can be obtained from the abstract, as to where it was published (the country), date, type of animal, and type of study in order to determine whether to keep the paper in the review. For pharmacological papers, knowing whether the study was funded by the manufacturer that may influence results, in which country it was performed, and to know whether the drug is legal and available to use in the USA.

   The results from the studies chosen to critically evaluate after this point should ideally be laid out in a spreadsheet for ease of comparison and further determine which of the papers are most useful for the clinical question. The factors evaluated will vary based on the question, but could include country, type of animal, number of animals included (higher numbers of animals results in higher statistical power), what the treatments including frequency and dose were, etc.

   It is also important to analyze the paper for sources of potential bias. Bias may affect the validity of the results, but can sometimes be difficult to discern from reading a paper. Bias includes the study selecting certain patients, loss of patients in follow up, mis-calibration of instruments, lack of blinding, subjective endpoints, non-randomization of treatment groups, publication bias for positive outcomes and conflict of interest (Buhles and Kass, 2012). Without a detailed knowledge of statistical methodology, fully evaluating the statistics used can be difficult. However, a practitioner can get a general idea of the validity of the statistics. Large sample sizes result in higher statistical power, which is the ability to reject the null hypothesis when it is false. The magnitude of the differences and effects of the results regardless of their significance, and the precision of the statistics (high precision demonstrated by low variation showed by low standard error or standard deviation) should also be evaluated.

4. Step 4 is conclusions of the findings of the paper in order to answer the question. In order to formulate the question the practitioner should weigh their clinical expertise in with the results of the studies and with an understanding of the clinical case (e.g. is a study performed in Europe on housed dairy calves applicable to answer a question regarding stocker calves in Texas, even if the study compared the 2 drugs and the outcome of choice).

5. Step 5 involves self-assessment. In must be remembered that often we must make decisions in a short time frame which precludes performing a full literature search. In these situations, recent expert opinion (e.g. by calling a colleague or
looking in a text book) is usually sufficient with evaluation of the literature performed retrospectively in order to develop knowledge for the next time.

Conclusion
In performing steps 1-4 for a personal clinical question, it becomes a clinically appraised topic, or a mini systematic review. Best Bets for Vets (www.bestbestsforvets.org) is a website comprised of many clinically appraised topics for a variety of species. This site, along with collections of systematic reviews published in Veterinary Clinics North America are excellent resources and examples of Evidence Based Veterinary Medicine. In practice however, the optimum outcome will need to be balanced using both best evidence, the clinical state, circumstances and client preference. (Holmes and Ramey, 2007).

Figure 1. Pyramid of evidence

Table 1. Examples of questions asked in order to evaluate papers

<table>
<thead>
<tr>
<th>Question</th>
<th>Thought process</th>
</tr>
</thead>
<tbody>
<tr>
<td>What is the hypothesis (the clinical question being answered)</td>
<td>Is it applicable to my clinical question</td>
</tr>
<tr>
<td>What is the study design?</td>
<td>Is it appropriate to answer the question?</td>
</tr>
<tr>
<td></td>
<td>Is it a high level of evidence (see pyramid)?</td>
</tr>
<tr>
<td>How large was the sample size</td>
<td>How reliable are the results</td>
</tr>
<tr>
<td>How were patients enrolled</td>
<td>Is it applicable to my population of animals</td>
</tr>
<tr>
<td></td>
<td>Was it randomized</td>
</tr>
<tr>
<td>What were the clinical outcomes measured?</td>
<td>Multiple outcomes weaken a study</td>
</tr>
</tbody>
</table>

Table 2. Description of types of studies

<table>
<thead>
<tr>
<th>Type of study</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systematic review</td>
<td>Review the literature on a clinical topic and minimize bias and error by their methods for identifying, selecting and evaluating the studies. The standard for a systematic review is PRISMA</td>
</tr>
<tr>
<td>Meta-analysis</td>
<td>Summarize information and analyze the results of several clinical trials that have been included following specific eligibility criteria. Often, the raw data is re-evaluated all together.</td>
</tr>
<tr>
<td>Randomized clinical trial</td>
<td>Study that assigns patients randomly to one or more interventions and records the effects on health outcomes. It can be natural or experimental. Clinical trials are mandatory for FDA regulated products. The CONSORT statement provides recommendations for reporting of clinical trials, which has been modified by the REFLECT statement for livestock and food safety. (Toews, 2011)</td>
</tr>
<tr>
<td>Cohort study</td>
<td>Animals that have been exposed to a cause are followed over time. Used for prognostic studies and for causation studies. They tend to take a long time to complete.</td>
</tr>
<tr>
<td>Cross sectional survey</td>
<td>A whole population is assessed for those with and without disease. It is used for screening tests</td>
</tr>
<tr>
<td>Case control study</td>
<td>Animals that have the disease are identified and their exposure to a suspected cause is compared with</td>
</tr>
</tbody>
</table>
unaffected animals. Used to study diseases with long incubation period or rare diseases

| Case reports and case series | Useful for new diseases or new presentations of disease or new treatments, are retrospective |

References and further reading
Toews, L. 2011. The information infrastructure that supports Evidence-based Veterinary Medicine: a comparison with human medicine. JVME. 38:123-134
Arlt, A and Heuwieser, W. 2011. Training students to appraise the quality of scientific literature. JVME. 38:140
Ultrasound examination of the abdomen is a useful diagnostic tool to support physical examination and rectal palpation findings in cattle and small ruminants. Ultrasound can easily be performed standing with the animal restrained in a chute, or using a stand or by hand in the case of small ruminants. Due to the large volume of the ruminant abdomen, low frequency linear or convex transducers (3.5-5MHz) are needed to visualize deep structures. 3.5mHz transducers can obtain 20cm depth, but at this frequency resolution of the image is compromised. Higher frequency probes are preferred to examine superficial structures in more detail. Typically, large animal practitioners will not have access to large powerful machines, however, using late gestation mode on rectal ultrasound scanners (typically 5 mHz) can be utilized to visualize specific structures. Applications for the rectal ultrasound transducer can be examination of the reticulum for traumatic reticuloperitonitis; the liver to assist with percutaneous biopsy; and the ventral abdomen to assist with abdominocentesis. Rectal probes can also be used to examine the pleural surface of the lungs, superficial masses; and the left kidney via rectal approach. When performing ultrasound, it is good practice to keep the marker cranial and dorsal to help with orientation.

Good contact is important in order to obtain clear images. Ideally the animal should be clipped and prepared with alcohol and ultrasonographic gel. However, in smooth coated animals images can be obtained using alcohol alone, or small windows clipped to facilitate ultrasound.

**Abdominal ultrasound**

**Left abdomen**

Figure 1 shows the superficial anatomy of the left abdomen. Due to the large gas and ingesta filled rumen occupying most of the left side of the abdomen, ultrasonography is limited to examination of the spleen. The spleen can be visualized in intercostal spaces 7-12. Common abnormalities include splenic abscesses.

**Right abdomen**

Figure 2 shows the anatomy as visible by ultrasound of the right abdomen. Specific structures to critically examine are:

Liver – the liver can be visualized from the last intercostal space cranially to ICS 5. The cranial portion of the liver is obscured by the lungs. The portal veins, caudal vena cava, the angle, position and size of the liver, the gall bladder (located ICS 9-11) and the parenchyma should all be assessed. Fascioliasis, hepatic abscessation, hepatic neoplastic, hepatic lipidosis and caudal vena cava thrombosis are all diseases for which ultrasound can be of diagnostic benefit.

Reticulum – the reticulum is best visualized from the ventral abdomen, just to the left of midline behind the xyphoid process. The reticulum and cranial ventral sac of the rumen should be visible and the motility of the reticulum can be assessed. Normal reticular motility is biphasic at approximately 1/minute. Decreased motility and evidence of fluid or fibrin cranial to the reticulum is suggestive of traumatic reticuloperitonitis.

Abomasum – the abomasum can be visualized on the right ventral abdomen. Of particular interest is the pylorus, which is typically situated around the entrance of the superficial epigastic vein into the body wall. Typically the abomasum will produce a gas shadow, with the folds of the mucosa visible. The pylorus appears as a thick semi-circular structure with gas shadow. Location of the abomasum can assist in diagnosis of displaced abomasums. Abomasal ulceration cannot be examined via ultrasound, except in the case of perforating ulcers where peritonitis may be evident.

**Figure 1. Left abdominal anatomy for ultrasound of the bovine.**

**Figure 2. Right abdominal anatomy for ultrasound of the bovine.**

Small intestine – normally the small intestine can be visualized from the tuber coxae to ICS 8 and ventrally to midline. The wall of the small intestine is
normally 2-3mm thick and luminal diameter 2-4cm. The descending duodenum can be imaged from the 10-12 ICS and in the paralumbar fossa. Abnormalities of the intestine for which ultrasound can be of diagnostic benefit include ileus, intussception and hemorrhagic bowel syndrome among others.

**Omasum** – the omasum is usually visualized ventral to the liver, at the 6-11 ICS as a hyperechogenic crescent shaped structure. The omasum does not have any significant motility and primary diseases are rare.

Other structures which may be imaged from the right abdomen include the cecum, particularly if it is fluid filled and the right kidney. Examination for free peritoneal fluid is also most easily performed from the right abdomen.

**Calves**

When scanning the abdomen of calves, it is important to remember that early in life, ventral anechoic fluid may be seen in the rumen, the omasum may also be fluid filled. However, the rumen, reticulum and omasum are of normal adult appearance by a few days of age. A fluid filled rumen after this age may indicate rumen drinker syndrome. The abomasum when filled with milk appears as hyperechogenic areas (clots) surrounded by hypoechogenic fluid. There may be a different appearance between whole milk and milk replacer. The folded walls of the abomasum may be visualized. Ultrasonography of the abomasum in calves can be used to time how long it takes for milk to appear in the abomasum post suckling (indicating esophageal groove closure and motility).

Ultrasound is commonly used to assess umbilical structures (vein, arteries, urachus) and the liver and bladder to assess omphalophlebitis.

**Small ruminants**

Small ruminants have very similar anatomy to cattle. Ultrasound can be performed in the same way, a small convex probe may be needed to image between the ribs. The urinary bladder is can be visualized in the inguinal region, and the left kidney can be imaged in the right paralumbar fossa caudal to the right kidney.

**Thorax**

Relatively high sensitivities and specificities (Se 79.4%, Sp 93.9%) have been reported for bovine respiratory disease (BRD) diagnosis using ultrasound (Ollivett and Buczinski, 2016). Most frequently implicated lung lobes in BRD are the right cranial, right middle and left cranial lung lobes. For examination of individual animals, complete thoracic examination should be performed, scanning from dorsocaudal to cranioventral following each intercostal space on both sides of the thorax. If multiple animals are being scanned, a shortened ultrasound at the 5th intercostal space (ICS) can be performed. The 5th ICS is at approximately the level of the elbow. In order to scan cranially, the limb may need to be extended forward. Use of a rectal probe can be more helpful than a sector scanner in order to get underneath the triceps muscle. Lesions observed are roughening of the pleural surface (‘comet tails’), small areas of consolidation (typically abscesses) on the surface of the lung that disrupt the smooth pleural surface, larger areas of lung consolidation (these typically carry a poor prognosis), pleural fluid, and pneumothorax.

**References and further reading**

In general terms, bloat can be defined as distension of the abdomen. From an owner’s perspective it can be difficult for them to differentiate between rumen distension versus abdominal distension. As an overall picture, there are 5 ‘F’s’ that can cause abdominal distension: fluid (intra-ruminal or intra-abdominal as in uroabdomen or ascites), feed, fetus, fart (gas in the gastrointestinal tract primarily) or fat (enlargement of the abdomen in overweight animals – most commonly appreciated in small ruminants). Due to the possibility of pressure on the diaphragm resulting in dyspnea and ultimately death, bloat should be considered an emergency until rumen bloat has been ruled out. This talk will focus on gastrointestinal diseases in adult bovines with distension of the abdomen caused by gas or feed.

**Examination of the abdomen**

Complete physical examination of the abdomen includes ausculting rumen contractions (for 2 minutes), feeling strength of rumen contractions, percussion (pinging), succussion (ballottment while listening for splashing sounds) of the entire abdomen bilaterally, the withers pinch or Eric Williams (bar test under the xiphoid), assessment of rumen fill, assessment of fecal output (amount, smell, color, texture) and a rectal examination. The withers pinch test is a non-specific test of cranial abdominal pain: animals with severe pneumonia may not dip on response to a pinch test. Ideally the animal should not be tightly restrained in a chute during the test, so that they can move naturally. A non-reproductive rectal examination comprises of assessment of the size of the rumen (position, shape, and firmness), palpation for any distended small intestine or cecal dilation (a cecal dilation and torsion typically results in a gas distended blind sac that may protrude caudally into the pelvic cavity), palpation of the left kidney, and assessment for any peritonitis. Chronic peritonitis results in adhesions and a ‘stove pipe’ rectum, preventing movement of the rectum and palpation of abdominal structures. Acute peritonitis may feel like free fluid, or slight ‘crunchiness’ over the surface of the organs. Assessment of the abdomen must be taken into consideration with the history. An animal that is off feed for more than 24 hours will typically have a ‘drawn’ left flank. An animal with a history of prolonged in appetence but has a ‘normal’ left sided fill is likely to have poor gastrointestinal motility or outflow. The animal should also be assessed from behind, one can divide the abdomen into quarters, to assess where there is abdominal distension. Some cattle may display colic like signs (usually less noticeable than a horse). Three rumen contractions should be heard and felt in 2 minutes, one of these should be associated with eructation. Rumen contraction frequency is modulated by gastric center in the medulla. Increased rumen motility may be seen in cases of early frothy bloat and some vagal indigestion cases. Decreased motility can be caused by severe ruminal distension, pain, fever, acidosis, hypocalcemia. Strength of the rumen contractions depends on fill, and amount of fiber present. Reticular contractions (1 per minute) may be heard at the level of the 7th intercostal space on the left. Heart rate can also be helpful in determining type of gastrointestinal disease and severity. Brady cardia (<60 bpm) may be seen with some forms of vagal indigestion. Elevated heart rates can reflect pain, severe distension or fluid imbalances.

Bloat can be considered ‘simple’ when passage of an oro or naso-gastric tube relieves the distension. Failure to eructate results in free gas bloat. Free gas bloat can be caused by intra luminal (intra esophageal) obstruction with feed material or a foreign body. Passage of a tube may allow this obstruction to pass into the rumen. Failure of passage of the tube may result in trocharization or a rumenotomy being performed to relieve the bloat until the obstruction is relieved. Obstructions can be relieved by repeated passage and lavage of the esophagus or by rumenotomy and manual retrieval of the object for distal obstructions. In smaller ruminants, endoscopy may be utilized under sedation. Extra-luminal obstruction can result in esophageal closure and preventing eructation. Common causes include bolus gun injuries, chronic respiratory disease resulting in large abscesses or lymph nodes in the thoracic cavity, cranial mediastinal tumors or abscesses. In cases of extra-luminal obstruction, bloat may need to be repeatedly relieved until the cause of the obstruction is treated, or a semi-permanent rumen fistula placed.

The second kind of ‘simple’ bloat is frothy bloat, caused by either excessive legume or grain ingestion. Passage of a tube and administration of a surfactant or neutralization of the pH typically relieves these cases. History is key in determining whether a frothy bloat is a primary problem or secondary to gastrointestinal outflow obstruction.

**Causes of gastrointestinal motility dysfunction**

Box 1 lists differentials for gastrointestinal motility dysfunction (primary GI disease) that may present with abdominal distension, or not, depending on duration of clinical signs. The list is broken down into parts of the GI tract which is significant when evaluating clinical parameters and diagnostic testing.
Box 1

**Esophageal (type I vagal indigestion)**
- Intraluminal (choke) with foreign body or feed material
- Extraluminal obstruction
  - Granulomas, or neoplasia of cardia

**Rumen**
- Frothy bloat (legume or grain)
- Acute rumen acidosis
- Subacute rumen acidosis
- Hay belly – poor quality roughage
- Hyperparakeratotic rumen wall

**Reticulo-omasal obstruction (type II vagal indigestion)**
- Foreign body
- Traumatic reticuloperitonitis
- Liver/splenic abscesses
- Toxic rumenitis

**Abomasal (pyloric outflow obstruction / type III vagal indigestion)**
- Displaced abomasum
- Lymphoma of pylorus
- Severe chronic abomasal ulceration
- Large fetus (type IV vagal indigestion)
- Abomasal impaction

**Small intestinal (type III vagal indigestion)**
- Intussception
- Mesenteric torsion
- Ileus of unknown cause
- Mesenteric fat necrosis
- Hemorrhagic bowel syndrome

**Cecum**
- Cecal dilation and torsion

**Peritonitis**

---

**Box 2. Causes of pings**

<table>
<thead>
<tr>
<th>Left sided</th>
<th>Right sided</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rumen collapse (may be a dull ping)</td>
<td>Right displaced abomasum</td>
</tr>
<tr>
<td>Left displaced abomasum</td>
<td>Abomasal volvulus</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>Cecal dilation</td>
</tr>
<tr>
<td>Physometra (caudal)</td>
<td>Small intestinal distension</td>
</tr>
<tr>
<td>Pneumorectum (dorsal and caudal)</td>
<td>Peritonitis</td>
</tr>
<tr>
<td></td>
<td>Physometra (caudal)</td>
</tr>
<tr>
<td></td>
<td>Pneumorectum (dorsal and caudal)</td>
</tr>
</tbody>
</table>

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**Working up the bloated adult ruminant**

The first step when presented with a bloated adult bovine is passage of a stomach tube to determine if esophageal obstruction is present. If significant free gas is not released, a rumen sample should be obtained by aspiration on the tube. In cases where the animal is not grossly distended, passage of a tube is also very helpful in determining whether there may be a primary or secondary GI problem. Rumen pH can be easily measured. Rumen pH obtained via a tube may range from 6-8 depending on dietary history and salivary contamination. Anorexic animals will have higher rumen pH. A pH of less than 5.5 indicates acidosis. Accurate pH can be determined by rumenocentesis in the left lower abdominal flank with a 16G 5 inch needle. In the clinic, rumen protozoal motility can also be assessed at low power (x10) under a microscope. Three sizes of protozoa are typically observed, large, medium and small. The larger protozoa die first. It is important to keep the fluid warm until it is viewed. Bacterial activity can be assessed by the redox reaction using a 1 part new methylene blue to 20 parts rumen fluid. Color should return to normal in 3-6 minutes. More rapid reduction indicates carbohydrate overload, prolonged indicates poor rumen function. On a gram stain, it should have a predominantly gram negative population. Rumen chloride can be measured on some chemistry analyzers. Normal rumen chloride is <30mEq/L. Elevated levels (>50mEq/L) indicate internal vomiting of chloride from the abomasum (type III VI).

If the animal has significant distension and passage of an orogastric tube to the rumen (thereby ruling out obstruction of the esophagus), does not relieve the bloat. A large bore tube (1 inch diameter and x cm long) passed through a wooden gag can allow the rumen to empty. A siphon may need to be created with a water hose first.
Serum biochemistry can be helpful in determining where an obstruction may be occurring. Vagal indigestion type III (obstructions distal to the omasum i.e. SI, pylorus) will result in failure of hydrochloric acid produced by the abomasum to be absorbed by the small intestine. As a result, a form of ‘internal vomiting’ occurs, resulting in evidence of hypochloremic metabolic alkalosis. Mild hypochloremic metabolic alkalosis and hypokalemia may also occur with prolonged inappetance. Severity of biochemical changes depends on duration of disease. Only slight elevations of total bilirubin are expected in inappetant ruminants. Hyperglobulinemia or A:G ratios <0.5 may indicate chronic inflammation consistent with TRP. Azotemia (elevations of creatinine and BUN) may indicate secondary pre-renal or renal disease. Assessment of the urine specific gravity (in a dehydrated animal, USG is expected to be higher than 1.024) will assist in determining pre-renal from renal azotemia. In male animals until urination has been observed, post renal azotemia secondary to urinary obstruction should be kept on the differential list. A simple packed cell volume and total protein can be helpful to assess hydration levels and possible increases in proteins consistent with chronic inflammatory disease (a plasma sample will contain albumin, globulin and fibrinogen fractions, therefore chemistry analysis should be performed on a serum sample in order to get an accurate calculation of globulin as fibrinogen removed).

Abdominocentesis can be performed blind in cattle safely in the right flank fold. It can however be unrewarding or non-diagnostic due to ruminants’ ability to wall off pockets of infection within the abdomen. Abdominocentesis can also be performed where ultrasound indicates a pocket of fluid. Normal peritoneal fluid should be clear to yellow (you should be able to read print through the tube), total protein <3g/dL. Some eosinophils are frequently noted on cytology, cell count should be less than 10,000 and an equal ratio of neutrophils to eosinophils.

Hematology. Few disruptions to the complete blood count may be expected with gastrointestinal disease. Fibrinogen may be elevated in cases of TRP. Hemoconcentration may be observed where the animal is dehydrated, and anemia in cases of abomasal ulceration.

Abdominal ultrasound may be helpful in determining whether there is any distended small intestine, free abdominal fluid, abomasal positioning, and assessing motility of the reticulum. Further description of abdominal ultrasound is provided in another lecture.

Radiographs. Confirmation of TRP prior to surgery or necropsy is by radiographs of the cranioventral abdomen. The technique is beyond the scope of most private practices. Therefore, left sided exploratory surgery or ultrasound can be key in assisting in the diagnosis of TRP.

Exploratory surgery. In cases where type III vagal indigestion is suspected based on bloodwork, or there is evidence of small intestinal, abomasal or cecal disease from physical examination, a right sided standing flank exploratory can be easily performed. The abomasum, small intestine (accessed from behind the omental sling), liver, kidneys, reproductive tract, cecum, spiral colon and omasum can be evaluated. In the author’s opinion, in cases of cattle with signs of colic, small intestinal distension, cecal dilation, right sided pings or elevated heart rate >100bpm, surgery is recommended sooner rather than later. The reticulum and adhesions to the diaphragm/body wall can also be assessed from the left. If TRP is suspected, a left sided exploratory is recommended, to allow for a rumenotomy to be subsequently performed.

Treatment of gastrointestinal disorders
Many disorders of the reticulum, abomasum, small intestine and cecum are surgical. Surgical correction of these disorders will not be discussed here.

Supportive therapy includes fluid therapy, prokinetic agents, transfaunation, slow feeding back (especially post resection and anastomosis), anti-inflammatories and analgesics. Intravenous fluid therapy in ruminants is most easily achieved using a ruminant electrolyte solution in a 20 liter carboy administered through a jugular or ear vein catheter. Box 3 lists ingredients to make an isotonic crystalloid electrolyte solution that is appropriate for bovines without acidosis.

There is no conclusive evidence on the effectiveness of prokinetic agents in cattle. The best evidence exists for the macrolide erythromycin acting on motilin receptors to increase abomasal emptying rate in adult cattle and calves.

Box 3. Contents of bovine electrolyte solution to be administered in a carboy with 20 liters of distilled water

<table>
<thead>
<tr>
<th>NaCl 140 grams</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCl 30 grams</td>
</tr>
<tr>
<td>CaCl 10 grams</td>
</tr>
</tbody>
</table>

Further reading

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Proper development of replacement females can be critical to the productivity and profitability of a beef operation. Replacement females are the future of any given beef cow herd and they are the mechanism by which genetic progress is made in a cow herd. It has been estimated that the development costs for preparing a heifer to calve at 24 months age average about 31% of her total lifetime expenses. Veterinary practitioners can provide an invaluable service to their clients by providing assistance to develop and implement a comprehensive heifer development program.

One of the primary goals of a heifer development program is to get a high proportion of the heifers to conceive early in the breeding season. Heifers that become pregnant early in their first season tend to breed early as cows and have greater longevity in the herd. Becoming pregnant early in the breeding season has several advantages that will be discussed later. In order to accomplish that goal, heifers need to reach puberty prior to or at the beginning of the breeding season. Heifers are more fertile on their third estrus cycle compared to their first estrus cycle so reaching puberty prior to the breeding season increases the chances of the heifer becoming pregnant early. Some producers will also breed heifers to calve prior to the mature cows. By placing these points on a timeline, it is easy to see the importance of heifer age and growth.

A heifer development program should include several key areas. These areas are selection, health, growth and nutrition, breeding, calving, and rebreeding. Each of these areas will be discussed in more detail below.

Selection
Most commercial producers are going to select replacements from heifers born on the operation. Having a planned program to produce potential replacements can offer significant advantages. The application of artificial insemination in the commercial cow herd gives the producer the opportunity to select bulls that rank high for maternal traits with the specific intent of producing heifers that will serve as future replacements for the herd. Using sexed or gender sorted semen can increase the proportion of heifers born from matings specifically intended to produce replacements.

The selection process for replacement heifers can begin at birth. Performance records of both the sire and dam should be evaluated if possible. Heifers sired by bulls with larger scrotal circumference measurements will reach puberty at an earlier age.

Heifers born to dams with poor feet and leg confirmation, excessive size, poor disposition, or poor udder conformation should not be considered as future potential replacements. Dams of potential replacement heifers should thrive in their environment while requiring minimum additional inputs.

Several authors have recommended that potential replacement heifers should not receive growth promoting implants. Some authors have reported that implants interfere with development of the reproductive tract or result in decreased fertility while others have reported that implants have minimal to no impact on overall reproductive performance.

Selecting heifers that are born early in the calving season offers several important advantages. Early born heifers were conceived early and, therefore, may be from more fertile dams and will have heavier weaning weights, reach puberty earlier and have higher pregnancy rates than heifers born later in the calving season.

There is growing evidence that nutritional and environmental factors during gestation can program the growth and reproductive performance of progeny heifers. Protein restriction during late gestation can affect age at puberty and reproductive efficiency of heifer progeny. Replacements should be selected from dams that were properly managed during gestation.

At weaning, heifers should be evaluated for growth potential and conformational soundness. Simply selecting the biggest heifers as replacements may not be optimum because this is likely to lead to a gradual increase in mature cow size. For seedstock producers, Expected Progeny Differences (EPD’s) are a useful tool for comparing animals based on their predicted production capabilities.

At approximately one year of age individual growth performance and confirmation should be reevaluated. Pelvic area measurement is a tool that is used in an effort to reduce the incidence of dystocia but there is some controversy concerning its use. The average pelvic area of the herd can be increased over time by using pelvic measurements but, as pelvic area increases, so does mature cow size and, potentially, calf birth weight. Also, differences in pelvic area observed in yearlings are not always present when those heifers reach two years of age. The current recommendation for using pelvic area measurements is to eliminate heifers that do not meet a minimum pelvic area measurement.

Reproductive tract scoring (RTS) is a tool used to subjectively classify pubertal status through evaluation of the reproductive tract via rectal palpation. The technique is typically applied a few months before the start of the breeding season. Briefly, heifers are assigned a score of 1-5 based on the size and tone of the uterus and the presence of palpable structures on the ovaries. Having a high percentage of heifers with RTS of 4 or 5 indicates that majority of the heifers have reached puberty. If a high percentage of heifers have scores lower than 4, nutritional adjustments must be made or heifers with low RTS can be culled.
Assessment of antral follicle count prior to breeding is a relatively recent addition to the replacement heifer selection process.14

Health
A solid preventive health program is a critical component of a heifer development program. A preventive health program should be tailored to each operation and should be focused on providing maximal immunity to the most important reproductive pathogens on that ranch. For most operations, these pathogens include bovine viral diarrhea virus (BVDV), bovine herpes virus-1, leptospirosis, vibriosis, and possibly trichomoniasis. Brucellosis vaccination may be considered if heifers as sold as replacements as some state still require brucellosis vaccination as part of entry requirements. Regardless of the vaccine program used, the primary goal should be to maximize immunity to important reproductive pathogens prior to the start of the breeding season. Parasite control programs will vary depending on location, environment and rainfall. Diagnostic testing for specific diseases may be warranted depending on the biosecurity program for the operation. The veterinarian is uniquely suited to assist producers with developing herd health programs that are specifically tailored to a given operation.

Growth and nutrition
Prior to weaning, nutrition for the growing replacement heifer is provided by her mother. If creep feeding is used, care must be taken to not allow the calves to become too fat. Heifers that become overconditioned as calves may have reduced productivity as adults.6

The nutritional management of beef replacement heifers has been reviewed.16-18

Important targets have been established to guide the nutritional management of replacement heifers. The overriding goal is for heifers to reach desired weights and attain puberty prior to the breeding season with minimum supplementation. The topic of replacement heifer target weight has been reviewed.18 Heifers that require significant supplementation may not be suitable as replacements.

Most beef heifers will enter their first breeding season at around 15 months of age. Heifers should reach 60-65% of their expected mature body weight prior to breeding.18 Most heifers will have reached puberty by the time they have reached this weight. Research has shown that this weight gain does not have to be consistent over the entire weaning to breeding period.18 Research has also shown that the source of nutrients does not matter as long as desired levels of gain are achieved.19 This information allows considerable flexibility in heifer feeding programs. Recent research has investigated the impacts of feeding heifers to a lighter target weight of 50-55% of mature weight prior to the first breeding season.20,21 This body of work has demonstrated that, in some cases, developing heifers to a lighter target weight can reduce development costs without effecting reproductive efficiency. Although there was no difference in overall pregnancy rate, heifers developed to 50% of mature weight reached puberty later and therefore conceived later in the breeding season.22

Once heifers are confirmed pregnant, average daily gain should be adjusted to insure that heifers reach 85% of expected mature weight prior to calving and calve in a body condition score (BCS) of 6. Reaching 85% of mature weight reduces the risk of dystocia and calving in a BCS of 6 gives the heifer needed energy reserves to return to estrus cyclicity in a timely manner. The ration should be adjusted to provide for rapid fetal growth during the last trimester of gestation.

Breeding
Several important milestones should have been met prior to the start of the breeding season. Heifers should have reached desired target weights and reached puberty prior to breeding. Some producers elect to breed heifers to calve 3-4 weeks ahead of the mature cows to allow closer observation of the heifers and to give the heifers more time to return to cycling for their second breeding season. This gives calves born to replacement heifers an advantage as well since they will be older at weaning.

Breeding replacement heifers is an excellent time to take advantage of artificial insemination. Estrus synchronization facilitates the use of artificial insemination and may give heifers an extra opportunity to become pregnant during the breeding season. Numerous estrus synchronization protocols are effective in heifers. If a significant proportion of the heifers are not cycling, use of a progestin based synchronization protocol may help induce puberty in some animals.

One of the most critical components of a successful heifer breeding program is bull selection. Artificial insemination is a great tool for breeding replacement heifers and improving the genetics of the herd. Proper bull selection is also one of the most effective dystocia prevention tools available to producers. Calving ease bulls with balanced EPDs exist in most breeds and careful evaluation of EPD’s can identify these bulls. Use proven sires with high EPD accuracy for breeding heifers.

The first breeding season is a great time to apply selection pressure for fertility. Restricting heifers to a confined, shortened breeding season ensures that only the most fertile heifers have the chance to remain in the herd as cows. The typical recommended breeding season length for heifers is 2 estrus cycles or 42 days. If estrus synchronization is used, this 42 day period may include 3 estrus cycles. Early diagnosis of pregnancy permits producers to select heifers that conceived early in the breeding season. As mentioned previously, heifers that become pregnant early in their first breeding season tend to become pregnant early in the breeding season as cows and have increased longevity in the herd.3 Retaining only those heifers that conceived early in the breeding season should increase the reproductive efficiency of the herd over time.
Calving
Heifers should calve at approximately 85% of their expected mature weight in a body condition score of 6. At 85% of mature weight, heifers have attained enough size to minimize the occurrence of dystocia provided they are bred to appropriate bulls. Calving in a body condition score of 6 helps insure adequate energy reserves to resume estrus activity in a timely manner following calving.

Heifers are at increased risk of dystocia compared to mature cows and therefore should be observed closely and often during the calving season. Keeping heifers confined for ease of observation must be balanced with the risk of disease in the calves if the animals are too crowded. The ideal calving area in most climates is a clean grassy pasture with shelter from harsh weather and easy access to facilities so that assistance can be provided when needed. Keeping other cattle out of the calving area prior to the calving season may help reduce the risk of infectious disease in the calves.

Calving assistance should be provided in a timely manner to minimize the effects of dystocia on both the calving heifer and the calf. Providing prompt assistance, when needed, improves return to cyclicity in heifers. If a heifer has been in active labor for more than 90 minutes without making progress, assistance should be provided.

Rebreeding
Achieving a female’s second pregnancy is often the most difficult of her life. The nutritional demands of lactation stacked on the demands for maintenance and growth may prevent some heifers from returning to estrus activity soon enough to get pregnant during the next breeding season. Developing heifers that reach puberty early, conceive early in the breeding season, reach target weight and BCS goals prior to calving and calve without dystocia have the best chance of remaining productive within the herd.

Resources
Readers who desire more in-depth information regarding heifer management are referred to the November 2013 edition of the Veterinary Clinics of North America: Food Animal Practice. The entire issue is devoted to beef heifer management and offers several excellent reviews on the subject. Another very recent review of heifer development has been published by Larson and colleagues.

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Diagnosing and Managing Vagal Indigestion Syndrome
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Vagal indigestion syndrome is an umbrella term used to describe a variety of disease conditions that present with a relatively consistent set of clinical signs. Interference with the vagus nerve at various locations can lead to development of the various forms of vagal indigestion. However, many, if not most, clinical cases have no direct involvement of the vagus nerve. Many of the conditions resulting in signs consistent with vagal indigestion are difficult to differentiate based on clinical findings alone. Understanding the pathogenesis and conducting a careful clinical investigation will usually lead to an accurate diagnosis. This paper presents the author’s approach to diagnosis and management of vagal indigestion syndrome.

Types of vagal indigestion

**Free gas bloat (type I)**
Type I vagal indigestion is characterized by chronic, sometimes intermittent, accumulation of free gas within the dorsal sac of the rumen. The bloat is usually mild to moderate in severity but can become life-threatening.

**Omasal transport failure (type II)**
Type II vagal indigestion is characterized by the accumulation of ingesta within the reticulorumen while the omasum and abomasum remain relatively empty.

**Pyloric outflow obstruction (type III)**
Type III vagal indigestion is characterized by accumulation of fluid ingesta in the abomasum with backflow of ingesta into the omasum and reticulorumen leading to distention of these organs as well.

**Indigestion of late pregnancy (type IV)**
Type IV vagal indigestion is very similar to type III except that it is thought to be caused by a large gravid uterus interfering with normal abomasal emptying.

Clinical presentation

**Free gas bloat (type I)**
- Mild to moderate distention of the upper left quadrant on the abdomen with free gas (bloat)
- Normal stratification of rumen contents may be palpable if bloat is not too severe
- Bloat is usually chronic and may be intermittent
- Decreased appetite and lethargy may be present
- Evidence of other systemic disease (respiratory disease, TRP) may be present
- Respiratory difficulty may be present depending on severity of bloat

**Omasal transport failure (type II)**
- Decreased appetite, weight loss and decreased milk yield may be noted
- Moderate to marked distention of the left side of the abdomen in early cases. As condition progresses, abdomen will take on a “papple” shape with distention of the upper and lower left quadrants and lower right quadrant
- Rumen motility is often increased due to activation of the low threshold tension receptors in the reticular wall
- Rumen contents lose the normal stratification and develop a frothy consistency
- Mild dehydration may be present
- Enlarged “L” shaped rumen is palpable upon rectal examination
- Decreased fecal volume with longer than normal fiber length. Feces may have a greasy or pasty appearance
- Significant bloodwork abnormalities are not usually evident

**Pyloric outflow obstruction (type III)**
- Decreased appetite, lethargy, weight loss and milk yield may be noted
- Abdomen usually takes on “papple” appearance
- Rumen motility is usually decreased due to more severe distention leading to activation of high threshold tension receptors
- Rumen contents lose normal stratification and become watery
- Dehydration is usually evident and may be severe
- Varying degrees of depression may be evident
- Spontaneous reflux occasionally occurs in severe cases
- Enlarged “L” shaped rumen is palpable upon rectal examination
- Fecal volume is decreased more than with Type II and may have longer than normal fibers
- Hypokalemic, hypochloremic metabolic acidosis is usually evident on blood work
Indigestion of late pregnancy (type IV)
- Clinical appearance is very similar to Pyloric Outflow Obstruction
- Large gravid uterus is evident upon rectal examination and may obscure evaluation of the rumen

Differential diagnoses

Free gas bloat (type I)
- Injury to vagal nerve leading to functional deficits of the reticulum and/or cardia
  - TRP, liver abscess, peritonitis
- Functional interference with normal reticuloruminal motility patterns
  - Adhesions from TRP, liver abscess, neoplasia
- Esophageal obstruction
  - Intraluminal foreign body – hedge apple, potato etc
  - Extraluminal mass – enlarged mediastinal lymph node, abscess
- Dietary changes

Omasal transport failure (type II)
- Injury to vagal nerve leading to functional deficits of the reticular groove, reticuloomasal orifice, or omasum
  - TRP, liver abscess, peritonitis
- Functional interference with function of the reticuloomasal orifice
  - Adhesions, abscess, peritonitis
- Neoplasia involving reticular groove or reticuloomasal orifice
  - Papilloma
- Foreign body obstruction of the reticuloomasal orifice
  - Placenta, plastic bag, baling twine

Pyloric outflow obstruction (type III)
- Injury to vagal nerve leading to motility deficits of the abomasum
  - TRP, peritonitis
- Abomasal volvulus
  - Stretching of the abomasal wall may injure nerve and/or muscle leading to altered motility patterns
- Lymphosarcoma involving the pylorus
- Abomasal impaction
  - Poor quality roughage diets
- Foreign body obstruction of the pylorus
  - Trichobezoar (young calves)

Indigestion of late pregnancy (type IV)
- Clinically indistinguishable from pyloric outflow obstruction
- Only occurs in with late term pregnancy

Diagnosis/differentiation of types

Free gas bloat (type I)
- Pass orogastric tube to rule out intraluminal obstruction
  - Extraluminal mass may still allow passage of tube
- Rule out dietary causes as much as possible
  - Evaluate rumen fluid for microbial activity
- Ultrasound examination of the cranioventral abdomen may provide evidence of TRP or adhesions
- Ultrasound examination of the thorax may reveal evidence of pleural or pulmonary disease consistent with pneumonia
  - Mediastinal disease cannot be assessed with ultrasound
- Thoracic radiography in smaller animals may reveal evidence of intrathoracic masses

Omasal transport failure (type II)
- “Papple” shaped abdomen is the classic clinical sign
- Rumen motility is usually increased but may be more quiet than normal due to frothy nature of rumen contents
- Frothy rumen contents
- Enlarged “L” shaped rumen noted on rectal examination
- Ultrasound examination of the cranioventral abdomen may provide evidence of TRP or adhesions
- Relatively empty abomasum when visualized with ultrasound
- Minimal abnormalities noted on bloodwork
Concurrent disease may cause bloodwork changes
- Normal ruminal fluid chloride level (<30 mEq/L)

Pyloric outflow obstruction (type III)
- “Papple” shaped abdomen is the classic clinical sign
- Dehydration is generally more severe than with Type II
- Rumen motility is generally decreased
- Fluid rumen contents
- Ultrasound examination of the cranioventral abdomen may provide evidence of TRP or adhesions
- Abomasum may be distended with fluid contents when visualized with ultrasound
- Ultrasound examination of the pyloric region may provide evidence of lymphosarcoma
- Hypokalemic, hypochloremic metabolic acidosis evident on bloodwork
- Elevated ruminal fluid chloride level (>30 mEq/L)
  - May approximate serum chloride level in severe cases
- May develop 2-3 days following abomasal volvulus correction
- Right flank exploratory laparotomy

Indigestion of late pregnancy (type IV)
- Rule out other causes of Pyloric Outflow Obstruction
- Large gravid uterus may obscure palpation of enlarged rumen

Treatment
Free gas bloat (type I)
- Treat underlying disease condition
  - TRP, pneumonia
- Screw-in rumen trocar or surgical rumenostomy may be needed
  - Normal function may return with time if bloat can be controlled

Omasal transport failure (type II)
- Treat underlying disease
- Decompress rumen via large bore orogastric tube
- Transfaunation
- Left flank exploratory laparotomy with rumenotomy
  - Remove penetrating foreign bodies by pulling back into reticulum
  - Drain abscess back into reticulum if firmly adhered to reticular wall
  - Remove foreign body/material from reticulorumen

Pyloric outflow obstruction (type III)
- Treat underlying disease
- Decompress rumen via large bore orogastric tube
- Correct dehydration, metabolic and acid-base abnormalities
  - IV fluid therapy
- Gastric motility modulation
  - Erythromycin
- Right flank exploratory laparotomy
  - Lymphosarcoma – euthanasia
- Left flank exploratory laparotomy with rumenotomy
  - Treat TRP/abscess as described above
  - Feed orogastric tube into abomasum via hand in the rumen
    - Administer DSS to help relieve abomasal impaction
    - Massage abomasum through rumen wall
- Abomasotomy to remove foreign bodies

Indigestion of late pregnancy (type IV)
- Induce parturition or caesarian section
- Repeated rumen decompression and supportive care until calving
Several infectious pathogens have the ability to cause infertility and/or abortion in cattle. The pathogens that commonly cause reproductive loss in cattle include bovine viral diarrhea virus (BVDV), bovine herpes virus-1 (BHV-1, also known as Infectious Bovine Rhinotracheitis or IBR), Leptospirosis, *Campylobacter fetus* subspp. *venerealis*, *Tritrichomonas foetus*, and *Neospora caninum*. Controlling and minimizing the effects of these pathogens requires sound herd biosecurity programs, one component of which is vaccination. The purpose of this paper is to briefly review the efficacy and safety of vaccination against reproductive pathogens in cattle.

**BVDV and BHV-1**

Do vaccines provide a benefit

Bovine viral diarrhea virus can effect reproduction in a variety of ways including alterations in ovarian function, early embryonic death, abortion, congenital defects, and the development of immunotolerant persistently infected (PI) calves. The most common reproductive effects of BHV-1 are alterations in ovarian function and mid to late-term abortion.

The efficacy of vaccination to reduce the reproductive impact of these pathogens has been recently reviewed. This review found that vaccination against BVDV and BHV-1 consistently provided protection of the fetus from virulent challenge even though the protection is not 100% in all cases. In a recent meta-analysis of the published literature regarding vaccination against BVDV, Newcomer et al found a decrease in abortion rate of nearly 45% and a decrease in fetal infection of nearly 85% in vaccinated cattle versus non-vaccinated cohorts. The meta-analysis included 46 studies reported in 41 separate papers. In a large study involving four different experiments utilizing Brazilian cow-calf operations, Aono et al reported that vaccination against BVDV, BHV-1 and leptospirosis resulted in reduced pregnancy loss and/or increased pregnancy rate in vaccinates versus non vaccinated controls. Results varied between the different experiments included in the report. In a similar study, Pereira et al reported reduced pregnancy loss or increased pregnancy rate (depending on the experiment) in vaccinated versus non vaccinated Brazilian dairy cattle.

**Efficacy of modified live versus killed vaccines**

Among the many choices that producers and veterinarians must make when establishing a reproductive vaccine program is the choice of whether to use modified live virus (MLV) or inactivated/killed viral (KV) vaccines. There is general agreement the MLV vaccines provide a more robust and longer lasting immune response. Modified live vaccines also typically generate a stronger cell-mediated immune response. In contrast, KV vaccines are generally considered to have a wider margin of safety, especially in pregnant cattle.

Both types of vaccines can be efficacious when used correctly. Inactivated BHV-1 vaccine has been shown to provide protection against BHV-1 challenge. In this study, 3 of 21 vaccinates and 14 of 14 non-vaccinated controls aborted when challenged with virulent BHV-1 at approximately 180 days of gestation. Inactivated BVDV vaccine has been shown to provide fetal protection when pregnant heifers were commingled with BVDV persistently infected cows during gestation. Fetuses were harvested via caesarian at approximately 150 days of gestation. BVDV was isolated from 4 of 15 vaccinated heifers and 14 of 14 control heifers.

A single dose of MLV BHV-1 vaccine given at either 13 or 8 months prior to challenge has been shown to be effective. Abortion occurred in 1 of 13 heifers vaccinated 13 months prior to challenge, 3 of 19 heifers vaccinated 8 months prior to challenge, and 18 of 19 non-vaccinated controls. A single dose of MLV vaccine has been shown to protect fetuses from the development of BVDV persistent infection. In this study, 0 of 39 calves born to vaccinated heifers were PI while 18 of 19 calves born to control heifers were PI.

Administration of two doses of MLV vaccine containing both BVDV and BHV-1 prior to breeding has been shown to be protective against challenge via exposure to PI cattle and cattle acutely infected with BVDV-1. No vaccinated heifers aborted following BHV-1 challenge compared 4 of 10 controls. None of 19 calves from vaccinated heifers were BVDV PI compared to 10 of 10 fetuses or calves from control heifers.

Rødning et al compared two MLV vaccines and one KV vaccine for ability to prevent BVDV persistent infection. All heifers received four doses of the respective vaccine prior to breeding and were subsequently challenged by exposure to PI animals. Two of 18 calves produced by KV vaccinates were PI compared to 0 of 19 and 0 of 18 calves produced by the two MLV vaccinate groups. Ten of 10 calves produced by non-vaccinated control heifers were PI.

The previously mentioned meta-analysis published by Newcomer et al demonstrated that vaccination against BVDV reduced abortions by nearly 45% and fetal infection by nearly 85% compared to non-vaccinated controls. While both types of products provided better protection than no vaccination, the use of MLV vaccines was more effective at reducing the risk of abortion and fetal infection compared to the use of KV vaccines.
Bovine viral diarrhea virus exists in two distinct genotypes described as BVDV1 and BVDV2. While the genotypes are clinically indistinguishable, antigenic differences do exist. Effective cross protection between genotypes is variable. The use of vaccines containing both genotypes has been shown to be superior and is recommended.\(^2,3\) Fortunately, most currently available commercial vaccines contain both genotypes.

**Safety of modified live vaccines**

Vaccines against reproductive diseases are most commonly administered to beef cattle prior to breeding or during gestation when females are checked for pregnancy. There are advantages and disadvantages to each approach. Prebreeding vaccines provide peak immunity during conception and early pregnancy when most reproductive loss occurs but often require extra handling of the cattle in order to administer the vaccines. Administering vaccines at pregnancy diagnosis is more convenient since cattle are already being handled but the timing may not provide optimal immunity at the time of greatest risk. Regardless of the timing of administration of MLV vaccines, there are some safety concerns that should be considered.

Necrotic oophoritis has been reported following intravenous administration of MLV BHV-1 vaccines.\(^12\) Chiang et al\(^13\) reported decreased first service conception rates when heifers were given MLV BHV-1 vaccines at estrus compared with non-vaccinated controls. It is important to note the differences in conception rate reported in this study were numerically different but not statistically different. The difference in overall calving rate was statistically significant.

Infection of ovarian tissue by BVDV following administration of MLV vaccine has been demonstrated.\(^14\) Infection with BVDV following estrus has been shown to effect ovarian follicular dynamics.\(^15\) Perry et al\(^16\) investigated the effects of vaccination with either MLV or KV vaccines containing both BVDV and BHV-1 when naïve heifers were vaccinated at the start of synchronization for fixed time artificial insemination (FTAI). Heifers that received the MLV vaccine had reduced pregnancy rate and an increased number of abnormal estrus cycles compared to heifers receiving KV vaccine or non-vaccinated controls. It is important to note that the heifers in this study were naïve at the time of vaccination and that administering the vaccine at the start of synchronization for FTAI is an extra-label use of the vaccine. Label instructions for most MLV vaccines indicate that prebreeding vaccination should not occur within 28 days of breeding.

In more recent work, Walz et al\(^17\) found no difference in serum progesterone concentrations or pregnancy rates when primiparous, previously vaccinated, dairy cows were vaccinated 17 days prior to the start of an estrus synchronization-FTAI protocol. The reproductive effects of MLV vaccine administration prior to estrus synchronization in previously vaccinated heifers have been investigated.\(^18\) Revaccination was performed 10 or 31 days prior to synchronized natural breeding. No differences in duration of interestrus intervals, proportion exhibiting estrus within 5 days of synchronization, serum progesterone concentrations, pregnancy rates, and pregnancies within the first 5 days were observed. No BVDV or BHV-1 was isolated from luteal, ovarian, or fetal tissues harvested between 44 and 62 days of gestation.

Safety of MLV vaccines administered during pregnancy is a current topic of debate. Currently several products are labeled for use in pregnant cows. Label instructions indicate the females must receive at least one dose of the same MLV vaccine prior to breeding and have been vaccinated within the preceding year before receiving MLV vaccine during pregnancy. When label instructions are followed, vaccination with MLV vaccines during pregnancy has been shown to be safe. In one study\(^19\), heifers vaccinated with KV vaccines twice as calves received MLV vaccine, KV vaccine or no vaccine at pregnancy check. Heifers were 60-120 days pregnant at the time of vaccination. One abortion occurred within each treatment group. Ellsworth et al\(^20\) reported the results of a large safety trial in which previously vaccinated heifers were vaccinated during pregnancy with 10 times the normal vaccine dose of MLV BVDV and BHV-1. Six of 11 BHV-1 seronegative controls (had not received prebreeding vaccine) aborted. Nine of 12 calves born to BVDV seronegative controls had precolostral antibody titers indicating intraluminal exposure to BVDV. ALL 59 previously vaccinated heifers delivered live healthy calves, 58 of which were negative for precolostral antibody titers (one calf nursed prior to sampling). The same report details the findings of three field trials in which previously vaccinated pregnant cows or heifers received MLV vaccine containing both BHV-1 and BVDV during the first, second, and third trimesters. No difference in abortion rate between vaccinates and controls were observed in any of the three trials.

Both BVDV and BHV-1 are important reproductive pathogens in cattle. When used correctly, vaccines can be an effective part of an effective biosecurity program. Both MLV and KV vaccines are effective although MLV vaccines are preferred. Administration of MLV vaccines prebreeding or during pregnancy appears to be safe as long as the animals have been previously vaccinated according to label directions.

**Leptospirosis**

Leptospirosis is an important bacterial disease of cattle. Reproductive consequences of Leptospira infections include abortions, stillbirths, early embryonic death and infertility.\(^21\) Non-reproductive manifestations of Leptospira infection include septicemia and nephritis. Leptospirosis is also an important zoonotic pathogen. Leptospira taxonomy is extremely complex and confusing. Leptospira are divided based on genetic sequencing with at least 16 genomespecies identified.\(^22\) Approximately 200 serovars of Leptospira have been identified. Leptospira serovars are typically associated with one or more maintenance hosts and prevalence
varies with geography. Leptospira serovars host adapted to cattle include *Leptospira interrogans* serovar hardjo (type hardjoprajitno) and *Leptospira borgpetersenii* serovar hardjo (type hardjo-bovis).\(^1\) Serovar hardjo type hardjo-bovis is found in cattle throughout the world and serovar hardjo type hardjoprajitno is typically found in cattle in the United Kingdom. Other serovars commonly associated with disease in cattle include pomona and grippotyphosa. Serovar hardjo is host adapted to cattle causing chronic infections of the renal and reproductive systems. Results of reproductive infection generally include infertility, early embryonic death and sporadic abortions. Serovar hardo type hardjo-bovis is the most common cause of Leptospirosis in cattle in the United States.\(^1\)

Infections with other serovars such as pomona tend to cause late term abortions occasionally occurring in abortion storms.\(^23\)

Traditional pentavalent leptospira vaccines used in cattle in the United States include hardjo (type hardjoprajitno), pomona, canicola, grippotyphosa, and icterohaemorrhagiae. These vaccines can provide good protection against serovars contained within the vaccine other than hardjo but traditionally have not provided adequate protection against hardjo.\(^1\)\(^,\)\(^24,\)\(^25\) However, one recent study\(^26\) demonstrated that administration of pentavalent vaccine containing hardjo type hardjoprajitno was effective at protecting 6 month old heifers from experimental challenge with hardjo type hardjo-bovis. Leptospira vaccines are bacterins and therefore require appropriate boosters when initially administered. Once the initial series of vaccines has been properly administered, once yearly boosters are usually adequate although more frequent boosters may be required in some areas.

Monovalent vaccines specifically targeting serovar hardjo type hardjo-bovis are available. These vaccines produce a strong cell mediated immune response\(^27\) and have been shown to be effective at preventing colonization of the renal and reproductive systems in experimentally challenged animals.\(^24\) Vaccination of calves as young as 4 weeks old has been shown to be protective against experimental challenge\(^28\) when challenge occurs up to 12 months after vaccination.\(^29\) Multiple vaccines containing serovar hardjo type hard-bovis are currently available in monovalent forms or in combination with pentavalent leptospira vaccines.

Although several studies have shown that vaccines containing serovar hardjo type hardjo-bovis are effective against experimental challenge, efficacy in field trials remains questionable. In a large study of beef cow-calf herds\(^30\), administration of a monovalent hardjo-bovis vaccine along with oxytetracycline did not significantly improve reproductive performance. Similarly, a large study\(^31\) conducted in a commercial dairy in California failed to demonstrate an improvement in reproductive efficiency or a reduction in urine shedding when cows received two doses of a monovalent hardjo-bovis vaccine along with oxytetracycline.

Leptospirosis remains an important reproductive disease of cattle with *L. borgpetersonii* serovar hardjo type hardjo-bovis being the most important Leptospira pathogen of cattle in the United States. Vaccines containing serovar hardjo type hardjo-bovis have been shown to be highly effective at protecting cattle against experimental challenge but efficacy in field trial settings remains questionable. At least one pentavalent vaccine containing serovar hardjo type hardjoprajitno has been shown to protect cattle against experimental challenge with type hardjo-bovis.

### *Campylobacter fetus subsp. venerealis* (Vibrio)

Vibriosis is a venereally transmitted disease of cattle causing transient infertility, early embryonic death or abortion. Infection generally does not produce any outward signs of infection in bulls and the only signs in cows are related to decreased reproductive efficiency. Vaccines targeting *C. fetus* subsp. *venerealis* are available in oil adjuvanted and aluminum hydroxide absorbed types. Oil adjuvanted vaccines are considered more effective particularly if only one dose is administered.\(^1\) Vaccination is generally effective if administered prior to breeding and appropriate boosters are given when an animal is first vaccinated.\(^32\) The response to vaccination against *C. fetus* subsp. *Venerealis* has been recently reviewed.\(^33,\)\(^34\) It has been recommended that bulls receive 2.5 times the normal dose of oil adjuvanted vaccine prior to breeding to produce protective immunity.\(^1\) There is some evidence that vaccination is effective in the treatment of infected bulls.\(^33,\)\(^34\)

### *Tritrichomonas foetus*

* *T. foetus* is a protozoal pathogen responsible for infertility, early embryonic death and abortion in cattle. Transmission is venereal. Infected bulls serve as the reservoir in most situations. Diagnosis and management of *T. foetus* infection is somewhat complicated and has been recently reviewed.\(^35\) There is currently one commercially available vaccine for *T. foetus* in the United States. The vaccine is labeled for use in cows and claims to reducing shedding of the organism. Vaccination against *T. foetus* has been described in recent reviews as being effective for prevention of infection and treatment of infection in bulls.\(^33,\)\(^34\) However, a recent meta-analysis\(^36\) of the published literature concluded that the quantity and quality of the published literature was insufficient to make conclusions regarding the efficacy of the vaccine. Vaccination against *T. foetus* appears to be most useful when working to clear up an infected herd or when other risk factors for *T. foetus* transmission cannot be fully controlled.\(^1,\)\(^32,\)\(^35\)

### *Neospora caninum*

*Neospora caninum* is a protozoal pathogen that can cause abortion in cattle. Cattle are incidental hosts and become infected when feed is contaminated with canine feces. Vertical transmission via transplacental infection can also occur in cattle. The diagnosis and control of Neosporosis in cattle has been recently reviewed.\(^37\) A vaccine for *N. caninum* was previously available but it was not effective and is no longer on the market. Currently there are no effective vaccines for *N. caninum* available in the United States.
References

Managing Preputial Injuries in Bulls
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Preputial injuries are common in bulls and can have a serious impact on the overall productivity of a cow herd. In single sire settings, preputial injury may have a significant impact on pregnancy rates if a bull is no longer able to breed cows successfully and a replacement is not provided quickly enough. Producers often make considerable investments in bulls as a primary way to improve the genetics of a herd and significant economic loss can be incurred due to serious preputial injury.

Preputial injury occurs most commonly in pasture breeding situations but can also occur in bulls housed in artificial insemination studs. Injury may occur during the breeding process or may result from habitual prolapse and subsequent trauma. *Bos indicus* influenced breeds are more likely to experience preputial injury due to their tendency for habitual prolapse. Anatomic factors such as a pendulous sheath, a longer prepuce and a larger preputial orifice lead to habitual prolapse in these bulls. Although less frequent, *Bos taurus* breeds commonly experience preputial injury. Polled bulls tend to have poorly developed preputial retractor muscles making them more prone to habitual prolapse. Breeding accidents can occur in bulls of any breed.

If the bull is able to retract the prepuce into the sheath following injury, the injury may not be noticed for several days. It is typical for these injuries to be discoverable by 7-10 days old when they are presented for veterinary care. The types of injuries include laceration, avulsions, and abscess formation. Any of these injuries may be complicated by significant edema and/or infection. Preputial injury in bulls has been reviewed.1,2

**Preputial laceration**
Laceration is probably the most common type of preputial injury. Lacerations may occur as a direct breeding injury or may occur secondary to preputial prolapse. Most lacerations occur on the ventral aspect of the prepuce. Most lacerations are not discovered acutely and are usually complicated by edema and infection. As the prepuce swells, prolapse tends to occur making the injury easier to see. If the swelling continues, paraphimosis may develop and the bull will not be able to retract the prepuce back into the sheath. This sets up a vicious cycle as the edema worsens due to gravity and further injury may occur. In cold climates, the prolapsed prepuce is highly susceptible to frostbite. If a preputial laceration occurs during the breeding season, the bull must be replaced because he will not be available for the current breeding season regardless of what treatment he undergoes.

The primary goals of initial treatment of this type of injury are to control infection and edema and get the prepuce placed back into the sheath where it will be protected. In many cases the prepuce is markedly swollen and dirty. To facilitate adequate examination, soaking the prepuce in warm water with a mild disinfectant may be useful. This will help remove scabs and other debris making examination easier. Warm water hydrotherapy for 20-30 minutes and massage may help reduce edema enough to get the prepuce back into the sheath. If the prepuce cannot be placed back into the sheath, it must be protected with a bandage. Support from some type of sling may also be useful by holding the prepuce close to the ventral abdominal wall to reduce the development of dependent edema. A soft plastic tube should be inserted into the preputial lumen to facilitate urination. The tube should be soft and flexible to prevent iatrogenic injury but must be rigid enough to maintain patency under a pressure bandage. A section of nasogastric tube or milking unit tubing will work well. The cut edges should be smoothed and adding multiple fenestrations to one end will facilitate urine flow. In addition to providing for urine flow, the tube can serve as a rigid structure to facilitate the placement of a pressure bandage. Bandaging the prepuce under moderate pressure will help reduce the edema more quickly. Prior to placing the bandage, a disinfecting ointment or emollient should be massaged into the prepuce taking care not to get the ointment on the hair. A mixture of anhydrous lanolin, scarlet oil and oxytetracycline works well. The bandage should be changed and the prepuce massaged daily until it can be placed back into the sheath. Broad spectrum antibiotics and anti-inflammatory therapy should be provided as needed.

Once the prepuce is back in the sheath, it should be retained there and allowed to heal for several days. The easiest method of retaining the prepuce is with a bandage. A tube is placed in the preputial lumen as previously described. Ideally, the tube should extend past the proximal aspect of the laceration to minimize urine contamination. Elastikon® is applied to the tube while the tube is clean and dry. The tube is then lubricated with ointment and placed in the preputial lumen. The tube should be inserted up to the previously applied Elastikon®. The Elastikon® is then wrapped onto the haired skin to hold the tube in place. For protection, the bandage is then covered with white athletic tape. It is critical not to get any of the ointment on the haired skin so that the tape will stick. If necessary, the top edge of the bandage can be sutured to the skin with a few interrupted sutures. The bandage is left in place for 7-10 days to give the prepuce a chance to heal. As an alternative to a bandage, a purse-string suture may be placed in the preputial orifice. The skin should be thoroughly cleaned to help prevent infection.

After 7-10 days, the prepuce should remain retracted into the sheath. If it does not, the bandage or purse-string should be replaced. If the prepuce is damaged too badly to save or if the owner elects to salvage the bull, amputation of the prepuce is a good option. A rigid plastic tube is placed in the preputial lumen and a rubber ring is placed around the prepuce proximal to the affected area. A
The prognosis for return to breeding soundness is generally good. The swelling is similar in appearance to that seen with a penile hematoma but it is located more distally. Significant abscessation may develop any time the injured prepuce is retracted into the sheath and the injury is not noticed. This most commonly occurs with lacerations or other injuries that occur near the fornix. Most affected bulls have a characteristic swelling at the level of the preputial fornix. The swelling is similar in appearance to that seen with a penile hematoma but it is located more distally. Significant

### Preputial Avulsion

Preputial avulsion is a breeding injury that occurs most commonly during semen collection with an artificial vagina. If the prepuce sticks to the inside of the AV when the bull thrusts during ejaculation, the prepuce may tear at the fornix. Occasionally the tear may encompass the full circumference of the prepuce. These injuries are usually noticed immediately. Primary closure following cleaning and debridement is usually an option for these injuries. If the injury is too old for primary closure, treatment is similar to that described for a preputial laceration. If left untreated, a preputial abscess may develop. Prognosis is generally good following primary closure and 3-4 weeks of sexual rest.

### Preputial Abscess

Abscessation may develop any time the injured prepuce is retracted into the sheath and the injury is not noticed. This most commonly occurs with lacerations or other injuries that occur near the fornix. Most affected bulls have a characteristic swelling at the level of the preputial fornix. The swelling is similar in appearance to that seen with a penile hematoma but it is located more distally. Significant

### Indications for Surgical Intervention

Indications for surgical intervention include persistent prolapse, preputial stricture, and phimosis. Occasionally an owner will request prophylactic surgery for a bull that is not injured but might be at high risk for preputial injury. Presurgical evaluation should include an assessment of tissue health, prepuce length, and proximity of the injury to the hairline. Surgery should be performed on healthy tissue. If infection remains or healing is not complete, surgical intervention should be delayed. Preputial avulsion is a breeding injury that occurs most commonly during semen collection with an artificial vagina. If the prepuce sticks to the inside of the AV when the bull thrusts during ejaculation, the prepuce may tear at the fornix. Occasionally the tear may encompass the full circumference of the prepuce. These injuries are usually noticed immediately. Primary closure following cleaning and debridement is usually an option for these injuries. If the injury is too old for primary closure, treatment is similar to that described for a preputial laceration. If left untreated, a preputial abscess may develop. Prognosis is generally good following primary closure and 3-4 weeks of sexual rest.

### Preputial Resection and Anastomosis

Preputial resection and anastomosis, also called reefing, is the preferred option in most cases. The prognosis for return to breeding soundness is higher following reefing than with circumcision. Preputial resection is performed with the penis extended. After aseptic preparation, a circumferential incision is made both distal and proximal to the affected area. Marker sutures should be placed both distal and proximal to the affected area to ensure proper alignment during the anastomosis. Care should be taken to insure, as much as possible, that adequate length of healthy prepuce is left. The incisions are extended through the preputial mucosa to the superficial layers of elastic tissue. A third longitudinal incision is made connecting first two incisions. The lesion and associated fibrous tissue are then removed. The mucosa is then apposed using absorbable suture in a simple continuous pattern. A Penrose drain is then sutured over the tip of the glans to protect the incision from urine. Topical antibiotic is applied to the incision and the penis is allowed to retract back into the sheath. A bandage or purse-string suture is placed to prevent prolapse. A scar revision surgical technique has recently been described. It is reported that this technique may be better suited to bulls that have a limited length of healthy preputial tissue remaining. In short, an elliptical incision is made around the scar and the scar removed. The defect is then closed longitudinally using a bootlace suture pattern. Postoperative care is similar regardless of the surgical technique used. Systemic antibiotics are provided for 5-7 days along with appropriate anti-inflammatory therapy. The bandage or purse-string can usually be removed in a few days as long as prolapse does not occur. Complete sexual rest is required for at least 6-8 weeks, at which time the bull should be evaluated for breeding soundness. The prognosis for return to breeding soundness is generally good.
cellulitis may be present. Drainage through the prepuce may be attempted by passing a rigid pipette through the preputial orifice to the fornix and penetrating into the abscess. Even though the abscess is subcutaneous, drainage through the skin is unlikely to be successful because adhesions will develop that will prevent extension of the penis. Antibiotics and anti-inflammatory therapy are indicated. Prognosis for return to breeding soundness is generally poor due to adhesion formation and immediate salvage may be the best option if severe cellulitis is not present. Recovered bulls could be used for semen collection for artificial insemination.

References

Effective management of pain resulting from husbandry practices or disease is a major challenge for food animal practitioners. The challenge is exacerbated by growing concern among consumers of food animal products about pain experienced by livestock animals. That concern is most notable regarding pain caused by routine husbandry practices such as castration and dehorning. In response to growing concern among consumers, producers, and veterinarians about pain experienced by livestock animals, considerable research has been conducted on the topic of pain alleviation over the past several years. A complete review of the published literature regarding pain in livestock is beyond the scope of this document. However, readers are referred to the March 2013 issue of the Veterinary Clinics of North America: Food Animal Practice for a very thorough review of the literature relative to this topic. That issue can be accessed online at [http://www.vetfood.theclinics.com/issue/S0749-0720(13)X0002-7](http://www.vetfood.theclinics.com/issue/S0749-0720(13)X0002-7).

**Challenges to effective analgesia**

One of the biggest challenges to providing effective analgesia to food animals is the lack of Food and Drug Administration (FDA) approved medications for pain relief for food animals. Currently there are no medications labeled for analgesia in food animals in the United States.¹ Flunixin meglumine is labeled for use in cattle for treatment of inflammation due to a variety of disease processes but it is not labeled for analgesia. Since there are no medications approved for analgesia in food animals, administration of any medication for the purpose of analgesia requires Extra-Label Drug Use (ELDU). In order for ELDU use to be legal, it must occur by or on the order of a veterinarian within the confines of a valid Veterinary Client Patient Relationship (VCPR). Readers are referred to the practice act for their respective state for clarification of what constitutes a VCPR. Food animal veterinarians perform ELDU routinely, if not daily, so this should not prevent veterinarians from prescribing medications for the purpose of analgesia. A primary reason that there are no medications approved for analgesia in food animals is that there is no consistent, reliable, repeatable way to measure pain in livestock species. Researchers have used a variety of methods to assess pain. Measurement of serum cortisol has been the traditional method used to assess pain response. Cortisol increases in response to pain but the response can be variable and other stimuli can affect the cortisol response. Other techniques for assessing pain in food animals include measurement of acute phase proteins such as substance P and haptoglobin, assessment of behavioral changes, heart rate or heart rate variability, assessment of feed intake and average daily gain, and even measurement of skin temperature. Techniques for assessing pain are being refined and new ones are being developed. Methods of assessing pain in food animals are discussed in detail in several recent review articles.²⁻⁴ Cost is cited as another common reason that analgesic drugs are not used in food animals. Costs can be measured in terms of actual costs of the medications or in terms the time required to appropriately administer analgesic medications. Many producers perform common husbandry practices such as castration and dehorning themselves. Involving a veterinarian to meet the requirements of ELDU can result in additional costs as well. The idea that pain reduces animal performance is well documented in the literature. It is logical that providing analgesia should improve animal performance thereby offsetting at least some of the costs of providing the analgesia. While few studies have been able to document a significant improvement in performance attributable to the provision of analgesia for practices such as castration and dehorning, recent studies have shown improvements in health and average daily gain (ADG) when analgesia is provided at the time of castration.⁵⁻⁶ In a very recent abstract, Roberts et al² investigated castration, castration + meloxicam and no castration (controls) in 3 month old beef calves. Calves castrated with meloxicam had decreased ADG for 2 weeks post castration compared to castrated calves receiving meloxicam and non-castrated controls.

**Motivations for providing analgesia**

Veterinarians and producers may have different motivations for providing analgesia to food animals. Possible motivations include improving animal performance, improving animal health, and improving animal welfare. As mentioned above, the evidence for improved performance or health attributable to analgesia is limited. The primary force driving consumer concerns about pain in food animals is concern about the impacts of pain on animal welfare. Improving animal welfare should be a motivating force for veterinarians and producers as well. Even in the absence of improved performance, improving welfare is justification for the adoption of analgesic practices in food animal production. In fact, providing analgesia for painful practices may become simply a cost of doing business in order to maintain access to markets and to maintain consumer demand for food animal products. Producers and veterinarians should be proactive in the adoption of practical analgesic practices in an effort to improve the welfare of livestock animals.
Preemptive analgesia
Preemptive analgesia is the concept of providing analgesia prior to the onset of a painful stimulus. Preemptive analgesia is often more effective than analgesia occurring after pain is perceived. While preemptive analgesia is not always possible, it can be effectively provided for procedures such as dehorning, castration and other elective or planned surgical procedures.

Multimodal analgesia
Multimodal analgesia is the application of multiple analgesic treatments, typically with different mechanisms of action. Multimodal analgesia is generally more effective than single analgesic treatments and is discussed in more detail later in this document.

Commonly used analgesic drugs
Food animal veterinarians have access to numerous drugs that have analgesic properties. These drugs include local anesthetics, non-steroidal anti-inflammatory drugs (NSAIDs), opioids, α2-agonists, and N-methyl-D-aspartate receptor antagonists (Ketamine). Gabapentin is another drug that may be useful for analgesia in some situations. Excellent reviews of the analgesic drugs available for use in food animals have been recently published. Table 1 at the end of this document lists commonly used analgesic drugs along with doses, routes of administration and withdrawal times.

Application of analgesia in food animal practice
The following provides a brief discussion of practical ways to provide analgesia in common food animal practice situations.

Castration
Studies investigating analgesia for castration are numerous and an excellent review of the topic has been recently published. Readers are referred to this review for a detailed review of the published literature.

Local anesthesia for castration can be provided in multiple ways. Lidocaine can be injected directly into the testicle or into the spermatic cord. The dose for intratesticular injection varies depending on the size of the testicle. The author typically injects 2-10 mls directly into each testicle. There is a notable increase in injection pressure and an increase in firmness of the testicle as the tunic fills with lidocaine. The author stops the injection once the testicle feels full. Injection of 5-10 mls of lidocaine directly into each spermatic cord can also be effective. Injection into the spermatic cord has the advantage of blocking the cremaster muscle which is not blocked with intratesticular injection. Subcutaneous infiltration of lidocaine into the distal aspect of the scrotum can be helpful for anesthetizing the skin at the site of the scrotal incision.

Local anesthesia has been shown to reduce the acute cortisol response associated with castration but the overall area under the curve for cortisol concentration was only modestly reduced. This suggests that the effects of local anesthesia are short lived. Administration of an NSAID has been shown to be beneficial for alleviating pain associated with castration. Administration of an NSAID prior to castration does not abolish the acute cortisol response but the total area under the curve is reduced to a greater extent than that seen with local anesthetics alone. Unfortunately, many study protocols resulted in the NSAID being administered 20 minutes or more prior to the castration procedure which makes application of those study findings difficult.

The combination of local anesthesia and NSAID resulted in the greatest reduction in the total area under the curve for cortisol concentration post castration suggesting that multimodal therapy is more effective than either therapy administered alone. More recent studies support this finding as well.

It is often recommended that calves be castrated as early as possible to reduce the negative effects of castration. Powell et al demonstrated the benefits of early castration on average daily gain at weaning. Calves castrated at birth had higher post weaning ADG than calves castrated at weaning. When calves castrated at weaning given a single dose of meloxicam, ADG was improved compared to calves not receiving meloxicam. Additionally, calves castrated at weaning demonstrated altered immune function. Meloxicam administered at weaning reduced haptoglobin concentrations compared to calves that did not receive meloxicam. These findings are consistent with those published by Coetzee et al demonstrating a decrease in the incidence of respiratory disease when calves received meloxicam prior to castration when entering the feedlot.

Dehorning
Studies investigating the effects of various analgesic treatments for alleviation of pain associated with dehorning are numerous. For a detailed review of the literature, readers are referred to an excellent recent review by Stock and colleagues.

Unlike castration, a simple nerve block can provide complete anesthesia of the horn in young calves. Local anesthesia has been shown to alleviate the initial rise in cortisol concentrations observed when calves are dehorned without anesthesia. The cornual nerve block is performed by injecting 5-10 mls of lidocaine subcutaneously just ventral to the temporal ridge of the frontal bone at a point halfway between the lateral canthus of the eye and the horn. With larger horns, a partial ring block may need to be performed on the caudal aspect of the horn to achieve complete anesthesia. A successful cornual nerve block alleviates the pain associated with the dehorning procedure and also makes the procedure easier for the operator.

Provision of an NSAID has been shown to be useful to help alleviate the pain associated with dehorning once the effects of the cornual nerve block wear off. Several studies have demonstrated a delayed rise in cortisol once the effects of local anesthesia have
subsided. This delayed cortisol peak can be attenuated by administration of an NSAID. As with castration, many study protocols administer the NSAID 20 minutes prior to dehorning making application of the results difficult. More recent work with meloxicam administered at the time of dehorning has demonstrated a positive effect.14

Sedation may also be useful for providing analgesia during dehorning. Whenever possible a multimodal approach to analgesia including local anesthesia, NSAIDs and sedation (if possible) is recommended.13

**Lameness**

The provision of analgesia for lameness has been recently reviewed.15 Providing effective analgesia for lameness is challenging because lameness is often chronic by the time the animal is presented for veterinary care. Chronic pain is less responsive to analgesic compounds due to central sensitization and wind up.8 Alleviation of the inciting cause of lameness is critical for alleviation pain. Corrective claw trimming and the application of claw blocks are useful in the alleviation of pain in many causes of lameness.15

Local anesthesia of the distal limb via regional intravenous anesthesia is a simple and effective method for providing analgesia during painful claw procedures or digital surgery. Briefly, a tourniquet is placed proximal to the area to be anesthetized, most commonly over the middle of the metacarpus or metatarsus. Lidocaine (20-30 mls) is then infused into any accessible vein distal to the tourniquet. The dorsal common digital vein is used most commonly but the abaxial sesamoid veins can be used as well. The procedure will provide complete anesthesia of the limb distal to the tourniquet. The author has left tourniquets in place for 30-45 minutes with no adverse effects.

Published reports of studies investigating analgesia for lameness are scarce. Listed below are a few studies demonstrating beneficial effects of analgesia for lameness in cattle.

**Chapinal N et al. J Dair Sci, 93 2010 pp. 3039-3046**
- Flunixin meglumine (2.2 mg/kg IV) administered immediately before corrective hoof trimming and 24 hours later
- No difference in gait scores but untreated cows spent more time lying down compared to treated cows

- Induced lameness model using intraarticular amphotericin B
- Flunixin meglumine (1 mg/kg IV) at the time of lameness induction and 12 hours later
- Treated calves had improved lameness scores and reduced recumbency compared to untreated controls

- Induced lameness model using intraarticular amphotericin B
- Treatments included placebo, meloxicam (0.5 mg/kg PO), or meloxicam + gabapentin (15mg/kg PO) administered 4 hours after lameness induction and repeated once daily for 4 days.
- Meloxicam alone or with gabapentin reduced lameness severity in the experimental model

**Surgical pain**

Whenever possible preemptive analgesia should be employed to manage pain associated with surgery.8 Since most bovine surgery is performed with the animal awake, clinicians must have a good grasp of local and regional anesthetic techniques. Reviewing these techniques is beyond the scope of this paper but an excellent review of common techniques has been recently published.8 Local anesthetics and NSAIDs are commonly used to provide intra- and post-operative analgesia. Opioids such as morphine or butorphanol are commonly used for post-op analgesia in the author’s clinic.

**Obstetrical pain**

Published studies investigating the use of analgesia are scarce. It seems plausible that providing analgesia, particularly following a difficult dystocia, would be beneficial to the cow but data on which to base those decisions is lacking. In a recent review on the topic, Laven et al concluded that available data on the use of NSAIDs post calving was insufficient to allow formulation of recommendations regarding NSAID use. The authors concluded that more research on the subject was needed. Also, there is some evidence that periparturient administration of NSAIDs may lead to an increased incidence of retained placenta.

The studies listed below demonstrate some of the recent work investigating the periparturient use of NSAIDs.

**Newby NC et al. J of Dair Sci, 96(6) 2013 pp. 3682-3688**
- Meloxicam administered 24 hours post calving (0.5 mg/kg of body weight)
- No difference in dry matter intake, milk production, blood metabolites, or health events
- Meloxicam increased feeding time and the frequency of bunk visits for 24 following injection

- No difference in the incidence of retained placenta in meloxicam treated versus untreated controls

**Stilwell G et al. J Dair Sci, 97 2014 pp. 888-891**
- Carprofen (1.4 mg/kg IV) administered immediately after calving
- Treated cows spent more time eating in the 24 hours after calving
- 305 day milk yield was higher in primiparous cows treated with carprofen
- Carprofen treatment increased the time from calving to conception
References


In order to improve productivity and profitability of a beef cow enterprise, an assessment of productivity based on production records must be performed. According to the 2007-2008 National Animal Health Monitoring System Beef Cow-Calf Report, over 83% of all beef cow/calf operations maintained some form of production records. In herds with more than 100 cows, the percentage increased to 90%. Just keeping records is of little value to the beef producer. For records to be useful, the data must be summarized and analyzed to aid in making management decisions that will affect the overall productivity of the operation. Analysis and interpretation of production records is a valuable service veterinary practitioners can provide to their clients.

Analysis of beef cow/calf production records does not have to be complex to be useful. Numerous record keeping software systems are commercially available. Most of these programs will conduct routine analysis procedures and provide a report to the user. Additionally, simple spreadsheet program can be used to conduct most of the useful calculations.

Production records analysis has two broad goals. The first is to evaluate the productivity of the cow/calf operation as a whole. This is done in an effort to identify areas where the operation is doing well and, more importantly, areas where changes might be made to increase the productivity of the herd. The second goal is to evaluate the performance of individuals within the herd to aid in selection and culling decisions. Selection and culling decisions are often based on visual appraisal which may not provide an accurate assessment of an animal’s true productive capacity.

The goal of this paper is to review the process of production records analysis for parameters that have a significant effect on the productivity of an operation. It will not include a complete discussion of all possible analysis techniques but will focus on those that are easy to obtain and use. For additional information, the reader is referred to a recently published review on this topic. Beef cow/calf production records are typically divided into three categories: breeding, calving, and weaning.

**Evaluation of breeding records**

Breeding and pregnancy performance is measured by several indices. The denominator used in calculation of most of these indices is the number of cows exposed to a breeding opportunity. It is important to accurately determine this number prior to calculating performance indices. In order to insure accuracy, a few adjustments may have to be made to arrive at the actual number of females exposed to a breeding opportunity. Any exposed pregnant females sold or transferred out of the herd prior to pregnancy diagnosis should be subtracted. Any exposed females or pairs purchased prior to pregnancy diagnosis should be included. Exposed females that die prior to pregnancy diagnosis should be included. Any exposed females that are intended to be sold but remain in the exposed female herd are subtracted from the number exposed when they are sold.

**Pregnancy percentage**

- Calculated as the number of exposed females diagnosed as pregnant divided by the number of exposed females X 100
- May also be measured by strata of interest such as cow age, body condition score, service sire, etc.
- **Key points**
  - Year-to-year variation will occur due to environmental stresses
  - This value should only be used in comparisons with similar operations
  - A low value may indicate a problem but it does not identify the cause of the problem
- **Targets:** 90-95% for cows with a 60 day breeding season, 80-85% for heifers with a 45 day breeding season

**Time to conception**

- Determines how quickly cows become pregnant after being exposed to a breeding opportunity
- Requires accurate estimation of gestational age at pregnancy diagnosis in natural service herds.
- Determined using the beginning exposure date and the days pregnant on the day of examination
- Allows creation of a pregnancy rate distribution

**Pregnancy rate distribution**

- Visualized using a simple histogram
- Distribution interval determined by evaluator (1 week, 2 week, etc)
  - Most common to break the breeding season into 21 day periods
- May be stratified according to parameter of interest
- Ideal distribution is strongly left skewed indicating that a high proportion of cows became pregnant early in the breeding season
  - Should expect 60-65% of the herd to become pregnant in the first 21 days if cows and bulls are fertile at the beginning of the breeding season
Flat or right skewed distribution may indicate delayed estrus or the presence of an infectious disease causing early embryonic death. An excellent review of interpretation of pregnancy rate distribution has recently been published.

**Pregnancy loss percentage or abortion percentage**
- Calculated as the number of cows that are diagnosed pregnant but fail to calve divided by the number of cows diagnosed as pregnant X 100
- May be stratified according to parameters of interest (cow age, body condition score, management group)
- Target: Less than 2%

**Evaluation of calving records**
The denominator for many of the gestation and calving performance indices is the number of cows exposed to a breeding opportunity. To insure accuracy, this number should be carefully derived as described above.

**Calving percentage**
- Calculated as the number of females calving divided by the number of females exposed X 100
- Is a good measure of breeding performance and gestational management
- Directly related to pregnancy percentage and pregnancy loss percentage
- Key points
  - May indicate that a problem exists but does not identify the cause
  - Year-to-year variation should be expected
  - Does not describe the calving distribution
- Target: 80-85%, National average reported as 91.5% in NAHMS 07-08 Beef Report.

**Pregnancy loss percentage or abortion percentage**
- See description above

**Calving distribution**
- Cumulative number of calves born by 21, 42, 63, and >63 days of the calving season divided by the total number of calves born X 100
- Beginning of the calving season is 285 days after bulls were first introduced or the date on which the 3rd mature cow calves
- Should closely mirror the pregnancy rate distribution with the ideal distribution being strongly skewed to the left indicating that a high proportion of calves were born early in the calving season
- Viewed a simple histogram
- Targets: 60-65% born in 1st 21 days, 85-90% born by 42 days, 100% born by 63 days
- May be stratified according to parameters of interest

**Calf death loss**
- Based on cows exposed: calculated as number of calves that died divided by the number of cows exposed X 100
- Based on calves born: Calculated as number of calves that died divided by the number of calves that were born X 100
- May indicate deficiencies in herd health, calving environment, nutrition, or breeding program
- Includes all calf deaths from birth to weaning
- Does not distinguish between calf deaths at birth versus during the suckling period

**Calving interval**
- Period from the birth of one calf until the birth of the next calf
- Target: 12 months

**Evaluation of weaning records**

**Calf crop or weaning percentage**
- Calculated as the number of calves weaned divided by the number of cows exposed X 100
- Encompasses breeding, gestation, birth, and rearing the calf
- Any calves purchased prior to weaning should not be included
- Target: >75%

**Measures of calf weight**
- Actual weaning weight
  - Calculated as total weight of weaned calves divided by the number of weaned calves
  - Usually stratified by contemporary group (bulls, steers, heifers)
  - Not standardized for a given age
  - Not useful for comparisons between operations due to differences in environment and management
 Target: 40% of cow’s mature weight;
National average reported as 559 lbs for bulls and steers and 515 lbs for heifers.

- Weight per day of age
  - Calculated as calf’s weaning weight minus the birth weight divided by age in days at weaning
  - Allows standardized comparisons between calves of different ages

- Adjusted 205 day weaning weight
  - Calculated by multiplying weight per day of age by 205 and adding birth weight
  - Allows standardized comparisons between calves of different ages

- Each of these measures are typically evaluated as averages for a given contemporary group

**Calf death loss**

- See calculation described above
- May be stratified as time of death relative to calf age

**Overall measure of herd performance**

**Pounds of calf weaned per cow exposed**

- Calculated as total pounds of calf weaned divided by the number of cows exposed
- Non-specific single index of performance of the cow herd
- May provide a single year-to-year index of herd performance
- Some year-to-year variation should be expected

**Measures of individual animal performance**

The indices described above evaluate performance of the cow herd as a whole. Objective evaluation of individuals within the herd is also necessary for the purposes of selecting animals as replacements, making breeding decisions, and making culling decisions. Comparisons of individual animals within their contemporary groups are usually made by the use of ratios. Ratios provide an evaluation of an individual’s performance relative to the average performance of their contemporary group. For example, adjusted weaning weight ratio is calculated as an individual’s adjusted 205 day weaning weight divided by the average adjusted 205 day weight of the contemporary group and the result is multiplied by 100. A calf with a weaning weight ratio of 100 is average for the group. A ratio of 110 indicates that that calf’s performance was 10% better than the average for his contemporaries.

**Most probable producing ability**

- Measure that allows accurate comparison of future production potential for cows with different numbers of progeny
- Can be calculated for various traits
- Calculated according to the following equation (weaning weight example):
  \[ \text{MPPA} = H + \frac{NR}{1 + (N-1)R} \times (C-H) \]
- Where: 
  - \( H \) = herd average weaning weight ratio
  - \( N \) = number of calves included in the cow’s average
  - \( R = 0.4, \) the repeatability factor for weaning weight ratio
  - \( C \) = average weaning weight ratio for all calves the cow has produced
- Value is calculated by most cow/calf records keeping programs
- Cows can be ranked within a herd by MPPA for adjusted 205 day weaning weight
- Should be used to evaluate cows within contemporary groups
- Interpreted as a ratio
  - MPPA of 100 is average
  - MPPA of 110 indicates production 10% above average for the group
  - MPPA of 95 indicates production 5% below average for the group

**References**

Cattle veterinarians and producers are frequently faced with the need to humanely euthanize animals due to illness or injury. Based on the Guidelines for Euthanasia of Animals published in 2013 by the American Veterinary Medical Association, cattle can be euthanized by injection of a barbiturate overdose or by physical disruption of the brain via gunshot or captive bolt device. While barbiturate overdose is usually the most aesthetically pleasing method of euthanasia, it is impractical for use in most on-farm euthanasia situations. Barbiturate overdose can only be performed by veterinarians due to the controlled nature of the medication, it is expensive and it leaves a potentially dangerous carcass/environmental residue if carcasses are not disposed of properly. That leaves producers and veterinarians with physical disruption of the brain as the only approved method of euthanizing livestock in most on-farm situations. Although gunshot and captive bolt devices have been around for many years, recent work over the last few years has aimed to improve the effectiveness and humaneness of these euthanasia methods.

Effects of captive bolt or gunshot on brain function
The correct application of a captive bolt or gunshot to the brain of an animal during euthanasia results in instantaneous loss of consciousness followed by death. It is believed that the concussive forces of the bolt or projectile striking the skull are responsible for the immediate loss of consciousness. Physical disruption and/or destruction of brain tissue then occur as a result of penetration of the bullet or bolt into the brain. This direct penetration of the brain causes a rapid increase in intracranial pressure, intracranial hemorrhage, and displacement of brain tissue. Additionally, cavitation, shockwaves and rotational forces applied to the brain result in further destruction of brain tissue. As a free bullet penetrates the skull and brain, it tends to expand causing more severe cavitation injury than what is typically seen with a penetrating captive bolt since the shape of the bolt doesn’t change.

Anatomic landmarks for captive bolt euthanasia
Traditional descriptions of the preferred landmarks for captive bolt euthanasia describe shooting at the intersection of two imaginary lines drawn from the medial canthus of each eye to the opposite horn or top of the opposite ear. Recent research has shown that this position is too far rostral resulting in failure to physically disrupt the brainstem and in some types of cattle results in missing the brain altogether. More recent guidelines from the AVMA and the American Association of Bovine Practitioners describe placing the shot at the intersection of two lines drawn from the lateral canthus to the opposite horn or top of the opposite ear. This position is also consistent with the recommendations of the World Organization for Animal Health. Based on further recent investigations into captive bolt shot placement, the author recommends placing the captive bolt on midline halfway between the top of the poll and an imaginary line drawn between the lateral canthus of each eye. (Figure 1) This position more accurately places the captive bolt directly over the brainstem in a variety cattle types and head shapes.

Application of the penetrating captive bolt
Due to the limited penetration depth of captive bolt devices, euthanasia via captive bolt requires precise shot placement and requires that the muzzle of the captive bolt device be pressed firmly against the animal’s head at the time of the shot. The device should also be held as close to perpendicular as possible relative to the animal’s head. Placing the shot correctly requires good restraint since the device must be in contact with the head. In recumbent animals the author prefers the use a halter to tie the head around to the animal’s hind limb. The author prefers to sedate ambulatory animals with a heavy dose of xylazine and allow them to become recumbent prior to euthanasia. Sedation is not required and animals may be euthanized while restrained in a chute but removing them from the chute post-euthanasia can be problematic.

Behavioral responses of the animal are used to confirm loss of consciousness following captive bolt shot. An animal that is standing when shot should collapse immediately. The eye should be centered within the orbit and display a dilated pupil with lack of a corneal reflex when the eye is touched. The lack of a corneal reflex is the most sensitive indicator of unconsciousness. Additionally, there should be immediate loss of coordinated respiration and no coordinated vocalization. Limb movement is common following captive bolt shot and is not a sign of returning sensibility. In some cases limb movement can be quite dramatic and care should be taken to avoid being injured by flailing limbs.
Because a penetrating captive bolt creates less brain trauma than the typical free bullet, it is recommended that the captive bolt shot be followed with a secondary step to insure death once loss of consciousness in confirmed based on the reflexes described above. Possible secondary steps include exsanguination, pithing, pithing or intravenous injection of a saturated salt solution such as potassium chloride (KCL). Pithing can be performed by inserting a metal rod or similar device into the hole created by the bolt. The rod is inserted into the brainstem and possible into the proximal spinal cord creating additional damage to the brain tissue. Potassium chloride solution can be created by dissolving KCL into warm water until the solution becomes saturated. Sixty to 120 mls injected intravenously will readily stop the heart of most cattle. Potassium chloride should never be injected into a conscious animal because it induces cardiac arrest but does not induce unconsciousness.

**Euthanasia via gunshot**

Although gunshot has been used as a euthanasia method for many years, there is relatively little published research regarding the topic. There is a tremendous number of possible combinations of firearm type, caliber, and bullet type. This variety coupled with limited research makes it difficult to make recommendations regarding the ideal equipment for gunshot euthanasia of cattle. A recent study conducted at Kansas State University investigated the effectiveness of a variety of firearms for euthanasia of feedlot cattle. This study included rifles, handguns and shotguns. The authors concluded that all of the firearm/bullet combinations tested were effective for euthanasia of feedlot cattle except for the .22 caliber hollow-point and the 9mm pistol firing a full metal jacket bullet.

In the absence of data to make specific recommendations regarding caliber and bullet type, some general recommendations should be made. Caliber selection should be balanced with the goal of having enough power to penetrate the skull and brain without having a significant risk of the projectile exiting the target animal thereby increasing the safety risk to other animals or people. Selection of an appropriate bullet type is critically important. Soft nosed lead bullets are preferred over hollow-points or bullets with a full metal jacket. Some hollow-points will fragment prior to penetrating the skull. The distortion of the shape of the bullet transfers energy into the brain and creates the profound cavitation effect that makes gunshot so effective. Bullets with a full metal jacket retain their shape and transfer less kinetic energy in the brain. These bullets also increase the risk of over penetration allowing the bullet to exit the target animal. Shotguns firing birdshot are very effective at close range and have minimal risk of overpenetration.

In most cases, shot placement for firearm euthanasia is similar to that described above for captive bolt. Just as with captive bolt, the goal is to disrupt the cerebral cortex and brainstem. However, one advantage of firearm euthanasia is that it does not require close contact with the animal. In some cases, the point of entry of the bullet may need to be adjusted to get the bullet to reach the target area of the brainstem. The brainstem lies on midline on the floor of the calvarium. Imagining a line drawn between the ear holes and shooting for the point where that line crosses midline will result in the bullet penetrating the brainstem. If the animal is at some distance from the person conducting the euthanasia with its head raised the entry point of the bullet will need to be lower on the head in order to reach to brainstem. The behavioral responses of the animal following gunshot are virtually the same as those described above for captive bolt. If the caliber and bullet type have been selected appropriately, a secondary step to ensure death is usually not needed following gunshot delivered to the brain.

**Conclusion**

Both gunshot and captive bolt euthanasia are effective and can be readily applied in on-farm situations where cattle require euthanasia. When performed correctly, both methods result in immediate loss of consciousness followed shortly by death.

**References**

Ruminants are large animals with similar bone structure to equine species. Long bone fractures are common in these species and primary cause of fractures in these species is thought to be traumatic in origin. The decision to treat a fracture in ruminants is made by evaluating the severity, the cost, success rate and the economic value/genetic value of the animal after repair. Once a fracture is suspected, further traumatization to the fracture site should be prevented by stabilization of the fracture. Stabilization will prevent further trauma to the fracture itself as well as the surrounding soft tissue. Compromise to all these structures can have a significant impact on the success of the fracture repair and may change the way you are going to approach your fracture repair.

The basic principles of fracture healing are similar to other species such as equine species. Fracture healing is associated with factors associated with the fracture itself as well as patient factors. Fracture configuration, stability and presence of infection as well as age of animal, body condition, temperament, and pregnancy can influence the healing of a fracture. Therefore, the healing of any fracture can vary tremendously between animals. Fractures in young animals have a tendency to heal faster than fractures in older animals.

When selecting the type of fracture repair there are certain factors to keep in mind such as location and type of fracture, degree of soft tissue damage, patient temperament, budget and the experience of the veterinarian. For example, open fractures have a more guarded prognosis than closed fractures. In some patients, a successful treatment of the fracture is not possible due to severity of trauma or overwhelming contamination/infection of fracture site. In these cases, euthanasia of the patient or limb amputation might be considered.

The methods of repair can be divided into external fixation methods and coaptation methods and internal fixation techniques. External fixation/coaptation methods are casts, cast bandages, splints, transfixation pin casts and external skeletal fixators. Internal fracture fixation can be accomplished with intramedullary pins and cerclage wire, intramedullary interlocking nails and bone plates and screws.

Not all fracture configurations can be repaired with each technique. Plus, each of these techniques has its advantages and limitations. In some cases, a combination of techniques might be used for a successful outcome. External fixation methods can cause malalignment, delayed union and non-union of the fracture site. Intramedullary pins have been reported to be limited primarily to humeral and femoral bone fractures. Short oblique, transverse, comminuted or segmental fractures are also not candidates for this type of fracture repair. The intramedullary interlocking nail construct is best applied in femoral, tibial and humeral fractures. This technique most likely requires the most specialized surgical skill and equipment. Fracture repair with bone plates and screws is similar to the technique in other species. Whatever technique is used for fracture repair, once the fracture is healed the orthopedic implants typically do not need to be removed unless they cause lameness or other problems.

In conclusion, the choice of repair of fractures in ruminants depends on many factors; however, we feel that external fixation techniques are often an economical and sufficient way to stabilize fractures in ruminants. Internal fixation provides more stability and allows near perfect anatomical reconstruction of the bone, which favors healing with minimal complications.
Dairy practices perform fewer cesarean sections compared to beef practices where they are numerous and more frequently in late winter and early spring compared to all year round. The basic goal of a cesarean section is preservation of the dam and calf and ensure a future reproductive efficiency for the dam.

Cesarean section

Indication for c-section

The indications for c-sections can be divided into maternal reasons and fetal indications. The main maternal indication/reasons for a c-section are inadequate dilation of the cervix, abnormalities to the dam’s urogenital tract, immature heifer, uncorrected uterine torsion, uterine tear, hydrops, prepartum paralysis and elective c-section. Fetal indications that could require a c-section are disparity between the fetus and the pelvis, irreducible malpositions and pathologic fetal conditions.

Surgical procedure for c-section

A c-section can be carried out under field conditions as occurs in many large animal practices. Patient positioning and approach is decided by the veterinarian based on a number of factors, such as patient attitude, facilities available, conditions of the fetus (position, viability and such).

Surgical procedure

There is not one best approach to perform a c-section. A clinician chooses the best approach for the present circumstances and what they are comfortable with at that time.

The main option to choose from first is whether to perform the c-section standing or in recumbent position. If standing is elected, the left paralumbar fossa approach is the most common approach to perform a C-section. With this approach the intestines are retained by the rumen and the uterus is easily accessible. A right paralumbar fossa approach or a left oblique paralumbar approach have also been described.

A recumbent approach can be on ventral midline, paramedian, low flank or paramammary approach to the abdomen. The paramammary approach can be useful in dairy cows because it avoids the caudal epigastric veins ad the ventral edema. The midline approach requires the longest incision since the linea alba is very inflexible.

Also due to the dorsal recumbency the respiratory ability is reduced due to the fact of the heavy gravid uterus being pressed on to the diaphragm and aspiration of rumen content may be a concern.

The surgical technique for performing a c-section are well described. Some important things to remember are:

- A small abdominal incision increases the level of difficulty in removing the fetus as well as increases the risk of subcutaneous emphysema. Therefore, it is important to make a large enough incision in the abdominal wall.
- Breech of posterior presentations may require larger incision due to difficulty of exteriorizing the uterus.
- During ventral approaches the greater omentum has to be retracted cranially.
- The incision in the uterus should be made along the greater curvature to avoid blood vessels and caruncles.
- A small uterine incision increases the risk of uterine tears.
- The umbilical cord should be stretched and ruptured in a controlled fashion to allow contraction of the umbilical arteries.

Umbilical complications in calves are seen approx. in 30% of calves delivered by c-section.

Always check for a second calf and remove placenta only if it readily detaches from the caruncles.

The uterus can be closed in one or two layers. The abdominal wall can be closed in two to three layers.

Post-operative care

Antimicrobials are frequently given for prophylactic and therapeutic reasons, especially under field conditions. The most common organisms to be encountered during a c-section are susceptible to procaine penicillin G, oxytetracycline and ceftiofur sodium. Post-operative pain management is frequently done and the most familiar agent is flunixin meglumine. The appropriate milk and meat withdrawals need to be followed when giving any medications in cattle.

To help with the involution of the uterus and passage of the placenta oxytocin (10-20 IU) is administered.

Standing flank paralumbar incisions require far less postoperative care and attention compared to ventral approaches.

Complications

Development of complications have been reported and the list is extensive. The complications can be categorized into pre-operative, operative, post-operative and long term complications. More details will be discussed during the presentation.
Outcome

Important in the outcome after a c-section is the case selection. When a c-section is considered a last resort as a treatment option, a negative outcome is more likely. If a c-section is chosen early on in a dystocia case, the procedure might be more rewarding. The condition of the cow at the time of surgery is another major factor to determine outcome.

The overall pregnancy rate in dairy and beef cattle that had a c-section has been reported to range from 72%-91%. Beef cattle in general tolerate surgery better than dairy cattle and their outcomes are more likely better. The effect of a c-section on milk production is difficult to assess due to numerous confounding variables. In dairy cattle it is believed that lactation after a c-section to be reduced by 80-1500L compared with their previous lactation. The overall risk of being culled in dairy cattle is higher for cows which have undergone a c-section compared to control cows (cows with no c-section).
Inhalation anesthesia is seldom feasible and economically justified in the field, so therefore injectable field anesthesia in different forms has been used extensively to perform surgical procedures in ruminants.

Pre-anesthetic preparation
Before a ruminant is anesthetized there are some considerations to keep in mind. A physical examination should always be performed before any surgical procedure, especially if anesthesia is involved. This examination helps to determine if the animal is suitable for field anesthesia and will help to reduce the complications.

The site of the surgery should also be carefully selected. The site should have good footing, free of hazards in all directions, calm environment and close to water, electricity or your vehicle if necessary.

Placing an intravenous catheter before anesthetizing an animal is advisable, but it might not always be possible or necessary depending on the procedure. The catheter will give you easy access to the vein during the procedure and will already be present in case complications are encountered.

Ruminants produce a significant amount of saliva when they are anesthetized. During field anesthesia certain reflexes such as eye and laryngeal reflexes are still present and care should be taken to position the head that saliva can run out of the mouth rather than pooling near the larynx.

Oxygen supplementation is generally not required in field anesthesia since the duration of the anesthesia is usually short-term. Eye protection and muscle /nerve protection are recommended. Eye protection can be performed by placing ophthalmic ointment in both eyes and in addition protecting the down eye with a towel or a thin pad. By paying attention to detail during the positioning of the animal before surgery, muscles and nerves are protected to reduce the risk of muscle or nerve damage.

Fasting of the animal before surgery is not always feasible either, especially in emergency situations. Withholding food for 12-24 hrs. and water for 12-24 hrs. in elective surgical procedures will decrease the severity of the tympany and therefore reduce the negative effects on the cardiorespiratory system. Withholding food does not greatly affect the volume of the rumen content in small ruminants but it is more appreciated in larger ruminants such as cattle. Therefore, it might be better to fast an animal in cattle if the procedure is an elective procedure to decrease the complication risk.

Endotracheal intubation is preferred if injectable anesthesia is maintained for longer than 10 minutes, especially if dorsal recumbency is necessary for the surgical procedure. Intubation will prevent aspiration of saliva and ruminal content.

Anesthesia methods
Local and regional anesthesia with/without sedation
Regional anesthesia with or without sedation is a popular and useful anesthetic method in ruminant practice. Regional anesthesia is low in cost and there is no need for extensive equipment. If no or minimal sedation is used, the cardiovascular and respiratory changes are minimal, the procedure can be performed in the standing animal so the risk for regurgitation and aspiration is low as well.

The simplest method for regional anesthesia is to infiltrate the surgical site with the anesthetic. This is effective for procedures in the superficial layers of the tissue; however less effective for procedures such as abdominal surgery which require anesthesia of the whole body wall. In these circumstances inverted L blocks and paravertebral blocks are preferred. Lidocaine 2% is the most commonly used anesthetic, but Mepivacaine 2% can also be used and has a longer lasting anesthesia. It is advised to not exceed 10 mg/kg body weight.

The most common drug used for sedation in ruminants is xylazine an α2 agonist. Other α2 agonists such as detomidine, medetomidine and romifidine are also used but to a lesser extent. These sedatives have analgesic as well as muscle relaxant properties, but an additional regional block is necessary to achieve full analgesic effect for a surgical procedure. When administering a α2 agonist, certain adverse effect can be noted depending on the dose of the sedation. Respiratory depression, hypercapnia, hypoxemia, bradycardia and increased urine production can be seen after administration of a α2 agonist in an animal. Therefore, extreme caution should be used in animals with preexisting cardiopulmonary disease, hypovolemia or urinary tract obstruction. Xylazine should also be avoided during the end of pregnancy in animals because xylazine has an oxytocin effect on the uterus and can cause abortions. Luckily the α2 agonist effects can be reversed; however, the analgesic effects will also be reversed at that time.

Other agents used for sedation, especially in young calves are the benzodiazepines such as diazepam and midazolam. They have mild sedative, muscle relaxant and anti-convulsive properties; however not any analgesic properties. Therefore, an additional regional block is important for the surgical procedure. The cardiopulmonary effects of benzodiazepines are minimal and transient. These sedatives are excellent to use in animals with cardiopulmonary disease or compromise.
**Injectable anesthesia**

Injectable anesthesia can be used for short-duration anesthesia and animals may be premedicated with drugs used for sedation in smaller doses. Ketamine is commonly used either by itself but more often in a combination with α2 agonists or benzodiazepines. The combination with a α2 agonist or benzodiazepine improves the muscle relaxation and sedation, plus facilitates the placement of an endotracheal tube if necessary. The choice of which specific combination of drugs to use depends on duration of anesthesia required for the procedure, the degree of the analgesia required and the physical condition of the animal. Adding a small dose of ketamine to a more traditional chemical restraint combination can greatly enhance the cooperation of an animal. This technique is also known as the “ketamine stun”.

Propofol can also be used to maintain injectable anesthesia. This drug is mainly used in calves and small ruminants for induction and maintenance of general anesthesia. This can be with a single dose (3-7mg/kg) for a 5-10-minute anesthesia or with a constant infusion to maintain a longer anesthesia (0.3-0.6 mg/kg/min). The advantage of propofol is that it is noncumulative and the recovery is rapid and smooth; however no analgesic effect is noted. Side effects that can be seen with propofol are respiratory and cardiovascular depression. Apneu is noted with rapid IV administration and hypoventilation, hypoxia and hypercapnea have been noted when anesthesia was maintained with propofol. Endotracheal intubation with supplemental oxygen is advisable when using propofol in injectable anesthesia conditions.

Guaifenesin is not an anesthetic but is a centrally acting muscle relaxant with minimal cardiovascular and respiratory depression. It is not used alone in ruminants, but is used in a combination with ketamine (a double drip) or in a solution of xylazine, ketamine and guaifenesin. The latter is also referred to as a triple drip used for a constant rate infusion. The guaifenesin solution should be a 5% solution to avoid hemolysis and tissue necrosis in case of a perivascular injection.

**Recovery period**

Recovery of ruminants from surgical procedures in the field is usually uneventful. The animal should recover in a comfortable, dry and warm setting and preferable under supervision until most of the anesthetic is worn off. The duration of recovery depends on the type of drug used and the amounts that were used of that particular drug. If a procedure was performed in dorsal or lateral recumbency, the animal should be placed in sternal recumbency to avoid ruminal tympany and regurgitation during the recovery period. If an endotracheal tube was placed, it can be left in place until the coughing and swallowing reflexes are present.

In conclusion, anesthesia of ruminants in the field is frequently performed for routine small surgical procedures and with low risk with good preparatory work. If, however a complicated surgical procedure or a high-risk animal is considered, inhalation anesthesia in a clinic environment is preferable.
Teat Injuries in Dairy Cattle
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Teat injuries are common in dairy cattle. The injuries can be divided into external and internal injuries. These lesions can have greatly interfere with milk production and in some cases can compromise the production of the quarter or the whole gland. Therefore, it is important to treat these type of injuries appropriately and promptly. Teat injuries can be challenging to address; however in bovine practice this is a valuable service to offer to your clients.

Diagnosis
Diagnosis of a teat injuries when there is an external laceration is easy; however, for an internal injury some additional diagnostics might be of use. A step by step examination protocol can involve one or all of the following:

- Visual examination of the teat and description of the lesion if visible.
- Palpation of the gland and the individual teats.
- Insertion of a teat cannula if an obstruction is suspected internally in the teat.
- Milk cultures and sensitivity
- Additional imaging modalities such as ultrasound examination, radiography with and without contrast, theloscopy.

Internal injuries of the teat
Atresia congenital or acquired
Atresia is the obstruction of a duct or orifice. This can be present at birth (congenital) or acquired after an infection or traumatic incident. It may be present at the end of a teat, but could potentially extend into the gland itself. Treatment can be simple by opening up the skin with a needle or blade at the tip of the teat or may be more involved such as thelotomy.

Fibrosis of furstenburg rosette
Lesions in this area of the streak canal will result in a reduced milk flow. Depending if the fibrosis is associated with a tight streak canal, mucosal default or a total obstruction of the rosette, the diagnosis and treatment can be more or less complicated and challenging.

Localized hemorrhage
Self injury to the distal portion of the teat can cause damage to the vascular plexus in the teat. This damage will result in a local hematoma that can obstruct the outflow of milk while the teat appears swollen, painful and bruised. Conservative anti-inflammatory treatment should be started immediately together with a temporary dry period. After the acute inflammatory phase, the teat should be evaluated again and further treatment options such as partial teat amputation discussed.

Fibrosis in the teat wall
Fibrosis of the teat wall can be caused by previous trauma to the wall or from infection of a chronic nature. The cistern of the lumen can become so narrow that it is almost nonexistent. In these cases, contrast radiography can be extremely helpful. Surgical treatment is in most cases the treatment of choice. This can range from teat theloscopy to a vertical thelotomy depending of the extent of the fibrosis.

External lacerations of the teat
External lacerations are classified according to their duration, localization, conformation and thickness of the injury. It is important to take some time to make this evaluation since the prognosis of the injury is depended on this.

All teat lacerations are considered contaminated, so therefore cleaning of the injury is important. Before surgical repair a dose of antibiotics and pain medication is also recommended. Repair of teat laceration can be challenging, but with appropriate material/instruments and a good knowledge of the anatomy, practitioners can be very successful at it. During this presentation, we will discuss more in detail some of the aspects to keep in mind with these type of injuries to the teat.
The external umbilical cord consists of one vein, two arteries and one urachus. The umbilical vein joins the left portal vein and carries oxygenated blood during fetal life. The umbilical arteries originate from the internal iliac arteries and carry deoxygenated blood to the placenta. The urachus connects the fetal bladder to the allantoic sac, which contains diluted urine and other excretory products.

At birth, elongation of the cord breaks the umbilical cord and a stalk of 6-10 cm remains outside of the abdomen. A calf delivered by c-section has a shorter umbilical cord which could become a cause for complications. The umbilical cord shrivels in 3-4 days and should be totally gone by 3-4 weeks.

The umbilical vein becomes the round ligament of the liver. The paired umbilical arteries form the lateral or round ligaments of the bladder. The urachus atrophies and leaves no identifiable structure in later life.

**Conditions affecting the umbilicus**

**Hemorrhage**
Hemorrhage from the umbilicus can occur immediately after birth. It occurs more commonly after the cord is cut and tied than if it were allowed to contract naturally due to stretching.

**Patent urachus**
This condition is not something that is often seen in cattle. There may be a congenital or acquired condition. Animals with the congenital condition are clinically normal and the urachus failed to close at birth. Animals with an acquired form of patent urachus, the urachus opens up after a few days or weeks of age. This may be due to illness or improper care of the umbilical cord after birth.

**Umbilical hernia**
This condition may also be congenital or acquired as a result of an umbilical infection. Most of the hernias are notices days or weeks after birth. It is the most congenital defect in cattle and in Holstein cattle it may be hereditary.

Uncomplicated umbilical hernias should be differentiated from an umbilical infection on the basis of clinical signs. The hernia most commonly contains the omentum, followed by the abomasum, rumen and small intestine. In longstanding cases adhesions may develop between structures in the hernia sac and the sac itself. Strangulations occur rarely in cattle due to the fact that the cavity is blocked off by the omentum. Bovine hernias < 5cm can often be treated conservatively. Multiple conservative treatment options will be discussed during the presentation. Bovine hernias > 5cm should be repaired surgically under sedation with a local anesthetic or general anesthesia. The open and closed technique of herniorrhaphy will be discussed more in detail during this presentation.

**Umbilical abscess**
This condition is frequently a sequel to an omphalitis in calves. Improper care of the umbilicus after birth, environmental contamination or failure of passive transfer are considered predisposing factors. Calves are less likely to develop septicemia. Calf usually had an enlarged cord after birth and slowly a swelling developed that is large, warm and may or may not be painful. Diagnostics such as ultrasound may help to determine the nature of the swelling and its contents. Treatment is to lance the abscess and flush the cavity. Many animals with a defect in the body wall and extension of the abscess into the abdomen will develop a hernia.

**Infected umbilical remnants**
The history in calves is usually of an infected, draining umbilicus starting at 1-2 weeks of age. An enlarged external mass may develop over the following weeks as well. It is important to assess the whole animal with these conditions since the affected calves are often unthrifty and have other problems such as diarrhea, fever pneumonia or joint infections. A good physical examination and ultrasonographic examination are key elements in the diagnostic work-up. An ultrasound exam in 2 planes (transverse and longitudinal) can help identify the structures of the umbilical cord that are affected.

Surgical resection of the infected umbilical remnants under general anesthesia is strongly recommended. In young calves it may be attempted under sedation with local anesthetic and rope restraint; however, I would not recommend this since some of these surgeries can be quite involved and take longer than 1 hour. The majority of the calves undergoing surgery will be female because of economic considerations. During surgery there will be a rich blood supply since you are operating on inflamed tissue. The approach for the different structures as well as the marsupialization of the umbilical vein will be discussed in more detail during this presentation.
This lecture will review the risks posed to developing or nursing calves by a number of xenobiotics, including plant toxins and mycotoxins.

**Review of the physiology of parturition and lactation**
- Maternal recognition of pregnancy
- Sources of progesterone during pregnancy
- Changes in maternal physiology during pregnancy
- Embryonic and fetal morphogenesis
- Cascade of endocrine events leading to parturition
- Physiology of lactogenesis and galactopoiesis in cattle

**What can go wrong?**
- Embryonic loss
- Teratogenesis
- Abortion
- Prolonged Gestation
- Stillbirth
- Dystocia
- Subfertility
- Neonatal Loss
- Dysgalactia/Agalactia

**Modern view of teratogenesis**
- There are CRITICAL PERIODS of exposure corresponding to when susceptible structures/functions develop.
- NOT ALL exposed animals are AFFECTED.
- ALL DEVELOPMENTAL PROCESSES CAN BE TARGETED!!!
- TERATOGENESIS IS MANIFESTED BY EMBRYONIC OR FETAL MORTALITY, ALTERATIONS IN BIRTH WEIGHT, MORPHOLOGIC ABNORMALITIES, and/or IMPAIRED FUNCTION.
- The DOSAGE of toxicant → TERATOGENESIS → Generally LOWER than the DOSAGES → MATERNAL DISEASE.
- TERATOGENESIS → INTERACTIONS BETWEEN THE TERATOGEN AND GENOTYPE.

THE PLACENTAL “BARRIER” IS NOT AN EXTREMELY EFFECTIVE BARRIER TO THE MATERNAL TRANSFER OF XENOBIOTICS FROM THE MATERMAL TO THE FETAL CIRCULATIONS!!!

**Some important considerations**
- MOST xenobiotics reach pharmacologic concentrations in the fetus after exposure of the mother
- Umbilical vein concentrations of a given xenobiotic are several times higher than maternal circulating concentrations.
- Intoxications from topically administered insecticides MIGHT be more likely during pregnancy, because of increased dermal absorption.
- During pregnancy the amount of antibiotics necessary to maintain antimicrobial drug concentrations MIGHT increase and the elimination ½ life might decrease.
- Weak bases are more likely to pass into the milk than weak acids.

**Guiding principles for the administration of therapeutic xenobiotics to pregnant/lactating females**
- NO medications are without potentially toxic effects, BUT the anticipated benefit of a therapeutic approach MUST outweigh its associated risks.
- HIGHER doses and/or CHRONIC administration of medications are MORE likely to be “TOXIC” than lower doses and/or one-time use.
- Medications should be administered during pregnancy ONLY in accordance with product LABEL INSTRUCTIONS AND PRECAUTIONS.
• IDEALLY, fetal viability/gestational health should be MONITORED over the course of a treatment regimen or, at least afterwards.
• REMEMBER CATTLE ARE FOOD ANIMALS!!

Specific examples of poisons & medications impacting pregnancy & lactation
• Plant Toxins
• Mycotoxins
• Metals
• Pesticides
• Antibiotics
• Other “Medications”

How to prove and prevent problems
• For diagnosis of abortions submit BOTH fetus and fetal membranes!!!
• Samples fixed in neutral-buffered formalin and fresh/frozen!!!
The prevention and management of “fescue toxicosis” and exposure to other sources of ergot alkaloids, such as ergotized grasses and small grains, continue to be ongoing challenges to bovine practitioners and cattle producers in certain parts of the U.S. This session will briefly review ergot alkaloid intoxication and provide updates on animal and forage management approaches which can be utilized to minimize production losses associated with endophyte-infected tall fescue and grasses and/or grains infected with “ergot”.

What is “fescue toxicosis”?  
- Associated with endophytic ergot (ergopeptine) alkaloids in tall fescue grass.  
- The endophytic fungus (Epichloë coenophiala) is not visible to the “naked eye”.  
- There is a symbiosis between the endophyte and tall fescue (Lolium arundinaceum).  
- Ergot alkaloids (specifically ergovaline) cause hypoprolactinemia and vasoconstriction  
- Dry gangrene (“fescue foot) is observed in winter (cold temperatures).  
- “Summer slump” is observed during the summer (hot temperatures)

What is “ergotism”?  
- Associated with fungal ergot (ergopeptine) alkaloids produced by Claviceps purpurea.  
- Claviceps purpurea infects the seed heads of many grasses and small grains (NOT corn).  
- Claviceps infection occurs most frequently following cool, wet springs.  
- The “ergot bodies” (sclerotia) of Claviceps species are visible to the “naked eye”.  
- Ergot sclerotia can contain very high concentrations of ergot (ergopeptine) alkaloids.  
- These ergot (ergopeptine) alkaloids cause hypoprolactinemia and vasoconstriction.  
- The signs of ergotism can be indistinguishable from those of “fescue toxicosis”.  
- However, ergotism is less dependent on ambient temperature and can be fatal.

Forage management strategies for “fescue toxicosis”  
- “Fescue toxicosis is likely a reality in the beef industry for the foreseeable future.  
- Toxic endophyte-infected tall fescue is not “ALL BAD” and can be managed.  
- Several agronomic practices can eliminate/reduce the effects of tall “fescue toxicosis”.  
- “Testing” tall fescue pastures can provide important information.  
- Renovation of toxic endophyte-infected tall fescue can eliminate the problem.  
- Over-seeding tall fescue fields with clovers can dilute the effects of endophytic toxins.  
- Avoidance of high nitrogen fertilization rates reduces the severity of “fescue toxicosis”.  
- Removal (mowing) of seed heads can reduce ergovaline content.  
- The forage ergovaline concentration can also be reduced by drying (production of hay).  
- Ammoniation of hay has also been shown to reduce forage ergovaline content.

Forage management strategies for “ergotism”  
- Observation/“testing” of pastures for “ergot” can provide important information.  
- Removal (mowing) of seed heads can reduce the number of aerial “ergot bodies”.  
- Mature “ergot bodies” can be physically removed during the production of hay.

Animal management strategies for “fescue toxicosis” and “ergotism”  
- Be aware of the problems!!!  
- Most management strategies focus on the management of “summer slump”.  
- Feeding supplements has been reported to be “helpful” in minimizing clinical effects.  
- Rotation of cattle to warm-season forages in summer and stockpiled tall fescue grass in the fall has been proven to be a helpful management strategy for “fescue toxicosis”.  
- Recent research shows that animal tolerance to “fescue toxicosis” is possible!!!  
- Using tolerant livestock where exposure to toxic endophyte-infected tall fescue is unavoidable might give innovative producers a competitive edge!!!  
- GENETIC TESTING/STRATEGIC BREEDING ARE LIKELY TO BE THE WAVE OF THE FUTURE!!!
CAN THESE NOVEL STRATEGIES FOR “FESCUE TOXICOSIS” BE APPLIED TO “ERGOTISM”?

References
Joint mobilizations
When doing joint mobilization, one bone of the joint is stabilized while the second is motioned in specific patterns with a specific amount of pressure. Two terms are commonly used when describing what is going on, arthrokinematic and osteokinematic motion. Osteokinematic motion describes the big picture of what the bones are doing (ie. flexion, extension, abduction, adduction) where as arthrokinematic motion is what the bones are doing at the joint and depends on which bone is stable and which is having pressure applied to it. If the convex bone is stable and the motion is from the bone with a concave surface, the arthrokinematic and osteokinematic motion is in the same direction. If the bone with the concave surface is stable and it is the bone with the convex surface the arthrokinematic and osteokinematic motion are going in different directions and so the force you are applying is applied in the opposite direction you want the bone you are motioning to go. The easiest way to remember this is “males go the wrong way”. An example of this would be glide at the hip, because you are motioning the bone with the convex surface to improve hip extension, the force applied to the great trochanter would be cranial (the “wrong way” from the direction you want the femur to move).

Joint mobilizations can be separated into roll, glide, and spin. Traction can also be used to stimulate inhibitory nociceptive receptors, diminish pain, and move the synovial fluid in the joint to enhance joint nutrition. The amount of pressure or traction applied and the amount of motion at the joint are other important aspects that we will review. We will discuss each of these individually.

A consideration has to be when will joint mobilization and soft tissue techniques be appropriate or inappropriate. The first step is to define “End Feel”. End feel is what happens when you reach an end of range of motion. For simplicities sake, we will only cover four of these. The first is “firm” which has some spring at the end and is secondary to stretch of tendon, ligament, and joint capsule. An example of this would be felt on extension of your metacarpo-phalangeal joint. The second is “soft” which is when muscle restricts further motion. An example would be flexion of the stifle of a well-muscled mastiff. It is the physical presence of muscle that is stopping the motion of flexion. The third is “bony”, which is when bone physically stops the joint motion. This can be normal as felt when extending your elbow, or secondary to osteophytes surrounding a joint. The last is “empty”, where there is no physical structure that stops motion, it is purely a pain response.

Maitland Joint Mobilization Grading Scale is based on amplitude of movement and where the force is applied within available ROM. Grade I is a small amplitude rhythmic oscillating movement at the beginning of range of movement that is used to manage pain and spasm. Grade II is a larger amplitude rhythmic oscillating movement within midrange of movement that is used to manage pain and spasm. Grade I and II are often used before and after treatment with grades III and IV. Grade III is a large amplitude rhythmic oscillating movement up to the point of limitation in the range of movement that is used to gain motion within the joint by stretching the joint capsule and connective tissue structures. Grade IV is a small amplitude rhythmic oscillating movement at very end range of movement that is used to gain motion within the joint when resistance limits movement in absence of pain. Grade V is a thrust technique or manipulation that is a small amplitude, quick thrust at end of range that is often accompanied by a popping sound. This technique would be considered a chiropractic motion and should not be performed without specific chiropractic training.

Indications for grade I and II joint mobilization are for pain and stiffness and can be done daily. Grades III and IV mobilizations are primarily used to increase motion at the joint. Stiff or hypo-mobile joints can be treated 1-3 times per week. This should be used in conjunction with active motion exercises. Do not treat hypermobile joint actions with actions that will stretch structures that increase instability, treatment options include improving normal motions if there are restrictions in a different direction and then strengthening the muscles, tendons, and ligaments that support that joint. An example would be carpal hyperextension, improving restricted flexion, while not stressing extension, will allow more normal ROM so the supporting structures can be strengthened without destabilizing the joint further.

Roll is the movement that occurs when equidistant points on the moving surface come into contact with equidistant points on the opposing joint surface. These motions would include flexion, extension, abduction, and adduction of the shoulder, elbow, carpus, hip, stifle, and tarsal joints. If this motion is uncomfortable, often doing other motions in a small oscillating fashion within the patients “comfort zone” (grade I &II mobilizations) allows roll to become pain free.

Glide is the movement that occurs when the same point on a moving surface contacts with a new point on the opposing joint surface. We use this often in ball and socket joints like the shoulder and hip, but also use it in the stifle, carpus, tarsus, and digits. When this motion is done, one bone stays stationary and the other is motioned. At the hip the pelvis is stationary and the femur is motioned. We look at the arthrokinematic motion and will follow the rule of thumb that “males go the wrong way”. The hip is held in a somewhat neutral to flexed position. The femur is held approximately parallel to the cranial border of the wing of the ilium and gently oscillated 15-20 times. Pressure is applied in a cranio-dorsal, cranial, and cranio-ventral motion. This is not three different
directions, but a continuum of motion. With the shoulder, the humerus can be stabilized and the scapula mobilized or visa-versa. If the humerus is stabilized it is the concave or female joint surface that is being motioned. The action that is usually restricted is extension of the shoulder. The scapula, female or concave joint surface, would be motioned cranially (the “right” way for the direction you want the bone to move for correct osteokinematic motion). We can perform the exact same joint surface motion by stabilizing the scapula and mobilizing the humerus, but the motion would be caudally. As with the hip, there is a continuum of motion in a semicircle of direction to find the joint capsule restriction and correct it.

At the carpal joint, stretching the joint capsule of the inter-carpal and carpo-metacarpal joints aids in improving restricted carpal flexion and diminishing pain at the joint. By placing caudal pressure on the radial carpal bone and ulna carpal bone, the female surface of the inter-carpal joint, we can increase carpal flexion if the joint capsule is part of the cause of the restriction. We can also oscillate the 2nd through 4th carpal bones caudally, the female surface of the carpal-metacarpal joint, to further enhance carpal flexion. A glide of the accessory carpal bone can be performed by placing the joint in a neutral position and motioning the accessory carpal bone medially, laterally, proximo-lateral to disto-medial and back, and proximo-medial to disto-lateral and back.

Glide can also be used in the stifle as long as there is not compromised stability (i.e. injured cranial cruciate ligament). In this instance the female surface is the tibia. The femur is stabilized the motion of the tibia would be cranially (the “right” way for the joint) to increase extension and caudally to increase flexion. The most common time I work with the stifle is when there is loss of extension after cruciate rupture and surgery so this technique would not be performed until the patient is completely healed from the surgery (usually 8-12 weeks).

Another, less commonly thought of place to use glide in the canine patient, but one that is extremely helpful is at the radio-ulna-interosseous joint. By stabilizing the ulna and motioning the radial head at a 60 degree angle caudo-lateral to cranio-medial, with a component of proximal to distal, flexion, extension, pronation, and supination can be improved as well as comfort.

When gliding digits for either restricted flexion or extension, the proximal bone is stabilized and the distal bone is motioned. As the convex and concave surfaces are present but minimal, I will often move the distal bone cranially, caudally, medially, and laterally. This usually improves motion, but must be done very gently.

Another joint mobilization technique is spin. Spin is the motion of a point on the moving bone surface creating an arc of a circle on the stationary bone surface as the moving bone spins. This motion is demonstrated by internal and external rotation of the limb at the hip and shoulder joint, but can also be used in the digits. Spin can be visualized best with a ball and socket joints. If the proximal bone is stationary and the distal bone rotates around the center axis the joint capsule is stretched evenly, which is different then some of the other techniques we have discussed. Spin can be applied with or without traction. Spin and Traction are the two most important joint mobilizations to treat pain and are frequently used to treat the joint before the other motions are implemented.

Traction is extremely useful for painful or arthritic joints. The direction of traction is extremely important in some of the canine joints. For instance, if the elbow was held in a straight position and traction was applied by pulling on the radius and ulna, an excessive force is placed on the anconeal process. If the same joint is placed at a 90 degree angle and the radius and ulna are pulled down the line of the humerus, the joint capsule can be stretched effectively to decrease discomfort at the elbow without any abnormal forces. The hock should also be stretched at a 90 degree angle with the pressure following the line of the tibia to stretch the tibi-talus joint. Traction can be applied to the toes both in a straight motion or applied in a circular manner stretching the dorsal surface of the joint. Straight traction is beneficial for dogs that have sensitivity or pain at the toe joints. Toe traction can be taught to some owners to be used daily, especially before walks. Straight traction of the toe joints involves placing one finger or thumb behind the pad and another dorsally and applying gentle traction straight out from the foot. Circular traction involves the same hand position but the dorsal joint surface is stretched more by placing downward pressure with the finger above the toe causing the toe to “curl” around your finger. This is beneficial for dogs that have restricted flexion at the digits. For carpal traction, one hand is placed above the carpus to stabilize the limb and the other hand is below the carpus applying gentle traction. Many dogs have discomfort at the carpal joints as well as restricted flexion due to fibrous tissue. Traction not only relieves discomfort, but can be one of the methods used to remove the fibrous tissue. The hip, shoulder, and stifle are placed in a neutral position, as though the dog were standing, when traction is applied.

Contraindications for joint mobilization include: active inflammatory arthritis, malignancy, ligamentous rupture, herniated disks with nerve compression, bone fracture, some congenital bone deformities, joint effusion (may use type I & II mobilizations to relieve pain), bacterial infection, sutures over the area, cellulitis, fever, hemATOMA, open wound at the site, hyperesthesia, and constant severe pain.

Precautions include: osteoarthritis, pregnancy, severe scoliosis, poor general health, hypermobility, and anticoagulation therapy or the patient’s inability to relax. Grade III and IV mobs are contraindicated in artificial joints.

Treating a patient
When approaching a patient we have to assess each joint before treating, even if we have seen this patient recently, as its circumstances can change from day to day. By taking the joint through range of motion in normal active directions as well as in other directions (ALWAYS while in the comfort zone of the patient) we are stretching the joint capsule, mixing the joint fluid to enhance
cartilage nutrition, and stimulating conscious proprioceptive and nociceptive (pain) receptors in the joint capsule and surrounding fibrous structures (ligaments and tendons).

Starting with the toes (front and rear are treated the same), each digit is flexed and extended. If a restriction is found, traction and/or spin of the joint that is restricted are often enough to break up the fibrous tissue and improve or normalize motion. If pain is found, straight traction will often relieve the discomfort, allowing the examination of flexion and extension. Metacarpals and metatarsals can be motioned by stabilizing one bone at the distal aspect and motioning the adjacent one dorsal and ventral. If there is a restriction, stabilizing one and mobilizing the adjacent one will break up the fibrous tissue allowing normal motion. Loss of flexion at the carpus is a common finding. Mobilization of the accessory carpal bone, cross friction massage to remove fibrous tissue over the restriction, stabilizing one and mobilizing the adjacent one will break up the fibrous tissue allowing normal motion. Loss of flexion at this joint. Another technique utilized more for pain can be done by stabilizing above the carpus with one hand and grasping the metacarpals with the other, compressing the joint, and while compression is maintained, motioning the joint so that the tips of the toes make large circles and there is minimal motion at the joint itself. If the major part of the motion is at the joint, and not the toes, the joint will have a “grinding” effect that can be painful and not beneficial for the joint. When done correctly, this can relieve pain and enhance joint nutrition as well as aid in proprioception.

Loss of elbow flexion is often seen with fibrous tissue buildup at the medial joint surface, enthesopathy. Traction with the joint at a 90 degree angle, cross friction massage directly over the fibrous tissue, traction with your fingers as a fulcrum creating a long lever technique to stretch the caudal aspect of the joint, and grade III joint mobilization at the radial ulnar intersosseus joint can all relieve pain and enhance joint motion. True shoulder extension, when the scapula is stabilized, can be restricted by the joint capsule (which can be mobilized as described previously) or by excessive tightening, spasms, or knots/trigger points in the triceps or deltoid muscles. Manual therapy techniques can be utilized for the muscle issue to increase mobility. Once true shoulder joint restrictions have been addressed, the musculature around the scapula needs to be evaluated. With the dogs back straight and the head at a 90 degree angle, the forelimb should be able to be brought forward to touch the medial canthus of the eye. When the limb is stretched back, the forelimb toes should be able to come to the stifle when the rear limb is in normal standing position. If this is not possible there are multiple muscles that can be restricting this motion. We will review how to assess each muscle noting that the stretch for each muscle as well as the manual therapies described above can be used to enhance flexibility, allowing for more normal motion. Straight abduction with the limb in neutral evaluates/stretches the pectoral muscle. With the shoulder and elbow flexed and the limb externally rotated, the subscapularis muscle is evaluated/stretched. With the elbow and shoulder in extension, the limb is internally and externally rotated evaluating the caudal and cranial trapezius muscle respectively. If the forelimb is brought back and the scapula rotates on the body wall but the elbow does not extend fully, the biceps brachii muscle is the restricting muscle. These motions are repeated after each manual therapy until all motions move fluidly and without pain.

Flexion of the hock can be affected by multiple factors. Although most of the motion is at the tibio-talus joint, mobilizing the other joints can affect the overall flexion and health of the joint. Stabilizing the tarsal bones and supinating and pronating the foot motions the tarsometatarsal joints. Stabilizing the tarsal bones and “wiggling” the calcaneus bone evaluates and stretches the talus-calcaneal joint and capsule. Mobilizing the tarsal bones with cranial to caudal and caudal to cranial motions as done with the carpus (intermetatarsal joint capsule stretch) are all indirect ways to affect the joint. One way to affect the tibio-tarsal joint directly is to stabilize the tibia with the hock at a 90 degree angle and to mobilize the talus by holding the limb below the hock and oscillating it cranially 15-20 times and then caudally 15 to 20 times. The other method, a Mulligan technique, stabilizes the tibia with the tarsus in a neutral position, internally rotates the foot below the tarsus, and flexes of the tarsus repeatedly feeling tension with each oscillation. After about 15-20 oscillations, the tarsus should be fully extended and then fully flexed and then flexion can be re-evaluated. Soft tissue manual therapy techniques can be used if there is excessive tightening of the gastrocnemius, superficial digital flexor, or deep digital flexor muscles. Loss of flexion at the stifle can be affected by fibrous tissue restricting patellar glide. If this is the case, it can be detected and corrected by placing the stifle in full extension and mobilizing the patella medial to lateral, proximo-medial to disto-lateral, and proximo-lateral to disto-medial. Loss of flexion can also be caused by muscle problems in the quadriceps femoris or sartorius muscles. Manual therapy techniques can be used to restore motion if this is the case. It is very common to have muscle issues cause loss of extension of the hip. The sartorius, rectus femoris of the quadriceps, pectineus, and tensor fascia latae muscles are the most likely muscles, but the iliopsoas muscle can also play a role. The iliopsoas muscle is evaluated by extending the hip and internally rotating the limb. If this is uncomfortable, the iliopsoas is the culprit. The pectineus is palpated by abducting the limb. If this is restricted, manual therapy can be used to improve first abduction and then indirectly, hip extension. If the muscles are normal, the joint capsule is the most likely problem. A glide can be done by placing the femur parallel to the wing of the ilium and oscillating the greater trochanter toward the ilium from the caudal to cranial. Often the direction of the restriction can be felt and corrected by moving the oscillating contact point up and around the greater trochanter. Hip extension should then be improved and pain resolved. If hip extension is still painful, slight abduction and rotation of the limb (internally and externally) creates a spin that can relieve pain. Extension should be re-evaluated after this has been done several times. The next position to evaluate is to see if there is a restriction when the rear limb toes are brought to the elbow. The lumbar spine must remain in a straight position when this is evaluated or you
can put abnormal stress on the lumbar spine and not be stretching any of the muscles you want to evaluate and treat. The muscles that can be restricting this motion may be one of the hip extensors (biceps femoris, semimembranosus, semitendinosus, or gracilis muscle), the middle gluteal muscle, or the iliocostalis muscle. Each needs to be palpated during the stretch to be evaluated. Manual therapies can be utilized to restore motion if there is a muscle problem. The ilium may also have restricted motion on the sacrum affecting the toes reaching the elbow. The ilium can be evaluated and treated by motioning it cranial-caudal along the line of the ilium, clockwise, and counter-clockwise. When rotating the ilium it must be understood that there is a 20 degree angle between the sacrum and the ilium and if you do not take this into account you can cause more restrictions then are currently present.
The musculoskeletal exam
A thorough musculoskeletal exam begins with a thorough history and a general physical exam to identify or rule out physical ailments that may be mistaken for a musculoskeletal condition. In the history it is important to identify the duration of the problem, the progression (is it getting better, worse, or staying the same), what the patient can do (i.e. climbing stairs, jumping or climbing on the couch and/or bed), and what the patient does each day (walks- how frequent and how long, crated vs. free reign of the house, how often are they trained if they are a working dog…).

Digits
The forelimb toes are examined by assessing the digits for bony or soft tissue swelling, previous fractures, or pain on palpation, extension or flexion. The second and the fifth digit should flex to the same position while the third and fourth digit should flex to the same position, and all should be able to flex to be perpendicular to the line of the metacarpal or metatarsal bones. The most common condition seen at the digits is osteoarthritis with secondary restricted joint motion, flexion or extension, with or without pain.

Metacarpo-phalangeal joints
The metacarpo-phalangeal joints may hyperextend if the animal chronically pulls their weight forward, has chronically long nails, or if there is a metabolic ailment causing a generalized ligament laxity. The 2nd and 7th sesamoid bones are the most likely to be fractured. Clinical findings include thickening of the second or seventh sesamoid that correspond to the second or fifth digit. Surgical removal frequently destabilizes the digit and so support and strengthening are the treatment of choice. These joints will be chronically thick, but can become pain free and may have full range of motion with rehabilitation. Restricted flexion of the metacarpo-phalangeal joints can also be seen with osteoarthritis and immune mediated polyarthropathy.

Carpus
The majority of carpal joints issues commonly seen are loss of flexion secondary to chronic hyperextension and acute traumatic hyperextension injuries commonly seen from jumping off of heights. Hyperextension can be seen when standing or by assessing extension manually, making sure the elbow in extension when evaluating the carpus. Flexor carpi ulnaris tendon injuries may include strains, core lesions (significant thickening), calcification from chronic repetitive injuries, complete disruption or avulsion. Grade 1 strains are tender with out palpable thickening; Grade 2 strains are thickened, tender acutely on palpation and tender on stretch when chronic. Grade 3 strains are a full rupture and will need surgery.  With the carpus in flexion the edges of the carpal bones should be easily palpated. If not, either there is synovitis, thickening of the joint capsule with a firm, thick texture, or effusion with a fluctuant texture.  It is also common in carpal injuries to have edema in the extensor tendon peritendinous sheath. Diagnosis is achieved when tubular structures running parallel to the antebrachium are palpated across the joint.

Elbow
Elbow Dysplasia (ED) is, by definition, abnormal formation of the elbow joint and encompasses 3 three etiologies including fragmented coronoid process (FCP), ununited anconeal process (UAP), and osteochondrosis (OC). FCP of the ulna has a genetic component with the most common breeds affected being Labrador Retrievers (18-50%), Bernese Mountain Dogs, and Rottweilers. FCP occurs bilaterally in 50-90% of dogs where one side is affected and can be seen with OC in 60% of cases. Dogs usually present with discomfort at either 4-12 months of age or later in life secondary to osteoarthritis.  When dogs have clinical signs at a young age they typically include a lameness aggravated by activity where the dog shifts weight off of the more severely affected limb and may hold the limb in external rotation.  

There may be muscle atrophy in the forelimb and hypertrophy in the rear secondary to a caudal weight shift. Effusion, which would be found on the lateral aspect of the joint, is rarely present. Pain can usually be elicited in these cases by supinating the antebrachium and flexing and extending the elbow. Another common finding is pain on deep palpation over the insertion of the biceps brachii tendon at the medial aspect of the ulna. Chronic signs include fibrous thickening of the medial aspect of the elbow, crepitus, and loss of both flexion and extension of the elbow. Even when pain is resolved, a mechanical lameness may persist due to the inability to fully extend the elbow. Clinical signs for FCP and OC are identical and x-rays of the elbow are not sensitive to identify FCP, therefore, if these clinical signs are present and the owner wants to pursue surgical treatment, arthroscopy +/- CT or MRI are the diagnostic tools of choice. In a study comparing arthroscopy with conservative treatment (NSAIDs and exercises), where objective gait analysis was used to evaluate outcomes, at 4 and 8 weeks the dogs that had surgery had significantly worse limb function.
compared to the rehab group. At 6 and 12 months there was no significant difference between groups suggesting that a rehabilitation approach is superior to a surgical approach in these cases.

UAP is not as common as the other causes of ED and is only found in large breed dogs, as small breed dogs do not have a separate center of ossification of the anconeal process. Clinical signs include lameness between 5-20 months of age with palpable crepitus on flexion and extension of the joint and effusion at the lateral joint surface.

Traumatic fragmented medial coronoid process (TFMC) or “jump down syndrome” is the most common elbow injury seen in performance, working, or highly active dogs. This may be caused by repetitive subchondral micro-fractures from abnormal jumping or a repetitive pull of the distal biceps tendon where it inserts on coronoid process causing it to medially rotate into the radius causing micro-damage to the subchondral trabecular bone. Another theory is that these dogs are predisposed due to an incongruity in the elbow where the ulna is longer then the radius. This would cause more pressure at the coronoid-trochlear articulation, leading to fracture of this process. OA is inevitable as there is a continuum of cartilage softening, fibrillation, fissures, erosions, and subchondral cracks. Just as with FCP, there is an intermittent lameness exacerbated by exercise, but it is uncommon to be bilateral. The lameness progresses and is not responsive to NSAIDs and rest. Patients can be painful on direct pressure over the coronoid process, resist full or hyper-flexion of the elbow, are painful on carpal flexion with external rotation while extending the elbow (this puts load on the medial compartment), and/or have joint effusion at the lateral joint surface.

Shoulder
Trigger points and excessive muscle tightening can be identified in the biceps brachii, triceps and deltoid muscles. Trigger points are commonly found in the triceps muscles, especially in working dogs, and can be palpated as either bands of tight tissue or round hard areas in the long or lateral head. The biceps muscle is palpated while in a shortened position as well as during stretch. The muscle should be taunt on full stretch. If it is taunt before getting to a full stretch it indicates an abnormality in the tissue. To stretch the biceps brachii the shoulder is flexed and then the elbow is extended. The forelimb toes should touch the stifle if the rear limb is in a neutral standing position. If the scapula rocks back on the body wall, but the elbow does not extend, the biceps brachii muscle is affected. If the elbow extends all the way but the scapula does not rotate on the body wall, then the problem may be in the pectoral, brachiocephalicus, trapezius, rhomboids, or subscapularis muscles. When the pectoral muscle causes the restriction the limb will not be able to abduct easily. If the dog picks up its head each time the limb is rotated caudally, the brachiocephalicus muscle is affected. Locking the shoulder joint in extension and internally and externally rotating the forelimb will stretch the trapezius muscle, valuating it. The scapula should “rock off the body wall” at its cranial and caudal border. Tight bands in the trapezius of muscle will hold it down on the body. These bands can be palpated, stretched, massaged, or treated with laser to allow normal motion. The rhomboideus muscle is evaluated by moving the scapula dorsal and ventral on the body wall. Rhomboideus muscle tension can be eliminated with a quick stretch in the direction of the greatest restriction. The subscapularis muscle is evaluated by flexing the shoulder and externally rotating the forelimb, assessing if the distal aspect of the scapula can rock off the body wall. Massage, stretching, and laser therapy can bring this muscle back to normal length improving “Reach”. The most common shoulder lameness causes include the supraspinatus tendinopathy, biceps brachii tendinopathy, medial shoulder syndrome (MSS), OC of the humeral head, and infraspinatus fibrotic contracture. Supraspinatus tendinopathy is considered an overuse condition due to the loss of elasticity with discontinuous and disorganized tendon fibers and no active inflammation. These fibers, on diagnostic ultrasound, appear hypoechoic and are termed “a core lesion”. Mineralization may be observed on radiographs, but it cannot be differentiated from mineralization of the biceps brachii tendon. Supraspinatus muscle atrophy can be present if the condition is chronic. Lameness is usually a drop in the shoulder during the stance phase of gait, is exacerbated with exercise, and is non-responsive to NSAIDs. The tendon can be thickened on palpation and pain may be elicited on palpation of the tendon as the shoulder is flexed. Biceps tendinopathy may be primary, secondary to impingement from the supraspinatus or referred elbow pain from the muscle pulling at the point of insertion. This is also a repetitive, overstitch strain injury that can lead to degeneration of the tendon. Common clinical signs associated with biceps tendinopathy are trouble jumping, inability to “wrap”, or make tight turns, trouble with two-on-two-off contacts, and knocking bars with the forelimbs. Lameness may be intermittent subtle to severe and is exacerbated with exercise. Palpation of the biceps tendon usually elicits pain and the tendon itself is thickened. The biceps muscle belly is usually thickened, lobular, and unable to stretch; shoulder flexion with elbow extension.

If the shoulder flexes, the elbow extends, and the forelimb toes are able to be brought up above the dorsal surface of the back, a bicepital tendon tear or superglenoid tubercle avulsion fracture is diagnosed.

Medial Shoulder Syndrome (MSS), a cause of lameness especially in working dogs is defined by either a partial labral or capsule tear, ligament disruption, or subscapularis tendinopathy. This is considered an overuse or repetitive use injury and as such the lameness may be intermittent to constant, and exacerbated by exercise. To assess for MSS the shoulder is locked in extension, the scapula is stabilized, and the forelimb is abducted. If pain, spasm, or increased lameness after this test is performed, then MSS is likely.
Osteochondrosis (OC) of the shoulder is found on the caudal aspect of the humeral head in medium, large and most commonly giant breed dogs. Clinical signs typically appear between 4 and 8 months of age. Radiographs are typically diagnostic.

Infraspinatus fibrotic contracture is most commonly seen in hunting and working dogs and may be traced to an earlier short-lived lameness. Typical clinical signs include shoulder abduction with restricted extension, antebrachium external rotation, a mild lameness with the limb advancing via circumduction of the limb and the paw flipping into extension before striking the ground.

**Tarsus and musculature**

The tarsal joint is evaluated for joint effusion and soft tissue swelling. If joint effusion is present at the cranial surface, acute trauma, tick born, and autoimmune diseases should be considered. Testing for crepitus is most sensitive when pressure is applied between the talus and the tibia and full flexion and extension is performed.

Superficial digital flexor (SDF) luxation is a condition where the tendon luxates, usually laterally, off of the calcaneal bone causing lameness. Clinical signs may include swelling over the calcaneus and a “pop” as the tendon luxates upon flexion of the tarsus. This is most common in young Shelties and there may be a concurrent bursitis associated with it.

Damage to the common calcaneal tendon is usually traumatic in nature and can be a partial or full tear. The SDF is intact with a tear of the gastrocnemius when the tarsus is close, but not touching, the ground and the toes are in constant flexion. If the gastrocnemius has a partial tear at the origin there will be heat and a palpable hematoma or ball of fibrous tissue depending on chronicity. A full tear of the common calcaneal tendon will result in a plantigrade stance.

Hyperextension of the tarsus is found with dogs with chronic stifle disease or primary tarsal disease. The clinical appearance of the patient is a dog that stands with the pelvis tucked, the hips and stifles in flexion, and the tarsal joints in extension to hyperextension. During the stance phase of the stride the tarsal joint may extend past 180 degrees. This leads to lumbar pain in conjunction with the limb joint pain (tarsus, stifle, +/- hip).

**Stifle and musculature**

The patella is evaluated for medial and lateral luxation on extension of the stifle. Effusion at the stifle can create a temporary luxation, therefore if it is present when luxation is diagnosed, this should be controlled and the stifle reassessed. Medial patella luxation is more prominent than lateral in both small and large breed dogs, is bilateral in 50% of dogs, and is more prevalent in females then males. To establish luxation with the least amount of pressure the tibia is rotated externally when evaluating for medial luxation, and internally when evaluating for lateral luxation. Clinical signs of patella luxation include intermittent skipping and having the dog trying to replace the patella by extending the limb out behind it. There are four grades to patella luxation: In Grade I the patella is in the groove, can be manually luxated, but reduces as soon a pressure is removed; in grade II the patella moves in and out of the groove (out during flexion and in during extension); in grade III the patella is luxated most of the time but can be manually reduced; in grade IV the patella is outside of the groove and can not be manually reduced.

Anterior drawer and cranial tibial thrust are tests to identify CCR. In an anterior drawer assessment the proximal hand stabilizes the femur with the thumb and middle finger behind the femoral condyles and the pointer finger on the patella. The distal hand is placed with the thumb and middle finger on the caudal surface of the tibia with the pointer finger on the tibial crest. The proximal hand stabilizes the femur while the distal hand places the stifle at an approximate 120 degree angle and attempts to displace the tibia cranially. This is repeated with the stifle in flexion to assess for a partial CCR. If motion is found the cruciate may be ruptured. If there is a minimal amount of motion it should be compared to the opposite limb.

Cranial tibial thrust is performed with the proximal hand stabilizing the femur with the thumb and middle finger behind the femoral condyles. The pointer finger barely touches or is a hair width away from the tibial crest, the distal hand cups the tarsus in the palm and while keeping the stifle at the same angle, flexes the tarsus. If the tibial crest moves cranially into the pointer finger, the cranial cruciate ligament is compromised. The most common errors made during this assessment are allowing the stifle angle to change while assessing the joint and pressing/holding the tibial crest in place with the proximal hand pointer finger. This motion should be assessed in multiple positions. This test is more comfortable for the patient with a CCR then anterior drawer.

**Hip and musculature**

Quadriceps contracture is characterized by scarring of the muscle causing shortening and an inability to flex the stifle. It can be seen after a distal femoral fracture in a young dog, especially if the limb is immobilized in extension with a cast or splint. Fibrosis of the quadriceps may take place in as little as 5-7 days. Other causes of quadriceps contracture include Neospora and severe muscle trauma.

Sartorius and tensor fascia lata muscle trigger points should be assessed with the hip in extension as these are hip flexors. Lack of flexibility in these muscles can prevent full hip extension in the absence of hip joint disease. Once restricted hip extension secondary to muscle involvement is ruled out, pain on motion of the hip, sacro-iliac (SI) joint, and lumbosacral joint can be assessed. To isolate each of these areas, first the tuber ishii is stabilized while the hip is extended, isolating the hip joint. Pressure is maintained on the femur as the stabilizing hand moves from the tuber ishii to the sacrum and the femur is further caudally rotated, isolating the SI joint.
Pressure is maintained on the femur again as the stabilizing hand moves from the sacrum to L7 and the femur is further caudally rotated, isolating the lumbosacral joint.

The hip can be evaluated for luxation with two special tests. The first is performed by placing a finger between the greater trochanter and the tuber ishii and externally rotating the limb. In the normal hip the finger is displaced by the greater trochanter. The second test looks at the shape of the triangle formed by the most dorsal aspect of the tuber ishii, greater trochanter, and wing of the ilium. Presenting clinical signs of hip luxation typically include a dog with a non-weight bearing lameness with external rotation and adduction of the limb. Frequently there is a firm palpable swelling above the hip joint; this is the displaced greater trochanter. The cranial dorsal movement of the greater trochanter is secondary to the pull of the iliopsoas and the gluteal muscle groups that attach to the lesser and greater trochanter respectively. Hip luxation is common and usually secondary to an automobile accident though may be chronic and secondary to severe hip dysplasia.

A sensitive test to assess hip laxity can be performed by having the dog laying in lateral recumbency, lightly placing the center of the palm of the proximal hand over the most dorsal aspect of the greater trochanter, stabilizing the stifle and preventing limb abduction with the distal hand, and creating a long lever lateral force. The top of the greater trochanter should not move into the palm of the hand, if it does, there is laxity. Laxity is to be expected in puppies under 4 months of age and old lean dogs where there is not enough muscle to hold the femur in place. Laxity in any other dog is considered abnormal. With exercise, this laxity may disappear.

Evaluation of the deep and middle gluteal muscle is performed by assessing for consistency in shape from the wing of the ilium to the greater trochanter. It may be convex or concave, but not a combination, as this would indicate a trigger point.

The Ortolani test is another test to check for hip laxity. This test is performed by placing the limb in adduction, applying pressure dorsally, and then slowly abducting the limb. A positive test is if the head of the femur pops back into the acetabulum on abduction. This test is only valid for dogs under 6 months of age and the research shows that if the test is positive there is only a 50% chance of dysplasia on OFA radiographs at 2 years of age. 50% of dogs that have a positive Ortolani sign at 6 months of age have no dysplasia via OFA radiographs at 2 years of age. This is a test that should be done under sedation or anesthesia, as it can be very uncomfortable.

The pectineus muscle is just cranial to the adductor muscle and can be palpated as a tight band on the medial thigh when the rear limb is abducted and should soften with adduction. This is one of the major hip stabilizer muscles, along with the gluteal muscles, and when over worked, can have trigger points (palpated as peas in a pod) or be very tight. In some animals it may spasm and cause pain with ambulation.

Contracture of the semimembranosus, semitendinosus muscles, and/or gracilis muscles, aka Fibrotic Myopathy is commonly seen in German Shepherd Dogs that are competitive athletes or working dogs, especially police dogs and dogs trained in Schutzhund. This is a disease where micro tears are evident in the muscles leading to scar tissue formation and the inability of the muscle to stretch fully. This can be a crippling disease. Diagnosis is by not being able to stretch these muscles and having them have a fibrous feel, even when not in a stretched position.
Fun with Assistive Devices: What You Need to Know
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Assistive devices can be extremely beneficial
Independent locomotion is critically important in dogs. In fact, companion animals that have lost the ability to walk independently are at risk of being euthanized. Many geriatric patients are at greater risk and can benefit from assistive devices. Some can be as simple as boots that help prevent slipping on a home’s tile floors, others can be as complex as carts for dogs that can no longer use two or even four limbs optimally or prosthetics to allow arthritic dogs to ambulate after a partial amputation. Assistive devices may decrease complications present in recumbent patients including decubitus ulcers and urine scald. Assistive devices can give back independence for dogs that have lost the endurance, strength, or neurological capacity to ambulate fully on their own. These devices can be used to motivate dogs that are weak to allow them to build the strength to once again walk, run, or even jump again. Some devices are designed to prevent pressure sores or allow them to heal. They can also be an aid to pet owners to decrease risk to their bodies by allowing proper body position while assisting their pets. Some devices can be easily purchased by clients on the internet, but some require significantly more training and expertise. In order to succeed with prosthetic and orthotic devices, accurate assessment and a comprehensive knowledge of rehabilitation, orthopedics, biomechanics, and prosthetics is required.

Carts – pros of individual carts
Walkin’ Wheels make carts in two sizes, below 20 pounds and 20-180 pounds. They are adjustable and can be used in the clinic for many sized dogs. They are lightweight, allow the dog to use the rear limbs, and can be adjusted adjust to almost any dog. They have the ability to be counter balanced to allow the cart to take the weight off of the front limbs if they become weak as in a dog with degenerative myelopathy. Eddie’s Wheels is custom made to the individual pet and very specific measurements must be made and sent in to the company. They are able to make carts for dogs with problems utilizing the forelimbs, rear limbs, or all four limbs. They also have a cart that can be used over a land treadmill to support the dog’s weight while they use their limbs to ambulate. Doggon Wheels are also custom built carts that can be utilized for dogs with problems utilizing the forelimbs, rear limbs, or all four limbs. They have a neoprene saddle to decrease the chance of abdominal skin burn from rubbing. Dewey’s carts are less expensive custom made carts that also have a neoprene saddle for the dog to sit in. K-9 Carts were one of the first companies to produce carts for dogs. The saddle the dog sits in is a foam covered metal support. Evan’s Mobility Carts are made of PVC pipe and have different types of wheels. Some people even home make their own carts out of various materials due to the cost of the custom built carts. There are several sites on the internet that show how to make carts for the mechanically inclined. Many companies allow you to purchase used carts and then have them retrofitted to a new patient.

Harnesses
The Help-em-up harness is a great tool to have in the clinic as well as to send home with patients. There is a front and a rear piece, each with a handle, and each can be used independently or they can be connected together. It is easy to put on and take off and usually clients can easily adjust the straps for a good fit. If the patient is a male and the under piece affects the prepuce, there is a male adaptor piece that can be purchased that displaces the abdominal support to a different location for patient comfort. The Solv-it is less expensive, less physical material, and harder to figure out. It can be a good alternative when finances are an issue and the client is willing to work a bit harder at fitting and dressing the dog. The Ginger lead has a sling like base that attaches to the collar to aid in staying in place. As in all sling like devices this may put pressure on the prepuce, unintentionally express the bladder, and if too much upward pressure is applied, lifting the dog, places the spine in extension at the cranial border and flexion at the caudal border. The walk-a-bout sling design allows the rear limbs to go through the neoprene device, lifting the dog by the pelvic floor rather then by a section of the spine. This is a better option then a sling for dogs with IVDD or who have weak bladders that are easily expressed when a sling or towel is used to support weight. Bottom’s Up devices wrap around the rear limbs applying slight lateral pull slightly abducting the rear limbs. This may be beneficial for neurological patients that cross over in the rear limbs. In The Company of Dogs catalog there is a harness that has a handle in the thoraco-lumbar region allowing the handler to lift from the middle of the dog. This is beneficial if both fore and rear limbs are affected. There are also many homemade slings and harnesses that can be found on the internet with instructions on how to create your own.

Other aids
Stair lifts are available for sale, made by the people that build them for people, but they may be cost prohibitive.

Show foot is a spray that creates a slightly sticky surface when sprayed on the pads of the feet preventing or diminishing slipping. This usually has to be applied every 3-4 days or if the dog is in a frequently wet environment, more often. Rubberized socks and boots
can also help prevent skipping on slippery surfaces. Yoga mats can be laid down in slippery areas or, if bought by the role, can cover an entire floor surface. Long nails increase the chance of slipping as well as damage the tendons and ligaments in feet leading to an inability to flex the toes and grip surfaces. If nails are trimmed and there is still an issue with sliding of the feet on slick surfaces, Dr Buzyby’s Toe Grips may reduce sliding.

If the patient is small, unable to use the rear limbs, creating drag sores, and there is no hope that function will return, Drag Bags may prove helpful. This should not be utilized if there is hope of return of any function, as it will stifle it. There are many types of boots, each situation needs to be evaluated for the best product for that patient. Some are heavy but take a long time to wear through, some are light weight and thin so the patient can easily pick up the foot, some are breathable to decrease moisture buildup and dermatitis, and some have significant traction to aid in slipping or to allow the patient to ambulate over rough terrain.

If lateral sliding of the fore limbs, medial shoulder instability, hygroma, or ulceration of the elbows are an issue, Dog Legges products may be beneficial. They cover and cushion the elbow and can be connected ventrally to prevent abduction of the forelimbs which is imperative post medial shoulder instability surgery. Human or Doggy diapers may be used as a bellyband or to go around the back end. If human diapers are used for fecal incontinence then a tail hole must be added. If urine scald is present, there are multiple ointments available over the counter. Zinc oxide ointments (diaper creams), thought toxic in large doses, can be applied sparingly for quick results.

Orthoses may be used to prevent hyper-extension of carpal, tarsal, or stifle joints, add length to a short limb, or add dynamic pressure to a restricted joint. They can be made with a hinge to enhance functionality. Dorsal tarsal flexion assist devices can cue timing for muscle contraction by applying a variable tension that stimulates flexion of the tarsus upon initial swing phase of the stride. These can be attached from the most distal aspect of the foot to the proximal tarsus or to the harness. Using a cord with rubber bands is a quick and inexpensive way to determine if these devices will be beneficial. This is not a long term solution as they can stretch the digital flexor muscle and tendons and create discomfort if left on too long.

Air splints are utilized to maintain one or multiple joints in a stable and continuous position to allow a weight bearing stance when the dog would otherwise be unable to prevent buckling of the limb. Elevated feeders are beneficial to dogs that have cervical pain, weakness, or neurological conditions that make lowering the head to the floor difficult or uncomfortable. Stairs or ramps can allow dogs that are weak or unable to jump to get on furniture independently. Compression wraps and Thunder shirts can be beneficial for geriatric patients with cognitive dysfunction syndrome that become anxious when left alone or in new situations. An inexpensive alternative that sometimes works is using an Ace bandage in a cross-over pattern. This design is a Tellington Touch method that can have great results in some dogs.

Cooling mats, ones that contain water to conduct heat away from the dogs’ body can help dogs that are routinely hot and want to lay on the cold floor, but are stiff upon rising. Fans on the floor, especially in front of the air conditioning vent, allow dogs to cool off when they want and walk away if they get too cold. Bedding can also warm dogs that seek heat and create a soft comfortable place to relax. Some beds have a porous surface for those pets that are urinary incontinent. In these cases pee pads can be placed under the cot to absorb any urine that leaks during slumber.

References
Working dogs can be injured in many ways. Understanding what was injured, how it heals, and how to strengthen it to prevent re-injury, is extremely important. Sprains, strains, and tears are common injuries that once the healing begins, can be re-injured unless the damaged tissue can heal and then be strengthened fully before the canine patient is allowed to go back to a full work load. A strain, by definition, is damage to a ligament secondary to a stretch or tear of the ligament without an associated bone fracture or joint dislocation. Studies on ligaments show that at one year post injury the tensile strength is only about 80% of its original strength. Therefore when a ligament is injured, the rate of exercise intensity is much slower then if a muscle or tendon is injured. A muscle heals faster then a tendon as it has a significantly higher blood supply. A muscle strain, or muscle pull, is damage to a muscle or its attaching tendon, usually as a result of a strenuous activity. A strain can be accomplished during sports or during a course of normal daily living activities. As most working dogs are high drive, even going out to run in the back yard can be strenuous. A severe muscle strain can result in a muscle tear. The tearing of the muscle can also damage small blood vessels, causing local bleeding (with or without bruising) and pain (caused by irritation of the nerve endings in the area). Strains are graded on degree of damage to the muscle. A Grade 1 strain is mild damage (less then 5% of the fibers damaged) where there is pain at the muscle on palpation but no gross lesions are found. There is minimal loss of strength and motion. These injuries generally take about 2-3 weeks to heal. A Grade 2 strain is more extensive with more muscle fibers disrupted and a significant loss of strength and motion. These injuries may require 2-3 months to heal and come back to normal function. A Grade 3 strain is a complete rupture of the muscle or tendon. These may present with a palpable defect in the muscle or tendon, acutely a hematoma, or an area of thick fibrous tissue. Caught early, these may be surgically corrected.

The body’s reaction to an injury is to lay down fibrous tissue, first in an unorganized fashion, and then, if proper stretch and blood flow are available, to organize into a stronger bonded area. Depending on the degree of injury, this area may never have the strength of normal healthy tissue, but by building up the strength of the tissue around it, we can decrease the chance of the injured area being stressed to the point of re-injury.

There are changes that can be seen in muscle tissue with exercise. These changes take 3-4 weeks to start to be observed. In contrast, it takes 3-4 months to start to strengthen ligaments. One time when we see injury is when the dog starts to gain strength in the muscles, after the first four weeks of exercise, and before the twelve week period, when the tendons and ligaments gain strength to catch up to the muscles. We commonly see carpal hyperextension injuries or ligament stretch injuries during this time.

Type I muscle fibers are fibers that are considered slow twitch, oxidative (aerobic) fibers that are slow to contract. They are used in low intensity exercises such as light resistance work, used for muscular endurance and long duration aerobic activities like long runs (for example - field work). They have a slow contraction time, but are quite resistant to fatigue. Type I muscle fibers have a high mitochondria and capillary density. To strengthen Type I muscle fibers endurance, aerobic, exercises are performed. Endurance or aerobic exercises, over time, have local and systemic effects. At a local level, endurance exercises strengthen the Type I muscle fibers, increase fiber capillary density, increase oxidative enzymes in the muscle fibers, and increased lactate threshold in the muscle (intensity of exercise where lactic acid starts to accumulate). Systemically, endurance exercises improve cardiovascular and neurological efficiency. Adaptations include: lower resting heart rate, increased interventricular septal thickness and heart weight, stronger connections between neurons with enhanced firing frequency and spinal reflexes, improved VO2 max (maximal O2 consumption, the point at which O2 consumption remains the same even when workload is increased). Exercise is considered endurance work if the dog is trotted for at least 20 minutes or swims continuously for 5 minutes. The ideal gait for endurance ambulating is a trot (diagonal limbs move together), as this works the limbs evenly. A soft, forgiving surface (dirt, wood chips, or a rubberized track) should be used to decrease the chance of repetitive motion joint injuries. Land treadmills can be used as long as the length of the tread is at least 2.5 times the length of the dog. As the treadmill requires less muscle activity then running on land, treadmills should not be the only endurance exercise for athletes that require significant endurance in their sport (mushing, field work, and herding). Swimming can be done in a pool or lake with the dog staying in the water to retrieve or swim along with the owner. The underwater treadmill is a cross between the two with added buoyancy, but significantly more resistance then trotting in air. Even for dogs whose job it is to do short bursts of activity (utilizing Type II muscle fibers), it has been shown that endurance exercise makes them more competitive and less likely to be injured. When these muscles are the predominant muscle fibers we see small circumference high tone body musculature, much like we would see in a marathon runner. This is in comparison to when Type II muscle fibers are the predominant strengthened fibers where the body appearance shows significant hypertrophy with increased muscle circumference, as seen in body builders. It has been shown that no matter what the sport, a degree of cross training improves performance and decreases the chance of injury.
Type II fibers are fast twitch, high force, high power, high speed contractions with low endurance. The energy used by these fibers is anaerobic or glycolytic. With exercise, the local effect is an increase in glycogen storage in the muscle fibers to allow increased glycolysis. These exercises can be performed by having the dog perform repetitions of an exercise or by doing an endurance exercise with resistance of an external force. Exercises that can be done as repetitions for strengthening can be grouped by body part.

Examples of strengthening exercises for the trunk and neck include: Sit Up And Beg, Snoopies, Roll Over, Crawling, Balance Beam Or Blocks, Wobble Board, Backing Up on an Incline and then Decline. Exercises to strengthen the forelimbs include: high 5’s (straight and with abduction and adduction), play bow, digging, swimming, backwards crawling, and low tugging. Exercises that specifically strengthen the rear limbs include: Dance; Ball Work with the forelimbs on the ball, walking the ball forwards, backwards, and sidestepping around it; Beg-Stand-Beg; Sit-to-Stand on a Hill (facing left right and up); high tugging, and jumping. Examples of resistance training would be accelerating uphill against gravity, wearing an appropriate harness and pulling a cart or weights, moving with resistance (in water or against therapy bands), and braking against momentum (acceleration/deceleration exercise). The purpose of this training is to gradually and progressively overload muscle tissue to improve strength, often observed as improved muscle tone and hypertrophy.

Balance and proprioception exercises also decrease the chance of injury by allowing the athlete to barely clear a bar and land correctly as they are twisting for the next obstacle, wrap around a pole, and take off using just enough expenditure of energy to accomplish their goal. Proprioception, knowing where the body is in space, comes from information provided by both the sensory neurons in the inner ear, and the stretch receptors in the muscles and ligaments that support the joints. Connections between neurons can be strengthened, and the number of synapses increased, improving proprioception and balance to decrease the chance of injury. Examples of exercises that work on proprioception and balance include: walking over poles laid out in different directions and at different heights; using a wobble board; and having the dog walk over a ladder. This type of work will allow the dog to be able to perform complex tasks for longer periods of time without fatigue.

Skill training is training of the specific acts the dog will need to perform to compete in their sport. This may be long or high jumps, weaving, pulling a cart, running over a dog walk or A-frame, jumping off a wall, balancing on small surfaces or any other activity needed for their sport. It is important to remember that if a dog is injured or away from their sport for a significant length of time that they will need time to work back to their competitive level in their skill, not just in endurance and strength. Even if they remember how to do it, they will need to practice it multiple times before they compete so they can do it with agility, not with reckless abandon.

Rest and recovery is also essential to preventing injury. Warming up the muscles decreases the chance of injury\(^7\) and improves performance.\(^8\) Proprioception\(^9\) and flexibility.\(^10\) The warm up should mirror the activity that the dog will be performing in their sport. Perrier\(^11\) showed that active stretching was superior to passive stretching to improve athletic performance in sports that involved flexibility and jumping. For agility dogs, start with trotting, juiking (abrupt side to side motions), tugging, bows, spins in both directions, and beg or beg-stand-beg to prepare for using the trunk for balance and the rear limbs for propulsion.\(^12\) Cool down should consist of a trot, followed by a walk, to aid the body in preventing lactic acid build up in the muscles. This should be followed by a massage and passive stretching to also decrease lactic acid build up in the muscle fibers.

Overtraining in humans has been shown to increase the chance of injury, reduce resistance to infection, decrease performance, and increase depression.\(^13,14\) This has also been shown to affect performance horses. The variables we examine to classify proper training vs. over training include intensity, volume, and frequency. Intensity is the amount of work required to perform a task. Volume refers to the repetitions per set and the number of sets given in a specific workout. Frequency refers to how many training sessions are done in a given time period. To prevent overtraining, if we significantly increase one of these variables we need to decrease one or both of the others. If these variables are not kept balanced the results can be chronic soreness, general lethargy, illness, or acute trauma such as avulsion fractures.\(^15\) One common formula to prevent over training is to have one variable high, one medium, and one low.

Another recommendation is to alternate the level of the three variables, give the dog one day a week off, and one month a year off. Specific injuries can be grouped into muscle and tendon injuries or ligament injuries. Some muscle injuries rules of thumb include: gently stretch a chronic repetitive injury where fibrous tissue has been laid down and needs to become organized, do not stretch an acute injury as you may reinjure the tissue or prevent stabilization of the tissue, and do not stretch a hyperextension injury. Exercises can be broken down into concentric, isometric, and eccentric. Isometric exercises are those that the muscle is firing, but the fibers are not changing in length. These are the safest exercises post injury and should be what a patient is started with. Examples of isometric exercises include standing/weight bearing, rhythmic stabilization, standing with all four or front feet on a ball, sitting on a hill (facing up, left, and right), diagonal leg lifts (Snoopy), rocker and wobble board. Eccentric exercises are those that the muscle lengthens as it fires. These exercises build muscle the fastest, but are also the most likely to cause injury and should therefore be the final exercise utilized post injury. Concentric exercises are when the muscle shortens as it is firing. These would be the intermediary exercises. The force of gravity is the largest force on the body, therefore our muscles are used to fight gravity, pushing up off the ground. Muscles lengthen and shorten constantly, but when an added force (gravity, weight bearing, or resistance) is added, there is more energy/work needed to perform that movement. To determine which exercise to choose for each patient it is important to know...
what the muscle that is injured does. Examples of common muscle that are injured, with concentric and eccentric exercises for each
one, are listed below.

- Flexor carpi ulnaris
  - Concentric – up hill walking, stairs, digging, swimming, pulling a cart, bike…
  - Eccentric – down hill walking, walking backwards, tug of war

- Triceps
  - Concentric – walking, digging, stairs, swimming, pulling a cart…
  - Eccentric - down hill walking, walking backwards, tug of war

- Biceps brachii
  - Concentric – walking up a hill, hi 5’s, swimming
  - Eccentric – walking down a hill, cavaletti poles

- Supraspinatus
  - Concentric – walking up a hill, hi 5’s, swimming
  - Eccentric - walking down a hill, cavaletti poles

- Infraspinatus
  - Concentric – walking up a hill, swimming, crawling, cavaletti poles
  - Eccentric – walking backwards

- Subscapularis
  - Concentric – sidesteps to the ipsilateral side, medial leg lifts
  - Eccentric – sidesteps to the contralateral side, weave poles

- Gracilis
  - Concentric – sidesteps to the ipsilateral side, sit to stand
  - Eccentric – backwards walking

- Sartorius
  - Concentric – backwards walking, swimming,
  - Eccentric – walking, stairs, sit to stand

- Longissimus and transversospinalis
  - Concentric – sit up and beg, beg-stand-beg, crawling, stairs, weave poles, spin and twirl
  - Eccentric – stepping from one small surface to another, crawling

References
Soft tissue manual therapy utilizes stretching and massage of the muscles, tendons, ligaments, and joint capsule to relieve pain and enhance range of motion and flexibility. Soft tissue manual therapy can be used to eliminate or diminish trigger points, fibrous tissue, or edema in the surrounding structures of the joint, and relieve excessive muscle tightening. Fibrous tissue is produced by the body in an attempt to stabilize the joint when it senses joint instability, but it causes secondary restriction of motion in the joint. By reducing motion at the joint, the muscles stabilizing and moving that joint can not contract or stretch fully. By limiting the amount the muscle can stretch and extend the muscle can shorten, and strength is reduced. Tendons and ligaments can be strengthened or become weaker, but strengthening them is a significantly slower process then strengthening muscle. Loss of strength of the muscles, tendons, and ligaments lead to more instability. Pain can be associated with joint instability as there are nociceptive pain fibers in the joint capsule and surrounding structures. Weakness and pain, leading to inactivity and atrophy, can perpetuate a cycle of restriction and instability. By breaking down the fibrous tissue, reducing trigger points and tendon sheath or joint capsule edema, pain is diminished, often immediately, and the contractility of the muscle is restored allowing the patient to begin to increase muscle mass and strength. The stronger muscles, tendons, and ligaments improve joint stability, slowing down the progression of secondary degenerative joint disease. Due to less pain and secondary joint disease the patient often experiences improved quality of life.

This lecture will discuss muscle techniques or massage. Massage has many techniques and forms, we will discuss some science and some techniques so you can have a better understanding of what is going on in the tissue, what you can and can not alter, and what kind of results to expect. There are many forms of massage or manual therapy and each form of massage has its own techniques. There is not one right technique, many techniques may be beneficial. They are like tools in the toolbox, the more you learn the better able you are to treat the patient in front of you.

Massage comes from the Arabic word mass which means “to press.” In the human medical field massage is considered standard therapy for pain management and decreased mobility as well as preventative therapy for competitive athletes. Chiropractors, Physical Therapists, and Doctors of Osteopathy frequently incorporate massage into their work.

Massage has an effect on multiple tissues: skin, fascia, muscle, tendon, fibrous/scar tissue, joint capsule, lymphatic and vascular circulatory vessels and, in some cases, periosteum. The main type of tissue in all of these structures that we will be working with is collagen. Type I collagen are long crimped fibers that resist axial tension, gives support, resists tension and behaves following the stress-strain curve. It is found in the dermis and fascia. Most of the effect massage has on collagen is in the toe region of the stress-strain curve (The very beginning of the stress-strain curve, just where the crimps are being straightened, and way before there is damage to the fibers.)

Tissue has the properties of viscosity and elasticity. Together these properties form the viscoelastic property that accounts for the fact that the response of the tissue to loading depends on how quickly the load is applied or removed. The faster the loading and unloading, the stiffer the tissue behaves. Rapid loading and unloading creates friction, energy that dissipates as heat when the tissue returns to its original length. It is important to understand this as massage can affect the stiffness of the tissue. If there is a low constant pressure or repetitive load applied over a long period of time, there is “creep” or elongation of the tissue. Massage should be able, with low loads and some repetition, to create a non-permanent “creep,” which is what we are looking for. Rapid loading creates the undesirable effect of tissue stiffness.

The pressure of the massage therapist increases pressure in the tissue that causes pressure gradients in the tissue, lymph and blood vessels. During the massage fluid is pushed from the vessels in to the tissue, and from the tissue into the vessels, at many depths and in multiple directions, allowing significant fluid interchanges. This fluid exchange creates a flushing effect in the tissue bringing it more nutrients. Human studies have shown that chemical irritants (Substance P and prostaglandin) decrease the pain threshold by sensitizing the nerve endings. Flushing these substances can thereby reduce this effect, allowing a higher threshold (less chronic pain). Massage in young animals resulted in reduction of stress as shown by a reduction in the output of adrenocorticotropic hormone. Resistance to infection later in life can be beneficially influenced by cutaneous stimulation at a young age. It has also been shown that young animals that receive massage learn faster and have more advanced neural development then non-handled animals. Changes in blood constituents following massage provides us with some of the information of the biochemical changes happening in the tissue as a result of massage. In a human study it was found that post massage the pain threshold was significantly elevated suggesting that massage can be used to treat pain and soreness.

Techniques

Effleurage is a technique where the massage therapist employs unbroken long or short, slow rhythmic, light or heavy pressure in a straight or circular manner with the finger tips, thumbs, knuckles, or palms of the hands, conforming to the surface of the underlying...
structures. This technique is used at the beginning of the massage to evaluate the tissue and at the end of the massage to help flush lactic acid from the tissue. It stretches the muscle fibers when the stroke runs parallel to the fibers and diminishes adhesions when the stroke runs perpendicular to the muscle fibers. One benefit of effleurage is an increase in circulation and lymph flow.

**Pettrissage** is a technique of kneading, pressing, wrenching, lifting, and squeezing with the intent of pulling the tissue away from the body to improve blood and lymph circulation, tissue elasticity, and to relieve tension. It increases circulation, mechanically relaxes muscles, reduces stiffness, loosens adhesions, releases analgesic chemicals into the body, and stimulates the nervous system. Strokes can be longitudinal (with the muscle fibers), cross fiber (perpendicular to the muscle fibers), diagonal or circular.

**Friction** is a technique that is used to increase circulation, break up fibrous tissue leading to reorganization of collagen, and it provides pain relief via stimulating inhibitory nocioceptor receptors. It is used to treat tendonitis, release trigger points, and to aid in stretching and broadening muscles.

**Tapotement** - is a technique of percussion that can be fingertip tapping or coupage. Finger tip tapping is used to stimulate muscles to fire when done for a short time or it can be used to create relaxation or muscle fatigue with a longer treatment time.

**Vibration** can be a very small amount of motion in a specific muscle or it can be as large a motion as rocking of the whole animal. It increases circulation, stimulates muscle spindles causing tiny contractions that lead to relaxation of the muscle, relieves pain, releases trigger points, and relaxes myofascial tissue. One of the ways vibration is used in soft tissue manual therapy is shortening and lengthening a tight muscle in a rhythmic motion by moving it side to side. It can also be used in diagnosis as if a muscle is tight due to the animal being stressed, vibrating the limb will immediately relax it. If the muscle is tight due to over or abnormal use, vibration will not cause immediate relaxation.

**Ischemic Compression** is a technique applying 1-2 pounds of steady pressure (8-12 seconds) to the center of the trigger point, in effect squeezing out the blood from the tissue in an area of local hyperactivity. When pressure is released, blood flows back into the tissue stimulating the golgi tendon organs, frequently relieving the trigger point or area of spasm. It is also felt that this technique reduces overall muscle tension and promotes healing.

**Strain Counter-strain** is a technique utilized to stretch a tight muscle. The muscle is placed into full stretch, in order to do this you need to know action of the muscle as the stretch is the exact opposite motion. In a muscle that crosses more then one joint, the stretch should affect the part of the muscle that is the most tight. Once the muscle is stretched and held for 15-20 seconds, full shortening of the muscle is performed, not just allowing it to go into neutral position, and then re-stretch the muscle while massaging the tightest point of the muscle. This process may need to be repeated up to four times to relieve the muscle tightness. With these techniques and precisely timed stretching and shortening of the muscles, ROM and flexibility can be improved, pain can be relieved, and the animal’s quality of life can be greatly enhanced. Once comfortable with these techniques, some can be taught to your “hands on” clients so they can perform them at home as well. Discrimment is important in these instances.

**Myofascial Release** is a technique where the fascia is addressed. It releases tension in the fascia that is there secondary to trauma, posture, or inflammation. Fascia surrounds the muscles, bones, nerves, and organs of the body. When there is restriction of this tissue there can be pain and restricted motion. Freeing this tissue relieves pain and allows normal motion. This technique involves applying gentle sustained pressure into the Myofascial connective tissue restrictions to eliminate pain and restore motion. It is a low load (gentle pressure) applied slowly into the tissue to allow a viscoelastic medium (fascia) to elongate. By putting pressure into the tissue and then applying directional pressure, in the area of greatest restriction, the fascia stretches and the flexibility of the muscle may be returned to normal.

**Patient evaluation**

The first step to evaluating a patient or pet is to know what is normal. By finding a young healthy dog and looking at an anatomy picture you can get a feel for the direction of the muscle fibers, the topography of the muscles, and a feel for what the muscles should feel like. You will feel the muscle issue the most when the muscle is in stretch. The most common muscles that I find issues in are the:

- Triceps - caudal aspect of the humerus; stretched by flexing the elbow and then extending the shoulder.
- Biceps brachii - cranial aspect of the humerus; stretched by flexing the shoulder and then extending the elbow.
- Gracilis - most medial muscle of the inner thigh; stretched by extending the stifle and then flexing the hip (abduction can be added).
- Sartorius - most cranial muscle of the thigh; stretched by extending the hip.
- Tensor facia lata - directly caudal to the sartorius; stretched by extending the hip.
- Pectineus - medial and adjacent to the femur; stretched by abducting the rear limb.
- Gluteal muscles - Proximal and cranial to the greater trochanter; stretched by flexing the hip and adducting the rear limb.
Favorite massage techniques

- T-Touch circles and long dorsal midline strokes on the face.
- Thumb over thumb of the neck, in the neck triangle.
- Big hand circle petrissage down the spine with alternating hands.
- Gentle traction of the tail.
- Longitudinal strokes down the back, the limbs and on either side of the scapula spine.
- Cross fiber/perpendicular massage of the limbs and either side of the spine (apply even pressure to both sides at the same time).
- Rolling down the limbs.
- Fingers together, circles over the long thin muscles.
- Traction and medial and lateral circles at the toes (just the top of the foot if they are hypersensitive).
- Distal to Proximal effleurage flushing of the limb as the final act of touch for that limb.
- Skin rolling and cross fiber (crabs) of the body wall skin.
- Circles over the gluteal muscles.

References
Working dogs can be thought of in several different categories. Protection or aggressive working dogs, service and guide dogs, hunting and field dogs, bomb, search and rescue and detection dogs, and herding dogs. They all have their own “language” and inherent traits. We will look at behavior, injury, and common modalities for each group.

Potentially aggressive dogs
Protection dogs, Police K-9s, military dogs, or dogs that you may have difficulty handling may have issues because of being taught not to take food from anyone other then their handler, taught to bite if someone besides the handler approaches them, may either have a vast vocabulary (police dog or war dog) or none (junk yard guard dog). Your choice with these dogs is either to let the handler do any close work with them or to build a rapport with them. Before you choose the latter, make sure you trust the handler. A handler that doesn’t know their dog well or has trouble controlling their dog can put you into a dangerous situation with these dogs. An example of how to work with one of these untrustworthy dogs is a dog that I worked with that was a protection dog that was owned by two police officers that had not established dominance, or a healthy relationship, with their 7 year old intact male German Shepherd Dog. The way I was able to work with this dog was that “dad” would come in leather gloves, stand behind and to the side of the dog with his leg pushed into his neck, and hold onto the dogs pinch collar with both hands. “Mom” would hold a pillow next to the dog’s head on the opposite side as dad, and I would come from behind and only be able to work from the upper thoracic spine back. We were able to do laser, acupuncture, chiropractic therapy, and massage. This therapy regime allowed the dog to regain strength and stamina to pace the property at home each night for two years. When the dog was not able to rise independently he was euthanized because he would not allow assistance in rising from the owners.

If possible become involved with the police dogs in your area before they initiate or complete “bite” training. If they meet you and know they can get love and cookies from you before they start their bite training, working with them when they are injured is significantly easier. Injuries in these dogs are usually acute trauma when they are younger and of spinal nature when they are older. Putting a maintenance program together to keep them in shape allows them to stay on the force until they are 11 or 12 years old versus 8 or 9 years of age without it. Modalities that can be done with the highly aggressive dogs should be hands off as much as possible. This is less of a problem if you have been working with the dogs from a young age. When there is an acute injury, cryo-therapy, laser and Assisi are some of the safest modalities. Ice or moldable gel packs can be given to a handler and if necessary a “demo dog” can be used to show the handler how and where to apply the pack. There are wraps or devices that can hold gel packs in place. CanineIceer.com has devices to hold ice receptacles for multiple joints so the handler does not need to be holding the ice on the dog for any length of time.

Laser therapy can often be done with little or no contact to the patient. If contact is recommended, an increased power can be used to make up for the increase in reflection secondary to holding the probe off of the skin surface. Be careful if you are used to touching the surface that you do not hold the angle of the probe at greater then a 30 degree angle as this is the tipping point for significantly increasing reflection and thereby significantly decreasing penetration even into skin level tissue. Laser improves blood flow and can decrease pain and inflammation by decreasing Cox-2, IL-1, and TNF-α.

An Assisi ring, targeted Pulsed Electromagnetic Field Therapy (tPEMF) can be used to increase blood flow (angiogenesis) and decrease pain. The mechanism for decreasing pain is by increased Ca+/Calmodulin binding leading to increased short bursts of eNOS.

If Neuromuscular electrical stimulation (e-stim) is indicated, always do this before active exercises as it prepares the muscles for stronger contractions. When performing e-stim on aggressive dogs having them stand over a peanut ball if they are able to stand. Applying the pads to muscles of the rear limbs and paraspinal muscles offers a better contraction while they are standing. Most handlers that own a protection or other aggressive dog prefer that their dog not be shaved if possible. If there is a short coat (Rottweiler, Pitbull or Doberman) then don’t shave. If Ultrasound or e-stim is indicated and there is long hair, shave, but not down to the skin. Use a blade that will cut the hair to less then 1 cm in length and use enough gel to allow transmission of the electrical or sound energy through the remaining coat.

Instead of passively stretching these animals it is often easier to have them do active stretching if possible. In cases of Fibrotic Myopathy, this is not possible, as you need to be stretching the affected musculature as you laser or ultrasound the tissue.

The underwater treadmill is a great tool for strengthening these dogs and it can also be used to somewhat “tire them out,” to take the edge off, before working with them. If the dog is trained in another language, it is worth your while to learn certain words to be able to communicate what you would like them to do.
Service and guide dogs
The most common issue seen with service and guide dogs is a propensity to become overweight. Frequently these dogs have restricted exercise as their “person” frequently does not live an active lifestyle. Introducing these dogs to a land treadmill, teaching them and their handler how to use it, and helping the client pick out and purchase an incline treadmill for “at home” use is often all that is needed. Sometime diet restrictions or diet changes may also necessary. Back issues are the most common issue I see with these patients. Laser and manual work (massage, chiropractic, or other manual therapies) are frequently done at the clinic and an Assisi loop is sent home for when there is pain and the client can not come in right away.

Hunting and field dogs
Hunting dogs and field dogs commonly have the opposite living situation as the therapy dogs. Instead of sleeping in the bedroom with their people, they sleep outside in a run. If they are not the most valuable dogs in the pack, you are not likely to see them. The dogs that are highly prized are dogs are frequently brought to the surgeon or rehab facility and left there until they can be returned to work. These dogs need to be brought to full endurance and skill training before being returned to their handler or they may be put in a position to be re-injured. This may mean they have to work up to being at a fast trot on the incline treadmill for one to one and one half hours as they may go in the field for 3-6 hours of intermittent hard running when they return home or to the trainer’s facility. These dogs frequently do not understand typical “commands” and yet may have high drive personalities. Hunting and field dogs usually do not like to lie down or be still. Ways to help calm these dogs to allow you to work with them include: applying essential oils (lavender, Peace and Calming…), flower essences like Rescue Remedy (ingested or, if you can not get it in the mouth, then applied topically into the ear), a T-Touch Wrap (an Ace Bandage wrapped from the breast plate, over the back, around the chest, and back up over the lumbar spine tied in a knot, creating a compression “suit” to sedate the nervous system), or a Thunder Shirt (same principle as a T-Touch wrap). Laser is a better tool for the common muscle and tendon injuries then ultrasound as there is less likely hood of heating bone or damaging tissue if they are attempting to struggle as you treat them. These dogs are easiest to treat when they are tired, hungry, or distracted. Toys that they only see at your clinic may distract them, especially if they have an oral fixation (Labrador Retrievers and Field Golden Retrievers). These toys should be machine washable and washed between patients unless the handler brings the dog’s own toy. The owner/handler can skip feeding them at home and bring their meal for you, the owner, or an assistant to feed as you work with them. This food or treats can be frozen in a mug or “Kong”, fed piece by piece if it is a kibble diet, used as a distraction, used to mesmerize the dog as each piece is offered, or held in a hand, to be licked at through a hole made in the fist by the thumb and pointer finger. If you have healed the patient and are now working on strength rebuilding, then exercising for endurance (running, swimming, underwater treadmill) should be done before the manual or modality treatment. This will tire them our both mentally and physically allowing you to have a calmer working environment.

When a dog has had a muscle, tendon, or ligament injury we will treat them with laser until there is no pain, not only after the treatment, but also when they come in for their next visit. At this point we start introducing exercising back into their schedule. Laser has been shown to decrease IL-1, COX-2, and TNF-a, three important inflammatory mediators. By decreasing these mediators after exercise we can speed healing by preventing the damage that would ensue after their release. It also allows us to see them at their most tired, both from a medical prospective (are they lame after exercise?) and a treatment prospective (they will lay down and rest because they are tired, allowing us to do what needs to be done to help them). Manual therapy is usually performed after the laser therapy as this can be done on a standing dog, though frequently the dog will relax and laydown while this is being done. Strength and skill training should be done after manual therapy, when their body is “aligned” and the nervous system is functioning optimally. Active exercises are best with these patients. We will frequently use a peanut butter mug to guide or drive the dog through the strength training exercises. These mugs have peanut butter spread evenly on the inside surface and have been frozen. This creates a continuous positive reinforcement as well as attention getting device.

Jumping, when reinitiated into the training program, should be done on a leash if the dog does not have a superior recall. This is a time when the dog feels “free” and may hold onto the “free” feeling and take off, potentially doing activity to re-injure itself. Beside laser, the Assisi unit is another modality frequently used by my sporting clients as they can purchase them and have them in their emergency kit to use in the field. Anytime they feel heat or see swelling, this can be applied for 15-30 minutes up to four times a day.

Bomb, search and rescue, and detection dogs
Bomb dogs can are usually trained so that they are only fed when they make a “find,” and then only a small portion of their daily ration. Though they are mostly finding explosive materials that have been hidden by their handler, in their mind they are doing their job 10-20 times a day. That being said, since these are the rules, food cannot be used as a distraction or a lure. These dogs do usually have a large vocabulary and respond very well to “sit” and “down, stay” making treating them with modalities easy. As this population of dogs are not high drive and are not asked to do crazy or fast, agile exploits, they are much less likely to have acute injuries. They are more likely to have back pain on examination. Laser and manual therapies are most likely the treatment of choice for these patients.

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**Herding dogs**

Herding dogs are a cross between the aggressive dogs, only in that they are high drive and may or may not have an “off” switch, and the bomb and detection dogs, as they don’t have to perform sharp turns and huge jumps. This population of dogs is more likely to have chronic repetitive type injuries. Spinal, carpal and hip pain are common with the occasional stifle or elbow injury observed. The best words I learned to communicate with these dogs are “that’ll do.” That is universal for “you have played enough, now settle down.” The other thing that works well for these dogs is the frozen peanut butter mug trick mentioned above. Manual therapy and laser are frequently the modalities of choice for these dogs. If there has been a surgery or significant injury requiring more then 2-3 weeks of restricted activity, then underwater treadmill and exercises to bring them back to full strength, endurance, and skill training is a must. These dogs will injure themselves and frequently not even show it until they are done working or done working, slept, and then will get up lame.

Different types of dogs with different mindsets, body builds, and work ethics have been bred for different jobs. Understanding what they do, what they can and can’t do, how they do or do not communicate, where their common injuries lie, and what modalities may work best for them should aid you in your journey into working with the working dogs.
An adverse drug reaction (ADR) is an unfavorable and unintended effect that occurs after use of a medicinal product. Although awareness of some potential adverse drug reactions in veterinary medicine is widespread, others may not be promptly recognized by practitioners, either because they are rarely observed, because they are associated with newer pharmaceutical products and have not become entrenched in the literature, or because they mimic the clinical signs of other common diseases in dogs and cats. In human medicine, unrecognized adverse drug reactions may result in unnecessary patient morbidity and healthcare expense, and the term “prescribing cascade” has been coined to describe the situation of an unrecognized or misinterpreted adverse drug reaction resulting in the prescription of additional drugs to address the clinical signs.

Old age, chronic disease, and use of multiple drugs place a patient at risk for adverse drug reactions and a prescribing cascade in human medicine, and polypharmacy among elderly or critically ill hospitalized pets may create a similar scenario. Veterinary practitioners can avoid a prescribing cascade by increasing their awareness of potential adverse drug reactions (e.g. through periodic review of veterinary literature and perusal of package inserts for new products carried in their hospitals), and by considering that the cause of any new clinical sign in a pet receiving other medications could be a consequence of drug treatment. Examples of several veterinary adverse drug reactions that may initially be unrecognized for various reasons (some with the potential to trigger a prescribing cascade) are discussed below.

1. Phenobarbital and bone marrow toxicity
Most veterinarians are familiar with the phenobarbital-associated side effects of polyuria, polydipsia, polyphagia, sedation/ataxia, and potential hepatotoxicity. Hepatotoxicity with phenobarbital is dose- and duration-dependent (more likely to occur when phenobarbital concentrations are >35-40 mcg/mL). A less common adverse effect of phenobarbital in dogs that may be less familiar to practitioners is idiosyncratic bone marrow toxicity. Reversible neutropenia, thrombocytopenia, and anemia have been reported in dogs on chronic phenobarbital therapy and after an acute overdose. If not suspected to be an adverse drug reaction, the presence of pancytopenia in a dog receiving phenobarbital may result in unnecessary prescription of immunosuppressive medications, which will not correct the problem. A diagnostic workup to rule out other causes is often appropriate, but discontinuation of phenobarbital as soon as possible and initiation of an alternative anticonvulsant is indicated if another cause of bone marrow disease is not identified. Recovery from phenobarbital-induced bone marrow toxicity usually occurs within 1-3 weeks of drug discontinuation.

Phenobarbital has also been associated with dyskinesia (anxiousness and muscle twitching that appeared dose-dependent and resolved when the drug was discontinued), and superficial necrolytic dermatitis or hepatocutaneous syndrome.

2. Potassium bromide and coughing in cats
Potassium bromide shares with phenobarbital the potential to cause polyuria, polydipsia, polyphagia, sedation, and ataxia in pets. However, an additional species-specific reaction to this drug is coughing in cats. Chronic cough occurs in up to 50% of cats receiving potassium bromide and is often associated with a diffuse bronchial pattern on thoracic radiographs. Because of the frequency of this side effect, potassium bromide is not generally recommended as an anticonvulsant in cats. However, it may still be prescribed by some practitioners, with the subsequent prescription of medications for feline lower airway disease (“feline asthma”) when a cough develops. An adverse drug reaction may ultimately be suspected when treatment is unsuccessful, as the cough generally does not resolve until the drug is discontinued.

3. Methimazole and lymphadenomegaly or pyogranulomatous folliculitis
Gastrointestinal upset, facial excoriation, hepatotoxicity, and leukopenia are well-known as potential side effects of methimazole treatment, and some commercial laboratories offer hyperthyroid monitoring profiles specifically directed at detecting clinicopathologic changes associated with the latter two. However, rare cases of generalized lymphadenomegaly with or without concurrent cutaneous lymphoid hyperplasia have been described in both humans and cats receiving methimazole and may mimic lymphoma cytologically. In cats, the lymphadenomegaly resolves within a few days after discontinuation of the drug. Pyogranulomatous mural folliculitis was also diagnosed by skin biopsy in a cat receiving methimazole that developed non-pruritic alopecia with scales, crusts, and erythema.

4. Metoclopramide (or mirtazapine, or metronidazole) and neurologic signs
Hospitalized patients with systemic illness causing inappetence, nausea, or decreased GI motility may be prescribed a combination of promotility agents, antiemetics, anti diarrheals, and appetite stimulants. If neurologic signs appear in these patients, they may be
attributed to other possible disease sequelae (e.g. thrombosis, cerebral edema, electrolyte abnormalities). However, both infusion of metoclopramide and administration of mirtazapine can cause behavior changes and/or tremors, and recent medication history should be examined in patients exhibiting neurologic signs. Metronidazole can also cause vestibular and cerebellar signs in both dogs and cats at high concentrations. Either geriatric vestibular disease or primary neurological disease may be misdiagnosed if this is not realized.

5. Fluoxetine and weight loss
Fluoxetine may be prescribed to dogs and cats for various anxiety-related behavioral disorders. One of the most common side effects reported with a veterinary-labeled fluoxetine product in dogs was inappetence and weight loss (approximately 30% of dogs). This may trigger an extended diagnostic workup if it is not realized that the inappetence may be an adverse drug reaction. This scenario emphasizes the importance of educating oneself, other hospital veterinarians, and veterinary staff regarding potential adverse events associated with products prescribed or dispensed through the hospital. Package inserts are often a valuable source of information.

6. Any medication and adverse drug reactions involving the skin (e.g. erythema multiforme or toxic epidermal necrolysis)
Adverse drug reactions involving the skin are notorious for being attributed to other causes such as infection, allergy, or idiopathic immune-mediated disease. Drugs or classes of drugs that have been associated with cutaneous adverse reactions include antiparasitics (amitraz, metaflumizone, ivermectin), antibiotics (e.g. beta lactams, sulfonamides, tetracyclines), vitamin K, shampoos, contrast agents, NSAIDs, hydralazine, anticonvulsants, chemotherapeutic agents, griseofulvin, allopurinol, corticosteroids, and levophylline. A thorough drug history is important in any animal presenting with skin lesions and even drugs that the pet has received for many months should be considered as a potential etiology. More information about specific dermatologic syndromes and methods of diagnosis and treatment can be found in Reference 16.

Conclusion
The above are only a few examples of potential adverse reactions to veterinary drugs, and with the diversity of medications available in the human and veterinary marketplaces, it is not always possible for veterinarians to maintain full awareness of the occurrence of all potential adverse effects of all products used. However, recognition of adverse drug reactions and avoidance of an unneeded prescribing cascade can be facilitated by regular review of drug information (e.g. through regular perusal of package inserts and the veterinary literature, and utilization of product safety updates provided by the FDA and veterinary news sources). Veterinarians should also consider the possibility of an adverse drug reaction whenever new clinical signs appear in a patient receiving multiple medications, particularly geriatric or systemically ill patients. Involvement of the bone marrow, liver, or skin is also common with idiosyncratic adverse drug reactions and an adverse drug reaction should be on the differential list when these organs are affected. Whenever possible, polypharmacy should be avoided and the need for each medication re-assessed on a regular basis.

References
When selecting an antibiotic for use in dogs or cats, important considerations include the antibiotic’s spectrum of action (i.e. activity against likely pathogens) and its suitability for treatment of infections in a particular site (which may be affected by drug characteristics such as lipophilicity, degree of protein binding, and route of elimination). Details regarding antimicrobial spectrum and disposition may be easily recalled for frequently used antibiotics, but drug activity against emerging pathogens, or the spectra and pharmacokinetics of recently approved antibiotics, may not be as familiar. The goal of this presentation is to review of the characteristics and spectra of action of different antibiotic classes, together with questions that may arise concerning their use in light of newer options.

Antibiotics effective against gram-positive organisms
Penicillins, first-generation cephalosporins, monobactams, glycopeptides, macrolides, and lincosamides have activity primarily against Gram-positive organisms. For penicillins, these organisms include include streptococci, enterococci, some staphylococci (those without beta-lactamases), Arcanobacter, Actinomyces, Listeria, and spirochetes such as Leptospira and Borrelia. The Gram-negative spectrum of penicillins is very limited but includes Pasteurella (the predominant pathogen in cat bite abscesses), and aminopenicillins and first-generation cephalosporins are effective against selected Gram-negative organisms (e.g. E. coli, Proteus mirabilis, Salmonella). Penicillins, aminopenicillins, and lincosamides have good anaerobic coverage except against Bacteroides fragilis.

Among beta lactams, activity against beta-lactamase producing staphylococci requires an antistaphylococcal penicillin (e.g. methicillin), addition of a beta-lactamase inhibitor, or the bulkier side chain of a cephalosporin (excluding carbapenems, which should be reserved for life-threatening resistant infections). Potentiated sulfonamides, tetracyclines, and chloramphenicol also have activity against Gram-positive organisms, including staphylococci.

Q: Amoxicillin-clavulanate is often thought of as “broad-spectrum”. What does it NOT cover?
Amoxicillin-clavulanate is not reliably effective against Bacteroides fragilis, Bordetella bronchiseptica, Enterobacter and Citrobacter spp, Klebsiella, non-mirabilis Proteus spp., Pseudomonas, and Serratia. In addition, it is not effective against organisms (generally Gram negative Enterobacteriaceae) that have acquired beta lactam resistance. Therefore, amoxicillin-clavulanate (or ampicillin-sulbactam) should not be selected as a sole or first choice for potentially life-threatening infections with suspected involvement of Gram-negative organisms.

Q: What about MRSA/MRSP?
Recently, there has been an increase in the incidence of infections involving multi-drug resistant staphylococci (methicillin-resistant Staphylococcus aureus or pseudintermedius). These pathogens tend to be resistant to all beta lactams and multiple other drug classes, and preferred antibiotics include doxycycline, fluoroquinolones (variably) and aminoglycosides. Rifampin may also be used in combination with other antibiotics.

Q: Earlier generations of cephalosporins tend to have more Gram-positive activity, whereas later generations in general have greater Gram-negative at the expense of Gram-positive activity. How do the spectra of cefpodoxime and cefovecin compare with those of first-generation cephalosporins? Have they lost activity against common Gram-positive veterinary pathogens?
Cefpodoxime, like the first-generation cephalosporins, is effective against streptococci and staphylococci (other than MRSA/MRSP). It also has efficacy against beta-lactamase negative E. coli, Klebsiella, Serratia, Proteus mirabilis and vulgaris, Providencia, and Salmonella. Approximately half of human isolates of beta-lactamase- producing E. coli, Enterobacter spp, Citrobacter spp, and Morganella are susceptible. Therefore, it has slightly expanded Gram-negative coverage compared with first-generation cephalosporins, but maintains some Gram positive activity (e.g. against opportunistic skin flora causing pyoderma in veterinary patients).

Cefovecin is also a third-generation cephalosporin and is more active with lower MICs for many bacteria than first-generation cephalosporins. In studies conducted by the sponsor of a long-acting veterinary formulation, the MIC50 was 0.25 ug/mL for S. intermedius (vs. 2 ug/mL for cephalexin), and it had an MIC90 of 1 ug/mL (vs. 16 ug/mL for cephalexin and cefadroxil) for many Gram-negative organisms and very low MICs for feline Pasteurella isolates and Streptococcus canis. However, as noted by the manufacturer, this antibiotic is heavily protein-bound and free concentrations may not be sufficient to reach an appropriate time above MIC for E. coli in the plasma in vivo. Therefore, single administration may be effective only for highly susceptible pathogens (Gram-positive and Pasteurella) and for urinary tract infection (as the drug is eliminated in the urine.) It is labeled for treatment of pyoderma,
bite wounds, and abscesses in the United States, and additionally for E. coli UTI in Europe. It is not effective against Pseudomonas or enterococci.

In both cases, amoxicillin for Pasteurella or susceptible UTI, or amoxicillin-clavulanic acid or cephalexin for pyoderma, would be appropriate choices in lieu of third-generation cephalosporins unless compliance is an obstacle or Gram-negative involvement is suspected.

**Antibiotics effective against gram-negative organisms**

Aminoglycosides, fluoroquinolones, and parenteral third-generation cephalosporins (cefotaxime, ceftazidime) are generally used primarily for Gram-negative coverage. Aminoglycosides require oxygen-dependent transport into bacteria and are not effective against anaerobes, and among Gram-positive organisms, fluoroquinolones are effective against staphylococci but not streptococci. Fluoroquinolones (particularly enrofloxacin) have variable efficacy against Pseudomonas and antipseudomonal penicillins (e.g. ticarcillin) or selected third-generation cephalosporins (ceftazidime) might be preferable when fluoroquinolone resistance is encountered. Other options for treatment of Gram-negative infection include potentiated sulfonamides, tetracyclines, and chloramphenicol.

Pradofloxacin is a third-generation fluoroquinolone with expanded activity compared to other classes of fluoroquinolones. It is effective against a variety of Gram-positive, Gram-negative, and anaerobic pathogens and is labeled for treatment of dental as well as soft tissue infections in dogs.

**Anaerobes**

Drugs of choice for anaerobes include penicillins, clindamycin, metronidazole, and third-generation cephalosporins. Carbapenems and chloramphenicol also provide anaerobic coverage. Coverage is variable with tetracyclines.

**Q: Is there any rationale for using both a penicillin and metronidazole in animals with suspected anaerobic (e.g. clostridial) infections?**

Metronidazole’s coverage is exclusively anaerobic and in contrast to that of penicillins, it includes *Bacteroides fragilis*. However, the significance of this pathogen in veterinary medicine is questionable, and there is minimal difference in the effectiveness of metronidazole and penicillins against clostridia.
Because of the relatively small proportion of pharmaceuticals marketed for dogs and cats and the limited dispensary space in many veterinary hospitals, veterinarians frequently prescribe medications via an outside pharmacy (either human or veterinary). Confusion may arise during multiple steps in this process, e.g. during communication of the prescription (which often occurs via telephone), or during filling of the prescription and delivery of the medication to the client and ultimately to the pet. Veterinarians may also receive inquiries from clients about prescribed medications for which a seemingly comparable human or generic formulation is available.

The problem of prescribing confusion is not unique to veterinary medicine, and in human medicine, the nonprofit Institution for Safe Medical Practices (ISMP) has produced guidelines for hospitals and pharmacies to help reduce the likelihood of patient harm from medication errors. Application of these guidelines to veterinary prescribing can be helpful, as can an understanding of the distinctions between different formulations of human and veterinary drugs.

Accidental substitution during the prescription process: sources of error and measures to prevent them

“Look-alike/sound-alike” drugs

Considering the array of medications available for prescription through human pharmacies, it is not surprising that one drug may be inadvertently substituted for another based on a similar-sounding name, or a name with a similar written appearance. To prevent such a substitution, which can be life-threatening for the patient (e.g. azathioprine for azithromycin in a cat), the following (adapted from ISMP guidelines) are recommended.

1. Educate staff about common “sound-alike” drugs, and consider posting a list in an accessible area where telephone prescribing occurs. A list of “sound-alike” drugs from human medicine is periodically published by the ISMP and this can be reviewed to select those applicable to a particular veterinary practice. The list can be reviewed and renewed at least quarterly by a staff member.

2. The person placing a telephone prescription should either spell the name of the drug to the pharmacist for verification, or have the pharmacist spell it to the prescriber. The prescribing individual should also provide an indication for the drug, e.g. “for infection”. A drug indication that does not match the drug name will often trigger closer scrutiny.

3. When submitting a written prescription, be sure handwriting is legible, and, as for telephone prescriptions, be aware of drugs with similar-appearing names and provide an indication as part of the prescription. Do not abbreviate drug names.

4. If you routinely leave prescriptions on a pharmacy’s voicemail, make sure the client has the drug name (and potentially the strength and directions) in writing to verify at pickup

Dosing errors

Dosing errors can result from incorrect interpretation of two similar-sounding numbers (e.g. “fifty” for “fifteen”), from illegible written prescriptions or incorrect placement of decimal points, from incorrect calculations, or from unfamiliar abbreviations. To avoid dosing errors:

1. When communicating a number over the phone, repeat numbers in digits, e.g. “one five” in addition to “fifteen.”

2. As for drug names, make sure drug dosages are legible, with no ambiguity surrounding numbers or decimal points (do not use trailing zeros, and place zeros before decimal points), and avoid abbreviations (particularly SID, which is not used in human medicine) where possible.

3. Double check all calculated drug doses.

General communication obstacles

For either a spoken or a written prescription, the prescriber should always 1) be willing to clarify the prescription to the pharmacist, and 2) maintain a courteous attitude. In a survey conducted among human healthcare workers by ISMP, 83% of pharmacists said they had encountered a reluctance or refusal to answer questions or return calls by prescribers, and 40% of respondents reported that they assumed a medication order they had concerns about was correct, rather than seeking clarification from the prescriber, if the prescriber was perceived as intimidating. 7% of these individuals also acknowledged that they had been involved in a medication error in which intimidation played a role within the previous year.

Potential sources of error when the prescription is filled

Human pharmacists reviewing veterinary prescriptions may note differences in the veterinary and human indications and dosing regimens for certain drugs. When this information is communicated to the client at pickup, it can cause confusion. For example, mirtazapine is an antidepressant in humans; and clients picking up benazepril for treatment of proteinuria may be told that it is for
blood pressure management. When a medication is prescribed, attention should be given by veterinary staff to educating clients about the drug’s purpose, directions, side effects, and any notable differences from use of the drug in humans.

Substitutions among human formulations
Clients or pharmacists may ask a prescribing veterinarian whether a substitution of one human drug or formulation for another is acceptable, or pharmacists may assume it is unless otherwise specified. Generic human drugs may be assumed to be equivalent to name-brand formulations, but this is not always the case in pets, as bioequivalence has been demonstrated between human name-brand and generic drugs in human populations, but not in canine and feline populations. If a drug has an easily monitored clinical effect (e.g. amlodipine), if the consequences of lack of efficacy are not serious or life-threatening, if there is not a body of veterinary data supporting a specific formulation only, or if the drug is not a biological product such as insulin or levothyroxine, substitution of a generic for a name-brand human formulation may be acceptable. Hormone-based medications are more difficult to standardize among patients and clients should be aware that substitution of one brand of insulin or levothyroxine for another may require re-regulation of the patient’s disease.

Substituting a human drug for a veterinary drug
In some situations, clients may ask if a human drug formulation or human drug from the same class can be substituted for a veterinary prescription, for cost or convenience reasons. Clients may not be aware that human and veterinary-labeled drugs may differ in effectiveness despite having the same active ingredient, as other components of the product (excipients, enteric coating) can affect absorption and bioavailability. The approval requirements for veterinary-labeled drugs require substantial evidence of safety and efficacy, which generally entails clinical trials in the species of interest documenting appropriate pharmacodynamics (with or without pharmacokinetic data) and information about the incidence of adverse events with that formulation specifically in that species. The same is not true of human formulations used in pets.

For several human drugs with veterinary analogues, however, a large body of literature describing efficacy and nature of adverse events to be expected with the drug exists, and efficacy is not restricted to a specific formulation (e.g. cephalaxin, methimazole, amlodipine). Substitution of a human drug for the veterinary version in this case will likely result in similar efficacy, but may result in the loss of modifications specific to the veterinary product (most often improved palatability or precise dosage forms) and support from the veterinary sponsor in case of adverse reaction. With regard to drugs from the same class, substitution of ciprofloxacin for enrofloxacin is often requested by clients for financial reasons, but is not advised considering the wide variation in ciprofloxacin bioavailability among dogs, which has been demonstrated in multiple studies.

Substituting veterinary generic drugs or compounded drugs for veterinary name-brand formulations
Manufacturers of veterinary generic formulations are required to demonstrate bioequivalence of the generic product to the parent (name brand) drug. Generally, this means that the two products produced drug exposure (as assessed by maximum plasma concentration and area under the curve) that was similar enough it would not be expected to result in a clinical difference. Therefore, generic veterinary products can often be substituted for name-brand products with minimal repercussion, although for hormone-based formulations, re-regulation may then be necessary, as discussed above.

The same is not true of compounded products, for which no comparison with name-brand or generic products must be made and which are not subject to outside regulations regarding stability, efficacy, and safety. Although compounded products may be effective in some circumstances, they should be used only when approved alternatives are not available, and inquiries should be made of the compounding pharmacy regarding stability data and manufacturing practices for their products. Two recent veterinary studies have identified stability concerns and inadequate plasma concentrations with compounded doxycycline and itraconazole, respectively, in dogs, and numerous other examples exist.

Additional notes on prescribing through an in-clinic pharmacy
Several of the guidelines for avoiding communication errors with human pharmacies can be applied to medication orders within a veterinary hospital. Prescribing veterinarians can help to minimize medication errors by communicating medication orders clearly to staff, and by being courteous and receptive to questions. Additionally, “look-alike/sound-alike” drugs, especially injectable drugs, can be labeled as such and placed in different areas of the in-clinic pharmacy.

References


Physiology of neonatal (0-2 weeks), infant (2-6 weeks), and pediatric (6-12 weeks) dogs and cats differs from that of adults in several important ways that can impact drug metabolism and disposition. Proportion of total body water is substantially higher in puppies and kittens, leading to a higher volume of distribution and lower than expected plasma concentrations of water-soluble drugs; the reverse is true for fat-soluble drugs. Renal and hepatic function are also immature until at least 3-6 weeks of age. In addition, young animals have slower gastrointestinal transit time and a more permeable blood-brain barrier than adults. Because of the latter and because certain systems are still developing (e.g. physes, tooth buds) younger dogs and cats may be more susceptible than adults to certain adverse drug reactions.

For some drugs (primarily antiparasitics), specific information exists to support safe use in puppies and kittens as young as 4 weeks. Other drugs have been demonstrated to have increased risk of toxicity in young compared with mature animals. However, for most drugs, neither of the above is true. Labels for veterinary products may include statements specifying that the product is approved only for use in animals above a certain age. In some cases, a particular adverse effect was noted during safety assessment. However, because most veterinary pharmaceuticals are not specifically designed for puppies and kittens, safety testing is often performed in “healthy young mature animals” as per FDA guidance. Consequently, the label recommendation to avoid use in animals younger than a certain age may simply mean that its safety in pets below that age has not been evaluated. Approval for younger animals may be sought later if they form a substantial portion of the use class after initial marketing (e.g. maropitant). Only a very few medications are approved for pregnant or lactating animals.

Consequently, recommendations for use of specific drugs in puppies and kittens are often based on an amalgam of information from other species, theoretical support or concerns, and clinical experience. A collection of these recommendations, by drug class, is discussed below.

Antibiotics

B-lactams (penicillins and cephalosporins) are often suggested to be safe in young animals because of their wide therapeutic index, which alleviates concern about a slower elimination rate. However, in neonates, penicillins may be associated with suppression of normal GI flora and colonization by pathogenic organisms. The same does not appear to be true of cephalosporins, several of which are active against common pathogens causing neonatal septicemia. Macrolides are not overtly toxic, but undergo hepatic recirculation and may disrupt GI flora. Trimethoprim-sulfonamide has also been recommended as an initial broad-spectrum choice for puppies and kittens, although it has been associated with immune-mediated drug reactions in adults. Chloramphenicol has caused blood dyscrasias in 8-12 week old puppies at 50 mg/kg twice a day, and causes cardiac depression in neonates of other species (e.g. humans). Aminoglycosides have a narrow therapeutic index and young age has been listed as a risk factor for nephrotoxicity; however, these agents may still be used if adequate hydration is ensured. If metronidazole is used, very young puppies and kittens should be closely monitored for neurotoxicity. Tetracycline-induced tooth staining (which appears less common with doxycycline but has still been reported to occur) and cartilage damage with fluoroquinolones in large-breed puppies up to 18 weeks of age are often cited as reasons not to use these drugs in young animals. However, if life-threatening infection is present, fluoroquinolones may be appropriate as a means of Gram-negative coverage.

Analgesics and sedatives

Based on human literature, short-term opioid use is likely to be safe in pregnant, nursing, and neonatal dogs and cats. NSAID use is to be avoided in pregnant animals based on numerous studies in other species documenting fetal nephrotoxicity (as COX-2 is important for the developing kidney) and teratogenesis. Because most NSAIDs are lipid-soluble and highly protein-bound, they may not be transferred in milk in sufficient concentrations to affect nursing animals; however, this has not been demonstrated in dogs or cats, and meloxicam concentrations in the milk of rats were higher than those in plasma. NSAIDs are not recommended for animals less than 6 weeks of age because of ongoing hepatic and renal maturation.

Benzodiazepines have a large margin of safety and are often recommended as components of sedative or preanesthetic protocols for young puppies. Opioids alone have a sedating effect in pediatrics (<4 mo). Acepromazine may cause CNS depression in young animals and dose reductions to 0.005-0.025 mg/kg have been recommended. Lidocaine can be used as a local anesthetic at 3-6 mg/kg in kittens (with the lower dose to be used in neonatal animals) and 6-10 mg/kg in dogs. Similarly, some authors recommend bupivacaine at 2 mg/kg in older puppies and kittens and half of this dose for patients <12 weeks.
Anticonvulsants
Both phenobarbital and potassium bromide have been used to manage seizures in young puppies. Potassium bromide does not require hepatic metabolism, which may be advantageous in animals with immature liver function. Phenobarbital use in human children and young rats has been shown to interfere with brain development, and some authors recommend reduction of the initial dose for animal younger than 6 months (e.g. 0.5 mg/kg once a day for animals less than 3 months old, 1 mg/kg twice a day for 3-6 month old animals, and 2 mg/kg twice a day for animals older than 6 months). Gabapentin and levetiracetam have not been evaluated in young dogs and cats.

Antiparasitics
Pyrantel has a wide safety margin and has frequently been used in animals as young as 2 weeks of age. Many other antiparasitics and flea/tick control products have been marketed by veterinary pharmaceutical companies, and age recommendations can be found on the product label.

References
The relationship between administration of a drug to a patient and appearance of a clinical effect is complex, and is affected by both pharmacokinetic and pharmacodynamic factors. Pharmacokinetic (PK) factors include all processes involved in absorption, distribution, metabolism, and elimination of the drug; although these may be broadly similar within a species for a particular drug, they may also vary enough between individuals (or even within the same individual over time) to influence clinical effect. Likewise, pharmacodynamic (PD) processes such as receptor activation or enzyme inhibition vary among patients and may be responsible for differences in therapeutic response. Therefore, although published drug dose ranges may result in the desired clinical effect in some patients, they may not be ideal for others.

Therapeutic drug monitoring (TDM) refers to measurement of a pharmacologic or physiologic parameter at specific time points during drug treatment, with the goal of optimizing the dosing regimen for an individual patient. Traditionally, the parameter measured has been the plasma drug concentration (PK monitoring); however, TDM based on biomarkers or other indicators of drug effect (PD monitoring) may also be appropriate for some drugs. TDM can be thought of as a way to access otherwise hidden information regarding the chain of events that occurs between drug administration and drug effect. This information can be utilized preemptively (to assess the appropriateness of the dosing regimen before clinical signs of toxicity or lack of efficacy become apparent) or diagnostically (to investigate whether lack of efficacy or suspected toxicity are related to PK or PD problems, and to evaluate client compliance).

Obviously, TDM is not necessary or suitable for every drug that is used in veterinary medicine, as treatment goals can often be accomplished via empirical dose adjustment. TDM contributes to patient management primarily when:

- The drug being used has a narrow therapeutic index or a steep dose-response curve (e.g. aminoglycosides)
- Drug PK is highly variable among patients or in the same patient over time (e.g. cyclosporine, anticonvulsants)
- The drug is prone to significant drug-drug or drug-disease interaction (e.g. digoxin).

Even when the above criteria are met, TDM is generally employed only when the endpoint of therapy is difficult to monitor clinically, and the consequences of toxicity or lack of efficacy are serious. A serious consequence could mean actual harm to the patient, or it could mean a significant financial or emotional cost to the client of giving an ineffective therapy for an extended period of time. Limitations of TDM include the fact that plasma drug concentration may not reflect drug concentration at the active site or provide information about the pharmacodynamics of the drug in that patient, and individual patients may experience drug efficacy or toxicity at concentrations different from published ranges.

Because TDM is a “snapshot” of drug concentration or drug effect on a physiologic parameter at only one or two points in time, those points must be chosen carefully to maximize the information received. In understanding appropriate sample timing, two pharmacokinetic concepts are helpful: accumulation and steady state. When the next dose of a drug is given before the last dose has been completely eliminated from the body, drug accumulation will occur. The degree of accumulation is determined by the ratio of the half-life to the dosing interval; if this ratio is large (the half-life is very long compared to the dosing interval) only a small percentage of drug will be eliminated during each dosing interval and accumulation will be marked. Conversely, when the half-life is very short compared to the dosing interval, almost all the administered dose will be eliminated during the dosing interval and accumulation will be minimal.

Any drug that exhibits accumulation will eventually reach steady-state (in which the mass of drug eliminated from the body during each dosing interval has “caught up with” or become exactly equal to the mass of drug administered with each dose). At steady state, which occurs after 5-7 half-lives, the peak and trough plasma concentrations are very similar across dosing intervals. The difference between the peak and trough concentration within a dosing interval is also determined by the ratio of the half-life to the dosing interval; if this is large, the amount of drug eliminated during each dosing interval will be very small compared to the steady-state plasma drug concentration, and there will be only a small difference between peak and trough. For drugs with a shorter half-life:dosing interval ratio, a larger percentage of drug will be eliminated during each dosing interval and the difference between peak and trough will be larger.
The implications of these principles are that for the first category of drugs (extensive accumulation, e.g. KBr), a single missed dose will have little impact on the steady-state plasma drug concentration, and either peak or trough concentration can be measured for preemptive and diagnostic monitoring. For drugs with a shorter half-life:dosing interval ratio (e.g. phenobarbital), missed doses may impact steady-state concentration (i.e. if a history of recent missed doses is discovered on the day TDM was planned, rescheduling should be considered). Trough (before the next dose) is usually measured for preemptive monitoring or diagnostic monitoring for inefficacy (to avoid the influence of variability associated with absorption on peak concentrations), whereas peak is measured if toxicity is suspected. An exception to the latter is aminoglycosides, in which trough drug concentration correlates with toxicity and peak with efficacy.

For calculation of half-life, two samples (generally peak and trough) are necessary; for drugs expected to have half-lives substantially shorter than the dosing interval, “trough” samples should be taken two half-lives after the peak to avoid undetectable drug concentrations prior to the next dose.

A summary of monitoring recommendations for selected drugs in dogs is as follows

**Phenobarbital**
- Half-life: 32-75h
- Preemptive monitoring
- Trough for consistency (peak vs trough not important in 90% of patients)
- Post-load
- 2 weeks (steady-state)
- 3-6 months after starting (due to enzyme auto induction)
- Every 6-12 months (clinical judgment)
- Diagnostic monitoring
- Peak (4-5 h) if suspect toxicity
- Trough (before next dose) if suspect lack of efficacy
- Peak and trough if suspect short half-life

**Potassium bromide**
- Half-life: 14-21 days
- Preemptive monitoring
- Any time within dosing interval (i.e. peak, trough, or in between)
- Post-load
- 3 weeks (one half-life) after load – if concentration has declined from post-load, maintenance dose may need to be increased
- 3 months (steady-state)
- Every 6-12 months (clinical judgment)
- Salt content of diet must be kept constant
- Diagnostic monitoring
- Any time within dosing interval

**Levetiracetam**
- Half-life: 2-3.6 h
- Preemptive monitoring
- Peak (2 h) and “trough” (two half-lives, or 4-6h, after peak) when beginning therapy
- Goal: determine half-life and customize dosing regimen
- Diagnostic monitoring
- Peak if suspect toxicity (rare)
- Trough (+/- peak for half-life) if suspect lack of efficacy

**Zonisamide**
- Half-life 16-65 h
- Preemptive monitoring
- Peak (2 h) and trough (before next dose) when beginning therapy
- Steady-state 10 d
- Diagnostic monitoring
- Peak if suspect toxicity
- Trough if suspect lack of efficacy
Aminoglycosides
- Half-life
  - 0.9-1.3 h (gentamicin)
  - 1-3 h (amikacin)
- Preemptive monitoring
- Peak (0.75-1 hour) and “trough” 2 half-lives (4-6 h) after peak (to determine half-life in patient)
- Diagnostic monitoring
- Trough if suspect toxicity

Cyclosporine
- Half-life: 3-8 h
- Preemptive monitoring
- Trough
- 1-2 days (steady state) after starting
- Peak (2h) concentrations correlate best with total drug exposure in humans; PD monitoring (calcineurin inhibition) may be alternative to PK monitorin
- Diagnostic monitoring
- Peak if suspect toxicity
- Trough if suspect lack of efficacy
- Therapeutic range not well defined
- Trend in patient may be more important
- Trough <50 ng/mL associated with lack of efficacy

When interpreting the results of pre-emptive monitoring for drugs such as phenobarbital, the need for further investigation and/or dose adjustment can be determined based on 1) whether or not the plasma concentration is as expected from the administered dose, and 2) how the results of TDM fit the clinical picture. With regard to the first point, it is helpful to consider that drug dosage and plasma drug concentration should vary in a proportional manner for drugs with linear pharmacokinetics (most drugs utilized in veterinary medicine). Therefore:

\[
\text{Old dose} = \frac{\text{New dose}}{\text{Old } C_p} \quad \text{New } C_p
\]

where \( C_p \) = plasma drug concentration.

If a higher plasma drug concentration is desired, the new dose required to achieve that concentration can be calculated using this equation. Additionally, when plasma drug concentration is measured after a dose change, the new concentration expected can be calculated and compared with the actual results. A large discrepancy may indicate difficulties with owner compliance or changes in drug absorption or elimination, and these issues can be further investigated.

Information regarding practice management aspects of TDM will also be covered by a co-presenter.

References
Interventional Radiology (IR) is defined as the use of contemporary imaging and endoscopic modalities to gain access to various anatomic structures to deliver therapeutic agents. The advantages of minimally invasive techniques are well documented in the human medical field, and the benefits of decreased hospitalization and pain and faster recovery will likely be seen in veterinary medicine with improved proficiency in these skills and thorough research is conducted. Interventional radiology and endoscopy also provide alternative treatments to traditional management or even a last option for palliation and improved quality of life with some terminal disease states.

**Tracheal collapse & tracheal masses**

Medical management is the mainstay of management of tracheal collapse, and may include the use of any combination of cough suppression, anti-inflammatory medications, antibiotics, bronchodilators, and sedation. For dogs with progression of tracheal collapse, medical management may not be able to effectively control clinical signs of airway obstruction, resulting in life threatening respiratory distress.

For patients with intra-thoracic collapse, or patients with cervical collapse where there are increased risks associated with surgical prosthetic ring placement, intra-luminal tracheal stenting provides a rapid, non-invasive, permanent method to relieve tracheal collapse. Advantages of tracheal stenting include it being minimally invasive, surgical dissection is not required, anesthetic time is lessened, and it can treat disease of the intra-thoracic trachea.

The extent of tracheal collapse, including the presence of mainstem bronchi collapse is assessed with fluoroscopy and pre-operative tracheoscopy. At the time of the procedure, a thorough laryngeal examination and endotracheal wash are also performed, as tracheal infections are becoming more commonly recognized in dogs undergoing tracheal stenting. Stent size is selected under general anesthesia, and the stent is placed through the endotracheal tube using a bronchoscope adapter to permit continued delivery of oxygen during stent placement. Patients are generally monitored in the intensive care unit (ICU) for 24 hours after stent placement. Repeat thoracic radiographs prior to discharge are used to confirm stent positioning and evaluate for the presence of bronchopneumonia.

Lifelong continued medical management for cough control is essential for most tracheal stent patients to prevent complications associated with stents. Complications include stent migration, fracture, progressive collapse in the unstented portion of the trachea, and the development of intra-luminal inflammatory tissue cranial or caudal to the stent ends. Tracheal stents can also be used to increase tracheal lumen diameter in patients with strictures and neoplasia and have been used successfully in both cats and dogs.

**Percutaneous antegrade urethral catheterization**

When retrograde urethral catheterization is not possible, such as in small female dogs, patients with obstructive neoplasia, or patients with urethral tears secondary to trauma, antegrade urethral catheterization performed with the assistance of fluoroscopy can prevent the need for cystostomy tubes or emergency surgery.

With the patient anesthetized or heavily sedated, cystocentesis is performed with an over-the-needle catheter after aseptic preparation of the caudolateral abdomen. Urine is removed for urinalysis and culture (if indicated), and an iodinated contrast agent is injected into the bladder under fluoroscopy to delineate the bladder and urethra. An angled hydrophilic guidewire is advanced through the catheter and into the bladder. Under fluoroscopic guidance, the wire is advanced into the trigone and out the urethra. A urinary catheter is passed retrograde over the wire, positioned appropriately within the bladder, and secured routinely.

**Urethral stenting**

Intra-luminal or extra-luminal urethral obstructions and compressions can quickly become life-threatening emergencies. For many patients, surgical resection of the cause of the obstruction, particularly in the case of neoplastic obstructions, is not a viable option. Permanent cystostomy tubes are associated with complications and can negatively affect an animal’s quality of life.

The most common indications for urethral stents include transitional cell carcinoma, prostatic carcinoma, urethral strictures, and urethral compression from malignant enlargement of other abdominal organs, such as lymph nodes.

Urethral stenting provides a permanent, non-invasive, comfortable option to relieve urethral obstructions in dogs, and more recently, cats. Stent size is based on contrast retrograde cystourethrography generated measurements of normal and diseased urethral diameter and length. A repeat contrast cystourethrogram is performed after the stent is deployed to confirm patency of the urethra followed by abdominal radiographs to confirm stent positioning should future comparisons be needed.

The most significant complication associated with urethral stenting is incontinence; with approximately 25-30% of patients being affected by severe incontinence regardless of patient sex or length of urethra stented. Due to the risk of incontinence, the procedure is
transfusion. There are substantial financial implications for clients of patients requiring large amounts of blood products, and the strain placed on a veterinary practice’s blood product supply can impact future patients in need.

Selective, transarterial catheterization can be performed via access through the femoral or carotid artery with the aid of fluoroscopic guidance. Hemostasis can be achieved through placement of thrombogenic coils, gelfoam, or polyvinyl alcohol particles. This technique has been used for intractable epistaxis, hemorrhage from gastric ulceration, and traumatic laceration of the genicular artery in a dog with a femoral condylar fracture.

In patients with non-resectable neoplasms, particular hepatocellular carcinomas, intra-arterial delivery of chemotherapy with or without embolic polyvinyl alcohol particles is an emerging interventional option for tumor management. This technique has also been used for soft tissue sarcomas, tumors of the head and neck, and urinary tract neoplasias. Using arterial access (femoral or carotid), selective catheterization into the arterial blood supply of a given tumor is achieved with angiography and fluoroscopy. Depending on the nature of the mass, collateral blood supply, and other structures being supplied by the artery in question, chemotherapy with or...
without embolic particles are delivered under fluoroscopic guidance. Often, a systemic dose of chemotherapy is given locally to increase drug levels within the tumor. Embolic particles are added to achieve stasis of blood flow to the tumor.

Experience with intra-arterial chemotherapy and chemoembolization is still in its infancy in veterinary medicine, though preliminary experiences are promising and clinical trials are being conducted at several institutions to determine safety and efficacy.

References
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Too Hot to Handle:
Management of the Heat Stroke Patient
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Heat stroke is the most serious form of heat-induced illness, with the hallmark clinical sign being central nervous system dysfunction. Heat stroke results when the mechanisms for heat dissipation are overwhelmed by heat load. Cooling measures are the mainstay of emergency treatment, followed by intensive management of hypovolemia, bacterial translocation, coagulopathies, acute kidney injury, and nervous system dysfunction. Prolonged hospitalization, transfusion support, and intensive care are imperative for survival of these critically ill veterinary patients.

Pathophysiology
Heat stroke is defined as “hyperthermia associated with a systemic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates”. There are less severe versions of heat-induced illness, which are often under-recognized in veterinary patients, are heat cramp and heat exhaustion. Heat cramp occurs when sodium and chloride loss causes muscle spasms and tremors. Heat exhaustion is characterized by weakness, fatigue, vomiting, and diarrhea.

Cats rarely suffer from heat-induced illness, therefore the following discussion focuses mainly on dogs. Most dogs present when warm, humid weather begins and becomes less frequent later in the summer, which is likely due to fact that it takes 60 days for full acclimatization in animals. Exertion and exposure to warm, humid conditions does not automatically equate to heat induced illness. Heat induced illness results when the core body temperature increases the heat load in excess of the heat dissipating mechanisms.

The major heat loads for animals are:
- Warm, humid environment (non-exertional)
- Exercise (exertional)

The major mechanisms of heat dissipation are:
- Convection – heat transfer from the body to air as air is moved over the patient
- Conduction – occurs when heat is transferred from patient to a cooler surface via direct contact
- Radiation – body heat release into the environment
- Evaporation – conversion of water from its liquid form to a vapor
- As the ambient temperature increases above 89.6°F (32°C) evaporation becomes most important method for heat dissipation

Factors that diminish heat dissipation include:
- Confinement/poor ventilation
- Upper respiratory disease/anomalies – brachycephalic airway syndrome, laryngeal paralysis, collapsing trachea
- Obesity
- Thick haircoat
- Cardiac disease
- Lack of acclimatization

There are multiple protective mechanisms that are inherent to the physiology of buffering against an excessive heat load. In addition the mechanisms of heat dissipation listed above, other core thermoregulatory responses include tachycardia, vasodilation, and shunting of blood to the cutaneous circulation, all of which are designed to increase heat loss through the skin. In order to increase respiratory heat losses, minute ventilation (minute ventilation = respiratory rate x tidal volume) is increased. Heat shock proteins, which are intracellular proteins to protect and improve enzyme function during heat insults, and both pro- and anti-inflammatory cytokines are important to halt protein denaturation and damage.

Predisposing factors
While any animal can succumb to heat induced illness when their mechanisms of heat dissipation are exceeded by heat load, there are multiple predisposing factors that can make certain animals more susceptible to heat stroke. Since panting and evaporative heat loss is a major mechanism of thermoregulation in dogs, any airway disease that narrows the airway or impedes air-flow is a risk factor for heat injury. Dogs with brachycephalic airway syndrome, particularly laryngeal and palate disease that impedes ventilation, are at risk of heat stroke even in environmental conditions that would not be problematic for dogs with mesocephalic or dolicocephalic facial anatomy. Dogs with laryngeal paralysis, laryngeal collapse, and tracheal collapse are also susceptible to heat stroke from an inability to eliminate heat load due to ineffective panting from airway obstruction. Other disease processes that may impair heat dissipation include cardiovascular disease and neurologic or neuromuscular disease. Obesity and a thick haircoat can decrease radiant and convective heat loss, thereby increasing heat load.
Physical examination

- Temperature may be normal, increased or decreased depending on prior interventions and perfusion. When body temperature exceeds 105.8°F (41°C) permanent brain injury may result
- On cardiovascular assessment, sinus tachycardia common and ventricular arrhythmias are also possible. The capillary refill time (CRT) may be fast from vasodilation. Peripheral pulse quality is often weak due to fluid losses (panting, vomiting, diarrhea) and vasodilation
- On respiratory assessment, panting is common and upper airway noise may be heard from a distance or ausculted as referred upper airway noise on lung auscultation. Pulmonary parenchymal harshness and/or crackles may be heard if aspiration pneumonia or parenchymal hemorrhage secondary to coagulopathy are present.
- Mental depression is common, but mentation can vary from normal to comatose on neurologic assessment. Pupil size and PLR may vary and ataxia may also be present in ambulatory patients.
- Vomiting and diarrhea, with or without evidence of gastrointestinal hemorrhage (hematemesis, hematochezia, melena), are common findings on assessment of the gastrointestinal system.
- Urinary system findings can be variable since bladder size will depend on the patient’s last void and will not immediately reflect renal dysfunction. Urinary bleeding may be evident with coagulation derangements.
- Petechia and ecchymoses may be seen in the oral cavity, sclera, or anywhere on the skin secondary to thrombocytopenia or thrombocytopenia.

Common clinicopathologic findings

- Minimum database (MDB)
  - Packed cell volume (PCV) may be increased due to hemoconcentration, decreased from gastrointestinal blood loss, or may be normal due to a combination of these two effects
  - Total solids (TS) may be increased due to hemoconcentration, due to protein loss through diarrhea, or may be normal due to a combination of these two effects
  - Blood urea nitrogen (BUN) may be increased from pre-renal azotemia, renal azotemia, and/or gastrointestinal bleeding
  - Blood glucose (BG) can be decreased from increased utilization during hyperthermia or as an early marker of sepsis
- Electrolytes
  - Hypernatremia may be seen secondary to free water losses from excessive panting, urinary and/or gastrointestinal losses
  - Hypokalemia may be seen secondary to gastrointestinal and renal losses
  - Hypochloremia can be present due to losses from vomiting
- Lactate may be elevated from decreased perfusion during hypovolemic shock
- Serum chemistry
  - Increased BUN and creatinine may be present due to pre-renal or renal azotemia
  - BUN may also be increased with gastrointestinal bleeding
  - Alanine aminotransferase (ALT) and creatinine kinase (CK) can be increased secondary to muscle injury
  - Bilirubin may be increased with hepatic dysfunction and associated with cholestasis of sepsis
  - Cholesterol can be decreased from gastrointestinal blood loss and hepatic dysfunction
- Complete blood count
  - Increased nucleated red blood cells (nRBC) are present early after heat stroke and are an indicator of bone marrow insult
  - Thrombocytopenia can secondary to blood loss and/or consumptive processes
- Urinalysis
  - Glucosuria may be suggestive of recent hyperglycemia and/or tubular injury
  - Tubular casts on urine sediment can be indicative tubular injury
- Coagulation screening
  - Prothrombin time (PT) and partial thromboplastin time (PTT) are increased secondary to coagulation factor activation and consumption

Emergency management

Since several upper airway obstructive disease processes, such as brachycephalic airway disease, laryngeal paralysis, and tracheal collapse can impair heat dissipation and lead to heat induced illness, it is important to ensure adequate oxygenation and ventilation.
upon arrival to the emergency room. Oxygen supplementation should be provided if there is concern for the patient’s oxygenation saturation. Appropriate ventilation should be confirmed through assessment of the patient’s respiratory status, physical examination, and arterial or venous assessment of partial pressure of carbon dioxide (PCO2). For patients struggling to ventilate or with severe hypoxemia that is non-responsive to supplementation, sedation, intubation, and ventilation may be necessary.

Active patient cooling is one of the most important aspects of initial stabilization of the heat stroke patient. Since many clients recognize their pet is overheating, cooling measures may have already been instituted prior to hospital arrival. The safest cooling method is to wet the animal with water and then placing it in front of a fan. This technique utilizes evaporation, conduction, and convection. Concurrent muscle massage helps to maintain circulation and prevent peripheral vasoconstriction in response to surface cooling. For patients with very thick coat, shaving of the haircoat could be necessary to facilitate effective heat dissipation. It is very important to monitor the patient’s rectal temperature frequently during cooling measures and discontinue cooling when the patient’s temperature has reached 103.5-104°F (39.7-40°C) to avoid rebound hypothermia.

Ice water baths should be avoided since they cause marked cutaneous vasoconstriction and shunt warm blood to the core. Alcohol on the footpads is ineffective because of small surface area. It is also noxious to the animal and causes a fire risk in the event that defibrillation could be needed. Gastric lavage, peritoneal lavage, and cold water enemas should also be avoided due to invasiveness, risk for fluid balance and electrolyte complications, and risk for septic peritonitis and aspiration pneumonia.

Many patients with heat stroke are in hypovolemic shock, due to large fluid losses through panting, vomiting, and diarrhea, and vasodilatory shock from systemic inflammatory response syndrome (SIRS). Cardiovascular support was intravenous fluid therapy is essential for volume replacement and expansion and restoration of perfusion.

Room temperature fluids should be used since they are cool relative to the patient’s core temperature and avoid causing phlebitis that could be caused by cold IV fluid use.

Fluid therapy needs may require a combination of isotonic crystalloids, colloids, hypertonic saline, and blood products depending on the patient’s perfusion, intra-vascular volume, ongoing losses, PCV, and coagulation status. If appropriate intravascular volume does not improve perfusion parameters and blood pressure, vasopressor and/or ionotropic support with vasopressors and/or positive inotropic drugs may be needed. Overly aggressive fluid therapy should be avoided due to concerns for decreased intravascular oncotic pressure that can accompany protein losses, loss of vasomotor tone, and capillary leakage from systemic inflammation, all of which may cause fluid pooling and edema.

Once hypovolemia has been corrected, fluid therapy should be tapered to maintain hydration and address any ongoing losses, such as gastrointestinal and urinary losses. Cardiac arrhythmias can occur secondary to myocardial hypoperfusion as well as be induced by circulating cytokines. ECG monitoring should be performed and any arrhythmias negatively impacting perfusion should be treated.

Obtaining a urinalysis prior to induction of fluid therapy is valuable for the assessment of renal concentrating abilities. However, given that coagulopathies are one of the most common complications of heat stroke, obtaining this sample by cystocentesis should be avoided until normal primary and secondary hemostasis have been confirmed. Placement of a urinary catheter is ideal for accurate assessment of urine production and serial urinalyses for tubular injury monitoring. Indwelling catheters can be difficult to place in small female dogs, and are associated with ascending urinary tract infections. Infection risk increases with diarrhea, which is a common complication of heat stroke patients. For patients whom catheterization is not possible or contraindicated, urine production can be determined by free catch or weighing diapers. Patient body weight should also be measured regularly (every 6-8 hours) to aid in assessment of fluid balance. Frequent assessment of measures of renal function (BUN, creatinine, potassium, sodium, and acid-base balance) should also be performed regularly. For patients with acute kidney injury that is non-responsive to medical management strategies, peritoneal dialysis or hemodialysis may be indicated for oliguria or anuria refractory to medical management.

Since encephalopathy is the hallmark of heatstroke, serial neurologic examinations to assess for improvement with restoration of perfusion and tissue oxygenation should be performed and well documented in the record. Serum sodium monitoring is essential to avoid rapid fluid shifts in the CNS. Frequent glucose monitoring should also be performed to ensure normoglycemia. For patients with signs of increase intracranial pressure (ICP) despite medical therapy, medical management to decrease cerebral edema (mannitol, hypertonic saline, 15-30 degrees of head elevation, and avoidance of jugular venipuncture) should be instituted.

Broad-spectrum antibiotic coverage for bacterial translocation secondary to thermal injury and altered GI perfusion is indicated in many heat stroke patients. Additional gastrointestinal management considerations include therapies for treatment or prevention of gastric ulceration with acid production blockade and mucosal protectants such as sucralfate as well as nutritional support.

Frequent monitoring of primary and secondary hemostasis is very important in heat stroke patients. Many needed transfusion support with multiple blood (packed red blood cells, whole blood, and/or fresh frozen plasma) during recovery from severe heat induced illness.

**Prognosis/outcome**

Heat stroke can be difficult to treat and requires aggressive critical care and monitoring.
The outcome is often dependent on the dog’s prior health status and severity of heat insult. Factors associated with a more poor prognosis include presentation in a coma or progressive neurologic decline, hypothermia on presentation, persistent hypoglycemia, worsening azotemia in the face of fluid therapy or oliguria in the face of appropriate volume resuscitation, evidence of disseminated intravascular coagulation (DIC), refractory hypotension, increased total bilirubin, and ventricular arrhythmias. Hypoglycemia and PT prolongation at presentation were associated with death in another study. Other risk factors for death include creatinine > 1.5 mg/dL after 24 hours of hospitalization, delayed admission (>90 minutes), seizures and obesity. Number of nucleated red blood cells (nRBC) has also been shown to valuable at predicting death in heat stroke patients; >18 nRBC per 100 WBC at presentation had a sensitivity of 91% and specificity of 88% for predicting death in dogs. Mortality rates may be as high as 50% in dogs with heat stroke.

References
Veterinarians working in the ER and ICU are faced with treating hypotensive patients on a daily basis. Depending on the etiology of the hypotension, aggressive fluids resuscitation may not be an appropriate management strategy and the clinician must be familiar with vasopressor therapy options. Alternatively, vasopressors may be necessary in patients who have been maximally volume resuscitated and continue to be hypotensive and/or hypoperfused. Vasopressor agents’ pharmacology, indications, and complications will be discussed.

Blood pressure results from a tightly regulated balance of the neural inputs of the autonomic nervous system, cardiac function, peripheral vascular resistance (SVR) and endothelial signaling, and renal control of electrolytes and plasma volume. Sympathetic input, vasomotor tone, and intravascular volume, through their combined effects on heart rate, stroke volume (SV), and cardiac output (CO), together, determine blood pressure (BP) (BP = CO x SVR). Throughout the cardiac cycle, blood pressure varies resulting in systolic (SAP), diastolic (DAP) and mean arterial pressures (MAP), with the mean pressure being most closely related to diastole, as the majority of the cardiac cycle is in diastole. MAP can be calculated as MAP = ((SAP-DAP)/3) + DAP.

Canine normal blood pressure ranges from 131-150 mmHg (systolic) and 74-91 mmHg (diastolic) using direct, oscillometric, and Doppler technology. In cats, normal blood pressure ranges from 115-162 mmHg (systolic) and 74-91 mmHg (diastolic) using direct, oscillometric, and Doppler technology. In general, the average canine blood pressure is 133/75 mmHg (systolic/diastolic) and average feline blood pressure is 124/84 mmHg (Labato, 2009 and Labato, 2004). Hypotension is generally defined as MAP < 60 mmHg.

Methods commonly used in small animal clinical practice to measure blood pressure include both non-invasive and invasive technologies. For non-invasive blood pressure monitoring, oscillometric and Doppler ultrasonography are readily available. Doppler is particularly useful in small patients, cats, and those with cardiac arrhythmias. Oscillometric methods are convenient as they can be programmed to cycle at pre-determined intervals, such that repeated measurements can be obtained automatically. For both methods, cuff size selection in relation to limb diameter is essential for accurate results. Cuff diameter should be approximately 40% the limb circumference in dogs and 30% in cats. Cuffs that are too large will generate falsely low blood pressure results and falsely high results will be obtained from a cuff that is too small. In hypotensive patients, non-invasive methods have been shown to have the greatest variability as compared to direct measurements. Direct arterial blood pressure is considered the gold standard for blood pressure determination, and offers the additional benefits of continuous, real-time results that are accurate with arrhythmias and decreased perfusion. However, placement of an arterial catheter is technically challenging, especially in hypotensive patients and cats, uncomfortable for the patient during placement, and requires constant monitoring to ensure the catheter is not inadvertently removed. Care must also be taken to ensure that medications are not injected into the arterial catheter. In cats, necrosis of tissues distal to the arterial catheter is a concern and therefore arterial catheters should not be left in place in cats for more than 6-8 hours.

It is important to remember that hypotension is not a diagnosis or disease, but is instead a clinical manifestation of the underlying pathologic process(es). It is also important to remember that many patients with hypotension do not have a single cardiovascular dysfunction responsible for the decrease in blood pressure; their hypotension is often the result of a multitude of contributing factors. For example, a cat in septic shock is may be hypotensive from bradycardia, hypovolemia, and catecholamine deficiency or non-responsiveness resulting in vasodilation. Clinical signs recognized in most hypotensive patients correlate with end organ perfusion, and include decreased responsiveness, tachycardia, weak or poor pulse quality, cool extremities, pale mucous membranes with prolonged capillary refill time (CRT), and decreased rectal temperature.

Since hypovolemic shock is the most common cause of hypotension in veterinary patients, intravenous fluid therapy to provide intravascular volume resuscitation is the mainstay of treatment for many patients with hypotension. Improvement of intravascular volume status will improve stroke volume, which will in turn improve cardiac output and ultimately blood pressure if hypovolemia is the sole or largest contributor to hypotension (CO = HR x SV and BP = CO x SVR). Catecholamine therapy is rarely needed for patients with inadequate volume resuscitation unless there are other factors impacting blood pressure, such as general anesthesia. For patients with cardiogenic shock due to forward (left sided) or backward (right sided) cardiac pump failure or severe arrhythmias, fluid therapy to correct hypotension may be ineffective or even contraindicated. For these patients, improvement in heart rate, cardiac contractility and/or systemic vascular resistance may be needed to normalize blood pressure. For patients with hypotension that is refractory to fluid therapy, or in those where further volume resuscitation cannot be tolerated (as with fluid overload or severe cardiac dysfunction creating risk for fluid overload), vasoactive catecholamines or vasopressin can improve vascular tone and blood pressure, but may not normalize perfusion due to shunting and excessive vasoconstriction in certain tissues.
Catecholamines

Endogenous catecholamines (epinephrine, norepinephrine, and dopamine) are synthesized from tyrosine, and depending on location, can function as hormones or neurotransmitters. Dopamine is a precursor to norepinephrine. They act through stimulation of α- or β-receptors, and receptor type determines response initiated by the catecholamine. Individual catecholamines have variable effects on each receptor type, and depending on the outcome desired, different catecholamines may be preferable in certain situations.

**Dopamine**: Dopamine’s (Abbott Laboratories, Chicago, IL) target receptor and action is dose dependent. At low doses (1-4 mcg/kg/min), the drug predominantly affects D1 and D2 dopaminergic receptors, resulting in dilatation of renal, cerebral, coronary, and mesenteric vascular beds. The efficacy of its use for preservation of renal blood flow and diuresis at this dose range in renal failure is debated. At doses of 5-10 mcg/kg/min, the β-receptor effects of this drug predominate, resulting in improved inotropy and chronotropy. There is also some α-receptor stimulation within this dose range, though resultant vasoconstriction and increased systemic vascular resistance are less predictable. At higher doses of 10-20 mcg/kg/min the α-receptor effects are most prominent, resulting in potent vasoconstriction, which may compromise perfusion to the GI, peripheral, and renal circulations. Dopamine has a rapid onset and offset of action, with a plasma half-life of approximately 2 minutes, which is why constant rate infusion (CRI) is necessary. Its metabolism is through monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT) in the plasma, kidney, and liver. Side effects include nausea, vomiting, arrhythmias, hypotension, or hypertension. It can cause soft tissue injury and necrosis, so extreme care must be exercised to prevent extravasation. There are no strict guidelines for incremental dose increases or weaning of dopamine. In general, given the rapid onset of action, improvements in cardiac function or blood pressure should be seen quickly (within 5-10 minutes) at a given dose of dopamine. If insufficient improvement in cardiovascular parameters is seen, the dose is generally increased in increments of 10-25% until desired effect or maximum safe dose is reached, with the goal being to use to lowest effective dose. When cardiovascular parameters have stabilized, dopamine is generally weaned in 2-5 mcg/kg/min increments every 30 minutes to hour as tolerated by the patient’s blood pressure. Most vasopressors are less effective over time due to down regulation of adrenergic receptors and higher doses and/or additional vasoactive catecholamines may be needed.

**Dobutamine**: Dobutamine (Lilly, Indianapolis, IL) is more effective at therapeutic doses as a β1-receptor agonist than β2-receptor or α1-receptor agonist. It does not significantly alter systemic vascular resistance and does not contribute to norepinephrine release. It produces marked increase in cardiac ionotropy, and is therefore useful in patients with inadequate cardiac contractility. However, the increased contractility likely also increases myocardial oxygen demand, which may be detrimental in some patients, especially hypoxemic animals. Dobutamine has a half-life of approximately 2 minutes, is rapidly metabolized by the liver, and needs to be given as a CRI. Side effects secondary to dobutamine therapy include tachycardia, arrhythmias, and hypertension. In cats, seizures and tremors, particularly at higher doses, have been reported. For this reason, the dosing range for cats is 1-5 mcg/kg/min and the range for dogs is 2-20 mcg/kg/min. Due to the similar half-life and onset of action, the same considerations for dose escalation and weaning as used with dopamine can be used for dobutamine.

**Norepinephrine**: Norepinephrine (Teva, Irvine, CA) is primarily an α-receptor agonist with its main effect being increased vasoconstriction. It is able to provide significant vasoconstriction without compromising renal and cardiac blood flow, which makes it particularly attractive as a vital organ perfusion sparing vasopressor. It produces little effect on cardiac contractility and heart rate. The recommended dose is 0.05-2 mcg/kg/min. The same considerations for dose increase and weaning as with dobutamine and dopamine are used.

**Epinephrine**: Epinephrine (Abbott Laboratories, Chicago, IL) has both α- and β-receptor activity of similar magnitude at a given dose, and is most commonly used for cardiopulmonary resuscitation and anaphylaxis in veterinary medicine due to substantial side effects. Epinephrine causes increased cardiac inotropy and chronotropy, as well as increased systemic vascular resistance. Other effects include bronchial smooth muscle relaxation and increased glycogenolysis. After IV bolus, it can cause significant increase in blood pressure through vasoconstriction, as well as increased oxygen consumption at the capillary level, which can be deleterious with vasoconstriction that effects perfusion. It is rapidly absorbed when given intravenously, however, it can also be given subcutaneously (SQ) or intramuscularly (IM). It is metabolized in the liver and other tissues through MAO and COMT. Side effects include agitation, anxiety, hypertension, arrhythmias, and tissue necrosis at the site of repeated IM or SQ injections. The dose is 0.005-1 mcg/kg/min when used as a vasopressor CRI.

**Phenylephrine**: Phenylephrine (Baxter, Deerfield, IL) is primarily an α-receptor agonist with its main effect being powerful vasoconstriction which can result in reflex bradycardia. Vasoconstriction can be significant enough to decrease perfusion to the gastrointestinal, renal, and hepatic vasculature, however, coronary perfusion is often maintained or increased. It has a rapid onset of action, and is generally administered as a CRI even though its effects can persist for up to 20 minutes after injection. Side effects include agitation, bradycardia, arrhythmias, and skin necrosis in cases of vascular extravasation. The dose is 1-3 mcg/kg/min.

**Isoproterenol**: Isoproterenol (Abbott Laboratories, Chicago, IL) is reserved for cases of third degree AV block, as it causes increased conduction rate through the SA and AV nodes. It is a non-specific β-agonist that causes both improved contractility and heart rate through increased cAMP production. It has little, if any, α-receptor effects. Side effects include bronchial relaxation, arrhythmias, tremors, anxiety, weakness, and vomiting. The dose is 0.04-0.08 mcg/kg/min.
**Vasopressin**

Vasopressin, which is also known as anti-diuretic hormone (ADH), is a peptide synthesized in the pituitary that has a variety of systemic effects. With respect to vascular tone, vasopressin binds receptors on vascular smooth muscle, and depending on the dose, causes vasodilatation (low doses) or vasoconstriction (high doses). Vasopressin stores can become depleted with prolonged shock or sepsis resulting in vasoplegia despite intravenous fluid and vasoactive catecholamine therapy. Vasopressin deficiency has been documented in people with refractory hypotension, and positive benefit has been shown with the addition of intravenous administration of vasopressin. The use of vasopressin has also been described in 5 dogs with dopamine-resistant hypotension secondary to vasodilatory shock and shown to improve blood pressure without causing arrhythmias. Clinical experience in cats is lacking. It is not bound to plasma proteins, and is rapidly degraded in the liver and kidneys. The dose used in dogs is extrapolated from human doses and is 0.5-2.0 mU/kg/min IV CRI. There is no established dose in cats, however, similar doses as used in dogs have been used in cats.

When hypotensive patients fail to respond to fluid therapy to improve perfusion and hemodynamic status, or in patients intolerant of or for whom intravenous fluid therapy is contraindicated, vasopressors are often required to improve blood pressure. It is important to know whether enhancing α- or β-receptor response is preferred for a given patient, depending on whether cardiac contractility or vascular tone needs improvement, as this will impact the decision about which pressor and dose. Vasopressor agents can be associated with serious side effects, and patients receiving these medications require intensive monitoring.

**References**

Tracheal collapse is a common, frustrating disease process in small breed dogs. Signalment, history, and physical examination findings can support a tentative diagnosis of tracheal collapse but localization of disease along the trachea and lower airways may require pointed client questions and diagnostic imaging. Once the extent and severity of disease is understood, medical management is attempted in many cases except those so severely affected that discharge without intervention is not possible. Tracheal stenting has become a popular, non-invasive method for treating tracheal collapse in dogs. Initial experience with tracheal stenting was met with significant complications, leading to it being branded a salvage procedure. Design enhancements, progress in sizing protocols, and improved patient selection criteria has increased the success of their use.

**History and physical exam**

In all dogs suspected to have tracheal collapse, specific questions that should be directed to the client to help better understand the localization of disease and severity include:

1. Duration and progression of clinical signs
2. Nature of cough (honking, dry, moist, soft, expiratory/pushing)
3. How frequent are events and how long do they last
4. Has there ever been respiratory difficulty or distress during or after an event
5. Does a coughing event stop activity, or is the dog able to continue activity despite coughing
6. What are the triggers for a coughing event
7. Does the dog snore and can it sleep through the night
8. Is there a seasonal component to the coughing events
9. What is the home environment like (is there smoking, air conditioning, air fresheners, etc.)

Visual assessment of patient breathing at rest and during a coughing event to evaluate for nature of the cough, prolongation of inspiratory or expiratory phase of respiration, increased respiratory effort, abdominal push on expiration, and herniation of the cranial lung lobes out of the thoracic inlet on expiration should be performed first. Auditory assessment from a distance should assess for nature of abnormal respiratory noises (honking, high pitched, wheezing moist, stertor or stridor). The larynx, trachea, and entire thorax should be carefully ausculted with simultaneous observation of respiratory phase for air movement, fluid sounds, and crackles. The presence of an inducible cough on tracheal palpation is not pathognomonic for tracheal collapse as aggressive palpation can induce patients with normal tracheas to cough and patients with both cervical and intrathoracic collapse may not cough on tracheal palpation.

In animals with tracheal collapse, their diseased airway is not able to withstand deformation due to dynamic airway pressure changes during respiration. In general, patients with cervical tracheal collapse have clinical signs upon inspiration whereas those with intrathoracic collapse are affected on exhalation. Patients with thoracic inlet collapse can have signs during both or either phase of respiration. Patients with mainstem bronchial collapse are affected mostly during expiration.

The remainder of a complete history and physical exam are essential for ensuring there is not another disease process contributing to the cough and to assess for other disease processes which may exacerbate tracheal collapse signs (obesity, endocrinopathies such as hyperadrenalcorticism, chronic lower airway disease, valvular heart disease, etc.)

The ultimate goal of a thorough history and physical examination is to determine the primary source of clinical signs, as many dogs will have a combination of nasopharyngeal, tracheal and mainstem bronchial collapse. Honking dogs with airway obstruction and respiratory compromise need to be differentiated from the chronic coughing patient with no respiratory or activity compromise whom are mainly affected by bronchial collapse as the treatment and intervention recommendations are different in these two groups of patients.

**Diagnostics**

Thoracic radiographs are an important first line diagnostic in patients with suspected tracheal collapse. They are essential to rule out concurrent bronchopneumonia, bronchiectasis, lower airway disease, cardiomegaly, and to assess pulmonary vasculature size. The presence of tracheal collapse on films does not determine severity, the absence of collapse on films does not preclude a diagnosis of tracheal collapse, and thoracic radiographs cannot determine the dynamic nature of the collapse. Paired inspiratory and expiratory thoracic radiographs improves their utility, but can still underestimate severity and extent of disease. Radiographs misdiagnosed the location of tracheal collapse in 44% of dogs and failed to diagnose tracheal collapse in 8% of dogs when compared to fluoroscopy. Thoracic radiographs may not give a complete assessment of mainstem bronchial disease and nasopharyngeal collapse.
Tracheal fluoroscopy is an extremely useful diagnostic for thoroughly assessing the dynamic nature nasopharyngeal, tracheal, and bronchial collapse. It allows for real time understanding of the extent of tracheal collapse during all phases of respiration and during coughing in awake patients. It is especially helpful for determining the presence and extent of mainstem bronchial collapse.

Computed tomography (CT) can be very helpful in dogs with tracheal disease, especially in cases where a tracheal mass is a differential for the cause of clinical signs and when there is concern for severe tracheal collapse leading to ventral tracheal cartilage inversion (tracheal malformation). Unfortunately, dynamic disease processes like nasopharyngeal, laryngeal, and tracheal collapse will not be seen on sedated or anesthetized CT examinations. Studies evaluating a clear plastic patient positioning device (MouseTrap™) that restricts movement have shown promising results for dynamic CT evaluation of upper airway obstruction secondary to laryngeal, tracheal and bronchial disease without the need for anesthesia or sedation. Recent studies evaluating the utility of CT for tracheal diameter measurements to determine tracheal stent sizing show promise for improved measurement accuracy when compared to lateral radiographic or fluoroscopic measurements alone.

Endotracheal washes for cytology and aerobic should be performed in all dogs undergoing intubation for tracheal collapse diagnostics or definitive intervention. Dogs with tracheal collapse likely have abnormal airway clearance mechanisms due to their tracheal disease, so it is possible to have positive airway cultures without radiographic evidence of pneumonia. This is especially important for dogs undergoing stenting since they are managed post-operatively with steroids and cough suppressants, which could exacerbate infection, especially in the presence of a permanent implant. Airway culture and sensitivity is also important in dogs who may have been treated with multiple antibiotics while being medically managed for their tracheal collapse to guide therapy.

Trachoscopy is a very useful diagnostic in tracheal collapse patients, however, small patient size may mean that some patients must be extubated for trachoscopy, which can make anesthesia more difficult and dangerous in patients with compromised airways. Trachoscopy allows for direct visualization of the entire airway and for bronchoscopy if indicated. In patients with suspected or confirmed tracheal collapse where trachoscopy is being used for staging of collapse and complete airway assessment, the clinician must be prepared for intervention (prolonged intubation, prosthetic ring placement, or endoluminal stenting) should the patient be unable to be extubated due to their disease and/or iatrogenic irritation induced from trachoscopy. Therefore, in most cases of tracheal collapse, trachoscopy is reserved for immediate airway assessment before intervention, and in the case of tracheal stenting, immediately post-stent placement to assess position and mucosal contact before recovering the patient from anesthesia.

Medical management
Medical therapy is a mainstay of tracheal collapse management and efforts should be made to attempt medical therapies before surgical or interventional options are pursued. Oftentimes, “breaking the cycle” of dyspnea, distress, and anxiety with sedation, oxygen, cough suppression, and possibly corticosteroids is adequate to control tracheal collapse symptoms enough to permit discharge with medical management institution or adjustment or referral for further surgical or interventional care. However there are cases where respiratory distress cannot be controlled or there is significant patient compromise and immediate relief of the airway obstruction is needed. Prior recommendations that medical management should be “exhausted” before intervening with extra-luminal rings or a stent are falling out of favor due to the negative impacts of long term steroids and improved outcomes with stents.

In the emergency setting when tracheal collapse patients are dyspnic, sedation, anxiolytics, and oxygen are essential. Butorphanol (0.1-0.4 mg/kg IV or IM q2-4 hr) and acepromazine (0.005-0.01 mg/kg IV, IM, or SQ q4-6 hr) are effective initial anxiolytic and sedative options. Acepromazine should only be used patients for whom cardiovascular compromise is not a concern. The use of an oxygen cage where the percentage of inspired oxygen can be adjusted as dictated by the patient’s comfort is also very helpful during emergency stabilization. Corticosteroids may also be considered if there is concern for airway edema and inflammation secondary to respiratory distress and increased work of breathing. Dexamethasone sodium phosphate (0.05-0.1 mg/kg IV q12-24hr) is typically used in these instances.

Hydrocodone and butorphanol are effective antitussives, though it is imperative to educate owners that they better at preventing coughing than stopping an episode, so they should be used regularly in the beginning phases of medical therapy and in some patients, lifelong. Dosing is flexible (0.25-0.5 mg/kg PO q6-12hr) and dependent on patient response and will likely need to be increased over time should tolerance develop. Hydrocodone is available in tablets and an elixir, with the elixir allowing for smaller dose increment changes and titration by the owners. If a prescription for hydrocodone is filled at a human pharmacy ensure that the medication is hydrocodone/homatropine, and not hydrocodone/acetaminophen, which is an additional formulation available for use in people.

The use of corticosteroids such as prednisone in the long-term management of tracheal collapse is controversial. In patients recovering from an episode of dyspnea, a tapering course of anti-inflammatory steroids may be necessary to control the airway inflammation that results from cycling of the collapsing trachea during coughing and edema from increased work of breathing. However, when cough is controlled, airway inflammation from tracheal collapse should be minimal and may not require steroids long term. In addition, long-term steroids contribute to muscle weakness, weight gain and predispose to the development of pulmonary thromboemboli; all of which may exacerbate respiratory signs associated with tracheal collapse. Inhaled steroids such as fluticasone
have been shown to be beneficial in tracheal collapse dogs that present in respiratory crisis that is nonresponsive to medical stabilization. The use of bronchodilators is also controversial in managing dogs with tracheal collapse. Bronchodilators are generally reserved for dogs with confirmed bronchial collapse or concurrent lower airway disease. Methylxanthine derivatives, such as theophylline (10-20 mg/kg PO q12) or aminophylline (5-10 mg/kg IM, IV, PO q8hr) may be preferable to B2 agonists such as terbutaline or albuterol due to risk of tachycardia with sympathomimetic agents. When oral theophylline is used, the extended release formulation is preferred. Theophylline and aminophylline have multiple drug interactions, so drug compatibility must be confirmed before initiating therapy. Inhaled bronchodilators have also been used with some success as a rescue drug during coughing or long term in dogs with mainstem bronchial collapse.

**Tracheal stenting**

Tracheal stenting can be performed in dogs with both intra-thoracic and extra-thoracic tracheal collapse as well as those with cervical collapse that are deemed poor surgical candidates for extra-luminal rings or for those whom clients are unwilling to accept the complications associated with tracheal ring placement. Since tracheal stenting can be performed quickly and non-invasively, it has has been shown to be beneficial in tracheal collapse dogs that present in respiratory crisis that is nonresponsive to medical stabilization. Additionally, dogs who have required hospitalization for a respiratory crisis and whose clinical signs are consistent with airway obstruction can also be considered candidates for stenting after appropriate client counseling about the pros and cons of intervention.

Complications when balloon expandable and human biliary wall stents were used in research dogs and clinical tracheal collapse patients, such as foreshortening, migration, fracture, and excessive airway irritation, led to labeling tracheal stenting as a salvage procedure only, and hence the recommendation to exhaust medical management before considering stenting. Stents designed specifically for the canine trachea (http://infinitimedical.com) have gone through multiple design enhancements to improve their sizing and placement predictability and risk of fracture. Complications seen with tracheal stenting are believed to be greatly reduced when precise sizing is performed. Oversized stents that do not fully expand are at increased risk of fracture since stents are strongest when fully expanded. Alternatively, undersized stents are at risk of poor incorporation into the tracheal mucosa, leading to mucous accumulation, inflammation, and likely infection. Unequal tracheal mucosal contact, in conjunction with airway inflammation and infection, may also lead to granulation/inflammatory tissue formation but objective data correlating these circumstances is lacking.

Current sizing recommendations include positive pressure breath holds to 20 cmH2O to determine maximal tracheal diameter using digital radiography or fluoroscopy. Stent size is chosen by selecting a stent with a diameter that is 10-20% larger than the maximal tracheal diameter measurement obtained. In some cases, the tracheal diameter is not uniform along its length, with the most common scenario being a larger cervical and cranial thoracic inlet diameter compared to the intrathoracic tracheal diameter. Stent choice in these cases mandates that either the cervical trachea is undersized to accommodate the intrathoracic trachea, or the thoracic trachea is oversized to accommodate the cervical trachea when using traditional tubular stents. Since accurate sizing is thought to be paramount in the success of tracheal stenting, a self-expanding, nitinol stent with a tapering diameter where the cervical tracheal diameter stent portion is larger than the intrathoracic portion was designed. Initial clinical case experience with the tapered tracheal stent (VetStent Duality, http://infinitimedical.com) has been very promising.

Since measurements are performed under general anesthesia at the time of stenting, multiple sizes of tracheal stents should be readily available so that the appropriate size can be placed without having to attempt to recover a compromised patient. Tracheal stents are placed through an endotracheal tube and when a bronchoscope adapter is used, the patient can continue to have oxygen insufflation during stent positioning and deployment.

Patients are often discharged the day following stent placement, unless there is concurrent pneumonia requiring prolonged hospital care. All tracheal stent patients have thoracic radiographs taken prior to discharge. If no pneumonia is present, the first recheck examination is generally one month after stent placement. If pneumonia is present prior to discharge, but patients are only mildly affected and able to be discharged for care at home, recheck radiographs are usually performed 7-10 days post-operatively to ensure appropriate resolution of pneumonia. Patients are discharged with antibiotics (10-15 mg/kg enrofloxacin PO q24) pending airway culture, a 2-3 weeks tapering course of steroids, and regular (q6-8 hour) cough suppression. A short, dry, self-limiting cough is to be expected for 4-6 weeks post-stent placement and is something about which clients should be educated pre-operatively.

Long-term routine thoracic radiography monitoring is important to be able to detect migration, early fracture or the development of inflammatory tissue. For the first year post-operatively, radiographs are checked every 3-4 months. For every year after that, radiographs are taken every 6 months. If at any point post-operatively, there is a change in the patient’s cough or respiratory comfort, repeat radiographs are taken immediately. If radiographs and/or tracheal fluoroscopy do not reveal the explanation for changes in coughing and respiratory comfort, repeat endotracheal wash and tracheoscopy is indicated. Since tracheal stents are permanent implants, stent fracture causing clinical signs of airway obstruction and coughing can only be managed by placement of an additional stent within the original stent. Very limited experience with obstructive intra-luminal granulation tissue has also shown promising response to immunosuppressive steroid therapy, culture-guided antibiotic therapy, and in some cases, repeat stenting.
References

Don’t see emergency cases every day? Have a dog presenting to you with pale mucous membranes, a weak pulse, a heart rate of 190 bpm, and you’re not sure what to do next? Have a dyspneic cat fish-mouth breathing in front of you? This article discusses how to avoid 10 common errors in emergency patients that will save your patient’s life, including when to tap that dyspneic cat’s chest, when to reach for that “FAST” ultrasound, or the best time to do chest radiographs. Having practiced in the trenches of a busy inner-city emergency room to the ivory tower of academia, I’ve seen these mistakes made, and I’ve made them myself. Here, some common mistakes to avoid in the emergency room.

Not doing chest radiographs
One of the most common mistakes in the emergency room is not performing chest radiographs (a “met check”) as part of routine geriatric diagnostics. Geriatric patients (defined as a dog > 6–7 years of age [size-dependent] or a cat > 12 years of age) with, for example, hepatosplenomegaly, icterus, hemoabdomen, immune-mediated disease, or fever of unknown origin should have chest radiographs done at the same time as abdominal radiographs. Typically, a three-view chest set is the method of choice; however, this may be difficult in emergency patients with dyspnea. That said, a right- and left-lateral chest radiograph is also an effective way to screen for metastasis. While a met check is often a “low-yield test” (i.e., the likelihood of identifying chest metastasis is relatively low), it is an important screening tool that can help veterinarians counsel pet owners on end-of-life decision-making and overall prognosis.

Using the shock dose of fluids
The “shock dose” of fluids is extrapolated from the blood volume (60–90 ml/kg for dogs; 60 ml/kg for cats). More recently, emergency critical care specialists have moved away from using the entire shock dose when trying to stabilize hypovolemic patients—smaller aliquots (e.g., one-quarter to one-third of a shock dose) of intravenous (IV) crystalloids are preferred. A patient rarely requires replacement of the whole blood volume with crystalloid fluids.

Using the wrong dose of steroids
Traditionally, “shock doses” of steroids have been listed in emergency books (e.g., dexamethasone sodium phosphate [DexSP] 4–6 mg/kg). However, criticalists have moved away from giving steroids with trauma because of potential deleterious effects (including gastric ulceration in a poorly perfused “shock gut” in the dog, exacerbation of hyperglycemia, and delayed wound healing). More recently, we have moved to different doses of DexSP. Antiinflammatory doses of DexSP are generally considered 0.1 mg/kg, whereas immunosuppressive doses are as low as 0.25 mg/kg IV q 12 to 24 hours. For that reason, the 4-6 mg/kg dose for shock is no longer indicated. Remember that DexSP is approximately 8 to 15 times stronger than prednisone, and one is unlikely to need 40 mg/kg of prednisone in trauma cases.

Clinical application
1. For cases warranting immunosuppression (e.g., immune-mediated hemolytic anemia), consider using lower doses of DexSP (0.25 mg/kg IV q 12–24 hours).
2. Avoid the “shock doses” of steroids—if you are giving more than a few milliliters, it’s probably too much.
3. Concurrent use of nonsteroidal antiinflammatory drugs and steroids should still be avoided to minimize GI effects.

Giving steroids to head trauma patients
Recently, the use of steroids in both human and veterinary head trauma has been widely debated. Although research in this topic is voluminous, there are no experimental or clinical studies demonstrating a clear benefit of steroids in head trauma. In one human study, a meta-analysis of randomized, controlled trials did not show a beneficial response from steroid therapy (1). Unfortunately, steroids have been associated with the following deleterious side effects: gastrointestinal (GI) bleeding, hyperglycemia, immunosuppression, delayed wound healing, and perpetuation of a catabolic state. Currently, the brain trauma foundation guidelines state that glucocorticoids are “not recommended for improving outcome or reducing intracranial pressure in head-injured patients” (2). The “CRASH” (Corticosteroid Randomisation After Significant Head injury) study demonstrated that overall mortality was statistically higher in patients who were treated with steroids (3). Recent studies have shown that human patients with head trauma and hyperglycemia have a poorer return to cognitive function than do euglycemic patients. Why is hyperglycemia dangerous in head trauma, or in any case of brain ischemia? Unfortunately, elevated glucose concentrations provide a substrate for anaerobic metabolism and glycolysis in the brain, worsening brain perfusion via the accumulation of the by-product, lactic acid. Hyperglycemia is also associated with proconvulsant effects, which are due to
increased neuronal excitability. In a veterinary study by Syring and coworkers, 52 dogs and 70 cats with head trauma were compared with 122 age- and species-matched control dogs and cats. Severity of head trauma was classified as mild, moderate, or severe, and blood glucose concentrations were recorded within 1 hour after admission. The study found that the blood glucose concentrations were significantly associated with severity of head trauma in dogs and cats and were significantly higher in dogs and cats with head trauma than in the control animals. However, blood glucose concentration was not associated with outcome, which is divergent from human studies. This veterinary study may also differ from human medicine in that overall cognitive function varies between humans and dogs/cats. These studies reiterate that iatrogenic hyperglycemia must be avoided in patients with head trauma or cerebral ischemia and that severe hyperglycemia in head trauma should potentially be treated with regular insulin therapy if warranted and persistent. When in doubt, withhold steroid therapy in head trauma patients to prevent hyperglycemia and other detrimental effects. Instead, osmotic agents such as mannitol have been found to be helpful in decreasing intracranial pressure (ICP).

If IV fluid resuscitation alone does not reduce glucose levels in hyperglycemic patients with head trauma, a low dose of regular insulin (0.2 U/kg, intramuscular) may be given every 3 to 4 hours for the first few hours to help lower blood glucose. Blood glucose levels should be monitored frequently to ensure improvement and to preclude hypoglycemia, which would further complicate neurologic monitoring.

Therapies other than steroids to consider in head trauma patients include:
- Aggressive fluid resuscitation to help normalize or maintain blood pressure and maximize perfusion
- Oxygen therapy
- 15- to 30-degree head elevation (to lower ICP)
- Minimal jugular restraint or pressure (to prevent increased ICP)
- Tight glycemic control

Not assessing your patient more frequently with simple tests
In veterinary medicine, the temperature, pulse rate, respiratory rate, and weight are typically evaluated during the initial presentation. These simple, inexpensive physical examination parameters are an important part of serial assessment and often provide clues on hydration status, disease process, and response to treatment.

Temperature
When examining a hyper- or hypothermic patient, differentiate between exogenous and endogenous sources. Hyperthermia is typically caused by an exogenous heat source (e.g., sun exposure, humidity, locked inside a car, upper airway obstruction resulting in lack of ability to thermoregulate). This is semantically different from fever, which is caused by an endogenous heat source (e.g., neoplasia, inflammatory cytokines). With hyperthermia, patients should be cooled by using cold water baths, cold IV fluids, fans, and relieving the upper airway obstruction. Patients should only be cooled to 103.5°F (39.7°C) to prevent severe rebound hypothermia. The use of “fever-breaking” medications (e.g., dipyrone) is not indicated, as resetting of the hypothalamus may have already occurred. Patients with fever should not undergo cooling methods, as the fever is a physiologic response to an underlying pathology (e.g., viruses, bacteria). Three key differential diagnostics should be considered with fever:
1. Infection
2. Inflammation
3. Neoplasia

For hypothermia, it is important to determine whether it is due to an exogenous source (e.g., living in a cold environment with inadequate shelter, hair coat, or underlying hypothyroidism) or an endogenous one. Hypothermic patients should be warmed passively (e.g., blankets, concurrent warm IV fluids) and slowly. With hypothermic patients, it is important not to rapidly warm patients via surface warming alone (e.g., BAIR hugger), particularly if they are hypotensive, as rewarming can result in peripheral vasodilation. During states of poor perfusion or hypotension, patients should physiologically vasoconstrict peripheral blood flow to direct blood to more important organs—the heart and lungs. Rapid surface rewarming of hypothermic patients without adequate IV fluid replacement can result in inappropriate shunting of blood.

Weight
Weight is often underutilized as a means of assessing hydration. Because we can calculate dehydration (kg weight X % dehydration), we can also estimate appropriate weight gain, as 1 liter = 1 kg. Patients should be weighed daily while hospitalized, ideally on the same scale. This is important because it is an easy way to evaluate hydration and appropriate (or inappropriate) weight gain. For patients in which volume–fluid balance is tenuous (e.g., acute renal failure with anuria, congestive heart failure, volume overload), weight should be evaluated every 6 to 8 hours.

For example, if you determine that a 30 kg dog is 10% dehydrated, the amount of fluid required to hydrate him is:

Calculated dehydration: 30 kg X 0.1 (percent dehydration) X 1000 mls = 3000 ml = 3 L
In other words, a 30 kg dog needs 3L of IV crystalloids for rehydration alone and thus should weigh 33 kg after hydration (in 8–12 hours, depending on patient stability). If that same dog weighs 32 kg by the next day, he may still be inadequately hydrated. Likewise,
if the patient weighs 34.8 kg the next day, he may be overhydrated, volume overloaded, and retaining water inappropriately (e.g., acute renal failure).

**Pulse quality**
Assessing pulse quality frequently is imperative in unstable, shocky emergency patients. Palpating the femoral pulse enables assessment of pulse quality, which is the difference between the systolic and diastolic pressures. Pulse palpation, quality, and duration are a gross estimate of blood pressure and, indirectly, stroke volume. In a normal healthy animal, the pulses should be strong and synchronous, with a palpable pulse for each heart beat (therefore, make sure that you are simultaneously ausculting your patient and palpating for femoral pulses). A palpable femoral pulse is consistent with systolic blood pressure of at least 60 mm Hg. Poor femoral pulses typically indicate profound hypotension and should be treated aggressively and appropriately. A palpable dorsal metatarsal pulse is consistent with a systolic blood pressure of at least 90 mm Hg, and can be used as a basic “poor man’s Dinamap,” particularly during volume resuscitation.

Patients with systolic blood pressure < 90 mm Hg should be treated with IV fluids (if hypovolemic) and vasopressors (once adequately volume resuscitated) if evidence of shock (e.g., hypovolemic, septic, hyperdynamic, hemorrhagic) is present (provided cardiovascular shock has been ruled out as a differential). Patients with systolic blood pressure > 180 mm Hg (normal, 120 mm Hg) should be treated with antihypertensives, such as hydralazine, nitroprusside, amlodipine or enalapril, to minimize secondary complications from hypertension, such as detached retinas, cardiovascular and renal effects, and ischemic events. Frequent monitoring of blood pressure is imperative to ensure adequate care.

Serial physical examination is imperative to adequately evaluate a patient’s hydration status—checking for return of skin turgor, appropriate weight gain, and moisture of mucous membranes. However, physical examination findings are subjective, and <5% dehydration is subjective and difficult to assess on physical examination. The concurrent use of evaluation of PCV/TS, blood glucose, blood urea nitrogen (BUN or AZO) weight, UOP, and urine specific gravity (USG), and thirst can be used in conjunction with physical examination findings to better assess hydration status.

**Packed cell volume/total solids, blood glucose, and blood, urea, nitrogen (BUN/AZO) (“Big 4”)**
Patients on IV fluids should have a minimum database (including PCV/TS and blood glucose) measured daily, along with basic electrolytes to make sure Na⁺ and K⁺ are normal. Because patients often experience hemoconcentration when they are dehydrated (e.g., PCV/TS 55%/7.8 g/dl [78 g/L]), the goal of fluid therapy is to ensure that these numbers improve with appropriate therapy (consistent with hemodilution). Ideally, the PCV/TS in a normal, systemically healthy patient on IV fluids at sea level should be 35%/5.0 g/dl (50 g/L). In fact, oxygen delivery is maximal at such a “hemodilute” PCV/TS, as there is less viscosity of red blood cells and “sludginess.” We can still evaluate the PCV/TS in abnormal, metabolically inappropriate patients. Classically, a 10% to 12% dehydrated, cachectic, geriatric cat with chronic renal failure may present to you with a PCV/TS of 28%/7 g/dl (70 g/L). Once that patient is adequately hydrated, the PCV/TS may decrease to 20%/7 g/dl (70 g/L), unmasking the anemia from lack of erythropoietin.

**Urine specific gravity (USG)**
USG can be evaluated in patients on IV fluids to help assess hydration status. Ideally, USG should be measured before fluid administration to allow for evaluation of renal function. Dehydrated patients with concentrated urine demonstrate adequate renal function (cat > 1.040, dog > 1.025)—in other words, the kidneys are working and trying to absorb as much water from the urine as possible. Once started on IV fluids, normal, systemically healthy patients should have isosthenuric urine. Patients on IV fluids for > 6 to 12 hours should have adequate dilution of USG, and the ultimate goal of fluid therapy and adequate hydration should be USG of 1.015 to 1.018 on IV fluids. Patients on IV fluids with USG > 1.020 are still likely dehydrated and should be treated more aggressively with IV fluids if other parameters of dehydration persist (e.g., hemoconcentration). Hydration can be determined by assessing the color, volume, and USG of urine. A patient that is still dehydrated while hospitalized on IV fluids may have decreased UOP and dark-yellow urine (provided, for example, that no pigmentation, myoglobinuria, or bilirubinuria are present). This is a result of antidiuretic hormone release and renin-angiotensin stimulation, resulting in maximum absorption of free water and sodium.

**Urine output (UOP)**
UOP should be monitored carefully, particularly in azotemic patients. Fluid therapy should be directed toward achieving normal UOP (i.e., 1–2 ml/kg/hour). Again, one can assess the hydration status of the patient by evaluating the volume and USG of urine. Excessive urination with dilute, clear urine may indicate copious or excessive IV fluid therapy, whereas hypersentruria may suggest ongoing dehydration, and aggressive fluid resuscitation may be further warranted. If UOP is decreased (particularly in azotemic patients), fluid therapy and vasopressor support (to increase renal blood flow) should be initiated to prevent anuria (< 0.5 ml/kg/hour) or oliguria (< 1 ml/kg/hour). If UOP is decreasing and renal function is normal (based on creatinine, BUN, and pre–fluid therapy USG), the patient should be reassessed for hydration status, and fluid therapy adjusted as indicated. Classically, a cat with urethral obstruction may have a profound postobstructive diuresis. A sudden decrease in UOP should elicit assessment for reobstruction. If no obstruction is found, USG should be remeasured. If hypersentruria is found (>1.025 on IV fluids), decreased UOP is likely due to continued dehydration from a postobstructive diuresis— the patient is attempting to absorb as much free water as possible from the kidneys, resulting in decreased UOP. In this example, the IV fluid rate should be increased.
- Normal UOP: 1–2 ml/kg/hour
- Oliguria: 0.5–1 ml/kg/hour
- Anuria: < 0.5 ml/kg/hour

Note that underlying diseases such as postobstructive diuresis (posturethral obstruction); diabetes mellitus (with secondary osmotic diuresis due to glucosuria); hyperthyroidism (increased glomerular filtration rate due to increased metabolic rate); and chronic renal failure (inability to adequately concentrate and absorb water) may result in dramatic water losses through the kidneys, and these patients may need a higher rate of fluids to compensate for ongoing losses. Likewise, these disease processes prevent us from differentiating renal versus prerenal disease on the basis of USG alone, as these patients have isosthenuria due to metabolic disease. Regardless, appropriate fluid therapy and urine monitoring (e.g., “measuring ins and outs”) may be necessary, particularly in azotemic, oliguric renal failure.

**A water bowl**

Any hospitalized animal should always have access to fresh, clean water unless it is contraindicated due to vomiting, pancreatitis, fasting for anesthesia or sedation, or to maximize mannitol or furosemide effects (fasted for 20 minutes only). If a hospitalized patient on IV fluids continues to drink water in front of you, you should be concerned that the patient is still dehydrated. Due to the timidity of cats, they often will not drink water when stressed and hospitalized. If a dog or cat drinks in your presence, that patient is probably still dehydrated, and their thirst mechanism continues to be stimulated in an attempt to hydrate. Take that as a hint that your patient is trying to tell you to increase the fluid rate! Rare situations when hydration status cannot be based on the thirst mechanism include diabetes insipidus and psychogenic polydypsia.

**Not using enough SQ fluids**

We often use SQ fluids in veterinary outpatient medicine to help hydrate a patient. Because fluids are so slowly absorbed when given in this manner, SQ administration is not appropriate for hypovolemic or severely dehydrated patients. SQ fluids are ideally utilized for outpatient medicine (e.g., the vomiting patient that needs to be fasted overnight but still needs to maintain hydration). But just how much fluid can you give SQ? The calculation for how many ml/kg to give SQ is typically *maintenance fluids*. We do not adjust for dehydration or ongoing losses with SQ fluids.

**Example:**
- 5-kg, male castrated cat presents for 4 episodes of vomiting
  - Physical examination: no string on oral examination, nonpainful abdomen
  - Amount of SQ fluids to potentially give: 5 kg x 60 ml/kg/day = 300 ml SQ

- 40 kg, female spayed Labrador presents for 3 vomiting episodes in 12 hours after ingesting garbage
  - Physical examination: nonpainful abdomen; abdominal radiographs: no significant findings, no obstruction, but some fluid-filled loops of intestine
  - Amount to give: 40 kg x 50 ml/kg/day = 2000 ml SQ

Giving too small an amount of SQ fluids often does not benefit the patient. Having owners give < 50 ml/adult cat for SQ fluids is often not aggressive enough (not worth the needle poke!). That said, if a patient has a heart murmur (particularly in cats), this maintenance amount should be *reduced* to prevent volume overload.

**Not doing enough fast (focused assessment with sonography for trauma) ultrasounds**

The focused assessment with sonography for trauma (FAST) ultrasound is a 2-minute procedure that detects the presence of fluid in the abdominal cavity to allow for rapid therapeutic intervention (e.g., fluid resuscitation, abdominocectesis, cytology, clinicopathologic testing) (6). This has also been modified for the pleural (T-FAST) and pericardial space. This rapid method of ultrasound is designed to be used by health care professionals with limited ultrasonographic training and is not designed for extensive examination of the abdomen. The added benefit of the FAST examination is the ability to detect very small amounts of fluid. Typically, 5 to 25 ml/kg of fluid needs to be present to be removed by blind abdominocectesis; > 10 to 20 ml/kg of fluid has to be present before it can be detected by fluid-wave assessment on physical examination; and approximately 8.8 ml/kg of fluid needs to be present before it can be detected radiographically. On the contrary, as little as 2 ml/kg of fluid can be detected on a FAST examination, allowing for rapid diagnosis and identification of underlying pathology.

The FAST examination typically involves assessment of 4 sites of the abdomen: caudal to the xiphoid, cranial to the bladder, and the right and left dependent flank (6). The presence of fluid at any of the sites is considered positive. Evaluation of the xiphoid region allows you to check for fluid between the liver and diaphragm and the liver lobes, as well as for pericardial or pleural effusion (6). Evaluation of the bladder view evaluates for fluid cranial to the bladder and for the presence of a bladder (6). The right dependent flank allows for fluid detection between the intestines and the body wall, whereas the left dependent flank view allows for identification of the spleen, abdominal effusion near the spleen and body wall, the kidney and spleen, and the liver and spleen (6).
Reluctance to penetrate body cavities
The use of abdominocentesis or thoracocentesis is a benign procedure that is both diagnostic and therapeutic. Referring a stressed, hypoxemic, frantic, dyspneic cat with 300 ml of pleural effusion for a 1-hour car ride to a specialist can easily result in the cat’s demise. Shaving and surgically preparing a wide area near the umbilicus (abdominocentesis) or thorax (thoracocentesis) should be done quickly but aseptically. For the thorax, thoracocentesis should be performed either dorsally (for air) or ventrally (for effusion) at the 7th to 9th intercostal space (ICS). Likewise, an imaginary line can be drawn from the end of the xiphoid to the lateral body wall, which is approximately the 8th ICS. This will allow for rapid identification of where to perform an emergency thoracocentesis. Pericardiocentesis should be performed on the right side at the region of the 3rd to 5th ICS at the point of the flexed elbow. Abdominocentesis should be aseptically performed via a four-quadrant tap in the periumbilical region. The use of a 3-way stopcock, 20- to 60-ml syringe, extension tubing, and appropriately sized needles dependent on patient size and volume of effusate (usually 20–22 gauge for cats and 16–22 gauge for dogs) is indicated.

Conclusion
Veterinarians should avoid these key, common mistakes in emergency medicine. By avoiding these errors, the overall quality of care and survival of the emergency patient may improve. Simple, easy monitoring tools (e.g., Big 4, pulse quality, weight) can be used to more carefully monitor our critically ill patients in a cost-effective, simple, repeatable manner.

References

NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as Plumb's Veterinary Drug Handbook.
Gastric dilatation-volvulus (GDV), often known as “bloat” by the layperson, is a life-threatening syndrome seen most commonly in large or giant breed dogs. Immediate recognition, aggressive volume resuscitation, medical stabilization, and surgical intervention are required for best outcome. Life-threatening complications can occur – even with treatment – and include cardiac arrhythmias, sepsis, disseminated intravascular coagulation (DIC), peritonitis, multi-organ dysfunction syndrome (MODS), and death.1

Signalment
Certain breeds are over-represented or predisposed to GDV and include:1

- Great Dane
- Standard poodle
- Gordon setter
- Weimaraner
- Saint Bernard
- Irish Setter
- German shepherds

Risk factors
The following risk factors for developing GDV have been previously reported:1

- High thoracic depth-to-width ratio
- Older dogs
- Lean body
- Those with a first-degree relative having had GDV
- Fast or “greedy” eater
- Temperament (e.g., nervous, fearful or aggressive)
- Dietary factors
- Dry food
- Feeding of a single large meal
- Raised dog bowl
- Pyloric outflow mechanics
- Abnormal eructation
- Laxity or agenesis of perigastric ligaments
- Gastric foreign body

Pathophysiology
GDV can result in acute signs of hypovolemic shock and decreased oxygen delivery (DO₂) due to cardiovascular compromise. Severe gastric dilatation can cause compression of the intraabdominal veins [e.g., portal vein, splenic veins, caudal vena cava (CVC)], resulting in decreased CVC blood flow and secondary decreased venous return.1 Likewise, respiratory compromise can occur due to gastric distension and increased intraabdominal pressure; this results in diminished diaphragmatic excursion and decreased total thoracic volume.1

Clinical signs and physical examination findings
Clinical signs of GDV include

- Non-productive retching
- Agitation
- Restlessness
- Hypersalivation
- Attempting to vomit
- Tachypnea
- Distended stomach
- Tachycardia
- Collapse
Acute death

Common physical examination findings of GDV include:
- Sprung ribs
- Signs of early decompensated shock (e.g., prolonged CRT, pallor, tachycardia, weak pulses, depressed mentation)
- Tachypnea
- Pulse deficits
- Irregular cardiac arrhythmias
- Tympanic abdomen
- Splenomegaly
- Obtunded
- Comatose

Diagnosis

The diagnosis of GDV is typically based on a combination of history, clinical signs, physical examination findings, and a single right lateral abdominal radiograph. On abdominal radiographs, with GDV, the pylorus moves craniodorsal, and is separated by a soft tissue opacity from the gastric fundus (e.g., *Popeye sign, double bubble, reverse C*). If the radiograph is unclear, a dorsoventral (DV) or ventrodorsal (VD) radiograph may be performed; the pylorus will be found left of midline on the DV view (as compared to right of midline in a normal healthy dog). It is also important to take the time to perform three-view chest radiographs in any dog > 5-7 years of age to help rule out underlying metastasis or pathology. A recent study found that 14% of dogs had evidence of aspiration pneumonia on radiographs, reiterating the importance of chest radiographs in these pre-surgical candidates.

A minimum database (e.g., PCV/TS/BG/AZO and lactate) should ideally be performed on presentation (e.g., during IV catheter placement). Further clinicopathologic testing should include a CBC, biochemistry panel, venous blood gas, and possibly a coagulation panel (if DIC is suspected). Evidence of hemoconcentration and stress-related hyperglycemia (e.g., elevated PCV/TS/BG) are often seen, along with hyperlactatemia (due to hypoperfusion). Severe hyperlactatemia (e.g., initially reported at 6 mmol/L; now 7.4 mmol/L) has been associated with gastric necrosis and nonsurvival in dogs. More recently, Green et al found that initial plasma lactate > 6 mmol/L was not predictive of gastric wall necrosis or survival in dogs; rather, the trend of the lactate level to decrease by > 50% within 12 hours was a good indicator for survival. Further clinicopathologic testing may reveal a stress leukogram, mild thrombocytopenia (secondary to platelet consumption and blood loss), and coagulopathy. Mild elevation in hepatic transaminases (secondary to hepatocellular injury from hypoperfusion) and azotemia (secondary to pre-renal causes) may be seen.

Treatment

In the GDV patient, aggressive and prompt treatment should be initiated to ensure the best outcome. Treatment should be directed towards the following:
- Fluid resuscitation
- Gastric decompression
- Surgical intervention
- Treatment of life-threatening complications (e.g., cardiac arrhythmias)
- Analgesia
- Monitoring and supportive care

Fluid resuscitation

Depending on the stability of the GDV patient, immediate IV access should be established with two, large bore cephalic catheters. Fluid resuscitation with isotonic crystalloids should be initiated until clinical signs of shock resolve. Small crystalloid aliquots of the “shock dose” should be used (e.g., 20-30 ml/kg, IV) and repeated as necessary. Colloids (e.g., Hetastarch, VetStarch, 5-10 ml/kg, IV) can also be used if the patient fails to respond to multiple crystalloid aliquots. The patient should be stabilized prior to anesthesia and surgery. Following surgery, continued fluid therapy is warranted to maintain perfusion and colloid osmotic pressure (COP) until the patient is discharged.

Gastric decompression

Gastric decompression can occur concurrently while the patient is being fluid resuscitated. This author prefers trocharization as compared to orogastric intubation, as it is easy to perform, effective, has minimal side complications, and is less stressful to the patient. The clinician should locate the most tympanic region (estimating where the stomach is) and clip and prep the region. Aseptic technique should be used. A large gauge needle or catheter (e.g., 14 or 16 ga.) should be directed into this area to alleviate gas from the stomach; the sound of hissing gas indicates appropriate placement into the stomach. Rare complications can be seen secondary to trocharization including splenic laceration, gastric perforation, or septic peritonitis. Alternatively, once the patient has been
appropriate stabilized, orogastric intubation can be performed to aid in gastric decompression. Goodrich et al evaluated dogs undergoing orogastric intubation versus trocharization in 116 dogs and found that orogastric tube placement was successful in 77% of dogs, with 38% of the dogs requiring sedation. In comparison, trocharization was successful in 86% of the cases, with no need for sedation. In the author’s opinion, all dogs should be sedated for orogastric intubation unless comatosed or obtunded to prevent undue stress and anxiety to the patient.

**Surgical intervention**

Once the patient has been stabilized, immediate surgical intervention is aimed at decompressing and repositioning the stomach. A complete abdominal exploratory should be performed; viability of the stomach, spleen, and entire gastrointestinal tract (GIT) should be assessed. Lastly, a gastropexy should be performed to prevent future reoccurrence. The readers are referred to a surgical resource for additional information.

**Treatment of life-threatening complications (e.g., cardiac arrhythmias)**

Cardiac arrhythmias have been reported in GDV patients both pre- and post-operatively (11-38%, 50.6-77%, respectively). Ideally, continuous ECG monitoring should be performed. There have been conflicting reports of whether the presence of perioperative arrhythmias affects the outcome or prognosis. Regardless, the use of anti-arrhythmics (e.g., lidocaine, procainamide) should be used in the following situations:

- When perfusion parameters are affected by the arrhythmia (e.g., prolonged CRT, poor pulse quality, pulse deficits, hypotension),
- Heart rate > 180 bpm
- R-on-T phenomenon, sustained ventricular tachycardia, multiform ventricular premature contractions (VPC)
- Impaired cardiac output

**Monitoring and supportive care**

As the majority of GDV patients are critically ill, appropriate monitoring (e.g., continuous ECG, blood pressure monitoring, etc.) and supportive care is imperative. Therapy should be aimed towards improving perfusion and oxygen delivery (e.g., crystalloid and colloid therapy). PCV/TS, blood pressure, perfusion parameters, and level of pain should be carefully monitored. Often, the use of gastrointestinal support (e.g., anti-emetics, pro-kinetics, antacids) is warranted to prevent secondary complications (e.g., aspiration pneumonia, ileus, etc.).

**Analgesia**

Once the patient has been stabilized, analgesia can be considered. Ideally, this should be titrated to the lowest effective dose and should be reversible (e.g., opioids). The author prefers opioids for post-operative analgesia (e.g., buprenorphine, hydromorphone, fentanyl CRI, etc.). The use of NSAIDs is not generally recommended due to the severity of gastric injury and poor perfusion to the GIT during GDV syndrome.

**Prognosis**

Overall, the prognosis for GDV is fair to good with rapid recognition, volume resuscitation, gastric decompression and surgical correction. However, other parameters such as hypotension, presenting collapsed, combined splenectomy and partial gastrectomy, DIC, peritonitis, and sepsis have been associated with fatalities.

**Conclusion**

The clinician and veterinary team must be able to rapidly recognize and treat the GDV patient. With aggressive fluid resuscitation, gastric decompression, stabilization and supportive care, the patient will be a better anesthetic candidate for surgical correction and gastropexy.

**References**


NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb’s Veterinary Drug Handbook*. 
Leptospirosis, a thin, motile spirochete with a hook-shaped end, is a zoonotic disease that affects wildlife, companion animals, and livestock. There are over 200 serovars of *Leptospira*, with some being saprophytic while others are pathogenic. Numerous reservoir hosts exist for *Leptospira* including raccoons, voles, skunks, dogs, pigs, cattle and rats, resulting in growing exposure to humans and to the environment.

In dogs, leptospirosis is caused by *Leptospira interrogans* (including serovars icterohaemorrhagiae, canicola, pomona, bratislava, and possibly autumnalis) and *Leptospira kirschneri* (including serovar grippotyphosa). Prior to vaccines (developed in the 1960s), the most common serovars infecting dogs including *L. icterohaemorrhagiae* and *L. canicola*. More recently, different serovars seem to be more associated with canine leptospirosis, including *L. grippotyphosa*, *pomona*, *bratislava*, and possibly *autumnalis*. Currently, veterinary vaccines provide coverage against *L. icterohaemorrhagiae*, *L. canicola*, *L. grippotyphosa* and *L. pomona*. With canine leptospirosis, infection with certain serovars are thought to be associated with certain types and severities of clinical disease, although this is not definitive.

**Geographic distribution**

The prevalence of leptospirosis is higher in warm, tropical locations with high rainfall. The top geographical locations where humans are diagnosed with leptospirosis include the Caribbean, Latin America, India, Southeast Asia, Oceania, and Eastern Europe. In North America, Hawaii is the state with the highest human cases. In the United States, high antibody prevalence (>1,600) has been seen in dogs from the following regions: Hawaii, West coast states (e.g., northern California, Oregon, Washington), the upper Midwest (e.g., Minnesota, etc.), the Northeast, the mid-Atlantic coastal regions, and other regions (e.g., Texas, Colorado).

**Risk factors**

While canine leptospirosis used to be considered more prominent in large breed, male, working dogs that free roam in rural environments, more recent studies have found that urban areas have a growing prevalence, with smaller dogs < 15 pounds being one of the fastest growing populations of canine leptospirosis. Of increased concern are studies showing that >20% of dogs may be chronic healthy carriers (based on studies in Michigan). Additional risk factors for leptospirosis include exposure to slow-moving or stagnant water, conditions where higher rainfall has occurred, late autumn, exposure to urbanized wild animals, or rodent exposure. One hypothesis is that global warming has contributed to the growing prevalence of leptospirosis due to the creation of warmer, wetter (e.g., flooding) weather conditions. Likewise, urban sprawl – the invasion of humans into the environment of wildlife - has increased the prevalence of canine leptospirosis.

While rare, leptospirosis has been reported in cats. Serologic evidence of exposure has been confirmed in cats to *L. canicola*, *L. grippotyphosa*, and *L. pomona*. Exposure is thought to occur due to rodent exposure, and can result in clinical illness and histopathologic changes consistent with changes seen in dogs with leptospirosis.

**Transmission**

Pathogenic leptospires are shed from renal tubules of both domestic and wild animals, and can remain viable in the soil and environment for weeks to months. That said, leptospires are inactivated by UV radiation and freezing. Infection can also occur through intact mucous membranes or abraded skin with direct or indirect exposure to urine. Rarely, leptospirosis can be transmitted via bite wound, ingestions of infected tissue (e.g., eating raw meat), or by venereal or placental transfer.

**Clinical signs**

Canine leptospirosis classically presents with both acute kidney injury (AKI) and hepatic injury. Clinical signs include:

- Generalized malaise/listlessness
- Inappetence to anorexia
- Vomiting
- Halitosis (e.g., uremia)
- Hypersalivation
- Diarrhea
- Melena
- Icterus
- Febrile
• Dehydration
• PU/PD*
• Abdominal pain (e.g., secondary to AKI)
• Uveitis
• Conjunctivitis
• Oliguria/anuria
• Weight loss

*Note, the polyuria and polydipsia seen with canine leptospirosis may be seen irrespective if the patient is azotemic. This may be due to several causes: impaired renal concentrating ability secondary to a decreased glomerular filtration rate or decreased vasopressin responsiveness of the inner medullary collecting ducts (e.g., acquired nephrogenic diabetes insipidus).

Less common signs include hematuria, vasculitis (e.g., peripheral edema, pleural effusion, ascites, etc.), cardiac abnormalities, abortion (e.g., predominantly reported in cattle), and pulmonary signs. Pulmonary lesions may be secondary to leptospiral pulmonary hemorrhage syndrome (LPHS) or vasculitis. Clinical signs include tachypnea, dyspnea, acute respiratory distress syndrome (ARDS), and pulmonary hemorrhage. With leptospirosis, a secondary coagulopathy may also be seen due to hepatic failure (e.g., decreased production of activated Vitamin K factors II, VII, IX, and X), disseminated intravascular coagulation (DIC), or vascular damage (e.g., presumed to be secondary to the spirochetes). Clinical signs of coagulopathy include:

• Petechial hemorrhage
• Hemoptyis
• Melena
• Epistaxis
• Hematochezia
• Hematemesis

Diagnostic testing
The diagnosis of leptospirosis is based on clinical suspicion, clinical signs, and clinicopathologic results consistent with leptospirosis. Clinicopathologic findings consistent with leptospirosis include the presence of: neutrophilia, a left shift, lymphopenia, a mild to moderate non-regenerative anemia, hemoconcentration (seen with dehydration), hemolysis (seen with cattle), thrombocytopenia (seen in up to 58% of dogs), azotemia (seen in > 80-90% of dogs), increased liver enzymes (including increases in ALT, AST, ALP, and total bilirubin; these changes are almost always seen with concurrent azotemia with leptospirosis), electrolyte abnormalities (e.g., hypokalemia, hyponatremia, hypochloridemia, hyperphosphatemia), and increased creatinine kinase.

Additional findings consistent with leptospirosis include isosthenuria, bilirubinuria, hematuria, glucosuria, proteinuria, and evidence of coagulopathy (e.g., increased fibrinogen, FDP, FSPs). Prolonged PT or PTT may be seen in 6-50% of dogs with leptospirosis.

Other advanced diagnostics may include radiology (to look for evidence of pulmonary lesions secondary to leptospirosis, which may appear as a nodular interstitial or alveolar pattern) and abdominal ultrasound (to rule out other underlying disease processes such as neoplasia, etc.). Ultrasound findings may reveal non-descript findings including renomegaly, pylectasia, perirenal fluid accumulation, a medullary band of increased echogenicity, increased cortical echogenicity, and rare other findings (e.g., splenomegaly, mild abdominal lymphadenopathy, etc.).

The most “definitive” diagnosis of leptospirosis is typically based on serology by the microscopic agglutination test (MAT), which tests for antibodies to leptospires. The MAT tests for the highest serum dilution causing agglutination of 50% of the leptospires. MAT testing typically includes L. canicola, L. icterohaemorrhagiae, L. pomona, L. grippotyphosa, L. hardjo, and L. bratislava. Unfortunately, there are several limitations of the MAT, including the hazardous need to maintain live cultures of pathogenic serovars, difficulty in standardizing the test, expense, cross-contamination of serovar cultures, and false negatives (e.g., due to acute disease) or false positives (due to previous vaccination, exposure, etc.). Another limitation of the MAT is that some cross-reactivity may occur between different serogroups. Keep in mind that one of the key limitations of MAT is that during the first week of acute disease, dogs may test negative. For this reason, convalescent titters are generally recommended 2-4 weeks later (at the same laboratory) to look for the presence of seroconversion. Typically, a 4-fold increase in titer is suggestive of infection; however, recent vaccination or antimicrobial therapy may affect the results (e.g., antimicrobial therapy may blunt the expected response). Interpreting MAT results must be done with care, as a result. Titters post-exposure can persist for at least one year, but are thought to declined by 4 months secondary to vaccination.

There are other diagnostic tests that can be used to screen for leptospirosis including dark field microscopy, silver staining of biopsy specimens (e.g., renal), immunohistochemistry, PCR, in situ hybridization, culture, and Idexx’s leptospirosis PCR & antibody ELISA in-clinic test. Note that each has their limitations. Dark field microscopy is technically difficult and has low specificity; this has fallen out of favor and is rarely used now. Silver staining of renal biopsy tissue can be performed, but lacks sensitivity and can result in false negatives. Fluorescent antibody testing and PCR can be performed on urine or tissue. Note that culture, PCR, and even
antibody ELISA tests can all be affected by recent microbial therapy. For this reason, pre-treatment blood work should always be utilized for submission in the patient suspected to have leptospirosis.

Treatment
Treatment of the leptospirosis patient is aimed towards fluid therapy, antibiotic therapy, gastrointestinal support, supportive care, and monitoring.

Fluid therapy
In the leptospirosis patient, aggressive intravenous (IV) fluid therapy is indicated as many patients are often massively polyuric, dehydrated, and azotemic. In general, a balanced, maintenance, isotonic crystalloid (e.g., LRS, Norm-R) can be used at 2.5-4.5X maintenance, and monitoring of ins and outs may be necessary to guide treatment (based on the severity of polyuria seen in patients with leptospirosis). The patient should be assessed carefully to ensure that volume overload does not occur, particularly in patients with cardiopulmonary disease. Fluid therapy should be continued until azotemia and clinical signs resolve (typically 2-4 days); IV fluids should then be slowly tapered to ensure that polyuria has resolved and the patient can maintain hydration.

Goals of fluid therapy
Serial physical examination is imperative to adequately evaluate a patient’s hydration status—checking for return of skin turgor, appropriate weight gain, and moisture of mucous membranes. However, physical examination findings are subjective, and <5% dehydration is subjective and difficult to assess on physical examination. The concurrent use of evaluation of PCV/TS, blood glucose, blood urea nitrogen (BUN or AZO), weight, urine output (UOP), urine specific gravity (USG), and thirst can be used in conjunction with physical examination findings to better assess hydration status.

Packed cell volume/total solids, blood glucose, and blood, urea, nitrogen (BUN/AZO)
Patients on IV fluids should have daily blood work (including PCV/TS, blood glucose, electrolytes, renal or biochemistry panel) assessed while hospitalized. Because patients often experience hemoconcentration when they are dehydrated (e.g., PCV/TS 55%/7.8 g/dL; 78 g/L), the goal of fluid therapy is to ensure that these numbers improve with appropriate therapy (consistent with hemodilution). Ideally, the PCV/TS in a normal, systemically healthy patient on IV fluids at sea level should be 35%/5.0 g/dL. (50 g/L). In fact, oxygen delivery is maximal at such a “hemodilute” PCV/TS, as there is less viscosity of red blood cells and “sludginess.” Note that some patients with leptospirosis may have a mild to moderate non-regenerative anemia; the goal should still be to hemodilute the patient, and total protein/solids should be used as a more appropriate guide in this situation. We can still evaluate the PCV/TS in abnormal, metabolically inappropriate patients. Classically, a 10% to 12% dehydrated, cachectic, geriatric cat with chronic renal failure may present to you with a PCV/TS of 28%/11 g/dL (110 g/L). Once that patient is adequately hydrated, the PCV/TS may decrease to 20%/7 g/dL (70 g/L), unmasking the anemia from lack of erythropoietin.

Urine specific gravity (USG)
In normal healthy patients, USG can be evaluated in patients on IV fluids to help assess hydration status. Ideally, USG should be measured before fluid administration to allow for evaluation of renal function. Dehydrated patients with concentrated urine demonstrate adequate renal function (cat > 1.040, dog > 1.025) - in other words, the kidneys are working and trying to absorb as much water from the urine as possible. Once started on IV fluids, normal, systemically healthy patients should have isosthenuric urine. Patients on IV fluids for > 6 to 12 hours should have adequate dilution of USG, and the ultimate goal of fluid therapy and adequate hydration should be USG of 1.015 to 1.018 on IV fluids. Patients on IV fluids with USG > 1.020 are still likely dehydrated and should be treated more aggressively with IV fluids if other parameters of dehydration persist (e.g., hemoconcentration). Hydration can be determined by assessing the color, volume, and USG of urine. A patient that is still dehydrated while hospitalized on IV fluids may have decreased UOP and dark-yellow urine (provided, for example, that no pigmentation, myoglobinuria, or bilirubinuria are present). This is a result of antidiuretic hormone release and renin-angiotensin stimulation, resulting in maximum absorption of free water and sodium. Unfortunately, in the leptospirosis patient, PU/PD may occur due to acquired nephrogenic diabetes insipidus, so utilizing USG as a guideline for hydration status will be difficult.

Urine output (UOP)
UOP should be monitored carefully, particularly in azotemic patients with leptospirosis. Fluid therapy should be directed toward achieving a hydrated state and matching ins and outs, based on the patient’s UOP. Note that normal UOP is 1–2 ml/kg/hour, but many of these leptospirosis patients present with severe polyuria. Again, one can assess the hydration status of the patient by evaluating the volume and USG of urine. Excessive urination with dilute, clear urine may indicate copious or excessive IV fluid therapy, whereas hypersenturia may suggest ongoing dehydration, and aggressive fluid resuscitation may be further warranted. If UOP is decreased (particularly in azotemic patients), fluid therapy and vasopressor support (to increase renal blood flow) should be initiated to prevent anuria (< 0.5 ml/kg/hour) or oliguria (< 1 ml/kg/hour). If UOP is decreasing and renal function is normal (based on creatinine, BUN, and pre–fluid therapy USG), the patient should be reassessed for hydration status, and fluid therapy adjusted as indicated.

1. Normal UOP: 1–2 ml/kg/hour
2. Oliguria: 0.5–1 ml/kg/hour
3. Anuria: < 0.5 ml/kg/hour

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Note that underlying diseases such as leptospirosis; postobstructive diuresis (posturethral obstruction); diabetes mellitus (with secondary osmotic diuresis due to glucosuria); diabetes insipidus; hyperthyroidism (increased glomerular filtration rate due to increased metabolic rate); and chronic renal failure (inability to adequately concentrate and absorb water) may result in dramatic water losses through the kidneys, and these patients may need a higher rate of fluids to compensate for ongoing losses. Likewise, these disease processes prevent us from differentiating renal versus prerenal disease on the basis of USG alone, as these patients have isosthenuria due to metabolic disease. Regardless, appropriate fluid therapy and urine monitoring (e.g., “measuring ins and outs”) may be necessary, particularly in azotemic, oliguric renal failure.

**Antibiotic therapy**

In the patient suspected of having leptospirosis, prompt, appropriate antibiotic therapy should be initiated (ideally after pre-treatment blood work has been submitted). Goals of antibiotic therapy is to eliminate leptospiremia and to eliminate leptospires from the renal tubular cells and renal carrier state). Appropriate antibiotics include penicillins (e.g., including ampicillin, amoxicillin, amoxicillin/clavulanic acid, penicillin, etc.) and doxycycline. In humans, the use of ceftriaxone and cefotaxime are also efficacious. The use of fluoroquinolones is controversial, as efficacy in a hamster model failed to clear leptospires from the kidneys and blood. Based on the ACVIM Consensus Statement, the antibiotic of choice is doxycycline (5 mg/kg PO or IV q. 12 hours for 2 weeks). Leptospires can shed in urine for months if appropriate antibiotic use is not implemented.

**Gastrointestinal support**

Azotemic patients should be treated with phosphate binders (e.g., aluminum hydroxide) if hyperphosphatemic, along with gastrointestinal protectants (e.g., omeprazole, pantoprazole, famotidine, sucralfate, etc.) for presumptive uremic gastritis. Anti-emetics (e.g., maropitant, ondansetron, dolasetron) should be implemented for patient comfort and to treat nausea.

**Anti-emetics**

- Maropitant: 1 mg/kg SQ q. 24 hours
- Ondansetron: 0.1-0.2 mg/kg IV q. 8-12 hours
- Dolasetron: 0.5-1 mg/kg SQ, IV q. 24 hours
- Metoclopramide: 0.1-0.5 mg/kg SC, IV q. 8 hours or 1-2 mg/kg/day as CRI IV

**Gastric pH altering medication**

**H₂ blockers**

- Famotidine: 0.5-1 mg/kg IV, SQ q. 12-24 (least p-450)
- Ranitidine: 0.5-2 mg/kg, IV, PO, SQ q. 8-12 (moderate p-450)
- Cimetidine: 5-10 mg/kg IV, PO, SQ q. 6-8 (most p-450)

**Proton-pump inhibitors**

- Omeprazole: 0.5-1 mg/kg PO q. 24 hours
- Pantoprazole: 1 mg/kg IV q. 24 hours

**Anti-ulcer:**

- Sucralfate 100-1 g PO q. 8 hours

**Zoonotic risks**

In animals developing acute leptospirosis, caution must be taken to prevent zoonotic spread. The use of appropriate hygiene (including protective eye ware, gowns, gloves, etc.) should be used when handling the patient and bodily fluids while hospitalized. Pet owners should also be cautioned about the zoonotic risk. A 10% bleach solution, iodine-based disinfectant, accelerated hydrogen peroxide, and quaternary ammonium solutions can all be used against leptospires. Likewise, other pets in the house should be assessed for clinical signs, and if healthy, vaccinated to mount an immune response.

**Prognosis**

The prognosis for leptospirosis is fair to good, provided aggressive treatment can be initiated. The survival is reported to be approximately 80% in dogs, both with dogs treated conservatively (e.g., IV fluids) and those treated more aggressively with hemodialysis. In those dogs developing pulmonary complications, the prognosis is poorer, with reported mortality rates (from Europe) of 36-42%. Pet owners should be cautioned about the risks for chronic renal insufficiency as a secondary consequence of chronic renal inflammation.

**Prevention**

As shedding of organisms can persist (e.g., leptospiuria) for weeks to months, prevention is imperative. Despite the good prognosis for leptospirosis, aggressive preventative care is warranted in dogs. This will help minimize zoonotic risk to pet owners and veterinary professionals; help minimize the chronic carrier state in dogs (which can result in further spread); prevent costly hospitalization; and minimize the risk of chronic injury (chronic renal failure). A leptospirosis prevention package should be initiated with the following:

- Environmental changes: This should be initiated to include rodent control; appropriate fencing; and landscaping changes to remove stagnant/standing water.
• Annual vaccination: The decision to vaccinate should be based on an endemic area, exposure of the dog, and risk factors (e.g., access to streams/stagnant water or urbanized wildlife). Ideally, vaccination with a 4-way leptospirosis strain should be utilized. Vaccination is important to help prevent/aid in the prevention of shedding to reduce infection of other animals and possible human exposure.

References
2. Winzelberg SE. Leptospirosis treatment and prevention with data on an ongoing leptospirosis prevalence study at the Animal Medical Center in New York City. Atlantic Coast Veterinary Conference Proceedings, 2013.
Canine parvovirus (CPV) is a common pathogen affecting young dogs that are unvaccinated, under-vaccinated, or immunosuppressed. Without treatment, CPV can be life threatening due to severe fluid losses and electrolyte derangements secondary to anorexia, vomiting, and diarrhea. In order to ensure the best outcome, treatment should be aimed towards symptomatic supportive care, aggressive fluid therapy, anti-emetics, antibiotic therapy, and nutritional support. This lecture will review the etiology, clinical signs, treatment, overall prognosis and preventative measures for CPV.

**Etiology**
CPV was originally discovered in 1967 and resulted in mild diarrhea. Since then, the virus has evolved to CPV-2 in 1978, with additional evolution of subtypes CPV-2a, CPV-2b, and more recently, CPV-2c. CPV-2b is thought to be more pathogenic and has replaced CPV-2a as the cause of parvovirus throughout the United States.

**Pathogenesis**
CPV is a small, single-stranded, non-enveloped DNA virus that preferentially infects rapidly diving cells (e.g., bone marrow, gastrointestinal tract, myocardium, etc.). There is an increased prevalence during warm summer months (e.g., July through September). Spread occurs via ingestion of bodily fluids (e.g., vomitus, diarrhea, etc.) containing the virus. CPV replicates quickly and infects the intestinal crypt epithelium by day 4 of infection. Clinical signs are thought to appear within 4-10 days of exposure, while antibody development occurs approximately 5 days after exposure.

**Risk factors/signalment**
Parvovirus is often seen in more urban environments with affected pups coming from poor husbandry backgrounds. As a result, pet owners may also have financial limitations. Dogs affected typically are < 6 months of age, between 6 to 20 weeks of age. Typically, there is no gender predilection, although one study reported that in dogs > 6 months of age, intact, male dogs were overrepresented. Certain breeds are thought to be at increased risk:
- American Pit Bull terrier
- Rottweilers
- German Shepherd dogs
- Doberman pinschers

In studies, breed, age, gender, and body weight did not appear to correlate with outcome or duration of hospitalization in one study.

**Clinical signs**
Clinical signs seen with parvovirus include:
- Anorexia
- Lethargy/Listlessness
- Malaise
- Hypersalivation (e.g., secondary to nausea)
- Vomiting
- Abdominal pain
- Diarrhea*
- Hematochezia

In mild cases, diarrhea may not be seen.

**Physical examination findings**
Classic physical examination findings for the parvovirus patient include:
- Dehydration (e.g., prolonged skin tenting, sunken eyes, etc.)
- Cachexia
- Hypothermia
- Fever
- Tachycardia
- Tachypnea
- Pallor
- Prolonged capillary refill time (CRT)
- Hypersalivation
- Poor pulse quality
- Hypovolemic shock
- Fluid filled loops of intestine
- Malodorous diarrhea staining
- Dyspnea
- Death

**Differential diagnoses**
Other rule outs for patients exhibiting similar clinical signs include:
- Other viral (e.g., coronavirus) infections
- Other bacterial (e.g., *E. coli*) infections
- Parasitism
- Intestinal bacterial infection (e.g., *Salmonella, Campylobacter*)
- Intussusception
- Foreign body obstruction

**Diagnostic testing**
The use of a fecal antigen ELISA test is the most rapid, cost-effective way of diagnosing CPV for the practitioner. The fecal antigen ELISA is sensitive to detect both CPV-2b and CPV-2c. Other tests that can be considered include PCR, virus isolation, and hemagglutination inhibition, but these are less commonly performed. That said, in a dog that tests negative on an in-house fecal antigen ELISA test, a PCR (on feces) can be considered due to its high sensitive. A real-time PCR can improve the sensitivity and specificity, and allows for rapid detection of CPV-2.

The diagnosis of CPV can be more challenging if these diagnostic tests are not readily available, as the decision to put an immunocomprised, young, immunologically naïve puppy into isolation poses large risk if the patient truly does not have CPV. Note that the modified live (ML) vaccine for CPV also replicates in the mucosal epithelium of the GIT; theoretically, the presence of low levels of antigen can be detected by various tests, resulting in a false-positive result. However, a recent study showed that various types of ML CPV-2 vaccines did not produce levels of antigen that were detectable on a SNAP ELISA parvovirus antigen test within 7 days of vaccine.

Depending on the financial limitations of pet owners, the ideal “gold” standard for the parvovirus patient includes:

**Gold or cadillac™ standard**
- Parvoviral fecal antigen test
- Complete blood count + blood smear
- Biochemistry panel
- Venous blood gas (e.g., acid-base status, electrolytes)
- Fecal float/smear
- PCV/TS/BG/AZO
- Blood pressure
- PCR if negative fecal antigen test and still suspicious
- Abdominal radiographs
- Colloid oncotic pressure (COP)
- ± abdominal ultrasound (if intussusception is suspected)

**Silver or Honda™ standard**
- Parvoviral fecal antigen test
- CBC with smear evaluation
- Biochemistry panel or venous blood gas
- Fecal float
- Blood pressure

**Bronze or YUGO™ standard**
- Parvoviral fecal antigen
- Blood smear + PCV/TS/BG/AZO
- Venous blood gas with electrolytes
Clinicopathologic findings
As parvovirus affects the pediatric patient, blood work changes associated with young patients are observed (e.g., isosthenuria, mild anemia, hypoproteinemia, elevated alkaline phosphatase, hyperphosphatemia, etc.). Additional clinicopathologic findings seen with parvovirus include:

- Lymphopenia
- Neutropenia
- Overall leukopenia
- Left shift
- Hypoglycemia
- Hemoconcentration (for a puppy)
- Hypoalbuminemia
- Hypoproteinemia
- Elevated liver enzymes
- Electrolyte abnormalities (e.g., hyponatremia, hypokalemia, hypochloremia)
- Mildly increased blood urea nitrogen
- Thrombocytopenia (due to consumption/blood loss into the GIT or DIC)
- Prolongation of PT/PTT
- Acid-base abnormalities (e.g., metabolic acidosis)
- Azotemia (secondary to multi-organ dysfunction)

Goals
Treatment of the canine parvovirus patient is aimed towards fluid therapy, antibiotic therapy, nutritional support, gastrointestinal support, supportive care, and monitoring. Specific goals of pediatric medicine include temperature control, fluid therapy, nutritional support (with the goal of weight gain), and control of infectious disease and parasites. In the more critically ill pediatric patient, goals should be focused on the following 4H’s: Hypovolemia/Hydration, Hypothermia, Hypoglycemia, and Hypoxemia.

Hypovolemia/hydration
One of the most common causes of neonatal hypovolemic shock is dehydration, which can occur quickly in these small patients due to gastrointestinal losses or higher fluid requirements; therefore, aggressive fluid therapy is warranted because these small patients can deteriorate quickly. For neonates, maintenance fluid requirements are 120-180 ml/kg/day, while for pediatric patients, fluid requirements range from 60-100 ml/kg/day. In critically ill pediatric patients, fluid therapy for shock must initially be given by IV (or intraosseous) route. Intraperitoneal or SQ routes are not adequate due to slower absorption and, ideally, should not be used in the critically ill, dehydrated, or hypovolemic patient. In severely dehydrated or hypovolemic patients, initial shock doses of a balanced crystalloid such as 30-45 ml/kg should be used. Serial assessment should be done after the bolus to reassess response and to evaluate the need for further fluid resuscitation. Potassium and dextrose supplementation typically is required, and careful monitoring of blood glucose and electrolytes is warranted. Lastly, colloids can be used in pediatric patients; however, keep in mind that puppies have a lower colloid osmotic pressure (COP) than adult dogs. If necessary, a colloid (e.g., Hetastarch, 1 mL/kg/H; VetStarch, 2 mL/kg/H) can be used to keep colloid osmotic pressure above 15 mm Hg in non-azotemic patients.

Hypothermia
In pediatric patients, careful temperature regulation and awareness of normal homeostatic temperatures is imperative. Normal rectal temperature in the first week of life is 96° ± 1.5°F (35.6° ± 0.7°C), 98.6° - 100°F (37-38.2°C) in the second and third week of life, and by 7 weeks of age, reach normal adult levels. Hypothermia can lead to bradycardia and intestinal ileus.

Hypoglycemia
Young patients are prone to hypoglycemia, which can be aggravated by anorexia, vomiting, diarrhea, dehydration, and infection. Ideally, IV dextrose boluses should be used (0.5-1.0 g/kg or 0.5-1.5 ml/kg IV of 50% dextrose, diluted 1:2-1:3) preferentially over oral dextrose. Isotonic fluids supplemented with 2.5-5% dextrose as a CRI can also be used (i.e., not D5W); however, caution should be used to prevent over-supplementation as prolonged hyperglycemic can result in worsening of dehydration via osmotic diuresis (due to puppies having insulin insensitivity).

Hypoxemia
Young patients exhibiting clinical signs of hypoxemia (e.g., cyanosis, orthopnea, tachypnea, dyspnea, and abnormal auscultation) should be immediately treated with oxygen therapy. Because neonates and pediatric patients are “normally anemic,” it may be clinically more difficult to “see” cyanosis since detection of cyanosis is dependent on hemoglobin concentration. In dyspneic patients, initial first line therapy should include oxygen therapy via facemask, oxygen cage, incubator, or endotracheal tube. The FiO2 should not exceed 40-60% for more than a brief period due to the risk of oxygen toxicity.

Antibody therapy
In general, beta lactam antimicrobials are considered the safest choices in young, growing puppies. If possible, avoid chloramphenicol, aminoglycosides, tetracyclines, and drugs like clindamycin that undergo enterohepatic cycling. Metronidazole can be used, but dose interval should be prolonged. Finally, quinolones have been shown to result in cartilage lesions in puppies and should be used only with the benefit outweighs the risk and ideally avoided altogether in growing, large breed dogs. Commonly recommended dosages include:

- Amoxicillin 6-20 mg/kg IV, PO q. 12
- Amoxicillin + clavulanic acid 12.5-25 mg/kg PO q. 12
- Cephalexin/Cefazolin 10-30 mg/kg IV q. 8-12
- Cefoxitin 22 mg/kg IV q. 8
- Ampicillin 22 mg/kg IV q. 8
- Ampicillin/Sulbactam 22-30 mg/kg IV q. 8

Gastrointestinal support
Anti-emetics (e.g., maropitant, ondansetron, dolasetron) should be implemented for patient comfort and to treat nausea.

**Anti-emetics**
- Maropitant: 1 mg/kg SQ or IV q. 24 hours
- Ondansetron: 0.1-0.2 mg/kg IV q. 8-12 hours
- Dolasetron: 0.5-1 mg/kg SQ, IV q. 24 hours
- Metoclopramide: 0.1-0.5 mg/kg SC, IV q. 8 hours or 1-2 mg/kg/day as CRI IV

The use of gastric pH-altering medication is not necessarily warranted in the CPV patient; most are unlikely to have gastric ulcers. As these gastric pH-altering medications have no anti-emetic effect, the author believes these are not typically necessary. That said, therapeutic doses include:

**H₂ blockers**
- Famotidine: 0.5-1 mg/kg IV, SQ q. 12-24 (least p-450)
- Ranitidine: 0.5-2 mg/kg, IV, PO, SQ q. 8-12 (moderate p-450)
- Cimetidine: 5-10 mg/kg IV, PO, SQ q. 6-8 (most p-450)

**Proton-pump inhibitors**
- Omeprazole: 0.5-1 mg/kg PO q. 24 hours
- Pantoprazole: 1 mg/kg IV q. 24 hours

Miscellaneous therapies
Fresh or fresh frozen plasma from recovered dogs has been suggested in the past to provide anti-parvoviral antibodies, but recent studies have not found a beneficial effect and have found that even recently recovered animals have minimal anti-CPV antibody concentrations. Moreover, such treatment may prime the dog for future transfusion reaction at a later point in its life. Equine endotoxin antiserum, recombinant human granulocyte-stimulating factor (rhG-CSF), or anti-virals (e.g., Tamiflu) have not been shown to be effective in improving survival or outcome. In small studies, the use of feline interferon has been weakly associated with improved survival; however, this is not readily available in veterinary hospitals.

**Prognosis**
The prognosis for canine parvovirus infection is fair to good with treatment, with recent reports of 80-90% survival with various modalities of treatment. Perhaps surprisingly, severity of neutropenia is not a negative prognostic factor, but severity of dehydration and lymphopenia may be. Recently, studies have compared standard in-hospital treatment versus a modified outpatient treatment (using volume resuscitation followed by subcutaneous fluid therapy and supportive care). Both protocols can be successful, with a survival only slightly lower in outpatients. A modified outpatient protocol may be a good alternative for less severely affected cases or those with financial limitations.

**Infectious diseases**
In animals developing acute parvovirus, caution must be taken to prevent further spread. The use of appropriate hygiene (including protective eye ware, gowns, gloves, etc.) should be used when handling the patient and bodily fluids while hospitalized. A 10% bleach solution, iodine-based disinfectant, accelerated hydrogen peroxide, or quaternary ammonium solution can all be used against parvovirus. Pet owners should be made aware to quarantine all affected pets for several weeks and to avoid dog parks, doggy daycares, training schools, city p

**Prevention**
While vaccination against parvovirus is highly effective, failure of passive transfer, early weaning, lack of vaccination, inappropriate client education (e.g., frequency of veterinary visits), or maternal antibody interference can result in disease. Parvovirus can be easily prevented by appropriate client education and vaccination. As DHPP is considered a core vaccine, puppies should be vaccinated.
frequently while maternal antibodies are waning. In high-risk areas (e.g., shelters), a vaccine every 2 weeks is indicated until 16-22 weeks of age (depending on if the breed is at risk) to prevent outbreaks.

AZOSTICK = BUN, bpm = beats per minute, BPM = breaths per minute, BG = blood glucose, HR = heart rate, IO = intraosseous, IP = intraperitoneal, IV = intravenous, PCV = packed cell volume, SQ = subcutaneous, TS = total solids

Footnotes

References
Cardiogenic shock (CS) occurs when oxygen delivery to the tissues is inadequate as a result of cardiac dysfunction with adequate intravascular volume. While patients in cardiogenic shock are often in congestive heart failure this does not have to be the case. The decrease in cardiac output associated with CS can result from failure of the heart to eject blood during systole (forward failure) or from inadequate ventricular filling during diastole (backward failure).

Forward CS can occur due to true systolic failure (decreased inotropy) as seen with DCM in which cardiac is directly related to stroke volume, however forward CS may also occur when valvular integrity is suddenly lost as happens with rupture of one or more first order chordae tendineae. Cardiogenic shock uncommonly occurs due to abrupt onset bradycardia but can be seen with 3rd degree AV block or sinus arrest (sick sinus syndrome). Regardless of the underlying cause, forward failure and CS is characterized by an increased end diastolic volume. This form of CS is often exacerbated by pulmonary edema and accompanying hypoxemia.

Backward failure and CS results when a disease process prevents diastolic filling of either the left or the right ventricle. This form of CS can result from obstruction of the right ventricular outflow tract or severe tachycardia effectively shortening the time available for diastolic filling, but most commonly is the result of acute cardiac tamponade secondary to pericardial effusion.

Due to the mechanism of CS many of these patients have a chronic progressive heart disease with an acute exacerbation and onset of shock. Acute tamponade can occur in a patient that was apparently healthy with collapse, shock or even sudden death as the only clinical signs. Canine heartworm disease can lead to CS when a large worm burden (60-100 worms) matures rapidly resulting in caval syndrome.

Cardiogenic shock can occur in patients of any age although older middle aged to geriatric animals are likely at increased risk. Small breed dogs are more likely to have forward failure resulting from valvular disease while large or giant breed dogs (Boxers and Dobermans excepted) are more likely to develop backward failure due to pericardial disease. When CS develops in a young animal it is most often secondary to a severe congenital abnormality and is often a terminal event.

Common historical information provided by owners includes episodes often described as “seizures”. These episodes are in fact syncope and can be differentiated from true seizures with some detailed questioning. Syncope often occurs during periods of excitement or increased activity whereas seizures often occur when animals are quiet and resting. Syncope patients often have episodes lasting for a few seconds after which they return to normal within several minutes. Episodes of aborted syncope may also have occurred in which the patient looked like it was going to collapse but then was able to recover. Patients can urinate or defecate during syncope so these findings should not be used to differentiate between the two. All patients presenting in shock with a history of “seizures” should be carefully evaluated for causes of cardiogenic shock.

As a rule these patients have physical exam evidence of impaired perfusion including: pale or cyanotic mucous membranes, cool distal extremities and obtundation. These patients will also have at least one abnormality found during cardiac auscultation such as: bradycardia or tachycardia, abrupt onset or cessation of an arrhythmia, new or worsening murmur, absent heart sounds or pulmonary crackles.

Unfortunately a single diagnostic test does not exist to diagnose cardiogenic shock, rather results from several tests, physical examination and history will have to be reconciled. Often, patients with CS are not hemodynamically stable enough to withstand prolonged physical restraint so the evaluation may be conducted over an extended period of time. Proper prioritization of diagnostic tests can aid in reaching a diagnosis, for instance delaying blood sampling but performing a single lateral radiograph instead may be a better use of the patient’s minimal physiologic reserves. Brief thoracic ultrasound and lead II EKG can often be obtained with little to no physical restraint necessary. When performed a brief thoracic ultrasound should focus on identifying left atrial enlargement, tamponade and estimating systolic function. Formal echocardiography is often necessary to identify the specific cause of CS although clinical decisions can be made without it.

Treatment of CS differs fundamentally from all other forms of shock in which volume expansion and vasopressor therapy are the mainstays of treatment. In fact, volume expansion and vasopressor therapies are often contraindicated for CS patients and their use could lead to worsening of the patient’s condition up to and including death. Cardiogenic shock must therefore be managed primarily by improving cardiac performance first and altering vasomotor tone second. Oxygen should be administered at the highest FiO2 possible immediately upon recognition of a patient in shock to maximize arterial oxygen content as much as possible. Animals with confirmed or suspected pulmonary edema should be administered a loop diuretic (furosemide 2 mg/kg IV or IM) and the dose should be repeated every 30-60 minutes until the clinical signs improve. The use of mild sedatives such as butorphanol (0.2 mg/kg IV or IM) may facilitate placement of an intravenous catheter to optimize medication administration.
Positive inotropes should be administered to patients with known or highly suspected systolic dysfunction. Due to the critical state of these patients these medications are almost invariably administered as constant rate IV infusions. Dobutamine can be administered at a starting dose of 2.5 mcg/kg/min and can be titrated up every 5-10 minutes until clinical signs improve or a rate of 20 mcg/kg/min is reached. In some instances it is possible to use pimobendan to improve cardiac contractility. Unfortunately, in the U.S. pimobendan is only available as an oral medication making its use limited. An injectable form of pimobendan is available for use in Europe with a recommended starting dose of 0.15 mg/kg IV.

Vasoconstricting medications are almost never used to treat CS however, balanced vasodilators may be beneficial. The most effective medication available is nitroprusside which can be used at doses starting at 1 mcg/kg/min up to a maximum of 10 mcg/kg/min. Unfortunately, the cost of nitroprusside has recently made its use prohibitive. Injectable hydralazine may be used to reduce vascular tone with anecdotal doses reported from 0.05 to 0.2 mg/kg/hr. As with nitroprusside, hypotension may occur. Early signs that hypotension is developing include vomiting. When vasodilators are used the doses should be adjusted to target a systolic arterial blood pressure of 90 mmHg or a mean blood pressure of 70 mmHg. Sildenafil citrate may be useful for management of acute, severe pulmonary hypertension in dogs with a dose of 1-2 mg PO TID. Again this medication is generally available as an oral formulation making its use for CS patients limited. An injectable form of sildenafil is available and has been used anecdotally with success. When used a loading dose of 1 mg/kg is administered slowly IV followed by a CRI of 0.3 mg/kg/hr.

Cardiogenic shock secondary to tamponade is treated by pericardiocentesis. Ultrasound may be used to guide the catheter during this procedure or it can be done blindly following previously described landmarks and techniques. Removal of even small amounts of pericardial effusion can have a dramatic impact on cardiac performance since pressure increases exponentially in a sphere as the radius changes.

Arrhythmias can be managed in one of two major ways, conversion of the rhythm to normal sinus rhythm or control of the ventricular contraction rate. Anti-arrhythmic medications can be used in the setting of CS to achieve either of these two goals. Due to the urgency inherent in CS injectable medications are always preferred over oral formulations. Lidocaine should be considered the first line anti-arrhythmic of choice for treatment of ventricular tachycardia and should be administered as a 2 mg/kg slow IV bolus monitoring the EKG for conversion. If no response is seen a second dose can be administered. If conversion occurs but is transient then a CRI should be considered at rates of 50-100 mcg/kg/min. Occasionally patients with SVT present in CS in which case beta blockade would be recommended. Administration of beta blocking drugs should be withheld until all other causes of shock have been ruled out. Once again, injectable, ultra-short acting drugs are preferred. Esmolol can be administered as a bolus of 0.2 to 0.5 mg/kg followed by CRI of 50-200 mcg/kg/min. Diltiazem may also be used (5-20 mcg/kg/min) and may be more beneficial for managing SVT that is not sinus in nature. Some tachyarrhythmias are amenable to electrical cardioversion but this procedure should be performed only in a referral setting by someone experienced in electrical cardioversion as the risk for developing asystole is moderate.

Management of bradyarrhythmias resulting in CS is essentially limited to the use of temporary or permanent pacemaker implantation. Transthoracic or transesophageal pacemakers can be used with many currently available defibrillators using the pacing function. Temporary or permanent transvenous pacing requires a dedicated generator and fluoroscopy and is most likely to be found in a referral hospital setting.

The management of caval syndrome can be done in a private practice setting using a transvenous jugular approach. This technique has been well described in review articles and textbook chapters and provided the patient survives the perioperative period the prognosis appears to be good.

Unfortunately, published mortality rates for veterinary patients with CS are lacking but human literature suggests mortality rates of 50-80%. In most cases, CS develops secondary to severe underlying cardiac disease. For this reason the short and long-term prognosis for these patient should be considered guarded to poor. Dogs receiving pacemaker implantation for 3rd degree AV block or surgery for caval syndrome provide two notable exceptions.
In 2010 the American College of Veterinary Emergency and Critical Care undertook the task of developing cohesive, veterinary specific recommendations for the performance of cardiopulmonary resuscitation utilizing an evidence-based approach. The result of this effort was the publication of the Reassessment Campaign on Veterinary Resuscitation (RECOVER) in 2011, developed through the participation of over 100 veterinary specialists and are composed of 101 clinical recommendations spanning 6 categories.

The progression for severely ill to cardiopulmonary arrest (CPA) is complex. Although not all animals progressing to CPA follow the same clinical course some physical exam findings that may indicate impending CPA include decreasing level of consciousness, hypothermia, hypotension, bradycardia and changes in respiratory pattern. Although the underlying cause of CPA may not be immediately apparent to the treating clinician, the nature of the arrest can have a significant impact on the expected outcome. Although success rates for veterinary patients receiving CPR are low with published survival rates ranging from 3 to 27% for animals suffering in hospital arrest, neurological outcomes are generally good to excellent, making CPR a worthy endeavor. In order to maximize the likelihood of a good outcome every effort should be made to optimize the effectiveness of CPR.

The success or failure of CPR is often determined before the first chest compression or rescue breath is provided. Organized and pre-stocked crash carts should be located in the areas that CPR is most likely to be performed. In most veterinary practices one of these locations will be in the vicinity of the operating room or areas where anesthesia is most frequently performed. In addition to crash carts, an easily visible and legible poster or individually tailored form with pre-calculated doses of common CPR drugs should be available. The veterinary team should make efforts to develop leadership and communications skills for members of the staff to improve their effectiveness during CPR. Communication skills can be further honed, and performing a debriefing following each CPR event can reveal important learning points. During this debriefing the team should critically evaluate their performance and determine what aspects of the CPR went well and which areas need to be targeted to improve future performance. The adage “practice makes perfect” or at least “practice makes better” in the case of CPR holds true. Therefore, the use of high fidelity mannequins or veterinary CPR training dummies and regular training events is recommended as their use can improve effectiveness of CPR by developing psychomotor skills allowing the clinician to slow down the event and think in a more clear fashion.

Second in importance only to preparation is the provision of high quality basic life support that is the foundation of cardiopulmonary resuscitation. The rapid recognition and diagnosis of CPA is of utmost importance as success rates decline considerably the longer CPA goes untreated. Because performance of CPR in patients that are not in CPA rarely causes complications and almost never causes serious complications it is recommended that no more than 10 seconds be allowed for pulse or heartbeat detection in animals that are apneic and unconscious. If cardiopulmonary arrest cannot be ruled out during this 10-second assessment then chest compressions should be started immediately. While historical practice has emphasized establishing an airway as the first step in resuscitation, current recommendations are to begin chest compressions and attempt orotracheal intubation concurrently. The need to re-establish blood flow to the brain and heart, even if that blood is somewhat oxygen depleted, trumps the need to increase blood oxygen levels, at least initially. Chest compressions should be provided at a rate of 100 compressions per minute and should compress the thoracic diameter by 1/3 to 1/2 its diameter. Once an airway is established, rescue breaths should be provided at a rate of 10 breaths per minute. Higher respiratory rates do not improve blood oxygen levels but can have detrimental effects due to the increased intrathoracic pressure during positive pressure ventilation and impaired venous return. Once begun, every effort should be made to minimize interruptions to chest compressions. It is recommended that brief (5-10 second) interruptions happen at pre-planned intervals every 2 minutes to allow for ECG evaluation and compression provider rotation. Significant compression provider fatigue occurs quickly and leads to a significant degradation in the quality of chest compressions.

Advanced life support is comprised of any and all interventions beyond chest compressions and ventilation. The most frequently utilized advanced life support technique is provision of drugs intended to improve hemodynamics and ameliorate vagal tone. Epinephrine is arguably the most frequently administered medication during CPR. Its use is intended to cause peripheral vasoconstriction and centralization of the blood to allow better perfusion of the brain and heart. The currently recommended dose of epinephrine is 0.01 mg/kg IV every 3-5 minutes or every two CPR cycles. All anesthetic drugs should be reversed immediately upon recognition of CPA and correction of documented, severe acid-base or electrolyte disturbances should be considered. Defibrillation is only indicated in animals with a heart rhythm amenable to such treatment (i.e. ventricular fibrillation or pulseless ventricular tachycardia). When progression from a perfusing rhythm to ventricular fibrillation (VF) or pulseless ventricular tachycardia (PVT) is observed then immediate defibrillation should be performed. If the progression to VF or PVT is not observed then defibrillation should be delayed to allow for completion of one CPR cycle of two minutes. Once defibrillation has been performed, chest compressions should be resumed for a two-minute cycle before the ECG is evaluated for success. The application of open chest CPR...
can improve outcomes but comes with difficulties inherent with thoracotomies. Attempts at open chest CPR should only be attempted if the means for managing a patient post-resuscitation are readily available.

In order to optimize CPR it is necessary that patients be monitored for signs of success or failure. All patients with suspected CPA should be instrumented with ECG. Analysis of ECG may help to rule out CPA or may identify rhythms that are amenable to a specific treatment (i.e. defibrillation). In the ideal setting all patients undergoing CPR will have end-tidal CO₂ monitoring performed. While useful for confirming correct placement of endotracheal tubes within the tracheal lumen in non-CPA animals, E_rCO₂ should not be used as the sole confirmation of endotracheal intubation in CPA animals. Since E_rCO₂ is linearly associated with cardiac output in CPA it can be a useful monitoring tool to gauge effectiveness of chest compressions and can be the earliest indicator of return of spontaneous circulation. When performing chest compressions and maintaining the minute ventilation at a constant level, E_rCO₂ levels can predict the likelihood of success with E_rCO₂ level of less than 15 mmHg and 20 mmHg suggesting worse prognosis in dogs and cats respectively. Once ROSC occurs E_rCO₂ will undergo a rapid and sustained rise as the heart more efficiently delivers CO₂ laden blood from the periphery to the pulmonary circulation. If ROSC is achieved then post-resuscitative monitoring should be tailored to the individual patient’s needs.

The care of an arrest patient does not end with ROSC; rather this is when the true care of the patient begins. In the immediate post-resuscitation phase every effort should be made to maintain arterial oxygen content within the normal range. No evidence exists that supra-physiologic oxygen levels are beneficial and theoretical detrimental effects exist. While post-arrest therapeutic hypothermia has become the standard of care in human medicine it is still beyond the capabilities of most veterinary facilities. However, if hypothermia occurs during the course of the arrest then rewarming efforts should not be vigorous and the patient should be allowed to return to normothermia at a gradual rate. Hyperthermia should be avoided if at all possible. There is no evidence in support of the routine use of corticosteroids, hypertonic fluids (mannitol or hypertonic saline), or prophylactic treatment with anti-seizure medications. If patients exhibit signs of intracranial hypertension then hypertonic saline or mannitol can be considered. Finally, referral to a comprehensive care facility with 24-hour capabilities should be considered for ongoing care of the post-arrest patient.
North America is home to 2 different families of venomous snakes, the elapinae (coral snakes) and the crotalinae (pit vipers). Of the two, the crotalinae are much more clinically significant due to their less reclusive behavior and wide range. In fact, pit-vipers can be found throughout North, Central and South America and include all rattlesnakes, the cottonmouth/water moccasin, and the copperhead. Pit-vipers are characterized by having moveable, hollow fangs through which venom is injected into the victim. Mature pit-vipers are capable of metering the dose of venom that they inject in order to preserve venom for the purpose of obtaining food. Juvenile snakes are incapable of dose adjustment and envenomate in an all-or-nothing fashion.

Historically the venom of the pit-vipers was considered to be vasculotoxic or necrogenic. While these terms are still useful, pit-viper venom is complex and varies with the species and local habitat. Some rattlesnakes for example the Mojave Green Rattlesnake, have venom that is highly neurotoxic with little tissue damage accompanying the bite. Ultimately, the purpose of snake venom is to immobilize the victim and begin the process of digestion. Typical pit-viper venom is composed of 90% water with the toxic components divided into both enzymes and peptides. The enzymatic factors are responsible for degrading connective tissue and allowing spread of the venom while peptides cause endothelial cell damage and ultimately circulatory collapse.

In addition to the massive soft tissue injury and necrosis that can accompany pit viper envenomation, life threatening hemostatic injury can occur. Typically, the coagulopathy resulting from pit viper envenomation is secondary to degradation of fibrin and fibrinogen directly by venom components. Through an unknown mechanism, platelet counts are often low following pit viper envenomation. These findings mimic those of disseminated intravascular coagulation (DIC) although importantly, fibrin cross-linking does not occur with snake envenomation and microthrombosis is not typically present. Neurotoxins associated with pit vipers result in non-depolarizing post-synaptic neuromuscular blockade and flaccid paralysis. Direct cardiovascular injury is possible and is manifested by ventricular arrhythmias and endothelial injury with circulatory collapse.

Establishing a diagnosis of pit viper envenomation can be challenging and is often heavily dependent on the history provided by the owner since clinical signs can be delayed by several hours. Physical examination may reveal one or two small puncture wounds or fang marks, most often on the face but occasionally on the feet or legs. Envenomating bites are often severely painful with progressive swelling advancing from the site of the bite. Some hematologic findings are present early in an envenomating bite and include echinocytosis and thrombocytopenia. Common chemistry changes that accompany envenomation include elevation of CPK, AST and ALT. The coagulation changes occur slightly later and are correlated with the severity of the envenomation. Expected findings would include decreased fibrinogen, prolongation of PT and aPTT and elevation of fibrin degradation products with normal to mildly elevated D-dimer.

Evaluating the severity of the bite can initially be difficult as 25 to 30% of pit viper bites are non-envenomating. Most initial defensive bites are dry with subsequent bites being highly envenomating. Agonal bites should always be considered highly envenomating since the entire volume of venom remaining in the venom glands will be injected. Several methods for describing severity have been described ranging from a numerical scale evaluating multiple systems to a simple minimal-moderate-severe scale. Regardless of the scoring mechanism used it is imperative to remember that signs may be delayed and may progress rapidly. Serial monitoring is recommended even for patients with no signs initially. Measuring circumference of an appendage or outlining the progression of signs is usually absent when dependent redistribution is the occurring.

Owners may contact a veterinarian prior to presentation seeking first-aid advice. Currently the only accepted first aid measure to be taken is immobilization of the affected site if possible followed by rapid transportation to a veterinarian. The owner should be instructed not to attempt to lance the site or attempt to suck venom from a bite. Placement of a tourniquet as this could lead to severe tissue damage and large areas of necrosis as the normal dilution of spreading venom components is prevented. It is not unusual for a severely envenomated patient to present in hypovolemic shock. In this case judicious resuscitation with crystalloids should be performed. Maintaining clear endpoints for resuscitation such as normalization of heart rate, respiratory rate and mentation are important to prevent over-resuscitation and subsequent exacerbation of tissue edema. Utilization of goal directed endpoints such as lactate, base excess and central venous oxygen saturation may aid in determining when resuscitation is complete.

The dosing of antivenom is based on the amount of venom injected not on the size of the patient. Typically dogs receive between 1-5 vials of antivenom. Paradoxically, smaller dogs often require higher doses of antivenom than large dogs due to the smaller volume of distribution of the venom. Administration usually begins slowly to allow for monitoring of possible reactions but is ideally completed within 30-60 minutes. Treatment with antivenom is indicated if progression of venom effects is seen including progressive
swelling and pain, coagulopathy or systemic effects such as hypotension or altered mental status. When administered antivenom binds to venom components and can reverse some of the clinical signs while preventing or slowing progression of signs. Ideally, antivenom should be used early following envenomation (i.e. within 4 hours) but may be beneficial up to 24 hours after a bite. Two different types of antivenom are currently available, a whole molecule antibody derived from immunized horses and a partial molecule antibody (Fab) derived from immunized horses or sheep. Both types of antivenom can lead to immune reactions including anaphylactoid dose related reactions and delayed type III hypersensitivity reactions.

Comprehensive management of envenomated patients includes effective analgesia. Liberal administration of opioids should be considered provided the patient’s neurological status does not preclude their use. Adjunctive analgesic medications to consider would include ketamine, lidocaine or dexmedetomidine. Limbs should be maintained in a functional position. Areas of necrosis should be cleaned and covered with a sterile bandage and managed as any other open wound would be. The prophylactic use of antibiotics in pit viper envenomation is controversial but both the recent human and veterinary literature suggests that prophylactic antibiotic use is not beneficial. Rather, antibiotics should be reserved for treatment of a known or suspected infection. Administration of steroids is not beneficial and should be avoided. Non-steroidal anti-inflammatory drugs are useful for treating associated inflammation but their use should be restricted to patients that are hemodynamically stable. NSAID medications should not be given to patients with impaired renal function or myoglobinuria. Since the coagulopathy associated with pit viper envenomation is due to the presence of circulating venom molecules, the mainstay of treatment is antivenom administration. Fresh frozen plasma is not expected to be beneficial so long as circulating venom is present and when used should be given after all intended antivenom doses.
Recognition of dystocia is based on an understanding of normal parturition. The average gestational length for the dog is 65 days (57-72) and for the cat is 66 days (56-70). Once the fetuses are full term parturition begins. Normal labor can be divided into 3 stages. Stage 1 includes restlessness, nesting and panting. No detectable uterine contractions occur during stage 1 labor which usually lasts for 6-12 hours but may last as long as 24 hours. Stage 2 begins with strong abdominal contractions and clear discharge and is complete when a fetus is expelled. The first fetus should be expected 2-4 hours after the onset of stage 2 labor with subsequent fetuses being delivered every 30 minutes to 4 hours. This stage of labor may last as long as 12-24 hours in dog. Cats have the unique ability to voluntarily interrupt stage 2 labor if they are feeling distressed and stage 2 may last for up to 48 hours. Stage 3 labor consists of placental expulsion which should occur 5-10 minutes after delivery of a fetus. Generally stage 2 and stage 3 labor alternate between fetuses but it is possible for 2 fetuses to be delivered consecutively followed by 2 placentae, one from each uterine horn.

Dystocia occurs when parturition does not proceed normally. This can be due to a problem with either the fetus or the dam. Fetal causes of dystocia include fetal monsters, true oversize, fetal malposition or malposture or fetal death. Maternal causes of dystocia are more common and include small pelvic diameter, abnormalities of the caudal reproductive tract, primary uterine inertia (failure of the uterus to begin contractions), secondary uterine inertia (uterine fatigue) or malnutrition. Secondary uterine inertia is the most common cause of dystocia and results from uterine fatigue usually in small breed dogs with fewer fetuses of larger size relative to the dam.

Frequently, inexperienced owners will call a veterinarian concerned that parturition is not occurring normally. Generally, owners should be advised to have a patient evaluated if one of the following occurs:

1. Prolonged gestation (due date reached with no signs of labor)
2. Stage 2 labor > 4 hours with no fetus
3. More than 2 hours between fetuses (cats may interrupt labor so this may not apply if they have been disturbed)
4. Green or brown vaginal discharge for 2-3 hours with no fetus delivered
5. Strong contractions > 30 minutes with no fetus delivered
6. Signs of systemic illness in the dam
7. Fetal membranes or part of fetus protruding from the vagina

When a patient presents for suspected dystocia a detailed history should be obtained including last known breeding date and expected due date if available. Following the history a thorough physical examination should be performed focusing on identifying any underlying systemic diseases of the dam that may be causing dystocia. Digital vaginal examination should be performed using sterile gloves and liberal lubrication. Fetal positioning, vaginal anatomy (vaginal hyperplasia, septum, neoplasia), cervical dilation, soft tissue pelvic anatomy (lymph nodes, urethra) and presence of meconium should be evaluated. Light feathering of the dorsal vaginal wall can be performed to attempt to elicit a contraction.

Diagnostic imaging is often useful to aid in determination of fetal age if the owner is unaware of the breeding or due dates. A fetus should be considered full term when the caudal vertebrae, fibula, calcaneus, bones of the feet are visible radiographically. Visible teeth are the final radiographic sign of a full term fetus and are present at approximately day 61 of gestation. Beyond estimating fetal age, radiography can be used to evaluate fetal viability. Signs of fetal death include gas present in the fetus or placenta, collapse of the spinal column, overlapping of skull bones or loss of fetal flexion. Orthogonal radiographs should be evaluated for potential causes of dystocia including bony pelvic anatomy and fetal positioning. Fetal viability may be more reliably evaluated with ultrasonography. Lack of a visible heartbeat in a full term fetus is an indication of fetal death. Other ultrasound findings consistent with fetal death include: decreased placental fluid volume, increased fetal echogenicity and increased gas in the fetal stomach. Once viability is determined each fetus should be evaluated for evidence of distress. Normal fetal heart rates are greater than 180 bpm while rates between 150-170 indicate moderate distress and less than 150 bpm indicate severe distress. If a low fetal heart rate is identified it should be monitored for 30-60 seconds as fetal heart rates will be transiently depressed during uterine contraction.

Medical management can be considered for dystocia if the fetuses are viable with no evidence of distress, the dam is in good health, the labor has not been protracted, the cervix is dilated and the fetal size is consistent with a possible vaginal delivery. If medical management is attempted the dam should be well hydrated with normal electrolytes. This may require administration of intravenous fluids. Oxytocin should be administered 1-3 IU SC or IM and the dam should be monitored for progression of labor. If a fetus is not delivered after 30-45 minutes calcium gluconate may be administered slowly IV (0.5-1.5 mL/kg) while monitoring EKG. If no further progress is made the oxytocin dose can be repeated or may be increased to 5-20 IU SC or IM. Oxytocin dosing may be
repeated up to 3 times. If this fails to result in delivery of a fetus the patient should proceed to surgery for caesarean section. Only 1/3 of dogs with dystocia will respond to administration of oxytocin alone.

Caesarean section is required in approximately 60-65% of canine dystocia cases and 70% of feline dystocia cases and 60% of caesarean sections are performed on an emergency basis. The most common reason for proceeding to C-section is identification of fetal distress. When a fetal heart rate less than 150 bpm is identified the patient should proceed to emergency surgery, if the fetal heart rate is between 150-170 bpm surgical treatment should be strongly recommended above attempted medical management. If there is a doubt about the necessity for surgery C-section is usually better for both the dam and the fetuses. Pre-operative considerations include the dam’s volume status, shock status and intended future breedings. Prior to induction the owner should be asked whether to prioritize the dam or the fetuses in the case of an emergency.

The patient should have the abdomen clipped prior to induction to minimize the time between induction and delivery. Blood pressure should be monitored if possible since placental blood flow is dependent on maternal blood pressure. Anesthetic drugs that are reversible and/or short acting are preferred with pure opioid agonists and propofol being most common. Inhalant gas should be minimized until after delivery since elimination of the gas is dependent on ventilation and neonates delivered by C-section are often apneic. Line block of the intended abdominal incision with local anesthetic may aid in reducing the amount of inhalant needed for the approach. Epidural anesthesia/analgesia can be considered and is generally safe for neonates and dam. Pure opioids are the preferred analgesic drugs due to their complete reversibility.

Two surgical techniques have been described. Traditional caesarean section in which a hysterotomy is performed and the fetuses are delivered one at a time will allow for placental blood flow to be maintained in the undelivered fetuses increasing the likelihood of survival. When hysterotomy is performed the expected neonatal survival rate exceeds 90%. Following delivery of all the fetuses, an ovariohysterectomy can be performed or the uterus can be closed if future breedings are intended. An alternate method of delivery is en-bloc ovariohysterectomy in which each ovarian pedicle and the uterine body is clamped and the uterus is removed en-bloc with the fetuses still inside. The uterus is then handed off to an assistant who delivers the fetuses and begins neonatal resuscitation. If this method is used every effort should be made to limit the amount of time from the placement of the first clamp to delivery of the last fetus to less than 60 seconds. Survival rates using the en-bloc technique are reported to be 75% in the dog and 42% in the cat. This technique should be used preferentially if uterine infection is suspected, the entire litter is dead or if the maternal condition necessitates surgical brevity. When using this technique, care should be taken to ensure that no part of the fetus is clamped in the uterine body. This can be achieved by manually milking the fetus proximally into the body of the uterus. If any part of the fetus has descended beyond the vulva this technique will likely not be possible.

Following delivery neonatal resuscitation begins. Ideally, each neonate will have a dedicated assistant responsible for its resuscitation. The umbilical cord should be clamped if this has not already been done and the face and mouth should be cleared of placental membranes. The neonates should be rubbed vigorously with a clean, dry towel focusing on the face, thorax, genitals and umbilicus. The mouth and nostrils can be cleared by using a bulb syringe. Neonates should not be swung in an attempt to clear airways as this could lead to significant injury. Neonates should have a respiratory rate between 10-18 brpm. Manual, mouth to snout ventilations can be used and supplemental oxygen should be provided. Aggressive needling of the acupuncture point GV26 (at the philtrum of the nose) with a 25-gauge needle can stimulate ventilation in an apneic neonate. As long as a heartbeat is detectable resuscitation should continue even in the presence of prolonged apnea. In neonates that are responding poorly to resuscitative efforts, reversal of anesthetic/analgesic drugs can be attempted. Naloxone can be administered at 0.1 mg/kg. Dosing can be intravenous, intramuscular, intraosseous or sublingual. Vascular access can be obtained at the jugular vein or the umbilical vein. The administration of doxapram is not recommended due to the risk of cerebral acidosis in an apneic patient. Once resuscitation is complete and the neonates are vocalizing and vigorous, they should be evaluated for the presence of cleft palate, atresia ani and umbilical hernia prior to being placed in a warm environment. The dam and surviving neonates should be discharged from the hospital when anesthetic recovery is complete and milk letdown has been confirmed and all neonates have been
Hypernatremia: What to Do with the Salty Dog
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Under normal conditions total body water is divided into the intracellular and extracellular fluid compartments with 66% of total body water being located within the intracellular space. The remaining 33% of total body water is distributed between the intravascular and extravascular spaces. Sodium is the most ubiquitous electrolyte in the extracellular compartment. It is responsible for maintaining plasma osmolarity between fluid compartments and regulating water movement between the intracellular and extracellular spaces. Serum sodium and subsequently serum osmolarity is closely regulated by the kidneys and brain under the influence of several hormones the most important of which is antidiuretic hormone (ADH). Changes in serum osmolarity by as little as 1-2 percent are detected at osmoreceptors located in the hypothalamus. Stimulation of these receptors results an increase in thirst and the release of ADH from the posterior pituitary gland. Once released, ADH causes aquaporin-2 channels into the collecting tubules of the kidney resulting in free water retention and restoration of plasma osmolarity. Serum sodium is therefore a better reflection of total body water content than total body sodium content.

When serum sodium concentrations increase serum osmolarity also increases. When this happens, water moves down its osmotic gradient out of the intracellular compartment resulting in cellular dehydration and thus cellular shrinkage. While most tissues are capable to tolerating some change in cellular volume the brain is not. As serum osmolarity increases brain volume decreases. This is compensated for within the first several hours by accumulation of electrolytes within the brain. The movement of electrolytes into the intracellular space is sufficient to partially restore brain volume. This process is completed over the next several days as osmotic particles called organic osmolytes are generated re-establishing the normal balance between intracellular and extracellular osmolarity.

Hypernatremia, defines as plasma or serum sodium concentration above the reference range (145-155 mEq/L), is a relatively uncommon finding in dogs and cats but when present has been associated with a significant increase in mortality. The development of hypernatremia may be divided into two major causes: excessive free water loss, absolute sodium gain. Of these, absolute sodium gain is by far the less common but can result from ingestion of high salt materials such as homemade playdough or seawater. Hypotonic fluid loss on the other hand is more common and can result from insufficient water intake, increased insensible losses, renal causes, gastrointestinal causes and cutaneous losses.

Whether or not clinical signs are associated with hypernatremia depends on the degree of hypernatremia present (> 180 mEq/L) and the rate at which it developed. Most patients with mild to moderate hypernatremia that occurs slowly do not have clinical signs associated with the electrolyte abnormality itself, rather signs will be referable to the underlying disease. In patients with severe acute hypernatremia it is possible to see obtundation, head pressing, seizures, coma or death.

Since the normal physiologic response to hypovolemia is sodium retention through the action of aldosterone, free water deficit should not be calculated until normovolemia has been established. When a patient is hypovolemic the initial replacement fluid of choice should have a sodium concentration as close as possible to that of the patient. In most cases 0.9% NaCl will be a suitable choice. Many patients will have correction of hypernatremia when the volume status has been returned to normal. If hypernatremia persists in a normovolemic patient then a free water deficit exists. Free water deficit is calculated using the following formula: Free water deficit (L) = 0.6 x body weight in kg x (measured Na+/ideal Na+ - 1). When hypernatremia develops acutely the correction can occur relatively rapidly with the free water deficit being replaced to achieve a rate of change of plasma sodium of 1 mEq/hr. When chronic hypernatremia is present it must be corrected more slowly due to the generation of organic osmolytes in the brain. The maximum rate of correction of chronic hypernatremia is therefore 0.5 mEq/hr.

Management of mild to moderate hypernatremia in a patient with an intact thirst mechanism may be achieved by administration of oral water. When the thirst mechanism is no longer intact enteral water may still be provided through placement of a feeding tube with the calculated free water deficit being replaced over the number of hours calculated using the correction limits above. If provision of enteral water is not an option due to decreased mentation or vomiting, or is not likely to be sufficient the free water deficit can be replaced using 5% dextrose in water. When this approach is taken the free water deficit is replaced as above with the volume being administered over the number of hours deemed to be safe. Regardless of the method used to correct free water deficit it must be remembered that this volume is an absolute deficit and should be added to the patient’s maintenance needs keeping in mind that ongoing losses and underlying medical conditions must be accounted for.

Once correction of hypernatremia has been started serum sodium should be monitored every 4 hours and the rate of correction adjusted to maintain an appropriate drop of sodium without exceeding the established maximum rate of correction (0.5 to 1 mEq/hr). It is typical for correction of severe hypernatremia to require 24 to 48 hours to correct. In addition to serum sodium concentration, the patient should be monitored for neurological signs associated with too rapid a correction including mentation changes (obtundation, stupor, coma), head pressing confusion or seizures. When hypernatremia is corrected too rapidly the intracellular osmolarity exceeds
blood osmolarity and water moves back into the cells down its osmotic gradient. This cellular swelling manifests as cerebral edema and increased intracranial pressure.

If a patient develops any signs associated with cerebral edema the serum sodium should be immediately measured to determine if the sodium concentration has gone down. If the serum sodium is lower, even if the rate of correction has not been excessive, cerebral edema should be considered. If cerebral edema is suspected treatment with 7.2% hypertonic saline at a dose of 3-5 mL/kg over 20 minutes should be started. If hypertonic saline is not available, mannitol can be considered at a dose of 0.5 to 1 g/kg IV over 20 minutes.
Under normal conditions total body water is divided into the intracellular and extracellular fluid compartments with 66% of total body water being located within the intracellular space. The remaining 33% of total body water is distributed between the intravascular and extravascular spaces. Sodium is the most ubiquitous electrolyte in the extracellular compartment. It is responsible for maintaining plasma osmolarity between fluid compartments and regulating water movement between the intracellular and extracellular spaces. Serum sodium and subsequently serum osmolarity is closely regulated by the kidneys and brain under the influence of several hormones the most important of which is antidiuretic hormone (ADH). Changes in serum osmolarity by as little as 1-2 percent are detected at osmoreceptors located in the hypothalamus. Stimulation of these receptors results an increase in thirst and the release of ADH from the posterior pituitary gland. Once released, ADH causes aquaporin-2 channels into the collecting tubules of the kidney resulting in free water retention and restoration of plasma osmolarity. Serum sodium is therefore a better reflection of total body water content than total body sodium content. Consequently hyponatremia reflects free water excess. Whether this free water excess represents absolute fluid excess depends on the underlying cause.

When intracellular osmolarity exceeds extracellular osmolarity water will move down its osmotic gradient and into the cell. This is most important in the cells of the CNS, which are highly sensitive to changes in volume. When cellular swelling occurs in the CNS intracranial pressure increases and if not addressed will lead to brain herniation and death. In acute hyponatremia, the cells of the CNS actively pump intracellular ions out of the cell in an effort to match the osmolarity of the plasma. This process occurs over several hours but is insufficient to compensate for large changes in serum sodium over long periods of time. When hyponatremia is chronic, cellular generation of organic osmoles is down regulated. This process takes several days but is much more effective as a long-term solution.

Hyponatremia is defined as blood sodium less than the reference range (145-155 mEq/L). Similar to hypernatremia, the degree of clinical signs associated with hyponatremia is dependent on both the magnitude of sodium change and the rate at which that change occurred. Mild to moderate hyponatremia (Na+ > 120 mEq/L) is unlikely to result in clinical signs. Severe or rapidly occurring hyponatremia on the other hand may cause CNS signs consistent with the development of cerebral edema including: mentation changes (obtundation, stupor, coma), head pressing and seizures. Unlike hypernatremia however, the pathogenesis of hyponatremia is more complex and affects the approach to treatment so a much greater degree.

The first step in managing a patient with hyponatremia is determining effective plasma osmolarity using the following formula: eOsm = 2(Na+) + (glucose/18). Unfortunately this formula fails to account for unmeasured osmoles such as ketone bodies, mannitol molecules or ethylene glycol molecules. Measuring blood osmolarity may identify an “osmole gap” when compared to the calculated value. Based on the calculation or measurement of osmolarity a patient can be classified as hyperosmolar, normosmolar or hypoosmolar.

Hyperosmolar hyponatremia results from increased blood osmolarity causing water to move from the intracellular to the extracellular space diluting blood sodium concentration. This type of hyponatremia is most common in patients with diabetes mellitus and in it’s worst form can manifest as hyperosmolar hyperglycemic syndrome. Normosmolar hyponatremia is often called pseudohyponatremia and is a laboratory artifact that can be seen when a patient’s plasma compartment contains a large fraction that is not water as occurs with hyperglobulinemia or hyperlipemia. Psuedohyponatremia only occurs when flame photometry is used to determine sodium concentration but does not occur when an ion-selective electrode is used so familiarity with the laboratory methodology employed is important.

Hypoosmolar hyponatremia is further categorized based on the patient’s volume status. Hypervolemic hypoosmolar hyponatremia results from a decrease in effective circulating volume and is seen in three clinical conditions: congestive heart failure, severe liver disease, nephrotic syndrome. In all of these conditions a decrease in effective circulating volume results in non-osmotic release of ADH in an attempt to restore volume at the cost of decreasing osmolarity. As a rule glomerular filtration rate must be decreased for hypervolemic hypoosmolar hyponatremia to develop. When GFR is maintained excess volume is eliminated through an increase in urine production.

Normovolemic hypoosmolar hyponatremia develops due to an absolute excess of free water when GFR is maintained. Conditions that can lead to development of normovolemic hypoosmolar hyponatremia include psychogenic polydipsia, syndrome of inappropriate ADH secretion, myxedema coma or administration of hypotonic fluids.

Hypovolemic hypoosmolar hyponatremia is the most common category of hyponatremia encountered in daily practice. This type of hyponatremia develops due to volume depletion from either renal or non-renal causes. When volume is lost from the vascular space through the gastrointestinal tract (vomiting or diarrhea) or through loss into a third space (pleural effusion or peritoneal effusion) the
body again attempts to preserve volume at the expense of tonicity through release of ADH and free water retention. Renal fluid and sodium loss occurs from administration of loop diuretics or mineralocorticoid deficiency (hypoadrenocorticism).

Treatment of hyponatremia is dependent on the classification of hyponatremia and the underlying disease process as well as the magnitude and duration of hyponatremia. Hyperosmolar hyponatremic patients are treated for their underlying disease. Once the source of the unmeasured osmoles is removed (i.e. glucose, mannitol) the patient’s plasma sodium will return to normal. Management of hyperosmolar hyponatremic patients is more similar to management of hypernatremia due to the presence of a hyperosmolar state. In this situation, inappropriately rapid correction of hyperosmolarity will lead to formation of cerebral edema and the neurological sequelae that accompany it. The most appropriate rate of correction for hyperosmolar hyponatremia would be to reduce the patient’s osmolarity by 0.5 to 1 mOsm/hr (the same rate as sodium correction in hypernatremia). Normosmolar or pseudohyponatremia do not require treatment, as this type of hyponatremia is a laboratory artifact and should resolve when the underlying condition leading to the artifact is removed (non-lipemic sample) or the laboratory method is changed.

Correction of hypoosmolar hyponatremia can be more challenging. Due to the brain’s adaptive response to chronic or profound hyponatremia overzealous correction can lead to acute decrease in CNS cell volume. Myelin sheaths are particularly sensitive to acute changes in cell volume and demyelination can occur. Unfortunately, the neurologic signs that accompany this dehydration do not present immediately but rather, are delayed for several days. Common clinical signs secondary to overly rapid correction of hyponatremia include drowsiness, inability to stand, ataxia, extensor spasticity, coma and death. Although this condition is not uniformly fatal when it occurs the prognosis for return to normal is poor with most animals having residual neurologic deficits.

Hypervolemic hyponatremia is managed by restoration of effective circulating volume and GFR. For patients with congestive heart failure this will include a diuretic medication but must also necessarily include medications to improve cardiac output such as dobutamine or pimobendan. In the acute setting pimobendan can be administered at 0.25 to 0.5 mg/kg PO BID to improve cardiac output. The addition of afterload reducing drugs (hydralazine 0.5 to 2 mg/kg PO BID to effect) may also improve GFR. Vasopressin antagonists are available although their clinical use in veterinary medicine has been limited due to high cost and potential liver toxicity.

Normovolemic hyponatremia can be managed by careful water restriction to limit intake to a volume less than urine output. When restricting water intake it is important to monitor the patient for ability to concentrate urine. All medications that have potential antidiuretic effects should be discontinued.

Hypovolemic hyponatremic patients should be volume resuscitated with fluid containing sodium in concentrations as close to plasma sodium as possible. Once normovolemia (intravascular volume and interstitial volume) has been re-established the patient’s sodium should be rechecked. If hyponatremia is persistent then the addition of higher sodium containing fluids can be considered.

Regardless of the cause of hyponatremia, when correction is begun the patient should have serum sodium levels monitored every 4-6 hours targeting a return to normal of 0.5-1 mEq/hr. The late onset of clinical signs associated with correction of chronic or profound hyponatremia means that a more conservative correction rate is often preferred. Due to the delayed presentation of neurological signs owners should be instructed to monitor the patient carefully and to return if the patient seems to be declining in any way. The best way to manage central demyelination is to be aware of the possibility and to prevent it from occurring. Once demyelination occurs treatment becomes entirely supportive.
Trauma is a common cause of veterinary emergency room visits. Common causes of trauma include bite wounds and blunt injuries suffered from encounters with vehicles or falls from heights. Due to the high degree of variability possible in the injuries associated with different mechanisms of trauma a systematic approach at the time of presentation followed by a deliberate medical or surgical plan is essential. The initial evaluation of a trauma patient begins with a brief history in which the owner or agent conveys what transpired if known. This initial history should be kept brief and should focus on important facts about the traumatic event including mechanism of injury (fall, hit by car, bite wound), loss of consciousness during or after the event, time from event to presentation and voluntary ambulation post event.

Following this brief history an initial survey exam is performed focusing on identifying potentially life-threatening injuries. Evaluation of the airways is usually performed first. This is achieved by observation of the respiratory pattern paying special attention to the effort the patient is making and if an increase in effort is evident which phase of the respiratory cycle is affected. Stertor or stridor may be evident and if severe may necessitate immediate intubation to aid in stabilization. Mucous membranes should be evaluated next. The presence of cyanosis or pallor should raise concern for impaired oxygenating ability or shock. Capillary refill time may be prolonged or even absent if the patient is in the later stages of shock. Breath sounds should be carefully ausculted. Absence of breath sounds either unilaterally or bilaterally may indicate pleural space disease such as pneumothorax or hemothorax. Loss of breath sounds in conjunction with circulatory collapse and severe respiratory distress suggest tension pneumothorax. Crackles, indicating pulmonary contusion or hemorrhage may be detected.

If a life threatening condition is present steps should immediately be taken to correct or manage it. Severe stertor/stridor or respiratory arrest requires endotracheal intubation or emergency tracheostomy. Identification of tension pneumothorax is treated by rapid thoracocentesis. Circulatory collapse or cardiac arrest is treated with cardiopulmonary resuscitation. If an imminent threat to life is not identified the secondary survey is begun utilizing a systematic approach to ensure no occult injuries are missed.

The secondary survey typically begins with evaluation of the patient’s neurological status. The patient’s mentation and level of consciousness should be evaluated and the patient should be assessed for voluntary motor. Postural changes such as rigid extension of all four limbs (opisthotonus) or rigid extension of the forelimbs only (Schiff-Sherrington) are associated with severe forms of brain stem compression or cerebellar herniation. Animals may be obtunded to stuporous following trauma and this may be due to the presence of primary CNS injury or as a result of shock. Small patients that were lifted from the ground and shaken should be treated as if they have a spinal cord injury until proven otherwise, especially if they have not been observed to ambulate following the injury.

Evaluation of the thorax is performed next beginning with a visual inspection to identify any lacerations or puncture wounds that might penetrate the thoracic cavity. Repeated auscultation should be performed paying special attention to the quality of breath sounds in each hemithorax. Decreased breath sounds ventrally may indicate the presence of pleural effusion while decreased sounds dorsally is common with pneumothorax. Focused assessment of the thorax with ultrasound has been shown to be an easy and rapid method of evaluating both the pleural space and the lung parenchyma. Loss of a glide sign or identification of fluid indicate pneumothorax or pleural effusion respectively. So called “lung rockets” suggest the presence of primary parenchymal injury or contusion. This procedure may be performed in either lateral or sternal recumbency. The ultrasound probe is initially placed at the “chest tube site” (widest part of the chest) on the left and right to evaluate for pneumothorax or contusions. The probe is then relocated to the “pericardial site” directly over the heart on the left and right to evaluate for possible pericardial effusion.

Abdominal evaluation again begins with a visual inspection of any lacerations and puncture wounds to generally assess depth and possible penetration. Any puncture wounds over the abdomen, especially those resulting from animal bites have the potential to penetrate the abdomen even if the surface injury appears to be minor. Abdominal distension with the presence of a fluid wave may be present with traumatic hemoabdomen. Palpation of the urinary bladder should be attempted although the presence of a palpable bladder does not rule out the possibility of uroabdomen or uroretroperitoneum. Rectal exam should be performed to assess pelvic ring continuity and to assess anal tone. Abdominal focused assessment with sonography for trauma (AFAST) has been well reported and is widely accepted as a rapid and sensitive method for evaluating the abdomen for the presence of free fluid. This evaluation can be performed in lateral recumbency. The ultrasound probe is placed over one kidney and is fanned cranial to caudal, at which time the probe is turned 90 degrees and the fanning is repeated in a ventral to dorsal plane. The probe is then moved to the next site of evaluation and the process is repeated until all of the following sites have been evaluated: left kidney (spleno-renal), xyphoid (diaphragmatico-hepatic), right kidney (hepato-renal), urinary bladder (cysto-colic). The finding of free fluid is a positive result. If free fluid is identified then attempts should be made to collect a sample for evaluation either via ultrasound guidance of blind 4-quadrant tap. Fluid should be assessed for PCV/TS, creatinine and potassium to rule out hemoabdomen and uroabdomen respectively.
Orthopedic and cutaneous wounds are finally identified. Patients should be evaluated for puncture wounds, abrasions, lacerations or bruises. Any areas of possible cutaneous injury should be gently clipped to allow for complete evaluation. Long bones and joints are assessed for fractures or disarticulations.

Laboratory evaluation may include PCV/TS, lactate, BUN as separate tests or may be included as part of a venous blood gas analysis. PCV is often normal following trauma, even when hemorrhage has occurred, due to splenic contraction however TS will generally fall when significant hemorrhage has occurred and may be a better indicator of the degree of bleeding. Lactate is a byproduct of anaerobic metabolism and indicates that oxygen delivery to the tissues is inadequate to keep up with patient needs and is a sensitive indicator of shock. Routine radiography of the thorax following any blunt trauma should be considered to rule out occult pneumothorax or pulmonary contusion. Radiographs should be obtained of any identified long bone or joint abnormalities with the region of interest centered in the beam.

Treatment of trauma patients can be divided into primary and secondary phases. The primary phase or resuscitative phase of treatment focuses on restoration of systemic and local oxygen delivery to pre-injury levels. The second phase begins once normal oxygen delivery has been restored and lasts until the patient is discharged from the hospital. The focus of the second phase of management is maintenance of oxygen delivery and definitive treatment of wounds, fractures and pain.

The ultimate success or failure of managing the severely traumatized patient is often decided within the first hours of presentation during the initial evaluation and stabilization. Successful resuscitation begins with identification of shock. The main goal of resuscitation should be re-expansion of the effective circulating volume with re-establishment of systemic and local blood flow. Resuscitation should not be considered to be complete until certain endpoints have been reached. Traditional endpoints of resuscitation include normalization of heart rate, respiratory rate, pulse quality and blood pressure. Newer, goal directed endpoints have been useful in identifying occult shock and should be incorporated into resuscitation goals. These include normalization of blood lactate and base excess and central venous oxygen saturation.

Initial treatment of a patient in shock should be administration of supplemental oxygen with early volume expansion. Crystalloid only resuscitation has been the mainstay of treatment for shock for many decades. It has the advantage of being relatively cheap and is readily available. Typically a replacement crystalloid such as lactated Ringer’s solution or 0.9% NaCl is administered rapidly in aliquots of 20-25 mL/kg IV until endpoints are reached or the maximum dose of 90 mL/kg has been reached. While effective the duration of volume expansion associated with crystalloid only resuscitation is short (30 minutes); this can be extended by incorporating synthetic colloids into the resuscitation protocol. A useful technique is to alternate doses of crystalloids with doses of colloids in 5 mL/kg aliquots until endpoints are reached or maximum doses of 90 mL/kg crystalloid and 20 mL/kg colloid are reached. Low volume resuscitation has been utilized for several years and is effective at restoring oxygen delivery while limiting over-resuscitation and associated tissue edema that delays healing. This is accomplished by administering 4-6 mL/kg 7.2% NaCl with 10-20 mL/kg synthetic colloid followed by crystalloids as needed. This approach generally reduces the overall fluid needed to reach endpoints and can be used in any trauma patient that was not severely dehydrated at the time of the traumatic event but is particularly useful in animals with cavitary hemorrhage or brain injury.

Early analgesia should be considered an important part of the resuscitative phase of treatment. As soon as it is evident that death is not imminent analgesia should be administered. Since trauma patients are dynamic the best analgesic choice is a pure opioid agonist such as morphine or hydromorphone. These drugs are effective, have no ceiling effect and are fully reversible. Typical starting doses are 0.05-0.1 mg/kg hydromorphone or 0.2-0.3 mg/kg morphine IV, IM or SC.

The treatment of wounds during the resuscitation phase focuses on preventing further wound contamination and tissue injury. All visible wounds should be clipped and cleaned with an antiseptic solution and lavaged with saline or tap water. Following lavage the wounds should be gently probed for depth and extent and then covered with a sterile non-adherent dressing until stabilization has been achieved and definitive management is possible. Any confirmed or suspected fractures of the distal limbs should be immobilized by placement of a modified Robert-Jones bandage or splint ensuring that the joint above and below the fracture are included. Fractures of the proximal limbs are not stabilized with external coaptation due to the risk of creating a stress riser and causing injury to important nearby structures (arteries, veins, nerves).

The secondary phase of trauma management begins when resuscitation endpoints have been met and the patient is either admitted for further care or is moved to surgery for definitive management of wounds or fractures. Almost all patients that suffered trauma severe enough to require surgery or hospitalization will require some fluid therapy. Due to the large volumes of crystalloids that many of these patients receive during resuscitation care must be taken to adjust ongoing fluid plans to meet patient needs without exacerbating edema or causing fluid overload. To ensure that fluid administration is not exceeding patient needs, serial weights may be used with any weight gains likely reflecting retained fluid. Many severely traumatized patients are moderately to severely hypoproteinemic making interstitial edema more likely to occur. Administration of synthetic colloids can be considered to minimize fluid leakage from the intravascular space but significant controversy exists about their routine use.

As with the resuscitative phase, appropriate analgesia is an important part of the management of trauma during the secondary phase. Pain causes a neuroendocrine response that increases levels of catabolic hormones including cortisol while decreasing anabolic
hormones such as insulin and impairing healing. Patients suffering trauma should be considered painful even if they are not demonstrating overt signs of pain. The best approach to pain management in trauma is multimodal therapy. Pure opioid agonists are the mainstay of treatment and can be safely used in even severely traumatized patients due to their cardiovascular sparing characteristics and reversibility. Respiratory depression is uncommon in veterinary patients when appropriate doses are used. Ketamine is dissociative agent NMDA antagonist that has some analgesic properties and modifies central sensitization that can lead to chronic pain syndromes or disproportionate pain responses. When used ketamine should be administered for at least 24 hours as a CRI at a rate of 5-15 mcg/kg/min in conjunction with an opioid. Alpha-2 agonist agents such as dexmedetomidine are also useful analgesic drugs at doses lower than those used for sedation. Dexmedetomidine is typically used as a CRI at a rate of 0.5 to 2 mcg/kg/min/hr.

When possible local or regional anesthetic techniques should be used including epidural administration, nerve block or diffusion catheter placement.

Non-steroidal anti-inflammatory drugs may be used in trauma patients once hemodynamic stability has been returned and abrupt changes are no longer anticipated. Typically NSAID administration is delayed until 24 hours after trauma for patients admitted to the hospital. The decision to use NSAIDS should be deliberate following careful consideration of the possible negative side effects. NSAIDS should not be used in any patient with evidence of hematuria or pigmenturia. The routine use of corticosteroids should be avoided due to the potential side effects including immunosuppression and delayed wound healing.

Traumatized patients require adequate caloric intake to heal. Following admission to the hospital a nutritional plan should be formulated beginning with calculating the basal energy requirements of the patient [(30 x BW in kg) + 70]. If the patient is not eating voluntarily then a feeding tube may be required to meet nutritional needs. The goal should be to reach the full BER within 48 to 72 hours from admission.

The routine use of antibiotics is not necessary unless the patient presented with grossly contaminated wounds or has evidence of infection. When necessary, first generation cephalosporins are generally adequate for wounds not associated with animal bites. Patients that have been bitten by an animal should be treated with a potentiated penicillin or fluorquinolone to cover common oral pathogens. Ideally antibiotic therapy would be guided by culture and sensitivity results. The risk of infection can be reduced through careful handling of all catheter sites and by keeping wounds and surgical incisions covered with a clean and dry bandage at all times. As always, the use of barrier protections (i.e. gloves) is recommended to prevent inadvertent colonization of an immunocompromized patient with potential pathogens such as methicillin resistant staphylococcus species.

Perhaps the most important aspect of the secondary management phase is monitoring the patient for evidence of hemodynamic compromise, organ failure or infection. Regular monitoring of temperature, respiratory rate and heart rate will identify patients potentially developing complications. Monitoring of blood pressure will be useful if the patient has had episodes of hypotension. Serum biochemistry analysis and complete blood count should be performed every 2-3 days as the patient’s condition dictates to identify possible organ failure early. Traumatic coagulopathy can occur in severely traumatized patients and any patient with unexplained bleeding or bruising should be evaluated with a platelet count and PT/aPTT.

High quality nursing care is imperative for the successful management of a severely traumatized patient. Every effort should be made to keep the patient clean and comfortable. Fecal and urine contamination should be cleaned as soon as they are identified. Patients should be kept on deep, soft bedding and should be repositioned every 4 hours if they are not ambulatory or moving on their own to prevent decubital ulcers from forming. All incisions and wounds should be evaluated at least once a day and soiled or wet bandages should be changed immediately upon recognition. Early mobilization of the patient will maintain joint health and aid in the management of ileus associated with recumbency and opioid administration. Passive-range-of-motion exercises are also useful to maintain range of motion and lymphatic flow.

Determination of prognosis can be difficult due to the confounding effect of euthanasia. Factors that have been associated with poorer outcomes include: head trauma, vertebral fractures, hemobadomen and the need for mechanical ventilation. The use of scoring systems may aid in guiding decision making but care should be taken to prevent using scores as a decision making tool for individual patients. The animal trauma triage (ATT) score assigns a score from 0-3 in six categories (perfusion, cardiac, respiratory, eye/muscle/integument, skeletal, neurological) with a maximum total score of 18. The risk of death has been shown to increase by 2.3 to 2.6 times for every one-point increase in ATT score. The modified Glasgow coma score (MGCS) can be used to serially monitor patients with head trauma and traumatic brain injury by assigning a score from 1-6 in three categories (motor activity, brain stem reflexes, level of consciousness) with a minimum score of 3 indicating the worst possible neurological performance. Total MGCS scores of less than 8 have been associated with a 50% mortality rate at 48 hours. In general the prognosis for animals suffering trauma is good with survival rates above 90%.
Identifying patients that might be at risk for development of refeeding syndrome is the first step in the management of the condition. Certain risk factors have been identified in people and are also present in the veterinary reports. The classic risk factor for refeeding syndrome is chronic malnutrition in which total energy and nutrient quality are both deficient. An often-overlooked cause of chronic malnourishment is conditions that result in malabsorption of ingested nutrients. Dogs and cats with severe intestinal disease or pancreatic insufficiency may be unable to absorb adequate nutrients despite having a normal appetite and nutrient rich diet. Similarly, patients that have been completely anorexic (no caloric intake) for > 7-10 days have an increased risk of developing refeeding. The last significant risk factor for veterinary patients is obesity with rapid weight loss.

Under homeostatic conditions net nutrient balance is 0. Meaning that the sum of energy intake is equal to the sum of energy consumption by the body and energy excretion. In order for growth to occur a positive energy balance is needed. Similarly, for protein synthesis to occur, a positive nitrogen balance is necessary. When energy consumption exceeds that needed for maintenance of daily needs, excess energy is stored in the form of adipose tissue for use in times of decreased energy availability. This storage mechanism allows for an energy reserve to be established for use at a later date if needed. Excess nitrogen intake unfortunately, does not result in storage of nitrogen. Rather, excess nitrogen intake only results in an increase in nitrogen excretion. During times of starvation or inadequate nutrient intake it is possible for animals to be severely protein deficient but still have adequate caloric energy to meet RER in the form of stored adipose tissue. Therefore, animals that are consuming protein deficient diets may be at increased risk of developing refeeding syndrome despite having some enteral caloric intake in the form of carbohydrate or fat. When starvation conditions are present the energy needs of the vital organs are met in the following order: brain, kidneys, maintenance of blood sugar, physical activity and finally maintenance of body condition.

The physiology of starvation can be divided into an acute response (that occurring within the first 2 weeks of starvation) and a delayed response (that occurring greater than 10 days after the onset of starvation). During the initial phase of starvation or anorexia there is a voluntary reduction in physical work and an involuntary reduction in basal metabolic rate. The decreased metabolic rate is characterized by a decrease in protein turnover, and decreased levels of thyroid hormones and somatomedins. If these changes do not result in equilibrium of energy demand and energy intake then endogenous fuels (adipose tissue, muscle) are used to balance the equation. As mentioned above, triglycerides are the major form of fuel storage in mammals and the body’s fat supply is the major determinant of the length of survival under starvation conditions. Carbohydrates are not stored in any significant amount and are therefore of little value with liver and muscle glycogen stores accounting for less than one days worth of caloric needs. While body protein could account for up to 2 weeks of caloric requirements, its depletion would have profound adverse effects due to its role in essential non-fuel functions. The metabolic alteration that occurs under starvation conditions is mediated by changes in circulating hormone levels with decreases in insulin and triiodothyronine (t3) and increases in glucagon, growth hormone, catecholamines and plasma cortisol. The end result of these hormonal alterations is enhanced hepatic glycolgenolysis, gluconeogenesis and skeletal muscle proteolysis thereby facilitating lipolysis. The brain is subsisting on glucose generated from protein catabolism and gluconeogenesis in the liver. During the delayed response to starvation there is a major shift from glucose to fat as the main energy source. Gluconeogenesis is reduced during this period and protein catabolism is minimized. Ketone bodies from hepatic oxidation of fatty acids are used by most of the tissues for energy. At this stage the brain is reliant on ketone bodies as an energy substrate. It is also during this delayed response to starvation that the kidneys begin to assume the role of an important glucose-producing organ.

Besides the hormonal and metabolic changes taking place, compositional changes are also occurring. Weight loss during starvation is greatest during the first few days due to a disproportionate loss of water. At the same time, body fat and lean body mass are reduced. Bone mass is preserved in nearly all cases unless malnutrition or starvation is prolonged. The reduction in total body water is greatest during the first 10-14 days. Within the first 48 hours of complete anorexia natriuresis occurs leading to significant extracellular and subsequently, intracellular fluid loss. Urinary sodium excretion decreases significantly after 7-14 days of complete anorexia. Natriuresis can be prevented by consumption of even very small amount of dietary carbohydrate. In addition to sodium loss, potassium, phosphorus and magnesium are lost due to catabolism of cell mass for use as an energy substrate. Although total body potassium, phosphorus and magnesium levels are depleted, serum levels are maintained.

The metabolic response to refeeding following starvation or lengthy malnourishment results from a major shift back to the use of carbohydrates as the primary energy substrate instead of fat. When patients are fed with carbohydrate and protein there is an immediate increase in circulating insulin levels. This insulin secretion inhibits glycogenolysis, gluconeogenesis and fatty acid mobilization by reducing glucagon levels. The same insulin secretion results in enhancement of cellular uptake of glucose, potassium, phosphorus and magnesium. Abrupt cessation of natriuresis occurs causing rapid expansion of the extracellular fluid compartment.
When the diet used for refeeding is composed of a high proportion of carbohydrate the cessation of natriuresis is abrupt and can lead to the development of peripheral edema and fluid overload. Refeeding with fat or protein alone will allow natriuresis to continue and may prevent fluid overload or edema formation from occurring.

In addition to the overall, global metabolic consequences of starvation and refeeding, there are effects on specific organ systems. During starvation the cardiovascular system is affected as cardiac muscle mass is reduced in parallel with loss of lean body mass. Cardiac function is impaired as catabolism of contractile sarcoplasmic proteins occurs and left ventricular mass is reduced. Cardiac dysfunction is characterized by a reduction in cardiac output (primarily through reduced stroke volume) and decreased ventricular compliance (reducing pre-load). Hypotension and bradycardia are the body’s attempt to reduce oxygen consumption and are not reflective of circulatory collapse. Upon refeeding there is a dramatic increase in heart rate, blood pressure, oxygen consumption, cardiac output and plasma volume. This rapid increase in metabolic demand may exceed supply and can lead to the development of congestive heart failure. Although the same metabolic and cardiovascular changes likely occur in veterinary patients there are no reports to date of congestive heart failure secondary to “naturally occurring” starvation and refeeding.

The effect of starvation on the gastrointestinal tract is early reduction in enterocyte formation and nutrient absorption. Decreased levels of brush border disaccharidases and pancreatic enzymes occur due to mucosal atrophy, loss of intraluminal stimulation and generalized protein depletion. Gut atrophy with decreased crypt cell proliferation and reduced villus height occurs as starvation progresses. Intestinal mass is reduced and thickening and coarsening of small intestinal mucosal folds occurs. Gastric acidity is reduced and both gastric and intestinal motility is decreased. Diarrhea occurs due to impaired absorptive ability, bacterial overgrowth, presence of unconjugated bile salts, hypoalbuminemia and gut edema. The development of diarrhea exacerbates electrolyte loss and leads to further whole body depletion of phosphorus, potassium and magnesium.

Arguably, the most important metabolic consequences associated with starvation and refeeding are the effects on total body phosphorus (during starvation) and serum phosphorus (upon refeeding). Phosphorus is present in both an inorganic and organic form within the body with organic phosphate being the most abundant intracellular anion. Organic phosphate is found within phospholipids, nucleic acids and many enzymes. It is important for maintenance of plasma membranes, the electron transport chain. ATP and 2,3-diphosphoglycerate. Inorganic phosphate is found in the extracellular fluid and is used as a substrate for many important functions including glycogenolysis, 2,3-DPG production and oxidative phosphorylation. As mentioned above, during starvation, total body phosphorus becomes depleted despite serum phosphorus usually being maintained within the normal range. Upon refeeding phosphorus moves from the extracellular fluid compartment to the intracellular fluid compartment to be used for the synthesis of phosphorylated compounds. When hypophosphatemia occurs secondary to refeeding it can be dramatic and may be seen within the first 24-72 hours although delayed hypophosphatemia can be seen up to 10 days following reintroduction of food. The consequences of hypophosphatemia can be severe and most of the clinical consequences associated with refeeding syndrome are attributed low serum phosphorus. Some of the consequences of hypophosphatemia include cardiac arrhythmias and contractile dysfunction, central nervous system dysfunction (including seizures), cellular hypoxia (due to 2,3-DPG depletion and shift of the oxyhemoglobin dissociate curve), leukocyte dysfunction, thrombocytopenia and impaired clot contraction; all of which are due to limitations in ATP and 2,3-DPG levels. In addition to its importance in cellular respiration, ATP is important for the maintenance of red blood cell (RBC) membrane integrity, cell shape and RBC deformability. ATP depletion can lead to decreased cell deformability and osmotic lysis due to impairment of RBC sodium-potassium pump function. The subsequent increased red blood cell fragility leads to perhaps the most devastating consequence of hypophosphatemia: intravascular hemolysis.

Alterations in serum potassium and magnesium levels also occur upon refeeding and can be severe in patients that have whole body depletion of potassium and magnesium. When serum insulin levels increase secondary to refeeding extracellular potassium is shifted to the intracellular compartment. Trans-cellular shifting of potassium with subsequent hypokalemia can lead to muscle weakness, ileus, cardiac arrhythmias, and rhabdomyolysis. Magnesium plays an important role in many body systems, acting as a cofactor involved in energy storage and utilization, protein synthesis, CNS function and neuromuscular function. Serum magnesium undergoes a similar trans-cellular shift when refeeding occurs and new tissue synthesis begins. The clinical signs of hypomagnesemia are similar to those seen with hypokalemia making it difficult to determine what is responsible for the clinical signs that are present. Although hypophosphatemia, hypokalemia and hypomagnesemia are each capable of individually causing the clinical signs associated with refeeding it is probable that the combination of electrolyte abnormalities is ultimately responsible. Thiamine deficiency may play a role in the development of clinical signs associated with refeeding syndrome although no definitive evidence exists. Thiamine is an important cofactor in carbohydrate metabolism and thiamine deficiency can lead to CNS dysfunction, muscular weakness and cardiomyopathy.

The prevention of refeeding syndrome begins with awareness and recognition of patients that are at risk. As previously mentioned risk factors for veterinary patients to develop refeeding syndrome include chronic undernourishment, obesity with rapid weight loss, prolonged fasting or complete anorexia of greater than 7-10 days duration. When a patient with one or more of these risk factors is identified and it is imperative to formulate a comprehensive nutritional plan in addition to a treatment plan for any underlying medical conditions. Patients should have complete blood work performed and any electrolyte abnormalities should be corrected prior to refeeding.
The first step in formulating a nutritional plan is to determine the patient's caloric needs by calculating the basal energy requirement (BER) using one of the following formulas: 1. Kcal/day = 30(BW in kg) + 70 or 2. Kcal/day = 70(BW in kg)^0.75. Basal energy requirement is the amount of energy required to maintain the body’s minimum normal metabolic activity. When calculating the BER there is some controversy about whether the ideal body weight or actual body weight should be used. It is likely that either method is acceptable provided the patient is monitored for refeeding syndrome. The goal of the first week is to meet the calculated BER.

Glucose should be provided at 150 to 200 g dextrose per day and lipid should account for 20-30% of non-protein calories. Protein should be provided at 1.2 to 1.5 grams/kg/day. There should be no attempt made to achieve weight gain during the first week of treatment and any weight gain that does occur should be considered to be due to fluid retention rather than addition of lean mass. There is little evidence that supplementing electrolytes prevents the development of refeeding syndrome and should not be used as a replacement for development of a comprehensive nutritional plan. The provision of electrolytes should be guided by measurement of serum levels. Although not documented, thiamine deficiency is a theoretical concern and supplementation with vitamin B complex can be considered. Due to the risk of fluid overload, measurement of serial body weights should be considered mandatory and consideration should be given to measurement of central venous pressure.

Once refeeding has begun electrolytes should be monitored at least once a day and should include measurement of serum phosphorus, potassium, sodium and ionized calcium and magnesium. After all, early recognition of refeeding syndrome is essential for successful treatment. Refeeding syndrome is most likely to occur within the first several days of refeeding although its onset can be delayed up to 10 days. If electrolyte changes consistent with refeeding syndrome develop then nutritional support should be stopped immediately and aggressive correction of electrolyte abnormalities should be pursued. Supplementation of potassium may occasionally exceed the recommended maximum of 0.5 mEq/Kg/hr. Phosphorus supplementation rates of 0.3 to 0.6 mM/kg/hr. can be used and magnesium supplementation can be attempted at 0.75 to 1 mEq/kg/day. If hypophosphatemia is not recognized and hemolysis develops the patient should receive blood products as needed to prevent signs associated with anemia and phosphorus supplementation should be begun. Following the first week of nutrition the patient’s caloric prescription should be increased by 10 to 15 percent. Upon correction of electrolyte abnormalities, nutritional support can be restarted but should be reduced by 20-30%. Following the initial refeeding period of 7-10 days, the nutritional prescription can be increased to create an anabolic state and allow for lean body mass repletion.

Perhaps the most difficult aspect of preventing and treating refeeding syndrome is convincing the owners/rescuers and hospital staff that a slow and methodical approach to refeeding is in the patient’s best interest. A frank discussion with staff members is recommended immediately upon recognition of an at risk patient to ensure that a thoughtful nutritional plan can be formulated. Although it is with the best intentions that rescuers and staff offer food to dogs and cats rescued from starving conditions, that approach can have dire consequences. Veterinarians who are in a position to evaluate patients rescued from hoarding conditions or those responding to natural disasters should brief support personnel prior to seizure of pets to ensure proper introduction of nutrition. Once hospitalized, the best way to prevent overzealous feeding is to delegate responsibility of feeding to one person within the hospital. The attending veterinarian should continue to update staff on the expectations and possible consequences of refeeding syndrome ensuring the staff that they have the patient’s best interest at heart to prevent staff members, family or rescuers from sneaking food to the animal. Ultimately, the best treatment for refeeding syndrome is prevention, and prevention begins with awareness.
The use of targeted endpoints to guide resuscitation from shock has been in routine use in human medicine for the last 10 years with most evidence directed at resuscitation from septic shock. The utility of endpoints lies in their ability to alert the clinician to the possibility of incomplete resuscitation or occult shock. By evaluating specific endpoints the treating veterinarian is able to determine if a different approach to shock resuscitation is necessary or if addition of vasoactive agents is indicated. The ease with which endpoints are reached may also provide the veterinarian with prognostic information.

The determination of when resuscitation from shock is complete has traditionally relied on normalization physical examination findings. Evaluation of the patient’s mentation has always been (consciously or unconsciously by the veterinarian) the most utilized traditional endpoint. The goal is to have a patient that is bright and alert and, assuming that the neurologic and skeletal systems allow, ambulating. Evaluation of mentation is cheap, easy and non-invasive but may be complicated by co-morbidities including traumatic brain injury or metabolic disease. In addition the brain may be affected much later in shock than other organ systems due to the body’s ability to maintain cerebral perfusion over a wide range of blood pressures. Serial monitoring of the heart rate with the goal of normalization for species, age and breed is also cheap, easy to perform and non-invasive. Unfortunately, the heart rate can be elevated due to physiologic responses not secondary to shock, such as pain and anxiety. When heart rate is evaluated it may provide a better indication of volume status than actual tissue oxygen delivery. Mucous membrane color is often evaluated in conjunction with heart rate and offers the same advantages with the added advantage of providing an insight as to the oxygen content of hemoglobin. The major drawbacks to evaluation of mucous membrane colors are their lack of sensitivity and its subjective nature.

Traditional resuscitation endpoints that are more specifically used to evaluate perfusion include pulse quality, capillary refill time, blood pressure and body temperature. Evaluation of pulse quality is cheap and non-invasive but requires a certain degree of skill and experience and is highly subjective. While useful for providing a crude evaluation of macrovascular function, it does not provide any information about microvascular function and may be altered by regional perfusion disturbances. Capillary refill time is the only perfusion parameter evaluated on the physical examination that attempts to evaluate microvascular function but is also crude and non-specific. Measurement of blood pressure can be done with the use of Doppler technology or oscillometric monitors. Targeting a systolic blood pressure of greater than 90 mmHg or mean arterial pressure of greater than 60 mmHg is still one of the mainstays of resuscitation. Blood pressure determination gives the clinician a good overall estimation of macrovascular performance but again, does not correlate with microvascular perfusion. Additionally, depending on the technology in use, obtaining an accurate blood pressure reading requires both skill and experience on the part of the operator. Finally, body temperature can be used to evaluate perfusion indirectly. While core body temperature will be affected in the later stages of shock when compensation is no longer complete, the gradient between core body temperature and peripheral limb temperature may be abnormal earlier. As with most of the traditional resuscitation endpoints, evaluation of body temperature or temperature gradients can be affected by co-morbidities or by pharmacologic interventions.

Given the subjective and non-specific nature of most of the traditional resuscitation endpoints, alternatives were sought that would provide more specific information regarding the delivery and use of oxygen on a global level. Venous oximetry (evaluation of venous hemoglobin saturation) provides information about tissue oxygen utilization. As tissue oxygen levels decrease a greater portion of oxygen is extracted from hemoglobin as it traverses a tissue bed. This holds true insofar as lung function is normal and arterial hemoglobin is fully saturated with oxygen upon leaving the lung. Targeting a central venous oxygen saturation of greater than 65% ensures that blood oxygen content is not the factor responsible for shock. Unfortunately, venous oximetry has drawbacks including the impact of co-morbidities, the requirement for central venous catheterization and the need for blood gas measuring equipment. Additionally, the value of venous oximetry is highly dependent on the location the sample was collected from. The further away from the hear the sample is collected from the more it reflects the specific tissue beds it drains, meaning in that blood collected from a cephalic vein only reflect oxygen consumption in the limb distal to the venipuncture site.

Central venous pressure measures the amount of hydrostatic pressure within the cranial vena cava and provides a direct assessment of vascular volume and an indirect assessment of cardiac pre-load. Measurement and normalization of central venous blood pressure allows for the exclusion of hypovolemia as the cause of shock. When CVP is utilized as a resuscitation endpoint the goal should be to achieve a CVP of 8-12 mmHg (11-16 cm H20). Utilization of CVP as a resuscitation endpoint can help prevent morbidity associated with over-resuscitation. Although relatively easy to perform, measurement of CVP requires a water manometer or multiparameter monitor capable of measuring invasive pressures. A moderate to high skill level is needed to successfully and correctly place a central venous catheter in a patient in shock and is often a limiting factor in the use of CVP in veterinary medicine.
Assuming that no co-morbidities exist and that the patient was euhydrated at the onset of shock, urine output is a good resuscitation target. Normal urine production of greater than 1 mL/kg/hr indicates that renal perfusion is adequate to maintain normal GFR making it unlikely that a significant perfusion derangement exists. To be utilized however, the patient must have an indwelling catheter placed and a closed collection system attached. Although this is easy to achieve in most male dogs, it is much more difficult in female dogs and cats of both sexes making its use infrequent.

Finally, the use of biochemical markers of shock allows for the assessment of cellular respiration and utilization of oxygen. Base deficit and lactate have both been utilized as resuscitation endpoints in both human and veterinary medicine. No clear evidence exists that would justify the use of one over the other and in fact they may be most useful when evaluated in conjunction. Base deficit reflects the degree of intracellular acidosis and therefore the presence or absence of anaerobic metabolism. It is easy to obtain and is a good indicator of tissue hypoxia. Diseases affecting both the kidneys and acid-base status independent of cellular respiration can affect base deficit. Lactate is a byproduct of anaerobic metabolism and can be used to estimate the degree of anaerobic metabolism that is occurring. Similar to base deficit however, lactate can be elevated for reasons other than tissue hypoxemia (i.e. type B lactic acidosis). When biochemical monitoring is being used to guide resuscitation the goal should be to achieve a base deficit of no greater than 5 mmol/L and a lactate of less than 2.5 mmol/L.

Perhaps the most clinically useful approach to resuscitation incorporates both the traditional and goal directed endpoints. When abnormalities are present with the traditional endpoints it is reasonable to provide resuscitation until resolution of these abnormalities occurs. At that time the evaluation of goal directed endpoints could be performed to investigate the possibility of occult shock or ongoing oxygen debt. By having endpoints of resuscitation in mind prior to treatment of a patient in shock the clinician can better determine when resuscitation is complete or if more aggressive means must be implemented.
The premise for any dental extraction of flap procedure is to appreciate the anatomy as well as the surgical principles necessary for this procedure to be accomplished.

Before any dental extraction should be undertaken, it is ESSENTIAL that a veterinarian has prior appreciation of the root structure of the tooth being removed. The only way this can be accomplished is via dental radiography. While many times complete removal of the affected tooth can be accomplished without radiography, can the veterinarian absolutely claim that there are no tooth shards, bone fragments, or tooth root structures remaining? Failure to completely remove the tooth in entirety without any remnants is tantamount to malpractice. In many clinical situations, existing endodontic disease causing apical periodontitis (abscess) will remain unless the entire tooth root is removed.

The flap procedure is an important consideration, as the veterinarian must choose a flap that allows exposure of the affected tooth and TENSION FREE CLOSURE without disruption of the neurovascular component of the gingiva. The ENVELOPE flap is considered with marginal amount of buccal or labial bone is needed to be removed to accomplish this extraction. This flap, as with others, must extend beyond the mucogingival line to allow for unattached gingiva to be released and facilitate a tension-free closure.

Vertical releasing flaps (single or bilateral) are mucoperiosteal flaps that allow the veterinarian full exposure of the tooth to be removed, thus allowing more buccal bone to be removed to facilitate extraction.

Three (3) Principles of flap surgery are as follows: If a vertical release is to be made, make it on the adjacent tooth at the appropriate LINE ANGLE, not the tooth to be removed. This is important in that you do not want your suture line over the alveolus, but rather over healthy bone (no suture line over a defect). Secondly, preserve blood supply and thirdly, selection of instruments that minimize tissue damage.

**Line angle definition**

This is an imaginary vertical line forming the intersection of two adjacent vertical dental surfaces. This denotes a specific position on a tooth and are important surgical landmarks. (Verstraete F, Lommer M. Oral and Maxillofacial Surgery in Dogs and Cats.)

The flap exposure is best accomplished after extraction by excision of the mucoperiosteum. Utilization of a scalpel blade nick followed by sharp dissection of this is made to allow the flap to fully cover the surgical extraction site. Any marginal tissue that looks irregular and inflamed should be removed.

4-0 poliglecaprone-25 with a reverse cutting edge needle is chosen as the oral suture material. This suture is rapidly absorbed and the reverse cutting edge is used to minimize inadvertent tissue tears. Since it is a monofilament suture, it pulls freely through tissue, has good knot security, stays in the mouth longer than chronic gut, and does not require removal. Simple interrupted patterns (2-3 mm apart) are needed.

Scalpel blade selection should be via a #15 or #15C blade. Rarely, a #11 surgical blade is needed. Proper instrumentation is needed to adequately remove large teeth. It is recommended to have a winged elevator kit (2-8), which allows one variability in selection of elevators. Periosteal elevators are ESSENTIAL for adequate flap preparation and a couple of sizes are needed for both the small dog and cat as well as for large breeds.

In order to facilitate extraction of teeth, a high speed dental unit is a must. One cannot adequately remove large single rooted or multirooted teeth without the benefit of a good high speed unit. BEFORE undertaking any dental extractions, the veterinary would be wise to consult with his/her distributor for a unit. There are many good units in the marketplace, and each has its own benefit or even restriction. It is recommended, however, to go the extra mile and purchase fiberoptics in your high-speed handpiece. Swivel-tip handpiece is also recommended.

**Bur Selection:** #1/2, #1, #2, #4, #6 round; 701 cross cut; medium grit football diamond; medium grit round diamond (I recommend surgical length burs in addition to the regular length)

Bone graft materials aid in filling the open socket with bone and connective tissue rather than allowing it to collapse or granulate in with soft tissue. Collapse of the socket can further alter facial features slightly because there is no longer any crown structure to support that portion of the upper lip, which is now vulnerable to trauma by the mandibular canine tooth. (for maxillary canine extraction). If there is marginal amount of ventral cortex remaining from extraction of a mandibular 1st molar tooth, a graft is warranted. Graft of mandibular canine teeth is also recommended if stability of the bone is needed. However, there is no substitute for a good clot formation in the site to help promote new bone formation.
Tips for extractions of the following teeth

**Maxillary canine**
- Single vertical diverging releasing incision advised (between the maxillary canine tooth and the maxillary lateral incisor tooth or mesial line angle of the maxillary canine tooth)
- Use larger round bur (#4, #6) to remove buccal bone and DO NOT remove bone mesial or distal to the tooth itself
- Use #170, 701, 699 crosscuts OR #1 round bur to make moat. Make moat only wide enough to accommodate the winged elevators or luxators only.
- Start out by taking buccal bone 2/3 or greater to the apex of the tooth. (remember to follow the contour of the tooth) As you get better at removing teeth, you can take off less buccal bone
- Periosteal release is a MUST and freshen edges prior to closure
- Always perform alveoplasty (football or round diamond)
- If dog or cat has ONF, remove the epithelial downgrowth tissue PRIOR to closure, otherwise the flap will fail
- Tension-free flap (for all extractions)
- Simple interrupted pattern 2-3 mm apart. DO NOT USE PDS
- E-collar may be needed for cases

**Maxillary 4th premolar tooth**
- Single or bilateral diverging incision. Make sure you DO NOT make incision over tooth you are extracting
- Careful of the parotid papilla if making a bilateral diverging incision
- Remove buccal bone with #4 or #6 round bur
- Moat with same burs you use in canine
- Section all 3 roots prior to removal, regardless if tooth is mobile or not
- Remove caudal crown cusp adjacent to the maxillary 1st molar tooth to facilitate straight-line luxation/elevation. Care needed to avoid contacting the 1st molar tooth
- Remove middle section of tooth if need be to allow straight line removal (OR amputate crowns for better visualization of crowns to facilitate extraction)
- Remove interradicular bone between the mesiobuccal root and the palatal root (after removal of the mesiobuccal root)
- Make a moat around the palatal root carefully with very fine cross cut (#170, 701, or 699) or round (1/4-1/2 round)
- Surgical burs are a must in difficult extractions so keep some on board (#2, #1, #1/2, 701)
- Alveoplasty needed
- Avoid neurovascular bundle at infraorbital foramina

**Mandibular 1st molar tooth**
- Vertical incision at line angle of the mesial or distal aspect of the 4th premolar tooth, NOT at the mesial aspect of the 1st molar (as roots can diverge)
- Envelope flap if practical
- Amputate mesial and distal cusps of 1st molar to facilitate straight-line access
- Remove middle section of tooth to facilitate extraction if needed
- Alveoplasty after extraction, especially on the lingual mandibular marginal bone

**Mandibular canine tooth**
- Preservation of labial frenulum advised
- Vertical release from mesial line angle of the canine tooth
- Care with buccal bone removal to avoid the mental foramina (use periosteal elevators to protect this area and avoid the bur macerating/lacerating the vessels/nerve)
- Release of lingual gingiva needed
- Tension-free closure
- E-collar needed

**Bone grafts**
The most common types of bone grafts in veterinary medicine are osteoinductive and osteoconductive agents. Osteoinduction is a chemical process by which molecules contained in the graft (bone morphogenetic proteins) convert the neighboring cells into osteoblasts, which in turn form bone. An example of this is a decalcified freeze-dried bone allograft (DFBDA). Osteoconduction is a physical effect by which the matrix of the graft forms a scaffold that favors outside cells to penetrate the graft and form new bone. In veterinary dentistry, freeze-dried bone allograft (FBDA) or more commonly a synthetic bioactive glass are examples of this type of osteoconductive agent.
Equipment list

- Dental radiograph system (Progeny generator; digital dental sensor)
- High Speed/low speed dental system
- 4-0 and 5-0 Monocryl (or generic) or Chromic Gut
- #15 or #15c surgical blades
- Winged elevator kit (2-8)
- Periosteal elevators (EX8-108; EX9-108)
- Thumb forceps
- Assorted burs (#1/2, #1, #2, #4, #6 round); 701, 701L Surgical crosscut burs
- Diamond burs (medium grit football)
- Goldman-Fox scissors
- Kelly Straight Scissors
- #100C and 1.3S luxators
- Cawood-Minnesota retractors
- Root tip pick
- Small needle holders (with or without scissors)
- Consil or Oste-Allograph Perio Mix
When a dog has a malocclusion, it can affect that pet throughout their entire lifetime. Left undiagnosed and untreated, these pets suffer unnecessarily. It is the job of the family veterinarian to identify that pathology and recommend treatment options as soon as practical. Malocclusions that do not cause oral pain or discomfort may not need to be corrected, and the scope of this lecture is addressing those occlusion issues that indeed cause oral discomfort. Ethical considerations should be made when altering an abnormal bite for personal gain in the show ring or for masking an underlying genetic defect to further promote the gene pool.

The practitioner must know the dental anatomy of the dog regarding both primary and adult teeth. There are 28 primary and 42 teeth in the dog. **The dental formula is available in many veterinary dental textbooks.**

### Eruption location and dates

<table>
<thead>
<tr>
<th>Primary (deciduous teeth)</th>
<th>Incisors</th>
<th>3-4 wks</th>
</tr>
</thead>
<tbody>
<tr>
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<td>Canines</td>
<td>3 wks</td>
</tr>
<tr>
<td></td>
<td>Premolars</td>
<td>4-12 wks</td>
</tr>
<tr>
<td></td>
<td>Molars</td>
<td>No primary molars</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Permanent (secondary or adults)</th>
<th>Incisors</th>
<th>3-5 mos</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Canines</td>
<td>4-6 mos</td>
</tr>
<tr>
<td></td>
<td>Premolars</td>
<td>4-6 mos</td>
</tr>
<tr>
<td></td>
<td>Molars</td>
<td>5-7 mos</td>
</tr>
</tbody>
</table>

### Definition of normal occlusion

An ideal occlusion can be described as perfect interdigitation of the upper and lower teeth. In the dog, the ideal tooth positions in the arches are defined by the occlusal, inter-arch and interdental relationships of the teeth of the archetypal dog (i.e. wolf). This ideal relationship with the mouth closed can be defined by the following:

The maxillary incisor teeth are all positioned rostral to the corresponding mandibular incisor teeth. The crown cusps of the mandibular incisor teeth contact the cingulum of the maxillary incisor teeth. The mandibular canine tooth is inclined labially and bisects the interproximal (interdental) space between the opposing maxillary third incisor tooth and canine tooth. The maxillary premolar teeth do not contact the mandibular premolar teeth. The crown cusps of the mandibular premolar teeth are positioned lingual to the arch of the maxillary premolar teeth. The crown cusps of the mandibular premolar teeth bisect the interproximal (interdental) spaces rostral to the corresponding maxillary premolar teeth. The mesial crown cusp of the maxillary fourth premolar tooth is positioned lateral to the space between the mandibular fourth premolar tooth and the mandibular first molar tooth.

### Types of malocclusions

Neutroclusion (Class 1 malocclusion; MAL/1): A normal rostral-caudal relationship of the maxillary and mandibular dental arches with malposition of one or more individual teeth. Common abnormalities of MAL/1 include rostral and caudal crossbites, mesioversion, labioversion, lingoversion, disto evasion, and buccoversion. (You can also see each of these in other forms of malocclusion)

Mandibular distoclusion (Class 2 malocclusion; MAL/2): An abnormal rostral-caudal relationship between the dental arches in which the mandibular arch occludes caudal to its normal position relative to the maxillary arch.

Mandibular mesioclusion (Class 3 malocclusion; MAL/3): An abnormal rostral-caudal relationship between the dental arches in which the mandibular arch occludes rostral to its normal position relative to the maxillary arch.

Treatment of Malocclusions is based on a single premise…oral pain and discomfort. Orthodontic movement of maloccluded teeth simply for improvement in the show ring is unethical and is not recommended. Orthodontics should therefore be reserved for those patients with attrition (tooth on tooth contact) or oral discomfort due to contact to the mandibular or maxillary gingival mucosa.

**Basic tenets of maxillary/mandibular growth and orthodontic treatment in puppies:**

1. Growth of mandible and maxilla is under separate genetic control
2. Up to day 50 post partum, the increase in length of mandible occurs as a result of growth in ROSTRAL portion. After day 50, almost all increase in length in mandibular length is a result of growth in the region of the RAMUS. No change in distance b/w tips of central incisors and central cusps of mandibular 1st molar between 3-6 mos of age
3. More time between primary tooth extraction and permanent tooth eruption, better chances of success

**Treatment for MAL/1**
- **Rostral crossbite:** None if no attrition is present; Maxillary expansion device; labial arch bar
- **Caudal crossbite:** Extraction of the affected tooth
- **Lingoversion or linguodisplaced mandibular canines:** Crown reduction/vital pulpotomy; acrylic incline plane; crown extension of mandibular canines; removable orthodontic appliance (aka Kong therapy); extraction
- **Mesoverted maxillary canine teeth:** Active orthodontic appliance (maisel chain); extraction

**Treatment for MAL/2**
- Puppy or kitten: Extract mandibular canines and incisors (primary teeth)
- Adult:
  - Linguoversion or linguodisplaced mandibular canines can occur with this scenario and treatment is the same as MAL/1
  - Crown reduction and dentin bonding of offending mandibular incisors

**Treatment of MAL/3**
- Puppy or kitten: Extract maxillary canine teeth and incisors (primary teeth)
- Adult:
  - Linguoverted or linguodisplaced can still occur with this scenario and treat accordingly
  - Crown reduction and dentin bonding of the maxillary incisors that contact the mandibular gingival mucosa
  - Pay very close attention to the maxillary 3rd incisors to insure no attrition to the mandibular canine teeth. If so, extraction of the 3rd incisor is warranted. (or odontoplasty)

**Persistent primary teeth**
(Previously called retained deciduous teeth) should be treated immediately. A discussion in this lecture will address the permanent malocclusion ramifications of delayed treatment of persistent primary teeth. The Rule of Dental Succession will be emphasized: No successional and deciduous precursor teeth should be erupted simultaneously or in competition for the same dental arcade space AT ANY TIME.

Other clinical presentations requiring interceptive orthodontics: crowding, rotation, supernumary teeth.
Knowing What’s Abnormal on Dental Radiographs in Dogs and Cats
Barden Greenfield, DVM, DAVDC
MidSouth Veterinary Dental Referrals
Cordova, TN

When a practitioner opens his/her world to dental radiography, there will become a tremendous amount of pathology that becomes ‘visible’. This new awareness of oral pathology then puts the exclamation point on the ASSESSMENT part of the Oral ATP (Assessment, Treatment, Prevention). Otherwise, the practitioner is thinking their car is in working order by just a simple wash and wax. Neglecting to ‘check under the hood’ not only leaves potential oral pathology to linger causing potential oral pain and discomfort, but also gives the pet owner a false sense of security thinking the mouth is all good as their pet had their ‘dental’.

A benchmark study by Verstraete proved the value of intraoral dental radiography. His findings (226 dogs/116 cats) showed the following: This shows that almost 3/10 dogs and 4/10 cats that only receive a ‘dental’ go out of your door with oral pathology that needs to be identified. This shows that ‘where there’s smoke, there’s fire’ with regard to pathology under the gumline. 50% of both dogs and cats that have radiographically visible pathology have more findings that need attention.

In a recent JAAHA article, 16 predetermined categories of abnormal radiographic findings in 233 small breed dogs found almost 30% had abnormal findings. The most common tooth identified with pathology was the mandibular 1st molar (70%) followed by the maxillary 4th premolar (40%). Bone loss was noted in 15% of all findings. The conclusion was “full mouth radiographic evaluation should be performed to obtain important information for making accurate diagnoses”.

The indications for dental radiography are:
- Before and after extractions
- Periodontal disease
- Mobile teeth
- Discolored teeth
- Fractured teeth
- Gingival ulcers
- Missing teeth
- Malocclusions causing trauma
- Malformed teeth
- Gingival mass/bone swelling/soft tissue swelling
- Tooth Resorptions/root resorptions
- Pet dropping food
- Foul odor in mouth
- Reluctance to eat
- Reluctance to eat chews
- Nasal discharge

Before and after extractions
How would you know if the root has a curved apex or that there is indeed a root fracture? What if the mandibular 1st molar you plan to remove has its apex 1 mm from the ventral cortex of the mandible? A good dentist always knows the lay of the land before undertaking any procedure. As a radiograph is a legal document, this is the ONLY confirmation that the procedure you charged for was done to completion. Tooth root fragments/remnants are quite commonly left in the mouth with extractions.

Periodontal disease (PD)
Probing is the gold standard to assess bone loss and periodontal pockets. Radiography compliments this. There has to be 40% cortical bone loss for radiographic evidence of PD to be visible. The earliest sign of periodontal disease is the irregular alveolar margin between teeth. This should give the practitioner a sign that the periodontium is undergoing stress and needs treatment.

Discolored teeth
There is a 93% chance that discolored teeth are non-vital, but only 57% of those teeth will show evidence of premature maturation (tooth death). Regardless, it is important to radiograph all discolored teeth for evidence of apical periodontitis. Radiography will aid in deciding if this tooth is a candidate for endodontic therapy or extraction. Leaving a discolored tooth in the mouth without treatment is unwarranted and can be considered malpractice.
Fractured teeth
Even uncomplicated crown fractures (dentin exposure with no pulp exposure) can cause kill a tooth and cause apical periodontitis. Dentin is microscopically porous and bacteria can ingress through these pores and cause premature maturation (tooth death). Treatment options depend on the radiographic structures of the pulp cavity and the surrounding periapical tissue. Uncomplicated crown fractures can be restored with a composite restoration, or with evidence of tooth death, extracted or root canaled.

Complicated crown fractures (and crown-root or even root fractures), which are fractures extending into the pulp cavity, must be radiographed prior to treatment as mentioned above. Treatment options for this type of fracture is either extraction (exodontia) or root canal therapy.

Strategic teeth such as maxillary/mandibular canines, maxillary 4th premolars, 1st mandibular molars and even maxillary 3rd incisors should be endodontically treated if at all possible. Just because one knows how to extract a tooth doesn’t mean it’s the best option for this pet. Endodontic therapy saves the tooth and allows a pet to have a functional tooth for the lifetime of the pet**

Missing teeth/embedded teeth
The only way you can legally document a tooth is not present is by a dental radiograph. Once it is confirmed, it is noted on the dental record as a permanent document. However, many teeth do not erupt and become entrapped under the gumline. A bone-destroying cyst (dentigerous cyst) can occur from any tooth that has not erupted. The reason is enamel does not belong below the gumline. It becomes a foreign body causing an osmotic gradient which leads to bone destruction. Therefore, all missing teeth should be radiographed to either A) confirm they are absent, B) identify tooth root remnants that may need to be removed, or C) identify embedded teeth that need to be removed.

Malocclusions
When a tooth contacts another tooth (attrition) due to a malocclusion, those teeth can die due to the continued trauma occurring. Direct contact (as in maxillary and mandibular incisors traumatizing each other or the maxillary lateral incisor traumatizing a mandibular canine tooth due to a Class III malocclusion) should not occur and in most cases, bad things happen. Dental radiography is needed to assess if the attrition has caused premature maturation (tooth death). Regardless, it is advised to remove those teeth that are traumatizing another tooth due to a malocclusion.

Malformed teeth
Enamel defects (enamel hyocalcification or enamel hypoplasia) not only cause visible crown pathology, but also can cause root developmental pathology. Depending on what caused the enamel defect (localized trauma versus systemic disease), all teeth with enamel defects should be radiographed.

Any tooth that has any visible malformation of the crown should be radiographed. Dens invaginatus is a condition where the dentin is involutes into the pulp cavity and the tooth is endodontically compromised. In many instances, the crown has a lobular appearance and is not smooth.

Gingival mass/soft tissue swelling/bone swelling
In many instances, radiography assists the practitioner in discerning whether a mass is malignant or not. While histopathology is needed for definitive identification of any soft tissue mass, radiography can be a vital aid. If a destructive mass is present but the teeth are not deviated from their normal anatomical position(s), one can assume this is a malignant mass. Odontogenic tumors tend to ‘move’ teeth as they grow.

All areas where an oral mass occurs should be radiographed. The pathology group this author uses request radiographs and clinical images with each sample to provide the most accurate clinical and histopathological diagnosis available.

There is no radiographic difference in many instances between a neoplasia and osteomyelitis. Therefore, histopathology is the only way to confirm this.

Tooth resorptions (TR) and root resorptions
The AVDC (avdc.org) has a very good description of the different types of tooth resorption and the radiographic difference between them. It is beyond the scope of this lecture to discuss them in detail but a very important detail must be discussed:

- Type I TR has a visible periodontal ligament space around the tooth root. Therefore, the tooth MUST be removed.
- Type II TR does not have a visible periodontal ligament space around the tooth root and the root is being remodeled into the alveolar bone. Extraction (if practical and the practitioner follows the principles of exodontia with resorbing roots) is recommended, but crown amputation is allowed. HOWEVER, this must be recorded on the dental/medical record and should be radiographed at least every 6-12 months.
- Type III TR is a tooth that has 1 root as Type I and another as Type II. Treatment as above.
Root resorptions pose an interesting conundrum as to what therapy should be performed. A recent article was written describing each type of resorptions (external replacement resorptions, inflammatory root resorptions, etc…) While this is a human classification; it has relevance with dogs and cats. The practitioner should have a basic understanding among them all. The more common one seen is external replacement resorption. “This finding is the gradual disappearance of the periodontal ligament (PDL) with progressive replacement of root tissue by surrounding alveolar bone.” The long term prognosis for this tooth is not good. In many instances, the crown fractures and the owner or the practitioner notices a tooth missing.

Internal root resorptions involve the pulp cavity. Treatment is either exodontia (extraction) or root canal therapy. External root resorptions involve the surrounding dentin and the pulp cavity is not involved. The way one differentiates between the two is by adjusting the tube head of your dental radiographic machine. If the lesion remains in the pulp cavity, it is internal root resorption. If the lesion moves with the movement of the tube head, then external root resorptions is present. With external root resorption, if there is no co-existing endodontic or apical pathology, and the lesion is below the alveolar margin, the tooth can remain and be monitored every 6-12 months. Any crown resorption should be treated either via restoration or extraction.
Congratulations on the addition of dental radiography to your dental services! Without it, you are basically going to the moon without a navigation system, as almost 100% of all dental work requires radiography to assess the visible tooth and the underlying root structures. Utilization of conventional radiography falls considerably short in diagnostics and is truly wasting client’s money.

**Types of radiology generators and sensors**

For those still considering dental radiography for their practice, there are two radiographic generator options. Wall mounted/floor units provide the DVM the ability to retake less than perfect radiographs by adjusting the tube head direction or distance. That tube head isn’t moving unless you move it yourself. This is unlike the hand held dental radiography generators that are quite useful in tight quarters, providing your state allows these systems to be used. A disadvantage to this system is the tube head placement is hard to reproduce or adjust, but many overcome this issue quite easily. Most systems have set kVp and millilamperage (mA) values, while others allow the operator to select mA between 7-15 mA and kVP between 60-90 kVp. Remember from radiography that the higher the kVp and lower mA makes fewer X-rays with higher penetration. Low kVp and high mA produces images using more x-rays but with less penetrating ability. Exposure time may be in fractions of seconds as with digital systems or pulses. A pulse is 1/60th of a second.

Conventional film used to be the gold standard of dental radiography. However, digital radiography affords the DVM images that are easily manipulated, catalogued, and visualized, and in many instances, superior to film. There is direct digital (computer generated) or indirect digital (utilization of phosphor plate to transfer image to computer). Size 2 direct digital sensors are available. Indirect digital utilizes size 1, 2, 3, 4 and even 6 sensor plates.


**Tooth root maturation**

A recent study evaluated apical closure of mandibular 1st molar teeth (10 months) and canine (7 months) in the cats. Canine teeth apical closure is approximately the same. This is important to know with regard to tooth fractures of young dogs and cats concerning which endodontic therapy to choose. It is also important with regard to orthodontic movement. Orthodontic movement is easier with an immature tooth than with a mature tooth (sound familiar?).

Pulp continues to mature throughout the life of a pet. Therefore, narrow dentin walls/wider pulp cavities are younger pets. As the tooth ages, the dentin wall gets thicker and the pulp canal gets narrower. One way to assess the vitality of a tooth is to radiograph the contralateral tooth to assess pulp cavity width. Premature maturation (tooth death) results in a static pulp canal width.

Q: What is the earliest age you can radiographically identify adult precursors? 8-12 weeks

**Radiographic positioning**

Maxillary radiographs are positioned with the crowns facing DOWN. Mandibular views have crowns facing UPWARDS. View images as you are looking at the patient face-on. Right maxillary views have molars to the left and the canine to the right as you are viewing. For left maxillary, molars on the right and canines to the left. Mandibular views are the same (right arch has molars to left and canine to the right and canines to the left). Digital software packages have templates to allow images to be placed in their normal position. If you take an image and the template is set for a different quadrant, the image may be inverted or backwards, this telling you your image is in the wrong quadrant. (pretty cool!)

Maxillary 4th Premolar Teeth...how do I know which tooth root is the mesiobuccal and which is the mesiopalatal? There is a phrase, “Same Lingual, Opposite Buccal” or the SLOB rule that may be a bit confusing to some. What is a bit more understandable is this: The PALATAL ROOT of the 4th PREMOLAR is closest to the tube head (either from the mesial direction to the distal direction; or the distal direction to the mesial direction). Therefore, if you are taking an image of the caudal maxilla and the tube is facing slightly distally, the most forward (mesial) root is the mesiopalatal root. If your tubehead is slightly facing mesial (towards to nose), then when looking at the two mesial roots, the mesiopalatal root is the one closest to the tube head direction.

For maxillary and mandibular canine teeth, a rostral oblique radiograph provides the best view for potential vertical bone loss. An occlusal view or lateral view may not show bone loss due to the superimposition of the tooth over the alveolar bone.
Nomenclature to understand and use
This section will review common radiographic terms that are essential in understanding whether a patient has normal or abnormal radiographic pathology. Here are some terms one may not be as familiar with:

- Mesial, Distal, Apical, Coronal
- Dentin, Pulp chamber
- Cementoenamel junction
- Periodontal ligament space
- Lamina dura
- Interradicular space
- Alveolar margin (marginal bone)
- Furcation
- Mandibular canal
- Palatine fissures
- Mandibular symphysis

Anomalies
- Chevron effect
- 3-rooted maxillary premolars (2nd and 3rd) and 2nd molars
- Gemination tooth
- Fusion tooth
- Curved root tips
- Microdontia
- Fusion
- Supernumerary teeth
- Twinning
- Missing teeth

A study was performed evaluating the variations in the dentition of the domestic cat. The following was noted from this study:

- Maxillary 2nd premolar absent 7.9%; single rooted 27%; partly fused 55%; 2 fully formed roots 9%
- Maxillary 1st molar absent 2%; single rooted 35%; partly fused 34%, and 2 rooted 28%
- Maxillary 3rd premolar had supernumary roots in 10% of cases

Radiographic technical errors
Foreshortening and elongation are two common errors that can give the interpreter difficulty in adequately interpreting images. This is especially true with addressing endodontic disease, as foreshortened images make visualization of the apices more difficult.

Over and underexposing images are also quite common for the novice dental radiographer. Many times, increased contrast can be more appealing to the eyes, but at a cost. Septal bone may not be visualized with high contract, therefore, a lower contract which may be a bit less clear is preferred in many instances.

Overlapping roots is very common in the caudal maxilla, especially the distal root of the maxillary 4th premolar tooth overlapping the maxillary 1st molar. This non-diagnostic view needs to be modified by adjusting the tube head positioning (previously discussed). However, the two mesial roots of the 4th premolar tooth (mesiobuccal and mesiopalatal roots) may be visualized well, so keeping this image in the template may be worth while. But a disto-mesial tube head angulation must be used to adequately visualize the distal root of the 4th premolar tooth.

In brachycephalics, this can be very difficult, if not impossible due to rotated and crowded teeth. Probing should compliment this closely as sometime radiographic pathology may not be as prominent due to this process.
Oral Tumors 101: What You Absolutely Need to Know
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Oral tumors compromise 5.3% of all neoplasia in the dogs and 6.7% in cats. Therefore, it is necessary for the clinician to be diligent in oral examinations and diagnostics. This lecture will review the most common oral tumors in dogs and cats, and treatment plans for each.

The most common oral malignancies in dogs in order of occurrence: Malignant melanoma, squamous cell carcinoma, fibrosarcoma. In the cat, squamous cell carcinoma

Malignant versus benign tumors
Malignant tumors tend to destroy bone and soft tissue, while leaving teeth in their normal arcade positions. This gives the impression of teeth being suspended in soft tissue with marginal bone. Benign tumors can move teeth due to the soft tissue expansion, thereby redirecting teeth.

Malignant melanoma (MM)
This is the most common oral tumor in the dog. Sex predilection: Males with a male-to-female ratio of 1.4-6.0:1. Typically occurs in older dogs (mean 11 years). Cocker spaniels, Labrador retrievers, Golden retrievers and German Shepherds and dogs with heavily pigmented oral mucosa may be predisposed. Non-pigmented (amelanotic) tumors do occur as well (33%) Melanomas are rare in cats. Otherwise, dark pigmented raised masses are noted.

These tumors have focal infiltration, with early metastasis to regional lymph nodes. Metastasis to the lungs and liver are less frequent. Bone destruction is common.

Location – Any part of the oral cavity including the dorsal tongue surface and lips. Encompassing mandibular and maxillary together, 32% were located rostrally and 20% were located caudally,

Diagnostic tests- Intraoral radiographs are needed to assess bone involvement (57%). FNA of mandibular lymph node, incisional biopsy. Once MM has been diagnosed, conventional oncology workup is recommended (CT, CBC, Serum chemistries, UA, 3 view thoracic radiographs and abdominal US).

Treatment options – Curative intent surgery with wide margins (1-2 cm margins), even as a sole treatment often extends PFI (Progression free interval) and ST (Survival times). Metastasis at time of diagnosis carries a poor prognosis and a lower ST. Rostral mandibular and maxillary masses provide the surgeon a more favorable clean tumor-free margin. Also, tumor size directly affects the ability for a surgeon to achieve clean surgical margins. Other therapy: Maximum tolerable dosage (MTD) chemotherapy (Carboplantin), xenogenic canine melanoma vaccine, radiation therapy, metronomic chemotherapy (combination of doxycycline, NSAID, cyclophosphamide), and interferon.

Survival times – The survival time is short, ranging from <4 months to 5.8 months and 8 months in other studies. However, a longer survival time was noted with dogs with histologically well-differentiated melanocytic neoplasms (Mean survival time of 23 months and median survival time of 34 months after surgery)

Squamous cell carcinoma (SCC) – non-papillary / non-tonsillar
This is the most common oral malignancy in the cat and 2nd most common one in the dog (17-25%). This occurs in older dogs (mean 8-10 yrs) with larger dogs overrepresented. The gingiva is the most common site for this neoplasia. The gingiva usually appears ulcerated with secondary bone involvement (77%). Metastasis to regional lymph nodes is rare (<10%) and low to moderate metastasis to the lungs in dogs is noted (3-36%). Some facial changes (exophthalmos) can be noted. These masses are slow growing, locally destructive mostly on the buccal mucosa. (See comparison of this mass with papillary SCC)

Location- In the dog, the gingival mucosa is the most common site. In the cat, premolar / molar area of maxilla, premolar region of the mandible, and sublingual lesion. Metastasis is late to regional lymph nodes and distant organs. SCC is locally aggressive with bone involvement. Tonsillar and lingual SCC are less common but have a higher and earlier metastatic rate.

Diagnostic tests- Incisional biopsy and regional lymph node aspirates are recommended. Once the non-papillary SCC has been diagnosed, conventional oncology workup is recommended.

Treatment options- Wide surgical excision (1-2 cm margins). Rostral mandibular SCC is more favorable with cats but case selection prior to aggressive excisional surgery must be considered. Rostral mandibular provide a more favorable long-term prognosis. SCC is responsive to radiation therapy with a medium survival time (MST) of 16 months. Radiation is radiosensitive but not radiocurative. Cisplatin and piroxicam have been reported to be effective.
Papillary SCC (PSCC)
Previously thought to only occur with young dogs, this form of SCC can occur with middle to older aged dogs as well. The mean age is ~4 years (0.5-9.0 years) in a 9 dog study. CT of these lesions showed bone lysis with our without osteoproliferation. These masses are more infiltrated, rapid growth, and atypical cellularity. PSS do not metastasize.5
Location - Most were large breed dogs and the most common location was the rostral maxilla (7/9), however, tumors were noted in the rostral mandible and mid/caudal maxilla.2,3,5
Diagnostic tests – Same as SCC
Treatment options – Surgical wide margins (1 cm) provide excellent clinical results.

Fibrosarcoma (FSA)
This neoplasia is the 3rd most common neoplasia in dogs (7.5-25%) and 2nd most common malignancy in cats (13%). The median age of 7.3-8.6 years in dogs, and <25% of dogs are <5 years of age. In cats, the average age is 10.3 years. There is a sex predilection of male to female of 1.4-2.8:1. Larger breed animals >50# (Golden Retrievers) have a higher predisposition for FSA. Metastatic potential is low and can occur late in the disease process with lymph nodes (19-22%) and lungs (6-27%) in dogs. The low metastatic potential is the same in cats.
Clinical appearance show a firm, flat, multilobulated and deeply attached to the underlying tissue with rare ulceration noted. Bone lysis occurs in 72% of canine cases.
These tumors are histologically low-grade and biologically high-grade which potentially provides confusion to the DVM when interpreting an aggressive oral tumor. These tumors may be misdiagnosed as benign fibromas or low-grade sarcomas. High grade anaplastic oral FSAs have a more metastatic potential than do low-grade tumors.
Location – The site predilection in dog is maxillary arcade between the canines and carnassial teeth (56-87%), hard palate (7-17%) and buccal or labial mucosa (4-22%). There is no site predilection in the cat.
Diagnostic tests – After initial incisional biopsy, routine staging with FNA of mandibular lymph nodes, 3 view orthogonal thoracic images, serum chemistries/CBC/UA and CT.
Treatment options – Wide surgical excision (2 cm) is warranted. Local recurrence occurs more frequently than metastasis. Radiation therapy post wide excisional surgery, radiation therapy alone, and radiation therapy with local hyperthermia can prolong the survival times.
Survival times- Median survival time (MST) is approximately 11-12 months for both mandibular and maxillary FSA resection with local recurrence rate of 46%. Radiation therapy MST is 6-26 months.2,3

Osteosarcoma (OSA)
Oral osteosarcomas are the 4th most common oral tumor in dogs (6-18%). Feline oral OSAs are much less frequent (2.4%). Medium to large breed dogs that are middle aged to older are mostly represented. Females appear to be more represented.
Location – Most OSAs occur in the maxilla (43%) followed by mandibular (32%) and the calvarium (23%).
Diagnostic tests – After incisional biopsy, regional lymph node aspirates, along with conventional oncological workup.
The metastatic rate of oral OSA is lower than the appendicular counterpart. Occurrence in the mandible and maxilla are noted, with a more unfavorable outcome with OSA in the TM joint and caudal maxilla / mandible.
Treatment options – Wide radical excision (1-2 cm) of the tumor should be performed if possible. Dogs treated with surgical excision had a Median Survival Time (MST) of 329 days. Surgery resulting in complete excision improved prognosis, whereas calvarial tumor location and increased monocyte count were associated with a poorer prognosis. Radiation therapy and chemotherapy have not shown a decrease in hazard of death progression.3,6

Odontogenic tumors
These tumors are derived from ectodermal, ectomesenchymal, or mesenchymal components of the tooth forming apparatus. These include Canine acanthomatous ameloblastoma (CAA), peripheral odontogenic fibroma (POF), and focal fibrous hyperplasia (FFH). Of the three, POF and FFH are relegated to the gingiva only.7

Canine acanthomatous ameloblastoma (CAA)
In a recent study of odontogenic tumors, CAA occurred 45% (68/152). This is an aggressive benign odontogenic tumor that is non-inductive in nature; therefore, the cells of ameloblastic origin do not induce the surrounding mesenchmal cells. Therefore, no dental hard tissues formed and is a soft tissue neoplasia. These raised, lobulated masses also cause local bone infiltration and tooth displacement. Metastasis to regional lymph nodes or distant organs has not been reported. CT is recommended prior to oral surgery to establish bone involvement. 1-2 cm margins are recommended. Intralesional bleomycin has been documented to resolve this oral mass with no recurrence. Local side effects to bleomycin injections have been documented. Predilection to the rostral mandible is common.7,8,9
Peripheral odontogenic fibroma (POF)
These are slow growing masses. These benign masses are not locally invasive, and occur in 31% of odontogenic tumors. Clinically, they appear as rough-surfaced masses on the gingiva. Radiographically and histologically, there may be dystrophic calcification within the mass, but no alveolar bone involvement. As with other odontogenic tumors, tooth movement due to expansion of the mass is possible. Regional distribution is mostly the rostral maxilla (47%) and caudal mandible (21%), but these masses may occur anywhere along the gingival margin. There is controversy whether these tumors are actually remnants of the periodontal ligament, and whether removal of the tooth and adjacent periodontal ligament is warranted. Some recommend removal in the reactive zone and the surrounding pseudocapsule. Others recommend a more aggressive approach to remove the tooth and the PDL, which means removal of alveolar bone that supports the tooth, to achieve complete removal.3,7

Focal fibrous hyperplasia (FFH)
This encompasses 16% of odontogenic tumors in the dog. Clinically, these appear raised, smooth and sometimes very firm. Regional distribution of these masses are mostly relegated to the rostral maxilla (57%) as well as rostral (22%) and distal (17%) mandible. Surgical removal is similar with POF.3,7

References
9 Kelly JM, Belding BA, Schaefer AK. Acanthomatous ameloblastoma treated with intralesional bleomycin. Veterinary and Comparative Oncology. Vol 8 (2): 81-86
Clinical presentation
This is a syndrome associated with pain on eating and/or opening the mouth. There is noticeable pawing of the mouth, dysphagia, weight loss, bad general condition, grooming deficiency, ptalysm, and sometimes bleeding from the mouth. There is generally inflammation associated around the teeth, as well as in some cases caudal mucositis (caudal to the dental arch and lateral to the glossopalatine folds). This appears to be a multifactorial process of which the body cannot adequately respond to local inflammation of bacteria or viral pathogens. Calicivirus has been implicated with this disease process. While Bartonella has been discussed as a possible etiology to GS, it has been shown NOT to be involved with this disease process.

Clinically, the presentation of gingivostomatitis can be localized or generalized. Periodontitis and tooth resorptions (TR) may present with areas of inflammation so dental radiography is needed to differentiate among the three. Inflammation in the palatoglossal folds is a hallmark sign of generalized feline chronic gingivostomatitis.2

According to Lommer, “While it is believed that feline stomatitis results from an inappropriate immune response to oral antigenic stimulation, the initiating cause is usually not identified, may differ from case to case, and is likely multifactorial”.3 Although underlying immunological abnormalities have not been identified, an increase in mRNA for IL-2, IL-4, IL-6, IL-10, IL-12 and IFN-γ have been identified with chronic stomatitis. Most noticeable in caudal stomatitis are IgG plasma cells and cytotoxic T-cells which could support the possibility of viral etiology in the development of this disease process. Plaque bacteria can stimulate the immune system that appear to contribute to ongoing inflammation. Successful treatment of chronic stomatitis requires minimizing this plaque.3

Initial therapy
Sedation exam with periodontal cleaning, probing, and radiographs is essential. This may have to be done every 3-6 months. Initiation of home care is paramount if the client is going to try a more conservative therapy. Assessment of horizontal bone loss and aggressive gingivitis is needed. Home care should be initiated and clients should be told that this has to be done every day. Therapy is to include the following: daily tooth brushings, chlorhexidine oral rinses bid, VOHC-approved water additives, and a plaque-retarding polymer. However, home care is usually insufficient due to the pain of the inflammation, the unwillingness of the cat to accept therapy, or lack of owner to perform daily.

With most cases, full-mouth or caudal mouth (caudal to the mandibular and maxillary canines) is warranted. It is imperative that dental radiography be utilized with this treatment, as tooth remnants left will not allow proper healing of the gingival and caudal oropharyngeal lesions from healing. Therefore, crown amputation of teeth is not a viable treatment option. The effectiveness of dental extractions has been shown to be: 55% cure, 35% markedly improved, 10% no improvement. Therefore, approx. 90% of cats responds favorably to extractions.3 If the canines do not appear to be involved and the lesions are caudal to them, it is recommended to first consider caudal mouth extractions and spare the canines. However, if the canines and/or incisors have any inflammation present it is recommended removing them and the incisors (full mouth extraction).

Extraction technique is very important as proper flap technique, complete extraction of teeth, alveoplasty of marginal bone with an assortment of diamond burs (round and football), and tension-free closure utilizing 5-0 chromic gut, Poliglecaprone 25, or Polyglactin 910 with a P3 needle. Utilization of assorted winged elevators and small luxators help facilitate complete tooth extraction. Post extraction radiographs are essential to properly confirm complete removal.

Refractory cases (treatment beyond extraction therapy)
No one treatment has shown superiority to another with regard to refractory caudal mucositis. In a recent paper by Hennet at the 2011 Veterinary Dental Forum, he described a study (Harley et al 1999) in that there was a comparison of the effect of methylprednisone, spiramycin-metronidazole, sodium aurothiomalate and chlorhexidine over a 3 month treatment showed none of the agents were able to resolve the underlying pathology present in local gingivostomatitis cases at either a clinical or molecular level.

Antibiotics
These should be used sparingly in gingivostomatitis cases as a primary treatment regimen. However, during refractory cases (after extractions) there may be a need for a course (3 weeks) of antibiotics. The most commonly used drugs are clindamycin, amoxicillin-clavulanic acid, doxycycline, and spiramycin-metronidazole. This helps to decrease the oral bacterial load over a significant period of time and there is improvement clinically. While many have chosen Azythromycin in Bartonella-positive cats with GS, a recent study (Dowers et al. 2010) failed to show a correlation between GS and Bartonella.
NSAID’S and opioids
NSAID’s and opioids can certainly be used for pain therapy. With the addition of Robenacoxib, this approved NSAID therapy does have a place in pain management. NSAID’s are better used for inflammatory control of Calicivirus positive cats. Buprenorphine therapy (0.02 mg/kg) sublingually q 8-12 hrs provides analgesia as well.

Glucocorticoids/immune modulating drugs
These can be used but try to avoid high doses in Calicivirus-positive or herpes-positive cases. Taper a 3 week regimen. If cyclosporine therapy is to be initiated, avoidance of steroids is advised.

Immune modulating drugs besides glucocorticoids, aurothiomalate (gold salts), cyclosporine, Omega interferon, and chlorambucil have been used. Feline Recombinant Interferon (Verbagen Omega) has shown promise in some refractory cases.

Cyclosporine inhibits T-cell activation by blocking the transcription certain pro-inflammatory cytokines which include IL-2 and IL-4. Need to evaluate every 2 weeks to identify cyclosporine blood levels. After 6 weeks of therapy, 52.7% improvement SDAI (Stomatitis Disease Activity Index). Establishment of tough levels of Cyclosporine was noted. Whole blood cyclosporine levels >300 ng/ml (72% improvement) while cyclosporine levels <300 ng/ml showed only a 28% improvement. (cat had to have undergone either premolar/molar or full mouth extractions) It is important for tough levels to be done on an empty stomach. It is best to avoid previous corticosteroid usage. Potential side effects include toxoplasmosis for outside cats.

Dosage is 2.5 mg/kg of Cyclosporine (Neoral) compounded with 1 ml cod liver oil with tuna base (60 mls). Standard is 1.0 mls po bid x 6 weeks.

Stem cell therapy
Current study by UC Davis Dentistry and Oral Surgery Service and the Regenerative Medicine Laboratory: As stem cells are known to have anti-inflammatory and regenerative properties, this may have value going forward. The adipose tissue was harvested from an affected cat with GS and then injected back into the cat (IV) after culture expansion and characterization.

There is a potential side effect of blood clots and transfusion-like reaction so 48-72 hrs of hospitalization/monitoring needed post injection. Two sets of treatment, four weeks apart were performed on cats with non-responsive GS and the cat was rechecked monthly afterwards with ~50% success rate. The clinical trial is still ongoing and the group is investigating the use of an autologous, allogenic and intralesional administration of stem cells.

CO2 laser therapy
This has been written in the J Vet Dent (Lewis et al., 2007). This case required multiple laser therapy and also rescue corticosteroid and IV fluid therapy.

Therapeutic lasers have received attention with regard to healing post surgical tissue, but no information is presently available regarding refractory GS and therapeutic laser tx.

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Why “Let’s Keep an Eye on It” Won’t Work Anymore: Addressing Fractured and Discolored Teeth

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Fractured teeth

In a recent study (Golden, Harvey: *A Survey of Oral and Dental Diseases in Dogs Anesthetized at a Veterinary Hospital*. JAAHA Nov/Dec 1982; Vol 18: 891-899.) in JAAHA showed that 27% of dogs treated had some form of tooth fracture. That study has been a tested and true evaluation of the % that clinicians see in their general practice. Therefore, if you are not seeing that % of cases, you need to put your ‘diagnostic antenna’ up and start looking…for they are there.

The AVDC has an easy to follow nomenclature regarding tooth fractures in the dog and cat. [http://www.avdc.org/nomenclature.html#toothfracture] This lecture will address uncomplicated crown fractures and the different clinical manifestations presented to you the practitioner.

**Enamel fractures (EF)** are simply just that…an abrasion or fracture of the outer covering of the tooth. Enamel is the hardest substance in the body. It is also smooth and impervious to bacterial ingress. Enamel wall length is thin in a dog. In an enamel study, canine enamel was noticeable thinner than that of humans, varying from 0.1 mm to 1.0 mm. “A number of teeth showed a difference in enamel thickness between the buccal and lingual/palatal surfaces. This was noted on the carnassial and incisor teeth, with the enamel of some samples being 1.5 X as thick on one side as the other.” “A thick layer of enamel is disadvantageous in teeth that flex even a minute amount during use as they tend to chip and flake, leaving a weakened, rough surface. Therefore, natural selection is likely to favor those carnivores with thin enamel on strategic teeth.”

Prior to intubation (either pre-anesthetically or at induction), assess the occlusion of the pet. If there is attrition (tooth on tooth contact) from an opposing tooth, then selective interceptive orthodontics may be warranted to prevent further pathology or failure of any treatment plan.

**Treatment of enamel fractures** should be performed using an Aluminum Oxide Bur or fine diamond (conical or tapered) on a high-speed delivery system. Care is taken to bevel the edges slightly and remove the loose enamel present. In some instances, a composite restoration can be performed on this.

1. **Uncomplicated crown fracture (UCF)** is a bit more complex and there are multiple outcomes regarding tooth vitality with this category. A UCF involves the tooth fracture involving the enamel and the underlying dentin. Dentin, while hard, is not nearly as impervious as enamel. It is softer and is traversed by tubules leading directly to the dental pulp, and has a rougher surface. Exposed dentin wears faster than enamel. Dentin microscopically looks like swiss cheese, and the diameter of it is greater than oral bacteria. (Dentin diameter 0.6-0.8 µm @ DEJ to 3.0 µm @ pulp. Bacteria 0.50-1.0 µm in diameter) Therefore, bacteria could ingress through the dentin tubules, penetrate the pulp cavity, and cause irreversible pulpitis. Naturally occurring processes such as blood pressure and pressure gradients help prevent ingress of bacteria to the pulp cavity. Dentin is sensitive and contains neurofibers, which are directly involved in reacting to hydrostatic pressure gradients. Exposed dentin can respond to the fracture by producing reparative or tertiary dentin. This think thickening of dentin helps to protect the pulp by increasing the thickness of dentin between the fracture site and the pulp cavity. However, even with tertiary dentin, a tooth can become necrotic or infected.

Use a sharp dental explorer to pass over the fracture site to verify that there is indeed no pulp exposure. If the explorer drops into a hole in the center of the tooth, or if bleeding occurs upon the exploration, then it is a Complicated Crown Fracture requiring either endodontic therapy or extraction.

**Treatment of UCF**: If you don’t have a dental radiograph unit, you should not be performing treatments for this type of fracture. If you do, then radiograph this tooth and take multiple angulations if need be to make sure no apical or endodontic pathology is present.

1. **Unfilled resin only**

   Clean and polish the tooth. Use flour pumice only for polishing. The oils and fluoride in conventional polish can prevent adequate bone strength or polymerization of the resin. Odontoplasty with an Aluminum oxide bur of fine diamond (cylindrical). Acid etch for 15 seconds, then rinse with water using air/water syringe for 15 seconds. Apply a thin layer of unfilled resin on the tooth and light cure according to manufacturer’s recommendations.

   Recheck radiographically in 6-12 months. Discuss with owner lifestyle changes.

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2. Composite restoration
Clean and polish the tooth (flour pumice only). Prepare the tooth edges and floor to receive the composite restoration. Acid etch bonding agent (unfilled resin) composite restoration (in 2 mm increments) Light cure. Then use assorted coarse to fine finishing discs or Aluminum oxide bur. Use explorer to insure no roughened edges, raised composite, and no gaps between prep site and the composite restoration. Re-acid etch and then apply a final application of an unfilled resin and light cure. Recheck radiographically in 6-12 months. Discuss lifestyle changes.

3. Crown preparation and placement
With more crown damage or the propensity to have a composite restoration being removed by aggressive chewing, a full coverage or ¼ crown can be fabricated and placed. This entails extensive prosthodontics training and the tooth preparation must be very careful as this is still a vital tooth.

4. Indirect pulp capping
If you notice a very thin layer of dentin covering the pulp cavity, a very specific procedure can be performed placing calcium hydroxide or MTA (mineral trioxide aggregate) on the tooth surface followed by the placement of a composite restoration. This procedure entails advanced endodontic training.

5. Endodontic therapy (root canal) or extraction
If the tooth is non-vital (diagnosed radiographically) or has visible pulp exposure, root canal therapy or extraction is warranted. If the pulp is entered upon attempting to prepare the tooth for composite restoration, a vital pulpotomy can be performed if the tooth is endodontically sound.

Discolored tooth
A study by Fraser Hale in the Journal of Veterinary Dentistry (Hale FA. Localized intrinsic staining of teeth due to pulpitis and pulp necrosis in dogs. J Vet Dent 18[1]:14-20 2001 Mar) showed that over 92% of intrinsically stained teeth were non-vital (partial or total pulp necrosis). Amazingly, only 42.9% of those cases had radiographic evidence of endodontic disease.

Teeth can become non-vital via a variety of ways. The first is obviously via direct pulp exposure as in a complicated crown fracture which if not treated within 48 hours is considered non-vital. Other ways teeth can become non-vital is via trauma as in a subluxation, luxation or even avulsion of a tooth. This interruption of the blood supply can cause premature tooth maturation or tooth death. Finally, blood borne infection called anachoresis can affect a tooth at the apex and kill a tooth.

Why does a tooth discolor and what does this mean?
When the blood supply to a tooth is interrupted, either temporarily or permanently, hemoglobin in the pulp cavity is degraded into byproducts (hematoidin, hetoporphyrin, hemosiderin ) and leeches to the dentin wall. This gives a pink/purple hue. If this becomes irreversible pulpitis, the hemocomponents continue to breakdown and the tooth can appear to be blue/grey in appearance.

Reversible vs. irreversible pulpitis
Reversible pulpitis is pulpal inflammation that over time returns to a viable pulp cavity. It can be caused by inflammation or trauma. The tooth discoloration may lessen with time and return to a normal ivory color. Radiography may not be beneficial with reversible pulpitis until pulp canal or apical lesions occur. Pain, however is very common in humans with acute periapical periodontitis and acute periapical abscess so please consider this the same for dogs and cats.

Ideally, it would be great to know the exact time of trauma or inflammation but usually it is not known. However, if the tooth tip or the coronal ¼ of the crown becomes acutely discolored, aggressive therapy should be performed initially to see if the tooth would indeed revert to a normal, healthy pulp.

Irreversible pulpitis can be a sterile event or it can be bacterial related. It can also be acute, subacute or chronic in nature. If bacterial infection combines with the hemoglobin breakdown products, iron sulfide is formed which causes a dark grey or blue color to the tooth. Once that color appearance is noted, it is almost pathognomonic for irreversible pulpitis.

According the Hale study, “Inflammation within the pulp during the period of irreversible pulpitis stimulates nerves within the pulp leading to the perception of pain. Intact teeth undergoing necrosis are often more acutely painful compared with open-chambered, fractured teeth based on increased intra-cranial pressure. Over time, the pain diminishes with the onset of pulp necrosis. However, over time tissue-breakdown products, which include several mediators of inflammation, may extend beyond the apical delta into the LDL space leading to periapical periodontitis which is quite painful (dull pain). (Hale FA. Localized intrinsic staining of teeth due to pulpitis and pulp necrosis in dogs. J Vet Dent 18[1]:14-21 2001 Mar)

Radiographic evidence of a non-vital tooth
Dental radiography plays an important role in identification of irreversible pulpitis, but it is not soley. With regard to the above-mentioned study by Hale, radiographic signs of endodontic disease were not present in 42.4% of these teeth. Therefore, the eyes (seeing intrinsic staining) and radiography do make for a very powerful diagnosis of over 92%

Radiographic signs: Wide pulp cavity with relation to the contralateral tooth; apical rarefaction (apical lucency); Narrowing of the pulp cavity due to pulp calcification (localized or generalized); and root resorption.
Treatment for reversible and irreversible pulpitis

Reversible pulpitis: Therapy includes antibiotics (Ampicillin/Clavulinate or Clindamycin) for 7 days plus NSAID therapy for 7 days (providing renal and hepatic function is normal). If the discoloration does not return to normal after 2-3 months, it can be assumed that this tooth now experiencing irreversible pulpitis which requires immediate therapy.

Irreversible pulpitis: Root canal therapy or extraction is the two treatment options for a non-vital tooth.

Root canal therapy is highly recommended for strategic teeth in the mouth (maxillary and mandibular canines; maxillary 4th premolars and mandibular 1st molars) as well as lateral incisors in large breed pets. Root canal therapy removes the necrotic pulp, and hermetically seals the tooth root canal system with materials that allow the tooth to remain in the mouth for the life of the pet. Success rate of root canal therapy is over 90%. In many instances, a full coverage crown is recommended.

Surgical extraction is the other treatment option for an irreversible pulpitis tooth. While the success rate is 100% (provided dental radiography is performed after the procedure to confirm complete extraction), it does have limitations and consequences. Firstly, extractions can cause more pain than endodontic therapy and involve a longer recovery period. If the pet plays and retrieves or likes to chew, removing a strategic tooth may compromise that aspect of the pet’s life. Removing a mandibular canine tooth has its own unique set of risks involved such as mandibular fracture or trauma to adjacent incisors and premolars. The tongue may loll or fall to one side once the canine is removed and this becomes an undesirable side effect of the removal.
The word ‘dental’ by nature is an adjective, not a verb. There is more to a periodontal sedating exam than just cleaning and polishing. Cleaning and polishing alone is defined as a ‘dental prophy’, but 80% of dogs and cats over the age of 3 have some form of periodontal disease. Therefore, some form of additional therapy must be performed. Ergo, this procedure is not defined as a ‘dental prophy’.

Practitioners must be more proactive with oral care to include sedation examination and cleaning with younger pets. Waiting for oral disease to occur allows bone loss to develop unnecessarily. It is recommended to start sedation dentistry at 1-1½ years of age. Dental radiography should be performed to assess for any missing teeth or identification of a primary tooth (puppy tooth) with no adult counterpart.

Acceptable probing depths are ≤2 mm in small dogs and ≤3 mm in large breed dogs. The clinician will be introduced to the 3 furcation stages and how each one can be treated.

**Stage 1 periodontal disease (PD1)**
This is defined as gingivitis with no bone loss associated with it. Of the 4 stages of PD, this is only reversible stage. Treatment for gingivitis includes subgingival and supragingival scaling and polishing, along with any gingival curettage. In some instances, gingivoplasty is needed to recontour and smooth the inflamed gingiva.

Home care at this level is vitally important to avoid this progressing to a bone loss stage. These home care products will be discussed in the lecture. Of all stages of PD, this is the one that most practitioners avoid treatment. Going forward, this stage must be treated aggressively.

**Stage 2 periodontal disease (PD2)**
Bone loss of <25% occurs with this level of PD. While this may not be severe for a large breed pet such as a Golden Retriever or Rottweiler, it can be extremely severe in a pet under 5 kg. Treatment plan includes closed root planing, perioceutic application, and home care products.

**Stage 3 periodontal disease (PD3)**
Bone loss of >25% and <50%. This stage has a variable treatment plan based on the tooth size and amount of attachment loss. In many instances, open root planing and bone grafting is a possible treatment option. However, in other instances, extractions are necessary.

**Stage 4 periodontal disease (PD4)**
This is the terminal stage of PD (>50% bone loss). Extractions are the only treatment option for this stage.

**Other items**
Antibiotics should only be used in those severely diseased cases that are immunocompromised or have systemic disease. Pulse antibiotic therapy is not advised as a clinically relevant treatment option.
The focus of this two part lecture is to increase your familiarity with common auto-immune skin diseases, the diagnostic tests required to identify them definitively, and common treatment modalities. We will start with a review of diagnostic tests required when dealing with auto-immune skin disease.

The first test is cytology. Cytology is simple to perform but there are many common pitfalls. First, the clinician must consider whether it is more appropriate to use a glass slide or a piece of clear packing tape to collect the sample. Slides can be easily pressed onto moist or gooey lesions. Slides can also be used to lift the edge of a crust and slide underneath. Tape is most helpful in areas two small for the glass slide and lesions that are dry. The next step is staining your sample. Tape should not be flamed and does not require dipping in fixative. Lastly you must evaluate the cytology. If you have a dilapidated microscope then you are going to have more trouble and achieve poor results. The more cytology you perform the more comfortable you will become with the test.

Next on our list is the basic CBC and Chemistry that we all know. These tests may not seem particularly related to the skin disaster standing on your exam table. Especially when the dog is happy and energetic. However, these tests are important for ruling out underlying metabolic problems that could cause the skin disease. More often you will use these tests to guide the medications you choose to treat the animal.

Biopsy is the ultimate test for dermatology. However, performing a biopsy is not always going to get you a straight answer. There are some simple things you can do to get better biopsy results. First, collect multiple samples. In most cases try for 4-6 pieces of tissue. Second, center your biopsy punch on the lesion. Do not send normal tissue to a pathologist. If you send in the margin of a lesion and include normal tissue there is a risk that the lab technician will not “cut in” the diseased tissue for examination. Third, if you see infection on cytology then you should consider resolving infection before collecting biopsy specimens. Infection can obscure the primary disease and make the pathologist’s job much more difficult. Fourth, send a good history along with your samples. Fifth, include clinical photographs with the samples when possible. Lastly, send your tissue samples to a dermatopathologist. Who you send your samples to could make the difference between the right answer and a wrong answer or no answer at all.

Pemphigus foliaceus

Pemphigus foliaceus is one of the most common auto-immune skin diseases seen in dogs and cats. This disease is characterized by pustules and honey colored crusts. This condition is typically idiopathic but it can develop secondary to drug exposure. Pemphigus foliaceus is often seen in patients previously diagnosed with allergic dermatitis; however, no link between the two has been proven.

In pemphigus foliaceus the immune system is attacking a particular protein in the complex structure (called a desmosome) that links keratinocytes together. Destroying the bonds between keratinocytes is termed acantholysis and results in acantholytic cells. Acantholytic cells are typically plump and round because they are no longer connected to their neighbors. They stain darkly and have a clearly visible nucleus. Different forms of pemphigus exist and one of the primary differences between them is what layer of the skin this acantholysis occurs. For pemphigus foliaceus the damage occurs in the two uppermost layers (the stratum corneum and the stratum granulosum). More serious forms of pemphigus affect deeper layers of the skin and cause significantly more damage. As acantholysis occurs, vesicles and sterile pustules are formed. These are fragile and easily damaged because they are located in the uppermost layers of the epidermis. Depending on the intensity of the immune response, pustules can develop and rupture in under an hour or over the course of days. For comparison, pyoderma pustules develop more slowly and are more resilient (more difficult to break). In addition, pyoderma pustules are typically centered around a hair follicle. Both pemphigus pustules and pyoderma pustules will contain neutrophils but intact pemphigus pustules will not contain bacteria.

As already mentioned, the classic lesions of pemphigus foliaceus are pustules and crusts. These lesions can occur anywhere on the body but are commonly found on the face and trunk. Pustules can develop inside the aural opening resulting in serum leakage and crust debris falling into the ear canals. The result is typically a wicked otitis externa. In many cases the nasal planum is also abnormal. The planum typically becomes dry, thick, and crusted. Ulcerations of the nasal planum can occur secondary to crust being traumatically removed. However, pemphigus foliaceus does not cause ulceration of the oral cavity or mucus membranes. The paw pads may be affected as well. Discrete pustules may be seen on the pads but more often the pads are thickened, dry, and crusted. Some dogs will be reluctant to walk but that is uncommon with pemphigus (much more common with hepatocutaneous syndrome).

Diagnosis is via biopsy. Intact pustules are preferred because they offer the clearest picture of the disease process. However, crusts are also very useful biopsy specimens. When collecting biopsies for potential pemphigus foliaceus it is critical not to scrub the skin. In most cases it is advised to avoid shaving the animal’s fur as well. Even the slightest disturbance to the skin can damage the fragile pustules seen with this condition. In the event that no pustules are present, the proof of pemphigus might be in the crust on top
of the skin rather than in the skin sample itself. Consequently, always include crust debris in the formalin jar and request the crust be processed when you biopsy for pemphigus.

Treatment, which is really to say management, is almost always successful but required life-long. Some cases of drug induced pemphigus foliaceus will remain in “remission” even when immune suppressive therapy is discontinued. However, it is often difficult to prove which cases are drug induced which makes predicting which patients will be able to stop therapy nearly impossible. Initial therapy requires steroid administration. Oral daily prednisolone/prednisone dosages of 2mg/kg to 6mg/kg are often required. Steroid therapy often yields dramatic improvement in two to four weeks when dosed adequately. Some patients will respond better to other steroids such as dexamethasone or triamcinolone. Recheck examinations every two to four weeks are critical to assess response to therapy and tailor drug therapy. Secondary bacterial infection is common in pemphigus and your clients will not be able to discern the difference between a pyoderma pustule (which needs antibiotics) and pemphigus pustule (which would cause you to evaluate your immune suppressive plan). In general, the goal is to slowly taper steroid therapy once clinical “remission” has been achieved. Over the course of three to four months some dogs will achieve good clinical response and can be maintained with every other day steroid therapy. However, the majority of patients will experience significant steroid side effects (such as weight gain, polyuria, polydipsia, polyphagia, behavioral abnormalities). Because of steroid side effects and the fact that most patients require life-long immune suppressive therapy it is typically necessary to add another medication as a steroid sparing agent. First line drugs for this purpose are cyclosporine and azathioprine. Second line drugs include mycophenolate and leflunomide. In most cases, I will start a steroid and a steroid sparing drug at the beginning of treatment. All of the above listed steroid sparing drugs have a delay of four to eight weeks until they become clinically effective. By starting both types of drugs at the same time I am able to reduce steroid therapy sooner.

**Discoid lupus erythematosus**

Discoid lupus erythematosus (DLE) is sometimes referred to as cutaneous lupus erythematosus. DLE is a relatively benign auto-immune skin disease which does not affect other organ systems. Histologically there is no difference between DLE and systemic lupus erythematosus (SLE) which is why a good clinical history and blood analysis are important.

**Mechanism**

The first sign of DLE is typically depigmentation of the nasal planum. Often the planum becomes grey, white, or even slightly bluish in color. The nasal planum will then typically lose its cobblestone architecture. Erythema and scaling typically follow. In severe cases erosions, ulcerations, and crusting eventually develop. Lesions may also be seen around the eyes and on the ears. In rare cases lesions can also occur on the distal limbs, foot pads, and perianal region.

Cytology may reveal pyogranulomatous inflammation but secondary infection is uncommon. CBC and Chemistry are typically normal. ANA testing is usually negative. Biopsy samples should be collected from hypopigmented areas or intact crusts. Ulcers and erosions should not be biopsied.

Differential diagnoses include: Pemphigus erythematosus, pemphigus foliaceus, mucocutaneous pyoderma, dermatomyositis, uveodermatologic syndrome, contact dermatitis, and SLE.

DLE typically responds well to low level immune suppressive therapy. Specifically, it is estimated that 50-70% of DLE cases will respond to combination therapy with Tetracycline and Niacinamide. (Doxycycline and minocycline have both been effectively substituted for tetracycline.) However, tetracycline/niacinamide therapy is slow to take effect and the full benefit may not be seen for 8 weeks. Oral steroids can be used to provide initial relief while waiting for tetracycline/niacinamide to take effect. Another option which is often utilized is topical 0.1% Tacrolimus cream. Tacrolimus is applied once to twice daily until full effect and then tapered to the lowest effective dose. Life-long therapy is usually needed. The intensity of therapy may vary over time. DLE is aggravated by sun exposure so some dogs will experience a worsening of symptoms in the summer. Pediatric sunscreen can be used to help avoid sun exposure.

**Mucocutaneous pyoderma**

Mucocutaneous pyoderma (MCP) is not an auto-immune disease. However, I have included it in this lecture because it mimics DLE and is often a source of speculation and confusion. The first sign of MCP is typically edema and erythema of the lip margins. The commissures are especially susceptible. Over time exudate and crusting may develop. Depigmentation may occur as well. Similar lesions may develop on the eyelids, nares, vulva, prepuce, and anus.

The primary differential diagnosis is DLE; however, zinc responsive dermatosis, PF, PE, adverse drug reaction and uveodermatologic syndrome can all appear similar to MCP. Biopsy reveals changes similar to DLE except that the dermal-epidermal junction is not obscured and hydropic degeneration is minimal or absent. Unfortunately, the presence of ulcers and secondary infection can interfere with the pathologist’s ability to evaluate the dermal-epidermal junction. I have seen many patients referred to me after being biopsied with a diagnosis of either DLE or MCP. Choose your biopsy sites wisely and try to eliminate or at least reduce infection first.

MCP is a pseudo auto-immune disease caused by bacterial infection. In some cases topical cleaning and frequent use of a chlorhexidine wipe is sufficient. However, many cases need oral antibiotic therapy for 3-4 weeks. Topical therapy with Mupirocin
Erythema multiforme (EM)

There is some debate regarding the relationship between erythema multiforme, Stevens-Johnson syndrome and Toxic epidermal necrolysis. For the purpose of this lecture we are going to use a rather broad description of erythema multiforme as the previously mentioned debate is not critically important in a clinical setting.

The clinical features of erythema multiforme can be diverse. “Target” lesions are considered the classic representation of this disease but are only reported in 38% of cases. Coalescing erosions and ulcers which form an “ink blot” appearance are common in more advanced forms of the disease. Both target lesions and coalescing ulcers are most often seen on the caudal ventral abdomen. Other, less specific clinical features include acute development of symmetrical macules, papules, plaques, vesicles, and bullae which might result in ulceration. Lesions are occasionally painful but usually not pruritic.

There are numerous differential diagnoses for erythema multiforme due to the variety of potential clinical signs. Mild forms of erythema multiforme may look similar to urticarial allergic reactions, superficial spreading pyoderma, bacterial folliculitis, dermatophytosis, and demodicosis. Other, slightly more severe cases may resemble sterile neutrophilic dermatosis (Sweet’s syndrome). Severe cases of erythema multiforme must be differentiated from burns, lupus erythematosus, vasculitis, hepatocutaneous syndrome and zinc responsive dermatosis.

Individual keratinocyte apoptosis (death) with lymphocyte satellitosis in all levels of the epidermis is the most characteristic histopathologic feature of Erythema Multiforme. However, it is still important to have the tissue sample evaluated by a dermatopathologist so that this key feature is not overlooked. Other non-specific changes include interface dermatitis and mild inflammation which may affect hair follicles. Pigmentary incontinence is variable and also non-specific.

Erythema Multiforme often has an underlying trigger. Potential triggers include: drugs, bacterial infection, viral infection, and neoplasia. Many cases are considered idiopathic but this should not be assumed without thorough investigation. The diagnostic work-up should include thoracic and abdominal imaging as well as CBC, Chemistry, and Urinalysis. The owners should be questioned about any medications or supplements they gave prior to the development of lesions.

An “old dog” form of Erythema Multiforme also exists. Unlike classic EM, “old dog EM” usually produces lesions which are more exudative and proliferative (rather than ulcerative). In addition, the lesions are usually focused on the face and ears instead of the ventrum. An underlying trigger is rarely found in cases of “Old dog EM.”
Treatment involves immune modulatory therapy. Steroids are often the first line of therapy but Atopica and Azathioprine are also used frequently. Higher doses are often necessary initially to gain control over the disease. Life-long therapy, potentially at lower dosages, is usually required.

**Cutaneous adverse drug eruption/reaction**

An adverse drug reaction is most generically described as any un-intended effect of a prescribed medication. Cutaneous adverse drug reactions in dogs and cats are considered uncommon although the true incidence is unknown. Any drug may be implicated in adverse drug reactions but those most commonly involved are: sulfonamides, penicillins, cephalosporins, levamisole, and diethylcarbamazine. Reactions may occur the first time a drug is administered or after a long, previously uneventful course of administration. In other cases, reactions develop during subsequent courses of the same medication. No age or sex predilection is reported; however, drug reactions are more likely to develop closely following vaccination.

Cutaneous adverse drug reactions can demonstrate multiple clinical presentations and mimic almost any other dermatosis. However, the most common clinic presentations are: contact dermatitis, exfoliative dermatitis, pustular dermatitis, pruritus resulting in self-inflicted lesions, maculopapular eruptions and erythema multiforme.

Biopsy is critical for diagnosis but so is a thorough history. Any potential drug triggers should be stopped as quickly as possible. In many cases, stopping the offending drug will result in clinical resolution of skin lesions in 7-14 days. Prognosis is usually good unless internal organ involvement has occurred, another auto-immune disease has been triggered (pemphigus foliaceus, erythema multiforme), extensive skin sloughing has occurred, or a resistant bacterial infection has developed.

Most cases will benefit from immune modulatory therapy. This is usually achieved with steroid therapy because a quick response is needed. Patients with severe ulceration or pruritus will be prone to secondary bacterial infection. This can be a clinical challenge as one tries to avoid adding drugs which could further stimulate the adverse reaction. Topical antimicrobial therapy should be used whenever possible. Systemic fluoroquinolones are typically the least offensive antibiotic option. However, reactions have been documented to all classes of antibiotics. The inciting medication and related compounds should be avoided in the future.

**Cutaneous vasculitis**

At the most basic level, cutaneous vasculitis involves inflammation which targets blood vessel walls. Inflammation narrows the vessel lumen and interferes with blood flow. Peripheral vessels which are very small and locations with poor collateral circulation are most commonly affected. Lesions are also commonly seen in areas of “wear and tear” such as elbows, hocks, paw pads, and the muzzle. It should be noted that vasculitis can occur without an immune component in situations such as burns and trauma.

It is important to realize that vasculitis is actually a reaction pattern and not a specific diagnosis. Multiple diseases can result in vasculitis. Investigating for the underlying cause of the vasculitis is critical to long term treatment success. Drug reactions and infections are considered the most common triggers. However, other possible causes include: infections, dietary hypersensitivity, insect bites, neoplasia, certain drugs, and connective tissue disorders. Even with intensive testing some cases of vasculitis are considered idiopathic.

Biopsy is required to identify vasculitis. However, confirming the diagnosis can be difficult. Definitively diagnosing vasculitis requires finding the precise area of the blood vessel wall which is being targeted by inflammation. Considering the vast network of vessels in the dermis and epidermis this can be like looking for a needle in a haystack. In addition, the pathologist has to differentiate from inflammatory cells that are simply passing through the vasculature and into the tissue verses inflammatory cells targeting the vessel wall. To add even more confusion, there is a category of vasculitis described as “cell poor” which demonstrates far more subtle changes to the vascular walls. As such, pathologists often use clues found elsewhere in the biopsy to make the diagnosis. Those clues include: pale staining collagen, atrophy of hair follicles, and a cell poor interface dermatitis.

Diascopy is a very useful, rapid, in-clinic test in cutaneous vasculitis. Diascopy involves gently but firmly pressing a clear glass slide against the skin in an area of erythema/urticaria. Erythema associated with vasculitis will not blanch with diascopy because the blood is outside the vessels. Erythema associated with allergy, for example, will blanch with diascopy.

Clinical signs of vasculitis vary based on the severity of the inflammation and the length of time that passes between when the disease develops and when the owner brings the animal to you for examination. Acute cases will often demonstrate palpable purpura, pitting edema and the formation of bullae, eschar, and ulcers. Papules, plaques, pustules, and urticaria may also be seen. More commonly, vasculitis is seen as a chronic disease. Pinnal lesions are often alopecic, dry, and crusted. Lesions on the pinnae usually effect the apex or concave surface. Severe vasculitis on the margin of the pinnae can result in a jagged or “cut out” shape. Scabs and frequent bleeding may also be features of pinnal lesions. Paw pad lesions typically involve a “punched out” defect in the center of the pad. In less severe cases the center of the affected pad may demonstrate a shallow circular crater or hypopigmented macule. It is important to note that cases of vaccine induced vasculitis typically demonstrate a lesion at the site of vaccine administration as well as lesions elsewhere on the body. However, the vaccination site lesion may be very minor.

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Treatment typically involves modulating the immune response with steroids, cyclosporine, azathioprine or other similar drugs. Pentoxifylline is an alternative treatment option which often helps milder cases. Pentoxifylline is a phosphodiesterase inhibitor which can increase the flexibility of red blood cells making it easier for them to pass through narrow vessels. Pentoxifylline also has the ability (mildly) to reduce inflammation in the epidermis. Additionally, topical therapy can be helpful for small lesions. Tacrolimus 0.1% (brand name = Protopic) or a steroid ointment such as fluocinonide 0.05% are commonly used. Some dogs will only need treatment for 4-6 months. Other patients will require life-long therapy; however, over time the drug dosages and frequency of administration can usually be reduced.
Canine Atopic Dermatitis:
Integrating New Therapies into Your Strategy
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We will start with a basic overview of atopic dermatitis. Atopic dermatitis is a life-long condition which usually requires life-long therapy. Achieving a true clinical cure for atopic dermatitis is possible but rare. Atopic dermatitis typically manifests as pruritus and erythema. However, some animals may develop recurrent pyoderma or otitis externa instead. While it is difficult to estimate, the current assessment is that 10-15% of the canine population suffers from atopic dermatitis. (Many suspect the number is considerably higher.) In technical terms, atopic dermatitis is a genetic predisposition to hyper-react to allergens in the environment. Unlike what many of us were taught in Veterinary School, allergen exposure occurs mainly through the skin. True inhalant (only) allergic dermatitis is rare. Respiratory symptoms that occur along with allergic dermatitis may represent irritation caused by debris rather than a true allergic process. Allergen exposure is enhanced by defects in the epidermal barrier. One of the many functions of the epidermis is to “keep the outside out and the inside in.” Animals that are genetically predisposed to allergic dermatitis have genetically programmed defects in their epidermal barrier. These defects result in increased allergen exposure. It is important to realize, and communicate, that allergic inflammation causes more than just the itching we can observe in the exam room. Allergies increase transepidermal water loss, dermal and epidermal inflammation, and the risk of secondary infection.

The first step of treating an allergy patient is achieving an appropriate diagnosis. This begins with a good physical exam. Many allergy patients demonstrate erythema on the ventral aspects of the front paws and in the ear canals. Typically the pinnae themselves are not primarily affected. Pinnal lesions should raise concern for Sarcoptes scabiei and extension of infection from otitis externa. Dogs with environmental allergies are also known for not having lesions on their dorsal lumbar area. This is a location more commonly associated with flea allergy dermatitis. Superficial pyoderma, malassezia dermatitis, alopecia, and seborrhea are common with all types of allergy so observing these symptoms should trigger you to look at common allergy affected areas. Of course, the other crucial part of examining a dog is questioning the owner. Not every owner is observant or adept at communicating but it is our job to tease out as many important details as possible. Important allergy related questions to ask are: “At what age did your dog start having skin/ear problems?” “When did this episode begin?” “Have you observed any change with the weather or seasons?” “Do you know anything about your dog’s parents or siblings?” “Are there any other pets in the house?” “If so, are the other pets affected?” The last part of a good allergy exam is client education. Education is critical because allergies are chronic and frustrating. It is best to have educational information available in multiple formats. Paper hand-outs, informational emails, in-office videos, and internet resources are all readily available. Controlling where your clients obtain their information will not only save you frustration later but also convey your commitment and knowledge to your clients. You can find many useful handouts at www.animaldermatology.com

Now that you have collected a good history and performed a thorough physical exam the next step is working down the diagnostic pathway of allergic dermatitis. Unfortunately, there is not one single test that can diagnose allergies. Atopic dermatitis is a diagnosis of exclusion. This often needs to be explained to our clients. Proper diagnosis of atopy requires appropriate history, consistent clinical signs, and proof that the pruritus and skin disease are not caused by infections, parasites, metabolic disease, and endocrine disease. There are times when your diagnostic work-up is actually quite simple. Sometimes your client will bring you a pruritic dog that receives regular flea prevention, has a history of seasonal variation, and does not have active skin lesions. If you are very lucky they might even know that a parent or sibling is also affected in a similar way. In this situation you might go straight to talking about treatment options. However, in many instances, the allergy patient isn’t so obvious. Allergy patients often suffer from alopecia, seborrhea, pyoderma, and otitis. In addition, many clients cannot remember when the symptoms began, or worse yet, you might have a husband and wife who vehemently disagree about the history. In these situations you must start at the beginning and rule out infections, parasites, and metabolic/endocrine disease. Cytology from the ears and skin is the first step. Cytology is almost always indicated. Cytology allows you to quickly identify yeast and bacteria and provides a semi-quantitative method of monitoring progress. A dry microscope slide can be pressed on moist lesions, scraped under crusts, or used to break pustules. Another useful method of collecting cytology is with clear packing tape. Packing tape is most helpful for dry lesions, folds, and nail beds. When using tape you do not need to “heat fix” the sample or use the fixative step of your three step staining protocol. Skin scrape sample collection is often needed but not as frequently as cytology. We all know the basics of skin scraping. But here are a few tips to improve your success. First, shave the area you intend to scrape. Second, apply mineral oil to the sample site and pinch the area firmly. Third, scrape until you obtain capillary bleeding. You should observe red blood cells on the slide when you look at it under the microscope. Lastly, it helps to have mineral oil waiting on your slide so that all the debris you remove from the skin surface stays where you can view it. The last of our skin related tests is DTM culture. DTM cultures can be particularly frustrating for veterinarians and technicians so perhaps the best tip is to send them to an outside lab (such as Purdue ADDL or IDEXX) if you don’t enjoy checking them yourself. When collecting your samples for DTM culture it is best to collect samples from the edge of lesions. Broken hairs are especially...
helpful. It is also useful to use a fresh tooth brush to pick up dander, debris, and hair from the entire surface of the animal. If you choose to perform DTM culture in house please remember: 1) Use plate type media not jars or test tubes. 2) Do not close the culture tightly. 3) Keep the culture in a dark area with approximately 30% humidity and at 86 degrees farenheit. 4) Color change does not confirm diagnosis of a dermatophyte. Many contaminants can cause the medium to change from orange to red so microscopic examination of the fungal growth is essential to confirm dermatophytosis.

The one very obvious diagnostic that we have not discussed yet is allergy testing. At some point in time most pet owners who are dealing with allergy problems will inquire about allergy testing. It is important to have the facts about allergy testing so that you can guide your clients accurately. First and foremost, allergy testing is not a tool to diagnose allergies. In other words, it is not a screening tool. Rather, allergy testing is used to define the allergy more precisely, predict flares, direct environmental modification, and formulate immunotherapy.

Allergy testing is not a screening tool because positive results don’t immediately prove that allergy is the cause of the skin symptoms. Rather, you must have a supportive history and clinical signs along with evidence that you have eliminated other causes of skin disease. In regards to allergy testing specifically, there are two accepted and peer-reviewed methods: serum testing and intradermal testing. Serum testing only requires the collection of a blood sample. This type of test is quick and easy for the general practitioner and does not require any special equipment. Serum allergy testing is generally touted as not being affected by drug therapy such as steroids or antihistamines. However, these drugs can contribute to poor results in some dogs. In addition, serum allergy test results will be affected by season of the year. Within the past ten years numerous companies have begun offering serum allergy testing. I caution you not to choose an allergy testing company solely on cost. While all serum allergy companies use a similar testing model there are unique differences that can be quite important. Lastly, a paper published last year highlighted the difficulty with this testing method by sending samples from the same patient to multiple labs. Agreement between the labs was very poor. Intradermal allergy testing is typically only performed by veterinary dermatologists because of the need to keep expensive antigens in stock for testing and because of the learning curve necessary to read an intradermal allergy test accurately. Intradermal allergy testing also requires sedating the pet and shaving a patch of hair on the side of the thorax. Perhaps the most confusing factor in recommending intradermal allergy testing is knowing the drug withdrawal times required prior to the test. In general, the withdrawal time for oral steroids and antihistamines is two weeks. For injectable steroids like triamcinolone or dexamethasone the withdrawal time is 2-3 weeks. For Depo-Medrol, the withdrawal time is three months. Topical steroids should be stopped 48 hours prior to the test. Fortunately, there is NO withdrawal time for Atopica or Apoquel. There are many unique benefits to intradermal allergy testing. First, this test is not affected by season. Second, it allows a veterinarian to test the organ affected and observe the true intensity of an allergic reaction. Lastly, and perhaps most importantly, every intradermal allergy test has a built in scale. These are positive and negative reactions designed into every skin test. This helps us adjust for inevitable patient to patient variation.

Before you can develop a good allergy treatment plan you must realize that no single therapy is 100% effective. It is also important to understand that no two patients are exactly the same and that it is easier to prevent rather than suppress flares. Multimodal therapy is recommended because it allows intervention of allergic inflammation at multiple points in the disease process. It is easier to think of allergy therapy as core and supportive treatments. Most patients need a core therapy and one or two supportive therapies. However, severe patients need multiple core therapies and supportive therapies.

For ease of discussion we will consider five core allergy therapies: 1) antihistamines, 2) steroids, 3) Atopica, 4) Apoquel, and 5) Immunotherapy. We will focus on Immunotherapy, Atopica, and Apoquel today. Immunotherapy is still considered the “gold standard” of allergy therapy. Immunotherapy allows us to modulate the allergic response without drugs. This occurs via multiple mechanisms including the development of IgG blocking antibodies, a decrease in allergen specific IgE and an increase in the number of regulatory T cells. Consequently, immunotherapy provides many unique benefits that drug therapy cannot. Immunotherapy may also prevent new allergies from developing and is the only therapy that could potentially result in a clinical cure. Because immunotherapy is not a drug there are no major side effects or drug interactions. Anaphylaxis can occur during immunotherapy but this is rare. Immunotherapy is tailored to each individual so animals at higher risk for anaphylaxis can be induced more gradually. Risk for anaphylaxis is based on breed and the intensity of the allergy test reactions. Immunotherapy has classically been administered as subcutaneous injections. However, within the past three years, sublingual immunotherapy drops have become available for pets. Both routes of administration can be effective. Early publications suggested that oral immunotherapy would be more efficacious but my experience has been that the two forms are equally successful. Multiple schedules for administering these products are available based on the laboratory used and the dermatologist involved. When discussing immunotherapy with clients it is important to clearly communicate that immunotherapy is not a fast acting treatment with many dogs not showing significant benefit for 6-12 months. As a general rule, animals should receive immunotherapy for at least a year before deciding whether it is effective and worth continuing. Because of the slow onset, many patients need additional therapy in the beginning. This might include antihistamines, steroids, Atopica or Apoquel. While immunotherapy can provide a clinical cure, it is rare and most dogs require immunotherapy for life. As a general rule immunotherapy is considered approximately 70% successful with 45-50% of those dogs requiring some type of additional supportive therapy long term.
Fortunately, we have two safe and effective drug options for treating allergy symptoms. Atopica (modified cyclosporine) became available commercially for dogs more than ten years ago. Atopica works via suppression of IL-2, T-helper, and T-suppressor cells. By far the most common side effects of Atopica are vomiting and diarrhea. Usually these are mild and do not require specific therapy or cessation of therapy. Another side effect that sometimes occurs is gingival hyperplasia. Obviously gingival hyperplasia is not a life threatening side effect. It is typically seen only in patients receiving high doses of cyclosporine or after many years of therapy. In most cases gingival hyperplasia resolves when Atopica is discontinued. Atopica is a very useful drug but there are a few items to keep in mind. First, because Atopica may take 4-6 weeks to see full effect it is not helpful for immediate control of flares. I typically recommend a 30 day recheck so that I can evaluate the patient’s progress. To help prevent vomiting you can freeze the capsules, give the medication with a small meal, divide the dose throughout the day or start with a low dose and ramp up to your target dose over two weeks. Lastly, you will commonly want to combine Atopica with a steroid during the first two to three weeks of treatment. The steroid provides immediate relief while the Atopica ramps up.²

Apoquel is the newer drug on the market. Apoquel was released in January 2014 and then quickly went on backorder. Apoquel become more widely available this April although production and distribution are still less than ideal. Apoquel is a completely different medication than Atopica and it works via an extremely different mechanism. Apoquel (Oclacitinib) works via blocking IL-31 at the JAK-STAT pathway. IL-31 is the cytokine linked to the feeling of itch. By blocking IL-31 there is also suppression of Epithelial Langerhans cells and T-cells. Because of overlap in the Jak-Stat pathways Apoquel also suppresses IL-2, IL-4, IL-6, and IL-13 which are also involved in allergy. Apoquel’s serious side effects are linked to this overlap as well. The most concerning side effect to watch for is decreased hematopoiesis. One of the benefits of Apoquel is the speed on action. Most dogs will improve in 24-48 hours but I have had a few patients not respond until 5-7 days. Vomiting is far less common with Apoquel (as compared to Atopica) but it can occur and it can be severe.⁵

Another new product has recently achieved conditional release. Canine Atopic Dermatitis Immunotherapeutic is currently available through most dermatologists and some general practitioners. CADI is a once a month injection of a monoclonal antibody designed to target IL-31. Side effects are extremely uncommon. This product can be given to puppies and dogs with other health problems.

With all of the products available for treating atopy in dogs you might think it would be an easy task. However, every patient has different allergies, different primary signs, and different secondary problems. Consequently, you need to have a consistent treatment strategy.

Step one of this strategy is to eliminate current infections. Eliminating infections reduces pruritus and inflammation while also improving the patient’s odor and appearance. The relief that a patient derives from resolving infections may be dramatic.¹ This is also the time to impress upon the client the importance of secondary infections. In many cases our clients may ignore or be oblivious to the signs of infection. When present, infections can negate the improvement obtained by the actual allergy treatment. Another major problem of recurrent secondary infections is antibiotic resistance. For this reason it is imperative to prescribe an appropriate antibiotic for an appropriate length of time. It is also important to consider topical antimicrobial therapy. Topical therapy can provide immediate relief for the pet. More importantly topical therapy also works synergistically with the oral antibiotic / antifungal medication to reduce the risk of resistance. Of course, dealing with infections includes dealing with ear infections. Otoscopy is always a useful part of the allergy exam. Remember that otic cytology is important any time you suspect an ear infection. Cytology helps you determine which ears are inflamed due to allergy and which ears have infection. Cytology also helps you track the progress of your therapy. When managing otitis externa remember to choose both your ear wash and ear medication carefully.

Improving the epidermal barrier is step two. The epidermal barrier is composed of lipids and cornocytes in the stratum corneum. The dominant lipids in the stratum corneum are called ceramides. Free fatty acids and cholesterol are also found in the lipid portion of the stratum corneum. However, ceramides play a crucial role by helping align the other lipids. When intact, the lipid portion prevents water loss as well as allergen and antimicrobial penetration. Consequently there is less allergen exposure, less risk of infection and less pruritus. Ceramides are now available in multiple forms. You will find ceramides in shampoo, sprays, conditioners, and spot-on products.

Conscientiously choosing a core treatment is step three. In order to make a good recommendation to your client you must consider the patient’s underlying medical conditions, the severity of the allergy, the primary symptoms, and the limitations of the dog and owner. You also want to steer your clients to the safest therapy for long term use. This entails considering Immunotherapy, Atopica, Apoquel, and CADI.

Step four is adding supportive therapy as needed. What you add is based on what the patient requires. Supportive therapies include: antibacterial and antipruritic shampoos, wipes, and sprays as well as oral antihistamines, oral essential fatty acids, and topical ceramides.
Clinical Update on Dermatophytosis:
Better Ways to Fight the Fungus Among Us
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Review of clinical signs
Perhaps the most critical task in discussing the clinical signs of dermatophytosis in cats is to highlight the fact that feline dermatophytosis looks very different from human dermatophytosis. In fact, dermatophytosis in animals can cause a wide variety of clinical signs. Often, dermatophytosis in cats appears as one or more irregular patches of alopecia. Affected and surrounding hairs may appear broken or frayed. Alopecia may be localized or diffuse. Erythema, scale, crust, and papules may or may not be present. Pruritus is uncommon but may occur. Dermatophytosis may look very similar to stud tail, chin acne, milliary dermatitis, pemphigus foliaceus, or cutaneous lymphoma. Onychomycosis (infection of the nails) and kerions (deep nodules) can occur secondary to dermatophytosis but are uncommon. Due to the widely variable presentation of dermatophytosis in the feline patient, a DTM culture is indicated in most cases of feline skin disease.

Science of the details
Numerous species of dermatophytes are known to exist. In our companion animal species the majority of disease is caused by: *Microsporum canis*, *Microsporum gypseum*, and *Trichophyton mentagrophytes*. Of these three, *Microsporum canis* is the most common. Transmission occurs by contact with infected hair or scales. Fungal elements in the environment, on fomites or animals can cause infections as well. The source of *M. canis* is usually an infected cat. In comparison, *M. gypseum* is usually contracted from contaminated soil and *T. mentagrophytes* from rodents or rodent dens.

The infective portion of the dermatophyte organism is the arthrospore. Arthrospores can be carried on dust, air currents, fomites, and ectoparasites like fleas. Physical damage to the stratum corneum is important to facilitate invasion of arthrospores. Taking this into consideration one can easily understand the heightened concern for secondary dermatophytosis infection in patients suffering from allergies or flea infestation.

It is helpful to have an understanding of the cycle of an “average” dermatophyte infection when treating patients and advising clients. For this example we will consider *M. canis*. Lesions typically develop seven to ten days after inoculation. For the next six to eight weeks the lesions typically enlarge. Finally, lesions may self-resolve by twelve to fourteen weeks after initial exposure. Upon exposure to viable arthrospores hair shafts are in both endothrix and ectothrix infection. Fungal hyphae are formed and migrate downward to the hair bulb. This process continues until the fungus reaches the keratogenous zone (Adamson fringe). Because the fungus needs keratin it cannot proceed down past the area of the hair shaft where keratin is formed. In an actively growing hair (anagen phase) the fungus and hair might remain in equilibrium. In a resting hair (telogen phase) new keratin is not being formed and the fungus must stop growing. Eventually the fungus is expelled when the hair is shed. This situation is also important when considering Wood’s lamp examination (discussed below). Dermatophytes which are not actively growing will not fluoresce. Thus, only infected anagen hairs will glow.

Diagnosis
As with any dermatologic problem, an accurate history and thorough physical exam are important first steps. However, because dermatophytosis can mimic many other diseases we need to review the diagnostic options.

First, the trichogram can be a quick and helpful diagnostic test. Hairs from the lesion and the surrounding area should be collected. The hairs are placed on a slide along with mineral oil. A cover slip is added and the slide is gently heated for 15-20 seconds. Most dermatophyte infected hair with display ectothrix lesions. When looking through the microscope it is recommended to concentrate on fragmented pieces of hair that are larger in diameter than other hairs present. In addition, it is usually most rewarding to look near the hair bulbs. Infected hairs often appear fuzzy or swollen. One must remember that dermatophytes do NOT form macroconidia on tissue. Thus, any macroconidia retrieved from the hair coat represent contamination. A positive trichogram might guide your initial therapy but does not negate the need for further testing.

Second, the Wood’s lamp is simple, easy, and quick. However, the sensitivity and specificity of this test are both quite low. The wood’s lamp is basically a black light but to describe it scientifically it is a UV light with a wavelength of 253.7 nm that is filtered through a cobalt or nickel filter. Hairs invaded by actively growing *M. canis* will fluoresce bright yellow-green. It has been widely accepted for years that the Wood’s lamp needs to warm up prior to use. However, that is actually not necessary. What is necessary is exposing the hair to the light for three to five minutes. Infected hairs glow because of tryptophan metabolites produced by the fungus. Thus, only anagen hairs will glow because those are the only ones which contain actively growing fungus. One must realize that many other things will fluoresce such as soap residue, dander, carpet fibers and even certain bacteria.
Third, fungal culture is well known and commonly used. Most fungal cultures contain Sabourand dextrose agar or dermatophyte test media or both. Dermatophyte test media is basically Sabourand dextrose agar with cyclohexamide, gentamicin, and chlorotetracycline added to inhibit contamination by bacteria and other fungi. The pH indicator phenol red is also added. However, I often feel the red color change is more of a hindrance than a benefit. Dermatophytes use the protein in the growth media first and produce alkaline metabolites. These alkaline metabolites cause the media to change from yellow to red. Once the proteins are exhausted the dermatophytes use carbohydrates which yield acidic metabolites and turn the agar back to yellow. Many other fungi (contaminants) use carbohydrates first and proteins later. Such fungi result in a color change that occurs 10-14 days after the culture is started. This is one reason why fungal cultures need to be visually examined daily. The color of the agar as well as the color and morphology of the colonies should be noted on a daily log sheet. Color is important to note because dermatophyte colonies are not pigmented. They can be white, off white or buff color. When a suitable colony forms and causes color change at the appropriate time it must be identified. Usually macroconidia are not produced prior to 7-10 days of growth. Sampling the colony for macroconidia involves gently applying the sticky side of strip of clear packing tape onto the surface. The tape is then placed on top of a slide which already contains several drops of lactophenol cotton blue. A cover slip is then applied and the sample can be easily examined for macroconidia. If you find macroconidia but cannot identify them or if you fail to find macroconidia but have a suggestive white colony you must simply wait and repeat the microscopic examine in a few days. Microsporum canis typically produces white fluffy colonies. Over time the center may become depressed. The macroconidia of M. canis are spindle shaped with thick walls and six or more cells/segments. The terminal end has spines which make a knob like structure. Microsporum gypseum colonies are flat and buff to cinnamon in color. Macroconidia are spindle shaped with thin walls and less than six cells/segments. Trichophyton mentagrophytes colonies are white to cream colored with a powdery surface. The macroconidia cigar shaped with thin, smooth walls. Macroconidia may occur in clusters like grapes.

Fourth, PCR testing has recently become commercially available through Idexx labs (spring 2015). The Idexx PCR includes Microsporum spp, Microsporum canis, and Trichophyton spp. According to Idexx the PCR test has a 95% sensitivity and 99% specificity. Results are available in 1-3 days. You can also request the lab perform a DTM culture to further identify the dermatophyte if the PCR is positive. Submitting a sample for PCR testing is similar to the process for collecting samples for in-house culture. A clean, sterile toothbrush can be combed over the entire pet and placed in a clean, new Ziploc plastic bag. Hairs can be plucked and placed into an empty red-top tube. Nail clippings can be submitted in a red-top tube. Specimens should be refrigerated once collected. The clinical usefulness of this test is yet to be discovered but it could be immensely helpful.

Fifth, dermatophytes are sometimes accidently and sometimes intentionally found on tissue biopsy. Biopsy is very helpful in the diagnosis of dermatophytosis which infect the stratum corneum instead of the hair shafts. Kerions are another example of a dermatophyte infection which warrants biopsy. The success of diagnosing dermatophytosis with biopsy is difficult to pinpoint because it varies greatly upon the quality of the sample submitted. However, dermatophytes can be highlighted in tissue specimens using PAS stains. In addition, the presence of fungal organisms in the hair follicle or shaft is typically easily identified.

**Treatment**

Treatment is typically divided into topical and systemic modalities. No discussion on topical dermatophyte therapy would be complete without discussing shaving. Many veterinarians advocate shaving cats who have cultured positive for dermatophytosis. The purpose is obvious. Because the fungus lives within the hair shafts, removal of the hair shafts results in removal of a large amount of infective material. Shaving also allows more effective topical therapy with lotions, sprays, or shampoos. The three main problems with shaving cats with dermatophytosis are: 1) The act of shaving can produce micro-trauma to the skin and thus facilitate new lesions. 2) Who is going to shave the cat and where is it going to happen? Shaving introduces infective spores into the air and contaminates the environment. 3) What cat enjoys being shaved? And are you willing/able to sedate the cat for shaving? As a general rule I don’t recommend shaving cats infected with dermatophytes. Next we must discuss topical antifungal ointments. Multiple products are available over-the-counter and are a favorite of clients who like to self-diagnose and self-treat. When using ointments it is important to apply the product to the lesion and a wide margin around the lesion that appears normal (6 cm). Ointments should be applied every 12 hours. In general, I find ointments only marginally helpful in cats. However, I will recommend an ointment as adjunct therapy if the owner “needs” something to do. I also recommend ointments if there are immune compromised people in the house. Antifungal shampoos and sprays are also available. I find these more helpful than ointments as adjunct therapy because the entire animal can be treated. Even if we are not hastening resolution of the infection we are reducing contagion in the environment. Some clinicians caution against antifungal shampoos and sprays because of the risk of breaking fragile hairs and spreading spores around the animal’s body. The last category of topical therapy is antifungal dips. This category includes Lime sulfur and Enilconazole. Enilconazole dips are not available in the United States. Lime sulfur dips are administered once or twice weekly and are extremely effective. However, Lime sulfur is foul smelling and stains most items. Proper personal protective gear is essential to avoiding human side effects. This is a treatment best performed in the veterinary hospital. One final note on Lime sulfur. The dip...
is not rinsed off and instead must be allowed to dry on the animal. Many cats require an e-collar during this drying period to prevent ingestion of Lime sulfur.

Systemic therapy is typically the core treatment for dermatophytosis. Five antifungal drugs are commonly available but two yield the best results. Drug choices include: 1) Itraconazole, 2) Terbinafine, 3) Fluconazole, 4) Griseofulvin, 5) Ketoconazole. Many years ago Lufenuron was claimed to have antifungal activity; however, critical studies of the drug’s effects indicate it does not. Itraconazole is highly effective and has a low incidence of side effects. The dose is 5-10mg/kg once daily with food. Terbinafine is also highly effective and demonstrates low risk of side effects. The dose is 20-30 mg/kg once daily. Terbinafine is available in 250mg tablets at most pharmacies. Most cats will receive ¼ or ½ tablet once daily making this medication very cost effective. Fluconazole is in the same family as Itraconazole and Ketoconazole. However, it is the least effective of the three against dermatophytosis. It does penetrate the blood brain barrier and is excreted in high concentrations in urine. Thus there are certain specific situations where Fluconazole might be indicated. Generally, however, it is not used for dermatophytosis. Griseofulvin is an older antifungal drug. It is effective but has the highest risk for side effects including GI upset and myelosuppression. Persians, Siamese, and Abyssinians may be more prone to Griseofulvin side effects. Griseofulvin should not be used in breeding animals. Ketoconazole is effective against dermatophytes but generally less so than Itraconazole. Ketoconazole as has a higher incidence of causing vomiting, diarrhea, and hepatotoxicity in cats. Of course, using an appropriate drug is only half of the story when treating dermatophytosis. Treatment protocol and duration are also important. I recommend daily therapy with Itraconazole or Terbinafine until 2 weeks after the second negative DTM culture. DTM cultures are repeated every 2-4 weeks depending on the situation. At the time of diagnosis I explain to clients that their cat will likely receive antifungal medication for at least 3 months. It is also important to explain to clients at the beginning that their cat will appear healed long before it is actually free of the fungus. Stopping therapy too soon is the most common cause of “recurring” dermatophyte infection. In truth many of these represent a case that did not achieve complete resolution the first time.

Decontamination

Physical removal of hair and dander are essential for environmental decontamination. Hair shafts containing infectious arthrospores which are left in the environment can remain a source of infection for months or even years. (18 months for M. canis.) Environmental decontamination comprises three steps: 1) Mechanical removal of infective material. 2) General cleaning with detergent or soap until area appears clean. 3) Application of a disinfectant to kill any remaining spores. Steps one and two are fairly simple. However, care should be taken to disinfect vacuum cleaners as well as other cleaning tools which could spread the spores. Dilute bleach solution has classically been recommended for disinfection. Using dilute bleach is complicated by the fact that commercially available bleach is available in different concentrations and clients are expected to produce an appropriate dilution themselves. Dilute bleach is also considered unstable and needs to be made fresh daily. Bleach can irritate the skin and cause respiratory difficulties if not appropriately handled. Lastly, bleach is known to discolor fabrics and is thus only a good option for hard surfaces. In 2013, Karen Moriello published an article in Vet Derm evaluating the efficacy of commercial disinfectants against Microsporum canis and Trichophyton spores on textile surfaces. Eight products were evaluated. Surfaces received either 1 spray (1ml) or 5 sprays (5ml) and were left to dry for 10 minutes. Results were similar for both organisms and are summarized in the table below.

<table>
<thead>
<tr>
<th>Product</th>
<th>Growth after 1 spray</th>
<th>Growth after 5 sprays</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>Too numerous to count</td>
<td>Too numerous to count</td>
</tr>
<tr>
<td>Dilute bleach</td>
<td>No growth</td>
<td>No growth</td>
</tr>
<tr>
<td>Formula 409</td>
<td>No growth</td>
<td>No growth</td>
</tr>
<tr>
<td>Chlorox Clean-up</td>
<td>No growth</td>
<td>No growth</td>
</tr>
<tr>
<td>Lysol</td>
<td>No growth</td>
<td>No growth</td>
</tr>
<tr>
<td>Accel TB</td>
<td>No growth</td>
<td>No growth</td>
</tr>
<tr>
<td>Chlorox Anywhere</td>
<td>Too numerous to count</td>
<td>No growth</td>
</tr>
<tr>
<td>Simple Green</td>
<td>Too numerous to count</td>
<td>No growth</td>
</tr>
<tr>
<td>Fantastik</td>
<td>Some inhibition against M. canis but not Trichophyton</td>
<td>No growth</td>
</tr>
<tr>
<td>Trifectant</td>
<td>Some inhibition against M. canis but not Trichophyton</td>
<td>No growth</td>
</tr>
</tbody>
</table>

*Adapted from: Moriello, Efficacy of eight commercial disinfectants against Microsporum canis and Trichophyton spp. infective spores on an experimentally contaminated textile surface. Vet Dermatol 2013; 24:621-e152.

Quarantining infected animals is recommended to reduce the amount of cleaning required. It is ideal to wear dedicated clothing when in the quarantine area to prevent accidental spread around the house.
Patients with otitis externa generally present for head shaking, ear scratching, and odor from their ears. Pet owners may also notice purulent material coming from the ear canal(s), changes in their dog’s behavior, or whining and discomfort. Otoscopic exam typically reveals varying degrees of erythema, edema and debris. These changes can make visualizing the tympanic membrane very difficult or even impossible. Unless the patient is aggressive/dangerous you should try your best to see every tympanic membrane that enters your exam room.

Key points
1. Otitis externa is most often a clinical sign of underlying skin disease, not a diagnosis in and of itself.
2. Identifying and resolving / controlling the underlying cause is essential to long term success in otitis externa.
3. Allergy is the most common cause for otitis externa in the canine
4. Topical therapy is the most effective therapy for treating otitis externa
5. Cytology (clean) – Plan (persuade, purge) – Recheck (re-engage)

Identifying the underlying cause of otitis externa can be difficult. The PSPP system can help you work through the potential causes and also make it easier to discuss otitis with your clients. PSPP stands for Primary, Secondary, Predisposing, Perpetuating. The Primary category includes things that can cause disease in a normal ear. The list includes: allergy, auto-immune disease, foreign objects, mass/polyps, endocrine dysfunction, immune mediated disease, and parasites. The most common primary problem I see is allergy, but in general practice you probably see a fair amount of ear mites and foreign bodies too. Whatever the primary factor, it has to be resolved or controlled before you are going to achieve lasting success. The Secondary category includes things that create disease in an abnormal ear. The secondary list includes: bacteria, yeast (malassezia), fungi, medication reactions, and over-cleaning. Because of the way we commonly communicate about otitis externa it is easy for our clients to misunderstand and think that bacteria or yeast are primary causes of otitis. Sometimes it helps to point out that ear canals are not sterile. There is a normal flora in the ear canal just like on the skin. Infections must be resolved but they are not the root cause. Predisposing factors are fairly simple to understand and are typically what most clients blame for ear disease. Predisposing factors are present prior to otitis but cannot by themselves cause otitis. This list includes: conformation, excess moisture, obstruction, systemic disease, and treatment effects. Sometimes it helps to reassure our clients that there are Cocker Spaniels without ear disease despite their floppy ears and Poodles without ear disease despite their excess ear hair. The last category, Perpetuating, is the one most often neglected in veterinary practice. Perpetuating factors occur as a result of the otitis and increase the likelihood of another infection. These factors are: excess cerumen production, altered epithelial migration, edema of the ear canal, rupture of the tympanic membrane, and otitis media. I believe that the first three issues in the perpetuating category are most overlooked and least understood in private practice. Excess cerumen production occurs any time there is inflammation in the ear canal. The body’s response is to make more cerumen in an attempt to push out whatever is happening. Unfortunately, this excess cerumen can be a great growth medium for yeast and bacteria. Cerumen production can continue to be excessive for several weeks after the infectious component of the otitis initially resolves. For this reason, it is beneficial to continue ear cleaning even after ear infection has resolved. Altered epithelial migration also develops during otitis externa. Normal otic epithelial migration starts at the tympanic membrane and marches distally out the aural orifice. This too is designed to help move debris out of the ear canals. However, inflammation within the canal disrupts this process resulting in build-up of debris in the canal. Altered epithelial migration is another reason why stenotic ears and cobblestone ears demonstrate wax build-up. Again, it is necessary to continue ear cleaning until this process is re-established. Edema in the ear canal is at least a problem you can see through the otoscope. But, the importance of edema is often underestimated. Edema will also trap cerumen which can potentially lead to a better environment for bacteria or yeast growth. Edema can also cause discomfort and pain which could result in ear pruritus and trauma.

Treatment for otitis externa starts with the PSPP system. In most cases, you can run through the PSPP list in your mind just like you would a check list for any other disease. In most cases you can quickly rule out ear mites, foreign objects, and polyps. You might need to perform blood tests to look for endocrine disease if other suggestive signs are present. Similarly, you might need to perform skin biopsy to look for auto-immune or immune mediated disease if other supportive lesions are present. In the majority of cases you are not going to find any of the problems listed above in this paragraph. The majority of otitis externa in the canine is secondary to allergic dermatitis. In that case, the first question to ask yourself is whether the allergy is controlled or not. If the allergy is generally well controlled and the otitis externa is due to a flare or a dietary indiscretion then resolving the problem will be easier. Well controlled allergy patients may still have one or two episodes of otitis externa each year. If the allergy is unknown or un-treated then
you will have more work to do. Not the least of which will be convincing the owner that their dog has allergies. Still, you may choose to focus initially on the otitis and address the allergy in two to four weeks.

The first step toward treatment is otoscopic exam. You need to assess pain, pruritus, edema, erythema, constriction, and exudate as well as the tympanic membrane. Next you will need to perform ear swab cytology. It is best to collect exudate from both the horizontal and vertical portions of the ear canal. Obviously you are checking for *Malassezia*, coccoid bacteria and rod shaped bacteria. But you are also looking for nuclear streaming, white blood cells, red blood cells, and evidence of biofilm. Bacterial culture from the ear canal may also be necessary depending on the situation. Ear cultures are not universally helpful for two reasons. First, you might culture normal flora. Second, MIC’s are usually based on serum levels of antibiotics. In the ear we are concerned about topical / direct exposure to the antibiotic. The essence is of the problem is that some antibiotics to which the bacteria are listed as “Resistant” will actually be “Sensitive.”

Now that you have performed an exam and evaluated cytology you have to choose a therapeutic plan. I want to stress that there isn’t one universal plan for otitis externa. We can’t group treatment into levels such as easy, moderate, and severe either. However, asking yourself the following six questions can help you make better treatment decisions.

1. Is there an allergy and are you treating it now?
2. How much debris is in the ear canal?
3. How is the conformation of the ear canal
4. What type of infection is present?
5. How much edema and erythema are present?
6. How much pain and anxiety are present?

**Now, in more detail**

1. Is there an allergy and are you treating it now? You may not treat allergy at the first visit for otitis externa. But, you should at least start the conversation about allergy.
2. How much debris is in the ear canal? This will help you decide what type of cleaner to use and how often. For thick sticky wax you will probably want a micellar solution or one with squalene. For mucoid exudate you will probably want a Triz EDTA product with Chlorhexidine.
3. How is the conformation of the ear canal? Is it constricted? Cobblestoned? This too will help you decide what type of ear wash to use and whether to use a topical medication that is a gel, ointment, or liquid. The more the canal is constricted the more you need a wash that is better at dissolving cerumen. Ointments are less likely to travel deep into a constricted or cobblestoned ear canal so you probably want a liquid medication.
4. What type of infection is present? This will help you pick a topical treatment. The side note is that YOU have to know what drugs are in the products on your shelf. Infection with rod shaped bacteria will also encourage you to use an ear wash with Triz EDTA. Most rod shaped bacteria are gram negative. Triz EDTA damages the gram negative membrane and forms channels which allow antimicrobials into the bacteria.
5. How much edema and erythema are present? This will tell you what strength of steroid to use. Topical steroid therapy may be sufficient or you might need oral steroid therapy as well. If the ear canals are completely constricted then you will definitely need help from an oral steroid. Again, you have to know what ingredients are in the products on your shelf! Common steroid ingredients in otic medications, in order or potency are as follows:
   a. Prednisolone
   b. Betamethasone
   c. Mometazone
6. How much pain and anxiety are present? This will tell you if you need to prescribe additional pain relief or anti-anxiety medications. These medications are short term but can really help both the dog and the owner. This might require a prescription of Tramadol, Rimadyl or Xanax. Don’t underestimate the pain or anxiety related to ear infection! How many clients have told you that their dog runs away when they see the ear wash bottle or tube or ear ointment?
Cutaneous adverse food reaction

The incidence of cutaneous adverse food reaction (food allergy) in cats is difficult to pinpoint. One study of 61 pruritic cats found that 16% had cutaneous adverse food reaction. Interestingly, 42% of the cats with cutaneous adverse food reaction also had a history of vomiting or diarrhea. It is estimated that only approximately 50% of cats with adverse food reaction respond to glucocorticoids.

Unfortunately, the cause and pathogenesis of cutaneous adverse food reaction are unknown. It is known that Toxocara cati infection can enhance IgE response to orally administered antigens.

Researchers suspect that multiple factors are important. No age or sex predilection has been reliably reported in cats. However, multiple reports suggest that Siamese cats are predisposed. The most common clinical sign is pruritus. The pruritus is nonseasonal and typically severe. Pruritus is typically focused on the face, ears and neck but can be generalized. Eosinophilic granuloma complex, miliary dermatitis, otitis externa, angioedema, urticaria, and conjunctivitis can all be seen as a result of cutaneous adverse food reaction as well.

Diagnosis of cutaneous adverse food reaction requires eliminating other potential diseases by collecting a minimum database. In feline dermatology the minimum database includes: cytology, Skin scraping, dermatophyte culture, and Wood's lamp investigation. If gastrointestinal signs are present as well then baseline biochemistry tests and fecal analysis are also recommended. Once the appropriate tests have been performed then a dietary trial can be initiated. At this time, dietary trials are the only accurate means of diagnosing cutaneous adverse food reaction. The goal of a dietary trial is to observe whether avoiding ingredients that a cat previously ingested will result in clinical improvement. As such, it is important to have at least some basic knowledge of the patient's previous diets. Prescription novel protein or hydrolyzed protein diets are recommended because of their increased consistency and reduced risk of cross contamination during processing. Home cooked diets remain a good choice for capable clients. The trial food is continued for at least 12 to 16 weeks before assessing its potential benefit. Consistent application of a quality flea control product is recommended during the entire trial to avoid fleas as a potential flare factor. Obviously, cat treats and human food are not allowed during the diet trial. It is also important to avoid flavored medications, pill pockets, and hunting. I recommend using metal or ceramic bowls because they are more easily cleaned and plastic bowls can cause reactions in and of themselves.

Flea bite hypersensitivity (flea allergy dermatitis)

Depending on your location in the country, flea bite hypersensitivity may be extremely common or completely non-existent. Experimental models have shown that intermittent exposure to fleas results in more severe hypersensitivity reactions. This is consistent with clinical experience. While animals with high flea loads can develop hypersensitivity, it is usually the patient who has relatively rare or low intensity exposure that becomes allergic. It is also useful to note that non-allergic animals who are chronically exposed to fleas usually develop partial or complete tolerance to flea saliva antigens. On the other hand, fleas are a known flare factor for animals with any type of allergy.

Flea bite hypersensitivity can develop at any age. Pruritus is typically focused on the dorsal lumbar region, flanks, tail base, perineum and tail. Clinical signs include barbered hair, papules, and erythema. While flea burden, and thus flea bite hypersensitivity, often spikes in the spring and fall, this syndrome can be a non-seasonal problem. The pruritus caused by the flea can persist for many weeks after the flea has died.

Diagnosis of flea bite hypersensitivity is usually based on appropriate clinical signs and suggestive history. Fleas, flea dirt, and/or tapeworms are sometimes found but are not required for diagnosis. Allergy testing, either serum or intradermal, can be performed; however, a positive allergy test result does not prove flea bite hypersensitivity. The results must be correlated with the history and clinical signs.

Treatment for flea bite hypersensitivity focuses on aggressive flea control. This requires both environmental measures and cat-related treatments. For treatment of the patient I recommend combining a topical and an oral flea preventative. This allows you to use two different modes of administration and two (or more) active ingredients. When using two products it is ideal to space out the products so that they are administered two weeks apart. This way each product is still administered at a monthly interval. The other benefit of this type of protocol is that the patient is always within the first two weeks of any flea treatment. In general, flea prevention products are most effective during the first two weeks. Environmental products are plentiful and easy to find either via an exterminator or at a local home improvement store. If possible, keeping all cats in the household indoors is also helpful. Multiple investigators have attempted to desensitize cats to flea saliva via immunotherapy over the past several decades; however, all attempts have failed.
Feline atopy

It is probably not a surprise to learn that atopic dermatitis (or feline allergic dermatitis) is a poorly understood disease. Most of what we know about feline atopy is extrapolated from canines and humans. As we were all told in veterinary school, cats are not small dogs! As such, expect our understanding of feline atopy to evolve over the coming decades.

The majority of cats with atopy develop clinical signs between 6 and 24 months of age. As with atopy in other veterinary species, pruritus is the most common clinical sign. Pruritus is often focused on the face and neck; however, the ventral abdomen, groin, lateral thorax, and rear legs are commonly affected as well. Pruritus often results in closely "barbered" hair or complete alopecia. Macules, papules, and crusts can develop as well. Lesions can appear identical to those of adverse food reaction and flea bite hypersensitivity. In addition, two or more allergic conditions can occur in the same patient. In general, cats are less likely to develop secondary pyoderma than atopic dogs. However, cytology is still important because *Malassezia* spp. overgrowth can occur.

Non-dermatological symptoms may develop as well. Such symptoms can include sneezing, coughing, asthma, and conjunctivitis. When other body systems are affected it is important to expand your minimum database to rule out other diseases which might coexist with atopy.

Allergy testing, either serum based or intradermal is a useful test for feline patients with appropriate clinical signs. Admittedly, allergy testing is performed less often in cats because many cat owners are not willing or able to give antigen injections at home. Other treatment options include antihistamines, steroids, and cyclosporine. Antihistamines are generally not effective enough to control clinical signs of atopy in cats. However, they are safe and inexpensive. Some owners prefer to start with antihistamine therapy before proceeding with other more aggressive options. My two favorite antihistamines in cats are Chlorpheniramine (1-2 mg twice daily) and Amitriptyline (5-10mg once or twice daily). Antihistamines should be given consistently for at least 14 days before assessing the patient for response. Steroids are undeniably the fall back for allergy treatment. In some cases they are even the best choice. But, we need to be smart about steroid therapy. In general I recommend oral steroid therapy over injectable steroid therapy. Oral therapy allows you to adjust the dose more effectively over time. Oral therapy can also be stopped quickly if side effects are observed or another unrelated problem develops. Remember that Prednisolone is more effective than Prednisone at least in terms of treating dermatologic problems. However, my favorite oral steroid for cats is Triamcinolone. You can roughly translate your typical prednisolone dose to a triamcinolone dose by remembering that 5mg of Prednisolone is similar to 0.5mg of Triamcinolone. Often, Triamcinolone can be tapered to every 2-3 days whereas this is uncommon when using Prednisolone. This brings us to an important point about steroid therapy. The dose, duration, and frequency of steroid administration are all important in the development of side effects. Oral therapy allows for every other or every third day dosing which reduces risk to the patient. No discussion on steroid therapy would be complete without mention of Depo-Medrol. For some cats and some owners Depo-medrol may be your only reasonable choice. However, please keep in mind that Depo-medrol remains in the body for three months regardless of how effectively it controls the allergy symptoms. This means that when symptoms return a month after the Depo-medrol injection it is not because the steroid "wore off but rather because the patient is becoming less responsive to methylprednisolone. In cats the primary concerns with steroid therapy is the development of overt diabetes, congestive heart failure, or hyperadrenocorticism. Cats can demonstrate other symptoms such as poor hair coat, alopecia, seborrhea, and thinning of the skin. The last oral therapy for atopic dermatitis in cats is Atopica® (modified cyclosporine). Both Atopica liquid and capsules can be given to cats. Studies examining the long term effects of Atopica® on CBC and Chemistry analyses indicate that abnormalities are uncommon. However, it is recommended to collect a blood sample prior to starting Atopica® so that you have a baseline. Included in these tests should be CBC, Chemistry, FeLV, FIV, and a fecal float. I also typically perform testing for Toxoplasmosis although studies have shown that the label dose of Atopica® is unlikely to activate a dormant Toxoplasmosis infection. Repeat monitoring of these tests is recommended every six months depending on the patient's condition and Atopica® dose. In my experience most cats require daily Atopica® therapy to remain complete control of allergy symptoms. However, some owners may willingly trade less perfect allergy control if the result is that they can medicate their cat less frequently. Regardless of the treatment chosen, life-long therapy is almost always necessary.

Atopica®. In addition, cats can receive Atopica with or without a meal. Studies examining the long term effects of Atopica® on CBC and Chemistry analyses indicate that abnormalities are uncommon. However, it is recommended to collect a blood sample prior to starting Atopica® so that you have a baseline. Included in these tests should be CBC, Chemistry, FeLV, FIV, and a fecal float. I also typically perform testing for Toxoplasmosis although studies have shown that the label dose of Atopica® is unlikely to activate a dormant Toxoplasmosis infection. Repeat monitoring of these tests is recommended every six months depending on the patient's condition and Atopica® dose. In my experience most cats require daily Atopica® therapy to remain complete control of allergy symptoms. However, some owners may willingly trade less perfect allergy control if the result is that they can medicate their cat less frequently. Regardless of the treatment chosen, life-long therapy is almost always necessary.

Eosinophilic granuloma complex

Eosinophilic granuloma complex includes indolent ulcers, eosinophilic plaques and eosinophilic (linear) granulomas. These terms are used to describe a clinical sign, not a final diagnosis. An allergic condition usually underpins the development of these lesions. Possible causes include inhalant allergies, environmental allergies, food allergies, and insect hypersensitivities. Bacterial and viral infections can be a factor but are rarely the primary cause. It is suspected that some cats are genetically predisposed to this syndrome without having classical signs of allergic dermatitis.

What must be remembered is that these lesions usually coincide with an underlying allergy. In certain circumstances, observing one of these lesions can narrow your differential list. For example, if you are examining a cat that has been over-grooming its
abdomen and you notice that it also has a swollen lip or chin you should think allergic disease instead of a pain related condition or behavioral overgrooming. When speaking to clients it is important to mention the potential of an underlying allergy. If allergy is present then it will need long term control in order to keep the lesions from returning. So, when you observe one of these lesions you should have "the allergy talk" and consider: 1) What is the animal's flea prevention status? 2) Is a dietary trial feasible? 3) Is allergy testing feasible/useful? 4) Is Atopica® a good solution for this cat? 5) Is chronic steroid therapy the best solution in this situation?

A few additional practice tips
1. Always check a cat's lip margins and chin for lesions. "Fat chin" cats are displaying a form of eosinophilic granuloma which is likely secondary to some type of allergy.
2. Plaques, linear granulomas, and rodent ulcers almost always return because the underlying allergy does not self-cure.
3. Some lesions need to be biopsied to rule out neoplasia and infectious disease. Consider biopsy when lesions don't respond well to steroids, lesions cover a large surface area, the patient is older, or the patient is allowed outdoors.
4. While not part of the eosinophilic syndrome, milliary dermatitis can be thought of in much the same way. It is a unique reaction pattern in the feline that occurs most often secondary to some form of allergy. Milliary dermatitis develops without self-trauma (licking or scratching) caused by the patient.

Otitis externa
Thankfully, otitis externa is uncommon in cats. When present, otitis externa in cats is usually secondary to allergies, ear mites, or polyps/tumors. Obviously, cytology and otoscopic examination are critical to helping you identify infection, ear mites, and masses in the ear canal. We are going to focus on allergy related otitis externa because it is often overlooked. Allergy related otitis externa can result in bacterial otitis, malassezia otitis, excess cerumen production or simply otic pruritus. Allergic otitis can occur with or without other allergy symptoms. Or the symptoms may be mild enough that the owner doesn't mention them to you. As usual, a thorough examination and thoughtful questioning are important. In many ways, treating feline otitis is the same as treating canine otitis. However, here are some useful tips to remember:
1. Cats typically don't arrow deep ear cleaning unless sedated
2. Few medications are labelled in the United States for treating otitis in cats.
3. I typically use Posatex® because of once daily application. Posatex® is also less likely to cause deafness than gentamicin products.
4. If an underlying allergy is present it needs to be treated or you will "fight" the otitis forever.
How to Win Friends and Influence People with Topical Therapy

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Shampoo therapy is an important treatment modality for veterinary patients with dermatologic issues. Unfortunately, shampoo therapy is often neglected by the busy practitioner and pet owner. There are many reasons why veterinarians should have at least a basic understanding of shampoo therapy. First, many of our clients are curious about shampoo therapy. This provides the veterinarian with an opportunity to educate the client and provide better patient care. With the rise of the internet and the mega-box store environment, our clients are exposed to an ever expanding pool of information. Just like in other facet of veterinary medicine, the information that our clients receive about shampoo via marketing or the internet is often misleading or downright wrong. Taking this opportunity to educate the client reinforces the veterinarian's position in the client's life and as part of their pet's health care team.

Being able to quickly and confidently answer the most common shampoo questions will help both you and your staff. The most common question I am asked is, "How often should I bathe my dog?" The best answer to this question is that the frequency of bathing depends both on the dog’s health status (taking into account the hair condition, skin condition, and internal health) as well as the shampoo being used. When using medicated shampoos to treat a specific skin problem you should plan to bathe at least weekly to observe a benefit. Twice weekly or every other day bathing is even more helpful. The second most common question I am asked is, "Can I just use my shampoo?" The answer to this question is more straightforward, "No." Dog skin has a normal pH of 7 and human skin has a normal pH of 5. Human shampoo may seem to effectively clean the dog's hair but it is not ideal for the skin underneath.

Now that you have educated the client and convinced them that bathing their pet is a good idea you need to make sure they know how to bathe their dog properly. This is often assumed by our profession but it should not be. The first topic in discussing proper bathing is the water. The water should be cool to tepid because hot water will increase pruritus. In addition, a study confirmed that soft water allows the active ingredients in your shampoo to work better. Prewashing is another helpful tip that can reduce the amount of medicated shampoo your client uses per bath. Another big factor in the effectiveness of the shampoo you prescribe is the contact time. Having 10-15 minutes of contact time is critical for proper activity of medicated shampoos. Suggestions to make 10-15 minutes pass more quickly include: playing 3-4 of your favorite songs in the bathroom while waiting, taking your dog for a walk while it is soaped up, and wrapping your dog in a towel and watching television with them while they are soaped up. Once the bath is over it is important to encourage your clients to avoid using hair dryers. Even on the cool setting hair dryers will dry out the skin.

In order to confidently recommend medicated shampoo you need to understand why it is important. The two most common conditions for which you will prescribe medicated shampoo are allergic dermatitis and pyoderma. Shampoo therapy can act synergistically with oral medications to help eliminate infection, improve moisture in the skin, and reduce pruritus. Adding a shampoo might allow you to use a shorter course of oral antimicrobial therapy or a lower dose of steroid. In addition, topical treatments work quickly to remove bacteria and inflammatory mediators thus helping provide an immediate response. Lastly, using topical antibacterial products will help avoid antibiotic resistance.

Next, you need to have a basic understand of shampoo ingredients. It is beyond the scope of this lecture to discuss every possible shampoo ingredient. Consequently, we are going to focus on the most common and most useful active ingredients in three categories. The categories we will discuss today are: antimicrobial, antiseborrheal, and antipruritic.

The first ingredient we will discuss in the antimicrobial section is chlorhexidine. Chlorhexidine is a common antimicrobial agent in many veterinary products so most veterinarians feel comfortable and familiar with this ingredient. But there are many benefits of chlorhexidine worth mentioning. A study published in 2013 showed that chlorhexidine bathed hairs retained an antibacterial quality for several days after bathing. Thus, the antibacterial benefit of chlorhexidine extends for days even after the bath is over. Another study, this one published in 2012, indicated that chlorhexidine was bactericidal at lower concentrations than other ingredients. The study went on to show that chlorhexidine was also the fastest acting bactericidal ingredient. Second, we will talk about miconazole and Ketoconazole. Many of us are familiar with these ingredients for their antifungal properties. However, studies have shown that combining miconazole with chlorhexidine provides even more antibacterial activity. It is worth noting at this time that miconazole and ketoconazole shampoos are not effective as sole therapy for dermatophytosis. Emollients and lime sulfur dips remain the best choices for topical therapy of dermatophytosis. The next ingredient to know is ethyl lactate. Ethyl lactate is a useful antibacterial agent that also has degreasing action. The last antimicrobial ingredient to be familiar with is benzoyl peroxide. Benzoyl peroxide has a several useful properties in addition to its antimicrobial effects including: flushing follicular, reducing comedones, and reducing seborrhea. In some cases you will find it helpful to alternate between antibacterial shampoos such as Chlorhexidine and Benzoyl Peroxide. You may also find patients who are extremely sensitive to Chlorhexidine and thus require an alternative antimicrobial agent.

Antiseborrhea agents are easily the most confusing group of active ingredients. Two terms that are frequently used when talking about antiseborrhea shampoos are: keratolytic and keratoplastic. Keratolytic means that the ingredient breaks the bonds between corneocytes in the stratum corneum (the upper most layer of the epidermis). Keratoplastic means that the ingredient alters the
replication characteristics of the dividing cells in the stratum basale (the bottom most layer of the epidermis). These terms are not very helpful in general practice and in reality many of the active ingredients are both keratolytic and keratoplastic. From weakest to strongest the ingredients are: zinc gluconate, benzoyl peroxide, selenium sulfide, salicylic acid, sulfur, and coal tar. At this time you cannot purchase a veterinary prescription shampoo with zinc gluconate or selenium sulfide. Selenium is classically considered the ingredient in Selsun Blue®. This is not a shampoo I recommend for several reasons. First, there are many Selsun Blue® products and not all of them contain selenium. In addition, we want to discourage the use of human shampoo. So we'll talk about benzoyl peroxide first. It is important to remember that benzoyl peroxide can be an effective antibacterial agent as well as an antiseborrhea product. Benzoyl peroxide is also very drying so many shampoos will also include moisturizing agents. Salicylic acid can be found alone, or more commonly, combined with other active ingredients. Salicylic acid is a very effective keratolytic ingredient Sulfur is the strongest anti-seborrhea agent that you will find in a prescription shampoo. The effects of sulfur are directly related to the concentration used. This makes sense if you consider Lime Sulfur dip at most powerful end of the sulfur spectrum. Sulfur can be keratolytic and keratoplastic as well as antimicrobial. The last anti-seborrhea agent is tar. Tar is no longer available in a prescription shampoo but it needs to be mentioned because OTC tar shampoos can be easily purchased. Tar is an extremely potent antiseborrhea agent. However, part of the way it accomplishes this is by being severely keratoplastic. Remember that tar is a carcinogen! Tar is toxic to cats and also very drying. I do not recommend using tar shampoos except under special circumstances and under the supervision of a veterinarian.

Our next group of shampoo ingredients are aimed at reducing pruritus. This group includes diphenhydramine, pramoxine, and hydrocortisone. Diphenhydramine (Benadryl®) has weak topical activity in dogs and cats. Pramoxine is a topical numbing agent that dulls the nerve endings in the skin thus reducing the sense of itch. Some animals respond very well to Pramoxine. Hydrocortisone is a common ingredient in anti-pruritic shampoos and it can be effective in some patients. It is important to remember that hydrocortisone is the weakest of the synthetic steroids so you can't expect a topical hydrocortisone product to resolve all of your patient's itching. Lastly, one note about inactive ingredients. The quality and blending of the inactive ingredients is important for factors like fragrance, lather-ability, and rinse-ability. These factors should not be overlooked because these are things that our clients notice. To make the shampoo dilemma more complicated, inactive ingredients can also affect the efficacy of the active ingredients. This underscores the importance of using a reliable brand of shampoo.

Next I want to review relevant shampoo technology. This includes Novasomes® by Vetoquinol®, Spherulites™ by Virbac®, Triz EDTA from Dechra®, and ceramides. Novasomes® are microscopic droplets with an outer lipid membrane and an inner water core. These droplets stick to the hair shafts electrostatically during bathing and then degrade slowly over time. This provides a slow release of moisture to the skin. Spherulites™ are somewhat similar. However, Spherulites™ have multiple layers of active ingredient and water. During bathing the Spherulites™ adhere to the hair and then degrade slowly over time providing both further activity and moisture. Triz EDTA is an ingredient that many veterinarians are familiar with because of ear rinse solutions. Triz EDTA is helpful in the fight against gram negative bacteria because this agent is able to form pores through the lipopolysaccharide membrane. Active ingredients including antiseptics and antibiotics can then pass through the gram negative membrane to kill the bacteria. Ceramides are a group of natural oils that comprise the major component of the lipid layer in the stratum corneum. Cholesterol and free fatty acids make up the rest of the lipid layer. Studies in humans have shown that damaged skin and allergic skin have reduced ceramide levels. Ceramide levels also decline after a certain age (30 years old in people). Ceramides are important for a number of reasons. First, low ceramide levels cause dry skin and dry skin is itchy. Second, low ceramide levels allow penetration of bacteria and yeast into the deeper layers of the epidermis and dermis. This is one reason why bacterial infections develop rapidly and spread quickly in allergic patients. Lastly, ceramides are important for limiting antigen exposure. Over the past couple years we have learned that most allergen exposure in our companion animal species occurs via cutaneous absorption. This is one more way that allergies predispose our patients to an ever worsening cycle of skin disease. The genetic predisposition for allergic dermatitis equates to lower ceramide levels in the skin. Lower ceramide levels allow more antigen penetration which results in allergy related inflammation. That inflammation further lowers ceramide levels and allows more antigen penetration.

Before we finish shampoo therapy I want to offer my suggestions for the types of shampoo you should have in practice. There are many good companies selling quality veterinary shampoos. But, you don't need every shampoo that a company produces. You might also not have just one brand of shampoo. I suggest you try them for yourself or have a couple of your best clients try them and give you their opinions. I do recommend that you keep your shampoo inventory as lean as possible. This will avoid confusion among technicians and lay-staff when reinforcing your recommendations. The three types of shampoo that you absolutely need are: 1) An antimicrobial shampoo. I prefer a shampoo with Chlorhexidine, Miconazole, and ceramides because you can use such a product for any skin infection. 2) A benzoyl peroxide shampoo. You will use this ingredient most commonly in patients with demodicosis but it will also serve as a back-up for bacterial pyoderma. Benzoyl peroxide will also handle mild seborrhea cases. 3) A moisturizing general cleaning shampoo. Having a quality shampoo you can feel good about recommending will help keep your clients out of the shampoo isle at the pet store. If the shampoo you choose is packed with moisturizers then it will be good for some of your dry flaky dogs too.

We are fortunate to now have topical therapy options beyond the scope of shampoo therapy.
This includes medicated wipes, sprays, lotions, and mousse products. The most common situations to use these products are for infections and moisturizing. Just like with shampoos, many different companies offer good products. The benefits are similar to shampoo therapy with added convenience (which translates into improved compliance). Medicated wipes are great for small areas of infection (skin folds especially) and easily allow twice daily treatment. Sprays and mousse products are great for larger areas! I recommend using these products twice daily to help resolve infection then tapering for long term control of problem areas.
Calcinosis cutis
Calcinosis cutis describes the deposition of calcium salts into dermal tissue (usually calciumphosphate or calcium carbonate). Four types are recognized in humans: Iatrogenic, Idiopathic, Dystrophic, and Metastatic. There is overlap between categories and these labels are not particularly helpful in veterinary medicine. The most common cause of calcinosis cutis in veterinary species is hyperglucocorticoidism. Typically this is either secondary to hyperadrenocorticism (endogenous or exogenous) or steroid administration. Lesions typically develop on the dorsal neck and then spread caudally down the topline. Localized calcinosis cutis can occur secondary to chronic application of topical steroids. This is often apparent on the caudal ventral abdomen secondary to steroid sprays. Less commonly, calcinosis cutis can develop secondary to percutaneous absorption of calcium. Such exposure to calcium can occur when a pet comes in contact with certain floor cleaners, fertilizers, and ice melt products.

Calcinosis cutis can occur in any breed but it is more common in English Bulldogs. It is also more common in patients receiving Depo-Medrol injections. The clinical appearance of calcinosis cutis changes over the progression of the syndrome. Early lesions are chalky white to pink with indistinct margins. More advanced lesions are white, firm, and usually surrounded by intense inflammation. Pruritus is usually present and may be severe. Ulceration is common and secondary infection usually follows.

Diagnosis is easily confirmed with biopsy as the changes are unique and often dramatic. Collect biopsy samples from areas not affected by self-trauma and ulceration. Biopsy reveals diffuse ormultifocal calcification of dermal collagen. Epidermal thickening and dermal edema are typically present as well. Calcinosis cutis is one dermatologic condition that can be seen on radiographs. It should be noted that serum calcium levels are not elevated in this syndrome.

Treatment involves eliminating exposure to environmental calcium and discontinuing steroid administration. If neither of these are a factor then cortisol testing is recommended as hyperadrenocorticism is very likely. If the patient does have hyperadrenocorticism then that condition needs to be managed in order to eliminate the calcinosis cutis. Patients without hyperadrenocorticism or exposure to external calcium or steroid containing products may have other severe systemic disease such as renal disease. Alternatively, some cases will develop secondary to repetitive micro-trauma (lesions typically on the pressure points of the limbs).

No treatment directly removes the calcium (aside from surgical excision). DMSO gel can be used to dissolve the calcium deposits. However, DMSO should be applied twice daily and may require weeks to months of treatment. Most owners cannot tolerate the smell of DMSO in their house for that length of time. Without DMSO, the calcium deposits will dissolve in two to twelve months. Patients with a history of calcinosis cutis should not receive steroid therapy in the future.

Hepatocutaneous syndrome
Hepatocutaneous syndrome has also been called: Superficial necrolytic dermatitis (SND). Metabolic epidermal necrosis (MEN), Necrolytic migratory erythema (NME), and Diabetic dermatopathy. I recommend against using the term Diabetic dermatopathy because it is confusing and not descriptive. In addition not every dog with Hepatocutaneous syndrome has diabetes.

The pathogenesis of hepatocutaneous syndrome involves death of keratinocytes in the upper layer of the epidermis due to presumed amino acid starvation. Most affected dogs have a distinctive chronic hepatopathy; but, serum chemistry evaluation may not reveal any abnormalities. Potential causes of the hepatopathy include phenobarbital, primidone, mycotoxin, and gastro-enteritis. In humans this syndrome is almost always associated with glucagonoma. However, Glucagonoma is rare in dogs and accounted for only 8% of cases in one study.

Hepatocutaneous syndrome is generally a disease of older dogs. Only four cases have been reported in cats. Skin lesions are typically the first sign as opposed to more common systemic signs of liver disease. Crusts and erosions occur in areas of trauma I wear. Thus, the paw pads are usually severely affected. The elbows, hocks, and muzzle are frequently affected as well. Many affected patients are often reluctant to walk due to painful erosions and fissures on the paw pads.

Diagnosis requires biopsy of skin lesions with intact crusts. Histopathologically the changes are often described as a "French Flag". Abdominal ultrasound can also be very helpful. A classic Mhoney comb" pattern to liver is present in most cases of Hepatocutaneous syndrome. However, inexperienced ultrasonographers may misinterpret the liver changes. In addition, the degree of change found on ultrasound does not necessarily correlate to severity of skin disease. CBC, serum chemistry, and urinalysis are also recommended. Nonregenerative anemia is common due to chronic disease. As stated before, liver values may or may not be elevated. Hyperglycemia is common and may require insulin therapy. Glucagon levels are elevated in patients with glucagonoma. However, glucagonoma is rare in dogs and cats and glucagon measurement is not readily available.
Cutaneous lymphoma

Cutaneous lymphoma is an uncommon malignant neoplasia of the dog and cat. Two types of cutaneous lymphoma are recognized: epitheliotrophic and non-epitheliotrophic. Epitheliotrophic lymphomas are classified T cell lymphomas and include mycosis fungoides, Sezary syndrome, and pategoid reticulosis. Non-epitheliotrophic lymphomas are typically large cell lymphomas and can be either B or T cell in origin. Older animals are usually affected but this disease can occur at any age.

These neoplasms are important even though they are rare because they imitate many other diseases. Non-epitheliotrophic lymphoma typically manifests as single or multiple nodules. Exfoliative erythroderma may occur separately or in addition to nodular disease. Patients with exfoliative erythroderma can easily be misdiagnosed as allergy, scabies, or seborrhea. If the mucus membranes and/or muzzle are affected by non-epitheliotrophic lymphoma it can appear visually indistinct from lupus erythematosus, pemphigus vulgaris, and bullous pemphigoid.

The most common epitheliotrophic lymphoma is mycosis fungoides. This condition displays multiple clinical manifestations. Erythroderma is typically present (same as non-epitheliotrophic). Once again, this erythroderma may appear visually indistinct from allergy, scabies, and seborrhea. Focal lesions progress from patches to plaques to tumors. The final stage involves widespread dissemination of tumors with lymph node involvement. Multiple types of lesions can be present at the same time and the speed of progression is not predictable or consistent. Additionally, initial lesions can be very subtle. For example, a client may notice the development of dry flaky seborrhea. During examination you might find a couple small patches of alopecia without inflammation and a nodule which the owner cannot remember.

Diagnosis is relatively straightforward via biopsy. The point of this lecture is merely to encourage you to biopsy older animals or animals with sudden onset of disease more quickly. Cytology is always recommended as well. Occasionally you will find an unusually large population of lymphocytes on cytology when what you expected was neutrophils and cocci.

Therapy depends on the location and the extent of the disease. Consultation with an oncologist should always be recommended. Survival time varies greatly based on aggressiveness of the neoplasia and when the disease is diagnosed. In my clinical experience, most patients survive 2-3 months after diagnosis but this can range from a few weeks up to 18 months. For clients un-interested in oncology referral or classical "chemotherapy" I recommend steroids as monotherapy. Steroid monotherapy can provide 1-3 months of quality time by reducing the intensity of lesions and subsequent discomfort.
Pemphigus foliaceus

Pemphigus foliaceus is one of the most common auto-immune skin diseases seen in dogs and cats. This disease is characterized by pustules and honey colored crusts. This condition is typically idiopathic but it can develop secondary to drug exposure. Pemphigus foliaceus is often seen in patients previously diagnosed with allergic dermatitis; however, no link between the two has been proven.

In pemphigus foliaceus the immune system is attacking a particular protein in the complex structure (called a desmosome) that links keratinocytes together. Destroying the bonds between keratinocytes is termed acantholysis and results in acantholytic cells. Acantholytic cells are typically plump and round because they are no longer connected to their neighbors. They stain darkly and have a clearly visible nucleus. Different forms of pemphigus exist and one of the primary differences between them is what layer of the skin this acantholysis occurs. For pemphigus foliaceus the damage occurs in the two uppermost layers (the stratum corneum and the stratum granulosum). More serious forms of pemphigus affect deeper layers of the skin and cause significantly more damage. As acantholysis occurs, vesicles and sterile pustules are formed. These are fragile and easily damaged because they are located in the uppermost layers of the epidermis. Depending on the intensity of the immune response, pustules can develop and rupture in under an hour or over the course of days. For comparison, pyoderma pustules develop more slowly and are more resilient (more difficult to break). In addition, pyoderma pustules are typically centered around a hair follicle. Both pemphigus pustules and pyoderma pustules will contain neutrophils but intact pemphigus pustules will not contain bacteria.

As already mentioned, the classic lesions of pemphigus foliaceus are pustules and crusts. These lesions can occur anywhere on the body but are commonly found on the face and trunk. Pustules can develop inside the aural opening resulting in serum leakage and crust debris falling into the ear canals. The result is typically a wicked otitis externa. In many cases the nasal planum is also abnormal. The planum typically becomes dry, thick, and crusted. Ulcerations of the nasal planum can occur secondary to crust being traumatically removed. However, pemphigus foliaceus does not cause ulceration of the oral cavity or mucus membranes. The paw pads may be affected as well. Discreet pustules may be seen on the pads but more often the pads are thickened, dry, and crust. Some dogs will be reluctant to walk but that is uncommon with pemphigus (much more common with hepatocutaneous syndrome).

Diagnosis is via biopsy. Intact pustules are preferred because they offer the clearest picture of the disease process. However, crusts are also very useful biopsy specimens. When collecting biopsies for potential pemphigus foliaceus it is critical not to scrub the skin. In most cases it is advised to avoid shaving the animal's fur as well. Even the slightest disturbance to the skin can damage the fragile pustules seen with this condition. In the event that no pustules are present, the proof of pemphigus might be in the crust on top of the skin rather than in the skin sample itself. Consequently, always include crust debris in the formalin jar and request the crust be processed when you biopsy for pemphigus.

Treatment, which is really to say management, is almost always successful but required life-long. Some cases of drug induced pemphigus foliaceus will remain in “remission” even when immune suppressive therapy is discontinued. However, it is often difficult to prove which cases are drug induced which makes predicting which patients will be able to stop therapy nearly impossible. Initial therapy requires steroid administration. Oral daily prednisolone/prednisone dosages of 2mg/kg to 6mg/kg are often required. Steroid therapy often yields dramatic improvement in two to four weeks when dosed adequately. Some patients will respond better to other steroids such as dexamethasone or triamcinolone. Recheck examinations every two to four weeks are critical to assess response to therapy and tailor drug therapy. Secondary bacterial infection is common in pemphigus and your clients will not be able to discern the difference between a pyoderma pustule (which needs antibiotics) and pemphigus pustule (which would cause you to evaluate your immune suppressive plan). In general, the goal is to slowly taper steroid therapy once clinical "remission" has been achieved. Over the course of three to four months some dogs will achieve good clinical response and can be maintained with every other day steroid therapy. However, the majority of patients will experience significant steroid side effects (such as weight gain, polyuria, polydipsia, polyphagia, behavioral abnormalities). Because of steroid side effects and the fact that most patients require life-long immune suppressive therapy it is typically necessary to add another medication as a steroid sparing agent. First line drugs for this purpose are cyclosporine and azathioprine. Second line drugs include mycophenolate and leflunomide. In most cases, I will start a steroid and a steroid sparing drug at the beginning of treatment. All of the above listed steroid sparing drugs have a delay of four to eight weeks until they become clinically effective. By starting both types of drugs at the same time I am able to reduce steroid therapy sooner.
Hypercortisolism is seen when there is an excessive amount of glucocorticoids present in the patient’s body. This increase in steroid levels can either be due to endogenous or exogenous glucocorticoid sources. A similar clinical appearance will be present in both sets of patients no matter the underlying cause of the increased cortisol levels. To make it even more difficult, many times these patients present to the dermatology office with no evidence of PU/PD, panting, polyphagia, pot belly, and/or liver enzyme elevations to support the diagnosis. Some clinicians believe that unless these systemic signs are present, a diagnosis of hyperadrenocorticism is very unlikely and thus a patient should not be treated. However, many times cutaneous signs can precede systemic signs and one should always screen for this disease if the clinical suspicion is high.

Iatrogenic hyperadrenocorticism

In veterinary dermatology, oral, injectable, and topical steroid use is rampant, so sometimes it may be difficult to determine if a particular patient could have natural or iatrogenic cushings. It is very important to get a very accurate medication history from these patients. Clients many times will forget about topical medication administration which can sometimes contain very potent topical steroids. The author has seen two cases where the owners were using so much of a topical steroid powder that they both developed iatrogenic cushings and were misdiagnosed as having addisons disease after both patients experienced gastrointestinal distress due to a completely unrelated issue.

In order to test for iatrogenic cushings the recommended protocol requires the collection of a pre-ACTH serum sample, prior to administration of 5 µg/kg of synthetic cosynorphin, IV. A post-serum sample is then collected 1 hour later. A dog with iatrogenic HAC should have a flat-line response, generally with both baseline and 1 h post-stimulation values of < 1 µg/d. It is important to note that even short courses of glucocorticoids may inhibit the adrenal response to ACTH for up to a month or more.

Calciosis cutis

Calciosis cutis is an uncommon occurrence, in which inorganic, insoluble mineral salts are deposited in the dermis, subcutis or, rarely, the epidermis. When related to excess steroids it is considered dystrophic mineralization. Lesions are firm white-pink to yellow firm dermal papules to plaques. Overtime as this calcium remains in the skin the area will start to become ulcerated and create large crusts which can become very pruritic. These changes tend to occur on the dorsal neck, rump and axillary and inguinal regions. These areas can get frequently infected with both bacteria and yeast which can lead to pain. Anytime these changes are seen clinically, or via a biopsy, this patient must be screened for Cushings disease and ensure no exogenous steroids are being given. One must remember that this disease will get better before it gets worse. One must also warn the clients that the calcium deposits will continue to appear for some time after diagnosis and treatment for hyperadrenocorticism is started. Controlling the cortisol levels will be the best long term treatment but DMSO has been shown to help resolve the lesions as well. DMSO is generally applied daily to twice daily to the lesions but can have a very strong odor for some clients. I also always warn by owners to wear gloves when applying given the medications ability to help with topical drug absorption. It is recommended with more extended use that serum calcium levels should be monitored to ensure that excess calcium is not entering the blood stream. However, the author has never seen this occur clinically.

Depending on the extent of the calcium deposits sometimes patients will have clinical lesions for years and years and some owners elect to surgically remove the more problematic areas.

Adult onset demodex

Any patient that presents with skin lesions that first develop at a middle to older age should always be skin scraped to ensure demodex mites are not present. In one retrospective study 20% of patients diagnosed with adult onset demodicosis were later diagnosed with hyperadrenocorticism as the underlying disease. Clinical lesions are similar to those seen in juvenile onset demodicosis. Lesions include erythema, comedones, scaling progressing to partial to complete alopecia with papules, pustules, and hyperpigmentation. Severe cases may be associated with lymphadenopathy, lethargy and fever as well as furunculosis with scales, crusts, exudation and focal ulceration and draining tracts. Pedal demodicosis may be associated with significant interdigital edema and, particularly in larger dogs, may be painful. A secondary bacterial skin infection almost invariably accompanies generalized demodicosis and may lead to pruritus.

Diagnosis will be by multiple skin scrapings, trichograms, and/or biopsy. Treatment will involve management of secondary infections, control of primary hyperadrenocorticism and appropriate acaricidal medications. Once the cortisol levels have been lowered patients may be able to be cured and not relapse with disease unless cortisol levels raise again.
Dermatophytosis
Dermatophytes are transmitted by contact with infected hair or fungal elements on other animals, on fomites or in the environment. Canines are usually infected with *M. canis* (transmitted from an infected cat), *Trichophyton* spp. (exposure to infected rodents), or *M. gypseum* (from the soil). Typical lesions are collarettes, scales, crusts and/or papules. This is a disease of patients less than one year of age so when present in the mature patient one must determine if there is an underlying condition that is present causing immunosuppression.

Adult onset bacterial and yeast infections
This is perhaps the most common reason I screen patients for hyperadrenocorticism. As we know it is not common for a patient, with no previous history of skin disease, to suddenly start presenting with multiple geriatric onset bacterial and yeast skin and ear infections. These adult onset infections can sometimes be very severe and yet some are relatively non-pruritic.

These patients many times will do very well while they are on antibiotics and/or anti-yeast medications and then the infection will return shortly after finishing the treatment course. Patients are screened for cushings disease based on their re-occurrence rate. I generally will not screen patients if the infection resolves quickly and without incident. However, if the infection and pruritus returns in less than 3-4 months I will recommend a low dose dexamethasone suppression test or ACTH test to be performed.

Unless the cortisol levels are managed these infections will continue to occur. This patient will often need extended course of antibiotics rather than the standard 4 weeks since they are immunocompromised until the cortisol levels drop to a normal range.

Hypersensitivity dermatitis
These cases can be the most frustrating for the clinician and the client and there are two main presentations.

One presentation is where patients present at a younger age with pruritic skin disease and then are spot treated, or even placed on long term allergy medications. As this patient ages, gradually their allergies seem to significantly improve through time. Many times owners describe to me that they feel that their animal “has grown out of their allergies”. Then slowly these animals start to have re-occurrence of their secondary infections and pruritus. These patients were essentially self-medicating their allergies with their endogenous production of cortisol. To the owner they feel that the allergies are back when in actuality it is another clinical disease.

The other common presentation is patient that have a long history of mild skin disease and suddenly patients start to get worse and worse clinical lesions as they age. Once their infections are treated they do better for a period of time and then the cycle restarts. These patients will generally have lesions first and then develop pruritus after the rash is present.

Despite the historical presentation both of these patients may slowly start having their pruritus (from their allergies) return once their cortisol levels have dropped to a more normal range. Some clients will see no real clinical change in their patient’s comfort despite medical treatment. Their pet may still get itchy and secondary infections but now it is due to their uncontrolled allergies rather than the hyperadrenocorticism. Educating owners from the start about what to expect is key especially if you know a patient has a history of allergic skin disease. That way they understand that sometimes multiple medications may be needed to keep a patient’s cortisol and allergies at a tolerable level. There have been many times when the author has needed to use more potent allergy medications once treatment for cushings has started. This may also play a role in how low you want their cortisol levels while on therapy. Some clinicians have been known to use lower doses of trilostane and/or mitotane to gather clinical benefit for allergies and sometimes osteoarthritis.

Coat changes
Some of the first signs that astute owners will notice is that gradually the coat loses its luster and becomes a coarser texture. Clients that own dogs with long hair growth cycles (poodle) may also increase the time in between grooming appointments because the hair is no longer growing as fast. This will eventually lead to hypotrichosis and then complete alopecia. The hair on the head and the distal extremities tends to be spared whereas the trunk and tail experience a majority of the changes. However, non-truncal patchy alopecia can occur in up to 13% of patients with HAC.

Along with changes in the quality and the quantity of hair the color itself can change as well. Black hairs turn auburn or light brown colored and brown hairs lighten to tan or blonde. This change in pigmentation can occur along the entire length of the hair shaft or only at the distal aspect. In the cases where only the distal tip is affected patients can appear sun bleached.

Skin changes
Other cutaneous signs include: thin skin that can wrinkle very easily, generalized and/or localized hyperpigmentation, with atrophied sebaceous glands and hair follicles. These patients will also easily bruise and create petechiae, ecchymosis and even phlebectasias. Comedones, milia and poor wound healing are also important cutaneous changes that occur in the patient with hyperadrenocorticism.
Pruritus

This is perhaps the most unexpected findings in dogs with cushings disease. However, several studies have seen a small number of cases where pruritus (pedal and limb pruritus) was present at the time of diagnosis and resolved once treatment with mitotane was started. It is thought that the pruritus may be associated with neurosis as noted in people with cushings disease.

Treatment and conclusion

Hyperadrenocorticism is a clinical disease that warrants treatment many times in the dermatological setting. Anytime a patient is diagnosed with recurrent adult onset infectious diseases (parasitic/bacterial/fungal) or calcinosis cutis one must screen for the presence of HAC. Once confirmed the cortisol levels must be lowered for those patients to lead a more comfortable life long term. Most patient’s cutaneous signs (resolution of infectious diseases, alopecia, coat changes) will resolve within about 3 months of treatment initiation but calcinosis cutis may take many years, if ever, to resolve.

References

Demodicosis (aka follicular or red mange) is an inflammatory parasitic disease of dogs that continues to pose a challenge in clinical practice, regarding management. In October 2010, an international committee was founded to establish current evidence-based guidelines for treating canine demodicosis. These guidelines, supported by the American College of Veterinary Dermatology in addition to veterinary colleges and societies in Asia, Canada, Europe, and Australia, were published in 2012. Demodex spp. are normal inhabitants of the hair follicles in most species of domestic animals and man. They are host specific and are present in small numbers in healthy individuals. Demodex canis, Demodex injai and Demodex cornei have all been identified in dogs with demodicosis. However, more recent evidence shows that these different forms are may all be different sizes of D. canis. Published data indicate similar efficacy of reported treatments regardless of the Demodex type.

Demodex mites are transmitted from the bitch to nursing pups by direct contact during the first 2-3 days of neonatal life. The host's immune system appears to detect and tolerate the presence of these mites and also has an inhibitory effect on mite proliferation. There is some evidence that mite chitin can be recognized by toll-like receptors from keratinocytes; however, the exact immunological mechanisms that control mite populations in dogs are still unknown. The initial cause of the mite overgrowth in juvenile generalized canine demodicosis is also unknown. It has been hypothesized to have a genetic basis. Once the disease has developed in a dog, indicators of T-cell exhaustion, such as the low production of supportive/stimulatory cytokines (IL-2 and IL-21) and high levels of suppressive cytokines (IL-10 and transforming growth factor-β) along with low numbers of circulating CD4+ lymphocytes have been documented. T-cell exhaustion would also provide a plausible explanation for the lack of relapse of generalized demodicosis after treatment with macrocyclic lactones. In T-cell exhaustion, the decrease in antigenic load, as occurs during the gradual resolution of infection, helps the exhausted T-cell population to regain polyfunctional attributes and more closely match typical memory T-cells.

According to this perspective, the main function of acaricidal treatment would be to reduce the parasite load to reverse the T-cell exhaustion and thereby give the host immune system the opportunity to regain control of the mite proliferation.

**Demodex canis**

Canine demodicosis or demodectic mange is a non-contagious parasitic skin disease seen in dogs. This disease is usually divided into localized and generalized disease. In young animals, endoparasiticism, malnutrition, estrus, and debilitation may lead to an immunocompromised state that favors mite proliferation and development of skin disease. In adult animals with demodicosis, disorders such as hypothyroidism, hypercortisolism [naturally occurring or iatrogenic], leishmaniasis, malignant neoplasia [especially indolent lymphoma], and immunosuppressive treatments for cancer or autoimmune diseases have been recognized. In more than 50% of cases no underlying cause was found for the adult onset demodex. However, if initial screening tests (X-rays, thyroid panels, ACTH tests, and abdominal ultrasounds) were normal patients should still be monitored carefully because the primary illness causing the demodicosis may become evident months later.

In many publications, a juvenile-onset and an adult-onset form of the disease are differentiated. However, this differentiation may be difficult in individual cases. It is more important to identify and correct predisposing factors or underlying diseases independent of age, to achieve the best possible outcome.

Localized onset demodicosis occurs where there are only a few (generally less than six) areas of alopecia and scaling in a patient where mites are discovered on skin scraping. These patients generally do not require treatment and will spontaneously resolve.

Generalized demodicosis: Exact definition varies but generally a patient is considered to have generalized demodicosis when that patient has many localized lesions, involvement of an entire body region or has complete involvement of two feet or more. May be severe and a potentially life-threatening disease. Lesions include erythema, comedones, scaling progressing to partial to complete alopecia with papules, pustules, and hyperpigmentation. Severe cases may be associated with lymphadenopathy, lethargy and fever. Generalized demodicosis is thought to be a hereditary based disease. In addition, there appears to be a Demodex specific T-cell defect that does play a role in severity and this defect is thought to be hereditary.

**Diagnosis**

Very easily done with simple multiple skin scraping, trichograms or via biopsy.

There are four stages of Demodex canis mites that can be seen on tape cytology and skin scrapings. These stages include fusiform eggs that hatch into six-legged larvae, which mature into eight-legged nymphs and progress to eight-legged adults. If one were to scrape a normal puppy numerous times in areas around the mouth and paws you will probably see a mite or two, however the puppy will not have any clinical lesions.
The skin scrapings have the best yield when primary lesions [e.g. follicular papules, pustules] chosen and skin is squeezed. If one mite is found, additional skin scrapings should be performed. Finding more than one mite is strongly suggestive of clinical demodicosis.

Trichograms are useful in areas that are difficult to scrape, such as periocular and interdigital areas. Negative trichograms should be followed by deep skin scrapings before ruling out demodicosis. Skin biopsies may be indicated in Shar-Pei dogs or from very fibrotic lesions (especially interdigital). There has been no study that has validated the prognostic value of percentages of immature forms or alive parasites on samples so I look to see if the mites are dead or alive and how many total present as my way of evaluating treatment.

**Therapy**

Localized demodicosis does not require treatment but I highly recommend periodic follow-ups to ensure that the disease is self-resolving and that the patient is not progressing into the generalized form.

For many years, veterinarians relied on a short list of medications for the treatment of generalized demodex however this has recently changed.

**Amitraz:** Traditionally, patients had been treated with dips at concentrations from 0.025% to 0.05% once a week to every other week. It is effective in 50 to 86% of patients and the higher concentration and more frequent dips were probably associated with a higher success rate. Amitraz can be toxic and the use of gloves and one will need a well-ventilated room to perform the treatment. There was a spot-on which included Amitraz that was fairly effective at treating demodex however this medication is currently not being manufactured and was associated with the development of localized and generalized pemphigus. Macrocyclic lactones (avermectins and milbemycins) are a very common choice for the treatment of demodex.

Ivermectin is an avermectin with a gamma-aminobutyric acid (GABA) agonist activity. Ivermectin is given daily at 0.3 to 0.6 mg/kg until three negative scrapings at one month intervals are obtained. The bovine injectable form of the drug is administered orally as a solution. Ivermectin should not be used in Collies, Shetland sheepdogs, Old English sheepdogs, Australian sheepdogs, and their crossbreds due to the mutation in ABCB1 (formerly MDR-1) gene. Signs of toxicosis include mydriasis, ataxia, weakness, recumbency, coma and even lead to death.

Milbemycin (interceptor®) is a natural fermentation product produced by Streptomyces hygroscopicus aureolacrimosus. It is closely related to the avermectins, produced by Streptomyces avermitilis, differing only in one position. The anthelmintic activity is believed to result from disruption of invertebrate gamma amino butyric acid (GABA) neurotransmission. Daily doses of 0.5 up to 3.1 mg/kg per day have been shown to have a 60 to 96% cure rate in a few months (up to about 1 year). Moxidectin is derived from fermentation products of Streptomyces cyaneoroseus subsp. Noncyanogenus. Oral formulation has been used at 0.4mg/kg daily and shown to be effective. There is also a spot application formulation available for dogs which has 2.5% Moxidectin and 10% imidoclopramide. The product is licensed for application every 4 weeks, but more frequent application (weekly to every other week) is often needed when treating Demodex.

Isoxazoline: Are a novel class of parasiticides that are potent inhibitors of GABA-gated chloride channels and glutamate-gated chloride channels.

Afoxolaner: (NexGard®): Afoxolaner was administered to eight dogs at the recommended dose (at least 2.5 mg/kg) on Days 0, 14, 28 and 56. The topicalical combination of imidacloprid/moxidectin was given at the same intervals at the recommended concentration to eight patients as well. Clinical examinations and deep skin scrapings were performed monthly. The percentage reductions of mite counts were 99.2%, 99.9% and 100% on Days 28, 56 and 84, respectively, in the afoxolaner-treated group, compared to 89.8%, 85.2% and 86.6% on Days 28, 56 and 84 in the imidacloprid/moxidectin-treated group. Fluralaner: (Bravecto™) Sixteen dogs, all diagnosed with generalized demodectic mange, were randomly allocated to two equal groups. Bravecto™ chewable tablets were administered once orally at a minimum dose of 25 mg fluralaner/kg body weight to one group of dogs, while the second group was treated topically on three occasions at 28-day intervals with Advocate®. Mites were counted in skin scrapings before treatment and at 28-day intervals over a 12-week study period. A single oral administration of Bravecto™ chewable tablets, mite numbers in skin scrapings were reduced by 99.8% on Day 28 and by 100% on Days 56 and 84. Mite numbers in the dogs treated topically on three occasions at 28-day intervals with Advocate® were reduced by 98.0% on Day 28, by 96.5% on Day 56 and by 94.7% on Day 84.

Sarolaner: (Simparica™) Sixteen dogs with generalized demodicosis were randomly assigned to treatment with either sarolaner orally on Days 0, 30 and 60, or topical imidacloprid (10 mg/kg) plus moxidectin (2.5 mg/kg) solution every 7 days from Day 0 to Day 81. For sarolaner-treated dogs, pretreatment mite counts were reduced by 97.1% at 14 days and 99.8% by 29 days after the first dose. Weekly imidacloprid plus moxidectin resulted in 84.4 and 95.6% reduction at these two time points. Infection control: Generally patients with generalized demodex will also have superficial and/or deep infections. Appropriate systemic antibiotics and antifungals may be necessary for minimum 4 weeks treatment duration. Topical antimicrobial based shampoos should also be implemented to decrease treatment interval.
Glucocorticoids are absolutely contra-indicated, even topically (cutaneous or auricular topicals) even if pruritus is severe.\(^1\)

**Monitoring and prognosis**

It is not sufficient to rely on clinical appearance as the end-point of treatment. Clinically normal dogs may still harbor mites on deep skin scrapings. Microscopic cure, defined as multiple negative skin scrapings, in addition to resolution of clinical signs is needed to determine the therapeutic end-point. In general, it is recommended to scrape the three to five most severely affected areas and any new lesions monthly until all scrapings are negative. It is recommended to continue treatment for 1 month after the second negative monthly set of skin scrapings.\(^1\)

The prognosis for canine demodicosis is good, with the majority of cases achieving long-term remission.\(^8\) However, dogs with an incurable or poorly controlled underlying disease may never be cured and may require long-term therapy with the newer generation flea preventatives this is an easier situation than it was even 3 years ago. The current recommendation is to avoid long-term glucocorticoid therapy in dogs with a history of demodicosis.\(^1\)

**Demodex injai**

In 1997, Dr Andrew Hillier presented at the AAVD/ACVD annual meeting in Nashville a canine skin disease due to a novel Demodex mite. This mite has a much longer body than *Demodex canis* and in 2003 it was coined demodex injai.

**Clinical features**

Can cause a greasy seborrhea mainly on the dorso-lumbar and facial area. Breeds that are predisposed include the West Highland White, Shih tzu, and Scottish terrier. Mites counts tend to be very low so alopecia may not be evident but patients can sometimes be very pruritic. Excessive glucocorticoid therapy (for allergy and pruritus) and hypothyroidism have been reported as underlying causes.\(^3\)

**Diagnosis**

Skin scrapings, trichograms, and a biopsy may be needed in some cases due to scarcity of mites.

**Therapy**

See above

**Demodex cornei**

A short-bodied Demodex mite that may exclusively live in the stratum corneum. It could be a mutant of *D. canis* or a new species. So far the mite is present in cases of simultaneous infestation with *Demodex canis*. Therapy is the same as for *D. canis*.\(^2\)

**References**

4. P B. Variation in size in Demodex canis: from the longest to the shortest forms. *Veterinary Dermatology* 2010;21.
There are more than 2,000 species and subspecies of fleas and dogs and cats can be the transient host for any of these species. However, *Ctenocephalides felis felis*, *Ctenocephalides canis*, *Pulex spp.*, and *Echidnophaga gallinacea* are the ones of medical concern. Overall *Ctenocephalides felis* is the one of most concern in the United States and Worldwide.

**Flea life cycle**

The flea depend on the host for food and protection so they will spend the almost entire adult life of the animal. The female flea will lay her eggs on the host usually while the animal rests or sleeps. These eggs quickly fall off the host and contaminant the environment. Flea eggs usually hatch in 36 hours and some make take up to 10 days. Low temperatures (<0°C) for 24 to 36 hours are lethal to most eggs. The eggs then hatch into larva which move to the dark protected areas like deep into carpets due to their negative phototactics and positive geotropism. Larva are the most sensitive stage in the flea life cycle and only about 25% will survive in the environment due to lack of food, dryness, and extreme temperatures. The third instar of the larva will then form of a cocoon and form a pupa. The pupa is more hardy than all the other stages but will die at the extreme temperatures. The adult flea can remain in the cocoon for as long as 140 days before emerging with proper temperature and humidity. In most households, *C. felis* will take 3 to 4 weeks to complete its life cycle so it is very important for flea infested households to understand that despite starting flea control they may still see fleas that first month or two.

The newly emerged adults require a host for long-term survival. Once on a host, *c felis* initiates feeding within seconds to minutes. Feeding is so rapid that partially digested blood can be defecated in as little as 2-6 minutes after fleas acquire a host. Mating occurs on the host after feeding and the female *c felis* begin egg production within 24-36 hours after their first blood meal. With peak egg production a single female flea can lay between 40-50 eggs per day.

**Flea control**

Flea are essentially everywhere and depending on the area can either be a seasonal or year round issue for pet owners. All animals are considered at risk and those that frequent grooming facilities, doggie day care, dog parks, have or access to wildlife populations are at even higher risk. Effective treatments used on the animal can eventually eliminate environmental fleas, provided that untreated animals do not reinfest the area.

**External environment**

Because flea eggs fall to the ground, the infested pet’s yard can be seeded with fleas by the pet itself or by stray dogs, cats, opossums and raccoons. As discussed earlier flea larva are very sensitive to heat and desiccation and given this fact, adults should not develop on paved areas, on deck surfaces, or in short-cut, un-exposed lawn. The areas that are protected from the direct sun and have some form of shelter are the largest problem areas. This would include area under decks, overgrown brush, and crawl spaces. These are the areas to be concerned about when treating the environment. There are many different products out there that can treat the environment including carbamates or organophosphates that are available in liquid and powders. For those families wishing to have a more natural approach there is a company marketing beneficial nematodes (Fleabusters). This product contains harmless nematodes called *Steinernema carpocapsae*, which kill flea larvae and pupae in the grass and soil. The efficacy of this system is not known.

**Internal environment**

This is usually the hardest part of flea control and prevention and requires thorough cleaning. Vacuuming will help remove eggs, larva and adults but may also cause adults to emerge. Once must remember to empty the bag or canister after each process. Dog beds and carpets should be washed or replaced.

Hiring a profession may be the best recommendation since they have access to many products that we do not and they are familiar with the proper application. However, many products are available to treat the interior of a house. For the client that has significant issues with the use of these products sodium borate would probably be the best recommendation. The borate compounds have rapid ovioidal and larvicidal activity, which is suspected to be through a dehydrating mechanism. The professionally applied product (Rx for Fleas, Inc.) is guaranteed for 1 year, provided carpets are not cleaned, and has a reported efficacy of greater than 99%. For prevention of re-infestation another good option would be the use of insect growth regulators. These products prevent the larva from transitioning to the pupa stage. If these products are applied in the house before fleas are introduced, infestation should be aborted. Methoprene is degraded by sunlight and should be reapplied at least every 30 weeks. Fenoxycarb and pyriproxyfen are sunlight stable and last 6 to 12 months.
**For use on animals**

The following is a table of the most commonly used products that are administered to the patient themselves.\(^3,8,9\)

<table>
<thead>
<tr>
<th>Key Drug</th>
<th>Drug Class</th>
<th>MoA</th>
<th>Key points</th>
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| Spinosad          | Macrocyclic lactone | nAChR agonist               | Contraindicated with high dose ivermectin  
GI upset somewhat common |
| Afoxolaner        | Isoxazoline      | GABA and Glutamate antagonists | Effective against demodex and some tick species  
Highly palatable  
Minimal upset stomach  
Fluralaner = effective for 12 weeks |
| Fluralaner        |                  |                              |                                                                            |
| Sarolaner         |                  |                              |                                                                            |
| Selamectin        | Avermectin       | GABA agonist                 | Fleas continue to feed during first hour.  
Peak efficacy not seen for 36-42 hours  
Effective against sarcoptic mange when applied q 2 weeks x 3 applications |
| Imidacloprid      | Neonicotinoid    | nAChR agonist                | Efficacy within 6-12 hours  
Fleas stop feeding within 3-5 minutes |
| Fipronil          | Phenylprazole    | Insect GABA receptor agonist  | Fleas continue to feed during first hour.  
Toxic to rabbits |
| Dinotefuran       | Neonicatinoid    | nAChR agonist                | Vectra 3D product contains permethrin, do not use in cats |
| Pyriproxyfen      | Insect growth regulator | Juvenile hormone analog       | Mode of action similar to methoprene but not UV sensitive like methoprene |
| Permethrin        | Pyrethroid       | Target voltage gated Na\(^+\) and Ca\(^+\) channels | Kills ticks, biting flies, and mosquitos  
Toxic to cats  
Repellant action  
Most EPA reports of "major pesticide reactions" involved spot-ons containing pyrethrins |
| Nitenpyram        | Neonicatinoid    | nAChR agonist                | Onset within 30 minutes  
100% efficacy within 4-6 hours  
Little residual activity within 48 hours |

**Therapeutic plan for the flea infested pet**

When encountering a patient for the first time with either clinical signs related to flea allergy dermatitis or who is flea infested one must take an accurate history. Determine if that patient frequents areas where fleas could be commonly found (dog parks etc) and also discuss the outdoor environment at home. Question the owner about the possibility of wildlife in the area and if there is a lot shaded areas the patient likes to frequent. Then start prescription flea preventative for all pets in the household. Treat the pruritus and any secondary pyoderma with appropriate medications. Make sure you pick a preventative that fits with the client's preferences (oral vs. topical) any concurrent dz (food allergy, flea allergy, demodex mites), the pet's lifestyle (tick exposure, bathing frequency)

**Advice for clients**

Clients many times will say their flea control doesn't work. These clients will need to be educated about the flea lifecycle and feeding behavior. Advise them that while our prescription flea preventatives are highly effective, they take time to kill fleas and do not prevent all feeding\(^3\). Fleas will need to bite the pet before being killed by the adulticide. It is likely that owners will see live adult
Client who do not believe fleas are the problem
You do not have to see the flea for the flea to cause a reaction. Use a peanut allergy analogy to help them understand: If a person is allergic to peanuts, one peanut may touch their food and they will have a reaction. They do not have to see the peanut. Repeat the history they gave back to them, wait for them to validate that you have the story straight. Then, present the fact that itching and secondary infections in the back half of the body is textbook for flea allergy. Explain that every flea medication, just like every drug of any kind, wanes in efficacy from the time it is given to the time the next dose is due. A pet on good monthly flea control will never have an infestation, because fleas that infest the pet will die before they have enough time to lay eggs. However, for a pet that is sensitive, a few hours of feeding before the flea dies may be enough to cause a reaction. For this reason, hypersensitive pets do better on bi-monthly flea prevention. Compliment the owner on the cleanliness of their home and their pet, advise them you are sure their pet would never have an infestation. However, remind them that with all the good work they are doing and money they are spending on allergy work-up it would be a shame for a future flea bite to confound analysis of the itch level and potentially lead us astray in our diagnosis.

Client who are concerned about safety
Flea preventatives exploit a difference in the nervous system between insects and mammals. It is anatomically/physiologically impossible for these products to kill a mammal the same way they kill fleas. Adverse reactions may occur as they can with any oral or topical product of any kind, but the benefits far outweigh the risks. Point out that monthly or bi-monthly flea prevention is a lot safer than repeated courses of steroids and antibiotics to manage the symptoms. Explain that the majority of reported adverse reactions in pets and people are involve topical products containing pyrethrins or older organophosphates and carbamates. These products may or may not have been used appropriately by the owners (e.g. pyrethrins may have been applied to cats). Explain that some topical products such as imidacloprid are not systemically absorbed. While dermal hypersensitivity reactions can occur, point out that this is also true with any soap or lotion we would use on ourselves. Many dermal reactions are related to carrier ingredients rather than the insecticide. Inform clients of the diseases which can be caused and/or transmitted by fleas. These include iron deficiency anemia, rickettsia typhi, rickettsia felis, bartonella henselae, mycoplasma haemofelis, yersinia pestis, dipylidium caninum. Educate them that these diseases are worse than flea infestation and may require treatment that is less safe than prescription flea preventatives. Use of the broader term "parasites" can sometimes invoke greater willingness of the owner.

What about 'natural' flea preventatives?
These medications are typically ineffective, this is usually easy especially once you educate them and potentially point out evidence of fleas on their animal. Also 'natural' does not always mean safe, garlic can cause anemia and Tea Tree oil (melaleuca) can cause transient hind-limb paresis, hepatotoxicity and is commonly implicated in atopic disease. Other ingredients like peppermint, clove, cinnamon, lemongrass and thyme have been links to neurologic signs.

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Pruritus in puppies may be one of the most difficult diseases that can present in clinical practice. This is due to many factors including: client frustration, the relative lack of safe treatment recommendations to decrease pruritus, constant changing weight of the patient, and the fact that sometimes a clear diagnosis is not available/obtainable.

Client education
Many times owners are already very irritated by the time they make an appointment to be seen. The puppy they just adopted is showing signs of illness and this is not what they expected to be managing at such a young age. In addition, training and housebreaking is a much more challenging experience if the drive to itch is so intense that the puppy cannot focus on the task at hand which can frustrate the client even further. Empathizing with the client’s situation and explaining all the different possibilities/treatments that are available is a must.

History
Just because the patients are so young does not mean that your history should be limited. Knowing if the patient came from the shelter and/or a breeder and if any of the littermates are affected can be a valuable pieces of information that can help narrow down the possible differentials. The client should also be questioned about travel history prior and post adoption. In the particular area that I work I have seen numerous dogs flown in as young adults/puppies from Africa, South American, Mexico, Middle east and all through-out the United States. There is some dermatological disease that will be more common in foreign counties than in the US so this may change your differentials.

In addition, I always ask what type of life style does this patient have? With the growing trend of doggie day care, hiking/walking groups, and dog parks canines have much more of a social lifestyle than ever before. This type of interaction can lead to increased flea exposure and other contagious diseases and can give you information about any other animals affected within the same social group.

The client should always be questioned regarding the main areas of the body that the puppy is itching/licking, how severe the pruritus is, and response to any previous medications that have been given.

If the patient has any history of vomiting, diarrhea, decreased appetite, excess gas, mucous in the stool and how many bowel movements per day can help support the possibility of a food related allergy.

Physical examination
Many of the pruritic puppies I have seen have completely normal physical examinations. However, one must always perform a pinnal-pedal reflex in young patients on their first examination. The reflex is assessed by rubbing the tip of an earflap on to the base of the ear for several seconds. This test is considered positive if the ipsilateral hind leg makes a scratching movement. This test has been shown to have a sensitivity of 81.8% and a specificity of 93.8% of dogs in a study of 588 patients1. Along with the testing of the pinnal-pedal reflex the patient should be flea combed and one should ensure there is no evidence of flea dirt as well.

Diagnostics
1. Superficial skin scrapings should always be performed in all itchy young patients. For the superficial scrapings I will often scrape the pinna and the lateral elbows and hocks and the examine the specimen at 10x. If I am very suspicious of scabies, I will often take numerous scrapings (sometimes more than 5) to see if any mites/eggs/fecal pellets are found.

2. A deep skin scraping is also performed generally at the face, at least one of the paws, and then 1 or 2 other clinically affected areas. The material is placed on separate slides and viewed at 10X to help determine if demodex mites are present and if present is the disease localized or generalized.

3. Surface cytology: Any papules, pustules, crusts or areas of lichenification are sampled and examined at 100x for any signs of bacteria and/or yeast organisms and to evaluate the inflammatory cells present.

4. Fungal culture: Although generally not considered an itchy disease one must consider performing a fungal culture on young puppies that present with lesions consistent with a folliculitis and/or hair loss. I have personally seen extremely itchy dogs that had a primary dermatophyte infection and their pruritus resolved once the dermatophyte was cleared this is mainly seen with Trichophyton and M. persicolor infections in dogs2.
Diseases

Sarcoptic mange
Intensely pruritic, contagious disease caused by Sarcoptes scabiei var. canis. This is considered a zoonotic disease and clients should be warned about the potential for transient lesions. In canine mites prefer the skin of the ears, elbows, abdomen and hocks where there tends to be less hair. Patients obtained recently from the shelter and animals that attend dog parks and doggie day care are at increased risk. Patients tend to be mostly itchy at the ears and face and the entire ventral surface with the dorsum essentially spared.

Clinical lesions
Intense pruritus will be the main clinical sign and these dogs will sometimes have to be manually restrained in the exam room. The initial lesions are generally crusted papules that can progress rapidly to alopecia, erythema, and thick yellow crusts. Some patients do not show any lesions at all beyond pruritus. The level of pruritus is directly related to the number of mites present. The pruritus seen is from a hypersensitivity reaction to the mites themselves.

Diagnosis
Can be very difficult to diagnosis since even after numerous scrapings; anywhere between 20-50% of the time the mites, eggs and/or fecal pellets are not found. Many clinicians rely on response to adequate medications as their main way of diagnosing this disease. As we discussed earlier, a positive pinnal-pedal reflex is helpful for screening for potential cases. Fecal flotation can also sometimes find mites due to excessive grooming behavior is affected animals. An ELISA test exists for the diagnosis of scabies and is between 84- 92% sensitive and 90- 96% specific. However, it can take up to 5 weeks for an animal to start producing adequate antibodies to illicit a positive test.

Treatment
At this time there are many ways to treat this disease and choice of medication will depend on patients age and owner’s preference. If patient lives in a multi-dog household all animals must be treated for resolution. 2-4% lime sulfur dip weekly for a period of 4-6 weeks has proven to be effective and does have additional anti-itch properties for the patient. Ivermectin at weekly administration between 0.2-0.4mg/kg has also been used at every two week intervals for three injections or orally once weekly for 6 weeks. Other effective medications include selemetin, imidacloprid, moxidectin, and milbemycin, and several of the new generation isoxazoline flea medications like sarolaner.

Pelodera strongyloides
Small nematodes that lives in decaying material in the soil where it normally completes its entire life cycle. However, the third stage larvae are capable of penetrating human and mammalian skin and can cause a severely pruritic dermatitis. Generally, these animals live in the Midwestern United States and have access to damp straw bedding.

Clinical lesions
Creates a pruritic dermatitis that will occur on parts of the body in contact with the damp bedding. Commonly lesions will be on the ventral abdomen, chest, perineum, legs, lateral shoulder and thighs and spares the head and back. This nematode can cause alopecia, erythema and crusted lesions in sites infected.

Diagnosis
You can find these nematodes in skin scrapings and are generally between 600-750 µm in length and 30-40 µm in width. The clinician should ensure that the nematodes found are not a type of hookworm. One can also diagnosis this disease via biopsy where the larva are found inside hair follicles.

Treatment
Complete removal of the contaminated bedding is mandatory to reliably treat these animals. All areas must be washed and sprayed with insecticide. To treat the patient one can use Ivermectin but once the environmental is cleaned patients can undergo spontaneously resolution.

Ancylostoma and uncinaria
Larva are present on grasses and soils during the spring and summer season. They are seen patients that live in large capacity dog kennels and in patients that frequent dog parks that are not properly maintained.

Clinical lesions
Red papules on the skin that come in contact with the soil/grasses. As the disease progresses these areas become thickened and alopecic. The paws can become erythematous and may become swollen and pruritus is variable but always present.

Diagnosis
Positive fecal examination for hookworms and compatible clinical signs can usually lead to a diagnosis. However, if the fecal examination is negative one can perform a biopsy to aid in the diagnosis. Histopathology can reveal a perivascular dermatitis and recent larval migrations tracts can sometimes be seen. Larva are not usually present on biopsy samples

Treatment and prevention
The widespread use of heartworm preventives that also have anti-helminth activity has minimized this disease. Treatment should emphasize cleaning of the premises, frequent removal of feces. Once can use pyrantel pamoate tablets or fендbendazole for treatment but keep in mind the treatment cycle needs to be repeated in 2-3 weeks to resolve the infection.
Intestinal parasite hypersensitivity
Certain intestinal parasites (ascarids, Coccidia, hookworms, tapeworms, whipworms) can cause severe pruritus in certain canines. It is thought to be a type I hypersensitivity.2

Clinical signs
Can vary but some patients can have papules, crusts, seborrhea, and sometimes even hives related to this disease.

Diagnosis
Positive fecal and/or response to treatment

Treatment
Proper anti-helminth based off of fecal analysis

Flea allergies
Depending on your location flea allergies can be the most common allergic hypersensitivity dermatitis you see or not present at all.

Clinical lesions
Intense pruritus centered around the dorsal L-S region, tail, flanks and inguinal region. One may also see papules in the umbilical area as a primary clinical sign. This leads to alopecia, crusting, papules and pustules and acute moist dermatitis “hot spots”.

Diagnosis
Presence of adult fleas, flea feces or based off the majority of the clinical signs being in back half of the body.

Treatment
Client education about the flea life cycle. One must ensure that all animals in the house are on proper, consistent flea medications all year round in some areas. Question owner regarding feral cats and other wildlife that could contribute to the flea burden in their environment and necessitate environmental control as well. Sometimes you need to treat the patient as if it is a flea allergy and then see how patients responds to therapy.

Food allergies
It is noted that 33-50% of all dogs diagnosed with a food allergy are less that 1 year of age. There is no breed or sex predilection.7

Clinical lesions
Some patients do not exhibit any clinical lesions beyond pruritus. Some patients have chronic otitis externa as their only clinical sign of a food allergy. Erythema, papules, macules, excoriations, ulcerations, alopecia, lichenification, and hyperpigmentation can occur on the interdigital region, axillae, groin, perineal area and face. Some patients will also have concurrent GI disease which may include: vomiting, diarrhea, increased bowel movements, flatulence, and fecal mucus.

Diagnosis
Perform a strict 6-12-week diet trial with a hypoallergenic diet approved for growth. This includes Royal Canine Venison and Potato, Royal Canine HP, and Purina HA.

Treatment
Continue to feed the hypoallergenic diet or do individual ingredient re-challenge to determine exactly what patient is allergic to.

Atopy
Typically presents between the ages of six months and three years but clinicians have seen it appear as young as three months. However, many times it will take many months to come to this diagnosis since it is one of exclusion and other allergies and parasitic diseases must be explored.

Clinical signs
Pruritus, erythema, papules, excoriations typically occur, in the axillary region, inguinal, interdigital, muzzle, periocular, pinnae and the medial aspects of the thoracic legs. Patients may be year round or seasonal depending on the allergens they are sensitivity.

Diagnosis
Exclusion of other pruritic disorders. According to Favrot in order for a patient to be considered atopic they must satisfy five of the following criteria:

1. Onset of clinical signs younger than 3 years of age
2. Must live mostly indoors
3. Steroid responsive itchiness
4. Itchiness with no skin lesions
5. Front feet are affected
6. Pinna are affected
7. Ear Margins are not affected
8. Back is not affected

Treatment
Antihistamines, topical therapy, fatty acids, cyclosporine, steroids, immunotherapy, monoclonal antibody.

Idiopathic pruritus of puppies
First documented by Danny Scoot from Cornell University where he described ten puppies that started with pruritus between 2-7 months of age. All puppies had no history of a change in diet or environment and all were properly dewormed.
**Clinical lesions**
Pruritus with no clinical lesions

**Diagnosis**
Difficult to diagnosis properly since really a diagnosis by exclusion

**Treatment**
None in the puppies documented by Scott. The pruritus just resolved between 3 ½ to 5 months after onset⁹.

**References**
Oclacitinib (Apoquel®)

Oclacitinib is approved by the FDA for the control of pruritus associated with allergic dermatitis and control of atopic dermatitis in dogs at least 12 months of age. Apoquel® is the first selective Janus kinase (JAK) inhibitor developed for the dog. JAKs play a key role in cytokine signaling and the signal transduction of pro-inflammatory, pro-allergic, and pruritogenic cytokines. Although multiple JAKs exist, Apoquel® preferentially inhibits JAK-1, which is involved with the signaling pathways for IL-2, 4, 6, 13, and 31. Apoquel has the greatest affinity for inhibition of JAKs involved with IL-31 signaling, the cytokine recently demonstrated to play a central role in the development of pruritus. Apoquel® is administered at 0.4-0.6mg/kg twice-daily for the initial two weeks and then decreased to 0.4-0.6mg/kg once-daily for long-term use. The medication is available in 3.6mg, 5.4mg and 16mg tablets and it is recommended to administer in full and ½ tablet increments. The absolute bioavailability of oclacitinib maleate was found to be 89% and has been shown to have a rapid onset of action, significantly decreasing pruritus within the first 24 hours of administration.

This medication is not recommended for use in patients with previous history of neoplasia. Side effects seen with this medication over a study period of 4 months include pyoderma, non-specified dermal lumps, otitis, vomiting, diarrhea, histiocytoma, cystitis, anorexia, lethargy, yeast skin infections, pododermatitis, lipoma, polydipsia, lymphadenopathy, nausea, increased appetite, aggression, and weight loss. On average, dogs gained 4% body weight on oclacitinib maleate during one study period. Generalized demodicosis and viral papillomatus have also been seen post-approval and during initial dosing studies. Co-administration of this medication and cyclosporine and/or corticosteroids is not recommended for long term. However, a study was recently published where oclacitinib was administered with cyclosporine for a period of three weeks in laboratory beagles and there were no adverse events seen. However, long term studies will be needed before it is deemed safe to use these two medications concurrently.

It is off label to use the medication in cats but numerous veterinarians and specialists are experimenting with its role in both feline allergic dermatitis and feline asthma. There has been one published study where 12 suspected atopic cats were treated with a mean dose of 0.47mg/kg twice daily for 14 days and then once daily for another 14 days to judge clinical efficacy. Five of these cases developed immediate post-injection reactions, and the most commonly reported adverse events were vomiting, diarrhea, and lethargy. During the study period many patients were also on corticosteroids, cyclosporine, antifungals, antibiotics, immunotherapy, and/or oclacitinib and there were no complications found with administration of these medications together. Laboratory beagles have received 7 monthly subcutaneous injections at 3.3mg/kg and 10mg/kg and no side effects have been reported in this population.

CADI is available in 10mg, 20mg, 30mg, and 40mg sterile single-use preservative-free 1 ml vials that need to be kept refrigerated.

Canine atopic dermatitis immunotherapeutic®

The USDA conditionally licensed product CADI is a caninized anti-IL-31 monoclonal antibody developed by Zoetis. Monoclonal antibody therapy works by one of two mechanisms; either by binding a soluble molecule and preventing it from interacting with a cell surface receptor or by targeting the cell surface receptor directly. CADI works by “soaking up” circulating IL-31 produced by lymphocytes preventing it from activating cell receptors. It is administered as a subcutaneous injection given no more frequently than every 30 days. CADI therapy offers the several advantages in that it is a very targeted therapy whose adverse event profile appears to be very narrow at this time. It can be administered to any age of dog, and may be given with any concurrent medications. Studies at this time reveal administration of CADI significantly reduced pruritus upon IL-31 challenge for up to a month compared to a placebo. These studies also demonstrated that the efficacy was dose dependent with few anti-drug antibodies (ADA) produced. Finally, the efficacy and safety of CADI in field conditions compared to a placebo was evaluated and revealed that treated dogs had a significant reduction in owner assessed pruritus compared to placebo treated dogs. The clinical improvement can be seen within 1-3 days post injection and was effective in around 80% of patients. During initial studies no dogs developed serious adverse reactions, no dogs developed immediate post-injection reactions, and the most commonly reported adverse events were vomiting, diarrhea, and lethargy. Given this is a caninized monoclonal antibody it will not have a place in the treatment of the feline patient. During the study period many patients were also on corticosteroids, cyclosporine, antifungals, antibiotics, immunotherapy, and/or oclacitinib and there were no complications found with administration of these medications together. Laboratory beagles have received 7 monthly subcutaneous injections at 3.3mg/kg and 10mg/kg and no side effects have been reported in this population.

CADI is available in 10mg, 20mg, 30mg, and 40mg sterile single-use preservative-free 1 ml vials that need to be kept refrigerated.

MicroSilver BG™

Microsilver BG™ is a micronized form of silver that is found in a line of topical products made by Vetbiotek. For a long time, silver has been known for its ability to kill yeast and multi-drug resistant bacterial infections; but it’s main clinical advantage comes from its activity against biofilms. Biofilm organisms have an inherent resistance to antibiotics, disinfectants and germicides. It has been shown that up to 96% of Staphylococcus pseudintermedius isolates from canines have the ability to form biofilms. Unlike planktonic populations, bacterial cells embedded in biofilms exhibit intrinsic resistance to antibiotics due to several specific defense mechanisms conferred by the biofilm environment, including the inactivation of anti-microbial agents by exopolysachharide (EPS),
over expression of stress-responsive genes, oxygen gradients within the biofilm matrix and differentiation of a subpopulation of biofilm cells into resistant dormant cells. The intrinsic resistance of bacterial cells within biofilms to conventional anti-microbials has led to new technology for the treatment of biofilm-associated infections, including the use of silver preparations. Silvers inherit anti-bacterial properties and low toxicity towards cells has made it heavily used in the human field to reduce nosocomial infections.

Vetbiotek has brought this technology into a line of shampoos, sprays, mousses and wipes that contain a silver molecule that is 10 microns in diameter. This size molecule is large enough that it will stay on the surface of the stratum corneum and not penetrate into the layers of the epidermis. In addition, the microsilver BG molecule is porous and adheres very well to the skin and will stay in contact with the skin until mechanically washed off; this allows for sustained anti-microbial effect. The silver molecule is also present in their seborrhea and anti-itch products.

**Osurnia®**

This is an otic gel with 10 mg florfenicol, 10 mg terbinafine, and 1 mg betamethasone acetate per mL that has been manufactured for the treatment of yeast and bacterial canine otitis externa. It is prepared in a 1ml sterile flexible soft tip applicator and one tube should treat one ear. The medication must be refrigerated upon arrival to the hospital and to increase patient comfort it should be warmed up slightly before use. The medication is then instilled in a clean and dry external ear canal and then the base of the canal is massaged to allow for the product to adhere to the lining and then also travel down the ear canal. This entire administration is then repeated in one week. This medication can be used for a suspected otitis externa with mainly cocci and/or yeast found on cytology. However, if the cytology confirms the presence of mainly rods this medication has been shown to not be very effective given the antibacterial medication present. This medication, like claroTM, offers many advantages to the client and veterinarian in that all administrations could theoretically occur in the clinic. The medication would be administered at the initial visit and then the one week follow up. This increases client compliance and allows for the infection to be rechecked in a timely manner. Using a gel topical product also decreases the ability of the medication to exit the ear canal by intense head and ear shaking during the application process.

After application of this product it is recommended to not clean the ear for a total of 45 days. This recommendation stems from an ear swab depletion study that was performed with this medication in normal dogs. Normal dogs were administered the medication on day 0 and then repeated at day 7 and then drug concentrations of florfenicol, terbinafine, and betamethasone were gathered over the period of 45 days. It was concluded that after 45 days therapeutic concentrations of osurnia remained present in normal ears. However, it is suspected that concentrations of these medications in inflamed ears may be decreased due to increased absorption through the disrupted skin barrier and drug degradation secondary to inflammation.

This medication has not been tested in patients with a perforated tympanic membrane, or in animals that are pregnant or lactating. Safety studies have shown that when the 1ml product is re-administered once weekly for a total of five weeks there has been mucosal necrosis and ulceration seen of the lining of the middle ear cavity. At the recommended dosage administration patients have experienced elevations in aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), vomiting and hearing loss.

**Claro™**

This is an otic solution that contains 15.0 mg/mL florfenicol, 13.3 mg/mL terbinafine and 2.0 mg/mL mometasone furoate. It is prepared in a 1ml sterile tip applicator and one tube should treat one ear no matter what the size of the patients ear canal. Just like Osurnia™, this medication is mainly used for the treatment of otitis resulting from Malassezia pachydermatis and Staphylococcus pseudintermedius. If rods are seen on cytology and Pseudomonas spp. are suspect this medication should not be used.

This medication should be applied once in the veterinary hospital and does not need to be refrigerated. This medication has been found to remain active in the ear canal for 30 days so re-administration is not necessary.

This medication has not been tested in patients with a perforated tympanic membrane, or in animals that are pregnant or lactating. Safety studies have shown that when five times the recommended amount of medication is applied every two weeks for three applications patients can experience a dose dependent suppression of the adrenal cortical response to ACTH stimulation and have clear ear exudate and ear wetness. At the recommended dosing administration, no elevations were seen in the liver enzymes but thickening of the tympanic membrane was noted in one patient.

**Sublingual immunotherapy (SLIT)**

Allergen specific immunotherapy in veterinary and human patients for many years relied on the administration of subcutaneous injections. However, recently in both fields there has been evidence that allergens administered directly into the oral cavity can be well tolerated and effective for the treatment of spontaneous atopic dermatitis. In the veterinary field the clinical studies are limited to one in particular out of the University of Wisconsin where 10 dust mite sensitive dogs with Atopica dermatitis underwent a 6-month trial of SLIT. During the initial phase of this study, corticosteroids were needed to provide temporary comfort for patients while the immunotherapy was being introduced. However, after 4-6 months into the immunotherapy 4/10 patients stopped steroids all together and maintained comfortable and another 4/10 had a reduced steroid intake.
References


13. MORRIS DJDaM. Multicentre open trial demonstrates efficacy of sublingual immunotherapy in canine atopic dermatitis. 7th World Congress of Veterinary Dermatology, July 24–28, 2012.
The Pemphigus complex is a group of autoimmune skin diseases that can affect dogs, cats, horses, people, and rarely goats. By far the most common disease of this group is pemphigus foliaceus (PF), with pemphigus vulgaris (PV), pemphigus erythematosus (PE), paraneoplastic pemphigus (PNP) and pemphigus vegetans (PVeg) being considered rare. PF was first seen in the canine species in 1977 by Halliwell and Goldschmidt and the first reported case of Feline PF was in 1982. PF in animals is a pustular and crusting disease rather than a vesicular disease as seen in humans.

Incidence/prevalence
When looking at the Incidence of this disease it is difficult since there are only a few studies and there may be differences based on regions. It is suspected that PF accounts for up to 1/3 of all autoimmune diseases seen in the canine. Canine breeds that genetically predisposed include: Akitas, Chow Chow, Bearded collie, Newfoundland, Schipperke, English bulldogs and Dobermans.

Pathogenesis
PF is associated with acantholysis which occurs when there is a loss of adhesion between keratinocytes. This loss of adhesion results from disruption of desmosome junctions. Cadherins are the structures that bridge the gap between two adjacent keratinocyte cell membranes and are very important adhesion proteins. They consist of desmogleins (Dsg1–3) and desmocollins (Dsc1–3). These are the targets in pemphigus with the major antigen in canine PF appearing to be Dsc1.

Clinical lesions
The main clinical feature are pustules (vary in color) that develop in waves and evolve rapidly into crusts and sometimes erosions. They are present on the face, dorsal muzzle, peri-ocular skin, pinnae and planum nasale. Foot pads can also have extensive hyperkeratosis/crusting and fissuring. Pruritus can be present in 25-50% of cases and generalized erythema can also be seen. Some patients can also present with more systemic signs including anorexia, fever, depression and weight loss.

Diagnosis
Diagnosis requires biopsy. Since dermatophytosis can sometimes mimic clinical signs and histopathology (especially in cats) sometimes special stains are needed. In-house cytological examination of pustule contents will demonstrate acantholytic keratinocytes either individually or in rafts. There may also be extensive amount of non-degenerative neutrophils.

Treatment
The first step in the treatment of any autoimmune disease will be to perform baseline serum chemistry, complete blood count and a urinalysis in order to have a reference once treatments are started. In addition, one must ensure that any recently used medications that could have been implicated as a cause of a drug induced pemphigus reaction are stopped.

Antimicrobials
Many times at initial presentation these patients are actively infected with either bacterial or yeast infections. Ideally I like to base my antibiotic choice based off culture given these patients are going to be immunocompromised for quite some time and so medications may need to be extended. Many times patients will appear to not respond to initial coarse of immunosuppressive medications and it is because their secondary infections have not been properly addressed.

Glucocorticoids
The main stay and the first drug of choice when dealing with pemphigus patients. One of the few times steroids should not be considered as first line therapy is if a patient is concurrently diabetic which does occur on occasion and represents a clinical challenge. Generally, once the diagnosis is established that patient is started at immunosuppressive doses of a glucocorticoid in order to get that patient into remission. The choice of glucocorticoid depends on the patient and the clinician. If that patient already has a tendency to develop PU/PD on steroids or has concurrent heart disease, I will tend to gravitate towards the medications that have less or no mineralocorticoid activity. Also many times it is it uncommon for a patient to have a better clinical response with one steroid over another.

Dose
Prednisolone/Methylprednisolone/Prednisone are used between 2.2-6 mg/kg/day but this author rarely goes over 3mg/kg/day. The immunosuppressive doses of triamcinolone is considered to be 0.2-0.6mg/kg/day and dexamethasone at 0.2-0.4mg/kg/day. When used as sole therapy it is ideal to get to 1mg/kg/EOD for prednisolone/methylprednisolone; triamcinolone to 0.1-0.2 every 2-3 days.
and dexamethasone at 0.05-0.1mg/kg every 2-3 days at maintenance. It is not uncommon for feline patients to require significantly higher dose of oral steroids to achieve remission, immunosuppressive dosages range from 2.2 to 8.8 mg/kg/day for prednisolone. Two studies have shown that 33-38% of canine patients can respond with the use of glucocorticoids alone5,6. I personally feel that felines many times will respond to steroids alone and this percentage may be higher is this species (around 65% or higher). 7

Monitoring
In the ideal situation the immunosuppressive doses are continued for 10-14 days and then gradually tapered to maintenance level over a period of 6-12 weeks. This requires recheck examinations every 2 weeks for the first 2-3 months in the best case scenario. At each recheck I recommend serum chemistry/complete blood counts to be performed and a urinalysis and culture done at 2-3 months into therapy. The urine culture is to ensure that a patient has not developed a urinary tract infection that can commonly occur with long term steroid use8. The serum chemistry and CBC is to ensure there are no signs of hyperglycemia or severe hepatic dysfunction. At each recheck cytology is performed to evaluate the presence or absence of acantholytic cells and bacteria/yeast. If a large number of acantholytic cells are found generally I will continue the immunosuppressive doses for 2 more weeks and then recheck the patient two weeks later.

If unable to taper the dose of steroids due to lack of improvement of clinical signs or worsening of clinical disease upon taper one must either add in additional immunosuppressive medications or change the formulation of steroids.

Adverse events
Complications with steroids are vast given their general mechanism of action. Given the haptic changes that occur with glucocorticoids and many of the other immunosuppressive drugs it may be beneficial to also start hepatic support medications (SAM-E) at the time of diagnosis. Many patients will also suffer PU/PD/PP/panting, muscle atrophy, poor dull scaly hair coats, wt gain, behavior changes, increased risk for infections (bladder infections, demodicosis, dermatophytosis), comedones, atrophic skin, calcinosis cutis, atrophic scars, milia GI ulcerations, diarrhea and and decreased thyroid hormone, adrenal gland suppression, Diabetes mellitus.

<table>
<thead>
<tr>
<th>Medication</th>
<th>Glucocorticoid activity</th>
<th>Mineralocorticoid activity</th>
<th>½ life (hours)</th>
<th>Equivalent dose mg</th>
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<tr>
<td>Cortisol</td>
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<td>0.8</td>
<td>12-36</td>
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<td>4</td>
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<td>Triamcinolone</td>
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<td>24-48</td>
<td>0.5-1</td>
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<tr>
<td>Dexamethasone</td>
<td>25-30</td>
<td>0</td>
<td>36-72</td>
<td>.75</td>
</tr>
</tbody>
</table>

Azathioprine (Imuran®)
Azathioprine (AZA) is considered an antimetabolite agent whose main mechanism of action involves mimicking natural molecules utilized in DNA and/or RNA replication. As a group they are most active in the 'S' phase of the cell cycle (when DNA is being synthesized). Immuran serves as fraudulent purine bases during DNA and RNA synthesis, resulting in dysfunctional nucleic acid strands in proliferating cells. The T-lymphocytes, T-cell dependent antibody synthesis, and cell mediated immunity) are inhibited, with very little direct effect on B-cells. AZA is metabolized into 6-mercaptopurine (6-MP) by the liver and then 6 MP is metabolized by three different enzymes. Two of these enzymes are xanthine oxidase and TPMT (thiopurine methyltranferase) which produce inactive compounds. In general, patients with low TPMT activity, have a greater incidence of bone marrow suppression, but also greater immunuran efficacy. As a species cats have low activity of TPMT, hence this medication is not recommended for these patients. Dogs have variable amounts of TMPT activity levels similar to that seen in humans, which may explain why some canine patients respond better and/or develop more myelotoxicity than others. Giant Schnauzers are known to have very low TMPT whereas Alaskan Malamutes have very high TMPT activity1,9.

Dose
When used in combination with oral steroids the dose varies from 1 to 2.5mg/kg Q24 to 48 hours) or 50 mg/m². Supplied as 50mg tablets and there is a very slow onset of action. Typically, beneficial response in seen within 6-12 weeks

Monitoring
CBC/chem after 2 to 3 weeks, then again in 1 month, then quarterly. Some clinicians monitor more frequently (Q2 weeks for the first 8 week). Watch the lymphocytes and platelet counts for decreasing trends, which may indicate when to decrease the dose.
Adverse events
Include bone marrow suppression and hepatopathy (contraindicated in cats). There has been an association with pancreatitis when used concurrently with prednisolone. Diarrhea is the most common side effect along with opportunistic infections. There has been rare hepatotoxicity that does respond to drug withdrawal.6

Chlorambucil
This medication is considering to be an alkylating agent that affects disease by physiochemical interaction with pre-formed and developing DNA and RNA. These medications as a group exert their effect (cross-links, strand breaks) on all stages of the cell cycle (i.e., do not require an actively proliferating population). As a group they are cytotoxic to all lymphocyte populations (including B-cells) and very effectively suppress immunoglobulin secretion. The new formulation requires refrigeration. It is considered to be the drug of choice for cats. It can also be used in conjunction with steroids1.

Dose
0.1-0.2 mg/kg q 24-48 hrs ONLY 2 mg tablets

Monitoring
CBC at 2 weeks and CBC chem. 4 weeks, then CBC at 8 weeks; Delayed action 2-4 weeks.

Adverse events
Myelosuppression, anorexia, vomiting, diarrhea

Cyclosporine
Cyclosporine is considered a calcineurin inhibitor that leads to the blocking of IL-2 transcription which results in impaired T helper and T cytotoxic lymphocytes (remember the principal action of IL-2 is T cell proliferation). Reduces production of IL-2, IL-3, IL-4, G-CSF and TNF-alpha as well as reducing the clonal proliferation of the cell. This leads to decreased clonal proliferation of B lymphocytes and indirectly also effects other cells, such as granulocytes, macrophages, NK cells, eosinophils and mast cells. Although initial studies have shown that food affects absorption when clinical efficacy studies were done with atopic patients no change was seen10.

Dose
5-10mg/kg Po SID

Monitoring
CBC/chem/UA after first 1-2 months and then every 6 months there after

Adverse Events
GI disturbances (vomiting, diarrhea, decreased appetite), gingival hyperplasia, papillomatous growths, increased susceptibility to opportunistic infections, hypertrichosis

Tacrolimus
The topical calcineurin inhibitor preparation is used mostly in veterinary medicine for localized lesions of PF and PE. It is the first macrolide immunosuppressant discover. It was found in a soil fungus, although it is produced by a type of bacteria, Streptomyces tsukubaensis.

Dose
Generally used twice daily

Adverse events
Minimal

Tetracycline/doxycycline and Niacinamide
The mechanism of action of this pair of medications is not exactly known. Tetracycline/doxycycline have been known to have anti-inflammatory properties affecting complement activation, antibody production, chemotaxis, prostaglandin synthesis, and suppressing lymphocyte blastogenesis. Niacinamide blocks antigen and immunoglobulin E induced histamine release, inhibits mast cell degranulation and phosphodiesterase protease release. This pair does have a delayed onset of action and generally beneficial response is seen after 6-12 weeks1.

Dose
Doxycycline 5mg-10 mg/kg Po BID and Niacinamide is dosed at 250mg PO TID less then 10kgs; 500mg MG/KG greater then 10kg

Monitoring
Serum chem and CBC every 6 months

Adverse events
Vomiting, diarrhea are the most common Doxycycline can also cause esophageal stricture in cats and dogs
Mycophenolate Mofetil
Derived from the fungus *Penicillium stoloniferum* and is metabolized in the liver to mycophenolic acid (MP). This compound is a potent and reversible uncompetitive inhibitor of inosine-5’-monophosphate dehydrogenase (IMPDH). This inhibition leads to the synthesis of guanine being blocked and prevents DNA and RNA synthesis. Cytotoxic to cells that rely on *de novo* purine synthesis (such as T and B lymphocytes)\(^\text{11}\).

**Dose**
22-39 mg/kg daily divided into q8 hour dosing; available in 250 and 500 mg. tablets

**Monitoring**
No clear protocol set, Complete blood count and serum chemistry q2-4 weeks for the first 2-3 months then every 6 months

**Adverse events**
Myelosuppression, Gastrointestinal upset: vomiting, diarrhea, anorexia and Increased risk of infections (urinary tract, skin, etc.) The concurrent use of mycophenolate and azathioprine is not recommend based off their similar mode of action\(^\text{12}\).

Leflunomide
Is a pyrimidine synthesis inhibitor that works by inhibiting the mitochondrial enzyme dihydroorotate dehydrogenase. This enzyme is involved in the *de novo* synthesis of the pyrimidine ribonucleotide uridine monophosphate (rUMP). Also Antagonizes action of IL-3, IL-4 and TNF-\(\alpha\)

**Dose**
2-4 mg/kg per day\(^\text{13}\)

**Monitoring**
No set protocol, CBC serum chem UA after 2 weeks and then every 3 weeks for 3 months. In one paper leflunomide dosage was decreased by 25% every 4 weeks for the first 4 months in order to achieve a trough level of approximately 20 micrograms/mL (leflunomide trough levels were based on studies of the canine renal transplantation model); then the dosage was decreased every 8 weeks until discontinuation after 10 months of therapy\(^\text{14}\).

**Adverse events**
Hepatotoxicity, myelosuppression (leukopenia, anemia and thrombocytopenia), recurrent infections, pneumonia and cutaneous drug eruptions

**Human IVIG**
Sterile purified IgG that is pooled from human plasma, that may also contain traces of IgA and IgM\(^\text{15}\). The exact mechanism of action is not known so there are several theories on how this medication works. Blocks the Fc receptors on antibiotics and eliminates circulating immune complexes, suppressing anti-idiotypic autoantibodies, inhibits complement mediation damage, and blocks the cell surface death receptor Fas have all been discussed\(^\text{16,17}\).

**Dose**
0.5-2.2 g/kg given slowly IV over 6-12 hours\(^\text{14,18}\).

**Monitoring**
Has a similar set up and monitoring as a blood transfusion to ensure no signs of anaphylaxis.

**Adverse events**
Allergic reaction, In human’s hypertension, nausea, tachycardia can occur. There has only been limited studies on the re-administration of IVIG to canine patients and this should be done with caution.

References
Managing Chronic Otitis:  
The Keys to Getting Started on the Right Foot  
James Noxon, DVM, DACVIM 
Iowa State University 
Ames, IA

The 2013 and 2014 data from Veterinary Pet Insurance lists ear infections as the number TWO reason that dogs went to veterinarians (after “skin allergies”) for veterinary care. In addition, Banfield data indicates that otitis was the second most common diagnosis made in affiliated hospitals in 2011. This data has been similar every year since 2005, with otitis being the #1 or #2 most common presenting complaint in dogs, year after year.

There are many, many important concepts about otitis that can literally make the difference in practice. Knowledge can, in fact, change your entire attitude about dealing with ear disease.

Information needed to manage otitis (getting started on the right foot)

#1 –Understanding structure and function

The ear consists of the pinna, the external ear canal, the middle ear, and the inner ear. There are major variations in the anatomy from breed to breed, especially with respect to the length and diameter of the external ear canal. These variations will affect predilection for disease, diagnosis, and treatment. For example, it can be very difficult to fully examine the external canal of the ear of an Irish setter, for it can be very long!

The external ear canal consists of skin overlying the auricular and annular cartilages. It has a vertical component and a horizontal component. The vertical component is formed by the auricular cartilage. The annular cartilage is the rolled, tube-like cartilage that extends from the auricular cartilage at the base of the vertical ear canal to the temporal bone. The auricular cartilage overlaps the annular cartilage with a fibrous band, which allows for flexibility in movement.

Anatomically, the vertical canal is more open and larger in volume than the horizontal ear canal. There is a depression or pocket at point where the auricular and annular cartilages overlap (i.e., the “opening” of the horizontal canal). The entrance to the horizontal canal is often elevated and requires manipulation of the otoscope in order to pass it into the horizontal canal. There is actually a fold of skin (overlying cartilage) on the dorsal aspect of the canal that must be bypassed in order to slip the otoscope into the horizontal ear canal. Mechanical irritation (e.g., during otoscopic examination) of this fold will cause startle the patient and result in poor patient compliance with otoscopy.

The skin lining the ear canal has sebaceous glands and apocrine (i.e., ceruminous) glands throughout the length. Sebaceous glands are found in the superficial part of the dermis with the apocrine glands located deeper. These apocrine glands can open directly onto the surface of the skin or in the hair follicle. Hair follicles are found throughout the length of the ear canal in most breeds, but there is breed variation as to the type of follicles and their density.

Ear wax is the mixture of apocrine (cerumen) gland secretions, sebaceous secretions, and epithelial cells. There is a natural movement of sebum outwardly in the normal ear, facilitating natural cleaning and removal of sebum.

The lipid portion of ear wax is derived from sebaceous glands and contains various waxes and fatty acids, many of which are bacteriostatic and fungistatic. The lipid portion of cerumen is responsible for controlling microorganisms. The apocrine secretions (from “ceruminous glands”) produce a water-based secretion that contains phospholipids and IgA, which also contributes to the defense of the ear. Epithelial cells contribute to the texture and consistency of the wax. Increased epithelial cell production in the ear will produce a thicker, pasty ear wax.

The tympanic membrane is at the end of the external ear canal. On otoscopic examination, the tympanum appears as a vertically aligned structure, but it actually is sloped at approximately a 30° angle, with the top towards the viewer. The tympanic membrane consists of two parts. The pars tensa is the tightly stretched, clear to opaque whitish section of the tympanic membrane. Embedded within the pars tensa is the handle (aka manubrium) of the malleus, the largest ossicle of the middle ear. The malleus is curved, with the concave section pointing rostrally. The pars flaccida is the dorsal-rostral component of the tympanic membrane. It appears pink and there are often small capillaries visible on the surface of the membrane. The pars flaccida often bulges out and may be seem moving with respiration, in a movement that resembles the bulging throat of a bullfrog!

The tympanum in the cat is much more transparent, and thus is often thought to be absent. The malleus is straighter than in the dog and the pars flaccida is generally not visible. Cats also have a bony septum in their middle ear that runs rostral to caudal….and this septum tends to obstruct the view of the middle ear during otoscopic examination and creates an obstruction for materials inside the middle ear. The two-chambered nature of the middle ear in the cat impairs our ability to clean and perform various procedures in the middle ear in cats.
#2 – Understanding the pathophysiology of otitis
By now, everyone should know about the concepts on the pathophysiology of otitis as introduced by Dr. John August. He recommended dividing the pathogenic factors of otitis as follows:

1. **predisposing factors**: these are conditions that “set the ear up” for inflammation. They include conformational changes, behavior, and previous treatments.
2. **primary factors**: these are those conditions that initiate inflammation in the ear. They include allergic diseases, foreign bodies, ectoparasites, autoimmune and other inflammatory skin disorders, and trauma.
3. **perpetuating factors**: these factors keep the inflammatory process active and often make it significantly worse.

Perpetuating factors include bacterial infections, yeast infections, hyperplastic changes, and otitis media.

Simply put, there is a “WHAT” and a “WHY” when dealing with ear disease. Clinicians must address both or the problem will fail to resolve or recur.

#3 – Understanding pathologic changes in the ear
Once the otitis has begun, certain pathologic changes occur that initiate a cascade of events that make the ear more hospitable for microorganisms and reduce the lumen size of the ear canal. Inflammatory changes are accompanied by pain, and progressive disease leads to loss of hearing. It has been determined that the pathologic changes in the ear do reduce acuity of hearing, and that some of that hearing loss is reversible, as the pathologic changes are reversed.

With inflammation comes edema and infiltration of inflammatory cells. Secretion of various growth factors will result in epidermal hyperplasia and hyperkeratosis, resulting in microfissures on the surface of the skin and increased deposition of cornified keratinocytes in the lumen of the ear. As inflammation progresses, there is fibroplasia (i.e., fibrosis) of the dermis and subcutis. Chronic inflammation of the cartilage will result in ossification of these structures.

Within the dermis, it has been shown that apocrine glands increase in size in otitis externa. The intense inflammation around apocrine glands, combined with epidermal hyperplasia (papillary proliferation) results in occlusion of ductal openings on the skin and hair follicles and may predispose the gland to rupture. When the apocrine glands rupture, there is infiltration of lymphocytes, neutrophils, mast cells, and macrophages into surrounding tissue. It would appear that the disruption of these glands significantly contributes to the inflammation, pain, and fibrosis. Interestingly, sebaceous glands remain the same size, even in chronic otitis externa, though there is a qualitative change in sebum production. The net result is decreased lipid content of cerumen in ears with otitis externa. Since lipid secretions of the skin have barrier and antimicrobial functions, there is speculation that this change further contributes to secondary infections in otitis externa.

Finally, biopsy of the ear canal in chronic otitis externa will reveal folliculitis and furunculosis. With furunculosis there is release of keratinized materials into the dermis, and the net result is a foreign body-type reaction. Furunculosis is common in ceruminous otitis externa associated with familial seborrhea of the American cocker spaniel.

#4 – Knowing the goal: Restoring defense mechanisms of the ear
The ear does have an effective defense system. First there is an inherent self-cleansing mechanism. Debris, including desquamated keratinocytes and wax, naturally moves from deep into the canal to the opening of the canal. Anything that blocks this movement, such as a foreign body or scar tissue, will predispose to infections. Second, ear wax is an amazing antimicrobial material, along with its other functions. Third, the hair in the canal and at the opening of the canal does help to restrict access into the canal form the outside. (On the other side of things, it can also cause problems by holding material in the canal that should be extruded.) Fourth, the very conformation of the ear (canal and pinnae) serves to restrict access into the canal. Again, this has both good and bad aspects.

**Diagnostic approach to otitis**
Collection of a thorough dermatologic history is crucial to evaluate the patient for the primary factor (i.e., underlying cause, or the “why”). When it comes to managing the perpetuating factors (e.g., current infections), it is helpful to know what medications have been used in the past. This includes amounts, frequency, and duration of each treatment. The physical examination includes inspection and palpation of the entire ear canal. The mouth should be opened wide to evaluation for bullae pain, one possible indicator of otitis media.

Otoscopy should be performed on all cases, and repeated at each recheck of the patient. Both ears should be examined, even if the client believes the problem is unilateral (one ear is often worse than the other). Handheld otoscopes are very useful and there are different styles that have different levels of magnification. Several commercial video otoscopes are now affordable and they provide much better visualization down the canal.

Cytology is the key diagnostic procedure in otology. Cytology should be done on both ears and repeated at every recheck examination...because things to change in the canal. Samples are usually collected by passing a cotton-tipped applicator gently into the ear canal to the beginning of the horizontal canal. If resistance is encountered while passing the swab, it should not be advanced further! The swab is gently rotated then withdrawn and used to make “roll preps” on a clean glass microscope slide. The slide is then stained (with the stain of choice in your practice) and examined under the microscope.
A couple of tips for cytology:
1. Use a clean glass slide (wipe the slide with a gauze to make it is clean)
2. Use firm pressure to roll the swab (this will increase the adhesion of material)
3. Dip or place slides into jars of stain or fixative VERY gently. Do not move the slide up and down after the initial placement…you may gently sway the slide in the jar to distribute stain, if needed.
4. Rinse the slide immediately after the thiazine (blue) stain (when using Diff-Quik stain), BUT do not let the rinse water hit the sample directly.
5. Air dry or use a blow dryer with the heat coil turned off to rapidly dry the slide. Do not overheat (or the sample may be ruined!)

**Tips:**
1. Make two ear prep slides on each case and examine before cleaning the ears, in the event that a culture is indicated.
2. Stain one slide with Diff-Quik and save the other for a Gram stain, to be done if rod-shaped organisms are present on the initial slide.

A normal ear may contain low numbers of bacteria (usually cocci) and yeast. However, the absolute number (e.g., number of organisms per field of view) is not important, since we all make slides differently. The cytologic findings are correlated with the clinical findings and a decision made to treat is based on all data.

Bacterial culture is indicated when: 1) the cytology shows a uniform population of rod-shaped bacteria (probably *Pseudomonas* spp.), 2) the infection has failed to respond to “standard-of-care” therapy, 3) when you have a known resistant organism (generally based on previous culture results). Imaging of the ear can also be very helpful. Radiography may help identify bony changes in the bullae that might reflect otitis media. Computerized tomography provides much better detail and is the author’s imaging of choice, due to relative low cost and high degree of detail provided. Magnetic resonance imaging is also very helpful, however the cost is significantly higher than CT scanning.

**Client education and communication**
Client education starts on day 1….the first time you see a client/pet with otitis. Client education should include:

1. Some basic information about the pathophysiology of otitis (really important),
2. Information about your plan for their pet (i.e., identify secondary issues, treat those, then look for the underlying cause),
3. Diagnostic findings on their pet at the first visit,
4. Why the recheck exam is important and what will happen at that appointment (repeating diagnostics, switching from treatment to a maintenance plan, additions testing for primary factors, etc.),
5. The long-term picture of otitis

It is very helpful to use analogies when speaking to clients. The author uses the analogy of archeology: dermatology and otology problems are like archeology. That is, clinicians must keep on digging until they find the underlying civilization (i.e., primary factor). If the clinician does not address the underlying problem, then the perpetuating (i.e., secondary) factors will fail to respond to treatment or will recur. So, it makes sense to explain this to clients at the beginning, understanding that it is unlikely that a client will give consent to spend a lot of time and money searching for a cause of first-time otitis. But….you plant the seed by giving them the education about the pathogenesis of otitis. That way, when the problem recurs (notice I didn’t say “if”), they just may remember that you tried to explain this to them.

Ear models are great for explaining otitis. Several companies have provided these to veterinarians in the past, so ask your reps about one! It is especially helpful to explain the “L” shaped ear canal and why we have to medicate the way we do.

Last, video otoscopes also help the clients be more involved. Clients LOVE seeing their pet’s ears before and after cleaning or before and after treatment. Letting clients see the ears will definitely help to convince them that cleaning and medications are warranted.

The clinical effects of client education include: better client compliance, more cooperative clients, and better success. Everybody wins.
Managing Chronic Otitis: Tips to Maximize the Value of Your Treatment
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Topical therapy is the most commonly used treatment for otitis externa. Selection of active ingredients and treatment protocols for veterinary otic preparations tend to have been driven by the pharmaceutical industry based on guidelines to facilitate approval by various governmental and regulatory agencies. The term best practices implies a method or technique set forth by an authority that has consistently shown superior results to those achieved with other means, and that are used as a benchmark. Ideally, these serve as clinical treatment guidelines and are integral to evidence-based practice of medicine.

Important concepts prior to treatment
1. A reminder of the structure and function (anatomy and physiology) of the ear is crucial. Specifically, the shape of the ear canal provides some challenges for topical therapy.
2. A reminder of the pathophysiology of otitis is also important: we have predisposing factors, primary causes (or underlying factors), and perpetuating factors (secondary causes) in otitis. There is a difference between short-term management (and success) and long-term management (and success).
3. Client education is paramount. It is essential that clients understand the two points listed above AND the goals and expectations of treatment.
4. We need to have collected the right information to allow us (the veterinarian) to “choose wisely” the best topical medication for each possible combinations of problems that may be factors in each patient. We need to know “what” is going on in the ears.

Cleaning the ears: “Preparing to succeed”
Cleaning the ears is an important and crucial component of effective management of chronic ear disease in dogs and cats. Cleaning the ears is important for the following reasons:
1. Cleaning removes debris, such as wax, that may cause irritation of the ear canal.
2. Cleaning removes debris that will block movement of medication into the horizontal canal and the self-cleansing mechanism.
3. Cleaning removes debris (e.g., pus, biofilm) that can interfere with the activity of topical (and systemic) otic medications.
4. Cleaning may help to lower the burden of bacteria in the ear.

The cleaner you get the canal, the better the chances are that your topical medication will work. Keep in mind that the efficacy of some topical medications, such as polymyxin B sulfates and some aminoglycosides, is dramatically reduced in the presence of pus! So, it is to your patient’s and client’s advantage to start with an ear cleaning.

It is your choice, as the veterinarian, on which type of ear cleaning you select. For mild cases, it may suffice to use a basic technique of filling the canal with cleanser, massaging the canal, then removing excess cleanser and debris with a cotton ball…repeated until otoscopic exam confirms that most of the debris has, in fact, been removed. However, I recommend a deep ear cleaning (or ear flush) with the patient under general anesthesia if you are unable to definitively see the ear drum...or at least enough to confirm that it is intact.

A good rule of thumb: Deep ear cleaning or flushing (under general anesthesia) is indicated if you cannot definitively visualize the tympanic membrane prior to treatment.

Best practices for topical management of otitis externa
Overall, the long-term success of medical management of otitis externa depends on the following considerations:
• Obstructions, such as hair and wax, should be removed to allow distribution of medications deep into the ear canal
• Topical medications should be selected based on consideration of the active ingredients and data supporting the use of that agent for secondary infections or perpetuating factors
• The integrity of the tympanic membrane should be considered when selecting topical medications
• The formulation of the medication should allow the product to distribute deep into the canal and provide adequate coverage of the surface area of the ear canal
• Topical medications must be administered using proper technique to ensure delivery of medicine throughout the full extent of the external canal. This often includes “positional instillation” of medicine, which means positioning the animal (on its side, for example) to allow deeper movement of the agent into the ear canal.
• Adequate volumes of topical medications must be administered to reach the deeper aspects (proximal) of the ear canal...
Treatment of infections should be continued until the infection is cleared. Generally, this requires a treatment period of 3-4 weeks.

Ear medications are most often in the form of an ointment (emulsions of lipid in water) or as a solution (aqueous or other carriers). Emulsions containing lipids will enhance penetration of the active ingredient into the skin of the ear; however, most of these ointment formulations are so viscous, that they fail to penetrate down deeply into the ear canal. They are especially ineffective in the presence of a heavy growth of hair in the canal. Less viscous medications are more likely to allow medication to distribute deeper into the canal, especially when there is significant hair in the ear canal or when the canal is hyperplastic. There is little data on the overall effect of viscosity on “spreadability” or distribution of topical medications over the skin that lines the ear canal.

In all cases when topical therapy is used, the owners MUST be educated about application of medications. This should include having the owner instill medication, IN THE PRESENCE of the veterinarian or technician. Owners should be taught to massage ears for 15-30 seconds after instilling medications…and to use proper amounts of medications. Once-daily treatment is generally sufficient for most cases of otitis, though severe infections may benefit from twice daily treatment. Treatment should be continued until there is no clinical or cytologic evidence of active disease. The minimum recommended treatment time (with topical therapy) is 30 days.

**Dose (volume) recommendations**

- Small dogs (<15 kg) 0.4-0.5 ml
- Medium dogs (15 – 20 kg) 0.7-0.8 ml
- Large dogs (> 20 kg) 1.0 ml

The volume of medication applied into the ear during treatment appears to be critical. Dosing syringes work well to accurately measure volumes of otic medications. Failure to apply sufficient quantities to penetrate to these areas seems to be a major cause of treatment failure. Volumes recommended in this paper to achieve adequate penetration down the canal are based on existing literature and pilot studies performed by the author. You may increase movement of otic medicine deeper into the canal by using “positional installation” and by massaging the ear for 15-30 seconds after instillation.

Keep in mind that higher volumes of otic medication may increase the likelihood of absorption of otic medications, especially glucocorticoids. It is important to understand that there may be systemic side effects as more potent glucocorticoids are used.

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**Table 1. Commercial veterinary otic preparations**

<table>
<thead>
<tr>
<th>Product</th>
<th>Manufacturer</th>
<th>Drops/ml*</th>
<th>Label dosing</th>
<th>Maximum tx time (days)‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aurizon®**</td>
<td>Vétoquinol</td>
<td>?</td>
<td>10 drops once daily</td>
<td>7-14</td>
</tr>
</tbody>
</table>
| Baytril® Otic | Bayer Animal Health              | 30        | <35 lbs: 5-10 drops twice daily  
>35 lbs: 10-15 drops twice daily | 14          |
| easOtic®      | Virbac Animal Health             | NA        | 1 pump daily                                     | 5                       |
| Mometamax®    | Intervet/Schering Plough Animal Health† | 40     | <30 lbs: 4 drops once daily  
>30 lbs: 8 drops once daily | 7                       |
| Otomax®       | Intervet/Schering Plough Animal Health† | 37     | <30 lbs: 4 drops twice daily  
>30 lbs: 8 drops twice daily | 7                       |
| Posatex™      | Intervet/Schering Plough Animal Health† | 39     | <30 lbs: 4 drops twice daily  
>30 lbs: 8 drops twice daily | 7                       |
| Surolan®      | Vetoquinol                       | 45        | 5 drops twice daily                              | 7                       |
| Tresaderm®    | Merial                           | 40        | 5-15 drops twice daily                           | 7                       |

* Determined manually by author. Estimates ± 2 drops/ml.  ** Not available in USA  † Merck Animal Health USA  ‡ Label instructions
Table 2. Newer extended-activity otic preparations

<table>
<thead>
<tr>
<th>Product</th>
<th>Manufacturer</th>
<th>Active ingredients</th>
<th>Labeled dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>KetoCort®</td>
<td>TrilogicPharma</td>
<td>Ketoconazole, hydrocortisone</td>
<td>Clean ears and dry. Instill adequate amount and repeat as necessary.</td>
</tr>
<tr>
<td>Osurnia®</td>
<td>Elanco</td>
<td>Florfenicol, terbenifine, betamethasone</td>
<td>Clean ears and dry. Instill one tube, massage 1-2 minutes Repeat in one week.</td>
</tr>
<tr>
<td>Claro™</td>
<td>Bayer</td>
<td>Florfenicol, terbenifine, mometasone</td>
<td>Clean ears and dry. Instill one tube.</td>
</tr>
</tbody>
</table>

The integrity of the tympanic membrane is critical in determining the best treatment options for a patient with otitis. The possibility of ototoxicosis is greatly enhanced if the medication is instilled directly into the middle ear. The best practice is to avoid topical therapy, if the tympanic membrane is torn or absent. However, there are some clinical indications, based entirely on anecdotal evidence, that vinegar: water (1:2) and enrofloxacin (parenteral formulation) are fairly safe.

Topical therapy is considered sufficient to manage most cases of otitis externa, if the principles of therapy discussed early are followed. In general systemic therapy is indicated when:

- The infections are recurrent and severe
- There are concurrent infections elsewhere, such as the skin, that would respond to the therapy
- When the owners are incapable of treating topically (e.g., arthritis, elderly owner)
- When the patient is entirely uncooperative
- When there is severe hyperplastic changes in the canal that preclude the ability of topical medications to distribute deeper into the ear canal

Systemic antibacterial therapy is indicated when inflammatory cells are seen on cytology, when a pure infection of a gram-negative bacteria is present, in recurring bacterial infections, when ulcers are present in the external ear canal, or when systemic signs accompany the otitis. Systemic therapy may or may not be indicated when otitis media is present. The antibiotic selection depends upon the organism isolated. Drugs should be dosed at the high end of the recommended range…always go up on pill size, never skimp on systemic drug doses! Drugs should be administered for a minimum of three weeks, then the patient re-examined and evaluated with cytology and/or culture.

Lastly, you should consider the goal of your therapy. Practically speaking, it is the improvement of the clinical condition of otitis: reduced swelling, erythema, pain, and restoration of function. However, for longer-term success in managing ear disease, it is important to CLEAR the infections. This generally requires longer treatment periods and higher doses.

Future considerations

There are several areas where additional studies could be greatly beneficial. For example, there is little data on contact times required, in vitro and in vivo, for effective killing/clearance of various bacteria and yeast. In addition, it would be helpful to better understand the distribution of topical medications, both immediately after instillation and after 12 and 24 hours following administration. We have little data about the duration of inhibitory concentrations of antimicrobials over time after topical application.

Selected references and recommended readings

Most difficult problems in otology can be resolved if best practices are followed. However, there are several conditions which seem to be considered more difficult and frustrating, depending on the situation. They include:

1. management of allergic ears
2. management of recurring yeast infections
3. control of specific bacterial infections, such as Pseudomonas infections
4. management of ceruminous otitis (seborrheic ear disease)
5. control of severe hyperplastic changes
6. treatment of otitis media

In this session, we’ll address Pseudomonas infections and hyperplastic changes and talk about strategies to prevent recurrence.

**Pseudomonas infections**

*Pseudomonas aeruginosa* is a hydrophilic, beta-lactamase producing, gram negative bacterium that is commonly associated with otitis externa and media in the dog. Many strains of Pseudomonas (up to 40% of isolates) are known to be potent producers of biofilm, a matrix that coats the surface of the tissue and “protects” the organisms from antimicrobial activity. Biofilm-producing bacteria had significantly higher MICs for common pathogens isolated in canine otitis. Biofilm is known to physically block penetration of compounds, such as antibiotics and antiseptics, and create concentration gradients of these agents that reduce efficacy and may lead to antimicrobial resistance. Remove or reduction of biofilm is a key component of managing patients with *Pseudomonas* infections.

Proper cleansing of the ear, systemic therapy with an appropriate antimicrobial agent, and management of the primary factor are also part of managing *Pseudomonas* infections of the ear! Aggressive and thorough cleaning of the ear is crucial to remove the biofilm, and thus allow the treatment of choice to be effective. There are limited studies reported at this time demonstrating the effectiveness of various cleansers or otic medications in the presence of biofilm.

**Antibiotic therapy of Pseudomonas otitis**

Antibiotic treatment of recurring *Pseudomonas* infections (or other resistant gram negative infections) should be based on cytology and culture results. Appropriate antibiotic stewardship is strongly encouraged when making the clinical decision to use an antibiotic. Some topical treatment options include:

- Gentamicin (Otomax/Mometamax/Posatex-ScheringPlough-Merial) is an effective antimicrobial for many *Pseudomonas* infections. Unfortunately, the labeling on this product in the USA minimizes its effectiveness (dose volume, maximum treatment period).
- Topical tobramycin (available as generic ophthalmic drops)
- Polymyxin B (Surolan®-Vetoquinol). Many *Pseudomonas* isolates are sensitive to this antibiotic; however, polymyxin B sulfates are not active in the presence of suppurative inflammation. Synergy of polymyxin B and miconazole against *E. coli* and *Pseudomonas* isolates (but not *Proteus* isolates) from dogs with otitis externa has been demonstrated in vitro.
- Topical fluoroquinolone antibiotics (Baytril® Otic-Bayer, Aurizon®-Vetoquinol) are often effective for *Pseudomonas* infections.
- Other antibiotics from which topical otic medications may be formulated include amikacin, (1-2%), ceftazidime, imipenem and meropenem. The latter two drugs have restricted use in most hospitals (for life-threatening infections) to prevent resistance. Their use should be as a last resort and therapy should follow all principles of antimicrobial use to avoid contributing to bacterial resistance to these drugs.

**Additional topical therapy includes agents that may not have direct antibacterial activity, but that are used to support other antimicrobial products.**

- Tromethamine (Tris) edetate disodium dehydrate (EDTA), known more commonly as Tris-EDTA solution, is commonly used as adjunctive therapy for bacterial otitis. Several commercial products (e.g., TrizEDTA™ Aqueous Flush-Dechra, and T8 Keto® Flush-DVM) contain this solution. There is good evidence that the Triz-EDTA is highly effective for *Pseudomonas* when used concurrently with an appropriate antimicrobial (some fluoroquinolones or aminoglycosides), silver sulfadiazine, or chlorhexidine. Tris-EDTA alone is bacteriostatic in vitro, but is not bactericidal. Triz-EDTA has been shown in vitro to reduce the MICs for neomycin and gentamicin (but not enrofloxacin or polymyxin B) for biofilm-embedded bacteria. Additional studies show that Triz-EDTA enhances antibiotic efficacy of marbofloxacin and gentamicin against multidrug-resistant *Pseudomonas in vitro*. Clinically, these products are often
administered into the infected ear 15-30 minutes prior to an antibiotic; however, data suggests they may be administered concurrently. Tris-EDTA appears to be safe when instilled into the middle ear, but there is no evidence to support that clinical observation.

**Antiseptic therapy for Pseudomonas otitis**

Antiseptics are attractive alternatives to the use of antibiotics for control of bacterial skin and ear diseases. However, most studies involving their use for otitis are in vitro studies looking at MIC values. It is likely that the ultimate effectiveness of antiseptics will involve selection of the proper concentrations (to exceed MICs or minimum bactericidal concentrations) and also consider the contact time.

- **Silver sulfadiazine (SSD),** (Baytril® Otic-Bayer) or as a 1:9 dilution of the 1% silver sulfadiazine cream). SSD has been shown to be effective in vitro against *Pseudomonas.* Based on two studies, it appears that the MICs for *Pseudomonas* have increased in the past 30 years (from 7.5 ug/ml to 23.4 ug/ml); however, they are still low enough to easily treat ears topically with available products. Note: The addition of Tris-EDTA to SSD has been shown to decrease the MIC even lower.
- Acetylcysteine has been shown to have anti-*Pseudomonas* activity *in vitro,* with the MIC values for six isolates calculated to be 10.3 mg/ml. Clinical trials (in vivo studies) have not been reported.
- **Aluminum acetate:** Burow’s solution has been demonstrated to have activity against *Pseudomonas* in vitro and in some animal models of *Pseudomonas*-associated otitis media. Clinical studies are ongoing in dogs with *Pseudomonas* otitis. Interestingly, aluminum acetate is a component of many commercial products in the USA that are used for managing otitis.
- **Chlorhexidine** and other antiseptics / biocides have efficacy against *Pseudomonas* and have been combined in various ear cleansers with TrisEDTA for enhanced activity. There have been conflicting reports of the ototoxicity of chlorhexidine.

One very important key to successful treatment of *Pseudomonas* otitis, is the concurrent use of glucocorticoids, preferably systemically. Glucocorticoids reduce the pain that is associated with this condition- and thus will make application of topical medications easier and more effective. In addition, glucocorticoids reduce the inflammation, which also reduces the discomfort and swelling that accompanies this condition. The recommended dose of prednisone in dogs is: 1-2 mg/kg. PO once daily for 5-7 days, then every other day for 5 doses, then half of the dose every other day for 5 additional doses. Naturally, any allergy testing should be done prior to initiation of glucocorticoid therapy.

Patients with *Pseudomonas* infections tend to get other secondary infections, most often yeast infections, immediately after the *Pseudomonas* is cleared. Therefore, we often initiate prophylactic anti-yeast therapy as part of our maintenance therapy as soon as the bacterial component of the otitis is controlled.

Lastly, control of the underlying cause (or primary factor) is very important to prevent recurrence, and in some cases, may be necessary to get the problem under control.

**Hyperplastic changes**

Swollen ear canals are a major threat to the survival of the ear! Hyperplastic changes of the external ear canal include epidermal hyperplasia (e.g. lichenification), fibrosis, edema, glandular hyperplasia, and inflammation, especially folliculitis and furunculosis. Hyperplastic changes are perpetuating factors. They promote a microclimate favoring microbial growth (increased temperature and humidity) and physically prevent distribution of topical medications deep in the ear. In addition, they reduce the ability of veterinarians to adequately examine or clean the ear. Closure of the ear canal may be due to 1) edema and inflammation or 2) fibrous changes, including calcification of the canal. It is difficult clinically determine whether the hyperplastic changes are reversible (due to edema and inflammation) or permanent (fibrosis). To make that determination, the following is recommended:

1. Potent topical glucocorticoids are administered to reduce inflammation. The glucocorticoid is should be in a vehicle that allows and facilitates deep movement into the canal (e.g., Synotic®-Ft. Dodge containing DMSO and flucinolone). An adequate volume should be instilled 1-2 times daily to reach the deeper areas of the ear canal. If flucinolone is not available, mometasone (Claro-Bayer) may be infused into the ear if the canal is patent enough to allow infusion.
2. Since distribution of a topical drug may not reach deep into the canal, concurrent administrations of a systemic glucocorticoid is recommended to reduce edema and allow for a proper examination or to allow medication to gain access to the ear. Assuming there are no medical conditions that may preclude their use, prednisone or prednisolone may be administered orally (1-2 mg/kg daily for 5-7 days, then q 48 hour for 5 doses, then half the dose every other day for 5 additional doses).

Patients are re-examined in 3-4 weeks. If the ear canal has opened (indicating the changes were primarily edema and inflammation), efforts should be directed towards identifying and managing the primary and perpetuating factors. If the canal does
not significantly open with topical and systemic administration of glucocorticoids, triamcinolone may be injected (0.05 ml/site) in a 
spiral manner in the most severe areas, in an attempt to reduce edema and inflammation. Alternatively, long-term administration of 
cy closporine may benefit some of these ears. It is unclear whether any effect of cyclosporine is due to anti-inflammatory actions or 
control of underlying / primary factors of otitis. However, if the canal has become calcified, a total ear canal ablation is recommended.

Preventing recurrence
An important strategy for ALL ear infections is to aggressively treat and clear the infection using best practices as described 
previously. However, the next step in management of otitis is to recheck and re-evaluate the patient to determine if the infection has 
been reduced/suppressed, or cleared. There is a big difference.

A recheck examination should be performed in each patient at an interval when the veterinarian feels the infection should be 
cleared. At that time, the examination should include 1) history since treatment was initiated, 2) physical examination, 3) otic 
examination, 4) cytology of the ear, and in cases with recurring infection or gram negative bacterial infections, 5) repeated culture. If 
cytology and culture (when performed) are negative, we proceed to the next step.

Maintenance therapy is always begun on patients with chronic otitis as soon as the recheck examination suggests the infection is 
cleared. The goals of maintenance therapy include 1) keeping the ears clean, 2) control or decreasing pain and pruritus, 3) control or 
decreasing the number of infectious agents, and 40 promoting “normalization” of the ear. The principles include the intermittent use of 
cleansers and non-antibiotic therapeutics (e.g., antiseptics, non-antibiotic agents) to prevent recurrence of the infection OR to reduce 
the severity and frequency of secondary infections. Options maintenance therapy include use of cleansers proven to have 
antimicrobial activities (e.g. EpiOtic Advanced-Virbac; Malacet Otic-Dechra). Ingredients with antiseptic properties include 
salicylic acid, boric acid, parachlorometaxylenol, and chlorhexidine. Other ear products and “flushes” that contain benzoyl alcohol 
(T8Keto-Bayer), aluminum acetate (Burrow’s solution, CortAstrin-VedCo) also have antibacterial and/or antifungal activity. Some otic 
products (MalAcetic Ultra-Dechra) contain azoles for managing yeast infections. We do not fully comprehend the ability of 
Malassezia pachydermatis to develop resistance to intermittent exposure to these agents, so caution should be the rule when looking 
for long-term maintenance of yeast infections.

Ultimate control
In the final analysis, identification and control of the underlying or primary factor of the otitis will lead to the best long-term 
management. However, some clients are unwilling or unable to pursue those factors due to financial consideration, medical 
philosophy, distrust of the motives of the veterinarian, or a lack of will. Maintenance therapy is ALWAYS combined with client 
education to achieve the best results for each patient.

Summary
Most difficult cases of otitis externa are not due to resistant organisms or strange circumstances. Most difficult cases develop when 
there has been a breakdown in communication or failure to strictly adhere to the best practices of ear management. Proper and 
thorough cleaning of the ears is a necessity in the management of chronic otitis externa. Infectious components of otitis can be 
managed and controlled, though in some cases, repeated trials may be required to identify the best treatment. Long term control of 
otitis externa or media requires identification and management of the primary factors.

Selected references and recommended readings
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In an era where most restaurants have stronger Wi-Fi than coffee, it’s hard to truly comprehend, let alone stay on top of, the sheer ubiquity of modern technology. Not only is technology everywhere, but it’s also speeding up each day. If we want our practices to keep up, we’ve got to be on the move as well. The digital realm continues to integrate into most major aspects of our daily lives, ballooning into a practically palpable world where we can book flights and check sports scores, literally without lifting a finger. Meanwhile, major Social Media outlets like Twitter and Facebook keep on expanding their user bases consistently each quarter. The transition toward a world that's entirely online has already begun, and folks, we’re a long way from dial-up.

Need more proof? In the year 2013, Apple sold more iPhones than most countries have people. Yes, feel free to read that twice for impact. And if you follow the trends, every year at the Apple Worldwide Developers Conference (WWDC), they promise there will be no slowing down…

Now, close your eyes and imagine even more people, with even faster technology. Open your eyes and voilà! It’s already happening. This expeditious trend isn’t just an illusion. As long as the rate of technological paradigm shift continues to increase exponentially, things will only get faster. This is theorized as the Law of Accelerating Returns and continues to make technological gains quicker and smarter than ever before.

What does this mean for a practice like yours? Well, whether you’re well known in your neighborhood or a newly opened veterinary practice, your services, of course, come first. But your engagement with your current and prospective clients, general accessibility and online presence now all come in at a close-ranking second. This pushes business owners to adapt or lose relevance in the all but prolonged digital age.

Still, not everyone is sold on the rise of Social Media. You are not alone. In a recent study only 17% of veterinarians utilize technology, such as a smartphone, as compared to the general population (21%). Those who are not embracing technology may chalk it up as a temporary fad; others simply won’t budge when it comes to their tried and true marketing methods. While there are traditional methods of advertising and marketing that maintain their pertinence over time, (word-of-mouth marketing, for example, will never die) a growing amount of these dated-mediums are simply falling by the wayside.

On the other side of the equation, we can assure you that Social Media isn’t something that's going to fade away anytime soon. While the Social Media-Medium itself that’s used to communicate might wax and wane over time, it’s the interconnectivity itself that remains not only relevant, but increasingly imperative to utilize, and ultimately master.

With most adults now online, the time to build your company’s digital infrastructure is now. Social Media allows you to quickly engage with your audience in an absolutely seamless manner. If it’s a new skillset you’re boasting, a sale you’re offering, or even a simple news announcement, your Facebook Page will allow you to capture your client’s attention in a heartbeat. Deliver your content directly into their newsfeed, while measuring clicks, traction and response rates in real-time.

For those that have already mastered Facebook, Twitter is another fantastically savvy way to engage with your clients, friends and fans. It’s about leveraging the platform that suits you best, and presenting your audience with what matters most.

At the end of the day, you want to live and work happily, and ultimately grow your practice into the most rewarding and viable endeavor possible. With such a teeming array of Social Media websites in existence, it may just be a matter of figuring out which ones are right for you and your practice, and then getting down to it
A Look at Old and New Insulin Formations
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When choosing an insulin formulation, factors like duration of action, potency, species-source, convenience and price should be considered. Another important factor that had not received much consideration is day-to-day variability in insulin action. While onset of action, duration of action and overall time-action profile are important when attempting to mimic physiological needs, minimizing day-to-day variability is critical for minimizing the risk of hypoglycemia while still maintaining acceptable glycemic control throughout the day. With most formulations, a peak insulin action occurs after injection and that peak should coincide with post-prandial glucose absorption. Post-peak levels should meet the basal insulin requirements (fasting requirements) but should not exceed them. However, achieving complete congruity between exogenous insulin action and endogenous insulin requirements during these 2 different phases is challenging at best, even if assuming complete consistency between meals, post-meal activity levels, stress, etc. Thus, to avoid hypoglycemia with a practical twice-daily insulin therapy and relatively infrequent monitoring, there has to be a tradeoff. In veterinary medicine this tradeoff is typically loose glycemic control with blood glucose concentrations ranging between 100 – 300 mg/dL and some residual glycosuria and incomplete reduction of clinical signs. In people, because of the critical importance of tight glycemic control, the tradeoff is often a combination of more intensive blood glucose monitoring with a more intensive treatment protocol that combines ultra-short acting formulations with long-acting ones.

Insulin formulations: General principles
Suspensions versus solutions
Insulin molecules tend to form dimers and hexamers (especially in the presence of zinc) but only the monomers are biologically active. Protamine and zinc were traditionally used to prolong the duration of action of insulin by promoting formation of insulin crystals (Zinc in Lente formulations, Protamine in NPH formulations, and both Protamine and zinc in PZI formulations). These traditional intermediate-acting formulations (Lente, NPH, PZI) are injected as suspension: They need to be re-suspended by gentle rolling (or thorough mixing in the case of Vetsulin) prior to drawing up a dose from the vial. This contributes to imprecision in dosing of these formulations. The process of de-precipitation in the SQ depot is relatively erratic and unpredictable for Lente, NPH and PZI which also contributes to their relatively variable time-action profile when compared to novel insulin formulations. In contrast to traditional formulations, synthetic insulin analogs (Glargine, detemir, degludec) are supplied as solutions (and not suspensions) and have a more predictable time-action profile, a result of their more precise dosing and more predictable absorption from the SQ depot.

Insulin glargine
Recombinant human insulin analog (Asparagine at A21 is replaced by glycine and 2 arginines are added at B31 and B32). This synthetic molecule does not tend to hexamerize at pH of 4.0 but strongly crystalizes at pH = 7.2. Considered in people long-acting and “peakless”.

Insulin detemir
Recombinant human insulin analog (B30 replaced by myristic acid – a 14-carbon fatty acid). Considered in people long-acting but not “peakless” and still as effective as insulin glargine as basal insulin (fewer side effects because more predictable). The fatty acid bound to insulin Levemir prevents formation of regular hexamers and allows hydrophobic interactions between detemir molecules and with albumin. These interactions allow more predictable absorption from the SQ depot and buffering of detemir concentrations by albumin which leads to minimal variability in time-action profile from one day to the next and a better safety profile (minimal frequency of hypoglycemic events). Insulin detemir has other advantages that are likely related to its tendency to bind to albumin. After adjusting for its high potency and comparing at equivalent units of action in terms of glucose lowering effects, insulin detemir decreases endogenous glucose output and NEFA more than other insulin formulations. This means that SQ administration of insulin detemir resembles the physiological effect of insulin more than other insulin formulations do. Insulin secreted from the pancreas and into the portal system reaches the liver in high concentrations. It is then degraded by the liver and eventually reaches peripheral target tissues in much lower concentrations (about 3 fold difference in dogs) so that overall endogenous insulin has more effect on shutting down endogenous glucose production than on peripheral glucose uptake. By mimicking this differential effect insulin detemir causes less weight gain while maintaining the same degree of glycemic control.

Insulin degludec
Recombinant human insulin analog in which B30 is replaced by a fatty acid (hexadecandioic acid) that is bound to B29 via a glutamic spacer. These changes allow for multi-hexamers to form in subcutaneous tissues and a long acting and completely peakless time-action profile
Species source
Amino acid sequence in traditional formulations depends on the animal source (porcine, bovine, etc.). To date, the basic molecule of synthetic analogs is human insulin and that molecule is engineered (with varying modifications of sequence) to achieve desired PK/PD profiles. The importance of the amino acid sequence is questionable: While anti-insulin antibodies can potentially form, they do not seem to affect glycemic control.

Insulin formulations: Practical considerations
Most available insulin formulations that are considered intermediate-long acting are most consistently used as twice daily injections in dogs and cats. On average, some might be longer-acting than others but inter-patient variability (and perhaps intra-patient variability as well) precludes a safe prediction for the average patient. The average time-action profile of a formulation should be considered but may not be relevant for a specific patient on a specific day. Variability in time-action profiles limits our ability to balance safety (avoiding hypoglycemia) and efficacy (normalizing blood glucose). Day-to-day variability is crucial to consider when monitoring blood/tissue glucose concentration.6, 7 When performing blood glucose curves, the result of a single curve may be helpful when hypoglycemia is detected but otherwise, repeated curves from multiple days might be required to appreciate long-term patterns of insulin action in the individual animal.8

Factors contributing to apparent variability in time-action profile
1. Injection site (vasculature, temperature)
2. Injection technique
3. Dose inaccuracies (dependent on syringe type, insulin formulation, and operator proficiency)
4. Insulin absorption (dependent on the above but also inherent to each insulin formulation)
5. External factors: Meal composition and size, physical and emotional stress and activity level

Minimizing day-to-day-variability of insulin action
Consider use of synthetic formulations over traditional formulations. Synthetic analogs have more predictable time-action profiles because: 1. They are supplied as solutions (and not suspensions): increased accuracy in dosing. 2. They have more predictable absorption from SQ depot. 3. Detemir and degludec are buffered by albumin.

Synthetic analogs are U100 while traditional formulations that are used in veterinary medicine are supplied as U40. U40 syringes are generally more convenient and more accurate than U100 but at low doses they are not as precise.9 Injection pens increase precision and accuracy regardless of the insulin formulation being used.10

What do we know about the pharmacology of different insulin formulations in veterinary medicine?
The time-action profiles of different insulin formulations have been tested in cats and dogs mostly using glucose serial glucose monitoring in client-owned patients. On a population level, these are useful in estimating dose and frequency of administration in the naïve patient (Table 1). However, the inter-subject variability and day-to-day intra-subject variability have rarely been reported in veterinary medicine. In a study comparing inter-subject variability of lente, NPH and PZI in dogs it was concluded that “individual idiosyncracies in the absorption of SQ administered insulin of any form may be as important in determining the individual’s glucose response as the type of insulin that is used.” This was particularly true for PZI and NPH in that study.11 Interestingly, NPH showed lesser inter-subject variability than insulin glargine in a later study that used the is-glycemic clamp technique.12 The same group later reported on the inter-subject variability of insulin detemir in dogs using the same technique and although the value of this the comparison is very limited, it seems that insulin detemir had lesser inter-subject variability than NPH or glargine.13 In a clinical study on PZI in dogs, the day-to-day variability partially reported by comparing the time to minimum blood glucose concentrations between glucose curves of individual dogs.14 This study reported an unpredictable time to minimum blood glucose in dogs on PZI, with lowest blood glucose detected 54% of the time at either time zero or at 10h post insulin injection (in 10h long glucose curves).

Another problem with understanding time-action profiles in veterinary medicine is that most studies report means and do not report patient specific profiles which could result in under or overestimating some parameters. For example, an insulin formulation with a peak of action that vary greatly between individuals, when averaged would appear as “peakless”.11, 12, 15 This might become important when choosing insulin formulations in cats where a “peakless” insulin formulation might be advantageous in patients that tend to “graze” throughout the day (in contrast to consistent twice daily meals). Insulin glargine has been reported as “peakless” in people but in cats, using the iso-glycemic clamp method, the time-action profiles of insulin glargine varies between “peakless” to having a very pronounced peak.15
Table 1: Insulin formulations in cats and dogs: species source and type, syringe type, and typical dosing frequency.

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Species source/Type</th>
<th>Syringe</th>
<th>Dog</th>
<th>Cat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Humulin N</td>
<td>Human/NPH</td>
<td>U-100</td>
<td>q 8-12h</td>
<td>q 8h</td>
</tr>
<tr>
<td>Vetsulin</td>
<td>Porcine/Lente</td>
<td>U-40</td>
<td>q 12-24h</td>
<td>q 8-12h</td>
</tr>
<tr>
<td>Prozinc</td>
<td>Human/PZI</td>
<td>U-40</td>
<td>q 12-24h</td>
<td>q 12-24h</td>
</tr>
<tr>
<td>Lantus</td>
<td>Human recombinant/Glargine</td>
<td>U-100</td>
<td>q 12h</td>
<td>q 12-24h</td>
</tr>
<tr>
<td>Levemir</td>
<td>Human recombinant/Detemir</td>
<td>U-100</td>
<td>q 12-24h</td>
<td>q 12-24h</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Starting dose</td>
<td></td>
</tr>
</tbody>
</table>

References
I. Highlights
   A. Hypoadrenocorticism (HA, Addison’s disease): Two different syndromes:
      1. Glucocorticoid deficiency (“atypical” Addison’s disease, G-HA)
      2. Glucocorticoid + mineralocorticoid deficiency (“classic” Addison’s disease, GM-HA)
   B. HA is an uncommon condition (1:200 to 1:2,000 dogs). The prevalence of Atypical Addison’s is unknown but it is underdiagnosed
   C. The diagnosis is straightforward if you maintain a high index of suspicion. Especially for Atypical Addison’s however, the overlap of clinical signs with other diseases makes it really easy to miss!!!
   D. Key to diagnosis is maintaining a high index of suspicion (especially in dogs that do not have the classic electrolyte abnormalities)
   E. A lack of a stress leukogram is neither sensitive nor specific for the diagnosis of HA
   F. In theory, corticosteroid-induced ALP should not be increased in HA dogs. In practice, it is increased in about 20% of cases, making it an unreliable screening test.
   G. Screening for HA is added by measuring basal cortisol: Basal cortisol should be low in HA and if it is not low, HA can be ruled out. A low cortisol, however, cannot replace the ACTH stimulation test in confirmation of HA.
   H. Consider screening for HA in: chronic GI disease (including protein losing enteropathy), megaesophagus, non-specific illness, recurrent “renal failure” and many others…

II. Classification of hypoadrenocorticism
   A. Primary hypoadrenocorticism (ACTH concentration high) (> 95% of cases)
      1. Mineralocorticoid- and glucocorticoid-dependent primary hypoadrenocorticism (Addison’s disease)
         a. Idiopathic destruction and collapse of adrenal cortex (Suspected to be immune-mediated based on lymphoplasmacytic infiltration of the adrenal glands): Most common form of primary hypoadrenocorticism
         b. Other causes of destruction: Drug-related (mitotane, trilostane), bilateral adrenalectomy, infectious (fungal) and neoplastic (lymphoma).
      2. “Atypical” or glucocorticoid-dependent primary hypoadrenocorticism
         a. Normal sodium and potassium on presentation
         b. Not presented for “Addisonian crisis”: Signs related to hyponatremia (hypovolemic shock) and hyperkalemia (bradycardia) are absent
         c. Usually more chronic with history of GI signs
         d. Most affected dogs remain only glucocorticoid-dependent for a prolonged period of time (months to years)
         e. Some affected dogs progress to glucocorticoid and mineralocorticoid dependent disease (i.e. classical primary hypoadrenocorticism) in the months after diagnosis of “atypical” Addison’s and consequently long-term monitoring including serum electrolytes is necessary.
         f. Difficult to diagnose because clinical signs are vague and serum electrolyte concentrations normal (see later)
         g. In old literature it was reported as the less common form of primary hypoadrenocorticism (5 to 25% of cases of primary hypoadrenocorticism) however it is recognized that this form is underdiagnosed (can easily be misdiagnosed as Inflammatory Bowel Disease or just be missed all together)
   B. Secondary hypoadrenocorticism (ACTH concentration low) (< 5% of cases)
      1. Glucocorticoid deficiency only (serum electrolyte concentrations normal)
      2. Caused by congenital or acquired hypopituitarism (neoplasia, trauma, immune-mediated diseases, etc.)
      3. Because this form of the disease is rare and because the clinical signs, laboratory findings, and treatment are identical to “atypical” Addison’s, the 2 will mostly be discussed together.

III. Presentation
   A. Duration of illness: few days to several months
   B. Some have a history of a chronic disorder that follows a waxing and waning course (25-40% of cases)
   C. Others present in crisis with acute collapse (10% of cases)
   D. History of previous treatment with and favorable response to fluids and/or glucocorticoids (25-35% of cases) should increase your index of suspicion for hypoadrenocorticism
   E. Chronic recurrent non-specific signs (lethargy, decreased appetite, weight loss, dehydration). GI signs are common.
      Vomiting is less common in G-HA than in GM-HA. Polyuria, polydipsia and bradycardic shock are a consequence of electrolyte disturbances (seen in GM-HA)

IV. Laboratory findings
   A. Hemogram
      1. Anemia (25-35% of cases)
         a. Anemia of chronic disease and/or gastrointestinal blood loss
         b. Anemia may be masked by dehydration
2. Leukogram: Absolute eosinophilia (10-20% of cases) and absolute lymphocytosis (10-15% of cases) or a lack of a stress leukogram (i.e. normal lymphocyte and neutrophil counts) are all helpful clues BUT: A significant number of non-Addisonian sick dogs present without a stress-leukogram and a significant number of Addisonian dogs present WITH a stress leukogram. Thus, lack of a stress leukogram is neither sensitive or specific for diagnosis of HA.

B. Biochemistry
1. Mineralocorticoid deficiency leads to sodium wasting, potassium retention, and acidosis. These lead to severe volume depletion, dehydration and eventually decreased GFR (leading to increased urea, creatinine, phosphorus). Although chloride is lost in the urine with sodium, its loss is less significant than sodium’s, resulting in a hyperchloremic (relative) metabolic acidosis with normal anion gap. Decreased GFR and renal failure may lead to accumulation of organic acids and a shift towards high anion gap metabolic acidosis.
2. Hypercalcemia (total calcium) is due to hyperproteinemia caused by dehydration and hemoconcentration as well as decreased renal excretion of calcium (30% of cases). Increased ionized calcium is observed in about 20% of HA dog and the degree of elevation is inversely proportional to the decrease in blood pH.
3. Increased liver enzyme activity (aspartate aminotransferase [AST], alanine aminotransferase [ALT], alkaline phosphatase [ALP]) occurs in 20-30% of cases possibly due to decreased hepatic perfusion. Corticosteroid-induced ALP is increased in 20% (although only mildly and probably representing the cross reactivity of the assay with total ALP).
4. Although glucocorticoids increase hepatic gluconeogenesis and decrease glucose uptake and utilization in peripheral tissues, hypoglycemia is only observed in 15-20% of dogs with classical hypoadrenocorticism.
5. Hypochlolesterolemia is more common in those with glucocorticoid-dependent hypoadrenocorticism.
6. Serum proteins may be decreased (GI loss) or normal-increased due to dehydration. Hypoalbuminemia is found in about 45% of cases and is more common in those with glucocorticoid-dependent hypoadrenocorticism.

C. Urinalysis
1. In general, high urine specific gravity (USG) indicates normal renal concentrating capacity and typically rules out primary renal disease. However, low urine specific gravity often is found in dogs with hypoadrenocorticism (60% of cases have USG < 1.030) making primary renal disease a confusing differential diagnosis.
2. The low USG in dogs with hypoadrenocorticism results from renal medullary washout of solute (i.e., urea and NaCl are the primary solutes of the renal medullary interstitium responsible for maintaining normal renal concentrating capacity). The low USG in combination with pre-renal azotemia (secondary to volume depletion) may cause confusion with acute renal failure.
3. Renal function returns to normal after re-hydration and re-establishment of normal renal medullary solute concentrations.

D. Abdominal ultrasound examination:
1. In cases of idiopathic or immune-mediated destruction of the adrenal gland or secondary to ACTH deficiency, the adrenal glands are expected to be smaller than normal.
2. In rare cases of hypoadrenocorticism caused by infiltrative disease (lymphosarcoma, fungal infections) the adrenal gland will appear normal or enlarged on ultrasound but show decreased response to ACTH.
3. Reference intervals for adrenal gland size vary based on dog size and body weight but these are not routinely used. When evaluating the sensitivity and specificity of adrenal gland caudal pole thickness for diagnosis of HA, groups of varying body sizes were used and therefore the degree of overlap between diseased and controls was probably greater than it would have been if dogs were grouped into body weight categories.
   a. Left: <2.8 mm is 90% sensitive, 100% specific
   b. Right <3.05 mm is 82% sensitive and 90% specific
   c. Sensitivity and specificity might be improved if using body-size appropriate reference intervals
5. Although the accuracy of ultrasound in diagnosis of HA is limited, it may be helpful in cases of chronic GI disease to increase suspicion and justify further testing for HA.

E. Screening test: Some advocate use of resting plasma cortisol concentration to “rule out” a diagnosis of hypoadrenocorticism
1. Lennon et al. JAVMA 2007:
   a. A resting cortisol concentration of ≤ 1 μg/dL had a sensitivity of 100% and a specificity of 98%
   b. A resting cortisol concentration of ≤ 2 μg/dL had a sensitivity of 100% and a specificity of 78%
2. These 1 and 2 μg/dL cutoffs will vary to some degree between laboratories.
3. Remember “sPin” and “sNout” – “a specific test, if positive, rules a disease in” whereas “a sensitive test, if negative, rules a disease out”
   a. It is not unusual for sick dogs (with non-adrenal illness) to have an occasional low resting cortisol (about 22% of the time…) but these dogs will respond to ACTH adequately (post ACTH cortisol > 5 ug/dL).
   b. You may want to use a resting plasma cortisol concentration to “rule out” hypoadrenocorticism but you should NOT use this test to try and “rule in” hypoadrenocorticism – you must rely on the ACTH stimulation test for that purpose (i.e. the “gold” standard to “rule in” hypoadrenocorticism)
   c. Assuming a disease prevalence of 0.5% (1:200), the positive predictive value (i.e., percentage of those patients with a positive test result that indeed have the disease) of a resting plasma cortisol is approximately 2% whereas its negative predictive value (i.e. the percentage of those with a negative test result that do not have the disease) is 100%. Thus, you can see that this test must only be used to “rule out” hypoadrenocorticism.

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d. Consider measurement of basal cortisol in the following instances (and many more – these are just a few examples):
   i. Acute Renal Failure, especially if sodium is low
   ii. Increased liver enzymes with "synthetic liver failure" (decreased albumin, cholesterol and glucose) but normal bilirubin, especially if the lymphocytes are not low and before performing biopsies.
   iii. Megaesophagus: Addison’s is not a common cause but one of the only treatable ones
   iv. Worked up cases suspected of IBD/lymphangiectasia or chronic GI signs with hypoglycemia, (especially if the lymphocytes are not low), before performing biopsies

F. Confirmatory test: ACTH stimulation is the gold standard test and must be used to “rule in” a diagnosis of hypoadrenocorticism
   1. ACTH stimulation test (the “gold standard” for diagnosis)
      a. Method
         i. Plasma sample for resting cortisol concentration is not needed for the interpretation and should definitely not be repeated if a basal cortisol was already measured before and was low.
         ii. Administer 5 μg/kg (or more) synthetic ACTH (Cortrosyn) intravenously
         iii. After 1 hour, collect plasma sample for post-ACTH cortisol concentration
         iv. Normal reference range in dogs (varies with laboratory)
            (A) Resting cortisol concentration: 1.0-5.0 μg/dL
            (B) 1-hour post-ACTH cortisol concentration: 5.0-17.0 μg/dL
         v. The test is positive for hypoadrenocorticism: no response to ACTH (post-ACTH cortisol concentrations < 2.0 μg/dL in nearly 100% of cases)
         vi. In cases of hypoadrenocorticism due to chronic exogenous glucocorticoid drug administration (iatrogenic): low to normal resting cortisol concentration (≤ 5.0 μg/dL) with subnormal response to ACTH (≤ 5.0 μg/dL)
         vii. Perform before administering glucocorticoids that may interfere with test results.
            (A) Assay cross reactivity: Most glucocorticoid cross react in the cortisol assay. Dexamethasone does not and can be given if necessary
            (B) Biological interference over time: Dexamethasone, like other glucocorticoids, will suppress ACTH secretion, leading to atrophy of the adrenal gland and decreased response in an ACTH stimulation test. If suspecting primary hypoadrenocorticism in a dog that is already receiving glucocorticoids, a long (usually weeks, depending on the drug) wash-out period is needed before and ACTH stimulation can be done. In a dog that was previously receiving glucocorticoids (that have been discontinued long enough before the testing and are not cross reacting in the assay), normal response to ACTH (high cortisol concentration) rules out hypoadrenocorticism but a suppressed response should be interpreted cautiously.
      b. Endogenous ACTH
         i. Why measure eACTH: In atypical disease, differentiate between primary and secondary
            (A) Progression to mineralocorticoid deficiency has been reported with Atypical Addison’s: Primary atypical HA requires periodic testing of electrolytes for prevention of an Addisonian crisis (life threatening)
            (B) Secondary HA does not require further monitoring of electrolytes: Big money saver!
         ii. ACTH is high in primary hypoadrenocorticism
         iii. ACTH is normal to low (often undetectable) in secondary hypoadrenocorticism
         iv. Normal reference range in dogs (varies with laboratory): 6.7 – 25 pmol/L
         v. ACTH is fragile and sample must be handled and shipped appropriately (avoid contact with glass during collection, separation and storage; store and ship frozen). Sending a sample from a normal dog as a control will facilitate interpretation of results (if both return low, problems likely occurred in sample handling)
Diagnosing and Treating Feline Hyperthyroidism
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A. Diagnosis of hyperthyroidism in cats
1. Signalment: No sex predisposition, older cats (< 5% are < 8 years of age at diagnosis)
2. History: Weight loss (> 90% of hyperthyroid cats) often concurrently with polyphagia (60% of hyperthyroid cats), polydipsia and polyuria (45-50% of hyperthyroid cats), increased activity or restlessness (40% of hyperthyroid cats), gastrointestinal signs (vomiting, diarrhea), heat intolerance (5% of hyperthyroid cats)
3. Physical findings: Palpable enlargement of thyroid gland (91% of hyperthyroid cats), thin (only 71% of hyperthyroid cats!), cardiovascular abnormalities (Tachycardia, systolic murmur, gallop rhythm), unkempt coat, hyperactivity, easily stressed, mild hyperthermia (14% of hyperthyroid cats), systemic hypertension
4. Laboratory findings
   a. Hemogram (aka complete blood count or CBC): Erythrocytosis (40-50% of cases), increased MCV (20-25% of cases), stress leukogram (but eosinophilia and lymphocytosis have also been reported)
   b. Biochemical profile: Mild to moderate increase in liver enzyme (ALT, ALP, AST) activities (60-90% of cases). Increased BUN and phosphorus (10-30% of cases)
   c. Urinalysis: Wide variability in urine specific gravity (USG): Isosthenuria doesn't indicate renal failure necessarily, even if the BUN is increased.
   d. Tests of thyroid function:
      i. Serum total T4 concentration (TT4):
         (A) Increased in > 90% of hyperthyroid cats.
         (B) TT4 is normal in ≤ 10% of hyperthyroid cats:
            (1) In early disease, daily fluctuations in T4 can result in an isolated normal result. Often, repeating the test on another day (or week) is useful. Alternatively, you can measure free T4 (see below).
            (2) Non-thyroidal illness (especially chronic renal disease) can result in a normal serum total T4 concentration in a hyperthyroid cat.
      ii. Serum free T4 (fT4) concentration by equilibrium dialysis: More sensitive but less specific than total T4 (fT4 is sometimes increased in diabetes, GI disease, and other conditions)

V. Hyperthyroidism and the kidneys
A. Hyperthyroidism and renal disease are both common in older cats.
B. Hyperthyroidism leads to increased cardiac output and increased renal perfusion resulting in a relative increase in GFR.
1. Increased GFR leads to a decrease in creatinine (potentially masking an underlying renal disease).
2. Increase increased renal perfusion leads to increased glomerular capillary pressure → proteinuria → progression of renal disease.
3. Hyperthyroidism may mask CKD but it might also contribute to its progression
C. About 30% of cats will develop azotemia within 30 days after treatment of hyperthyroidism, but the azotemia tends to remain stable in the majority of cats.
D. In hyperthyroid cats, prior to treatment, there is no practical way to predict accurately:
1. In which cat kidney disease will be unmasked by treatment
2. In which cat kidney disease will develop after treatment
E. A combination of GFR, crea, T4, and USG might be somewhat predictive but cannot be used conclusively to predict response to treatment
F. Medical therapy is recommended in cats with pre-existing kidney disease because of the risk of worsening azotemia.
G. A Methimazole trial is the most reliable way to assess kidney function in euthyroidism
1. Confirm euthyroidism for a minimum of 4 weeks
H. There is some evidence that hypothyroidism might cause a decrease in GFR.
1. Any treatment of hyperthyroidism might result in hypothyroidism (but severity and reversibility differ)
I. Definitive treatment of hyperthyroidism in a cat with CKD might be detrimental, beneficial or neither…

VI. Treatment of hyperthyroid cats:
A. Medical, surgical, dietary and 131I therapy can all be effective. Side-by-side comparison of these various treatment modalities have not been performed in cats. Based on mostly retrospective, often uncontrolled studies, as a whole, 131I seems to be associated with the longest survival time. Usually, the choice between these treatments is made based on convenience, cost, and side effects.
B. With all treatment modalities, effective reduction of thyroxine concentration could lead to significant reduction in GFR. The magnitude of reduction in GFR is depends on how effective treatment is (as well as on intrinsic factors).
C. In cats with kidney disease.
D. Oral methimazole: Predictable response, quickly reversible
   1. significant side effects, and not uncommon:
a. Anorexia, vomiting, lethargy (10-15%)
b. Cutaneous excoriations (2-3%), often peri-auricular
c. Hematologic changes (3-9%): ↑ eos, ↑ lymphs, ↓ neuts, bleeding tendencies
d. Liver toxicity (↑ liver enzymes, rarely ↑ Tbil)

E. Topical methimazole: Inconsistent absorption? Owners must wear gloves. quickly reversible
   1. Side effects: Similar to oral methimazole except for reduced frequency of anorexia vomiting

F. Thyroidectomy: Predictable response, irreversible (except with oral supplementation of thyroxine). Need to verify that no ectopic tissues are present prior to surgery. High cost up front.
   1. Risks:
      a. Anesthesia (should ideally stabilize first with methimazole)
      b. Hypocalcemia (potentially prolonged hospitalization)
      c. Neurological damage
      d. Vagosympatheic trunk -> Horner’s syndrome
      e. Recurrent laryngeal -> change in meow/purr
      f. Recurrence in contralateral lobe (with unilateral)
      g. Hypothyroidism (with bilateral)

   1. 95% of cats are euthyroid within 3 months.
   2. Serum TT4 is low in 16% of cats after treatment but only 2% develop clinical hypothyroidism.
   3. <2% of cats fail to respond by 6m and require second treatment
   4. 2.5% of cats relapse within 1-6 years
   5. High cost upfront
   6. Isolation of a potentially unstable patient (should ideally stabilize first with methimazole)

H. Iodine deficient diets (Hill’s a/d): Significant reduction in T4 might take weeks. Quickly reversible (by feeding any non-iodine deficient diet). Palatability is sometimes an issue. Efficacy seems lower than other treatment modalities (inconsistent feeding?). Long-term survival studies of treated hyperthyroid cats are not available.
Diagnosing Hypercortisolism in Dogs
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VII. Etiology of HC (general term: Cushing’s syndrome):
- Most common: Pituitary adenoma (AKA pituitary-dependent HC or PDH = Cushing’s disease) secreting excess ACTH which leads to bilateral adrenocortical hyperplasia and excessive production of cortisol (80-85% cases of naturally occurring HC)
- Adrenocortical neoplasia (15-20% of naturally occurring HC cases), AKA adrenal-dependent HC or ADH: May be benign (adenoma) or malignant (carcinoma)
  - Benign and malignant tumors are not easily distinguished clinically, but:
    - Carcinomas tend to be bigger
    - Carcinomas tend to invade adjacent structures (e.g. caudal vena cava)
  - Tumors usually are unilateral and, although presumably atrophied, the contralateral gland typically does not always appear smaller on abdominal ultrasound examination
- Iatrogenic: Long-term administration of (or exposure to) glucocorticoids
- Food-dependent HC (ACTH-independent):
  - Rare
  - Likely the result of aberrant expression of receptors to GI hormones on cortisol-secreting cells

VIII. Signalment
- A disease of middle-aged and older dogs: Almost all are > 6 years of age; rarely reported in young dogs
- No strong sex predilection
- Can occur in any breed or in mixed breed dogs
  - Commonly affected breeds include Poodle, Dachshund, Boston terrier, Boxer, and German shepherd
  - Of all dogs with PDH, 75% are <20 kgs. Of all dogs with ADH, “only” 50% are < 20kgs. In small dogs PDH is about 10 times more likely than ADH. In big dogs, PDH is “only” about 4 times more likely than ADH. This difference is the root of the misconception that ADH is more common in large breed dogs (it is more common than in small breeds but it is not more common than PDH regardless of the breed).

IX. History and Physical Findings
- Generally considered “healthy” (“just getting older”) by their owners
- Clinical signs are due to the gluconeogenic, lipolytic, protein catabolic, anti-inflammatory, and immunosuppressive effects of excess glucocorticoids
- Polyuria and polydipsia (> 80% of cases)
- Polyphagia (> 60% of cases)
- Abdominal enlargement due to weak abdominal muscles and hepatomegaly (> 70% of cases)
- Decreased exercise tolerance
- Muscle weakness
- Lethargy
- Hepatomegaly (hepatocyte vacuolation due to glycogen accumulation)
- Panting
- Cutaneous problems (almost all cases have some dermatologic signs, BUT… it is rare to have just dermatologic signs without at least Pu/Pd or PP)
  - Bilaterally symmetrical truncal alopecia
  - Thin, dry, scaling skin
  - Hyperpigmentation
  - Easy bruising (e.g. after venipuncture)
  - Comedones (especially around teats)
  - Calciosis cutis (calcium deposition in the dermis; uncommon but very suggestive of hyperadrenocorticism)
  - Pyoderma (increased susceptibility to infection)
- Hypertension (> 50% of cases)
- Increased susceptibility to infections
- Poor wound healing
- Pulmonary thromboembolism (see complications)

Some clinical signs are very inconsistent with HC and should prompt you to think of other differentials for the above problems (or at least prompt you to delay testing for HC until they resolve): Decreased appetite, vomiting, diarrhea, sneezing, coughing, pruritus, icterus, pain, seizures, bleeding.
X. Laboratory findings

- Hemogram ("stress" leukogram)
  - Lymphopenia (cortisol causes lymphocytolysis and eosinopenia (cortisol causes increased sequestration of eosinophils in the bone marrow)
  - Leukocytosis due to neutrophilia and monocytopsis (cortisol causes capillary demargination of these cell types)
  - Lymphopenia and eosinopenia are observed most consistently (80% of cases)
  - Mild to moderate erythrocytosis (increased PCV) occasionally
  - Thrombocytosis (cause unknown)

- Biochemistry
  - Mild hyperglycemia - 35% of cases. Severe hyperglycemia, glycosuria and clinical signs of DM are unusual (<10% of cases).
  - Hyperlipidemia (high cholesterol and triglycerides) (75% of cases)
  - Mildly to moderately increased alanine aminotransferase (ALT) (50% of cases)
  - High bile acids
    - Indicating liver dysfunction
    - HC is associated with gall bladder mucoceles in dogs
  - Increased alkaline phosphatase (ALP) including total ALP and corticosteroid-induced isoenzyme of cALP (cALP is unique to the dog).
    - Approximately 90% of cases have increased ALP (and in 10% ALP is normal!)
    - cALP is very sensitive but not specific: normal cALP tends to rule out hyperadrenocorticism but a high value does not rule it in
    - No correlation between magnitude of increase in ALP and…
      - The likelihood of having HC
      - The severity of HC

- Urinalysis
  - Low urine specific gravity (1.001-1.020 in approximately 80% of cases)
  - UTI is common (approximately 50% of cases) but pyuria (i.e. white cells in the urine sediment) is not (< 20% of cases) because increased concentrations of glucocorticoids impair migration of white blood cells into urine. The anti-inflammatory effects of cortisol also may explain why few of these dogs have clinical signs of cystitis.
  - Avoid catheterization for urine collection because of the increased risk of infection; use cystocentesis instead
  - Proteinuria (UPC > 1.0 in 46% of HC)

- Serum total thyroxine (T4) and free thyroxine (fT4) concentrations
  - Often decreased (~50% of cases) due to decreased thyroid-binding globulin, increased metabolism of thyroid hormones and decreased peripheral conversion of T4 to T3. Glucocorticoids also suppress TSH secretion and cause secondary hypothyroidism.
  - Don’t mistake for primary hypothyroidism!!!
  - Treatment with T4 is not indicated!!!

- Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) secretion also are inhibited by cortisol and can contribute to failure to cycle in females or testicular atrophy in males

- Abdominal ultrasound examination
  - Useful mostly for differentiation of PDH (symmetric) vs. ADH (asymmetry)
  - Adrenal gland size is correlated to body weight: Most of the variation is in the length of the adrenal gland, less so in the width/thickness (or diameter, terminology varies between sources)
  - Normal adrenal gland thickness varies less and a cutoff of 0.75 mm was used until recently to differentiate normal from enlarged
  - Using this cutoff, especially for small dogs, adrenomegaly would only be present in about 80% of HC dogs
  - Recent data suggest different cutoffs based on breed/size might be useful in refining the accuracy of adrenal width measurement in the diagnosis of HC but more studies are required to assess the validity of these new cutoffs.
  - Hyperplastic nodules, non-functional tumors, and non-cortisol secreting tumors can all cause adrenomegaly
  - Could non-adrenal illness cause enlarged adrenal glands?
    - Yes: In cats with hypersomatotropism
    - Other diseases???
  - Alone or combined with the endogenous ACTH assay, identification of adrenal glands on abdominal ultrasound examination can be used to successfully discriminate between PDH, ADH and iatrogenic causes of HC
    - Bilateral symmetrical enlargement (i.e., adrenal glands retain their normal “peanut” shape) with normal echogenicity in a dog with confirmed HC suggests PDH
    - Non-symmetrical abnormalities (Unilateral enlargement, distortion of shape, invasion into adjacent structures, and a small contralateral gland) in a dog with confirmed HC suggests ADH
    - In a dog with signs of HC, small adrenal glands are suggestive of iatrogenic HC
  - Carcinomas usually are bigger than adenomas and may invade local structures (e.g. caudal vena cava)

- Screening tests
  - Used to decide if an animal has hypercortisolism or not (i.e., answers the question, “Cushing’s or not?”)
- Resting cortisol concentrations are not valuable because many dogs with HC have normal cortisol concentrations at any given moment due to the episodic secretion of ACTH, and dogs with stress due to non-adrenal illness can have higher than normal resting cortisol concentrations.

- The first and most important screening test is taking a thorough history and doing a thorough physical exam!!

- From the American College of Veterinary Internal Medicine (2012) consensus statement on diagnosis of HC:
  - Avoid testing if other serious illness is present – any serious illness will cause false positive results for HC
  - Postponing testing for HC is recommended until concurrent illness is resolved or at least well controlled
  - Reference ranges for cortisol-related tests are outdated and all cutoff values should be interpreted cautiously (Below, cutoff values are approximate for normal, positive and negative results).

- From the American College of Veterinary Internal Medicine (2012) consensus statement on diagnosis of HC: “The LDDST is the screening test of choice unless iatrogenic HAC is suspected”

- Low dose dexamethasone suppression test (LDDST)
  - Best used as a screening test but sometimes can provide discriminatory information (see below)
  - Obtain resting plasma cortisol concentration
  - Give 0.01 mg/kg dexamethasone IV
  - Obtain 4- and 8-hour post-dexamethasone plasma cortisol concentrations
  - The 8 hour sample is used for diagnosis of HC. The zero and 4 hour samples are only useful as part of the discriminatory test (see below)
  - In normal dogs, plasma cortisol concentration is suppressed to \( \leq 1.0 \, \mu g/dL \) at 4 and 8 hours post-dexamethasone. Failure to suppress at the 8 hour time point, (i.e. 8 hour cortisol \( > 1.0 \, \mu g/dL \)) suggests hypercortisolism but does not differentiate between etiologies (PDH, ADH, etc.)
  - Using 8 hour cortisol concentrations as a screening test (at a cutoff of 1 \( \mu g/dL \)) the LDDST is very sensitive (approximately 95%) but not very specific (approximately 70%).

- For use of LDDST as a discriminatory test: See below.

- ACTH stimulation test
  - Old protocol: Obtain resting plasma cortisol concentration, give 0.25 mg synthetic ACTH IV or IM, obtain a 60-min post-ACTH plasma cortisol concentration
  - Normal resting cortisol concentration: 1-5 \( \mu g/dL \) but fluctuates considerably, especially in ill animals and has no diagnostic value.
  - Up-to-date protocol: No need to obtain a resting plasma cortisol concentration!
    - Give 5 \( \mu g/kg \) cosyntropin (synthetic ACTH) IV or IM
    - 5 \( \mu g/kg \) is sufficient, higher doses increase the cost unnecessarily. Cosyntropin is expensive. It is supplied as a 250 \( \mu g \) vial. To decrease cost, divide the vial into 5 aliquots of 50 \( \mu g \) and use as many aliquots as needed to reach a minimum dose of 5 \( \mu g/kg \)
  - Obtain a 60-min post-ACTH plasma cortisol concentration
  - normal post-ACTH cortisol concentration: 8-20 \( \mu g/dL \) (reference range will vary among laboratories)
  - Interpretation
    - An exaggerated response to ACTH (i.e. post-ACTH cortisol > 20 \( \mu g/dL \)) is consistent with PDH or ADH but also can be observed in animals with non-adrenal illness
    - A post-ACTH cortisol below the normal range (<5 \( \mu g/dL \)) is the result of atrophied/suppressed adrenals caused by iatrogenic HC, treated HC or Addison’s disease (Usually <2 \( \mu g/dL \) in Addison’s)
    - For diagnosis of HC: The ACTH stimulation is a reasonably specific test (approximately 90%) but NOT a sensitive one (60-95%). The sensitivity is reasonably high (95%) when testing PDH but abysmally low (60%) when testing ADH. Normally though, the screening test is done BEFORE it is clear if the dog has PDH or ADH…
    - Because of its low sensitivity: If negative, cannot rule out HC (especially if ADH is suspected)
    - Chronic stress of non-adrenal illness can cause abnormal results on ACTH stimulation (but not as much as it would in the LDDST and UCCR)
    - Acute stress has no effect on the test (the test mimics maximal acute stress – overdose of ACTH)
    - Advantage of ACTH stimulation test (over LDDST): Takes less time than LDDST and is less affected by non-adrenal illness
    - The ACTH stimulation test is the only diagnostic test available to identify dogs with iatrogenic hypercortisolism
    - The ACTH stimulation test is the test of choice to monitor dogs being treated for hypercortisolism with mitotane or trilostane (see later)
    - 17-hydroxyprogesterone (17-OH Prog) measurement: Can be measured before and after ACTH stimulation.
      - Used for diagnosis of “atypical Cushin’s syndrome”, i.e. Cushin’s like signs that are caused by an excess in sex hormones

Sex-hormone secreting adrenal tumors causing signs of Cushing’s have been described rarely but increased sex hormones concentrations (including 17-OH Prog) are frequently found in “regular” HC. Overall, measuring 17OH Prog is less sensitive and less specific than measuring post-ACTH cortisol (“regular ACTH stim)
Urine cortisol/creatinine ratio

- In this test, urine cortisol (and some of its degradation products) and urine creatinine are measured on the same urine sample. Dividing the cortisol concentration by the creatinine concentration corrects for the effect of concentrated or dilute urine on the urine cortisol concentration.
- The main advantage of this test is its simplicity: the owner need only bring in a morning urine sample from the dog.
- The main disadvantage is that, although the test is very sensitive (> 90%), it has very low specificity (20-40%) and is easily affected by stress (of any kind).
- Thus, it is useful to rule out a diagnosis of hyperadrenocorticism, but it is not helpful in ruling in the diagnosis. Remember sPin and sNout: a specific test, if positive, rules the diagnosis in; a sensitive test, if negative, rules the diagnosis out.
- Clinicians sometimes use this test if they don’t think hypercortisolism is very likely and want to remove it from their differential diagnosis list. However, if the test comes back positive, the diagnosis must be pursued by other screening tests (e.g., ACTH stimulation test, LDDST).
- The urine cortisol/creatinine ratio is affected by non-adrenal illnesses more so than the LDDST and the ACTH stimulation test.

Discriminatory tests: used to differentiate PDH from ADH

- Abdominal ultrasound: Identification of adrenal glands on abdominal ultrasound examination can be used to successfully discriminate between PDH, ADH and iatrogenic causes of HC.
  - Bilateral symmetrical enlargement (i.e., adrenal glands retain their normal “peanut” shape) with normal echogenicity in a dog with confirmed HC suggests PDH.
  - Non-symmetrical abnormalities (Unilateral enlargement, distortion of shape, invasion into adjacent structures, and a small contralateral gland) in a dog with confirmed HC suggests ADH.
- In one study of dogs with inconclusive asymmetry between glands, adrenal width < 5 mm on the contralateral gland was highly sensitive and specific for the diagnosis of ADH. (Benchekroun et al. JVIM 2010)
- In a dog with signs of HC, small adrenal glands are suggestive of iatrogenic HC (combine with ACTH stimulation).

Dexamethasone suppression test (DST)

- Low dose [LDDST]: 0.01 mg/kg (used primarily as a screening test).
- High dose [HDDST]: 0.1 mg/kg (used primarily as a discriminatory test).
- The discriminatory aspect of the test is based on the principle that a dose of dexamethasone would sometimes suppress cortisol concentrations in dogs with PDH but never in dogs with ADH.
- Thus, if dog with suspected hypercortisolism suppresses on the DST that result is consistent with PDH, but if the dog fails to suppress, you cannot draw any conclusions and must use other discriminatory tests.

Four criteria for suppression:

- 50% decrease in [cortisol] (from time 0) at 4h (LDDST, HDDST).
- 50% decrease in [cortisol] (from time 0) at 8h (LDDST, HDDST).
- [Cortisol] < cutoff value (1.0 ug/ml?) at 4h (LDDST, HDDST).
- [Cortisol] < cutoff value (1.0 ug/ml?) at 8h (HDDST only).
- These 4 criteria apply to HDDST. In the LDDST, only criteria 1-3 apply for differentiation of PDH from ADH. The 8h sample in the LDDST is used for diagnosis (see above) and by definition, a cortisol concentration below the cutoff at that time point rules out HC and therefore cannot be used to differentiate PDH from ADH.

- Using these criteria, dogs with PDH will have suppressed cortisol 63% on LDDST and 75% on HDDST. Hypothetically, higher doses of dexamethasone will lead to suppression of more pituitary tumors (thus increasing the sensitivity of the test), however, it is not practical to repeat the test with higher doses (diminishing return...).

Endogenous ACTH concentrations

- Normal: 6.7-25 pmol/L.
- eACTH is completely suppressed in ADH but is measurable (and frequently high) in PDH.
- Adrenocortical tumor (ADH): < 5 pmol/L (assay detection limit).
- Pituitary-dependent hypercortisolism (PDH): > 6 - 1250 pmol/L.
- ACTH is fragile and sample must be handled and shipped appropriately (avoid contact with glass during collection, separation and storage; store and ship frozen). Sending a sample from a normal dog as a control will facilitate interpretation of results (if both return low, problems likely occurred in sample handling).
- Out-dated assays had low sensitivity and were frequently unable to detect ACTH in cases of PDH, misclassifying PDA as ADH.
- A recent study (using a new ACTH assay) reported that for the diagnosis of ADH, the eACTH has a sensitivity of 85-100% and a specificity of 97-100% (Rodríguez Piñeiro et al. JVIM 2009).
- Performed correctly, both ultrasound and eACTH are highly accurate as discriminatory tests however:
  - eACTH is not widely available.
  - eACTH is VERY fragile (sample must be collected, handled, and shipped very carefully, constantly frozen).
- Abdominal U/S is more expensive
- Abdominal U/S provides additional information on other organs and on the presence of metastasis and local invasion of adrenal tumors and

- Other imaging modalities (e.g. magnetic resonance imaging [MRI])
  - Normal pituitary gland height is approximately 5 mm
  - Approximately 50% of dogs with untreated PDH and no neurologic signs have normal MRI findings and 50% have identifiable pituitary tumors (4-13 mm in height)
  - Neurologic signs typically are associated with tumors > 10 mm (so-called “macrotumors”) that expand dorsally beyond the sella turcica
  - Abdominal CT has no advantage over ultrasound for differentiation of PDH from ADH.
Overview of calcium homeostasis
Parathyroid hormone (PTH) is the principal hormone involved in the minute-to-minute fine regulation of blood calcium concentration through effects on tubular reabsorption of calcium, intestinal absorption of calcium mediated indirectly via calcitriol, and bone resorption of calcium. PTH is secreted by chief cells in the parathyroid glands. Overall, PTH increases plasma calcium concentrations. The parathyroid glands are exquisitely sensitive to fluctuations in ionized calcium (iCa), especially when iCa is low. If the parathyroid glands are responding appropriately, PTH secretion will dramatically increase when iCa is low; when iCa is high, PTH secretion will decrease. Parathyroid hormone has direct effects in the kidneys and bones and indirect effects in the intestines:

- In bone, PTH induces osteoclastic bone resorption, which increases calcium and phosphorus resorption.
- In the kidney, PTH increases calcium reabsorption and phosphorus excretion. It also increases the activity of 1α-hydroxylase, the enzyme responsible for converting 25-hydroxyvitamin D (25OHD) to the active form of vitamin D: Calcitriol (1,25-dihydroxyvitamin D3).
- Calcitriol increases calcium and phosphorus absorption in the gastrointestinal tract.

Tissues involved in calcium regulation (parathyroid gland, thyroid gland, kidney) express a Ca2+-sensing receptor (CaSR). In the parathyroid gland, the CaSR insures that the increase in secretion of PTH is proportional to the decrease in plasma iCa in the physiologic range of iCa concentrations. In that range, a small change in iCa causes large changes in PTH concentrations. However, outside of that normal range the response of the parathyroid gland to changes in iCa is remarkably different: Above the normal iCa concentrations, PTH secretion is quickly and mostly inhibited. Below the normal iCa concentrations, PTH secretion quickly reaches maximum:

The set-point for PTH secretion is defined as the concentration of circulating calcium that results in half the maximal PTH secretion that can be achieved. In some diseases, abnormalities in iCa concentrations are caused by shift in this set point. For example, in people with Familial Benign Hypocalcemic Hypercalcemia (FBHH), a genetic defect in the CaSR causes decreased sensitivity to iCa so that the set point is increased: PTH secretion is “normal” at abnormally high concentrations of iCa. This genetic disturbance may be a good model for Feline IHC.

Figure 1. Measurement and interpretation of PTH concentrations

Differential diagnoses for hypercalcemia in cats
H = Hyperparathyroidism (primary)
A = Addison’s disease
R = Renal disease (acute or chronic kidney disease)
D = vitamin D toxicity
I = Idiopathic
O = Osteolysis
N = Neoplasia or Nutritional
S = Spurious or Systemic granulomatous disease

From the most common to least common: Idiopathic, renal disease, neoplasia… all the rest…

Diagnostic tools for calcium-related disorders: Major challenges
PTH is released from the PTGs as an 84 amino acid single-chain peptide. This intact active form of the hormone (PTH1–84) is inactivated by hepatic and renal metabolism (plasma half-life approximately 2–4 mins). The 1–34 N-terminal region is essential for the biological activity of PTH and cleavage at that site by endoproteases renders the hormone inactive. In this process, fragments of various lengths are produced and released into the blood. These fragments are then cleared from the circulation by renal excretion. The analysis of ‘true’ PTH1–84 is not straightforward – there exists a surprisingly heterogeneous range of PTH fragments. Both the exact composition and possible biological functions of PTH fragments remain to be fully elucidated as does the variable influence of these...
fragments on currently available analytical methods for PTH testing. Accumulation of PTH fragments in patients with renal failure for example can be so pronounced that the intact 1-84 hormone accounts for only 5-20% of the measured PTH depending on calcemia status and stage of renal disease.

Second-generation (or ‘intact’), and third-generation (or ‘whole’) two-site PTH immunoassays use 2 antibodies: one directed at the C-terminal region (amino acids 39-84) and one directed at the N-terminal region of the intact 1-84 PTH molecule. The use of a two-site assay eliminates detection of inactive PTH partially (in ‘intact’ 2nd generation assays) or entirely (in ‘whole’ 3rd generation assays). Third generation assays are directed against the well-conserved region of amino acids 1-4. This is also a region that is necessary for activation of the PTH receptor, making these assays specific to the active PTH molecule. In contrast, second generation assays detect amino acids 12-32. With these second generation (‘intact’) assays the specificity to the active PTH is incomplete. Another important issue in cats is that commercial PTH assays use antibodies raised against human PTH. Feline PTH however differs in amino acids 16, 18 and 26 which are detected by second-generation assays. Thus, the affinity of these human PTH assays to feline PTH is decreased, rendering these assays insensitive. To date, most studies in which PTH was measured in cats used second generation human PTH assays with sensitivities so low that the “reference range” of PTH concentrations overlapped with the low limit of detection of the assay. This reduces the ability of these assays to detect PTH concentrations that have been suppressed below normal by hypercalcemia. This is crucial in conditions in which a low PTH concentration is the key finding for diagnosis (e.g., differentiating primary hyperparathyroidism from idiopathic hypercalcemia).

**Feline Idiopathic Hypercalcemia (IHC)**
An “emerging” disease, first described in a retrospective study by Midkiff et al (JVIM 2000). Defined as persistent hypercalcemia in a cat in which no diagnosis (from the list of differentials above) can be made.

- Mostly middle age to older cats but has been reported in cats as early as 2 years of age.
- Male ≈ female, various breeds
- Unclear relationship to commercial diets
- Mild-to-moderate hypercalcemia (total and ionized calcium)
- Hypercalcemia may be detected incidentally: about ~50% of cats present with no clinical signs
- Reported clinical signs might be incidental. In a recent retrospective study comparing cats with IHC to a control population of cats referred to a teaching hospital for a variety of conditions, no clinical sign emerged as more likely to occur in IHC. In that study, IHC cats were more likely to present with lethargy and hypercalcemic cats in general were less likely than controls to present with signs of lower urinary tract disease.
- Previously reported association between urolithiasis and IHC was based on anecdotal evidence. CaOx are frequently found in cats with IHC but their frequency in this disease is not higher than the frequency in a control population.
- Gastrointestinal signs (including vomiting, weight loss, decreased appetite, constipation) have been reported with IHC but they do not occur more frequently in IHC than in a control population.
- IHC is likely an umbrella diagnosis: The lack of obvious association between certain clinical signs and other potential disease consequences (as explained above) may be representative for IHC as a group but not necessarily for an individual cat.
- To date, there is no evidence that treatment of idiopathic hypercalcemia has any benefits in cats.
- In people with Familial Benign Hypocalciuric Hypercalcemia (a disease that resembles feline IHC in many ways), treatment of the hypercalcemia is generally not indicated. In FBHH, mutations in the CaSR cause decreased sensitivity of the target tissue to the effect of calcium. In the parathyroid gland this leads to the “perception” that iCa is not as high as it “should be” which leads to increased secretion of PTH. Similarly, the renal tubules do not sense calcium normally in this disease, leading to increased calcium reabsorption, increased plasma calcium concentration and decreased urinary calcium excretion.
Insulin Resistance in Cats
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Insulin Resistance

- In the treated diabetic patient in practice, insulin resistance is defined by response to treatment (i.e. high insulin dose required to achieve glycemic control):
  1. >3U/inj/Cat
  2. >1.5 – 2.0 U/kg/inj in dogs
  3. In the non-diabetic patient: Insulin/glucose ratio
  4. In the research settings: hyperinsulinemic-euglycemic clamps

- Pathophysiology of insulin resistance:
  1. Circulating insulin antagonists (related to stress, inflammation, obesity)
     - ↑ Counterregulatory hormones (e.g. glucagon, cortisol, GH)
     - ↑ Cytokines (TNF-α, IL-6)
     - Insulin antibodies
  2. Target tissue defects (obesity, congenital, others)
     - Insulin-receptor defects
     - Post-receptor defects

- Common causes of insulin resistance:
  a. Bacterial infections (periodontal disease, UTI)
  b. Major organ failure (heart, liver, kidney)
  c. Pancreatitits
  d. Concurrent endocrinopathies (obesity, hypercortisolism, acromegaly, hyperthyroidism)
  e. Administration of any drugs which antagonize insulin (eye, ear Rx common culprits)
  f. Heat cycles
  g. Insulin-induced hyperglycemia?
     1. Result of too much insulin
     2. Response to either hypoglycemia
     3. (BG < 60 mg/dl) or too rapid a decline in BG
     4. Secretion of counter-regulatory stress hormones
     5. Can results in an overall increase of BG with worsening of Pu/Pd and increased fructosamine

- Diagnosis of insulin resistance:
  1. Correlate results of glucose curves with clinical signs
  2. Is it really insulin resistance, or are owners not giving insulin properly?
     a. Handling of insulin & syringe, administration technique (Watch owners administer injection!!!)
     b. Check type, dose and expiration date
     c. If using a diluted insulin, switch to non-diluted
     d. Storage of insulin
  3. Is there any concurrent disease?
  4. If none of the above applies, consider insulin-induced hypoglycemia and decrease insulin dose

Hypercortisolism in cats

- Uncommon
- Females > males?
- Mostly similar to the syndrome in dogs
- Valentin et al. JVIM 2014: multicenter retrospective (1990 – 2011), N = 30
  a. Females = males
  b. Age range: 4 – 17y (median 13y)
  c. 90% PDH, 10% AT
  d. Dermatological signs (100%) including Skin Fragility Syndrome
     i. Thin skin (70%)
     ii. Alopecia (60%)
     iii. Skin lacerations (57%)
     iv. Dull coat/scaling/seborrheic (13%)
- Mellett JVIM 2013: N = 15, Dermatological signs (73%)
- Polyuria/Polydipsia (87%)
- Polyphagia (70%)
- Abdominal distention (67%)
- Muscle wasting (67%)
i. Lethargy (47%)

j. Weight loss (47%), weight gain (23%)

k. Unregulated DM (90%)

l. Other concurrent diseases:
   i. Bacterial infections (56%), including UTI’s, skin abscesses, rhinitis
   ii. Pancreatitis (30%)

m. CKD (36%)

n. Laboratory tests:
   i. Anemia (48%)
   ii. Hypochloremia (41%)
   iii. Hypertriglyceridemia (71%)
   iv. Normal TT4 (84%), low TT4 (16%)
   v. Normal ALP (80%)

o. ACTH stim: Variety of protocols
   i. Sensitivity: 56% (46% at 60 min)
   ii. All AT cases negative
   iii. Specificity???
   iv. 89% but not in diabetics

p. Dexamethasone Suppression test:
   i. 0.1 mg/kg
   ii. Sensitivity: 96%
   iii. Specificity?? No disease free group

- Abdominal ultrasound:
  - Valentin et al. JVIM 2014: 90% sensitivity (3/30 normal)
  - Combes et al. JFMS 2013: PDH, N = 4, 2/4 normal, 2/4 slightly enlarged
  - Combes et al. Vet Rad Ult 2012: The adrenal glands were significantly larger in hyperthyroid cats compared to normal

- Other tests ACTH precursors for diagnosis of PDH (Benchekroun et al. JVIM 2012)
  a. 229 pmol/L cut off: 89% sensitivity (8/9); 100% specificity in DM and in DM+acromegaly
  b. Not commercially available

- Medical management:
  o Valentin et al. JVIM 2014:
    - Mitotane is generally considered ineffective in cats but one cat survived > 5 years
    - Trilostane 0.5 – 12 mg/kg, q12-24h, survival: < 1 – > 21m
  o Neiger et al. JVIM 2004: Trilostane reduced clinical signs and improved endocrine test results in all cats, but insulin requirements did not change and all continued to have some signs of hypercortisolemia.
  o Mellett JVIM 2013: N = 15
    - Trilostane 10 – 30 mg/cat q24h or 10 – 20 mg/cat q12h
    - Clinical signs and ACTH stims improved in 13/15 cats.
    - Insulin requirements decreased by 36% within 2 months in 6/9 diabetic cats.
    - Median survival time: 617 d (range 80–1,278).
    - Hypocortisolemia was documented in 1 case.

- Surgical/radiation therapy
  - Adrenalectomy (uni or bilateral)
    - Smith et al. JAVMA 2012: Laparoscopic adrenalectomy for AT in a cat
  - Hypophysectomy (Utrecht)
  - Radiation Therapy (Sellon JVIM 2009): Variable response. DM remission is uncommon. Low complication rate

Hypersomatotropism (Formerly known as: Acromegaly) in cats

- Cause: GH-secreting pituitary adenomas
- Much more common than previously recognized
- Often causes diabetes without causing over signs of acromegaly
- Excessive GH secretion leads to:
  - Insulin resistance
  - Increased IGF-1 secretion from the liver → Bony (growth of extremities) and soft tissue overgrowth (causing HCM, renomegaly, hepatomegaly, enlarged endocrine glands, DJD, Spondylosis and more…)
  - Signs develop slowly

- Signalment:
  - Males and females are equally represented
  - No breed predisposition
  - Middle age and older
- Clinical signs:
Signs of uncontrolled DM often appear first
- The “typical” physical manifestations of acromegaly develop slowly and are often not present at all or at least not obvious when diabetes is already present (Renomegaly, hepatomegaly, heart murmur, plantigrade stance, progranthia inferior, broad face, stridor/stertor)
- Weight loss is sometimes seen but often not and many cats experience weight gain despite uncontrolled DM.
- Insulin resistance that could be extreme (insulin dose: median = 7U/cat BID [range 1-35])
- PuPdPP: 100%

### Diagnosis
- IGF-1 in uncontrolled diabetics is often high (low specificity) but above a certain cutoff, the positive predictive value of IGF-1 is high (95% PPV for IGF-1 > 1000ng/ml)
- IGF-1 in an untreated diabetic secondary to hypersomatropism can be low (low sensitivity) and increases with insulin treatment
- IGF-1 is a good screening test and should be performed in all diabetic cats that have not experienced diabetic remission (assuming appropriate treatment of DM with insulin and low carb diet for at least one month).
- Confirm diagnosis with brain imaging

### Treatment
- Radiation therapy (Sellon JVIM 2009, Dunning JVIM 2009)
  - Variable response. DM remission is uncommon.
  - Low complication rate
  - IGF-1 levels are not useful for monitoring
- Hypophysectomy
  - Most often curative and leads to diabetic remission
  - Available in a handful of centers around the world (WSU, OSU, RVC, Utrecht)
- Somatostatin-analogs (Niessen, JVIM 2013 abstract):
  - Pasireotide, once monthly injections.
  - Improved glycemic control and decreased IGF-1. No side effects except for hypoglycemia in one cat!
  - EXPENSIVE!
Diabetes mellitus: Pathophysiology
When hyperglycemia is persistent and severe enough to exceed the renal capacity for glucose reabsorption (180 – 200 mg/dl in dogs, 260 – 280 mg/dL in cats) glycosuria ensues leading to osmotic diuresis with free water, electrolytes and energy losses. Hyperglycemia and free water loss cause an increase in serum osmolality which in turn stimulates thirst. Insulin is an important anabolic hormone. A relative or absolute insulin deficiency will lead to breakdown of fat and muscles and weight loss. Lipolysis in peripheral adipose tissue leads to hyperlipidemia, mobilization of free fatty acids to the liver and hepatic lipidosis.

Glucose toxicity
- Is the result of prolonged and persistent hyperglycemia
- Causes impaired insulin secretion and down-regulation of glucose transport systems
- Leads to overt diabetes
- Resolves with insulin therapy
- May explain transient diabetes “honeymoon period” in cats

Diabetes remission
In cats, Type II DM is considered the most common type and although most cats require insulin therapy on initial presentation, periods of insulin independence (“remission”) may occur with appropriate dietary and insulin therapy. Remission in dogs is very unlikely, even in cases that seem secondary to steroid administration or in gestational diabetes although remission has been documented in dogs (gestational paper). In cats, remission occurs in approx. 50% of cases. Most studies did not find a correlation between remission rates and insulin type, duration of disease prior to starting treatment etc. One study found 100% remission rates with insulin glargine but these results were not reproducible and in other studies insulin type was not a significant factor. A canned, low fiber-low carbohydrate diet has been associated with higher remission rates (68%) when compared to a canned, moderate carbohydrate-high fiber diet (41%). Some studies suggest that longer duration of illness prior to starting insulin therapy decreases likelihood of remission. In 2 studies, an intensive treatment protocol (3-5 measurements of blood glucose daily and adjustment of insulin dose with the goal of reaching a blood glucose of 50 – 100 mg/dL) was associated with remission rates of almost 70% but all cats were fed a canned, low fiber-low carbohydrate diet. Importantly, it is unknown currently whether any protocol decreases the rate of complications of DM or prolongs survival.

Diagnosis of glycemic dysregulation
1. Based on appropriate clinical signs (polydipsia, polyuria, polyphagia and weight loss), persistent hyperglycemia, and glycosuria. A complete blood count (CBC), biochemistry panel, a urinalysis and a urine culture are recommended to rule out concurrent disease for any newly diagnosed diabetic and for the unregulated-treated diabetic. Serum total thyroxine concentrations should be included for cats over 9 years of age.
2. Serum fructosamine concentrations are useful for diagnosis of persistent DM and ruling out stress-related hyperglycemia/glycosuria.
3. For the treated, unregulated diabetic, an important component in the diagnosis of the cause of dysregulation is evaluation of therapy-related causes:
   a. Handling of insulin & syringe, administration technique (observe owners administer the injection?)
   b. Check type, dose and expiration date of insulin
   c. If using a diluted insulin, switch to non-diluted
   d. Appropriate storage of insulin vial

Monitoring of DM
Goals
- Avoid hypoglycemia
- Prevent DKA
- Resolve the clinical signs of diabetes
- Euglycemia is not a treatment goal in dogs (Maybe it should be in cats?)

Observation of changes in clinical signs is the corner stone of DM monitoring
- Consider changes in clinical signs since the last time therapy was altered

Serum fructosamine concentrations (SF)
- Reflect glycemic control over the course of 2 – 3 weeks.
• Indicated for routine monitoring of the treated diabetic. Small changes in glycemic control may not be apparent clinically but could still be reflected by changes in SF. Thus, it is recommended to obtain SF on diagnosis and every time a change is made in therapy: before (for baseline) and 2-3 weeks after.

• Trends in SF are more useful than absolute numbers (the latter don’t always fit the patient)

• SF are affected by the half-life of serum proteins. Thus, when protein turn-over is increased (e.g. hyperthyroidism, protein-losing enteropathy) SF can be falsely decreased.

• Hemolysis (either in-vivo or in-vitro) affects the laboratory’s ability to analyze SF and should be avoided.

Blood glucose curves (BGC)

• AAHA Diabetes Management Guidelines 2010: For a stable diabetic, advise owners to perform a blood glucose curve at home once a month.

• Results of BGC vary significantly from day to day even when factors like diet, meal size, insulin (formulation and dose) and site of injection are unchanged

• Day-to-day variability in insulin action precludes reliance on a single BGC for decision making unless hypoglycemia is documented.

• Day-to-day variability in BGC’s has been documented in studies of dogs in a hospital environment but also in cats when BGC’s are done at home with minimal stress.

• Factors associated with day-to-day variability in insulin action:
  o Meal composition and size
  o Stress
  o Activity level
  o Injection site (vasculature, temperature)
  o Injection technique
  o Dosing inaccuracies (especially with low doses)
  o Insulin absorption

• Dosing inaccuracies and variability in absorption from SQ depot are less of a problem with novel insulin formulations that are supplied as suspension (e.g. detemir and glargine) and more of a problem with traditional formulations that are supplied as suspensions (NPH, Lente, PZI)

• Measure BG at least q 2hr for at least as long as the insulin should last (12-24h depending on the insulin type)

• To determine the nadir: Identify at least 2 points on the upswing

• Interpret BGC’s carefully! Always correlate results with clinical signs and SF

• A diagnosis of hypoglycemia is useful: Reduce dose or start over with a different insulin formulation

• Diagnosis of duration, nadir, efficacy and “insulin resistance” based on a single BGC is problematic: Consider these diagnoses only if repeatable in multiple BGC’s

• More data points = decreased effect of day-to-day variability (consider home monitoring and continuous glucose monitoring).

Causes of inadequate glycemic regulation in the treated diabetic

• Insulin formulation and administration:
  o Check type and dose of insulin
  o Handling of insulin & syringe
  o Injection technique (observe owners administer injection!!!)
  o Storage, and expiration date of insulin
  o If using a diluted insulin, switch to non-diluted

• Concurrent diseases and causes of insulin resistance:
  o Bacterial infections (periodontal disease, UTI)
  o Major organ failure (heart, liver, kidney)
  o Pancreatitis
  o Concurrent endocrinopathies (Hypercortisolism, Acromegaly, hyperthyroidism, hypothyroidism)
  o Administration of any drugs which antagonizes insulin (eye, ear Rx are common culprits)
  o Heat cycles
  o Insulin-induced hyperglycemia
    • Result of too much insulin
    • Response to either hypoglycemia (BG < 60 mg/dl) or too rapid a decline in BG
    • Hypoglycemia leads to secretion of counter-regulatory stress hormones
    • Can results in an overall increase of BG with worsening of Pu/Pd and increased fructosamine

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Incretin-based therapies are revolutionizing the field of diabetes therapy by replacing insulin therapy with safer and more convenient, long-acting drugs. Incretin hormones (GLP-1 and GIP) are secreted from the intestinal tract in response to the presence of food in the intestinal lumen. GLP-1 augments insulin secretion and suppresses glucagon secretion during hyperglycemia in a glucose-dependent manner. Clinical data have revealed that incretin-based drugs are as effective as insulin in improving glycemic control while reducing body weight (GLP-1 analogs, specifically) in patients with type 2 diabetes. Furthermore, incidence of hypoglycemia is relatively low with these drugs because of their glucose-dependent mechanism of action. Another significant advantage of these drugs is their duration of action. While insulin injections are administered at least once daily, long-acting GLP-1 analogs have been developed as once-a-week injections and could potentially be administered even less frequently than that in diabetic cats. This review considers the physiology of incretin hormones, and the pharmacology and use of GLP-1 analogs in people. We will also review up-to-date research data on GLP-1 analogs in veterinary medicine.

**Incretin physiology**

Incretin-secreting cells (K and L cells) are interspersed in the epithelium of the gut. Their physiological role is to sense the type and quantity of digested nutrients in the gut. They then secrete incretin hormones as preparatory signals to other remote organs (e.g. brain, pancreas etc.). In the pancreas, the incretin signal is translated to increased sensitivity to the stimulatory effect of glucose. This effect is responsible for the observed difference in insulin secretion between oral and intravenous glucose and is defined as the “incretin effect”; oral glucose leads to much greater insulin secretion compared to intravenous glucose even when blood glucose concentrations are equal. The incretin effect is thought to be exclusively mediated by 2 peptide hormones: Glucagon-like peptide-1 (GLP-1) which is secreted from L cells, and glucose-dependent insulinotropic polypeptide (GIP) which is secreted from K cells. Incretin hormones also promote expansion of pancreatic beta-cell mass: They promote differentiation of pancreatic ductal cells into beta cells and increase beta cell proliferation. Importantly, incretin hormones protect beta cells from apoptosis induced by various cytotoxic agents or dexamethasone. They also prevent apoptosis secondary to glucotoxicity and lipotoxicity.

Incretin hormones affect glucagon secretion (GLP-1 inhibits, GIP stimulates), slow the rate of gastric emptying (GLP-1), increase satiety (GLP-1), and increase the sensitivity of adipose tissue to insulin (GIP). Thus, they play a major role in glucose homeostasis. Active GIP and active GLP-1 are degraded by the enzymes dipeptidyl peptidase-4 (DPP-4, also known as CD26) and neutral endopeptidase 24.11 (NEP-24.11) into inactive forms, thereby modifying or inhibiting their activity. DPP-4 and NEP are ubiquitous in tissues, and DPP-4 is also present in a soluble form in the blood. Consequently, when injected intravenously, active GLP-1 and GIP are quickly degraded into inactive forms. When injected intravenously, the half-life of GLP-1 is only about 1-2 minutes and the half-life of GIP is only about 5 minutes. The inactive GLP-1 and GIP are quickly cleared by the kidneys.

**Incretin hormones in diabetes and obesity**

The incretin effect is severely reduced or absent in people with type 2 DM, contributing to glucose intolerance and post-prandial hyperglycemia. In diabetics, the secretion of GIP is normal or slightly reduced but its effect on the pancreas is markedly decreased. In contrast, GLP-1 retains its insulinotropic effects in type 2 DM (at least in supraphysiologic concentrations), but its secretion is decreased. Whether a blunted incretin response is a primary process in type 2 DM or a secondary process caused by diabetes, is debatable. There is some evidence that obesity contributes to attenuation of the incretin effect independently of diabetes. In a recent study, obesity was associated with decreased incretin effect including decreased beta-cell response to GLP-1 but also decreased GLP-1 secretion. In some studies, GLP-1 secretion was normal in obese people but GIP secretion was increased during fasting and early after a meal. Insulin resistance in itself, regardless of obesity or diabetes, has been associated with abnormal secretion of GIP and GLP-1.

Increased insulin concentrations in the insulin-resistant subjects might down-regulate incretin secretion. Traditional treatments for diabetes are either just partially effective (diet change, oral drugs) or associated with significant side effects (insulin therapy). Owner compliance is a major problem because twice-a-day injections are required with most commonly used insulin preparations. Weight gain may indicate a good response to therapy initially but can eventually become a problem. Hypoglycemia is a common complication of insulin therapy and can be life-threatening. These side effects have been significantly reduced in human medicine with the introduction of incretin-based treatments. GLP-1 stimulates glucose-dependent insulin secretion. Its effect is blunted in euglycemia and therefore they are less likely to cause hypoglycemia. GLP-1 also promotes weight loss. GLP-1 analogs are not only effective in controlling blood glucose in diabetics but they are also associated with weight loss in diabetic and non-diabetic obese people. One major advantage of incretin-based therapies is their potential to reverse the course of the disease. Incretin-based treatments in diabetic people correct not only hyperglycemia but also all the major markers of beta-cell dysfunction.
The very short half-life of GLP-1 limits its clinical use. There are numerous treatment strategies that overcome this problem and take advantage of incretin-hormone physiology. Novel GLP-1 analogs have a prolonged half-life and are used as SQ injections at intervals of up to one week.

**Incretin-based therapy strategies**

There are several treatment modalities that overcome the problems associated with direct administration of incretin hormones. Long-acting, DPP-4-resistant, synthetic GLP-1 mimetic peptides are most commonly used. Available formulations are all for SQ injections but other delivery systems are being developed (including enteral, pulmonary, or sublingual). Also under development are non-peptidic GLP-1 receptor agonists. Oral DPP-4 inhibitors that prolong the half-life of incretin hormones are also commercially available. Non-peptidic drugs that activate free fatty acid receptors in L cells and stimulate GLP-1 secretion are being developed (GRP120 and GRP119 agonists). Diet manipulation should also be investigated.

Drugs that are commercially available or that are currently in advanced clinical trials are described below.

**GLP-1 mimetics and analogs**

**Exenatide**

The peptide exendin-4 was first isolated from the poisonous venom of the Gila Monster (*Heloderma suspectum*). Exendin-4 is a 39-amino-acid peptide that shares only a 53% sequence homology with GLP-1 but its affinity for the GLP-1 receptor is 1000 times greater than the affinity of GLP-1. Unlike GLP-1, exendin-4 is not a substrate for DPP-4 and NEP. Exenatide is a synthetic exendin-4. Resistant to degradation, exenatide is eliminated by the kidneys and has a half-life of 3-4 hours in people. Its biological effect lasts about 8 hours after subcutaneous injection and it can be detected in the plasma for up to 15 hours. Multiple studies, both *in vitro* and *in vivo*, have shown that, in general, exendin-4 has the same physiologic effects as GLP-1 in the pancreas, GI tract, and brain.

Exenatide is associated with improvement in some of the earliest and most fundamental abnormalities of type 2 diabetes: diminished “first-phase insulin response” and proinsulin/insulin ratio. Acute administration of exenatide in type 2 diabetic patients corrects the abnormal insulin secretion pattern after an IV glucose bolus (first phase and second phase insulin responses) and restores the ability of beta cells to respond to rapid changes in blood glucose concentrations. Exenatide also improves proinsulin/insulin ratio after 30 weeks of treatment.

Exenatide has been shown to be as effective as insulin glargine in the treatment of DM but with less side effects (e.g. hypoglycemia and weight gain). In a 2-year follow-up of patients receiving exenatide, patients achieved sustained and significant reductions in glycosylated hemoglobin, accompanied by significant weight loss (instead of weight gain commonly seen in diabetics receiving insulin) and improvement in serum liver enzyme activity and blood pressure. Most importantly, treatment with exenatide improved beta cell function as measured by homeostasis model assessment of beta cell function (HOMA-B). Exenatide has minimal side effects in people. It is mostly associated with nausea and less frequently with vomiting. Infrequently, it might cause hypoglycemia. Severe hypoglycemia (requiring assistance) was reported rarely (only 5 of 2781 patients) and only in patients who also received sulfonylurea drugs. Antibodies to exenatide developed in 67% of patients but this did not affect outcome and was not associated with side effects.

When first discovered, exendin-4 was shown to potentiate amylase release from rat acinar pancreatic cells in response to other hormones such as cholecystokinin. This was shown *ex vivo* and in high doses. A possible association between GLP-1 analogs and pancreatitis as well as medullary thyroid cancer has been suggested and lead to issuing of a warning by FDA. However, multiple studies have refuted these concerns and it is widely accepted that the proven enormous benefits of GLP-1 analogs outweigh its hypothetical risks.

Exenatide is commercially available in the USA under the trade name Byetta®.

**Exenatide in cats**

In healthy cats, exenatide was quickly absorbed after a SQ injection and caused glucose-dependent insulin secretion. Increased glucose tolerance, however, was not observed after a single SQ injection. At a dose of 1.0 mcg/kg SQ (about 10 times the dose that is used in diabetic people), exenatide injection did not cause any side effects in healthy cats, except for hypoglycemia in 1 out of 9 cats. Exenatide has led to significant weight loss in healthy cats of 7.0 ± 4.9% (from 4.78 ± 1.5 kg to 4.48 ± 1.5 kg) with a dose of 1.0 mcg/kg SQ BID for 28 days.

**Exenatide extended-release**

A long-acting sustained-release formulation of exenatide (Bydureon®) has recently been approved by the FDA as the first once-weekly subcutaneous injection for treatment of type 2 diabetes people. It consists of injectable microspheres of exenatide and poly(D,L-lactic-co-glycolic acid), a common biodegradable medical polymer with established use in absorbable sutures and extended-release pharmaceuticals, that allows gradual drug delivery at controlled rates. In people, Exenatide plasma concentrations are sustained at an effective concentration (50 pg/mL) for longer than 60 days after a single injection at doses of 5mg, 7mg or 10mg.

It has been shown in a recent clinical study to be more effective than once-a-day insulin glargine in achieving glycemic control with decreased risk of hypoglycemia and with reduction (instead of gain) in body weight. This extended release formulation was also
more effective than regular exenatide (Byetta) in achieving glycemic control with no increased risk of hypoglycemia, decreased side effects like nausea, and with similar reductions in body weight.23

Exenatide extended-release in cats
We have studied Exenatide Extended-Release in healthy cats at a dose of 0.13 mg/kg. Three weeks after a single subcutaneous injection fasting BG was decreased and during a hyperglycemic clamp at that time glucose tolerance improved, insulin concentrations increased and glucagon concentrations decreased. No side effects were observed throughout the study.24 Further evaluation is needed to determine its efficacy and duration of action in diabetic cats.

Liraglutide
Liraglutide is a GLP-1 synthetic analog with 2 amino acid substitutions and a fatty acid acyl group that enables noncovalent binding to albumin, thereby extending the pharmacokinetic profile of the GLP-1 molecule. Liraglutide exhibits a prolonged pharmacokinetic profile after a single injection, and exhibits all of the actions of native GLP-1.4 Liraglutide (once-a-day) was recently compared to exenatide (twice-a-day). Liraglutide provided significantly greater improvements in glycemic control than did exenatide and was generally better tolerated.25 Liraglutide once-daily was also more effective than Exenatide-ER once-weekly in achieving glycemic controlling but nausea, vomiting and diarrhea occurred less frequently with Exenatide-ER.26 Liraglutide has been used successfully to treat obesity in non-diabetic patients.27

Liraglutide is commercially available in the USA and Europe under the trade name Victoza®.

Liraglutide in cats
We have studied liraglutide in healthy cats at a dose of 0.6 mg/cat once daily for 7 days. Liraglutide caused significant weight loss in all cats at day (9% ± 3). Appetite was subjectively decreased in all cats and one cat was withdrawn on day 4 because of 48 hours of anorexia. During a hyperglycemic clamp, liraglutide was associated with a trend towards improved glucose tolerance, higher insulin concentrations and lower glucagon concentrations. Fasting glucose concentrations were not affected.28

Albiglutide
Albiglutide is a recombinant GLP-1-albumin fusion protein that exhibits a reduced affinity for the GLP-1R, but displays a broad spectrum of GLP-1R-dependent actions in preclinical studies, including inhibition of food intake and gastric emptying and reduction of glycemia excursion after meal ingestion. Importantly, it has the potential to be used as a very long-acting drug: It has been investigated in people for use as a weekly, biweekly and monthly injection.29 Albiglutide was approved by FDA in 2014.

DPP-4 inhibitors
DPP-4 inhibitors (e.g. sitagliptin, vildagliptin) are administered orally. In people, vildagliptin and sitagliptin are well-tolerated and not associated with hypoglycemia when used alone. Both agents increase plasma concentrations of GLP-1 and GIP after meal ingestion, enhance glucose-stimulated insulin secretion and reduce ratios of proinsulin:insulin, consistent with an improvement in beta cell function. They are, however, less potent than other oral hypoglycemic drugs.3 In contrast to GLP-1 analogs, DPP-4 inhibitors are not associated with nausea or vomiting in people, but they are associated with weight gain. DPP-4 inhibitors are also associated with increased risk of nasopharyngitis, urinary tract infections and headaches.30 Increased risk of infections might be related to the action of DPP-4 in T-cells as a co-stimulatory molecule (CD26).

A DPP-4 inhibitor has been used experimentally in cats and was effective in enhancing insulin secretion and inhibiting glucagon secretion after an intravenous glucose challenge. Glucagon inhibition was also observed after a meal challenge.30 In that study, the drug was administered subcutaneously and not orally.

References
Advances in Neonatal Abdominal Ultrasound
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Gastrointestinal disease is one of the most common problems neonatal foals face during the first weeks of life. The most frequently encountered problem in this age group is contagious gastroenteritis which has been well described. Less well understood and less common is the gastrointestinal (GI) dysfunction which often accompanies neonatal encephalopathy (also referred to as hypoxic ischemic encephalopathy, asphyxia syndrome) and neonatal nephropathy during the first days of a foal’s life. This triad of neurologic, renal and GI syndromes often with signs of sepsis is the most common complaint of foals less than 48 hours old admitted to our Neonatal Intensive Care Unit. The GI component has a wide range of clinical signs and severity form very mild dysmotility to severe necrotizing disease. The necrotizing disease we see has some similarities (and some differences) to the disease well recognized in human infants called Necrotizing Enterocolitis (NEC). This talk will describe our experience with two recent studies which have brought attention to the occurrence of necrotizing gastrointestinal (GI) disease and asymptomatic intussusceptions in neonatal foals.

The relevance and association of clinical and sonographic findings in neonates with gastrointestinal disease will be discussed. During this session we will not attempt to review the technique, normal or abnormal findings in abdominal sonograms in foals. However, we want to point out 2 technical notes. The sonographic examination of the neonate with abdominal disease can be done with the foal standing, in lateral or sternal recumbency. It is important to realize that lesions tend to localize in the most dependent part of the abdomen and that these lesions can be mobile. Therefore it is important to scan carefully the gravity dependent area. This implies to place the probe under the foal to scan the ventral abdomen if the foal is sternal, to scan the left side if the foal is in left lateral recumbency and the right side if the foal is in right lateral recumbency. The author finds microconvex probes to be the most useful probes. These probes typically have a frequency of 5-11 MHz. The frequency varies depending on the equipment used. To assess carefully the echogenicity of the wall, the wall layering or to be precise when measuring the thickness high frequency linear probes (usually 8-15 MHz) are required. Soaking the hair with alcohol is, in most foals, enough to obtain adequate images for ‘fluid checks’ or to detect severe abnormalities. To obtain detailed images clipping may be necessary. Clipping and using gel reduces the scanning time although it increases the preparation time.

Abdominal ultrasonography has been used for years as a valuable diagnostic aid and has become routine in the assessment of foals with critical gastrointestinal disease, however information about its usefulness, accuracy and limitations is not available. Abdominal ultrasound examinations are performed commonly by non-radiologists in human and veterinary intensive care units. In human critical care units, the time from presentation to operative care, length of hospitalization, complications and cost decrease when emergency abdominal sonograms are performed, potentially due to a more rapid diagnosis.

Intestinal wall layering and color doppler
The characterization of the layering of the GI wall has received little attention in equine medicine. There are specific case reports that have shown that this has potential increase the precision of diagnosticians. Sonographic to pathologic correlation has been demonstrated for several disease processes including pediatric colitis and GI emergencies. In these cases the careful assessment of the echogenicity and layering patterns can give information about potential specific diagnoses often helping patient management. Of particular interest to the diagnosis of GI disease in human neonates was the study by Baud et al. In this study different patterns of abnormal intestinal echogenicity, based on the loss/maintenance of stratification and folding helped in obtaining a precise diagnosis. In our opinion the wide use of high quality portable or hospital based ultrasound machines makes possible the assessment of intestinal wall layering. We performed a study to define the normal layering pattern of the GI of normal neonates. Equine neonates showed distinct layering of the stomach and large intestine, and indistinct layering of the small intestine. The reason why the layering of the stomach and large intestine was distinct, but not in the small intestine, is uncertain. It is known that rapid changes in the intestinal wall occur in the first days of life, including marked changes in total and relative thickness of the different histologic layers with the muscularis layer changing quantitatively the most in this period. We believe that the evaluation of the changes in layering patterns could help clinicians be more precise about diagnosis in neonates with GI disease and hopefully help in the management of these cases. Multicenter studies are needed to prove this and to investigate the association of different sonographic patterns with specific diagnoses in foals.

Sonographic evaluation of GI blood supply using color flow Doppler is well correlated with histology and outcome in human neonates with GI disease and allows the differentiation of focal from diffuse necrosis in the setting of NEC. Increased flow is reportedly obvious to the untrained observer. Color Doppler sonography was found to be more accurate than clinical examination and plain abdominal radiography in the prediction of necrosis in human neonates with NEC. The usefulness of this technique in equine medicine had not been evaluated. In our prospective study of normal neonates diagnostic color Doppler signals of mural blood flow could not be obtained. It is possible that different ultrasound equipment, different settings, sedating foals to decrease motion artifacts,
or the use of this technique in foals with abnormal patterns might help in obtaining diagnostic color flow Doppler readings. The authors believe that this modality has potential in the evaluation of GI in horses but further work is needed before it this potential can be defined.

Necrotizing enterocolitis (NEC) and pneumatosis intestinalis

The recognition in our hospital population of hyperechoic echoes of gas within the gastrointestinal wall (pneumatosis intestinalis, PI) of foals hospitalized with GI or critical illness led us to retrospectively investigate this phenomenon within the context of all sonographic examinations performed in foals with GI disease. The presence of PI is considered virtually pathognomonic for NEC in human neonates with a compatible clinical picture.12-13 NEC is a disease process of the GI tract of human neonates that is marked by inflammation and bacterial invasion of the bowel wall. NEC occurs frequently in human newborn intensive care units;14-15 primarily in premature neonates16-17 and had been described in foals in isolated case reports.18-21 We have the clinical impression, and several texts have also suggested, that this may be an under recognized syndrome.22-25 Based on our retrospective study we concluded that: 1-PI was diagnosed frequently in severe cases. 2-Foals with PI have a worse prognosis than foals without PI. 3- Variables associated with the presence of necrotizing GI disease were: prematurity, blood in the feces, gastric reflux, abdominal distention, abnormal colon echogenicity and leucopenia.

Several similarities exist between human NEC and GI disease in equine neonates with a necrotizing component resulting in gas in the wall of the intestinal tract. We chose to describe this group of foals as having a necrotizing component of the GI and avoid call this process NEC as the pathogenesis may differ. This nomenclature has been used in these proceedings and referring to NEC we refer to the GI disease in human babies.

It is important to realize that PI is not a diagnosis or a clinical problem per se, but a pathological or imaging finding that is a consequence of the underlying disease.15 PI has been attributed to at least 58 causes in mammalian species but it is virtually pathognomonic of NEC in human neonates with compatible clinical presentation.13 Therefore, and as for all imaging findings, PI has to be interpreted in the context of the clinical picture. The process of gas production and accumulation within the intestinal wall is poorly understood. The composition of the gas in human neonates is variable but it is often 30% hydrogen.11 Three possibilities have been proposed as the source of the gas: (1) intraluminal GI gas that migrates due to injury, (2) bacterial production of gas due to bacterial invasion, and (3) pulmonary gas. The pattern or extent of PI does not correlate with the severity of the signs or the severity of the underlying diseases in human neonates and the presence of intramural gas can even precede the presence of any clinical signs.28

Epidemiology

An understanding of the epidemiological aspects of NEC may help to place into context our clinical observations of necrotizing gastrointestinal disease in foals. The precise etiology of NEC, which is an ischemic and inflammatory necrosis of bowel,29 is unknown but three aspects are felt to be major risk factors for NEC to occur in human neonates: ischaemic/hypoxic mucosal injury, feeding and the presence of bacteria. Prematurity is the only independent determinant of human NEC. 90–95% of cases occur in infants born before 36 weeks of gestational age and the incidence varies inversely with birth weight and gestational age. Those most susceptible appear to be infants weighing less than 1000 g at birth and under 28 weeks’ gestation.16 Less than 10% of NEC cases occur in term infants.17 This close correlation with prematurity does not occur in foals in our experience as almost half the foals (46.2%) we identified were not premature. However, in our retrospective study, there was a difference (P = 0.03, OR 5.1) in the presence of necrotizing disease between premature foals with GI disease (53.8% of premature foals) and term (18.75%) foals with GI disease. In infants the clinical signs usually start between day 3-10 but NEC can be diagnosed up 90 days of age.11 In our clinical experience delayed onset beyond the first week does not occur in foals. Approximately 25–33% of all infants diagnoses with NEC die and 27–63% require surgical intervention.16 Only 33% of our cases with necrotizing disease survived but this figure includes fatalities because of economic concerns as well as humane considerations and so direct comparison might be misleading. 17/85 of foals that had abdominal sonograms due to signs of GI disease were diagnoses as having necrotizing GI disease. This frequency seems subjectively more than what is seen in other veterinary hospitals. This could be a reflection of regional characteristics, the type of cases that present to different hospitals or the specific concern of clinicians about these particular problems. Foals with PI have a worse prognosis than foals without PI. However, it is important to realize that many foals in which PI was diagnosed survived and foals with PI were likely to have more severe disease which introduces the confounders of financial limitations and humane perceptions. The survival rate of foals for which PI was observed sonographically was 37% vs. 72% in foals in which PI was not observed. When comparing the disease seen in foals to that seen in infants, the equine disease may be more similar to NEC seen in full term infants which in general has an earlier onset, shorter course and is less severe (less frequent perforations) and has been speculated to have a different pathogenesis.30-32

Clinical presentation

Clinical manifestations of NEC in infants can be specific to the GI tract (such as feeding intolerance, vomit, blood in the feces, abdominal distention, painful or discolored abdominal wall) and others can be vague: apnea, hypoxemia, bradycardia, lethargy, temperature instability, blood pressure instability, erytema or edema. In many cases NEC may be indistinguishable from sepsis.11,15
Laboratory values of babies with NEC are often the reflection of infection, activated coagulation and fluid retention. Leukopenia or leukocytosis, anemia, thrombocytopenia, hypo or hyperglycemia, electrolyte abnormalities and metabolic acidosis are common in this population. Bacteremia is reported in approximately 35% of cases of human NEC. Variables associated with the presence of necrotizing GI disease in foals are: prematurity, blood in the feces, gastric reflux, abdominal distension, abnormal colon echogenicity and leukocoria showing some parallelism between NEC and necrotizing GI disease in equine neonates. Bacteremia was observed in 55% of foals with necrotizing GI disease. This was not statistically different to the group of foals with GI disease without a necrotizing component in which 49% were bacteremic. In general the clinical picture seen in foals with necrotizing GI disease is consistent with sepsis perhaps reflecting that the final common pathway of intestinal injury is from the activation of the inflammatory cascade as has been speculated in infants.

Diagnosis
The diagnosis of NEC can be challenging and it seems that it is often made based on clinical and imaging findings. Classically radiographs showing PI, a thickened intestinal wall, free air in the abdomen (pneumoperitoneum), and portal venous gas were the basis of the diagnosis. The imaging findings (radiography, ultrasonography and computed tomography) have been described. Radiographic PI has been described in equine neonates. A recent review in infants described 11 sonographic features of NEC: 1. Increased intestinal echogenicity reflecting wall edema, inflammation or hemorrhage, 2. Intestinal thickening (2.7 mm or more), 3. Intestinal thinning (1 mm or less), 4. Increased intestinal perfusion, 5. Absent bowel perfusion, 6. PI, 7. Portal venous gas, 8. Free gas, 9. Anechoic free intraperitoneal fluid, 10. Free intraperitoneal fluid with echoes, and 11. Focal fluid collections. In our group of foals the presence of PI was part of the definition and apart from this finding only the presence of colonic thickening was different between the group with and the group without necrotizing disease. PI was most frequently imaged as scattered hyperechoic foci within the intestinal wall, although large clusters of hyperechoic foci within the bowel wall were also imaged in some foals. The intramural gas was imaged more frequently in the small intestine (58%) than in the large intestine (32%) and infrequently (11%) in both. The presence of increased peritoneal fluid was somewhat common (25%) but the presence of focal accumulations were rare (5%). The presence of hypo or dysmotility is also frequent in equine neonates with necrotizing GI disease. In our retrospective study only sonography was used. Comparisons with radiography would be interesting. Our clinical impression is that sonograms tend to be more informative when evaluating foals with GI disease. When compared to CT, sonograms have the advantage of being stall side and less expensive. We defined necrotizing disease as the presence of signs of GI disease (colic, diarrhea, gastric reflux or abdominal distension) and sonographic evidence of PI or pathologic presence of necrosis. In foals there are no reliable clinical signs that correlate with the presence of necrotizing GI lesions. Even dramatic signs such as hemorrhagic or coffee ground reflux or hemorrhagic or melanotic diarrhea may occur without any gross or histologic lesions. The major sequela in foals surviving with necrotizing lesions is segmental strictures which can be found in the small intestine, large colon or small colon. These strictures respond well to surgical resection.

Treatment
Medical treatment of NEC in infants typically consists of GI rest and decompression, broad spectrum antibiotic therapy, supportive care to manage electrolyte or acid base imbalances and parenteral nutrition. Although full thickness necrosis and secondary peritonitis may occur in affected foals, unlike infants, perforations are not seen in our experience. We have had anecdotal success treating foals at risk of or recovering from necrotizing disease with trophic feeding initially using fresh or frozen colostrum (even in foals up to 20 days old) or fresh mare’s milk and with judicious gradual volume increases dictated by feeding tolerance. The prevention of NEC is a complex matter. Avoiding preterm birth, judicious use of antenatal steroids in preterm deliveries, breast-milk feedings (vs. formula) and trophic feedings have been suggested as potential strategies to reduce the incidence of NEC in human neonates. The prophylactic use of oral antibiotics or the use of probiotics have received attention but its general use has not been adopted for reasons such as the emergence of antibacterial-resistant organisms or the questionable efficacy of probiotics. It’s been hypothesized that NEC can be a contagious disease and in some reports clusters of cases have occurred. Klebsiella, E. coli, Enterobacter, Salmonella, Pseudomonan, Clostridia, Staphylococci, coronavirus, rotavirus and enterovirus have been implicated as potential causative organisms. There are reports associating C. difficile, C. perfringens and Rhodococcus equi with necrotizing GI disease in foals. We feel that the necrotizing lesions in these reports are a direct result of bacterial pathogenic properties whereas the necrotizing disease we are reporting occurs as a primary problem which may then predispose the foal to secondary pathogenic colonization. In the group of foals that we reported blood cultures, PCR’s for detection of Salmonella in feces and fecal testing for the detection of clostridial toxins were performed frequently and a positive result was not associated with a necrotizing component to the gastrointestinal disease. In fact the percentage of foals positive to clostridial toxin detection analysis was lower (not statistically significantly higher) in foals with necrotizing GI disease.

Asymptomatic intussusceptions
As a part of our investigations in equine gastrointestinal disease we designed a prospective study in which normal neonatal standardbreds were scanned. Incidentally asymptomatic intussusceptions were found in 10/18 neonates. Finding jejuno-jejunal intussusceptions in these normal foals was unexpected, as asymptomatic intussusceptions had not been previously reported in horses.
Jejuno-jejunal intussusceptions are the most common type of intussusception in foals with abdominal pain, but intussusceptions are reported to uniformly require surgical correction in horses.\textsuperscript{36-38} Dysrhythmic peristaltic activity has been proposed as the cause of intussusception in horses and people. Sonographic signs that have been associated with transient, nonclinical small intestinal intussusceptions in small animals and infants are the absence of identifiable intestinal lesions, normal wall thickness, length of less than 3.5 cm, normal undilated proximal bowel, normal vascularity on color Doppler, respected layering, and the intussusception being compressible.\textsuperscript{40,41} We did not recognize pathologic lead points, increased wall thickness, or altered echogenicity in any of the asymptomatic intussusceptions we observed. Compressibility, length or mesenteric, and mural blood flow in the area of the intussusceptions were not evaluated. The wall thickness of the intussusciptens was 0.1–0.2 mm thinner than the lower end of the reference range for normal jejunal wall thickness in 4 cases and the average wall thickness of the intussusciptens was less than the average jejunal wall thickness. Clinical intussusceptions in humans\textsuperscript{42} are characterized by an increased thickness of both intussuscipiens and intussusciptens, and the marginal thinning observed in foals with asymptomatic intussusceptions is of uncertain relevance. It is possible that asymptomatic intussusceptions are a normal occurrence in foals of this age range because of initial development of gastrointestinal function and motility. This unexpected finding helped us realize that the finding of intussusceptions in equine neonates should not be considered an absolute indication for surgery and that the clinical, clinicopathologic and rest of imaging findings should be used to decide the course of therapy. Recheck sonograms may be useful in this subset of foals.

References
ECG interpretation

The ‘seven questions of the equine ECG’ is an example of a clinical approach to evaluation of ECGs. The goal of this simplified approach is to get information that will allow you to make clinical decisions in horses. It is an oversimplification in some aspects but helps to diagnose common arrhythmias by understanding the mechanisms that cause the rhythm alteration. You will often be able to obtain a rhythm diagnosis by ‘eyeballing’ the ECG and without answering these questions. I would recommend that you use a protocol. This will build up your reading skills and will decrease the number of misdiagnoses. This is very similar to what you have learnt for ECG interpretation in dogs and cats. Mean electrical axis calculations have little to no utility in equine electrocardiography due to the explosive nature of the conduction system causing cancellation of electrical vectors.

What is the heart rate?

The normal heart rate of a horse is 28-42/min. This breaks down rhythms in the ones that are fast or tachycardic (common tachycardias are sinus tachycardia, ventricular tachycardia or supraventricular tachycardia), slow or bradycardic (the common ones are rhythms with frequent AV block, sinus arrests or pauses) and the ones that have a normal rate (for example normal sinus, sinus arrhythmia or atrial fibrillation). Note that this is the second big difference (after the lack of use of the mean electrical axis) that we have mentioned between ECGs in horses and most other species. Atrial fibrillation occurs, most of the times, at normal heart rates in horses and without the presence of underlying disease ( lone atrial fibrillation). If the heart rate during atrial fibrillation is high you should suspect underlying systemic or cardiac disease predisposing to the arrhythmia and the tachycardia.

Sick sinus syndrome is rare in horses but the principles that you learnt for small animals apply to horses. Very long pauses are often followed by scape rhythms or tachycardias. Horses with sick sinus syndrome can collapse and the implications of collapse in horses are very different than in a small animals, particularly from the stand point of human safety.

If you are using a printed ECG you can calculate the HR by extrapolation from the paper speed. 25mm/sec is the most common paper speed. 60 divided the RR interval (in seconds) will give you the heart rate (ventricular rate) for a particular beat (this is called the instantaneous heart rate). If you do the same using the interval between two consecutive P waves you can calculate the atrial rate. In an ECG trace each of the smallest squares is 1 mm. At 25mm/sec each of the little squares is therefore 0.04secs (1sec/25mm=0.04sec/mm). A large square (5 small squares) is 0.2 secs and 25 small squares (5 large squares) is 1 second. A standard old fashioned pen measures approximately 15 cm. This is 6 secs assuming 25 mm/sec paper speed (6x25mm=150mm=15cm). If you put a pen on a 25mm/sec ECG trace and count the RR intervals that fall under a pen and add a zero after that number this approximates the HR. Remember that if you have scanned copies of an ECG there may be magnified or reduced in size so the ‘pen rule’ may not work. When using digital systems sometimes the size of the squares (especially in old systems) will not be adjusted to the paper speed so you may need to use the electronic calipers in the computer. For most digital systems a screenshot at 25mm/sec is 15 or 16 secs so if count the RR intervals in a computer screen and multiply by 4 you get an approximate heart rate.

Is there a QRS for every P?

If the answer is NO, some or all the P waves are not conducted to the ventricles. Common causes of this in horses are 2nd and 3rd degree AV block, ventricular tachycardia or non-conducted APCs.

In the case of second degree AV block some P waves are conducted and others are not. They are blocked at the level of the AV node. Second degree AV block is very common and most of the times is physiologic in horses due to their intrinsically high parasympathetic tone. The 2nd degree AV block should disappear with exercise or excitement. If it does not, it is concerning. Often trotting the horse up and down or exposing it to some situation that would create excitement is enough. Note that the AV block can come back very quickly after the exercise so in questionable situations you may need to record the rhythm while the horse is exercising. Sinus arrest (or pause) is also caused by high vagal tone, also common, and also should go away with exercise or excitement. In this case it is the SA node activity stops transiently and therefore the next P wave is delayed. If the pause is very long a scape beat can occur but this is rare in horses. If the pause is a multiple of the RR interval it is called sinus block. If it is longer than two RR intervals is called sinus arrest.

Figure: Example of second degree AV block-
This is a base apex ECG and the paper speed is 25mm/s
ectopic beat this is trigemini. Couplets, triplets, bigeminy and trigeminy are somewhat more concerning than single VPCs. Ventricular tachycardia is one of the most common rhythms requiring antiarrhythmic therapy in horses but not all horses with VT waves are buried in the QRS complexes or T waves and not visible. Increasing the paper speed and increasing the gain may help you follow a sinus beat, followed by an ectopic beat and so on are called bigeminy. If the series are of two sinus beats and one need antiarrhythmic. We will talk later about how to decide.

Are all QRS complexes normal and of equal morphology? If the answer is NO, likely there is a ventricular focus for the abnormal QRSs. Aberrant ventricular conduction will change the shape of the QRS complex but this is rare in horses (except in atrial fibrillation during exercise or at high rates but forget this for now). Assume that a QRS complex with a morphology different than the one from the SA node is of ventricular origin.

**Ventricular premature complexes (VPCs)**

In the case of a ventricular premature complex the QRS will come earlier than expected (has a high instantaneous heart rate). Most people will consider a complex premature if the RR interval is ≥20% earlier than the previous one. The number is somewhat arbitrary but it means that if you calculate all instantaneous hear rates the one from the premature beats will be ≥20% faster than the sinus beats. There is no P for this QRS and often the following P will not have a QRS as the ventricles will ‘be caught’ in a refractory period. This will cause a prolongation of the R-R interval between the ectopic QRS and the following sinus complex called a ‘compensatory pause’. This is a somewhat confusing nomenclature and almost a misnomer as ‘no one is compensating’, the SA node its doing its job, the ventricles are ‘misfiring’ and they are dissociated. But think about it this way: the word compensatory refers to the fact that the prolonged RR interval (the pause) compensates for the shortened RR interval of the premature beat. In some occasions the P wave after a VPC is conducted and there is no compensatory pause. In this cases the VPC is called interpolated. See the example below.

The QRS is of different morphology than the sinus QRSs and the morphology will be often ‘wide and bizarre’. This is often the case but keep in mind that the closer the ectopic focus is to the normal conduction system the less ‘wide and bizarre’ the ventricular complex will be. In other words, in horses some ventricular complexes are not wide and bizarre and just different to the normal QRS complexes. Sometimes one lead will not clearly reflect the ectopic origin, this means that in a particular lead the ectopic QRS may be similar to the sinus QRS. This is one of the reasons why it useful to have more than one lead. If there is more than one abnormal QRS morphology the rhythm is called multifocal or polymorphic, i.e. there are more than one origins (foci) for the VPCs and therefore they have different shapes (morphologies).

**Ventricular tachycardia (VT)**

4 or more VPCs in a row is defined as ventricular tachycardia. It is important to remember that there are P waves during ventricular tachycardia. The key is that this P waves are not associated with the QRS complexes. Ventricular tachycardia is a dissociated rhythm and the SA node will keep working ‘at its own pace’ (and generate P waves) while the ventricles work at a different (ectopic) rhythm and generate QRS complexes. The P waves are not conducted to the ventricles because the ventricular rhythm is faster and the ventricles are refractory when the P wave goes through the AV node. Occasionally the P waves go through the AV node when the ventricles are not refractory and will be conducted. This is called a capture beat. Sometimes the capture beat and the ectopic beat happen at the same time and the QRS becomes a combination of the sinus and the ectopic QRS. This is called a fusion beat. It is a very common misunderstanding to think that during VT there are no P waves. It is important to try to identify the P waves. Often the P waves are buried in the QRS complexes or T waves and not visible. Increasing the paper speed and increasing the gain may help you identify P waves. As the sinus rhythm is most of the times regular using calipers can also help you find the hidden or buried P waves. Ventricular tachycardia is one of the most common rhythms requiring antiarrhythmic therapy in horses but not all horses with VT need antiarrhythmic. We will talk later about how to decide.

Two VPCs in in a row is a couplet and three in a row a triplet. Series in which there are one sinus beat followed by an ectopic beat, followed by a sinus beat, followed by an ectopic beat and so on are called bigeminy. If the series are of two sinus beats and one ectopic beat this is trigemini. Couplets, triplets, bigeminy and trigeminy are somewhat more concerning than single VPCs. Ventricular premature complexes/ventricular tachycardia and idioventricular beats/idioventricular rhythms are to names used to characterize for
ventricular ectopy according to their rate. A rhythm is called idioventricular if the rate is similar to the prevailing sinus rate. The definition is somewhat non-specific. Idioventricular rhythms are typically more benign than ventricular tachycardias.

**Torsades the pointes, torsades or torsades-like VT**

This is particular type of polymorphic ventricular tachycardia that gives the illusion of a twisting (torsades) of the QRS complex around the isoelectric baseline. The QRS peaks first pointing up, then get smaller, then point down… This is a very unstable electrical system that can lead to sudden death. If you detect this rhythm go and get antiarrhythmics fast. Magnesium sulfate is the first choice antiarrhythmic for torsades in horses.

**Are all P waves of normal and same morphology?**

If the answer is NO there is a supraventricular premature complex (SVPCs), supraventricular tachycardia (SVT) or a wandering pacemaker.

**Supraventricular premature complexes (SVPC)**

In the case of a supraventricular premature complexes the P wave will come earlier than expected (has a high instantaneous heart rate and a short P-P interval). Most people will consider a complex premature if the P-P interval is $\geq 20\%$ earlier than the previous one at rest (the number is somewhat arbitrary). There is a P for this QRS and often the P is of abnormal morphology although not always (see example above). There are three versions of a SVPCs depending on how premature the complex is but no real difference in their clinical implications.

- **Conducted = Not very premature**
  - The P wave will be conducted normally and therefore the QRS will have normal morphology. There is no compensatory pause as the SA node will be reset by the ectopic P wave. In other words the following P-P and R-R interval will be normal. This is the common type.

- **Non-conducted = Very premature**
  - The P wave is so early that the ventricles are still refractory. The premature P wave will not be conducted to the ventricles and therefore will not be followed by a QRS complex. This is called a ‘non-conducted SVPC. It is common for electrocardiography students to want to call non-conducted SVPCs 2nd degree AV block. It looks similar to 2nd degree AV block but the difference is that in this case the P wave is premature. They have different implications.

- **Aberrantly conducted = Somewhat in between**
  - The ventricles are only partially repolarized and therefore the conduction through the ventricles is somewhat abnormal (aberrant). This causes the QRS complexes to have abnormal morphology. They are called SVPCs with aberrant conduction. They have different QRS morphologies when compared to sinus complexes similarly to VPCs. They are, however associated to P waves and they are not commonly as ‘wide and bizarre’.

  Four or more SVPCs in a row is called supraventricular tachycardia. Two in in a row is a couplet and three in a row a triplet. Series in which there are one sinus beat followed by an ectopic beat, followed by a sinus beat, followed by an ectopic beat and so on are called bigeminy. If the series are of two sinus beats and one ectopic beat this is trigemini. Couplets, triplets, bigeminy and trigemini are somewhat more concerning than single SVPCs.

**Is the R-R interval regular?**

This will help you break rhythms into regular rhythms, regularly irregular rhythms and irregularly irregular rhythms. The concept of regularly irregular or irregularly irregular if often misunderstood. Let’s break it down: the second word just refers to the presence or absence of an arrhythmia: regular rhythm means no arrhythmia vs. irregular rhythm (regularly irregular or irregularly irregular) means an arrhythmia is present. The first word qualifies the arrhythmia by describing the underlying rhythm: is there an underlying pattern to the rhythm? Common regular rhythms are normal sinus, sinus tachycardia…. Common regularly irregular rhythms (arrhythmias with an underlying regularity or pattern) are blocks, supraventricular or ventricular tachycardia and common irregularly irregular rhythms are such as atrial fibrillation or sinus arrhythmias. The most common misdiagnosis is equine electrocardiography is to diagnose atrial fibrillation at a fast rate (an irregularly irregular rhythm) as ventricular tachycardia (a regularly irregular rhythm). An irregularly irregular rhythm on a horse is atrial fibrillation until proven otherwise. This is the most common clinically relevant arrhythmia of horses. The other common irregularly irregular rhythm is sinus arrhythmia. Sinus arrhythmia often comes and goes and is vagally mediated. As the other vagally mediated rhythms we talked about before (2nd degree AV block, sinus block or arrest) it will disappear with exercise or excitement and has no clinical consequences. Sinus arrhythmia is not associated with respiration in horses. Atrial fibrillation will not disappear with exercise or excitement. Other characteristics of atrial fibrillation are the lack of an isoelectric line and the presence of ‘f’ waves. Be careful when the rate is fast as the ‘f’ waves may be difficult to see.

**Figure. Example of a base apex ECG showing atrial fibrillation. Note the RR interval changes with each beat. The paper speed is 22mm/sec.**

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Figure: Base apex ECG showed atrial fibrillation at a higher rate. Note that the RR interval changes with each beat but this is harder to 'eye ball'. It is easy to notice if you use calipers like the ones in the picture. Note that due to the higher rate the 'f' waves are more difficult to see.

Are the PR interval, QRS duration and QT interval of normal duration?
The normal intervals in horses are: P-R= 0.2-0.5s, QRS <0.14s and Q-T ≤0.6s. PR defines the AV conduction. QRS defines ventricular depolarization and QT ventricular repolarization. If abnormal they can help you make sense of the rhythm disturbance. For example, a very short PR interval may be caused by accelerated conduction through an accessory atrioventricular pathway. A very long PR interval may be caused by prolonged conduction associated with AV node/conduction system disease. A prolonged QRS duration can be seen in VPCs or be drug induced (quinidine) etc. Electrolyte abnormalities (hypo or hyperkemia being the most common, but not only) will also change conduction rate and excitability.

It is likely that answering a single question will not give you a rhythm diagnosis but it is unlikely that after answering all 7 you will have one or a very short list of possibilities. Remember this is a set or simplified rules and there are exceptions to all rules.
Interpretation of Equine Murmurs: When Should I Worry?
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This document is written to support the presentation about how to interpret heart murmurs. The objective is to give an easy to follow mental chart useful to field practitioners. To a certain degree the chart is an oversimplification but provides rules that hold true in the majority of cases.

A murmur is a cardiac sound generated by turbulent blood flow. It is useful to think about murmurs from the perspective of the reason for the turbulent flow. There are three main broad categories, these are: regurgitations, stenosis and shunts. Another relevant group of murmurs in horses are the ‘physiologic = ejection = flow murmurs’. Physiopathologically it is useful to place these ones under the category of stenosis. These are not generated by stenosis but mainly caused by ejection of blood through a major vessel and therefore sound similar to pulmonic or aortic stenosis. These are very common murmurs in horses due to the large size of the heart and stroke volume. Physiologic murmurs are particularly common in athletic horses with a large heart and a thin thoracic wall. Physiologic murmurs tend to be more frequent in circumstances that predispose to a turbulent flow during ejection such as anemia, fever, excitement or the immediate postexercise period. Pathologic murmurs vary from asymptomatic and not affecting performance to causing exercise intolerance, heart failure or affecting safety of riders.

The timing of the murmur is the first key. We will pause for a second to make sure the timing of the cardiac cycle in relation to auscultation is clear. There are two loud sounds that are S1-S2. These are traditionally described as ‘lub-dup’. The pause or silence between lub (S1) and dup (S2) is systole. This is the short pause. The pause or silence between dup and the following dub is diastole and is the long pause. In a horse with a high heart rate, or a foal, it can be difficult to differentiate systole vs. diastole as diastole shortens with the increase in rate. In adults the easiest artery to reach is the facial at the level of the medial aspect of the mandible and in recumbent foals the median in the medial aspect of the elbow region. Being able to differentiate systole from diastole is critical to the rest of the explanation. For most experienced clinicians the difference becomes intuitive. If you have questions about systole vs diastole ‘exercising’ your ears at the same time that you think in your head ‘lub-dup’ and tap your foot following the heart rhythm is a good practice.

Murmurs are described based on 7 characteristics. This may sounds superfluous but gives relevant information to diagnosis, prognosis and follow up of these cases. Small animal cardiologists do not usually follow this classification. I feel that the reason is that it is often impossible in a small animal with a high heart rate. Fortunately for us it is much easier in a horse with a normal resting heart rate. Giving 7 adjectives to a heart murmur requires practice and purposeful auscultation but it is very useful.

1. **Grade**- Tells you how loud the murmur is: There are different scales. I am familiar with the following.
   - Grade 1/6 – soft, only audible in a quiet environment
   - Grade 2/6 – heard easily when placing stethoscope over point of maximal intensity.
   - Grade 3/6 – audible immediately when placing stethoscope over chest wall.
   - Grade 4/6 – loud murmur with a faint thrill.
   - Grade 5/6- loud with a strong thrill
   - Grade 6/6 – loud with a strong thrill and heart with the stethoscope away from the chest wall.
2. **Timing**
The most important characteristic. Divides murmurs in systolic, diastolic or continuous. See the description above.
3. **Duration**
   ‘Holo’ - between S1-S2 or viceversa (S1-S2 are audible). Pan- extends over S1 and S2 and these are not audible. If shorter than holo can be classified as early, mid, mid to late etc.
4. **Character**
Blowing, coarse or harsh, musical, honking.
5. **Shape**
Crescendo (louder at the end), decrescendo (less loud at the end), band shaped (as loud beginning, middle and end) o crescendo-decrescendo- gets louder and then less loud. The shape is characteristic more difficult to precise.
6. **Location or PMI (point of maximal intensity)**
It refers to the area where the murmur is loudest but not necessarily to the structure that is affected.
   - Mitral valve area: Left fifth intercostal space approximately half way between the shoulder and the sternum. The most common mistake of is to auscultate to low. It is the only PMI in which the head of the stethoscope is visible. For all others the head of the stethoscope is buried under the triceps muscle.
   - Aortic valve area- Left 4th ICS under lower than the point of the shoulder.
• Pulmonic valve area- Left 3rd ICS. This is very cranial. You need to have the horse point the leg forward or push very hard under the triceps. Hard enough that the horse will be bothered by you pushing. Once the horse becomes bothered you need to push harder. I believe many veterinarians have never placed the stethoscope over the pulmonic valve area.
• Tricuspid valve area- Right 3rd and fourth intercostal space midway between the shoulder and the sternum. Also the head of the stethoscope is buried under the triceps.

7. Radiation
Murmurs can radiate cranially caudally or across the chest. It is common that loud, musical or honking murmurs radiate left to right. In the case of murmurs that are less than a grade 3 and not musical if a murmur is heard in both sides of the chest it is more likely that there are two murmurs. A murmur radiates with the same shape and character.

An example of a description of a typical murmur of aortic regurgitation would be:
‘Grade 3/6 holodiastolic decrescendo musical murmur with the point of maximal intensity over the aortic valce and radiating cranially caudally and to the right side of the chest’.

Another important key for murmur interpretation is variation in intensity. There are two types of murmurs that classically vary in intensity: ejection murmurs and mitral valve prolapse. There are easily differentiated as prolapses are mid to late crescendo systolic and ejection murmurs are early to mid crescendo decrescendo systolic. Exercise, excitement, pain or sedation are common circumstances that would make this murmurs louder. Murmurs that vary a lot in intensity are usually benign.

Below are example of a diagnostic algorithms for diagnosis of murmurs in horses.
*CHD= congenital heart disease, AC= aortocardiac, PDA= patent ductus arteriosus.

**Diastolic murmur**
A diastolic murmur in a horse is aortic regurgitation until proven otherwise. Pulmonic regurgitation is commonly seen echocardiographically but it is rarely audible due to the low pressure in the right side of the heart. Pulmonic regurgitation is rarely clinically relevant. If you hear a diastolic murmur you have to assume aortic regurgitation is the reason and the next immediate step is to feel the peripheral pulses. If peripheral pulses are bounding the aortic regurgitation is likely moderate or severe. The grade of the murmur does not correlate with the severity of the disease but the character of the pulse does. Aortic regurgitation murmurs are almost always decrescendo and often musical or blowing and if musical tend to radiate widely

A particular type of murmur/physiologic sound is the 2 year old squeak. This is a 1-3/6 early diastolic squeak heard in the left or right side of the chest. It is more commonly heard in young horses with big hearts and more commonly in young TH horses; hence the name ‘2 year old squeak’. It can be heard, however, in horses of any age and breed. It is likely caused by ventricular stretching during rapid ventricular filling and does not have clinical relevance.

Many horses with aortic regurgitation will have successful athletic careers for years and normal life expectancy. Aortic valve degeneration is very common in teenage horses. It is recommended to do an echocardiogram with bounding pulses, young horses or horses with other signs of heart disease apart from the murmur. In other horses the decision to make an echocardiogram will vary depending on the circumstances. Horses with moderate or severe aortic regurgitation are predisposed to ventricular arrhythmias and an exercising ECG is indicated in these horses.

**Right sided systolic murmur**
A right sided systolic murmur (only right sided) is tricuspid regurgitation until proven otherwise. Careful auscultation over the pulmonic valve area needs to be performed to rule out the presence of a ventricular septal defect. The severity of tricuspid regurgitation is correlated with the grade of the murmur. It is very common in Standardbred racehorses. It rarely affects performance or life expectancy. If the right atrium becomes enlarged in can predispose to atrial fibrillation. In horses with a murmur louder than 3/6, pansystolic or musical or in horses with other signs of cardiac disease (poor performance, jugular pulses, edema, exercise intolerance, arrhythmias…) an echocardiogram is recommended. In horses with murmurs 3/6 the recommendation will vary depending on the occupation of the horses are circumstance but significant disease is unlikely.
**Bilateral murmurs/Congenital heart disease**

An echocardiogram is indicated in all horses with suspected congenital heart disease. Ventricular septal defects (VSD) are, by far, the most common congenital heart defects of horses. The most common combination of murmurs in congenital heart disease is a 4-6/6 pansystolic band shaped and coarse murmur with the point of maximal intensity over the tricuspid valve area and a murmur less loud than the one on the right that is holosystolic or pansystolic crescendo decrescendo and blowing or coarse with point of maximal intensity over the pulmonic valve area. This combination reflect the left to right shunt (right sided murmur) and the left sided murmur of ‘relative pulmonic stenosis’. The second murmur is caused by ejection of an increased amount of blood in the right ventricle due to the shunt. If the left sided murmur is louder than the right sided murmur complex congenital heart disease is suspected. Tetralogy of Fallot is the most common complex congenital heart disease of horses. It is a common misunderstanding to say that in horses with VSD ‘the murmur can be heard bilaterally. These are two murmurs caused by two different processes: shunt + relative pulmonic stenosis.

Horses with small VSDs can have normal life expectancy and sometimes normal athletic careers. The echocardiogram is critical in these horses. In horses with large VSD or complex congenital heart disease the life expectancy may be shortened. They can be exercise intolerant and they may not be safe to exercise.

**Left sided systolic murmur**

A loud left sided systolic murmur should be considered mitral regurgitation until proven otherwise.

- The grade is not correlated with the severity of the disease. The PMI can be the mitral or aortic valve areas.

  - **There are 3 main variations:**
    - Holo or pansystolic band shaped and coarse or blowing- it is the most common one.
    - Musical or honking- raises concerns about a ruptured chorda tendinae often associated with severe disease.
    - Mid to late crescendo systolic murmur- suggests a mitral valve prolapse. Mitral valve prolapse in horses is usually associated with mitral valve disease that progresses very slowly, if at all, over the years and good prognosis.

  - There is one exception to the rule above. If the murmur is ≤3/6 early to mid-systolic crescendo-decescendo blowing or coarse over the pulmonic or aortic valve area and variable with hemodynamic changes it is likely a physiologic ejection murmur. Particularly with exercise or excitement an ejection=flow=physiologic murmur is suspected.

  - If the murmur is ≥3/6 holosystolic crescendo-decreseceudo blowing or coarse and over the pulmonic valve are pulmonic stenosis or more commonly relative pulmonic stenosis (PS) associated with a VSD is suspected. Isolated PS is rare in horses. Aortic stenosis is almost unheard of in horses.

  - Mitral regurgitation is both commonly asymptomatic and the most common valvular disease associated with poor performance and shortened life expectancy in horses. In the scenario of a prepurchase examination further diagnostics (echocardiogram) is recommended in horses with left sided systolic murmurs that do not fit the description of an ejection murmur. Particularly if the murmur is 3-6, pansystolic or there are other signs of heart disease. Clinical signs that suggest the MR may have clinical relevance are exercise intolerance, increased resting heart rate, respiratory rate or effort or an irregularly irregular rhythm consistent with atrial fibrillation.

**Continuous murmurs**

Continuous murmurs are rare in horses. Patent ductus arteriosus are common in new born foals and should close during the first days of life. A continuous murmur in a foal older than 5 days old warrants investigation. Aortocardiac fistula is the most common, and almost only, differential diagnosis in and adult horse with continuous murmurs. Horses with aortocardiac fistulas are not safe to ride as they are predisposed to life threatening arrhythmias and collapse. All adult horses with continuous murmurs should have an echocardiogram.
Prepurchase Examinations and Interpreting Arrhythmias: When Should I Worry?
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Clinical decisions after rhythm diagnosis: black white and grey
There will be times when the decision of how to manage an arrhythmia will be black or white. Other times it will not be that easy are many factors apart from the rhythm will play a role.

White
Vagally mediated arrhythmias: 2nd degree AV block and sinus blocks, pauses or arrests that go away with exercise or excitement and sinus arrhythmias can be considered benign. Sick sinus syndrome or 3rd degree AV block are the pathologic and black version of these physiologic arrhythmias and are very rare but very concerning as can make the horse collapse. They will need therapies similar to the used in small animals and a consultation with a specialist.

Light grey
Rare or occasional SVPCs or unifocal VPCs do not have, in and of themselves, clinical consequences in the resting horse. They would rarely need antiarrhythmic therapy. Electrolyte abnormalities, systemic inflammation, systemic disease or underlying heart disease can predispose to these arrhythmias. So the arrhythmia may be a sign that something else is wrong with the horse that you may need to address. SVPCs are one of the triggers for AF but the risk is difficult to quantify. VPCs are somewhat more concerning than SVPCs but do not panic when you identify VPCs in an ECG strip. It is often said that VPCs should be interpreted considering the ‘company that they keep’. Meaning the rest of the evaluation of the horse, the heart and other ECG abnormalities are critical to make decision about how to manage horses with VPCs. Another layman’s way of explaining the significance of ventricular ectopic beats to an owner is that a few single VCPs are not dangerous but the concern is that they can ‘gang up and try to kill you’.

Depending on the clinical scenario you will need to decide if you can ignore this arrhythmias or you should investigate them further. Take home message: worry about why the arrhythmia is present and worry about the potential for worsening.

For example:
1. You are treating a horse for acute colitis (or post-operative colic, pneumonia…) and you detect SVPCs. It would be advisable to make sure that electrolytes and acid base status are normal and evaluate the severity of the inflammatory response (do a CBC for example).
2. You are auscultating a high level three day eventer prior to sedating it to float his teeth. You hear a loud left sided systolic murmur and some premature beats. It would be advisable to explain the owner that the loud murmur is likely the cause of mitral regurgitation and that the premature beats could be a sign that there is cardiac remodeling or enlargement due to the mitral valve disease. You could recommend doing an echocardiogram and a holter and exercising ECG to evaluate the underlying heart disease, prognosis and risk for progression.

Grey: Atrial fibrillation (AF)
The most common clinically relevant arrhythmia in the horse. You definitely need to take this arrhythmia into account for the management of the horse but atrial fibrillation is seldom an emergency. The first step is to assess electrolytes, acid base status and a potential of an inflammatory response similar to the explained above for premature complexes. The next question to decide if the horse should be converted to normal sinus rhythm (cardioverted). There are many considerations that play a role in this decision like: the duration of the arrhythmia, the presence of underlying cardiac disease, the type of exercise the horse does, the potential complications of the cardioversion, the cost of the evaluation and treatment etc. The decision is multifactorial and would be topic for a full instructional video. These are some facts about AF to keep in mind:
- Not all horses need to be converted to normal sinus rhythm.
- The success rate for treatment is in general terms high, approximately 85-95%.
- If you decide to treat the horse you have to main choices: antiarrhythmic drugs (most common choice is Quinidine) or transvenous electrical cardioversion. Both have pros and cons.
- For any cardioversion attempt the main complication is sudden death. This is very rare but should be mentioned.
- Paroxysmal atrial fibrillation may occur in young racehorses. It will often spontaneously cardiovert within 48 hours of fibrillation onset.
- AF causes decreased performance in high intensity athletes (racehorses, eventer, field hunters, elite jumpers) and is often unnoticed in pasture pets or low intensity athletes (dressage, show hunters, conformation…)
- Treatment may not be necessary in older horses performing up to expectations if exercising ECG is acceptable (rate and rhythm) or in ‘pasture pets’. Horses that exercise at high intensities will not be able to do successfully while on AF.
- Echocardiography is needed to assess cardiac function and the presence of underlying cardiac disease.
- Echocardiogram and exercising ECG are mandatory if a horse is going to be exercised while on AF.
Cardioversion is contraindicated if there is heart failure or severe underlying disease.
The recurrence rate varies between 25% and 100% depending on the situation.
The cost of evaluation and therapy is variable but can range from 1000-5000$.

**Dark grey: Ventricular tachycardia (VT)**

Ventricular tachycardia is a serious arrhythmia. Horses with ventricular tachycardia always need close monitoring, ideally with telemetry and often need antiarrhythmic therapy. However, not all horses with ventricular tachycardia need antiarrhythmic therapy. It is mandatory to assess electrolytes, acid base status and a potential of an inflammatory response similar to the explained above for premature complexes and atrial fibrillation.

There are 5 classic scenarios in which antiarrhythmic treatment is indicated for horses with VT:

1. Clinical signs of cardiovascular collapse (e.g. weakness, syncope…). This applies to all arrhythmias and in general terms to all medical problems. Look at the patient first.
2. Heart rate >100 beats/min. The number is somewhat arbitrary but good place to start.
3. Multifocal rhythms - Implies there is more widespread cardiac involvement. Also has prognostic implications.
4. R on T phenomena- This happens when the premature QRS is so early that ‘comes out’ of the previous T. Creates a very unstable electrical system that predisposes to more malignant arrhythmias (ventricular fibrillation) and sudden death.
   
   Figure: Base apex ECG of a horse with VT. Note that the 6th QRS complex ‘comes out’ of the 5th T. This is R on T phenomenon.
5. **Torsades de Pointes**- This is an emergency. Run and get the emergency drug box (or tell someone to do so) and put an IV catheter ASAP. Start pulling magnesium sulfate (25 g from a 500 kg horse given over 25 minutes) and lidocaine (0.5 mg/kg slow and repeat up to 3 times q 5 minutes). If the horse is standing be aware that it may collapse at any time so have unnecessary or untrained people out of the stall and everybody else ready to get out of the way.

The most common drugs to treat ventricular arrhythmias in horses are lidocaine and magnesium sulfate. Magnesium sulfate is the drug of choice for Torsades. Magnesium sulfate should only be used in horses with low or normal magnesium. Hypermagnesemia is very rare unless it is iatrogenic in origin.
Cardiac auscultation is often the first clue for the detection of valvular, arrhythmic or congenital disease that can affect performance, safety or life expectancy. The most common physical examination findings that alert clinicians about cardiovascular disease are murmurs and irregular rhythms. The importance of careful auscultation and description of the heart rhythm and murmurs cannot be overemphasized. In some occasions diagnostic aids such as electrocardiograms, echocardiograms or measurements or cardiac biomarkers will be useful to the equine clinician.

The electrocardiogram (ECG or EKG) is the recording of the electrical activity of the heart. This can be printed in a paper, displayed on a screen or stored digitally. Units that display the ECG in real time are called telemetry units and the 24-hour continuous ECG recording is called Holter. The base-apex lead is, however, the most commonly used and often gives you the information that you need. To obtain a base-apex ECG place the left arm electrode (LA=Left Arm, positive, usually yellow) behind the left elbow- this is the apex. The right arm electrode (RA=Right Arm, negative, usually red) in front of scapula and the ground electrode (RF=Right Foot, usually black) attached at a site remote from the heart. For example over the right jugular groove. If you have position the leads correctly (and the horse has a normal rhythm) leads one and two will show a positive P wave followed by a QRS complex with formed by a small positive deflection and a large negative deflection (rS morphology) and followed by a T wave that is the typical base apex ECG of horse. If you decide that you want to see additional leads you can easily apply more self-adhesive electrodes and clip the leads to them or use alligator clips.

Currently is common to record ECGs digitally. A system called Televet is the most popular system worldwide for horses. It records ECGs without the need of clips and the rhythm is transmitted to a computer via Bluetooth. It can be used for continuous or exercising recordings.

Another popular option to obtain an ECG is to use a case that can be adapted to a smart phone (Alive core™). This will quickly give you a non-standard lead that you can interpret on the screen, print or email to a colleague. It is a good option for field veterinarians, for short recordings and for owners of animals with recurrent arrhythmias. It is less useful for the hospital setting in which continuous recording are more commonly needed.

When premature beats are auscultated during a prepurchase examination an electrocardiogram is almost always indicated. A continuous electrocardiogram (Holter) may be important as occasional premature contractions (average of less than 1/hour) is consider ‘normal’. Arrhythmias cab be intermittent and often short recordings do not demonstrate all arrhythmias present in one individual. In other species the prognosis and relevance of some arrhythmias is correlated with their frequency over a 24-hour period.

In horses that are to be ridden performing an exercising electrocardiogram is often indicated. Auscultation of an arrhythmia or a diastolic murmur are the most common indications for an exercising electrocardiograms. In general SVPCs are less concerning than VPCS although the increased risk for atrial fibrillation needs to be considered. Premature beats that are overdriven during exercise are considered more benign. Aortic regurgitation and exercise make the perfect arrhythmicogenic cocktail; the ventricular enlargement and remodeling, the decreased coronary perfusion caused by the aortic regurgitation, the shortened diastole caused by the increase in heart rate, the increased oxygen demand and the increased sympathetic tone caused by the exercise combine to predispose to arrhythmias. Horses with moderate or severe aortic regurgitation that continue to exercise should have an exercising electrocardiogram to investigate if exercising arrhythmias are present and an echocardiogram to assess the cardiac structure and function. Mitral or tricusid regurgitation are less commonly associated with exercising arrhythmias.

The interpretation of continuous or exercising electrocardiograms is usually simple. However it requires practice, is often time consuming and in some cases the interpretation is complex. For practitioners that see cardiac cases very frequently or that integrate exercising electrocardiograms in a busy sports medicine practice it may be useful to learn how to interpret exercising rhythms. For many others learning to acquire recordings and seeking advice for interpretation is the highest yield approach. The equipment is currently affordable (2.5-3000USD) and after an initial investment the electrodes and other supplies needed to acquire a continuous or exercising ECG may cost 2-3USD/recording.

Echocardiograms are indicated when significant valvular disease is suspected, to determine if arrhythmias are associated with structural heart disease and in all cases in which congenital heart disease is suspected or continuous murmurs are heard in adult horses. Some general rules follow.

A horse with aortic regurgitation needs an echocardiogram when:

- When the arterial pulses are bounding
- When it is young
- When the horse has ventricular premature beats
• When the horse has other signs of heart disease:
  o mitral regurgitation murmur
  o VSD murmur is present
  o atrial fibrillation
  o exercise intolerance

• A horse with a right sided murmur needs an echocardiogram when:
  • When the murmur is grade 4-6/6 and holosystolic or pansystolic
  • When other signs of heart disease:
    o Poor performance
    o A fib
    o MR or VSD (relative PS) murmurs

• A horses with a left sided systolic murmur needs an echocardiogram if:
  • If the murmur is 3-6/6 and holo or pansystolic
  • If the murmur is band shaped and holo or pansystolic
  • If the horse has other signs of heart disease:
    o Increased respiratory rate or effort
    o Atrial fibrillation
    o Exercise intolerance/poor performance

A horse with an irregularly irregular rhythm should be considered to have atrial fibrillation until proven otherwise. **Atrial fibrillation** is the most common clinically relevant arrhythmia in humans and horses. Atrial fibrillation affects performance in horses that practice high intensity exercise but many horses used for pleasure riding or low intensity equestrian sports can do so while in atrial fibrillation. The decision to convert a horse to normal sinus rhythm is multifactorial. Duration of the arrhythmia, presence of previous episodes, presence of underlying heart disease, economic factors, risk aversion of the owners and the use of the horse are some of these factors. There is ongoing debate about the safety/risk for collapse in horses with AF. If a horse is not going to be converted an echocardiogram and an exercising electrocardiogram are needed to determine if it is safe for the horse to exercise. Current recommendations in horses with sustained atrial fibrillation are that these horses should only be used by informed adult riders and exercise should be limited to a level considered relatively safe based on the exercising ECG.

The evaluation of horses with murmurs or arrhythmias is centered on echocardiograms and electrocardiograms. In the work up a horse with poor performance evaluation of the cardiovascular system is often better done in conjunction with the evaluation of the musculoskeletal and respiratory (upper and lower) systems and the assessment of the fitness status and progression of training. The protocol often includes evaluation of fitness, musculoskeletal system, upper respiratory tract, lower respiratory tract and cardiovascular system by means of: historical questionnaire, general physical examination, lameness examination and gait analysis using gyrosopes, resting and dynamic upper airway endoscopy, bronchoalveolar lavage, echocardiograms, exercising electrocardiograms and measurements of lactate, PCV, heart rate, CK and sweat response before, during and after exercise. I believe that a team approach for the simultaneous evaluation of different body systems and fitness is useful for the evaluation of poor performance and in some cases as a preventative medicine approach for the management of high level athletes.
Cardiac disease is a cause of poor performance in equine athletes. The musculoskeletal and respiratory systems, are the body systems more commonly involved in poor performance in horses participating in any equestrian disciplines, followed by the cardiovascular system [1]. Sudden death (SD) during sports is a rare event but has catastrophic consequences for the horse, the safety of the human partner and the public perception of welfare during equestrian sports.

Cardiac auscultation is often the first clue for the detection of valvular, arrhythmic or congenital disease that can affect performance, safety or life expectancy. The most common physical examination findings that alert clinicians about cardiovascular disease are murmurs and irregular rhythms. Careful description of the cardiac auscultation may seem superfluous but is key to the diagnosis of heart disease in horses.

**Mitral valve disease**
The most common equine valvular disease causing poor performance or shortened life expectancy. A left sided systolic murmur should be considered mitral regurgitation until proven otherwise. The exception to this rule is the physiologic ejection (or flow) murmur. Auscultation is often sufficient for the differentiation. A physiologic ejection murmur is usually 1-2/6 (sometimes 3/6) early to mid-systolic blowing or coarse crescendo-decrescendo murmur with the point of maximal intensity over the pulmonic or aortic valve areas. Ejection murmurs do not radiate and often vary in intensity with exercise or excitement. Physiologic murmurs are common in horses and this leads to many horse owners to the false conclusion that a cardiac murmur in a horse is never a problem. Mitral regurgitation murmurs are 1-6/6 holo- or mid- to late systolic blowing, coarse, musical or honking band shaped or crescendo murmurs with the point of maximal intensity over the mitral or aortic valve areas. Murmurs of mitral regurgitation may or may not radiate and usually do not vary in intensity unless they are caused by a prolapsed valve. Systolic murmurs that are variable in intensity (with excitement, exercise sedation or pain) are frequent. Physiologic ejection murmurs and murmurs of mitral valve prolapse are the common variable murmurs and the clue to their differentiation is their timing and shape: physiologic ejection murmurs are early to mid-systolic crescendo-decrescendo and mitral valve prolapse are mid to late crescendo. When the auscultation is not clear and to determine the severity and prognosis of mitral valve disease an echocardiogram is needed.

**Aortic regurgitation**
A common disease of teenage horses. Aortic valve disease is frequently an incidental finding but some horses with aortic regurgitation can develop exercising arrhythmias, exercise intolerance or heart failure. A diastolic murmur in a horse should be considered to be aortic regurgitation until proven otherwise. The presence of a diastolic murmur, and the consequent suspicion of aortic regurgitation, should prompt the clinician to feel the peripheral pulses. A horse with a diastolic murmur and strong or bounding peripheral pulses likely has moderate or severe aortic regurgitation. Moderate to severe aortic regurgitation predisposes horses to ventricular arrhythmias particularly during exercise. Aortic regurgitation and exercise make the perfect arrhythmogenic cocktail; the ventricular enlargement and remodeling, the decreased coronary perfusion caused by the aortic regurgitation, the shortened diastole caused by the increase in heart rate, the increased oxygen demand and the increased sympathetic tone caused by the exercise create ideal conditions for arrhythmias. Horses with moderate or severe aortic regurgitation that continue to exercise should have an exercising electrocardiogram to investigate if exercising arrhythmias are present and an echocardiogram to assess the cardiac structure and function. **Pulmonic and tricuspid regurgitation** rarely cause performance problems or affect safety unless tricuspid regurgitation causes atrial enlargement that predisposes to atrial fibrillation.

**Ventricular septal defect (VSD)**
The most common congenital heart disease of horses. A VSD can be identified on cardiac auscultation by the presence of a characteristic combination of murmurs. The left to right shunt causes a loud right sided systolic murmur. The second murmur is a systolic crescendo-decrescendo murmur with the point of maximal intensity over the pulmonic valve area that is less loud than the one on the right. This second murmur is caused by the ejection of blood (increased due to the shunt) out of the right ventricle and is called the murmur of ‘relative pulmonic stenosis’. The pulmonary artery is relatively small to the amount of blood ejected by the right ventricle. It is key in horses with right sided systolic murmurs to carefully auscultate the pulmonic valve area to differentiate tricuspid regurgitation (only right sided murmur) from VSDs (right and left sided murmurs). It takes conscious effort to place/push the stethoscope on the third intercostal space well under the left triceps to auscultate the pulmonic valve area. If this is not done the murmur of relative pulmonic stenosis is easily missed. Echocardiograms are needed to determine the prognosis and level of exercise that will be tolerated by a horse with a VSD.
Atrial fibrillation

The most common clinically relevant arrhythmia in humans and horses [2,3]. Atrial fibrillation affects performance in horses that practice high intensity exercise but many horses used for pleasure riding or low intensity equestrian sports can do so while in atrial fibrillation. The decision to convert a horse to normal sinus rhythm is multifactorial. Duration of the arrhythmia, presence of previous episodes, presence of underlying heart disease, economic factors, risk aversion of the owners and the use of the horse are some of these factors. There is ongoing debate about the safety/risk for collapse in horses with AF [4-7]. If a horse is not going to be converted an echocardiogram and an exercising electrocardiogram are needed to determine if it is safe for the horse to exercise. Current recommendations in horses with sustained atrial fibrillation are that these horses should only be used by informed adult riders and exercise should be limited to a level considered relatively safe based on the exercising ECG. Other cardiac diseases that can cause decreased performance are myocarditis, poor myocardial function, aortocardiac fistulas, complex congenital heart disease, 3rd degree AV block, sick sinus syndrome, severe pericarditis etc.

The field of exercising arrhythmias deserves special mention. The presence of arrhythmias is common in normally and poorly performing horses [8-13]. The influence of exercising arrhythmias on performance or tolerance to exercise is intuitive and extrapolated from basic physiologic studies documenting the associated cardiovascular changes. However this influence is not proven and in many cases it is not possible to ascertain if arrhythmias are incidental or the cause of poor performance. The fact that a malignant arrhythmia has the potential of causing collapse or sudden cardiac death (SCD) complicates the decision making processes [14]. Collapse and SCD can affect the health of horses, the public perception of welfare during equestrian sports and most importantly the safety of riders. The incidence of malignant arrhythmias causing collapse or SCD is fortunately low [15,16] but likely 50-100 fold higher in horses than in analogous human athletes [17]. Conditions to which equine sudden cardiac death has been attributed include: cardiac failure, arrhythmias, pulmonary hemorrhage or idiopathic blood vessel rupture. Idiopathic blood vessel rupture (particularly aortic rupture) is a classically described cause of sudden death in horses, reported in 9–24% of sudden death cases. The presence of abnormalities in the aortic root or signs of pulmonary hypertension should alert clinicians about an increased risk for sudden death. Many of the leading causes of SCD in humans, such as hypertrophic cardiomyopathy, coronary anomalies, arrhythmogenic right ventricular cardiomyopathy (ARVC), channelopathies, Marfan Syndrome, commotio cordis. [16-20] or coronary artery [18,21] are not reported or frequent in horses.

The evaluation of horses with murmurs or arrhythmias is centered on echocardiograms and electrocardiograms. In the work-up of a horse with poor performance, evaluation of the cardiovascular system is often better done in conjunction with the evaluation of the musculoskeletal and respiratory (upper and lower) systems and the assessment of the fitness status and progression of training. The protocol often includes evaluation of fitness, musculoskeletal system, upper respiratory tract, lower respiratory tract and cardiovascular system by means of: historical questionnaire, general physical examination, lameness examination and gait analysis using gyroscopes, resting and dynamic upper airway endoscopy, bronchoalveolar lavage, echocardiograms, exercising electrocardiograms and measurements of lactate, PCV, heart rate, CK and sweat response before, during and after exercise. The simultaneous evaluation of different body systems and fitness is useful for the investigation of poor performance and as a preventative medicine approach for the management of high level athletes. We believe that each member of a team composed of a primary care veterinarian, trainer and specialists in internal medicine, surgery and sports medicine and rehabilitation can solve a part of the poor performance or preventative medicine equation.

References


Acute Non-Weight-Bearing Lameness
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Diseases may result in acute non-weight-bearing lameness because of severe pain and/or instability. Causes of pain severe enough to result in non-weight-bearing lameness are often related to restriction of inflammation or infection within a closed space resulting in distension (i.e. synovial structure, facial plane, etc.). In these cases, reducing the inflammation, resolving the infection, and relieving the distension are the keys to improving the comfort of the horse. Horses with instability of their limb resulting in acute non-weight-bearing lameness often have pain associated with the cause of instability, but are additionally distressed due to their inability to bear weight. Providing stabilization, most commonly with external coaptation is important to minimize stress, discomfort, and further damage prior to definitive treatment.

The first step in evaluation of a horse with a non-weight-bearing lameness is performing a triage examination. A triage exam is designed to determine if a patient is in shock and requires immediate treatment or if the remainder of the physical examination and additional diagnostics can be performed prior to treatment safely. The triage examination focuses on rapid evaluation of three organ systems: neurologic, respiratory, and cardiovascular. The clinical signs are primarily related to either poor perfusion (cardiac output) or the body’s compensatory mechanisms to improve perfusion and arterial oxygenation. Although relatively uncommon, shock due to hemorrhage or severe dehydration can occur in horses with non-weight-bearing lameness. The cardiovascular triage examination should include heart rate, mucus membrane color, capillary refill time, jugular refill, pulse quality, and temperature of the extremities. Clinical signs of pain can be difficult to differentiate from signs of inadequate perfusion. For example, both can result in increased heart rate, pale mucus membranes, and cool extremities as a result of increased sympathetic tone. Signs such as poor pulse quality, prolonged capillary refill time, and poor jugular refill should not occur as a result of pain and are likely signs of hypovolemia. Hemorrhage associated with fractures and other injuries resulting in non-weight-bearing lameness can be difficult to identify and quantify. Some of the most severe hemorrhage can occur with fractures of the upper limb, including fractures of the humerus, femur and pelvis. These fractures are in close proximity to major vessels and surrounded by a large amount of musculature making significant blood loss without external evidence of blood possible. Lacerations in the upper limb are also more likely to result in severe hemorrhage. It is also important to remember that the trauma that caused the limb injury may have involved other regions of the horse such as the abdomen or thorax where bleeding could occur without external evidence. Horses that sustain a fracture or injury far from a source of water can become dehydrated. As noted, luckily, most horses will be stable enough to continue examination.

Causes of non-weight-bearing lameness associated with pain can often be identified rapidly on physical examination. As noted, many of the causes are associated with inflammation and/or infection being trapped within a confined space. One of the most common examples is a sub-solar abscess. Diagnosis of sub-solar abscess is based on clinical signs localizing the lameness to the foot including lack of other obvious findings within in the limb (swelling at the coronary band, pastern region can occur uncommonly), increased digital pulses, heat, and sensitivity to hoof testers. While these findings most consistently indicate an abscess, clinical signs will be identical in horses with fractures of P3 and this should be considered as a differential in cases with suggestive history or not responding to therapy. Another important example is infection of a synovial structure. Depending on the structure affected heat, pain, and swelling (effusion and periarticular) can be used to localize the problem. In adult horses, infection is generally secondary to a wound or injection/surgery. Careful palpation for un-noticed wounds and a good history can provide important information for these cases. Definitive diagnosis will require cytology and culture of the affected synovial structure. Other, less common, examples include myositis and cellulitis, which can often be identified with palpation and examination. Causes of non-weight-bearing lameness associated with pain without instability that are not the result of inflammation and/or infection being trapped within a confined space include non-displaced and/or incomplete fractures and lacerations to other soft tissues. Non-displaced and/or incomplete fractures are often difficult to diagnose and can become catastrophic if not suspected. Veterinarians must have a high index of suspicion based on history and physical examination findings and recommend strict stall rest (with or without tying) while awaiting definitive diagnosis or rule out of the fracture. Nuclear scintigraphy is the most accurate imaging modality for these types of fractures. Uptake will be greatest once remodeling is underway (7-10 days). Multiple radiographs in many planes will often miss these fractures until fracture remodeling and periosteal callus formation has started.

Causes of non-weight-bearing lameness associated with instability of the limb are generally not a diagnostic challenge and frequently examination even from some distance will give a diagnosis. Sources of instability that are frequently easily diagnosed on visual examination include complete, displaced fractures, disruption of tendons or ligaments required for weight bearing (ex: suspensory apparatus breakdown), and joint luxation. However, some causes of instability are less obvious including intermittent or sub-luxation of a joint and nerve paralysis/paresis. Fractures may be less obvious in the upper limb where palpation for swelling and
crepitus, examination for symmetry, and rectal examination and within the hoof (P3 fractures) where radiographs may be necessary to aid in diagnosis.

Due to the level of anxiety most horses with instability of the limb experience, appropriate administration of sedation and analgesics is vital to the safety of the horse, owner, and veterinarian during evaluation and stabilization. When choosing the type and dose of sedation to use it is important to balance the need for analgesia and a calm and quiet patient during manipulation with the detrimental ataxia that can occur with sedation. Cardiovascular compromise and increased sympathetic tone due to pain can alter the amount of sedation required to achieve the desired effect from what would be expected in a normal horse. In most cases, cardiovascular compromise is minimal while sympathetic tone is high. This can result in resistance to sedation. In horses that are painful and panicked, small amounts of sedation produce little effect, which results in the administration of multiple doses. This can lead to a large amount of sedation being given by the time an effect is seen. Once the horse becomes sedated all of the sedation given takes effect and the horse can be extremely sedated. Giving one good dose of sedation is preferable to repeated small, less effective doses. It is likely that assessment, wound treatment, and stabilization will require at least 1 hour. For this reason, I recommend using a combination of detomidine and butorphanol. Nonsteroidal anti-inflammatory drugs should be given for analgesia in addition to sedation.

Definitive diagnosis of boney and soft tissue causes of instability will require diagnostic imaging. Diagnostic imaging prior to stabilization can be difficult and time consuming. For these reasons, I usually start with stabilization after a rapid inspection of the overlying skin for any wounds and palpation of the limb distal to the injury for temperature if damage to the blood supply is suspected. If a break in the skin is identified, the wound should be cleaned, a light sterile bandage placed, and the horse should be started on broad-spectrum systemic antimicrobials. The owner should be informed that having an open injury is likely to decrease the prognosis. If the fracture is closed, antimicrobial therapy should not be initiated until the perioperative period if surgery is required to achieve maximum concentrations at the time of repair.

Following evaluation and sedation, the goal is to stabilize the limb to provide the horse with the ability to bear weight as quickly as possible. Stabilization is generally achieved with external coaptation. This is most commonly performed with splints, but casts or bandage casts or splint/cast combinations can be used if the veterinarian is comfortable with casting and has casting material available. The application of casts will not be discussed in this presentation. The ideal splinting material is strong but lightweight and radiolucent to allow diagnostic imaging to be performed without compromising stabilization. It should also be easy to cut to the correct size, inexpensive, and readily available. PVC tubing fulfills these criteria and is frequently recommended for splinting horses. The length of the tube should be the height of an adult horse’s withers or hip to be used for full limb splinting. The splint can be easily shortened for half limb splinting. The diameter of the tube should be large enough to provide 3-4 splints that are approximately 4-6 inches wide with a gentle curve. If a splint is too curved it creates gaps between the splint and the bandage. If PVC is not available, alternatives include wood boards or multiple broom handles bound together. Casting material can also be used to make splints. It is easily molded to the correct length and shape. Casting material is particularly useful when making splints for foals.

There are some important general principles that can be used to optimize stabilization with splints. They provide guidelines for bandaging, splint length, and placement.

1. **A splint should span the joint above and the joint below the fracture.** In most cases splints are started at the ground. Ending a splint or bandage at or below the fracture should be avoided at all costs because it results in a lever arm that will increase motion at the fracture.

2. **If possible, two splints should be used to stabilize the limb preventing motion in the dorsal to palmar/plantar or cranial to caudal plane and the medial to lateral plane.**

3. **The bandage beneath a splint must be composed of multiple thin layers to avoid compaction and shifting of excessive padding. Each layer should be composed of cotton padding (sheet cotton, roll cotton, or combine) wrapped with brown gauze to make a smooth, even layer. Layers that are <2 cm thick are recommended.** I prefer using roll cotton because I can create the most even bandage that way. A layer of vetwrap or elastic can be placed over the second layer. A bandage that is 2-3 layers provides adequate padding and results in the leg being a uniform tube to minimize dead space. Stabilization is optimal when the stabilization is as close to the center of gravity of the limb as possible (i.e. internal fixation). Additional layers of padding (as previously recommended) provide little/no additional stabilization and allow more compression, shifting, and/or bunching of the bandage.

4. **Motion at the fracture is minimized by preventing movement of the splint.**
   a. **The shape of the splint should match the shape of the bandaged limb.** The bandage should be used to provide a smooth tube so that the splint is in close contact along the entire bandage. Increased padding can be applied in areas of indentation in the contour of the leg (like the palmar/plantar aspect of the pastern).
   b. **The material used to affix the splint to the underlying splint should be inelastic.** Inelastic materials that can be used include white athletic tape and duct tape. Duct tape is much less expensive, but does not breathe and the limb will sweat more.

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c. The splint should be attached to the underlying bandage over as much of the length as possible. The attachment cannot be stopped at the level of the fracture for the same reason that the bandage and splint cannot be stopped at that level.

The above principles provide the basis for understanding how to splint specific regions/injuries in horses. The principles are adapted to the anatomy and mechanics of the region affected.

1. Instability at the level of the first and second phalanx of the forelimb and hindlimb are managed with splints from the ground to the level of the proximal metacarpus or metatarsus. It is vital to align the boney column—the dorsal aspect of the phalangeal bones and the metacarpus or metatarsus should form a straight line. This can be accomplished by placing the horse on its toe and applying two splints (dorsal or palmar/plantar and lateral) to the limb. A Kimsey splint will align the boney column, but only provides stabilization in the dorsal to palmar/plantar plane (which may be all that is necessary with a soft tissue injury). Medial to lateral stabilization can be augmented with the addition of a lateral splint. Kimsey splints are very easy to apply and are available for forelimbs and hindlimbs. Disadvantages include expense and weight of the splint. Additionally, the Velcro loses it’s stickiness with repeated use and needs to be reinforced with tape along the length of the splint.

2. Disruption of the suspensory apparatus causes instability only in the dorsal to palmar/plantar plane. Breakdown of the suspensory apparatus is the injury that sparked the design of the Kimsey splint.

3. Third metacarpal fractures are stabilized with a caudal and lateral splint from the ground to the level of the elbow. Aligning the boney column will require placing the horse on the toe similar to what is needed for P1 and P2 fractures.

4. Third metatarsal fractures are stabilized with a lateral splint from the ground to the level of the stifle. When a straight splint is used, in order to support the center of weight bearing of the limb, it will start just plantar to the heel and pass cranial to the hock and caudal to the stifle. Due to the anatomy of the hindlimb, it is not possible to place a caudal or cranial splint that will truly immobilize the hock. However, a caudal splint can be placed from the ground to the level of the proximal aspect of the calcaneus. Although it does not immobilize all of the joints of the hock, it does restrict the movement of the distal joints and decrease the motion of the tarsocrural joint. Additionally, the caudal splint is helpful to align the boney column of the metatarsus and phalangeal bones. If custom splints are used, they can be created to fit the contour of the limb.

5. Radius fractures are stabilized with a lateral splint from the ground to the shoulder or withers. The bandage and the attachment of the splint to the bandage should go as proximal as possible. It is vital that they are proximal to the location of the fracture. It is not possible to firmly affix the splint to the body above the elbow. Some have recommended figure eight bandaging, but the restriction in motion appears minimal. Although a caudal splint cannot completely immobilize the elbow, a caudal splint is frequently placed to the level of the proximal olecranon to decrease the motion at the elbow joint.

6. Carpal instability can be stabilized in a manner similar to radial fractures.

7. Tibial fractures are stabilized with a lateral splint from the ground to the level of the hip. The splint will follow the center of weight bearing of the limb. The bandage and the attachment of the splint to the bandage should go as proximal as possible. It is vital that they are proximal to the location of the fracture. It is not possible to firmly affix the splint to the body above the stifle. The addition of a customized cranial or caudal splint may be useful.

8. Tarsal instability can be stabilized similar to tibial fractures.

9. Olecranon fractures, some humeral fractures and radial nerve paralysis result in a “dropped elbow appearance”. Olecranon fractures do not require stabilization of the fracture to allow the horse to bear weight. The olecranon is not a weight bearing bone, but it is integral to weight bearing because of it’s role in the stay apparatus and locking the carpus. Horses with olecranon fractures that have a “dropped elbow” will be able to bear weight if a splint is placed to lock the carpus in extension. A caudal splint from the ground to the elbow is appropriate. It is important to differentiate a horse with a “dropped elbow” due to an olecranon fracture from one due to a distal humeral fracture because splinting a humeral fracture with a splint that ends at or below the elbow will provide a lever arm and increase motion at the humeral fracture.

10. Humerus and femur fractures are not amenable to splinting. Splints do not provide adequate stabilization of the joint above or below these fractures. Splinting may allow the horses to bear more weight, but without stabilization of the fracture this can lead to more damage.
The goals of evaluating a horse showing signs of colic in the field are to localize and categorize the cause of colic and to use that information to determine whether treatment in the field, referral, or euthanasia are most appropriate. Most frequently, the cause of colic can be localized to the gastrointestinal tract, more specifically to either the small or large intestine. Less commonly, diseases of the stomach, non-gastrointestinal organs, or peritoneum can result in colic. Within the small and large intestine, the majority of causes of colic can be categorized as non-strangulating (simple) obstructive, strangulating obstructive, or inflammatory. History, signalment, and a directed colic examination, including physical examination, nasogastric intubation, and examination per rectum, are the core of a basic colic work-up. In most cases, these basics will provide enough information for you to localize and categorize cause of colic and make treatment recommendations to your client. Advanced diagnostics that can be performed in the field include ultrasound, abdominocentesis, bloodwork, and fecal diagnostics. Knowing how to perform and interpret these tests in a field situation is valuable.

However, it is important to realize the limitations of these tests and that not all of the information from some of these diagnostics will be available immediately, i.e. on an emergency basis, in the field. The use of advanced diagnostics should be reserved for cases where the results are required to localize and characterize the lesion or, more importantly, allow you to help you client make the decision to treat, refer, or euthanize their horse.

A complete history should include detailed information about the current episode of colic as well as previous medical history and management and performance history. Information about the current episode of colic should include duration and severity of signs, changes in quantity, frequency, and quality of manure production, and any medications administered. Questions about previous relevant medical history should determine frequency, severity, and cause of previous episodes of colic, breeding, pregnancy and parturition details, deworming and vaccination protocols, ongoing illnesses, and any medications or supplements. Examples of associations between history and lesion localization and characterization include: horses with strangulating obstructions are typically acutely, severely painful whereas horses with inflammatory lesions are typically depressed and mildly painful, stallions with inguinal hernia may have a history of recently breeding, antibiotics can lead to antibiotic associated colitis, and alfalfa hay has been associated with cantharadin toxicity and enterolithiasis.

The signalment of a patient includes age, gender, and breed. Signalment has been associated with the prevalence, cause, and clinical signs displayed during colic. Examples of associations between signalment and lesion localization and characterization include: variations in gastric ulceration in foals of different ages and the tendency for older horses, gaited breeds and draft horses to behave stoically and show less overt signs of colic than expected with the underlying disease. As with any emergency, the first part of the examination of a horse showing signs of colic is a triage exam. A triage exam is designed to determine if a patient is in shock and requires immediate treatment or if the remainder of the physical examination and additional diagnostics can be performed prior to treatment safely. The triage examination focuses on rapid evaluation of three organ systems: neurologic, respiratory, and cardiovascular. The clinical signs are primarily related to either poor perfusion (cardiac output) or the body’s compensatory mechanisms to improve perfusion and arterial oxygenation. Signs of shock are more likely to occur in horses showing signs of colic associated with strangulating obstruction and inflammatory lesions and are uncommon in horses with non-strangulating obstructions. In my experience, large intestinal strangulating and inflammatory lesions are particularly likely to result in shock.

Once the triage examination has been completed, a complete physical examination should be performed. Examples of associations between physical examination findings and lesion localization and characterization include: abdominal distension is more common with lesions of the large intestine than the small intestine, horses with inflammatory lesions are the most likely to have a fever, and some changes in mucus membrane color such as cyanosis may occur with non-gastrointestinal causes of colic such as cardiac (CHF) or respiratory (pleuropneumonia) diseases.

The quantity and quality of nasogastric reflux should be determined in all horses showing signs of colic. A nasogastric tube should be passed immediately if the heart rate on initial examination is greater than 60 bpm. Greater than 2L of nasogastric reflux is considered abnormal. Stomach contents should normally be green with some flecks of feed material present and smell of fresh grass. In general, small intestinal diseases are more likely to result in net nasogastric reflux. Within small intestinal disease, the highest quantity of reflux occurs with inflammatory disease (anterior enteritis). Additionally, reflux from horses with anterior enteritis may be bloody and fetid smelling. The amount and time from initial signs of colic until onset of reflux for horses with obstructive small intestinal lesions will depend on the location of the lesion.

Unless there is a contraindication, such as risk to the horse or veterinarian, all horses showing signs of colic should have an examination per rectum. When performing examination per rectum, the first thing to determine is if the examination is normal or abnormal and the second is to determine if the abnormality is distension of the large or small intestine. Identification of a specific
underlying cause of colic is uncommon. Distension of the large intestine is characterized by the size of the distended structure the presence of tight bands on the distension. Distension can be caused by gas, fluid, or ingesta. Specific causes of large intestinal colic that can be identified on examination per rectum include left dorsal displacement of the large intestine and impactions of the small colon, cecum, or pelvic flexure. Fluidy distension suggests colitis. Other large intestinal diseases generally result in non-specific distension. Small intestinal distension is also characterized by the size of the distended structure and the absence of bands. Most causes of small intestinal disease are not associated with specific findings on examination per rectum. Ileal impactions can be palpated in some cases. Distension with anterior enteritis is generally less turgid and the intestine may feel thickened. Strangulated small intestine can also be thickened.

Portable ultrasound machines have become widely available and increasingly affordable for use in equine practice. While the same machine can be used for transrectal, musculoskeletal, transabdominal, and thoracic imaging, using the most appropriate probes will optimize the quality and types of images you are able to obtain. In horses showing signs of colic, transabdominal ultrasound examination is most commonly performed. In some cases, transthoracic ultrasound and thoracic ultrasound are indicated as well. For transabdominal ultrasound, a low frequency (3-5 MHz) curvilinear probe is recommended to provide the maximal depth penetration. Alternatively, higher frequency probes such as a 5-7.5 MHz transrectal linear probe or a 5-8 MHz Microconvex probe can be used for transabdominal image acquisition, but will not be able to reach the same depth as the 3-5 MHz curvilinear probe. Regardless of the probe used, the best resolution of the image will be obtained if the minimal depth required is maintained and the focal distance is appropriately adjusted. Any of the probes discussed related to transabdominal ultrasound will work well for most aspects of transabdominal ultrasound because relatively minimal depth penetration is required with the exception of echocardiography. Transrectal ultrasound is performed with a linear probe designed for that purpose. Appropriate restraint (physical and chemical) and technique must be used when performing transrectal ultrasound just as for any examination per rectum.

Transabdominal ultrasound is non-invasive and safe to perform in most horses showing signs of colic. If possible, ultrasound should be performed in a dark area and seeing the image will be quite difficult in direct sunlight. For the best image quality, the skin should be clipped and cleaned and coupling gel should be used. However, an adequate image can usually be obtained by soaking the hair with alcohol. Horses with thick, long, and/or dirty coats are more likely to require clipping. Image quality is also decreased in horses with excessive fat. Similar preparation considerations should be used when performing transthoracic ultrasound.

As with any examination, each clinician should develop a systematic and consistent method for ultrasound. A complete transabdominal ultrasound involves imaging each rib space (from approximately ICS 7/8 caudally) and the paralumbar fossa as well as the ventral abdomen and inguinal areas on both left and right sides. Depending on the amount of gas within the viscera and several other factors such as age, portions of the gastrointestinal tract (stomach, small intestine, cecum, large colon, and small colon), spleen, liver, kidneys, and bladder as well as peritoneal fluid can be imaged. With regards to the gastrointestinal tract, ultrasound is useful in evaluating wall thickness, motility, distension, and contents. Depending on the information that is being sought, the question being asked, an abbreviated, targeted transabdominal ultrasound may be more useful in cases of acute colic. A complete transthoracic ultrasound involves imaging each rib space on each on both right and left sides. Thoracic ultrasound is useful for evaluating surface of the lung, the pleural space, and the heart.

Transabdominal ultrasound is indicated when it can provide information that a basic colic examination cannot or when it is necessary to improve confidence in a diagnosis and help an owner make a decision. One set of indications is for situations where performing an examination per rectum is dangerous for the patient or veterinarian. It can be safely performed in patients too small for examination per rectum like foals and some miniature horses and ponies. It may also be safely performed in recumbent horses and fractious horses with appropriate sedation/restraint. For gastrointestinal lesions, transabdominal ultrasound is particularly useful for identifying and evaluating the distension, motility and wall thickness of small intestine, intussusceptions, fluid contents within the large intestine, and ruling out left dorsal displacement of the large colon when the spleen and kidney can be imaged adjacent to one another. Transabdominal ultrasound is more sensitive than examination per rectum for identifying small intestine and is indicated to identify small intestine not palpable on examination per rectum if other findings suggest a small intestinal lesion. Distended, amotile, thickened small intestine is a typical appearance for a strangulated segment of small intestine. Increased wall thickness occurs with large colon volvulus and may also occur with inflammatory lesions. Increased fluid contents of the large intestine are consistent with colitis. When trying to differentiate gastrointestinal lesion location, ultrasound is most useful for small intestinal identification. For non-gastrointestinal causes of colic, transabdominal ultrasound is useful in quantifying and qualifying peritoneal fluid and evaluating the architecture of the liver and spleen. It can be particularly useful identifying the characteristic swirling fluid associated with hemoabdomen and can provide valuable improved confidence and decision making is a horse with ruptured gastrointestinal tract locating a site for abdominocentesis to ensure the sample is not an enterocentesis.

Transthoracic ultrasound has limited indications in horses showing signs of colic. The only cause of true colic that it is particularly useful in identifying is diaphragmatic hernia. However, several thoracic diseases can present as colic—either due to the tissue hypoxia they create or an inability to distinguish thoracic pain from abdominal pain. Examples of thoracic disease that can present as colic are congestive heart failure and pleuropneumonia. Transrectal ultrasound should be considered in two types of cases. The first
is when the reproductive tract (particularly in mares) is the suspected source of colic. The other is any case where it may help characterize an abnormal finding from examination per rectum, such as a mass.

Site selection for abdominocentesis can be empirical or ultrasound guided. The most common locations selected empirically are just behind the xiphoid on midline or a hands breadth to the right of midline at the most ventral aspect of the abdomen. Ultrasound can be used to identify a pocket of fluid or to avoid gastrointestinal structures or spleen to be confident that the fluid is not an enterocentesis or splenic sample. It is important to note, that in a normal abdomen there may be no fluid or only small pockets seen intermittently on ultrasound. Despite this, abdominal fluid can be obtained in most cases. Once the site is selected, it should be clipped and prepared in aseptic fashion.

There are two basic techniques for performing abdominocentesis: using a needle or with a blunt tipped catheter such as a teat cannula or bitch catheter. In most horses, an 18 gauge, 1.5” needle can reach an adequate depth, but a spinal needle may be required in very fat horses. When using a needle, it is not necessary to block the site with local anesthetic. The needle should be advanced slowly until fluid is obtained. In some cases, due to the negative intra-abdominal pressure, it may help to use a second and third needle for sampling. When using a teat cannula or bitch catheter, the site is blocked with local anesthetic and a blade is used to incise the skin, subcutaneous tissue, and nick the external rectus sheath (off midline) or linea. Off midline, the teat cannula or bitch catheter must be pushed through the external, rectus abdominus muscle and internal rectus sheath. At either location, the peritoneum must be penetrated. The peritoneum cannot be blocked and most horses react when the teat cannula or bitch catheter tents it prior to puncture.

The sample should be collected in a purple top (EDTA) tube and a red top (serum tube). If possible, a purple top with liquid EDTA should be used and excess EDTA should be shaken out so that it does not falsely elevate protein readings. Information that can be gained from the peritoneal fluid in the fluid includes gross appearance and possibly total protein and lactate if a refractometer and lactatometer are available. Cytological analysis and bacterial culture and sensitivity will require submission to a laboratory. Important diagnoses that are suggested by gross appearance include peritonitis, hemoabdomen, and strangulating lesions. Fluid from horses with peritonitis will be opaque and white/yellow/pink in color. Fluid from a hemoabomen will have a high PCV (allowing the tube to settle will help estimate this) and will not clot (it will if it is a splenic sample). Fluid from a horse with a strangulating lesion will become serosanguinous. Rarely horses with inflammatory lesions can also have serosanguinous fluid. It can be difficult to differentiate this from blood contamination. Increases in total protein (>2-2.5 g/dl) occur in both strangulating and inflammatory diseases. Minimal or no increase in total protein is seen with non-strangulating lesions. In inflammatory diseases, the increase is more marked than the increase in WBC count, but that number will not be known in field conditions. Increases in peritoneal lactate (>2 mmol/L), especially if they are greater than the increase in lactate in peripheral blood, are most common in strangulating lesions. However, non-strangulating and inflammatory lesions can increase peritoneal lactate in some cases.

Abdominocentesis is indicated if the information gained will alter the client or veterinarian’s decision for referral. If an owner is unable or does not wish to treat a serious condition such as hemoabdomen or strangulating lesion, euthanasia may be recommended over referral based on abdominocentesis. If gastrointestinal rupture can be diagnosed in the field, the horse may not have to endure the trip to the referral center prior to euthanasia. Abdominal fluid analysis is also useful in characterizing small and large intestinal lesions, although typically changes occur more quickly with small intestinal lesions. Strangulating lesions are typified by a serosanguinous appearance, increased total protein, and increased lactate. Inflammatory lesions increase total protein and may rarely result in serosanguinous appearance. Non-strangulating lesions result in little to no change in the abdominal fluid.

Most bloodwork requires submission to a laboratory and is therefore rarely indicated in cases of acute colic in the field. Blood lactate can be measured in the field and may help support a diagnosis of a strangulating lesion. However, in my opinion, decisions should not be based on a single blood lactate measurement. It is nearly impossible to be sure that the increase in blood lactate is due to ischemia of the gastrointestinal tract rather than total body poor perfusion. In cases of horses with more chronic or recurrent colic, complete bloodwork can be useful in identifying inflammation and impairment of renal or hepatic function.

As with bloodwork, most fecal diagnostics require submission to a laboratory and are therefore rarely indicated in cases of acute colic in the field. One test that can be performed in the field is placing feces in a rectal sleeve, adding water, and determining if sand sediments in the fingers. This test has significant limitations in that it is not standardized and does not reliably predict significant sand when it is positive nor reliably predict no sand when it is negative. In cases of horses with more chronic or recurrent colic, fecal diagnostics for infectious diseases can be performed.
Field Treatment of Colic

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Treatment of horses with colic in the field will fall into two main categories—horses that require treatment to allow evaluation and stabilization prior to referral or euthanasia and horses with diseases that can be managed effectively, economically, and efficiently in the field. The determination of which category the horse you are attending falls into can be made based on lesion localization and characterization. Field evaluation of colic is the topic of a separate presentation, but will include a minimum database of history, physical examination, nasogastric tube intubation, and examination per rectum. Most frequently, the cause of colic can be localized to the gastrointestinal tract, more specifically to either the small or large intestine. Less commonly, diseases of the stomach, non-gastrointestinal organs, or peritoneum can result in colic. Within the small and large intestine, the majority of causes of colic can be categorized as non-strangulating (simple) obstructive, strangulating obstructive, or inflammatory.

Horses with strangulating lesions obviously fall into the first category, treatment for evaluation and stabilization prior to referral or euthanasia. Most horses with inflammatory lesions will also fall into the first category for several reasons including: they are at high risk for hypovolemic and/or distributive shock, once stabilized they will likely require intensive medical management, and the risk of infectious etiologies makes biosecurity a priority. However, if the owner is willing to manage the biosecurity risk, some horses with mild inflammatory lesions can be treated effectively in the field. Field treatment of horses with inflammatory lesions should be restricted to horses that are not in shock, relatively comfortable, and able to take in oral fluids (not refluxing). Thus, most of the horses that fall into the second category, with diseases that can be managed effectively, economically, and efficiently in the field, have non-strangulating (simple) obstructions. The decision of referral or field treatment in these cases must be made based on the clinical signs of the horse (pain, response to analgesics, duration) combined with the owner’s level of comfort and financial considerations. Always remember when considering costs that, although in the end it is your choice, you should be charging for your services and time.

Treatments required to allow evaluation and stabilization prior to referral include analgesics, anti-spasmodics, and intravenous fluid therapy. Treatments for horses that can be managed in the field can be divided into non-specific, symptomatic therapies of analgesics, anti-spasmodics, and trocharization and specific therapies for impactions, mild inflammatory lesions, and left dorsal displacement of the large colon. Analgesics used in the field treatment of colic include non-steroidal anti-inflammatories, alpha-2 agonists, and opioids.

Non-steroidal anti-inflammatory drugs inhibit cyclooxygenase and decreased the production of prostaglandins and thromboxane. Flunixin meglumine is the most common non-steroidal anti-inflammatory used in horses with colic. Flunixin meglumine has analgesic, anti-inflammatory, anti-endotoxic, and anti-pyretic effects. Other non-steroidal anti-inflammatory drugs include phenylbutazone and firocoxib. In one study firocoxib was as good as flunixin meglumine at controlling postoperative visceral pain. In addition to their role inflammation, prostaglandins are important for acid regulation in the stomach and blood flow to the gastrointestinal tract and kidney. As a result, toxic effects of non-steroidal anti-inflammatory drugs include stomach ulceration, right dorsal colitis, and renal tubular necrosis.

Alpha-2 agonists activate α2 (and to a lesser extent α1) adrenoreceptors in a variety of tissues. The α2 effects in the central nervous system are responsible for the desired effects of sedation and analgesia. They also produce muscle relaxation and decreased reactivity to painful stimuli and procedures. The α2 agonists that are currently widely available for use in horses and most practitioners should be familiar with are xylazine, romifidine, and detomidine. The main differences between the drugs are in their duration of action and potency (xylazine<romifidine<detomidine). α2 agonists produce an initial period of hypertension associated with vasoconstriction followed by hypotension due to bradycardia (with the potential for AV block) and resultant decreased cardiac output. α2 agonists are respiratory depressants decreasing respiratory rate and tidal volume and increasing upper airway resistance. α2 agonists markedly reduce intestinal motility. α2 agonists also produce hyperglycemia and diuresis (inhibit ADH). In horses with colic, α2 agonists are valuable for facilitating diagnostics and assessing severity of pain. Due to the cardiovascular, respiratory, and gastrointestinal effects of α2 agonists, these systems should be assessed prior to administration with a brief physical examination. Once given, it is important to monitor the duration of sedation and pain relief. I typically expect pain relief for ≥ 30-40 minutes with romifidine, and ≥ 60 minutes with detomidine. To allow for timely assessment, xylazine is generally chosen for initial sedation.

The most commonly used opioid in equine practice is butorphanol. Butorphanol is an opioid agonist (κ)-antagonist (μ) that produces analgesia through κ opioid receptors primarily located in the brain and spinal cord. Because of its greater activity at κ opioid receptors, it is believed to be effective in treating visceral pain. In horses, stimulation of opioid receptors can result in hyperexcitability and increased locomotion. These effects can be minimized by coadministration with α2 agonists. Butorphanol has the potential to induce respiratory depression, but this effect is generally minimal in horses without respiratory compromise that are not heavily sedated or anesthetized. Butorphanol decreases gastrointestinal motility, but the clinical relevance of this side effect in
horses with colic is controversial. Butorphanol is generally used in combination with α₂ agonists for a synergistic effect on sedation and analgesia. The combination is more potent and longer acting than α₂ agonists alone and minimizes side effects of each. In my experience, butorphanol given without α₂ agonists is a fairly weak analgesic and provides little sedation.

In the U.S., N-butylscopolammonium bromide (NBB) is the only commercially available anti-spasmodic. NBB is an acetylcholine receptor antagonist that inhibits muscarinic receptors in the parasympathetic autonomic nervous system. In the gastrointestinal tract, this inhibition results in decreased tone and motility. The parasympatholytic effects of NBB on the cardiovascular system result in tachycardia, hypertension, and rare cardiac arrhythmias. NBB is used to reduce abdominal pain. It is particularly effective in horses with spasmodic, or gas, colic where the pain is associated with hypermotility. It is also effective at reducing rectal pressure and rectal straining during rectal examination and, therefore, can be used to decrease the risk of complications.

Intravenous fluid therapy may be indicated in the field for stabilization of horses showing signs of shock based on triage examination in the field prior to referral. The goal of emergency fluid therapy is rapid restoration of preload, cardiac output, and oxygen delivery. This requires use of the intravenous route of fluid administration. A wide diameter (10-14g), short catheter with large bore fluid administration sets will maximize the speed of delivery. Depending on the type of fluid chosen and packaging, pressurized administration may be possible. The two main alternatives for rapid volume resuscitation are: 1) using a relatively small volume of hypertonic saline (2-4 ml/kg or 1-2 L/adult horse [not generally recommended in foals]) and/or synthetic colloids (5-10 ml/kg or 2.5-5 L/adult horse or 250-500 ml/foal) to borrow/pull fluid from the interstitium into the vascular space followed by administration of larger volumes of isotonic crystalloids (at least 20 ml/kg) or 2) using isotonic crystalloid fluid alone. In either case, the "shock dose" of 80-90 ml/kg can be used as a guideline for the maximum amount of isotonic crystalloids to administer as a bolus. In general, it is rare to need the entire “shock dose”. I recommend starting with a 20 ml/kg bolus (10 L/adult horse or 1L/foal), reassessing clinical signs/physical examination parameters, and determining if additional boluses are required. In horses with diseases that are associated with protein loss, the use of colloids in the resuscitation plan might have additional benefits. In the case of hemorrhagic shock, whole blood should be considered as part of the fluid resuscitation plan.

Trocharization of the large intestine can be performed in the field in selected cases. Trocharization may be effective in relieving pain and distension of the large intestine in cases of non-strangulating lesions of the large intestine. For cases of severe spasmodic colic, this treatment may be definitive. Whereas in cases of displacement or impaction, trocharization may provide enough relief to allow further treatment and time for resolution. Although complications of trocharization appear to be rare, the potential for severe complications (rupture and abscess formation) is possible. These risks must be clearly explained to the owner prior to performing trocharization particularly in cases where referral (and/or surgery) is an option. The procedure is simple to perform. A site is chosen, generally the right flank, using clinical exam (ping, rectal, distension) and/or ultrasound. The site is clipped, prepared aseptically, and blocked. A 14 gauge catheter (over the needle) with an extension set is placed into a cup of water and slowly advanced with the stilette in place for 2-3 cm past where bubbles start exiting. Some clinicians will advance the catheter past the needle or remove the stilette at this time. My preference is to leave the stilette and catheter in the same location. Then, wait… Once the bubbles have stopped the catheter is removed. Some clinicians recommend injecting procaine penicillin G or gentamicin during removal. Some clinicians will also place the horse on a short course of antimicrobials.

Impactions can be effectively treated with oral laxatives. There are several mechanisms of action that cause loosening and/or softening of gastrointestinal contents by laxatives. Laxatives used in horses include bulk laxatives that provide volume and help retain water (psyllium), surfactants that help incorporate water and fat (dioctyl sodium sulfosuccinate [DSS]), lubricants that are slippery and decrease water absorption (mineral oil), and hydrating agents that help attract and retain water (isotonic balanced electrolyte solution or magnesium sulfate). DSS can be irritating and may facilitate the absorption of mineral oil or magnesium sulfate and associated toxicities. Overdoses of magnesium sulfate alone can also result in magnesium toxicity.

An isotonic balanced electrolyte solution has been shown to result in the largest increase in fecal water. Water has also been shown to effectively hydrate ingesta. Psyllium is frequently used for the prevention and treatment of sand impactions. Mineral oil can be used as a marker for intestinal transit in addition to it’s laxative activity. My preference for treating horses with large intestinal impactions is the use of isotonic balanced electrolyte solutions and/or water. When administering oral fluids/laxatives, I never exceed 6-8L of fluids at one administration time and always check for reflux prior to administration.

Specific treatments for horses with mild colitis include anti-endotoxic therapy with flunixin meglumine and di-tri-octahedral smectite. In blocking prostaglandin production, flunixin meglumine minimizes some of the inflammatory effects of endotoxin that contribute to the development of systemic inflammatory response syndrome. Flunixin meglumine can be administered at a low (quarter dose) every 8 hrs. Di-tri-octahedral smectite has been shown to adsorb endo- and exo-toxins. It can be administered via nasogastric tube or as an oral paste formulation and is thought to bind these toxins preventing them from entering the circulation and stimulating a cascade of inflammation.

Non-surgical treatments for left dorsal displacement of the large colon have been described and reported to have high success rates. Physical methods of jogging and rolling attempt to “jostle” the colon off of the nephrosplenic ligament. In order to roll a horse, the horse is induced into general anesthesia and rolled from right lateral into dorsal (jostled around) and then into left lateral recumbency.
During jogging and/or rolling, there is the chance of gastrointestinal rupture. The administration of phenylephrine has also been describe alone or combined with the physical methods. When administering phenylephrine horses should be monitored for reflex bradycardia and other arrhythmias. Fatal hemorrhage has been reported in horses administered phenylephrine. Most horses with hemorrhage were older, but owners should be informed of this risk.
There are a wide variety of indications for fluid administration in horses. They include volume restoration, rehydration, maintenance, ongoing losses, diuresis, electrolyte and/or acid base imbalance, parenteral nutrition, support of colloid osmotic pressure, immunoglobulin or coagulation protein supplementation, and improving oxygen carrying capacity. In general, fluids can be groups into crystalloids and colloids based on their composition. When developing a fluid plan, one of the first things to decide is whether the horse requires rapid, emergency fluid therapy or if you are developing a 24-hour fluid plan. The underlying disease and reason for fluid administration will dictate the appropriate options for fluid type, amount, and route of administration.

Crystalloid fluids are categorized based on their tonicity. Tonicity refers to the gradient of osmotic pressure between two solutions across a semipermeable membrane. In the case of fluids, the tonicity is determined by the impermeable osmoles in the fluid relative to the permeable osmoles within cells. Hypertonic fluids have more effective osmoles, isotonic fluids have a similar effective osmoles, and hypotonic fluids have less effective osmoles than within cells. In plasma and in fluids, the main osmole that determines osmotic pressure is Na⁺. Glucose and BUN also contribute to osmotic pressure, but because the concentration is so low in comparison to Na⁺ in plasma and in fluids, it is generally ignored. The formula for calculating plasma osmolarity is 2[Na⁺ (mmol/L)] + [Glucose (mg/dL)]/18 + [BUN (mg/dL)]/2.8.

In addition to being defined by tonicity, crystalloid fluids can also be categorized based on their electrolyte and buffer composition as well as whether they are replacement or maintenance fluids. Most crystalloids are either straight NaCl or balanced electrolyte solutions with a buffer (acetate or lactate). Replacement fluids generally mimic the electrolyte composition of extracellular fluid (high Na⁺ and low K⁺, Ca²⁺, and Mg²⁺) while maintenance fluids generally mimic electrolyte composition of intracellular fluid (low Na⁺ and high K⁺, Ca²⁺, and Mg²⁺). Due to the low Na⁺ concentration, maintenance fluids many contain dextrose to make them isotonic or are hypotonic.

Whether crystalloid fluids are being administered intravenously, subcutaneously, or orally/intragastrically, they are being added primarily to the extracellular fluid space. The extracellular fluid is composed primarily of plasma/intravascular fluid (1/5) and interstitial fluid (4/5). In some conditions transcellular or third space fluids are also included. Although vessel walls separate the plasma and interstitial fluid, the solutes can move fairly freely within the extracellular fluid space. For this reason, regardless of the route of fluid administration, redistribution within the extracellular space is expected. Most importantly, this results in retention of only 20% of intravascularly administered crystalloids within the intravascular space within approximately 30 minutes.

Colloids contain relatively large insoluble molecules that exert oncotic or colloid osmotic pressure (COP). Colloid solutions are grouped based on whether the molecules are natural or synthetic in origin. Natural colloids are blood products and albumin is the main molecule responsible for their COP. There are a variety of synthetic colloids, but the most widely available products for veterinary use currently are the hydroxethyl starch solutions (HESS). There are several HESS commercially available that are differentiated by the molecular weight of the hydroxethyl starch molecules as well as the carrier solution (isotonic NaCl or balanced polyionic solution).

Starling’s Equation is a simplified, theoretical explanation of the factors that control movement of fluid across the capillary wall. Fluid movement from the capillary into the interstitium is related to capillary wall permeability and area and the balance between hydrostatic and oncotic pressures within the capillary and the interstitium. The main force maintaining fluid within the capillary is plasma oncotic pressure.

Colloids are administered intravenously and the relatively large insoluble molecules remain within vascular space. As a result, administration of colloids contributes to the oncotic pressure. The increase in oncotic pressure not only decreases the movement of fluid from the vascular space into the interstitium, but can also pull fluid from the interstitium into the vascular space.

The most common indications for rapid, emergency fluid therapy are conditions that result in shock associated with decreased stroke volume due to decreased preload. Preload must be restored rapidly in order to prevent progression to decompressed shock and death. Less common indications for emergency fluid therapy include rapid correction of life threatening electrolyte and acid-base abnormalities or hypoglycemia. Most other conditions requiring fluid therapy can be treated less rapidly and frequently are best treated by designing a 24-hour fluid plan.

Shock occurs when the energy needs of cells are greater than the energy being delivered by the blood. Most commonly, there is a deficiency in oxygen delivery. Oxygen delivery is a product of oxygen content and cardiac output. Oxygen content is primarily dependent on [Hb] and SpO₂ and cardiac output is the product of heart rate and stroke volume with stroke volume determined by preload, afterload, and contractility. Although conditions that affect either component can result in shock, in horses it is more
common for shock to be caused by decreased cardiac output. Specifically horses are affected by conditions that result in decreased preload. The two main types of shock seen in horses that are due to decreased preload are hypovolemic and maldistribution of fluids. Examples of hypovolemic shock are hemorrhagic shock and fluid loss associated with large volume diarrhea or nasogastric reflux. Examples of maldistributive shock are endotoxic and septic shock that result in massive vasodilation and venous pooling of blood.

Clinical signs of hypovolemic and maldistributive shock are similar and associated with poor perfusion and decreased intravascular volume as well as the body’s attempts to improve oxygen delivery. Physical examination findings associated with poor perfusion and decreased intravascular volume are cool extremities, prolonged jugular refill, prolonged capillary refill time, poor pulse quality, and decreased/absent urination. Physical examination findings associated with the body’s attempts to improve oxygen delivery are increased heart rate, increased respiratory rate, decreased/absent urination, and production of concentrated urine. If available, handheld lactatometers and/or blood gas monitors can be used to assess plasma lactate concentrations to support inadequate oxygen delivery.

The goal of emergency fluid therapy is rapid restoration of preload, cardiac output, and oxygen delivery. This requires use of the intravenous route of fluid administration. A wide diameter (10-14g), short catheter with large bore fluid administration sets will maximize the speed of delivery. Depending on the type of fluid chosen and packaging, pressurized administration may be possible.

The two main alternatives for rapid volume resuscitation are: 1) using a relatively small volume of hypertonic saline (2-4 ml/kg or 1-2 L/adult horse [not generally recommended in foals]) and/or synthetic colloids (5-10 ml/kg or 2.5-5 L/adult horse or 250-500 ml/foal) to borrow/pull fluid from the interstitium into the vascular space followed by administration of larger volumes of isotonic crystalloids (at least 20 ml/kg) or 2) using isotonic crystalloid fluid alone. In either case, the “shock dose” of 80-90 ml/kg can be used as a guideline for the maximum amount of isotonic crystalloids to administer as a bolus. In general, it is rare to need the entire “shock dose”. I recommend starting with a 20 ml/kg bolus (10 L/adult horse or 1L/foal), reassessing clinical signs/physical examination parameters, and determining if additional boluses are required.

In horses with diseases that are associated with protein loss, the use of colloids in the resuscitation plan might have additional benefits. In the case of hemorrhagic shock, whole blood should be considered as part of the fluid resuscitation plan.

There are three main components to consider when calculating the volume of fluid for a 24-hour plan: maintenance, dehydration, and ongoing losses. In adult horses, maintenance requirements are 50 ml/kg/day and in foals, maintenance requirements are 80-100 ml/kg/day. In horses, estimating dehydration is very difficult. Traditional clinical signs of dehydration such as skin tent and dry/tacky mucus membranes have been shown to be completely inaccurate. Ongoing losses may be easy to measure, such as with horses that are producing nasogastric reflux, or difficult to determine, such as with horses with diarrhea or polyuria.

The route of administration for a 24-hour fluid therapy plan can be intravenous or oral/intragastric (as long as the gastrointestinal tract is functional). Oral administration of fluids is as effective for dehydration and more effective for hydration of ingesta compared to intravenous fluids.

Depending on the underlying disease, the electrolyte composition and colloid requirement will be variable. Although isotonic, polyionic crystalloids are generally a safe first choice, clinical pathology is useful for tailoring fluid therapy.
The bifurcation of the trachea divides the upper from the lower respiratory tract. The upper respiratory tract includes the nares, nasal passages, pharynx, larynx, and trachea. The lower respiratory tract is separated into the airways (bronchi, bronchioles, and alveoli), vasculature, interstitium, and pleural space. Differential diagnoses for respiratory distress can also be divided by anatomic location. Within the upper respiratory tract, bilateral nasal passage obstruction, mechanical and functional pharyngeal or laryngeal obstruction, and less commonly tracheal obstructions are possible. Examples of nasal passage obstructions include congenital choanal atresia or masses within the nasal passage or sinus (infectious, neoplastic, sinus cysts). Examples of pharyngeal obstructions include neurologic dysfunction or physical obstruction associated with swelling within the pharyngeal musculature (bleeding with longus capitus rupture), guttural pouches (typany), or retropharyngeal lymphadenopathy. Examples of laryngeal obstruction include bilateral laryngeal paralysis associated with systemic disease or toxins or physical obstructions such as artenoid chondritis or subepiglottic cysts. Examples of tracheal diseases include intratracheal masses and tracheal collapse. Within the lower respiratory tract, infectious and non-infectious diseases of the airways, vasculature, interstitium and pleural space can result in respiratory distress. Inflammatory diseases of the airway such as RAO and SPAOPD can result in respiratory distress. Infection of the lower respiratory tract in horses affecting the parenchyma and airways alone (pneumonia) or involve the pleural space as well (pleuropneumonia) can also result in respiratory distress. Other diseases of the pleural space that can result in respiratory distress include hemotherax, pneumothorax, or pleural masses. Vascular causes of respiratory distress are very uncommon, but would include persistent fetal circulation and pulmonary hypertension secondary to congestive heart failure.

Observation of clinical signs and physical examination should be performed rapidly. They are generally sufficient to determine which portion of the respiratory tract is affected and often significantly further localize and prioritize differential diagnoses. The most obvious, consistent, and important clinical sign is respiratory noise. Horses that are making a respiratory noise have an upper respiratory cause of distress. Respiratory noise is the result of turbulent airflow. Turbulence, and respiratory noise, is most likely to occur during inspiration in the entire upper respiratory tract except the intra-thoracic trachea. This is because intra-airway pressure is negative, thereby promoting collapse and airflow disruption, during inspiration in the extra-thoracic airway. With more complete, rigid obstructions, respiratory noise may not be limited to the inspiratory phase. Further localization of the upper respiratory obstruction can be achieved with auscultation along the upper respiratory tract with a stethoscope to determine the point of maximal intensity, palpation along the upper respiratory tract for swelling, and dull percussion of the sinuses. Phase of dyspnea and symmetry of airflow and nasal discharge are important physical examination components for both upper and lower respiratory causes of distress. As with noise, dyspnea of the extra-thoracic airway will occur during inspiration and of the intra-thoracic airway will occur during expiration most frequently. Diseases of the pleural space decrease lung capacity and will result in a rapid, shallow breathing pattern. Changes in airflow can be detected by placing your hands or a mirror in front of the horse’s nostrils. Decreased/obstructed airflow and nasal discharge will be unilateral rostral to the nasal septum. Diseases of the pharynx that are related to the guttural pouch may also result in unilateral signs. Diseases caudal to the pharynx will uniformly result in bilateral decreased airflow and/or nasal discharge. Thoracic auscultation is the most important physical examination component for further localization of lower respiratory causes of respiratory distress. In horses in distress, rebreathing examination should only be performed if adequate examination is not possible without increasing respiratory effort and it is does not cause excessive distress to the horse. Abnormalities detected on auscultation may include increased bronchovesicular sounds, crackles, wheezes, or decreased/absent bronchovesicular sounds. Crackles are heard when fluid filled airways snap open and closed. This means that they occur when alveoli are filled with fluid preventing surfactant from minimizing surface tension appropriately. Wheezes occur when the airway is narrowed. Regional increases in bronchovesicular sounds (harsh or tubular lung sounds) can occur in areas of airway inflammation or consolidated lung. Regional decreases or absence of lung sounds can occur when portions of the lung are obliterated by abscesses or masses or, more commonly, secondary to accumulation of fluid (ventral) or air (dorsal) in the pleural space. Thoracic auscultation in horses with upper respiratory causes of distress will result in hearing referred upper airway noise. Once the upper airway obstruction is relieved, full thoracic auscultation should be repeated to ensure there is no lower airway involvement. Percussion of the thorax is an additional component of the physical examination that may be useful for localizing lower respiratory tract problems. It is useful to detect air/fluid or air/tissue interfaces such as detecting the extent of fluid in the pleural space in horses with pleuropneumonia or localizing areas of pulmonary abscesses, masses, or consolidation.

Once the cause of respiratory distress has been localized, emergency treatment can begin. Temporary tracheostomy is a life saving emergency procedure for horses with upper respiratory obstruction of the nares, nasal passages, pharynx, larynx, and trachea proximal to the tracheostomy site. Horses with lesions of the trachea distal to the temporary tracheostomy will not respond unless a tube can be
passed through the tracheostomy site past the obstructed area. In horses with lower respiratory tract causes of respiratory distress, intranasal oxygen therapy, if available, may improve oxygenation and clinical signs of distress. Which other emergency treatment is indicated will depend on the further localization and characterization of the cause.

When done on an “elective” basis, temporary tracheostomy is simple to perform and has a low incidence of complications. When performed in horses with severe respiratory distress, the risks of complications may increase because steps to ensure asepsis are often skipped and careful dissection is forgone in order to establish an airway as quickly as possible. For these reasons, I recommend performing the tracheostomy sooner than later in several situations to avoid difficulty, complications associated with a tracheostomy, and, most importantly, death. First, in horses that are at risk for developing respiratory distress during diagnostic procedures and/or awaiting improvement with treatment. Second, in horses that are at risk for developing respiratory distress and are going to be out of the direct supervision of a person (i.e. veterinarian) qualified to perform a tracheostomy, such as on a trailer or at a farm. Additionally, I strongly recommend that practitioners keep a preassembled kit, including include local anesthetic, gloves, a scalpel handle and blade, scissors, hemostats, 4 x 4’s, and several sizes of tracheostomy tube, for performing an emergency tracheostomy accessible in their truck and clinic. At minimum, it is valuable to have several options of sterilized, commercial tracheostomy tubes. Although a makeshift tracheostomy tube can be made from items a veterinary typically carries, they do require some manufacturing and therefore are not immediately available. Additionally, it is almost impossible to make one with a diameter as large as the commercially available tubes. They are also more difficult to place and maintain.

When performing a temporary tracheostomy, if the horse is excitable and/or distressed, sedation can be used in patients with upper respiratory distress. In fact, decreasing the respiratory effort with sedatives can reduce the degree of obstruction by decreasing the degree of negative intra-airway pressure. However, it should be noted that it also impairs pharyngeal and laryngeal function. If distress is very severe horses are frequently panicked and dangerous. In these cases it may be necessary to wait for the horse to pass out/fall down. This is obviously not a good situation for the horse or the veterinarian. Temporary tracheostomy is most commonly performed at the level of the junction of the cranial 1/3 and caudal 2/3 of the cervical trachea. Anatomically, this is located just cranial to the point where the sternocephalicus splits. In this location, there is less musculature overlying the trachea making tracheostomy easier to perform. The location may need to be modified if the horse has had a tracheostomy in the past or if it is likely that the horse will be having a permanent tracheostomy in the near future. If a permanent tracheostomy is expected, the tracheostomy should be performed as close to the split of the right and left sternocephalicus muscles to avoid interference with the surgical procedure. The selected area should be clipped and prepared aseptically. A line block with local anesthetic should be performed on ventral midline approximately 12-15 cm in length. In horses with severe obstruction, these steps may need to be brief or even omitted. The procedure begins with a 10-12 cm incision through the skin and cutaneous coli muscle on ventral midline. Division of the underlying paired sternothyrohyoideus muscles on midline will minimize bleeding. Pale fibrous tissue separates the left and right sides and can be incised with a scalpel or bluntly divided with scissors. Blunt dissection will minimize bleeding and prevent damage to the underlying trachea, but use of a scalpel is faster. The sternothyrohyoideus muscles are separated for a length of approximately 8 cm. This exposes several tracheal rings and the connecting annular ligaments. There is a loose connective tissue that overlies the trachea. Blunt dissection with scissors or using a sterile 4 x 4 results in better visualization. However, this step should be skipped in animals with severe respiratory distress. A scalpel blade is used to make a transverse stab incision through an annular ligament. It is very important to use a stab incision to enter the trachea to avoid separating the mucosal layer from the outer layers. Air will escape when the mucosa is incised. The incision is then extended with the scalpel blade or scissors. The tracheostomy should not exceed 1/3 to 1/2 of the circumference of the trachea. This is important to avoid injury to the adjacent vessels and nerves as well as stenosis of the trachea after removal. A variety of commercial temporary tracheostomy tubes are available. Tubes can be made of plastic or metal and may be self-retaining or may necessitate suturing to the skin or securing with gauze around the neck. If an inflatable cuff is present on the tube, it SHOULD NOT be inflated because pressure necrosis of the mucosa is possible. The largest tracheostomy tube that will fit should be used. My preference is the metal self-retaining tubes. If softer, plastic tubes are used, those that have a tapered end are generally easier to place. If no commercial tube is available a shortened plastic syringe or syringe case (as large as possible) can be used. A short piece of syringe or syringe case can be used to maintain separation of the rings in combination with a nasogastric tube to allow for a more flexible tube within the trachea. Other materials that have been used include the handle of a gallon milk jug and garden hoses. If tracheal collapse distal to the site of the tracheostomy is the cause of respiratory distress, a long endotracheal tube can be passed through the area of collapse to prevent collapse. Sutures encircling the tracheal rings adjacent to the tracheostomy site can be placed to make placement and replacement of the tube easier. Although they are not generally required, they can be useful when using stiff tubing. Upper respiratory obstruction can result in pulmonary edema due to alterations in intra-thoracic pressure, capillary permeability, and pulmonary vasculature tone. If present, foam may be evident in the trachea, crackles may be heard on thoracic auscultation, and the respiratory distress may not respond as expected to the tracheostomy. Treatment for pulmonary edema secondary to upper respiratory obstruction includes intravenous furosemide (1-2 mg/kg) (with caution in animals that are dehydrated) and oxygen if available. Following periods of respiratory distress and hypoxia, supplemental intranasal oxygen may benefit the patient.
Emergency treatment of horses with respiratory distress associated with inflammatory lower airway disease includes bronchodilators, corticosteroids, and environmental management. Bronchodilators can be administered intravenously, orally, or via inhalation from a metered dose inhaler with commercially available masks for horses. Bronchodilators are important to provide symptomatic relief, but do not treat the underlying cause of the disease. In an emergency situation, oral bronchodilators are the least rapid option and therefore not particularly useful. Intravenous anticholinergic drugs such as n-butylscopolamine and atropine are potent bronchodilators. Inhaled bronchodilators act rapidly, but also have a short duration of action and require repetitive dosing. Clenbuterol and albuterol are β-2 agonists and ipratropium bromide is an anticholinergic available as inhalers. Corticosteroids are the most potent anti-inflammatory drugs available and are a mainstay of treatment for inflammatory lower airway disease. As with bronchodilators, intravenous and/or inhaled administration will be preferred in an emergency situation. Inhaled corticosteroids may avoid some of the complications associated with systemic corticosteroids while providing high local levels of anti-inflammatory therapy. Environmental control is vital in the long-term management of patients with inflammatory airway disease and should begin immediately.

Emergency treatment of horses with infectious airway and interstitial disease (pneumonia) should include appropriate antimicrobials and anti-inflammatories. However, it is important to remember that these will take time to work and that the horse’s level of distress may not improve much immediately beyond what it does with oxygen therapy.

Emergency treatment of respiratory distress associated with diseases of the pleural space may result in rapid improvement of the respiratory distress if the space occupying material in the pleural cavity can be removed (fluid or air, rather than a mass). Due to the effect of gravity, fluid accumulated ventrally and air accumulates dorsally. The site for fluid removal can be chosen empirically in the cranioventral lung field avoiding the region of the heart or with the ultrasound guidance. The site for air removal is generally chosen empirically and placed dorsally around the 13th intercostal space. Removal of fluid or air can be achieved with intermittent thoracocentesis using a teat cannula or continuous or intermittent drainage with an indwelling chest drain (8-10 french catheter). Removal can be active (applying suction to the end of the tubing) or passive. Placement of a teat cannula or indwelling drain is performed in the same manner. Placement at the cranial aspect of a rib avoids the intercostal vessels that run along the caudal edge of each rib. The area selected is clipped and prepared aseptically and local anesthetic is infused in the subcutaneous tissues and intercostal muscles. An incision just long enough to accommodate the teat cannula or drain is made in the skin, subcutaneous tissue and external fascia of the intercostal muscles. The teat cannula or drain is then inserted and advanced into the pleural space bluntly. Care should be taken to assure a slow and controlled advancement into the pleural cavity and precautions should be taken to avoid air being sucked into the pleural space during inspiration (negative pressure within the pleural space). When using a teat cannula, extension tubing should be connected to a three way stop-cock and the skin should be closed when the teat cannula is removed. When using an indwelling chest drain, hemostats, a one way valve (Heimlich), or active suction can be applied to prevent air entering the pleural space and the tubing should be secured with a purse-string suture and Chinese finger trap to prevent leakage around the tube. Distress associated with bilateral accumulation of fluid or air may or may not respond to unilateral drainage depending on the amount of fluid or air that has accumulated and whether the mediastinum is incomplete or complete (in horses with pleuropneumonia, inflammatory proteins frequently clog fenestrations in the mediastinum). Complications can be associated with draining the pleural cavity, particularly if rapid drainage is performed. “Re-expansion pulmonary edema” is reported secondary to rapid drainage of pneumothorax in people. Evacuation of the air with suction applied at no more than 20 cm H2O is recommended. Cardiovascular compromise associated with rapid removal of fluid from the third space can occur when large quantities of fluids are removed. Intravenous fluid support and controlled drainage should minimize this effect.

Once emergency treatment has been performed to improve stability and make the horse as comfortable as possible, further diagnostics can be performed to identify the underlying cause and choose the most appropriate therapy. For the upper respiratory tract, the most useful diagnostic for all causes will be upper airway endoscopy because it allows direct visualization of the obstruction. Additional diagnostics that may be indicated include radiographs, ultrasound, histopathology, hematology, and microbiology. Similarly, for the lower respiratory tract, diagnostic imaging, histopathology, hematology, and microbiology may be indicated. Diagnostic imaging of the lower respiratory tract frequently involves the combination of radiographs and ultrasound. Ultrasound can be performed rapidly with portable ultrasound machines whereas radiographs of an adult horse’s chest requires large x-ray units generally only available in referral hospital settings. Ultrasound provides information on the pleural space and the periphery of the lung while radiographs provide information on the lung parenchyma. Samples from the lower respiratory tract may include fluid obtained via transtracheal wash, bronchoalveolar lavage, and thoracocentesis or tissue obtained via biopsy.
The first question to answer is: what is shock? By definition, shock occurs when cells are unable to produce enough energy (adenosine 5'-triphosphate [ATP]) to meet the demands of the cell to function. Cells metabolize nutrients, such as glucose and volatile fatty acids, to generate ATP. To produce the most ATP per unit of nutrient, this process requires oxygen. For this reason, energy production can be limited by decreased availability of either nutrients or oxygen. It is far more common for a reduction in ATP production to be due to decreased oxygen availability than to insufficient nutrients. Specifically, most often shock is the result of inadequate amounts of oxygen reaching the cells (oxygen delivery [DO2]). In rare cases, adequate nutrients and oxygen reach the cells, but the cells are unable to perform aerobic metabolism or the cells use more energy than normal due to a disease process.

Because most shock is caused by decreased DO2, understanding DO2 is the key to understanding how diseases result in shock, how the body compensates for shock, which clinical signs are associated with shock and compensation, and how treatments will improve shock. Oxygen delivery (DO2) is the product of cardiac output (Q) and arterial oxygen content (C\textsubscript{a}O\textsubscript{2}) (DO2=Q×C\textsubscript{a}O\textsubscript{2}). Q is the product of stroke volume (SV) and heart rate (HR) (Q=SV×HR). SV is affected by filling of the cardiac chambers (i.e., preload), the force against which the heart contracts (i.e., vascular resistance or afterload), and the strength of cardiac contractions (i.e., contractility). SV is directly proportional to preload and contractility and inversely proportional to afterload. C\textsubscript{a}O\textsubscript{2} is the product of oxygen carried by hemoglobin and dissolved in the blood (P\textsubscript{a}O\textsubscript{2}). The amount of oxygen carried by hemoglobin is the product of hemoglobin concentration in the blood ([Hb]) and the saturation of that hemoglobin with oxygen (S\textsubscript{a}O\textsubscript{2}). It is very important to recognize that the vast majority of oxygen in the blood is carried by hemoglobin and that very little is dissolved.

(C\textsubscript{a}O\textsubscript{2}=1.34×[Hb]×S\textsubscript{a}O\textsubscript{2}+0.003×P\textsubscript{a}O\textsubscript{2}).

There are a variety of ways to classify the causes of shock. One of the more complete and simple methods divides the causes of shock into 5 classifications: hypovolemic, distributive, obstructive, cardiogenic, and metabolic. Hypovolemic, distributive, obstructive, and cardiogenic shock all cause decreased DO2 by decreasing Q. In contrast, metabolic shock causes shock by a variety of mechanisms that can either decrease energy production or increase energy requirements by cells. Keep in mind that this classification scheme is overly simplified and often does not completely explain what is occurring in the patient due to the complexity of the diseases that cause shock with multiple pathophysiologic changes occur concurrently.

Diseases that result in a decreased circulating (intravascular) volume are associated with hypovolemic shock. Decreased intravascular volume causes a decrease in preload, leading to a decrease in SV leading to a reduction in Q and a decrease in DO2. The decreased circulating volume can be due to losses of whole blood (hemorrhage) or free water or due to severe and/or prolonged lack of fluid intake. Fluid can be lost outside of the body or sequestered within the body, but outside of the circulating volume. The latter situation is referred to as ‘third space’ loss. In horses, diseases such as middle uterine artery rupture, guttural pouch mycosis, or large artery lacerations secondary to trauma can result in hemorrhagic shock. Examples of diseases in horses that result in fluid loss include gastrointestinal losses in diarrhea in animals with colitis or nasogastric reflux with anterior enteritis or losses in sweat. In horses, fluid can be sequestered within the colon in horses with a large colon volvulus or within the pleural space with pleuropneumonia.

Diseases that result in relative hypovolemia (i.e., a normal amount of intravascular fluid is distributed throughout an expanded intravascular space) are associated with distributive shock. Like true hypovolemia, relative hypovolemia causes a reduction in preload, which leads to a decreased SV leading to reduced Q and a decrease in DO2. The expansion of the intravascular space is most often due to widespread vasodilation associated with the release of cytokines and inflammatory mediators from damaged or inflamed tissue. In horses, infectious and non-infectious triggers can result in a systemic inflammatory response syndrome (SIRS) and severe vasodilation. One trigger of SIRS is endotoxin. Endotoxin, also known as lipopolysaccharide (LPS), is a component of the cell wall of gram-negative bacteria that is released during times of rapid bacterial death or reproduction. These endotoxin molecules gain access to the systemic circulation, where they initiate a strong inflammatory response. Endotoxemia is common in diseases such as colitis and retained placenta due to increased availability of endotoxins combined with compromise in the integrity of the normal mucosal barrier to their absorption. Other causes of SIRS in horses are bacteremia secondary to failure of passive transfer in neonates and ischemic bowel associated with strangulating obstructions.

Diseases that cause physical obstructions of blood flow to or from the heart are associated with obstructive shock. When blood flow returning to the heart is obstructed, preload decreases, whereas obstruction of blood flow from the heart results in an increase in afterload. In either case, this leads to a decrease in SV leading to a decreased Q and decreased DO2. In horses, diseases that obstruct blood flow returning to the heart are more common than those obstructing blood flow from the heart. Severe gastrointestinal tract distention can increase intra-abdominal pressure and compress the caudal vena cava, limiting venous return to the heart and reducing preload. Pericardial limits the heart’s ability to expand, effectively decreasing preload.
Diseases that affect the heart primarily are associated with cardiogenic shock. Cardiogenic shock can be due to a decrease in HR or SV (due to a reduction in preload, increase in afterload, or decrease in contractility) leading to decreases in Q and DO2. In horses, reductions in heart rate (i.e., bradyarrhythmias) can be associated with 3rd degree AV block or hyperkalemia secondary to ruptured bladder. Cardiac diseases that can be associated with decreased preload include AV valve regurgitation with or without cordae tendinorum rupture, and tachyarrhythmias such as ventricular tachycardia. Cardiac diseases that can be associated with decreased contractility include influenza and Strep. equi-related cardiomyopathy and monensin toxicity.

Most diseases in this category of shock reduce [Hb], SaO2, or PaO2 leading to decreased CieO2 leading to decreased DO2. Less commonly, diseases can cause decreased nutrient availability to the cell, impair utilization of oxygen by the cell, or increase energy requirements of the cell. In horses, diseases that cause decreased [Hb] included red maple leaf toxicosis and immune mediated anemia. Diseases that decrease SSO2 and PaO2 are related to impaired oxygen uptake due to severe respiratory disease such as pneumonia. Other causes for metabolic shock include insulin resistance, which decreases the nutrients available to the cell, cyanide poisoning, which impairs the ability of the cell to utilize oxygen, and sepsis, which increases the energy requirements of the cells.

There are three stages of shock determined by how well the body is compensating in order to meet the energy demands of the cells. During shock, the body focuses on meeting the needs of the most “vital” structures, such as the heart and the brain, potentially to the detriment of less “vital” structures such as the gastrointestinal tract.

The body’s response to shock is triggered by detection of a variety of abnormalities associated with decreased DO2 (acidosis, hypercapnea, and hypoxemia), decreased intravascular water (detected by osmoreceptors in the hypothalamus), and decreased arterial blood pressure (detected by baroreceptors in the aortic body and carotid sinus). Responses mediated through the sympathetic nervous system and renin-angiotensin pathway are key to the body’s ability to compensate for these abnormalities. Through a relatively complex series of events, the body is able to improve DO2 and nutrient delivery and also limit energy requirements by minimizing non-essential functions. Improvement in Q is through increasing preload (shifting interstitial fluid to intravascular space, retaining fluid at the level of the kidney, and vasoconstricting to maintain blood pressure), increasing contractility, and increasing heart rate. Improvement in CieO2 occurs by increasing the respiratory rate (thereby maximizing PaO2 and SaO2) and splenic contraction to increase [Hb]. Nutrient delivery is increased with stimulation of gluconeogenesis and protein synthesis.

The clinical signs of each stage of shock are associated with the compensatory mechanisms and how well they maintain Q and organ function. Stage I is compensated (a.k.a. hyperdynamic) shock. During stage I, blood flow to the heart and brain is maintained through compensatory mechanisms. Due to these mechanisms, clinical signs include tachycardia, tachypnea, hyperemic mucous membranes, decreased capillary refill time, and bounding pulses. During this phase, mentation should remain fairly normal because the brain is receiving enough energy. Stage II is early decompensated shock. During stage II, energy demands are not being met, resulting in increased anaerobic metabolism and lactate production as well as organ dysfunction. The clinical signs associated with the compensatory mechanisms, including tachycardia and tachypnea, are still present. However, during stage II, signs of inadequate Q (increased CRT, cold appendages, decreased blood pressure) and organ dysfunction (decreased urine production and abnormal mentation) become apparent. Stage III is late decompensated (a.k.a. Irreversible) shock. During stage III, anaerobic metabolism continues and sympathetic compensation with vasoconstriction is overwhelmed, resulting in blood pooling in venules, fluid leaking into the interstitium, and activation of inflammation, as evidenced by SIRS. With inflammation, coagulation is also activated throughout the body resulting in disseminated intravascular coagulation (DIC). Microvascular thrombosis associated with DIC combined with dwindling energy delivery leads to organ failure and death. Because compensatory mechanisms have been overwhelmed, clinical signs of compensation are no longer present. Instead, clinical signs are related to cardiovascular collapse (marked hypotension, bradycardia, pale/gray mucous membranes) and organ failure. Once stage III is reached, recovery is rare even with aggressive treatment.

These proceedings are an adaptation of an iBook created for teaching purposes at the University of Georgia.
Accurate and thorough wound evaluation is vital to being able to provide the owner with information about treatment options, possible complications, and prognosis. The single most valuable tool for the veterinarian when evaluating wounds is a solid knowledge of anatomy. Although the appearance, configuration and location of the skin wound are obviously an important component, involvement of underlying structures often is the limiting factor in prognosis. As with all emergencies, the first step will be to evaluate the horse’s systemic status. Although relatively rare, any systemic compromise should be addressed before proceeding. In order to evaluate a wound fully, the horse must be appropriately restrained and the wound must be properly prepared. After these steps are taken, based on anatomic location evaluation of the appropriate underlying soft tissues (vessels, nerves, muscles, tendons, and ligaments), bone, and synovial structures can be performed.

In this presentation, interactive case discussions will be used to illustrate the techniques required to fully evaluate wounds. The details of the cases are not provided to stimulate more discussion.
Emergency treatment of horses with limb wounds varies dramatically in complexity depending on location and involvement of underlying structures. The single most valuable tool for the veterinarian when treating wounds is a solid knowledge of anatomy. Treatment of the wound can begin only after the horse’s systemic status has been assessed, the horse has been stabilized if necessary (rare), and the extent of the wound has been determined. Considerations for emergency wound treatment will include addressing limb instability, inflammation, contamination or infection, wound repair, dead space management, and bandaging/coaptation.

In this presentation, interactive case discussions will be used to illustrate the techniques required during emergency wound treatment in several locations. The details of the cases are not provided to stimulate more discussion.
The variety of acute wounds presenting to the equine practitioner offers a challenge to select the most appropriate management to facilitate healing. A complete wound history along with anatomic and specific medical considerations for each patient provides the basis of decision making for wound management. It is essential to evaluate the entire animal and apply an evidence-based approach. The practitioner should consider each wound individually in order to create the optimal conditions for wound healing.

**Patient assessment**

Assessment of any patient with suspected acute traumatic injury should be systematic and thorough. The size of the wound does not necessarily dictate severity and very small wounds are often overlooked when a large traumatic wound is present. Particularly in horses, the small wound can be the real danger and significant often fatal consequences result.

A useful tool for patient assessment following trauma follows the acronym “A CRASH PLAN” and will be used for this series of case evaluations.

- Airway
- C- Circulation/Cardiovascular
- R- Respiratory
- A- Abdomen
- S- Spine
- H- Head
- P- Pelvis
- L- Limbs
- A- Arteries
- N- Nerves

**Trauma types**

- Blount trauma: can be very high energy; falls over a jump, vehicular trauma, etc.
- Penetrating wounds: most common; lacerations, puncture wounds, impalements, foreign body
- Combination of Blount and Penetrating: often times deceiving in extent of injury

**Assessment of specific wounds**

- Mechanism of Injury
- Depth/Size
- Contamination
- Age of wound
- Structures involved (superficial and underlying)
- Tissue vitality
- Tension
- Temperament of patient
- Environment
- Follow-up care

Use all available information to determine the most appropriate treatment plan. Consider referral if hospital conditions are more appropriate for management or advanced diagnostics are needed.
Puncture wounds and lacerations are a common occurrence in equine practice. No matter how pristine a pasture is, horses seem to find some way to injure themselves. This session will discuss particular wound locations that should warrant greater concern.

**Wounds affecting synovial structures**

Wounds of the limb are at risk of entering synovial structures (joints, tendon sheaths, and bursae). If left untreated, these wounds can result in infection within the synovial cavity leading to permanent disability of the horse. Knowledge of the specific anatomy of the limbs is essential to identifying potential synovial involvement early (Figure 1). Synovial structures that are most often penetrated include: digital tendon sheath, metacarpo/metatarsophalangeal joint, and tarsal joints.[1-3]

Lacerations or puncture wounds into synovial structures often directly introduce bacteria and contaminants (e.g., hair, dander, dirt, etc.) into the synovial space. Once bacteria overwhelm the natural defenses of the synovial structure, detrimental effects on cartilage, synovium and other associated synovial structures happen very quickly. The longer the duration of the infection, the greater the likelihood for permanent damage to the synovial structure. Multiple retrospective studies have provided mixed reports regarding the effect of time until treatment on the outcome of horses with infected synovial structures due to wounds.[4-9] Though early treatment is best; cases that have had synovial sepsis longer than 24 hours can still carry a good prognosis with appropriate treatment.

All wound near synovial structures should be cleaned thoroughly, and the hair from the wound edges should be clipped. Once the wound is adequately cleaned the wound should be explored with sterile gloves. Synoviocentesis should be performed for all neighboring synovial structures. The sample should be submitted for synovial analysis in an ethylenediamine tetraacetic acid (EDTA) blood tube. Normal synovial fluid should have nucleated cell counts less than 1000 cells/L, of which most are mononuclear cells (macrophages and lymphocytes).[10] Total protein concentration is approximately 20-25% of plasma protein of the same animal.[11] Generally this should be less than 2.0 g/dL. A differential cell count is also important for a complete assessment. There should be less than 10% neutrophils.[10] A sample should synovial fluid should also be submitted for culture and sensitivity whenever possible.

Confirmation of communication between a wound and synovial structure may not always be straightforward. Synovial fluid analysis is beneficial but those results may not be available immediately. The most common method of confirming communication is to distend the synovial structure and watch for fluid to flow from the wound. Other methods include radiography and ultrasonography.

Management of wounds that involve synovial structures should be targeted at efficient removal of contamination and inflammation. Lavage of the synovial structure is usually necessary for removal of bacteria from the synovial space. This is often combined with regional and/or systemic antimicrobial therapies. Prognosis can be variable. Factors associated with a poor prognosis are increased intrasynovial total protein concentration at the time of admission,[4] positive culture results from synovial fluid,[2] and evidence of osteolysis.[12]

**Wounds affecting bones, tendons, and ligaments**

If a wound is distant to a synovial structure, you may not be able to take a sigh of relief yet. The force by which many of these wounds are afflicted can cause damage to structural components of the musculoskeletal system. Figure 2A depicts a kick wound to the medial antebrachium further examination reveal incomplete fractures of the radius (Figure 2B). Sharp lacerations, even when small, can also cause significant damage to the underlying soft tissue structures. Imaging is an crucial component of many wounds on the limbs of horses, especially if the degree of lameness is worse than what is expected for the size of the wound.

**Hemorrhage**

Hemorrhage in the horse can be classified based on rate (acute or chronic), severity (controlled or uncontrolled), location and cause. With these classifications in mind, treatment is based on clinical assessment, resolution of cause, prevention of ongoing losses and treatment of resulting circulatory perturbations.

**Diagnosis and estimation of blood loss**

Estimation of blood volume loss can be difficult to determine particularly when the hemorrhage is partially internal or external blood loss was not witnessed. Initially, packed cell volume and plasma protein levels may be normal due to blood components lost in equal volumes. Additionally, the erythrocyte storage capacity of the equine spleen and resulting splenic contraction in acute hemorrhage may mask initial changes in the hemogram. Changes in red blood cell numbers and total protein may remain within the normal reference range for up to 24 hours even in cases where a large volume of blood is lost. However, after vascular equilibration does
occur, a decrease in packed cell volume, hemoglobin, and panhypoproteinemia will be apparent. A neutrophilic leukocytosis may also be present by 3 hours after hemorrhage.

Blood loss can be estimated based on clinical signs and other dynamic indices even in the absence of visualized hemorrhage. Hypovolemic shock resulting from hemorrhage is divided into 4 classes based on percentage loss of total blood volume. The clinical signs (sweating, tachycardia, an anxiety) described in these classes are also consistent with pain and it is important to differentiate the underlying cause. Class I involves blood loss of less than 15% blood volume and can be compensated with little to no change in physical parameters or clinical signs other than decreased urine output. Class II (15-30% blood loss) results in hyperdynamic shock and the inability of the body to completely compensate for the volume of blood lost. Clinical signs include tachycardia, tachypnea, and bounding pulses. The patient may be anxious and sweating. Blood pressure may be normalized but a metabolic acidosis will result with increased blood lactate levels. Class III-IV (>30% or continued uncontrolled blood loss results in compensatory shock with marked tachycardia and tachypnea present. Capillary refill time and jugular filling are prolonged, peripheral vasoconstriction causes cold extremities and urine output is diminished. Metabolic acidosis is severe and blood lactate levels may be profoundly increased. Without intervention cellular hypoxia and acidosis results in complete decompensation, failure of cardiac function, circulatory collapse and death.

**Treatment for acute blood loss**

The first steps in treatment of acute hemorrhage are location determination and reduction or cessation of further blood loss. When hemorrhage is external, treatment involves application of direct pressure and/or ligation of lacerated vessels. Internal hemorrhage may be more difficult to diagnose. Epistaxis warrants airway endoscopy to evaluate upper and lower airway sources of hemorrhage including guttural pouches (mycosis or ventral straight muscle rupture) and pulmonary vessel rupture. Hemothorax and hemaabdomen can be diagnosed with radiography, ultrasound and thoraco- or abdominocentesis. In cases of abdominal hemorrhage, an exploratory celiotomy should not be performed unless there is confirmation of the source and a high likelihood of successful ligation.

Autotransfusion from body cavities permits the body to reuse blood components and approximately two thirds of the erythrocytes lost into the abdomen or thorax are resorbed within 24 to 72 hours. The remaining one third are lysed or phagocytized and the iron and protein are reused.

Hemorrhage may be reduced by administration of aminocarproic acid, an anti-fibrinolytic lysine derivative, which may enhance clot maintenance possibly through a reduction in partial thromboplastin time (PTT). Due to its short half-life, this medication has been shown to provide adequate therapeutic levels using a constant rate infusion of 3.5 mg/kg/min for 15 minutes, followed by 0.25 mg/kg/min. In the field, a bolus dose of 30 mg/kg may provide some benefit, if a CRI is not possible.

**Conclusion**

Acute wounds in the equine should be evaluated as quickly as possible and all scenarios with regards to involved structures should be assessed. Very small and seemingly insignificant wounds may be overlooked especially when multiple wounds are present. Additionally, in the equine patient the size of the external wound often has very little effect on the ultimate prognosis. The direct puncture wound into the carpus is much more dangerous than a large pectoral laceration and will often get the attention of the owner more quickly. It should also be noted that horses with severe gastrointestinal disease may present with self-induced trauma. If the overall system presentation does not fit with a seemingly simple wound, reevaluate! It is important to educate clients to these facts so that prompt notification of the equine practitioner occurs and appropriate therapy is initiated. All body systems should be evaluated for potential involvement and the overall status of the patient should not be forgotten. Cardiovascular, respiratory and neurologic status can play a major role in treatment plans.

**References**

Chronic Wounds:
New Options for Old Wounds
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Chronic, non-healing wounds can be particularly frustrating for the practitioner. It is often difficult to have access to a complete history with respect to the wound. Regardless, the evaluation of chronic wounds should follow the same systematic process used for all wounds. The way in which a wound is managed in the early stages has a dramatic effect on the quality and speed of healing. Delayed appropriate treatment of wounds limits treatment options and will likely affect the prognosis and quality of healing.

Exuberant granulation tissue
The development of exuberant granulation tissue (EGT) or “proud flesh” has long been a thorn in the side of the equine practitioner. Despite extensive evaluation of this phenomenon in the equine, there is no universal treatment option and multi-modal treatment is often required for the chronic wound with excessive granulation tissue formation. Fibroplasia, or the formation of granulation tissue, is a required phase of wound healing but exuberant production is difficult to predict. There are a number of factors which are known contributors to the development of proud flesh. Individual inflammatory response, wound location, presence of infection, and initial treatment are all contributing factors.

Treatment options for EGT include both chemical and physical means. Small to moderate amounts of granulation tissue can be managed with the application of topical corticosteroid cream which inhibit inflammation and fibroplasia. The use of corticosteroids is controversial in that they are also known to inhibit angiogenesis, wound contraction and epithelialization. Therefore, the application should be infrequent and limited to the EGT, sparing surrounding tissues. Alternatively, sharp excision is an excellent means of treating EGT. This is particularly helpful in chronic wounds with large proliferative masses of EGT.

Epithelialization cannot extend “uphill” upon the edge of the EGT and becomes stagnant over time. Quite often chronic wounds will appear larger than they actually are due to the mushroom-like proliferation of tissue extending over wound edges. Removing the excessive tissue may reveal a wound of a smaller size than expected. Removal renews the potential for epithelialization along wound edges. The excess granulation tissue should be trimmed flush with the healing epithelial edge. Granulation tissue is aneural so local anesthesia is not required when the excision is limited to the EGT. Additionally, it is highly vascular so a tourniquet may be used to reduce hemorrhage and preserve visualization. Blood loss is unlikely to be of a dangerous volume but it can be quite unsettling for a client observing the procedure. The wound will likely continue to bleed until a padded bandage is placed. Multiple trimmings of the granulation tissue is necessary in many cases. However, if the wound fails to progress toward healing other causes of delayed healing should be investigated.

Differential diagnoses
Exuberant granulation tissue is often confused with tumors in the equine, particularly sarcomas. Contributing to this is the reality that an equine wound can transform into a sarcoma or other tumor. The history of a known wound at the site does not preclude the sarcoma diagnosis. This transformation can occur at any wound site but distal limb wound sarcomas are fibroblastic in nature, mimicking the presence of EGT. Horses with sarcomas present at other sites are more prone to sarcoma transformation of wounds. Sarcoma or other neoplasia should be suspected in any chronic wound which is not healing with what is deemed to be appropriate management. In these cases biopsy and histologic evaluation is warranted.

Habronemiasis and the presence of “summer sores” can occur in open wounds and take on the appearance of EGT, sarcomas or squamous cell carcinoma. With this condition the wound may also have calcified granules contained within and be pruritic. As with any non-healing wound a biopsy is appropriate for diagnosis.

Excessive motion
Excessive motion can inhibit healing when all other aspects of the wound are appropriate and healing well. Measures to reduce motion include stall confinement, bandaging and splint or cast application. Often these measures result in a dramatic increase in the speed and quality of healing. Owners are often reluctant to stall confine but the treatment time may be so significantly reduced with a short period of bandaging and stall confinement, it should be encouraged when possible.

Conclusions
The management of chronic equine wounds in the field can be challenging and require dedication and commitment on the part of practitioner and owner, alike. The importance of diligent wound care and bandaging especially in the lower limb cannot be over emphasized. Many horribly chronic, seemingly hopeless wounds can be successfully managed with the appropriate tools.
Colic in Foals:
Never too Young
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Evaluation of colic in foals is particularly challenging for several reasons. Foals differ from adults in presentation of clinical signs, etiologies and in some diagnostic tools available to the practitioner. The essential elements of the colic exam are still present and additional diagnostics unique to the foal may be helpful. The decision for exploratory celiotomy in foals is a particularly difficult decision to make due to the risks involved in surgery at that age.

Signalment and history
The signalment and history can be particularly important in differentiating the cause of colic in foals. Age is an important factor pointing toward certain etiologies. For example, meconium impactions are seen most commonly in the newborn while ruptured bladder or congenital anomalies such as atresia coli usually cause clinical signs in 2-5 day old foals. Small intestinal volvulus, intussusceptions and gastric outflow obstructions are more common in older foals while ulcers and enteritis can be found in foals of any age.

Some causes of colic are more common or exclusively found in particular breeds such as lethal white syndrome in overo Paint horses. The gender of the foal may also indicate risk for certain types of colic. For example, colts are more likely to be diagnosed with ruptured bladders.

Physical examination
A complete and thorough physical examination is imperative in the evaluation of foals with colic. Underlying issues such as failure of passive transfer/sepsis, neonatal encephalopathy, fractured ribs, musculoskeletal problems and congenital abnormalities can result in colic signs or make diagnosis more difficult. A foal that is unable to stand due to profound weakness, sepsis or musculoskeletal abnormalities with be more difficult to assess. Colic signs may be more subtle if the foal is weak. Physical examination should be directed at the entire foal with evaluation of all physiologic parameters. Evaluation of the foal should include temperature, pulse, respiratory rate, mucous membrane color and capillary refill time. The adequacy of passive transfer and measurement of immunoglobulins should be evaluated and a complete blood count, serum biochemistry and blood culture should be obtained whenever possible. Low serum IgG concentrations will be an important factor in management of the colicky foal and more than one plasma transfusion may be necessary during the course of treatment.

Gastrointestinal examination: diagnostic techniques
Abdominal circumference can be serially measured in order to establish a baseline and appreciate increasing or decreasing abdominal distention through the course of treatment. Abdominal distention may be due to peritoneal fluid or gas or fluid accumulation in the gastrointestinal tract. GI tract distention is more evident in the foal than the adult horse and can result from either small intestinal or large colon distention.

Digital rectal examination is an important diagnostic tool particularly when meconium impaction is suspected in the neonate. The impaction can often be palpated in the pelvic canal. Meconium impaction may result in partial or complete obstruction, the latter causing marked abdominal distention on physical examination. Diarrhea in foals may also be preceded by abdominal distention and signs of colic.

Nasogastric intubation should be performed in any colicky foal. It is more difficult to perform than in the adult and it is helpful to measure the distance to the stomach before beginning intubation. A stallion catheter with stylet in place is the appropriate size for gastric intubation in most neonates. As is true in adults showing colic signs lack of nasogastric reflux does not rule out small intestinal disease in the foal.

Abdominal ultrasonography is particularly helpful in evaluation of the colicky foal and has become routine. It can be performed with the foal standing or recumbent. Clipping the hair may be necessary for good probe to skin contact and visualization. Ultrasound is useful for visualization of distended loops of small intestine including enteritis, intussusceptions, and other obstructions. A large portion of the small intestine can be visualized in neonates but it is more obscured in older foals as the large colon develops at around 4 months of age. Enteritis will appear as hypermotile, fluid-filled small intestinal loops. Intussusceptions may appear as a target lesion or multiple concentric rings on a single cross-section of the GI tract. It is also helpful in the diagnosis of uroabdomen and the bladder defect can occasionally be visualized. Ultrasonographic identification of peritoneal fluid increases the safety of abdominocentesis in foals and is used routinely for this purpose. While a 3-5 mHz probe is most useful for abdominal evaluation, any commonly used probe has the ability to penetrate the abdomen of the foal and can be useful.
Abdominal radiography is much more commonly utilized in the foal than it is in the adult colic but use has declined with the availability of quality ultrasound imaging. Abdominal radiography remains particularly helpful in evaluation of large bowel obstructions such as sand or meconium impaction as well as in the presence of peritoneal effusion.

Abdominocentesis is a useful diagnostic in the foal but should be performed with caution due to the increased risk of bowel laceration and peritoneal contamination in the foal when compared to the adult. Abdominal ultrasonography is useful to determine the presence of peritoneal fluid and aid in sampling success. Abdominocentesis is not always necessary to decide that surgery is warranted especially if diagnostic imaging is available to the practitioner.

**Common causes of colic in the foal**

**Congenital anomalies**

Congenital Anomalies must always be considered in the neonate. Atresia coli, atresia recti, and atresia ani have each been described in the foal. Most foals are born asymptomatic and begin to show signs of colic and abdominal distention at 24-48 hours with a history of no fecal production. In some cases abdominal radiography may reveal a blind stump of colon filled with gas. Many are diagnosed at exploratory celiotomy and successful anastomosis of bowel segments is rare.

Overo Lethal White Syndrome or uleocecolic agangllosis is a genetic defect recognized in homozygous, overo, paint foals. Usually these foals are completely white but may also have a few small spots of color.

In this genetic disease an endothelin receptor B mutation results in incomplete aganglosiosis of the myenteric and submucosal ganglia of the intestinal tract. Affected foals appear normal at birth but rapidly develop signs of colic, profound ileus, and intestinal distention. A diagnosis of lethal white syndrome is based on clinical signs coupled with ultrasonographic evidence of profound ileus. Unfortunately, there is no treatment and euthanasia is strongly recommended. A genetic test is available to identify heterozygous individuals and should be recommended in future breedings.

**Meconium impaction**

Meconium impaction is the most common cause of colic in the neonate. Clinical signs of meconium impaction include decreased nursing, straining to defecate with an arched back, flagging of tail, and lack of meconium production. Particularly early on or with incomplete obstruction foals may show intermittent colic signs that responds well to analgesia. A digital rectal examination may reveal hard fecal material in the rectum. Complete obstruction may cause significant and grossly evident abdominal distention to develop. In severe cases profound abdominal distension can result in respiratory compromise. Radiographs or ultrasound may reveal fecal material in the distal colon or rectum. A barium enema can be performed for further evaluation and to attempt to rule out focal atresia of the intestinal tract. Treatment of meconium impactions includes enemas, analgesics, oral laxatives, IV fluids and supportive care. An enema consisting of 200-500 ml of warm soapy (Ivory or other gentle soap) water is the first line of treatment. This should be performed using a small, well-lubricated catheter and gravity flow. Multiple enemas with a detergent such as dioctyl sodium succinate (DSS) are discouraged due rectal and colonic irritation and resulting edema as well as DSS toxicity. Retention enemas using acetylcysteine can be performed with caution if soapy water enemas prove unsuccessful in reducing the impaction. The foal should be sedated for this procedure as distention of the rectum may cause discomfort. A Foley catheter is inserted 2-4 inches into the rectum and the balloon is distended, using caution not to overinflate. Infuse 100-200 ml of 4% acetylcysteine solution into the rectum and clamp the Foley catheter. The fluid should remain in place for 15 minutes before deflating and removing the catheter. Manual evacuation of meconium using forceps should never be performed due to the risk of iatrogenic tissue damage. Occasionally, surgical reduction via celiotomy is required.

**Inguinal hernias**

Foals may be born with inguinal hernias or they may develop in the first 2-3 days of life. Foals born with congenital inguinal hernias are often not colicky particularly if the content of the hernia is easily reducible. Many can be successfully treated by repeated manual reduction or application of a truss bandage. If the contents becomes strangulated it will be firm on palpation and not reducible. Most of these occur as direct hernias when the vaginal tunic has ruptured and bowel passes through to subcutaneous tissues. The foal will quickly show signs of discomfort and emergency celiotomy is the required treatment.

**Uroperitoneum**

Uroperitoneum is most commonly diagnosed in foals at 2-3 days of age although the rupture may have occurred at the time of parturition. Uroperitoneum can occur from rupture of the urachus, ureter or bladder with the same resulting clinical presentation. Foals most often present with abdominal distention, low-grade pain and frequently posture to urinate. Symptoms can be similar to those exhibited by foals with meconium impaction and further diagnostics are often required for differentiation between the two. Subcutaneous rupture at the urachus may cause yellowish skin discoloration and subcutaneous swelling. The serum chemistry abnormalities of foals with uroperitoneum classically include hyponatremia, hypocloremia and hyperkalemia. Foals receiving intravenous fluids and supportive care for other abnormalities may not exhibit these electrolyte derangements and diagnosis may be more challenging. A peritoneal fluid/serum creatinine ration of >2:1 is diagnostic. As stated earlier, ultrasound is helpful in obtaining peritoneal fluid and a tear in the bladder can sometimes be visualized. Medical treatment involves stabilization and correction of electrolyte abnormalities. Intravenous fluids should not contain potassium as marked hyperkalemia is common. While uroperitoneum...
is an emergency which ultimately necessitates abdominal surgery for correction, surgery should be delayed until the patient is stable, serum potassium levels are below 5.5 mEq/L and thus a better anesthetic candidate.

**Diaphragmatic hernias**

Neonates may present with diaphragmatic hernias and are often associated with birthing trauma. The integrity of the ribs should be evaluated and any foal in with rib fractures could have a resulting diaphragmatic hernia. Congenital diaphragmatic hernias are also possible and the presenting foal may be up to several weeks old. Diagnosis is usually suspected with plain radiographs and thoracic ultrasound and confirmed on exploratory celiotomy.

**Indications for surgical treatment**

The indications for surgical exploration of foals are similar to those in adults. Persistence of pain despite appropriate analgesia, progressive abdominal distention or surgical diagnosis on ultrasound or radiography are all common indications for surgical management.
Dealing with Thoracic and Abdominal Trauma
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Trauma or lacerations of the abdomen and thorax have the capacity to heal remarkably well provided deeper structures are not involved. However, wounds involving the thoracic and abdominal cavity are particularly worrisome due to the potential for significant internal damage with minimal external trauma. Definitive investigation of the depth of these wounds is essential for appropriate management.

Aseptic technique is imperative for evaluation in these areas if thoracic or abdominal penetration is suspected.

Thoracic wounds
Thoracic trauma may involve rib fractures or penetration of the pleural space with pneumothorax or hemothorax. It may be possible to hear a “sucking” sound if the environment is quiet indicating pleural space involvement. Thoracic auscultation may also be helpful but radiography or ultrasonography may be necessary for definitive diagnosis. If the pleural space is involved pneumothorax may cause respiratory distress and removal or air by thoracocentesis may become necessary. These wounds should be explored for foreign bodies and only closed primarily if the wound is deemed clean of contamination and there is adequate tissue to do so. Second intention healing is a viable option in most thoracic wounds not involving deeper structures and allows for an adequate cosmetic outcome.

Abdominal wounds
Abdominal wounds may be challenging to diagnose with regards to depth and thus peritoneal cavity involvement. Tissue planes may shift after wounding and depth may not be immediately apparent. An abdominocentesis may be of benefit in the diagnosis particularly if hemoabdomen is suspected but normal parameters do not rule out peritoneal penetration. Additionally, large wounds with extensive tissue loss are at risk of herniation even if the wound does not penetrate the abdomen. Initial management should involve cleaning the wound aseptically and evaluating for foreign materials and depth. Like thoracic wounds, many non-penetrating abdominal wounds heal by second intention with acceptable cosmesis.

Inguinal and axillary wounds
Axillary wounds in horses have the potential to induced widespread subcutaneous emphysema, due to entrapment of air in the wound. As the horse advances the involved forelimb the wound gaps open filling with air. This air becomes trapped and is then “pumped” into the SQ by further motion of the forelimbs. Depending on the configuration of the wound SQ emphysema can lead to pneumomediastinum which can rarely advance to become a pneumothorax. If pneumomediastinum or pneumothorax develop they must be treated appropriately (see above). The SQ emphysema is usually limited and resolves once the wound is sealed. Packing the wound may decrease the severity of emphysema but the best way to decrease severity is to limit the movement of the horse to only what is absolutely necessary. Once it forms it cannot be removed and must be resorbed over time. Primary closure of these wounds is often difficult due to loss of tissue and may not be sufficient to prevent subcutaneous emphysema form forming. Wound dressing which promoting granulation tissue development should be used.

Inguinal wounds commonly involve fence post or tree branches and should be evaluated extensively for depth of penetration and foreign body material. Due to location it may be necessary to explore these wounds under general anesthesia in dorsal recumbency. The extent of the wound may not be evident with the horse in a standing position.

Head and neck wounds
Wounds involving the head are most frequently the result of horses running into a stationary object, tossing the head into overhanging objects or being kicked. The list of structures that can be involved in head trauma is extensive and involves many body systems. Many of these can be extremely cosmetically or athletically detrimental or even fatal. It is important to inspect for possible fractures and sinus involvement. Additionally, injuries to the head may result in neurologic signs or respiratory distress due to swelling or impaired function. It is important perform a thorough evaluation for neurologic signs as this may be the most significant indicator of prognosis. Respiratory distress may occur at some time after the initial injury due to inflammation and swelling occluding the upper airway. A temporary tracheotomy may be necessary in these cases.

Lacerations of the head are common and are usually presented with less contamination than other areas of the body. The head has an excellent blood supply and many lacerations will heal well given tissue loss is not too extensive. Lacerations of the eyelids, nostrils, lips, tongue and ears present more of a challenge because preservation of function is of paramount importance. This relies heavily on reconstruction and primary or delayed primary closure should be attempted whenever possible.
Wounds involving the facial bones, sinuses and orbit may be more extensive than initial examination indicates particularly when the skin is minimally affected. Fractures may be present even when the skin isn’t open. Radiography is warranted to evaluate the extent of these fractures and determine involvement of sinuses or orbital rim. Overlying soft tissues should be preserved whenever possible to facilitate closure and protection of underlying osseous structures. It is important to remember the anatomically position of sinuses, nasolacrimal duct and facial nerves when evaluating and repairing wounds of the head.

Neck wounds are most commonly caused by barbed or high-tensile wire fencing. They are usually presented in a horizontal orientation and often occur at the base of the neck. Usually only the skin, subcutaneous tissues and underlying muscles are involved. Involvement of the trachea, esophagus or underlying neurovascular structures is possible and may change the prognosis associated with these wounds considerably. Like head trauma, a tracheotomy may need to be performed if there are signs of respiratory distress. If the neck wound involves only skin, subcutaneous tissues and some underlying muscle the prognosis is favorable. Primary closure should be performed wherever possible. A neck cradle may be necessary to reduce tension on the sutures and prevent dehiscence.
The equine upper airway is a high, resistance, low-capacity path between the nares and lungs. Proper function of the equine upper airway requires the coordinated effort of a large number of anatomical structures. Very small changes in diameter of the upper airway are magnified in their effect on the ability of the horse to move air into the lower respiratory system and oxygenate tissue effectively. For example, a 20% decrease in airway radius doubles airway resistance. Disease diagnosis of the equine upper airway can be a challenge due to the dynamic nature of many abnormalities. The airway may appear mildly affected or even completely normal on resting endoscopy and have severe functional abnormalities at times of increased respiratory effort. Additionally, some abnormalities are intermittent in their appearance and only manifested under certain situations such as increased head and neck flexion. (McGorum 2007)

The use of radiography and resting endoscopy has been the mainstay of upper airway diagnosis for several years. Many abnormalities affecting the upper airway can be diagnosed using these modalities. More recently the use of such modalities as dynamic endoscopy, either on a treadmill or over-ground, and ultrasonography has increased the tools in the diagnostician’s arsenal. Resting endoscopy permits the diagnosis of more advanced grades of laryngeal hemiplegia, arytenoid chondritis, persistent dorsal displacement of the soft palate (DDSP), persistent epiglottic entrapment, subepiglottic cysts and fourth branchial arch defects. Radiography is a valuable secondary tool to evaluate epiglottic and palatopharyngeal arch position as well as subepiglottic cysts and laryngeal cartilage mineralization.

Dynamic over-ground and treadmill endoscopy
Dynamic endoscopy has several advantages for diagnosis of upper airway abnormalities. Some abnormalities only occur during exercise particularly at maximum-exertion when negative pressure is highest. Also, the actual effect of pathology on athletic performance can be observed and response to treatment documented. Often multiple, related abnormalities are causing poor performance or noise while only one may be diagnosed on resting endoscopy. Upper airway abnormalities detectable only under dynamic, exercising conditions include some grades of laryngeal hemiplegia, axial deviation of the aryepiglottic folds, intermittent DDSP, intermittent epiglottic entrapment, pharyngeal collapse and epiglottic retroversion. In approximately 30% of horses with upper airway abnormalities, more than one abnormality is actually present. (Lane, Bladon et al. 2006)

Dynamic upper airway endoscopy can be performed either on a treadmill or over-ground using an endoscope fitted to the patient. Both modalities allow for the detection of dynamic abnormalities but there are differences between them. Treadmill upper airway endoscopy allows direct manipulation of the patient’s exercising speed and incline but it is much less flexible in other important variables which can be introduced to the examination. Additionally, a period of training is required for the patient to decrease the risk of injury during examination. Advantages of over-ground dynamic upper airway examination include the ability to exercise the horse in the normal environment where the abnormality occurs. It is a useful tool for both sport horses and race horses. Variables that can be introduced include exercise under saddle, exercise with significant head-neck flexion, or training on the track in the company of other horses. Both modalities represent significant advances in the diagnosis of equine upper airway disorders.

Laryngeal ultrasound
The use of laryngeal ultrasound for the diagnosis and management of upper airway disorders in the equine has become a valuable tool in recent years. It is most useful in evaluation of the laryngeal cartilages and intrinsic musculature. Advantages of this diagnostic modality are expense and non-invasive nature. Unlike MRI or CT the technique does not require general anesthesia but rather mild sedation. In combination with resting upper airway endoscopy, laryngeal ultrasound is useful for diagnosis and follow-up of arytenoid chondritis and diagnosis of left laryngeal hemiplegia. (Garrett, Woodie et al. 2011)

The technique is performed with a 7-10 megahertz microconvex or linear transducer. Both lateral and ventral image windows can be obtained. The lateral image window orienting the probe in a longitudinal and transverse plane is used to assess the echogenicity of the cricoarytenoideus lateralis and cricoarytenoideus dorsalis muscles for identification of laryngeal hemiplegia. Neuromuscular dysfunction results in hyperechogenicity of these muscles evident on ultrasound.

Computed tomography
Computed Tomography (CT) is particularly useful for the evaluation of diseases within the paranasal sinuses particularly masses, tooth root abscesses, fractures and sinusitis of other etiologies. The detail of osseous structures obtained with CT is greater than that for magnetic resonance imaging (MRI) and CT has become a valuable tool for planning surgical intervention in these cases. The acquisition time is short and three dimensional reconstructions can be quickly created.
References
Sedation and Local Anesthesia for Standing Surgery
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The ability to adequately sedate and offer pain management is an essential part of veterinary care of the equine patient. Unfortunately, these areas do not appear to have advanced as rapidly in equine medicine as they have in the treatment of small animals. Several limitations exist which may explain this dichotomy. High expectations exist for sedation and analgesia in the equine patient. Sedation should keep the horse still with all four feet on the ground but not induce recumbency. There should be no or little effect on motility of the gastrointestinal tract. Likewise, analgesia should not inhibit normal gastrointestinal function. In the case of orthopedic disease, pain relief should be sufficient enough to allow full and even weight distribution, less support-limb laminitis develop. Dosage and route should be practical for the field situation. Finally, these compounds need to be economical for the average size horse. Despite these requirements, certain areas of sedation and analgesia have made recent advances.

Many surgical procedures can be performed in the standing horse in either a field or hospital situation. Regardless of the procedure, meticulous planning and adequate restraint with appropriate analgesia are essential for a successful outcome. Additionally, thorough knowledge of what complications can occur and how to deal with them are just as important as technical skills when any surgery is performed. With these tools in hand, standing surgery in a field situation can be rewarding for the equine practitioner.

Sedation and chemical restraint

Phenothiazine tranquilizers
Phenothiazines are primarily used for the calming effect and are often combined with other drugs. They are not thought to have analgesic properties themselves and are thus quite useful for calming the fractious horse during a lameness exam, training or traveling where analgesia (and ataxia) are not desired. Phenothiazines act by blocking the action of dopamine both centrally and peripherally. This action can lead to arterial hypotension and they should not be used in horses with cardiovascular compromise from hemorrhage or dehydration. There is concern for use with stallions as persistent penile paralysis has been rarely reported. Acepromazine is the most commonly used phenothiazine and is available as a 1% solution (10 mg/ml) which can be delivered by IM, IV or oral route. The onset of action is around 30 minutes and lasts for 4-6 hours.

Alpha-2 agonists
Alpha-2 agonists produce a variety of effects including sedation, muscle relaxation and analgesia by binding to and stimulating alpha-2 adrenergic receptors both peripherally and in the central nervous system. Xylazine, detomidine and romifidine are approved for use in the United States. The level of sedation is pronounced and they are each useful for performing standing procedures although they vary in the length of sedation and degree of ataxia produced. Additionally, the horse will take on a saw-horse stance but still may still become rapidly aroused and kick. For this reason, alpha-2 agonists are often augmented with opioids, particularly butorphanol for more balanced sedation. Romifidine is the most recent alpha-2 agonist to be approved for use in horses and is reported to cause less severe ataxia while maintaining adequate sedation.

Opioids
Opioids are often combined with alpha-2 agonists for a more profound and longer lasting sedation. The use of opioids alone can cause excitement and nervousness and are thus not used for sedation in this manner. The most commonly used opioid is butorphanol, a synthetic agonist/antagonist, which can be combined with detomidine for a variety of standing procedures. Opioids provide significant analgesia but still need to be combined with local anesthesia for standing surgical procedures.

Butorphanol administered at a dose of 0.01-0.1 mg/kg IV or IM is the most commonly used opioid for the equine patient. There is considerable variability in the amount of analgesia produced with this drug but under the best circumstances analgesia can be excellent and last up to 4 hours.

Recently advances have been made in the delivery of opioid analgesics. The use of transdermal fentanyl has been reported in horses although the drug is not approved for use in this species. The most common dose is two 100-microgram/g patches per 450-kg horse which can provide analgesia within 4 hours of application. The patches are applied to a clipped area of the skin and secured with a bandage for good skin contact. Reapplication should occur every 48 hours.

Epidural use of morphine is a reliable, inexpensive route for analgesia to the hindlimbs. Any medication introduced into the epidural space must be done with strict aseptic technique but this is worthwhile when a balanced and significant analgesic plan is required. The recommended dose is 0.1 to 0.2 mg/kg diluted in a volume of 20ml with 0.9% saline. The onset of action is 20-30 minutes and analgesia can last up to 16 hours. This route of administration offers the benefit of morphine analgesia without the side effects of CNS excitement which can occur with systemic administration. Epidural analgesia is particularly helpful in cases where treatment for hindlimb injury is underway but risk of support-limb laminitis is apparent.
Gabapentin
The newest area of interest for analgesia in the equine patient is that of neuropathic pain. Neuropathic pain is pain which is initiated or caused by a primary lesion or dysfunction in the nervous system. It is associated with chronic disease and cannot usually be alleviated with traditional analgesics such as opioids. This area is of particular interest in treating diseases such as laminitis where pain appears to be upregulated and is unfortunately difficult to manage with more traditional medication. Gabapentin, a gamma-aminobutyric acid (GABA) analogue, originally developed to treat epilepsy in humans has shown promise as an adjunct therapy in refractory, chronic pain in the equine patient.

Sedation protocols for standing procedures
Alpha-2 agonists are likely an important part of any standing surgical procedure. Xylazine sedation has a duration of 20-30 minutes and, while useful for brief sedation, does not last long enough for most surgical procedures. Detomidine is approximately 100 times as potent and lasts twice as long as xylazine. Detomidine can be used alone or in combination with an opioid such as butorphanol for standing surgery. It can be administered as a single or “as needed” dose of 0.01-0.02 mg/kg IV and provides reliable sedation for approximately 60 minutes. Additionally, it can be administered as an IV variable rate infusion which offers prolonged sedation while decreasing the total dose infused. For IV infusion, an IV catheter is placed and 6 micrograms/kg is administered as a bolus. Butorphanol can be used with this initial bolus at a dose of 0.01-0.03 mg.kg IV. The infusion is prepared by adding 24 micrograms/kg of detomidine to a 250 ml bag of 0.9% saline and is administered with a 15 drop/ml IV administration set. Begin the infusion at 2 drops/sec and serially half the rate every 15 minutes. The rate can be adjusted depending on the sedation level of the individual patient and butorphanol can be administered as needed.

This will provide adequate sedation and analgesia for restraint but local anesthetic will likely be required depending on the procedure to be performed. Local anesthetic plans can include perineural infusion, epidural and regional infusion and will depend on the location and specifics of the surgery. Both lidocaine and mepivacaine are routinely used for perineural and local infusion. Mepivacaine is probably the most widely used of the local anesthetics and has less vasodilatory activity and causes less local edema than lidocaine. Anesthesia can last for 1-2 hours with lidocaine and 2-4 hours with mepivacaine.

Surgeries of the eyelid and ear

Eyelid
The frontal nerve, a branch of the supraorbital nerve, provides sensory innervation to the medial and middle two thirds of the upper lid. This nerve is anesthetized as it emerges from the supraorbital foramen, palpated as a depression 2.5 cm above the supraorbital process, by inserting a 25 gauge needle into the foramen and depositing 1-2 ml of local anesthetic.

Other nerves providing sensory innervations to the skin around the eye include the infratrochlear, lacrimal and zygomatic nerves which can be anesthetized as they cross the rim of the orbit or by local infiltration in the skin at the surgical site.

Motor innervation to the periocular cutaneous muscles which are responsible for closure of the lids is provided by the palpebral nerve, a branch of the auriculopalpebral nerve. This nerve can be blocked where is crosses the zygomatic arch dorsolaterally by infusion of 3-6 ml of local anesthetic with a 25 gauge needle.

Ear
Local anesthesia of the ear can be performed by blocking the internal auricular nerve and great auricular nerve. The location of the internal auricular nerve is palpable as a notch or depression on the lateral aspect at the base of the auricular cartilage. At this location, anesthetic is deposited by directing a 25 gauge needle into the notch and depositing 2 ml of local anesthetic. The great auricular nerve is palpable at the caudal aspect of the base of the pinna. Again 2-3 ml can be infused over the nerve using a 25 gauge needle.

Surgeries of the paranasal sinuses
Trephination and lavage of both the frontal and maxillary sinuses can readily be performed in the standing patient with adequate sedation and local anesthetic. After infusion of local anesthetic both in the subcutaneous tissue and periosteum at the location of trephination, a stab incision is made at the location of trephination. A Steinmann pin and hand-held chuck or Michelle trephine are used to create a hole in the sinus adequate in size for the insertion of the infusion port of a standard drip-set. The location of trephination sites are as follows: for the frontal sinus, 60% of the distance in a lateral direction from midline to the medial canthus and 0.5 cm caudal to the medial canthus; for the caudal maxillary sinus, 2 cm rostral and 2 cm ventral to the medial canthus; and for the rostral maxillary sinus, 50% of the distance from the rostral end of the facial crest to the level of the medial canthus and 1 cm ventral to a line joining the infraorbital foramen and the medial canthus. The surgical site can be left open and bandaged for repeat lavage.

Surgeries of the perianal area and vulva
Local infusion of anesthetic or epidural anesthetic will be necessary for any surgery involving the tail, perianal area or vulva. Surgeries such as a Caslick’s procedure or small mass removal can be easily performed with local anesthetic infusion however, the time spent to perform epidural for more invasive or prolonged procedures is time well spent. Injection is performed in the space between the first and second coccygeal vertebrae. The site is located cranial to the tail hairs and is determined by moving the tail up and down. After aseptic preparation of the site, bleb of local anesthetic is placed to facilitate the procedure. The epidural is performed with an 18 gauge spinal needle with stylet. The needle is introduced on midline with the bevel in a cranial direction, perpendicular to
the skin. A “popping” sensation may be felt when the ligamentum flavum is penetrated and an audible “hiss” may be apparent when the epidural space is entered. A drop of sterile saline placed in the hub of the needle will be aspirated into the space when encountered. A maximum of 10 mls is usually used to prevent paralysis of the lumbosacral nerves. A combination of lidocaine (0.22mg/kg) and xylazine (0.17 mg/kg) expanded to a total volume of 8 mls with sterile saline offers reliable analgesia. Sedation and ataxia may occur with epidural administration. A tail-tie is usually enough to provide support in the face of mild to moderate ataxia. Recumbency is a rare but possible result of epidural anesthesia.

**Surgeries of the distal limb**

Surgery of the distal limbs including wound evaluation, laceration repair, or coffin bone debridement can be performed with use of routine perineural nerve blocks and adequate sedation.

**Conclusion**

A combination of sedation and local anesthetic will allow the performance of many surgical procedures in the standing horse. The individual animal temperament and environmental conditions as well as safety of the patient, veterinarian and assistants must be evaluated when deciding if standing surgery is appropriate. Regardless of the surgical procedure, appropriate antimicrobial and anti-inflammatory medications should be used and tetanus toxoid should always be current. Most importantly, aseptic technique is as important in the standing patient as it is under general anesthesia. Keeping those facts in mind, many procedures can be successfully performed standing negating the risks and expense of general anesthesia.
Standing Surgical Procedures:
No GA? No Problem!
Laura Riggs, DVM, PhD, DACVS
Louisiana State University
Baton Rouge, LA

Many surgical procedures can be performed in the standing horse in either a field or hospital situation. Regardless of the procedure, meticulous planning, appropriate patient selection and adequate restraint with appropriate analgesia are essential for a successful outcome. Additionally, thorough knowledge of what complications can occur and how to deal with them are just as important as technical skills when any surgery is performed. Many surgeries are simple as long as nothing goes wrong. With these tools in hand, standing surgery can be rewarding for the equine practitioner.

Sedation and restraint
Alpha-2 agonists are likely an important part of any standing surgical procedure. Xylazine sedation has a duration of 20-30 minutes and, while useful for brief sedation, does not last long enough for most surgical procedures. Detomidine is approximately 100 times as potent and lasts twice as long as xylazine. Detomidine can be used alone or in combination with an opioid such as butorphanol for standing surgery. It can be administered as a single or “as needed” dose of 0.01-0.02 mg/kg IV and provides reliable sedation for approximately 60 minutes. Additionally, it can be administered as an IV variable rate infusion which offers prolonged sedation while decreasing the total dose infused. For IV infusion, an IV catheter is placed and 6 micrograms/kg is administered as a bolus. Butorphanol can be used with this initial bolus at a dose of 0.01-0.03 mg/kg IV. The infusion is prepared by adding 24 micrograms/kg of detomidine to a 250 ml bag of 0.9% saline and is administered with a 15 drop/ml IV administration set. Begin the infusion at 2 drops/sec and serially half the rate every 15 minutes. The rate can be adjusted depending on the sedation level of the individual patient and butorphanol can be administered as needed.

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Paranasal sinuses
Standing surgery of the paranasal sinuses offers several advantages over performance under general anesthesia. Complications and cost associated with general anesthesia is avoided. Less hemorrhage occurs and there is better visualization of sinus anatomy. Successful standing surgery requires adequate sedation and restraint. The use of stocks is recommended. Adequate steady support for the head is a must and this can be accomplished by a trained assistant or head stand. The practitioner must have a thorough understanding of the anatomic structures involved.

Trephination and lavage of both the frontal and maxillary sinuses can readily be performed in the standing patient with adequate sedation and local anesthetic. After infusion of local anesthetic both in the subcutaneous tissue and periosteum at the location of trephination, a stab incision is made at the location of trephination. A Steimann pin and hand-held chuck or Michelle trephine are used to create a hole in the sinus adequate in size for the insertion of the infusion port of a standard drip-set. The location of
Subsequent damage to the periosteum and a loss of blood supply leads to necrosis of a superficial segment of bone. The result is sequestrum formation, which is a common sequelae to trauma common to the equine limb such as kicks, lacerations, and penetrating wounds. Sequestrectomy is the surgical removal of the diseased pedal bone and debrided to remove the sequestrum and abnormal bone. The defect is then packed with betadine soaked gauze and the foot is appropriately bandaged and covered with an impermeable dressing such as duct tape. The defect must be treated and allowed to heal by second intention.

Surgery of the distal limbs including wound evaluation, laceration repair, or coffin bone debridement can be performed with use of routine perineural nerve blocks and adequate sedation. Once the location of disease is confirmed pedal osteitis and sequestrum formation can be treated using standing debridement with the following technique.

After adequate sedation an abaxial sesamoid block is performed with mepivacaine on the affected foot. The foot is surgically prepared by scrubbing and soaking in a dilute betadine solution. The distal limb proximal to the foot is wrapped to limit contamination. A burr or sterilized hoof knife is used to create a defect at the determined location. The defect is extended to the level of the diseased pedal bone and debrided to remove the sequestrum and abnormal bone. The defect is then packed with betadine soaked gauze and the foot is appropriately bandaged and covered with an impermeable dressing such as duct tape. The defect must be treated and allowed to heal by second intention.

Sequestrum formation is a common sequelae to trauma common to the equine limb such as kicks, lacerations, and penetrating wounds. Subsequent damage to the periosteum and a loss of blood supply leads to necrosis of a superficial segment of bone. The result is devitalized bone and sequestrum formation. This may take several days to weeks to occur and the owner may be unaware at that point of the inciting traumatic incident. Reactive bone forms around the necrotic sequestrum and is commonly referred to as the involucrum. Although wounds involving loss of soft tissue coverage, periosteal damage and desiccation of bone are most at risk of sequestrum development the diagnosis is not made until lysis around the bone becomes extensive enough to be evident radiographically. Often the diagnosis is not made until a wound fails to heal appropriately or begins to drain after antimicrobials are discontinued. Diagnosis is frequently delayed when the failure to heal appropriately is blamed on inappropriate antimicrobial choice or length of treatment. Radiographs may be unrewarding until the lysis becomes more apparent. A cloaca or draining tract often develops which leads the practitioner directly to the area of bone to be addressed. A small sequestrum may be treated successfully without surgical intervention given a prolonged period of time with appropriate antimicrobial therapy but treatment may actually be less expensive with definitive surgical removal of the sequestrum.

Standing surgical removal of a sequestrum requires definitive radiograph diagnosis, a patient amenable to standing surgery and knowledge of the underlying anatomy. It is helpful to use pre-surgery radiographs to measure the size of the sequestrum before surgical exploration to ensure the entire sequestrated bone has been removed. Post-operative radiographs are also helpful to ensure removal. With proper debridement, intensive local antibiotic therapy and long-term systemic antimicrobials are usually not necessary.
As with any standing surgical procedure, adequate sedation and local anesthesia is crucial for success. In the distal limb a tourniquet is helpful to limit hemorrhage and aid in visualization. However, there is often a lack of direct visualization of the sequestered bone and surgery is more based on feel with appropriate instrumentation.

Conclusion
A combination of sedation and local anesthetic will allow the performance of many surgical procedures in the standing horse. The individual animal temperament and environmental conditions as well as safety of the patient, veterinarian and assistants must be evaluated when deciding if standing surgery is appropriate. Regardless of the surgical procedure, appropriate antimicrobial and anti-inflammatory medications should be used and tetanus toxoid should always be current. Most importantly, aseptic technique is as important in the standing patient as it is under general anesthesia. Keeping those facts in mind, many procedures can be successfully performed standing negating the risks and expense of general anesthesia.
The equine upper airway is a high, resistance, low-capacity path between the nares and lungs. Proper function of the equine upper airway requires the coordinated effort of a large number of anatomical structures. Dysfunction of these structures results in profound changes in airway dynamics, decreases in air moving into the lungs and ultimately decreased tissue perfusion. Very small changes in diameter of the upper airway are magnified in their effect on the ability of the horse to move air into the lower respiratory system and oxygenate tissue effectively. For example, a 20% decrease in airway radius doubles airway resistance. For this reason, upper airway abnormalities must be addressed appropriately to maximize the equine athlete’s performance.

**Dorsal displacement of the soft palate (DDSP)**

DDSP most commonly occurs in racehorses, but can occur in other types of performance horses, particularly those required to over flex at the poll. It is an expiratory obstruction and the presenting complaint is often “choking down” or making a “gurgling” noise. These horses are often observed to be open-mouthed breathing during episodes and once the palate displaces they are unable to breathe sufficiently, which leads to rapid slowing or stopping. At this time, they usually swallow and replace the palate into normal position, causing the gurgling noise to dissipate and the open-mouth breathing to stop. Substantial exercise intolerance occurs during DDSP due to disruption in airflow. While gurgling is relatively common, DDSP cannot be ruled out in a horse that is exercise intolerant, but does not make a noise. Approximately 30% of horses affected with DDSP reportedly do not make a noise. Parente, Martin et al. 2002

This causes of this condition are complex not completely understood resulting in several treatment options both conservative and surgical.

Conservative management includes rest, tack changes such as a figure-eight noseband or tongue-tie and anti-inflammatory medication. Failure to respond to these treatments may necessitate surgical intervention in the form of thermal palatoplasty, sternohyoideus and/or sternothyroideus myectomy, or laryngeal tie-forward.

The laryngeal tie-forward replaces the thyrohyoideus muscle which is thought to be dysfunctional in many cases of DDSP. The surgery resulted in significant improvement for 80-85% of cases on which it was performed and recovery time was minimal. Woodie, Ducharme et al. 2005

**Laryngeal hemiplegia**

The most appropriate treatment for laryngeal hemiplegia depends on the severity of disease, presenting complaint and the intended use of the horse. The non-racehorse presented for upper respiratory noise without a history of exercise intolerance is a candidate for ventriculocordectomy or ventriculectomy. This results in resolution of clinical signs in most cases but is unlikely to have a significant effect on exercise intolerance. Prosthetic laryngoplasty or “tie-back” is necessary to improve cases of exercise intolerance or poor performance particularly in racehorses. In situations of maximal exertion the increased airway diameter gained from the “tie-back” is required. The prosthetic laryngoplasty technique has been modified recently resulting in a more effective stable situation which may prove to be more effective with less relaxation of the suture and affected arytenoid.

**Axial deviation of the aryepiglottic fold (ADAF)**

Axial deviation of the aryepiglottic fold is a dynamic condition which can only be evaluated and diagnosed using dynamic endoscopy. The aryepiglottic fold is a membraneous piece of tissue that extends between the cornicate process of the arytenoids and the lateral edge of the epiglottis. On resting endoscopy the upper airway will appear completely normal but the condition can result in a greater than 80% reduction in inspiratory airway. It tends to occur at near maximal exertion when the airway pressures are most negative. ADAF can be seen in combination with other upper airway abnormalities or alone. It can be unilateral or bilateral and most often the right aryepiglottic fold exhibits the most deviation. Most commonly surgical treatment involves resection of a portion of one or both aryepiglottic folds using a laser as a transendoscopic procedure. Recovery time is minimal and success rates are high.

**References**


Zoological small mammal species are commonly encountered in veterinary practice and include those kept as pets, maintained by laboratories and zoological collections, and those encountered as free-living species. Effective protocols for analgesia and anesthesia are critical to the provision of a high level of medical and surgical care for these species. Pain can have serious negative implications for healing and well-being in small mammals, especially those susceptible to stress-associated morbidity. Pharmacologic protocols customized to small mammal species in order to control noxious stimuli at multiple points within the pain pathway are useful tools when providing analgesia. Additionally, sedation and anesthesia can provide a safe and effective means to facilitate diagnostic testing and to perform treatments that would otherwise be difficult or unsafe to perform.

When developing practice protocols for anesthesia of exotic mammals, factors pertaining to equipment availability and skill level of the animal care team must be taken into consideration. The small size of most exotic mammals often correlates with increased metabolic rates compared to dog and cat patients which can predispose them to hypothermia and hypoglycemia during anesthesia. Additionally, responses to medications and anesthesia are less predictable in small mammals. Maintaining a patent airway via endotracheal intubation, achieving vascular access for fluid and drug administration, and close patient monitoring is optimal but often difficult to achieve in these patients. Development and implementation of established, well-practiced protocols will minimize time and risk to the patient associated with anesthesia.

**Pre-anesthesia: Patient evaluation and preparation**

Prior to anesthetizing a patient, a complete medical history, including assessment of husbandry, should be collected. Obtaining a detailed description of the patient’s husbandry, including information pertaining to diet and enclosure, may provide valuable clues as to the cause of the pet’s presenting complaint which should be considered prior to anesthesia. Additionally, questions should be posed to the owner as to the signs the animal has been exhibiting, the duration and progression, as well as any therapies or care that have been implemented at home or by another veterinarian. Whenever possible, a complete physical examination should be performed. It is important to visually assess the patient prior to a hands-on physical exam. Many small mammals are stressed when in unusual situations and are intolerant of handling. As patients in critical condition may decompensate from a simple physical examination, decisions must be made as to how much the animal can tolerate at any given time during treatment. It may be necessary to perform diagnostic testing and treatment administration in a stepwise fashion, allowing the patient recuperation time between handling periods. A balance must be achieved which minimizes the stress of the patient while allowing a thorough, efficient physical assessment.

Sedation or general anesthesia may be warranted but is not without challenge or risk. At a minimum, an accurate weight on a gram scale and assessment of biological parameters including heart rate and respiratory rate should be obtained. Measurement of body temperature should also be recorded if feasible.

Ideally, pre-anesthetic assessment would include a complete blood count and biochemical profile. Minimally, the packed cell volume, total protein, and blood glucose should be measured. A volume corresponding to 1% of the total body weight (kg) can be collected safely from small mammal patients and commercially available analyzers have the ability to process small samples. However, consideration of the expected blood loss during upcoming surgical procedures may dictate that pre-anesthetic laboratory assessment be performed in advance of planned surgery to allow for replenishment of total blood volume and components. Additionally, sample collection in non-anesthetized patients is difficult or unsafe in certain species, such as small rodents and hedgehogs, making pre-anesthetic hematology impractical.

Patient fasting prior to anesthesia is neither practical nor necessary when working with exotic mammals. Rabbits and rodents lack the ability to vomit, although regurgitation can occur. Therefore, the goal of limiting food consumption prior to anesthesia is to keep the mouth clear of food which might be aspirated during intubation or during anesthetic maintenance. Small exotic carnivores should be fasted, but the fast is limited to 2-4 hours prior to anesthesia which allows the stomach to empty while avoiding complications associated with hypoglycemia. The pre and post anesthetic environment should be quiet, warm, with minimal visual and auditory stimulation to prevent stress induced catecholamine release. If blood loss is expected during the procedure, administration of fluids, usually by subcutaneous route, is recommended 2-3 hours prior to anesthesia (10 ml/Kg). If the procedure is likely to cause pain or if the patient’s condition warrants analgesia, preemptive pain management is implemented ½-1 hours prior to anesthesia. Pain should be managed post operatively at the same dosages. Frequency of dosing depends on the level of pain and sedation of the patient but is usually every 4-8 hours.

Establishing vascular access for parenteral fluid and drug administration is necessary based on the patient’s condition and length of anesthesia. Ideally placed prior to anesthetic induction, placement of catheters in non-sedated, fractious animals may not be feasible. In those cases, pre-anesthetic sedation may be used or the catheter should be placed after induction. Intraosseous catheter placement is
Anesthetic induction and maintenance

Sedation protocols may provide enough of a calming effect on exotic mammal patients to allow necessary exams, testing, and procedures. Therapeutic combinations using an opioid with a short-acting benzodiazepine, such as midazolam, are well tolerated. Clinical effects are generally apparent within 15 minutes after administration and last 1-2 hours. Sedation protocols may be used in lieu of anesthesia or may be used as a premedication to decrease the amount of inhalation agent necessary for anesthetic maintenance.

Due to the small size of many of these patients and the need for rapid adjustment of anesthetic depth or recovery, inhalation agents are preferred for use in exotic mammal patients. Isoflurane gas is well tolerated, cost effective, and readily available. Sevoflurane is also commonly used and offers more rapid induction. Mask induction is preferred in small mammals as it allows for close control of the patient. Rabbits will breath-hold during induction if exposed to high concentrations of isoflurane. To minimize this effect, induction is best done with incremental increases in isoflurane concentration. Many exotic mammals display an excitation phase when being induced. Secure restraint of the patient is required to prevent injury during this period. Conversely, anesthetic chambers may be used for induction of stressed or fractious animals but should only be used in well ventilated areas. Sterile ophthalmic lubricant should be applied to the eyes of the anesthetized patient to prevent corneal drying or abrasion.

Mask administration of maintenance anesthesia is acceptable in exotic mammals if the total anesthetic time is brief or if intubation of the patient is impractical. However, if an extended duration of anesthesia is necessary or if there is any concern regarding the patient’s ability to self-ventilate, intubation should be performed. Intubation of ferrets is performed in the same manner as in cats, using a 2.0-3.0 ID endotracheal tube. Because of the long, narrow oral cavity in rabbits, visualization of the larynx is difficult. Intubation may be performed blindly or with the aid of an endoscope using a non-cuffed, transparent, 2.0-3.0 ID endotracheal tube. The endotracheal tube is secured in place routinely but care should be taken to avoid impinging on the tube diameter. Alternatively, a tape butterfly secured to the tube can be sutured to the patient’s lip to prevent the tube from being displaced during patient manipulation. Intubation of rodents is often not feasible due to their small size. Small rubber tubing or large IV catheters can be converted into micro-endotracheal tubes for these patients. Laryngeal masks, developed to be inserted into the oropharynx of small mammals and fit over the larynx, have been used with some success in patients where intubation was not possible. Rabbits and rodents are obligate nasal breathers. Administration of inhalation agents via a mask covering the nose allows for access to the oral cavity during dental and other oral procedures. If a mask is used and assisted ventilation is necessary, the mask should fit tightly around the nose and mouth as manual ventilation is performed. In these instances, gas is likely to enter the stomach and will need to be expelled.

Injectable anesthetic agents

Injectable anesthetic agents are often used in combinations to achieve muscle relaxation, loss of consciousness, as well as anesthesia in small mammal patients. Drugs such as ketamine, telazol, dexmedetomidine, xylazine, and propofol are easy to administer, cost effective and require little equipment to use. However, disadvantages to injectable protocols include the inability to modify anesthetic depth once the drugs are administered and relatively long anesthetic recovery times if reversal agents are not administered. Additionally, many anesthetic pharmaceuticals are controlled, requiring specific storage and licensing. Dosing is available for many common anesthetic agents used in exotic mammal practice and practitioners should consult an appropriate formulary when developing individual patient protocols.

Anesthetic monitoring

Cardiopulmonary arrest associated with anesthesia is often treatable in veterinary patients if detected rapidly. Trained personnel dedicated to patient monitoring is critical to safe anesthesia. The same basic principles of monitoring anesthesia in domestic animals apply to monitoring of exotic mammals. Reflexes are often good indicators of anesthetic depth including the pedal withdrawal reflex.
and auricular reflex (rabbits). The loss of the palpebral reflex indicates medium anesthetic levels. Eye position is also an indication of anesthetic depth. As the animal is induced, the pupil is centrally located. As anesthetic depth reaches a light to medium level, the pupils move ventrally. Once deep surgical anesthetic depth is achieved, the pupil returns to a central location. This may indicate a depth that is deeper than needed for the actual procedure.

Monitoring of the patient is begun at the onset of induction or as soon as practical. Equipment used for monitoring small mammal patients includes an ECG, thermometer, pulse oximetry, Doppler monitor, pediatric stethoscope, +/- esophageal stethoscope. Charting anesthetic parameters helps the anesthesiologist to identify trends in the patient which could foretell a problem. Emergency resuscitation medications should be available as well as any reversal agents if injectable anesthetic agents are being used. Doses are either posted or calculated for the patient prior to anesthesia. Emergency medications used for resuscitative measures in small mammals are the same as those used for other species. However, glycopyrrolate is used in rabbits suffering from bradycardia as they are less responsive to atropine.

**Anesthetic recovery and post-operative care**
Following anesthesia, patients should be monitored closely and securely restrained until they can maintain a sternal posture and are responsive to mild stimuli. The oral cavity should be cleared of debris or mucus that may have accumulated during the procedure. Once fully recovered, food should be offered. Patients that do not self-feed within a few hours should receive assisted feedings. Small herbivores will also benefit from gastrointestinal prokinetic agents if fecal production does not resume. Analgesia, antibiotics, and other therapeutics should be provided as necessary including continued fluid therapy and supplemental heat.

**The pain pathway**
As an unpleasant sensory, and emotional, experience associated with actual or potential tissue damage, pain is associated with surgery and trauma, but is also associated with many disorders and diseases of small mammal patients. Physiologically, individuals feel pain to allow for behavioral responses to avoid painful stimuli and to identify physical ailments. However pain also has negative effects on homeostasis and healing, including immune system modulation and decreased food consumption. Ongoing pain results in guarding or altered use of the injured area leading to disuse atrophy. Pain may also lead to hypoventilation, causing hypoxia and respiratory acidosis.

**Recognition of pain in small mammal patients**
Assessment of pain is affected by species differences, individual tolerances, and the ability of animals to mask signs of pain. Many times the only indication of pain in small mammals is a behavioral change. Behaviors associated commonly with pain in ferrets and rabbits include depression, immobility, hiding, and decrease in grooming activities. Anorexia and disinterest in the surrounding environment may also be noted. Small herbivores will sometimes exhibit bruxism in response to pain. Recognition of pain is often underestimated due to the reliance upon observed behaviors to detect pain and the subjective nature of these observations.

Palpation is a useful tool for pain detection. The fleshy part of the 2nd and 3rd fingers are best suited for palpation due to the more sensitive nature of these digits and digital pressure required to blanch the ends of the fingernails should be applied. A systematic approach to palpation will allow for detection of areas that generate a reaction which should then be explored through additional palpation, range of motion manipulations, or other diagnostic modality. It is important to learn the normal behaviors of the species being evaluated so that deviations from this behavior can be recognized. Ideally, a pain scoring system should be developed for consistent patient evaluation and staff use. As patient status changes, so can pain perception making re-evaluation necessary. In general, if an animal’s lesion would be painful to a human with the same lesion, if the lesion is damaging to the tissues, or if the animal is displaying behavior that may be indicative of pain, then the presence of pain should be assumed and analgesia provided to the patient.

**Approach to analgesia**
Analgesic modalities target the pain pathway at one or more levels. A multimodal approach to analgesia provides optimal pain management by impacting multiple steps along the pain pathway including the transduction, transmission, modulation, and perception of a noxious stimulus. Multimodal analgesia allows individual analgesic modalities to become synergistic, or additive, when used together which prevents unwanted side effects associated with larger drug doses necessary for one drug to achieve the same effect. Thermal therapy (ice, heat) and massage therapy target the pathway at the level of transduction of the noxious stimulus, preventing the conversion and perpetuation of the electrical impulse. Local anesthesia impedes transmission of the impulse from the periphery to the spinal cord. Nonsteroidal anti-inflammatory drugs modify both transduction and transmission by inhibiting steps in the arachidonic acid inflammatory pathway. Opioid analgesics have effects at all levels of the pain cascade. Administration of analgesia before pain is perceived by the patient helps to stabilize the maintenance phase of anesthesia, reduces the total amount of analgesic medication needed to control pain, prevents wind-up pain, and decreases patient morbidity associated with surgery or anesthesia.
Analgesic drugs used in small mammals

Opioids – Three types of opioid receptors are expressed in veterinary patients; μ (mu), κ (kappa), and δ (delta). Understanding the distribution of these receptors helps guide analgesic protocols however continual patient assessment and therapeutic adjustment is warranted when working with individual cases. When administered appropriately, opioid analgesics are safe and effective in managing pain. Fentanyl, hydromorphone, morphine, buprenorphine, and butorphanol are the most commonly used opioids in veterinary medicine. Tramadol, a synthetic opioid, is also gaining popularity as an effective analgesic. Opioid analogesics tend to have a rapid onset and are reversible, making them ideal for controlling pain in critical patients. Butorphanol, an agonist-antagonist, is a short acting, fast-onset opioid that provides analgesia for visceral pain but not somatic pain. Due to its short action, re-dosing is frequent (2-4 hours); otherwise a constant rate infusion is necessary for prolonged analgesic effect. Buprenorphine is a mixed agonist-antagonist that is slow in onset but longer acting, generally re-dosed every 4-8 hours. Transmucosal dosing has been reported in small mammals but the efficacy of buprenorphine administered by this route is problematic and the dose administered is significantly higher than parenteral routes. Tramadol provides an oral option for opioid administration and may be compounded into suspension but is bitter tasting, requiring use of a strong flavoring for oral dosing to small mammals. While all opioids may have sedative effects in small mammal patients, buprenorphine is associated with decreased gastrointestinal motility. Despite historical concerns however, opioid analogesics do not cause respiratory depression in small mammal patients if dosed appropriately.

NSAIDS – Nonsteroidal anti-inflammatory drugs inhibit cyclo-oxygenase (COX) enzyme in the arachidonic acid pathway, thereby inhibiting production of inflammatory mediators such as prostaglandin. COX-1 has historically been associated with homeostatic functions in the body such as protecting the gastrointestinal tract and kidneys, maintaining platelet function and macrophage differentiation. COX-2 is involved in inducing prostaglandins that mediate inflammation, pain, and pyrexia. This clear functional distinction between these enzymes is unlikely but the negative side effects associated with NSAID administration that include gastrointestinal ulceration, renal damage, and bleeding, are associated with COX-1 inhibition. Therefore, many NSAIDS used today are COX-2 specific (COX-1 sparing). NSAIDs should not be used in small mammals with known renal disease or those with gastrointestinal bleeding. Meloxicam is the most commonly used NSAID in small mammals and it has primarily COX-2 inhibition. Oral and injectable formulations are available. Dosing is variable but published doses are available for all small mammals. Ferrets possess limited ability for glucuronide conjugation which prolongs the duration of action of NSAIDs, therefore caution must be used when using these products in ferrets. Carprofen is available as an oral option for opioid administration and may be compounded into suspension but is weak inhibitor of both COX-1 and COX-2 pathways. However, it has good anti-inflammatory activity and is safe for use in ferrets, rabbits, and rodents. Ibuprofen is well tolerated by rabbits and rodents, but should not be administered to ferrets. Gabapentin is used primarily for neuropathic pain and is synergistic with other NSAIDs when used concurrently.

Local Anesthetics – Local anesthetics, such as lidocaine and bupivacaine, provide good regional analgesia as well as local anesthesia. These can be used as an infiltrate at an incision site, in ring blocks, as a tissue infusion or topical wash, and in epidural application. The total dose used should not exceed published doses for systemic administration of either drug (1 mg/kg). The addition of an opioid to the infusion may also extend the duration of the local anesthesia provided. Preservative-free morphine (0.1 mg/kg) combined with bupivacaine 0.123% (0.1 mg/kg) is used for epidural anesthesia which is performed routinely.

Nerve Blocks – Dental disease is common in rabbits and rodents, often necessitating oral surgery and dental extractions. Dental blocks administered using lidocaine and bupivacaine are an important component of multimodal analgesia perioperatively. Controlling pain in these patients will allow them to recover faster and return to self-feeding. Regional anesthesia is achieved through 5 nerve blocks in the rabbit patient; infraorbital, mental, mandibular, maxillary, and palatine. Techniques for applying these blocks are described in Lichtenberger and Ko.

Ancillary therapy

Thermal therapy, using both heat and cold, provides targeted therapy to painful tissue. Cryotherapy is often used during the first 24-48 hours post tissue injury to provide analgesia and control inflammation. Ice packs applied to a surgical incision or inflamed tissue for 10-15 minutes 3-4 times daily controls edema and postoperative swelling. Applying heat to damaged tissue will promote tissue healing and increase circulation once the acute inflammatory phase has resolved. Thermal therapy is especially useful when preparing a patient for physical therapy. Both heat (prior to) and cold (following) can be used in conjunction with physical therapy to improve function and build strength in a limb following an injury. Passive range of motion exercises helps preserve normal joint function while the patient is recovering from an injury but care must be taken to prevent exacerbation of the injury or discomfort to the patient. Message therapy provides pain relief, reduces stress, and can reduce edema. It should be performed in a controlled, gentle manner. In small herbivores, gentle massage of the abdominal wall increases vagal activity which improves gastric motility. Acupuncture has modulating activity on the peripheral, central, and autonomic nervous system with studies in animals showing that those patient undergoing treatment experienced analgesia and more rapid return to function. Therapeutic laser therapy has been shown to have a positive impact on wound and bone healing by altering oxygen utilization in mitochondria, blood flow through angiogenesis and
vasodilation, and axonal sprouting both centrally and peripherally. Associated with few risks, this modality controls pain and inflammation while encouraging normal neurological function.

References
Providing veterinary care to exotic pets is both rewarding and challenging, offering the veterinary care team the chance to work with a wide variety of species while also requiring species-specific knowledge in order to provide quality care. Veterinary hospitals engaged in providing routine and critical care for exotic pets must adopt special accommodations and a commitment to educating all members of the veterinary care team about these unique pets. Physical assessment and diagnostic evaluation of exotic pets is often time intensive due to the need to obtain an in-depth history and the sometimes fragile state of these patients. Critical care is an integral part of disease management of exotic pets. The nature of these species and the fact that they are fairly recently domesticated dictates that they hide their illness until they have decompensated. Owners need to be aware that their pet is in serious condition but by providing critical care, the veterinary care team is able to provide them with the best chance for their pet’s recovery.

Preparedness
Many exotic pets requiring hospitalization and nursing care are presented in critical condition. Immediate and efficient implementation of triage and care protocols requires a dedicated, well-stocked, and organized clinical space with established roles and protocols for care team members. These preparations should be established well before the first critical patient is presented. Work with your care staff to create a plan to respond to medical emergencies. Most of the supplies and equipment used for emergency care of traditional pets can be used to care for exotic pets, but some additional supplies will be needed. Prepare a space in your facility that is ready to care for exotics. Include small gauge needles and small syringes, small endotracheal tubes, and drug doses for exotic species in your crash cart. Organize and hold training sessions to practice emergency techniques on exotic species, using cadavers or models to hone your skills and those of your support staff. Every good team has members that know their role in an emergency and are prepared to perform it. Make sure your team members are ready.

Critical care of the exotic small mammal herbivore
Rabbits and rodents, such as guinea pigs, chinchillas, degus, prairie dogs, rats, mice, hamsters, and gerbils, are often presented to small animal or exotic animal practices for routine care, basic and advanced surgical procedures, as well as for critical care. An important component of veterinary care is providing the owner with information regarding appropriate husbandry, dietary needs, preventative health care, and discussing clinical signs that, when observed in their pet, constitute the need for emergency and critical care. Small exotic herbivores, such as rabbits, guinea pigs, and chinchillas, are exquisitely sensitive to environmental and physiological stressors. Because of the resultant catecholamine induced physiology, gastrointestinal motility disorders often compound the animal’s clinical condition, regardless of the underlying pathology or etiology. In these situations, implementation of critical care early after the onset of clinical signs can improve the patient’s chances of stabilization and effective disease management.

Husbandry and history
Obtaining a detailed description of the patient’s husbandry, including information pertaining to diet, enclosure, and environment, may seem minimally important in the face of an emergency, but valuable clues as to the cause of the pet’s presenting complaint may be gleaned from the information provided. Additionally, questions should be posed to the owners as to the signs the animal has been exhibiting, their duration and progression, as well as any therapies or care that have been implemented at home or by another veterinarian. A detailed history can be collected by hospital staff while a triage exam is performed so as not to delay the provision of care to a critical patient. Developing a pre-printed history form will facilitate this process by identifying problems in husbandry to help focus your exam and make specific care recommendations. Additionally, if concerns regarding the patient’s husbandry are addressed prior to the onset of problems, you have provided your client with excellent preventative health care for their pet.

Triage exam
Patient assessment is part of a good triage examination whether the patient has just arrived for an emergency or it is being evaluated after a night of hospitalization and care. It is important to visually assess the patient prior to a hands-on physical exam. Many exotic herbivores are stressed when in unusual situations and are intolerant of handling and the stress it induces. As patients in critical condition may decompensate from a simple physical examination, decisions must be made as to how much the animal can tolerate at any given time during treatment. It may be necessary to perform diagnostic testing and treatment administration in a stepwise fashion, allowing the patient recuperation time between handling periods. Some animals benefit from sedation or anesthesia to minimize perceived stress.

Physical examination should be performed with the animal resting on a firm, stable surface. Wrapping the patient in a towel can provide comfortable restraint. Small rodents such as mice and hamsters may need to be scruffed to achieve secure restraint but this
will also increase the patient’s stress level. Conversely, the handler may wear a pair of thin leather or thick gardening gloves to handle particularly fractious rodents. While rodent teeth can still penetrate through these gloves, the animal is more likely to bite the glove and not the examiners hand. A balance must be achieved which minimizes the stress of the patient while allowing a thorough, efficient physical assessment. Sedation or general anesthesia may be warranted. Unfortunately, anesthesia of these small patients is not without challenge or risk. In all but the larger patients, intubation is not feasible and it is more difficult to achieve vascular access, prevent hypothermia, and generally monitor the patient while under anesthesia. Inhalant anesthetic agents, such as isoflurane or sevoflurane, offer the advantage of allowing rapid adjustments to anesthetic depth and patient recovery but are also associated with potential cardiopulmonary depression. Combining inhalant anesthesia with sedatives helps to ‘balance’ the anesthesia to provide desired affect (unconsciousness, decreased response to pain, muscle relaxation) while minimizing potential adverse effects. Midazolam, a short-acting benzodiazepine, is useful in rabbits and rodents as a pre-anesthetic agent or as a sedative for non-painful procedures or handling. When combined with an opioid analgesic such as buprenorphine or butorphanol, many small herbivores will become calm and comfortable enough to allow for diagnostic sample collection, positioning for imaging, and catheter placement. Alpha-2 agonists such as dexmedetomidine may also be used to provide good muscle relaxation but are associated with respiratory depression and bradycardia.

Basic biological parameters including heart rate, respiratory rate, body temperature, mucous membrane color, capillary refill time, and blood pressure should be evaluated, if feasible, to assess the patient’s overall condition and perfusion status. Table 1 summarizes biological parameters for common small herbivore pet species. Obtaining an accurate weight, best performed with the use of a digital gram scale, is essential for complete patient assessment and for implementing medical therapy.

### Table 1: Biological parameters for exotic pet herbivore species.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Life Expectancy</th>
<th>Heart Rate (bpm)</th>
<th>Respiratory Rate (breaths/min)</th>
<th>Temperature (°F / °C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rabbits</td>
<td>6-13 years</td>
<td>130-325</td>
<td>30-60</td>
<td>100-103°C (37.8-39.4°C)</td>
</tr>
<tr>
<td>Guinea pigs</td>
<td>5-7 years</td>
<td>240-310</td>
<td>80-120</td>
<td>99-103°C (37.2-39.5°)</td>
</tr>
<tr>
<td>Chinchillias</td>
<td>10-20 years</td>
<td>100-150</td>
<td>80-120</td>
<td>96.6-100.4° (37-38°)</td>
</tr>
<tr>
<td>Prairie dog</td>
<td>6-10 years</td>
<td>83-318</td>
<td>40-60</td>
<td>95.7-102.3° (35.4-39.1°)</td>
</tr>
<tr>
<td>Hamsters</td>
<td>1.5-2 years</td>
<td>250-500</td>
<td>35-135</td>
<td>-98.6-100.4 (37-38)</td>
</tr>
<tr>
<td>Gerbils</td>
<td>3-4 years</td>
<td>360</td>
<td>90</td>
<td>98.6-101.3 (37.0-38.5°)</td>
</tr>
<tr>
<td>Mice and Rats</td>
<td>1.5-3.5 years</td>
<td>250-780</td>
<td>35-135</td>
<td>96.6-103.1° (35.9-39.5°)</td>
</tr>
</tbody>
</table>

Skin tenting and moistness of mucous membranes give indications of the hydration status of the animal. Small herbivores with increased skin tenting, dry oral mucus membranes, but normal pulses may be considered to be 4-6% dehydrated. Those patients with more pronounced skin tenting, very dry mucous membranes and dry, sunken eyes are closer to 10% dehydrated. Hypovolemic shock accompanies dehydration greater than 10%. Small herbivores are less likely to demonstrate clinical signs associated with compensatory shock, making detection of even mild dehydration states critically important. In rabbits, once bradycardia (<200 bpm, ref. range 200-240 bpm), hypothermia (<98.0°F, ref. range 100-102°F), and hypotension (<90 mm Hg, ref. range 90-120 mm Hg systolic) is detected, the patient is in early decompensatory hypovolemic shock and in need of aggressive fluid resuscitation.5

Mentation and respiratory character can be assessed prior to handling, in addition to stance and gait in the ambulatory patient. As obligate nasal breathers, rabbits and rodents with respiratory disease should be observed closely for nasal and thoracic movement. Signs of dyspnea generally correspond with a poorer prognosis. Physical assessment of small herbivores is performed in a routine manner but must include examination of the oral cavity and palpation of the gastrointestinal structures. A transilluminator or other light source and nasal speculum will facilitate a thorough oral exam in rabbits, chinchillas, prairie dogs, and guinea pigs. When a nasal speculum is not available, an otoscope may be used. In small rodents, oral exam is limited to the incisor teeth unless the patient is anesthetized. Digital palpation or the maxilla and mandible will allow for detection of apical elongation of the cheek teeth which is a common finding in patients with dental disease. Abdominal palpation should include detection of the location and degree of distension of the stomach, detection of bloating in the intestines, and presence of fecal material in the colon. Many exotic herbivores suffering from GI disease will demonstrate a pain response during abdominal palpation. While any gastrointestinal disease can lead to life-threatening complications in small herbivores with hind-gut fermentation, the presence of diarrhea or fluid accumulation in the gastrointestinal tract are poor prognostic indicators.

### Stabilization and resuscitation

Critically ill small herbivore patients often present in early or late stages of decompensatory shock and require aggressive therapy to stabilize and reverse their declining condition. Animals exhibiting bradycardia will have low cardiac output resulting in hypothermia which in turn exacerbates the bradycardia and perpetuates the worsening of clinical signs. Fluid resuscitation as well as providing thermal and respiratory support are the hallmarks of management of shock.
Complete fluid therapy protocols provide for administration of fluids for resuscitation to correct perfusion deficits and restore oxygenation of tissues, rehydration to correct interstitial deficits, and subsequently maintenance to support body functions and replace ongoing losses until the patient is eating and drinking normally. Crystalloids fluids are preferred for rehydration and maintenance. Combination therapy using a crystalloid and a colloid is preferred for treatment of shock in small herbivores. Physiological data of the patient should be frequently monitored throughout therapy including assessment of mentation, capillary filling and mucous membrane color, pulse quality, heart rate, respiratory rate, body temperature, and production of urine. A small mammal suffering from hypovolemic shock requires placement of an intravenous or intraosseous catheter. Resuscitation should be initiated with administration of an isotonic crystalloid (10-15 mL/kg) bolus infusion followed with a colloid (5mL/kg) infused over 5-10 minutes. Thermal therapy should be implemented concurrently. The resuscitation fluid boluses are repeated every 15 minutes until systolic blood pressure is >90 mm Hg. Patients that demonstrate persistent hypotension in spite of therapy may be administered hypertonic saline (5 mL/kg) by bolus infusion over 10 minutes. Due to its hyperosmotic property, hypertonic saline pulls fluid from the interstitial and intracellular spaces to increase intravascular volume. Because of its effect on the extravascular tissue, however, it should be administered concurrently with crystalloids to prevent worsening of dehydration. Once the patient has stabilized, with normal physiologic parameters, rehydration therapy can begin. Rehydration fluids take into account the degree of patient dehydration and body weight. The volume to be administered equals the hydration deficit (%) multiplied by the patient weight (kg) multiplied by 1000 mL. A rabbit weighing 1.2 kg that is 8% dehydrated will require 96 ml (0.08 x 1.2 x 1000=96) to correct its deficit, 80% (76.8 ml) of which can be administered in the first 24 hours after resuscitation. Hetastarch can also be continued as a constant rate infusion (20 ml/kg/day) for those animals that require intravascular osmotic pressure. In general, maintenance fluid therapy for small herbivores is 3-4 mL/kg/hr or 75-100 mL/kg/day.5

Severe hypothermia in small herbivores indicates that the animal is in decompensatory shock and is a poor prognostic indicator. Aggressive, rapid warming of the patient should be performed over the initial 1-2 hours after presentation. Administration of warmed fluids will help heat the body core while external heat sources provide environmental warmth. Caution should be exercised when using a focal heat source such as a lamp or heating pad as a non-ambulatory animal will not remove itself from the heat source and may suffer burns. Assessment of body temperature should be performed frequently to ensure that the animal does not become hyperthermic.

As obligate nasal breathers, rabbits and rodents will become respiratory compromised rapidly when suffering from both upper and lower airway diseases. Oxygen supplementation is indicated for patients experiencing respiratory distress. The small size of exotic herbivores is well suited for oxygen chambers however oxygen cages are expensive and often unavailable. Oxygen concentrators absorb nitrogen from room air to increase the concentration of oxygen emitted, often >95% fractional inspired oxygen (FiO2), and can be used to achieve therapeutic levels of oxygen (>40% FiO2) in small enclosures used to hospitalize exotic patients.

Nebulization as an adjunct therapy for management of respiratory is not only beneficial in delivering medications directly into the airways, it helps maintain the hydration status of respiratory membranes to allow clearance of secretions. Nebulizing hypertonic saline decreases edema associated with the pulmonary tissue resulting in an opening of the airways. Albuterol inhalation solution developed for children with asthma has also been effective for management of respiratory disease in rabbits and rodents. Its bronchodilator action opens airways and alleviates bronchospasm. Dosing is empirical and extrapolated from human use (0.042% Albuterol Sulfate Inhalation Solution or 0.083% solution diluted with 3 ml saline, 15 min. q8-12h for 14 days). Antimicrobial therapy may be incorporated into nebulization therapy.

Nutritional support must be provided to hospitalized patients, especially those with high metabolic rates where anorexia rapidly results in cachexia. The caloric requirements of the patients must be calculated and those requirements met on a daily basis through self or supplemental feeding. As in domestic species, metabolic rates are measured in kilocalories per day (kcal/day). Once the number of kilocalories needed is determined, the amount of food needed to meet that requirement can be calculated and divided over the day’s feedings. The best way to determine if a patient is receiving appropriate nutrition is to weigh the patient daily on an accurate gram scale. Supplemental feeding can then be adjusted as appropriate. Oral administration of a gruel diet will also help hydrate the patient. Several commercial diets are available. Patients should be fed approximately 50 ml/kg/day divided into feedings every 4-6 hours.

References
Adrenal disease
Adrenal-associated endocrinopathy in ferrets, known as adrenal disease, affects ferrets from 1-8 years-of-age, although the average age of onset in ferrets is 3-4 years-of-age. First described in the 1980’s, the etiology of adrenal disease remains elusive. Suggested etiologies include early sterilization of ferrets, consistent and prolonged exposure to protracted photoperiods or lack of variation (seasonality) of photoperiods, and genetics. It has been proposed that in the United States, the prevalence of adrenal disease in ferrets is 20-25%, with age of onset an average of 3.5 years after sterilization surgery.

Clinical signs in affected ferrets are associated with hyperadrogenemia and hyperestrogenemia, with cortisol elevations rarely seen. The most common presenting clinical sign of adrenal disease is progressive bilaterally symmetrical alopecia, usually starting at the tail and dorsal lumbosacral region. Hair loss may initially be seasonal before becoming persistent. Other signs may include vulva swelling in females (55-89%), stranguria or dysuria in males with prostatic tissue swelling 1-7%, pruritis (9-40%), and muscle atrophy (4-56%). Aggression and/or sexual behavior, lethargy, and polyuria/polydipsia have also been associated with adrenal disease in ferrets.

Many clinicians are comfortable diagnosing adrenal disease in ferrets based on history and clinical presentation. Ancillary testing helps to support the diagnosis and histopathology of the adrenal tissue is confirmatory. Studies evaluating the circulating blood levels of sex hormones in ferrets with adrenal disease have demonstrated that concentrations of estradiol, 17-hydroxyprogesterone, androstenedione, and dehydroepiandrosterone may be independently or concurrently elevated. A commercial serum hormone assay which measures estradiol, androstenedione, and 17-hydroxyprogesterone is available (Clinical Endocrinology Laboratory, University of Tennessee College of Veterinary Medicine). Elevations in one, or more, of the three hormones has a reported 96% sensitivity for diagnosing adrenal disease in affected ferrets.

Cortisone levels and adrenal stimulation tests are not helpful in diagnosing adrenal disease in ferrets as this condition is not associated with hypercortisolemia. Anemia and thrombocytopenia may be found on hematological evaluation in chronic cases due to the prolonged presence of circulating estradiol hormones, however serum biochemistry evaluation is typically normal. Cytology of the prepuce in male ferrets may be a useful screening test for adrenal disease as there is a correlation between adrenal disease and the presence of >70% cornified preputial epithelial cells.

Radiographic imaging is rarely diagnostic for adrenal disease but is useful for screening patients for concurrent disease. Ultrasound evaluation is useful for evaluation of the size, shape, and parenchymal architecture of the adrenal glands, as well as evaluating the animal for unilateral versus bilateral disease. Measurement reference ranges have been established for normal adrenal glands in ferrets. Normal adrenal glands are larger in males than in females but may be challenging to locate using ultrasound. In one study of normal ferrets, size is reported as length (7.2 ± 1.8 mm left, 7.6 ± 1.8 mm right) and thickness or width (2.8 ± 0.5 mm left, 2.6 ± 0.4mm right), with glands tending to be ovoid. Abnormal adrenal glands become rounded, with enlargement of one or both poles. Normally hypoechoic to the surrounding retroperitoneal tissue, diseased adrenal glands may display a heterogenous structure and increased echogenicity. It is important to evaluate both adrenal glands to determine the presence of unilateral or bilateral disease as this will affect treatment planning.

Histologic evaluation of adrenal gland tissue from affected ferrets demonstrates a range of lesions, from hyperplasia to adenoma to adenocarcinoma, with some evidence of progression. Intact unbred female ferrets may present with clinical signs consistent with adrenal disease, as will those jills with remnant ovarian tissue. However, in these cases, the age of onset of clinical signs is associated with the onset of estrus, usually at a year-of-age. Likewise, male ferrets may demonstrate seasonal alopecia that is unrelated to adrenal disease, making this a differential diagnosis to consider.

Historically, an adrenalectomy of the affected gland is the preferred treatment for unilateral disease, however medical management may be preferred due to economic reasons or for ferrets with concurrent disease that makes them an unsuitable surgical candidate. The
right adrenal gland is more difficult to remove than the left due to its close proximity to, and possible involvement with, the vena cava. Surgical debulking, cryosurgery, and laser surgery are other options in these difficult cases. When bilateral disease is present, removal of the larger gland and debulking of the smaller gland is recommended. Serious complications and death can occur after bilateral adrenalectomy surgery. Up to 8% of ferrets, and perhaps more, possess accessory adrenal tissue as encapsulated nodules comprised of cortical tissue embedded in the perirenal fat, however temporary administration of glucocorticoids and/or mineralocorticoids may be necessary following adrenalectomy. Hobs with urinary obstruction secondary to prostatic cysts improve rapidly (1-2 days) post-surgery and should remain catheterized while obstruction of the urethra is resolving. Reoccurrence of disease within 3-18 months occurs in 17-38% of ferrets undergoing unilateral adrenalectomy.

Medical management of adrenal disease is palliative. Therapies developed for treating Cushing’s disease in dogs are ineffective and potentially dangerous in ferrets. Melatonin administration has been shown to temporarily ameliorate clinical signs. GnRH analogs have proven effective in temporarily relieving the clinical signs of adrenal disease. GnRH agonists, such as leuprolide and deslorelin, initially stimulate LH release, but subsequently promote negative feedback inhibition to downregulate GnRH receptors in the pituitary gland due to their binding properties. This leads to decreased LH release and decreased sex hormone production by the adrenal tissues. Leuprolide acetate (Lupron – Abbott Laboratories, Northbrook, IL) is a long-acting GnRH agonist depot that alleviates clinical signs beginning about 2 weeks post injection. Dosing of 100-150 µg/kg IM every 4-8 weeks is recommended. Higher doses (250) have been used without adverse effects on ferrets suffering from prostatic disease. Lupron can be divided into aliquots and frozen for at least a year. Deslorelin acetate was developed as a canine contraceptive and comes as a slow-release subcutaneous implant in two sizes (3 mg and 4.7 mg). Suprelorin F (Virbac Animal Health, Inc., Fort Worth, TX) is available in the United States. The implant is inserted subcutaneously on the dorsum over the shoulders and is associated with alleviation of clinical signs within 2 weeks and a duration averaging 17.6 months. Anesthesia or heavy sedation may be necessary when placing the implant due to its large size and the associated discomfort of placement. Adrenal disease in ferrets is not a life threatening disease except in the case of a hob with urinary obstruction. Both medical and surgical treatment options are expensive and owners may elect benign neglect as a treatment for their ferret.

**Insulinoma**

Insulinomas are tumors of insulin-secreting pancreatic islet cells and is considered one of the most common neoplasm affecting ferrets. There is no sex predilection but the average age of onset is 4-5 years. Contributing factors to the development of insulinomas in ferrets include genetic factors as well as diet. Metastasis is uncommon but possible. In a normal ferret, a low blood sugar level stimulates the release of glucagon, cortisol, epinephrine and growth hormone. These in turn stimulate gluconeogenesis and glycogenolysis in the liver to raise the blood sugar. In addition, these hormones inhibit the release of insulin and decrease the utilization of glucose by cells. Once blood sugar rises, pancreatic beta cells secrete insulin to provide negative feedback and stimulate the uptake of glucose by cells. A ferret with an insulinoma secretes excessive amounts of insulin which inhibits gluconeogenesis and glycogenolysis while stimulating the uptake of glucose by tissues, which results in hypoglycemia.

Clinical signs of insulinoma may develop over weeks to months. Conversely, owners may be unaware of any manifestation of disease until a hypoglycemic crisis occurs. Clinical signs of this disease include lethargy, weakness, ptosis, pawing at the mouth, weight loss, peripheral neuropathies, collapse, ataxia, and seizures. Seizures occur in the majority of dogs suffering from insulinomas, however only 14% of ferrets develop seizures. This may be reflective of the ferret’s relative tolerance of hypoglycemia and the slow onset of this disease. Diagnosis is based on a fasting blood glucose level, correlated with clinical signs. Most practitioners agree that a level of 60 mg/dl is suggestive of an insulinoma although other causes of hypoglycemia must be ruled out (hepatic disease, sepsis, paraneoplastic syndrome, hypoadrenocorticism). Hand-held glucometers may be inaccurate, generally providing a value lower than the actual blood glucose level. Additionally, glucometers are inaccurate in patients with high hematocrits making this tool problematic for use in ferrets. Suspected hypoglycemic samples should be confirmed with conventional laboratory methods. Fructosamine, a glycosylated protein, has been used in dogs to measure the degree of glycosylation which would be expected to be low in hypoglycemic patients. However, these proteins have not been measured in ferrets and the results are unreliable. Biochemical testing, with the exception of glucose levels, as well as hematology are usually unremarkable. Additionally, diagnostic imaging has low sensitivity for diagnosing insulinoma. Serum insulin levels can be measured and must be simultaneously compared to a fasting glucose level. A normal or high insulin level in the face of hypoglycemia is diagnostic. Ferrets presenting for emergency complications of hypoglycemia must be treated symptomatically until stable. A slow bolus of 50% dextrose followed by a constant rate infusion of 5% dextrose spiked fluids will stabilize the hypoglycemia. Diazepam (1mg/ferret IV) may be needed to control seizure activity. Once stabilized dietary, medical and surgical therapy is indicated singularly or in combination. The specific therapy implemented is dependent on severity of signs, age of patient, presence of concurrent disease, and owner’s wishes. The recent consensus is that surgical treatment is preferred to extend the patient’s expected life span. An exploratory laparotomy is performed and both lobes of the pancreas are visually and manually examined. While care should be exercised when handling pancreatic tissue, the ferret pancreas tolerates manipulation well. Insulinomas may be solitary or multiple.
and can range from non-detectable to 2 cm in size. Individual nodules can be locally resected. Diffuse disease is best treated with pancreatic lobectomy or partial pancreatectomy. Post-operative fasting for 12 hours is indicated. A blood glucose level, measured post-operatively, will often be high and will stabilize over the next few days. Serial glucose testing each day will determine the need for implementation of medical management. Medical management controls clinical signs by blocking the effects of insulin, not by treating the neoplasia. Prednisone (0.5-2.0mg/kg q 12 hrs.) is started at the lowest dose and raised as needed to maintain normal blood glucose levels and control clinical signs. If prednisone therapy is unable to control the disease, diazoxide (Proglycem, Baker Norton Pharmaceuticals, Inc. Miami, FL) can be added starting at an initial dose of 5mg/kg q 12 hrs and can be increased as needed to a maximum dose of 60mg/kg divided BID. Regardless of medical or surgical therapy, ferrets diagnosed with insulinoma should be fed many small meals throughout the day containing high quality protein and moderate to high levels of fat. Carbohydrates and treat food should be avoided. Due to the microscopic nature of some insulinomas, cure is generally not possible. Ferrets that undergo surgery tend to have longer survival times (1-3 years) than ferrets managed with medical therapy (6-12 months). Fasting (4 hr.) blood glucose levels should be evaluated regularly in these ferrets.

References
Antibiotic usage is wide spread in veterinary medicine and is indicated for a variety of reasons ranging from prophylactic prevention of bacterial colonization during surgery to treatment of systemic bacterial infections. The choice of antibiotic is based on factors pertaining to the patient, the clinic, the medication, and the individual practitioner. The location of infection, ease of handling of the patient, frequency of administration, and route of administration must be considered prior to developing antibiotic use protocols.

Questions to answer prior to implementing antibiotic therapy
Prior to electing to implement therapy with antimicrobial medications, several questions must be answered in order to judiciously select and use these drugs.

“What pathogen is most likely involved?”
Bacteria are classified as gram positive or gram negative based on their appearance after being exposed to the staining process. The peptidoglycan layer of the cell wall is not covered by a cell membrane in gram-positive cells so the methylene blue stain is retained and these bacteria appear blue-purple. Gram-negative cells do have a cell membrane which prevents the stain from being retained. The methylene blue stain is washed off and replaced by the safranin in gram-negative cells resulting in their pink-red appearance. Bacteria are also classified by their use of oxygen; aerobic organisms requiring oxygen to reproduce and anaerobic organisms requiring little to no oxygen to multiply. When possible, a sample collected from the infected tissue should be evaluated by Gram staining to provide the clinician with expedient and inexpensive preliminary clues pertaining to the causative organisms. Gram staining does not replace the need for microbe culture, nor does it provide clues regarding antibiotic susceptibility.

“Does this patient have a treatable bacterial infection?”
While seemingly obvious, this is the most important question to be asked. There are certainly some instances where antibiotic therapy is warranted when the animal is not suffering from a bacterial infection but this should not be the norm. Prophylactic use of antibiotics may be indicated peri-operatively, when surgical contamination is likely and unavoidable. Practitioners may also elect to implement antibiotic therapy when the patient is immunocompromised or severely debilitated and therefore exquisitely susceptible to opportunistic infections. However, antibiotics should only be used when necessary to treat a specific and susceptible condition. In healthy hosts, the body’s natural defenses are often sufficient to ward off infection. It is only when these mechanisms become compromised, or there is a change in pathogenicity of the microbe that infection develops. Signs of bacterial infection include suppurative exudate, inflammation, odor, and dysfunction of the affected tissue.

“Where is the infection and can I effectively treat this animal?”
The answer to these questions will allow the clinician to begin evaluating the treatment options at their disposal. Many antibiotics have a variety of formulations allowing for diverse routes of administration. Others are very limited in how they may be administered and what tissues they penetrate to reach therapeutic doses. Animals with skin infections may be treated systemically or topically, depending on the severity of the lesion. In some cases, patient handling will be the limiting factor. Particularly fractious or stressed patients will be better treated with longer acting therapeutics, while patients that are amenable to handling may be treated more frequently without adverse effects. Owner compliance and potential complications must always be considered when implementing antibiotic therapy.

“What antibiotic should be used?”
Focused antibiotic therapy directed against gram-positive organisms may include the beta-lactam antibiotics in the penicillin and cephalosporin classes. Antibiotics with specific efficacy against gram-negative organisms include the fluoroquinolones, 3rd generation cephalosporins, aminoglycosides, and phenicols. Anaerobic organisms are susceptible to metronidazole, penicillins, cephalosporins, phenicols, and macrolides. When sepsis is suspected, broad-spectrum antimicrobial therapy is indicated. Relatively few

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pharmacokinetic (PK) and pharmacodynamics (PD) studies have been done using these drugs in exotic animals and most dosing information is anecdotal. However, what has been elucidated by the studies that have been performed is that there can be significant species differences in PK and PD parameters when administered the same drug. Reported dosing ranges are often broad and caution must be exercised when extrapolating between species. In general, animals with a larger mass require lower doses and less frequent dosing than those with small mass. Likewise, dosing tends to be higher in animals with high metabolic rates compared to those with lower basal metabolism. Rabbits, rodents, and ferrets are often used as animal models for human drug studies making dosing information for these species more available. However, absorption, distribution, metabolism, and elimination are dependent on the health status of the animal, anatomical and physiological parameters, and variability in drug metabolites, enzymes, and protein binding properties.

**Extra label drug use**

Few drugs are approved for use in exotic animals. The U.S. Food and Drug Administration oversees the use and administration of pharmaceutical agents in animals through the Federal Food, Drug, and Cosmetic Act (CFR 21 USC 360) and the Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994 (CFR 21, Chapter 1, Part 530) which outlines the parameters for extra-label drug use. "Extra-label use" is defined as the "Actual use or intended use of a drug in an animal in a manner that is not in accordance with the approved labeling. This includes, but is not limited to, use in species not listed in the labeling, use for indications (disease and other conditions) not listed in the labeling, use at dosage levels, frequencies, or routes of administration other than those stated in the labeling, and deviation from labeled withdrawal time based on these different uses."

Off-label administration must be administered under oversight of veterinarian, there must be a valid veterinarian-client-patient relationship, the use is limited to cases where health of animal is threatened and suffering/death will result if not treated. Compounding of approved animal and human drugs is legal but is regulated under 21 CFR 530.13.

Wildlife with defined hunting seasons are considered food animals and have additional AMDUCA regulations: 1) Extra-label drugs may be used only when no approved drug is available 2) Requires veterinary assessment of patient 3) Extended withdrawal period for animal products should be established based on known withdrawal times for domestic species 4) Identification protocols must be Institutes to ID treated animals and 5) Measures to ensure that withdrawal time frames are met prior to release must be taken. Extra-label drug use is strictly regulated for poultry and game birds as well. The food animal drug residue database may be found at [http://www.farad.org/eldu/prohibit.asp](http://www.farad.org/eldu/prohibit.asp).

**What’s new**

**Long-acting formulations**

As new antibiotics are developed, efforts continue to increase their efficacy, duration of action, and spectrum. Several long-acting antibiotics have been developed for use in companion animals however their PK/PD parameters are unknown when used in exotic species. It has long been known that some antibiotics that are short-acting in domestic animals have long-acting properties when used in extra-label species (i.e. 3rd generation cephalosporins in reptiles). The definition of ‘long-acting’ is somewhat ambiguous and applies only to those species for which the drug is labeled. In most cases, the duration of long-acting antibiotics ranges from 48 to 150 hours depending on the drug and the species receiving it. Time-dependent antibiotics are most often suited for long-acting formulations as their mechanism of action requires that tissue levels remain above a minimum therapeutic threshold for an extended period of time rather than reaching a specific peak. Beta lactam antibiotics have been targeted because of these properties. In order to make a formulation long-acting, the drug must be changed in some manner; either in its chemical structure, its protein binding capability, or its carrier which keeps it in tissues.

One of the first long-acting antibiotic formulations that has recently been developed is a 3rd generation cephalosporin, cefovecin (Convenia, Zoetis). In dogs and cats, an 8 mg/kg dose provides therapeutic levels for 14 days. The duration of action in exotic species has been variable, with the formulation not having long-acting effect in reptilian and avian species. Conversely, another 3rd generation cephalosporin, ceftiofur crystalline free acid (Excede, Zoetis) does have long-acting properties in birds and reptiles. This formulation has decreased water solubility, retarding dissolution of drug particles and the injection site. In helmeted guinea fowl and American black ducks, a 10 mg/kg dose administered in muscles produced therapeutic levels for 3 days. A 15 mg/kg dose in ball pythons resulted in 5 day dosing recommendations.

Tulathromycin (Draxxin, Zoetis) is a long-acting bacteriostatic semisynthetic macrolide that achieves long-acting properties through extensive absorption and tissue distribution which results in decreased clearance. This product has some potential as an intraleisonal antibiotic for treatment of abscesses although is it labeled for small ruminants and pigs.

Azithromycin (Zithromax, Pfizer) is a macrolide that concentrates in the host’s cells. In ball pythons, 10 mg/kg oral dosing provided therapeutic levels for 2-7 days. Dosing in Amazon parrots and blue and gold macaws for uncomplicated, non-intracellular infections is 10-20 mg/kg every 48 hours for five doses. However, dose and duration of therapy increases for intracellular infections such as Chlamydioides, with dosing frequency increased to every 24 hours. Azithromycin has also been used as a component of...
antibiotic therapy to treat Mycobacteriosis in birds, in combination with rifabutin and ethambutol, although treatment is controversial due to the pathogens zoonotic potential. Use of azithromycin has not been studied in passerine species.

Tetracycline is bacteriostatic, with good oral absorption and tissue penetration, however its use in treating bacterial infections has declined over time due to the development of antimicrobial resistance. Long-acting tetracycline, LA-200 (Liquamycin) contains oxytetracycline with a co-solvent (2-pyrrolidone). When injected into muscle, the solvent is carried away while the drug precipitates at the injection site and is slowly dissolved to provide minimum inhibitory concentrations of antibiotic for a minimum of 72 hours in approved species. Tissue necrosis has been reported at the site of injection. Dosing of 20 mg/kg in a macropod species (tammar wallaby) did not provide reliable data regarding duration of efficacy. Five day dosing, using 10 mg/kg, has been recommended for American alligators. Recently, studies in humans have shown efficacy of long-acting tetracyclines in treating methicillin-resistant *Staphylococcus aureus* infections.

Florfenicol (Nuflor, Merck Animal Health) is a long-acting bactericidal phenicol labeled for large animals. It contains polyethylene glycol, propylene glycol, n-methyl pyrrolidone as solvents. When used in cattle, minimum inhibitory concentrations of drug are achieved for a minimum of 48 hours. Phenicol antibiotics provide broad spectrum antibacterial coverage, including anaerobic spectrum, making these drugs particularly useful in some small herbivores in which β-lactam antibiotics have limited use. However, chloramphenicol administered orally may result in anorexia in those patients. Florfenicol administered at 30 mg/kg every 24 hours as a subcutaneous injection in guinea pigs produced no adverse effects in this author’s experience.

**Formulation and delivery**
A significant amount of work has been done evaluating the efficacy and usefulness of antibiotic-impregnated beads for the delivery of sustained release of antimicrobials. New work is being performed to evaluate the potential of this method for delivery of other therapeutics including chemotherapeutics and anti-fungals. Antibiotic-impregnated beads embedded into tissues elute high concentrations of the antimicrobial at the implant site. A wide array of antibiotics have been used including aminoglycosides, β-lactams, macrolides, metronidazole, fluoroquinolones, and neomycin. Antibiotic powder may be incorporated into polymethylmethacrylate (PMMA) or plaster of Paris (calcium sulfate) to customize spectrum and size of implant. Care must be taken to use aseptic technique and gas sterilization is needed to avoid decreasing antibiotic potency.

Enrofloxacin, a fluoroquinolone, has long been used as an extra-label antibiotic in exotic species due to its efficacy against gram-negative bacteria. As a concentration-dependent antibiotic, less frequent dosing is permitted as compared to other antibiotics. Enrofloxacin is available as an injectable solution in two concentrations (22.7 mg/ml and 100 mg/ml) or as a tablet in three sizes (22.7 mg, 68 mg, 136 mg), but no oral liquid formulation is available. However, compounded oral suspensions or solutions remain stable at room temperature for 8 weeks. The 2.27% injectable solution may be administered by oral route. The large animal injectable product may be used to create compounded solutions but should not be administered orally undiluted as oral ulcerations have been observed. Extra-label use of fluoroquinolones is prohibited in food-producing animals. Besides enrofloxacin, few studies have been done using fluoroquinolones in exotic animals.

Aminoglycocides, including amikacin and gentamicin, are concentration dependent, bactericidal antibiotics with broad gram-negative spectrum. The lack of oral bioavailability and association with nephrotoxicity of these drugs make this class of antibiotic not ideal for use in exotic animals. Nebulization therapy is an effective means of administering therapeutics into the large and small airways. Incorporating an aminoglycocide into nebulized solutions delivers antibiotic to the site of infection allowing for local treatment without systemic absorption.

**References**
Staying Outside the Box: Strategies for Managing Unusual Exotic Pets
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Provision of quality care to zoological species requires familiarity of the clinician with the unique natural history, husbandry, anatomy and physiology of individual species. However, application of the principles of veterinary medicine transcends species differences. More and more people in today’s society are choosing to own exotic pets in addition to or in lieu of the traditional dogs and cats commonly seen in veterinary practice. Their small size and housing, ease of care, and human-animal bond potential are optimal for some pet owners. Often owners of exotic pets will make every effort to provide their pet with the best veterinary medicine has to offer. By offering service to exotic animal patients, you are meeting a tremendous demand that will be highly valued by your clients and/or employers.

Role of the veterinary team
Exotic pet practice offers the practitioner an opportunity to work with a large variety of species. While professionally rewarding, this also represents a significant challenge. The diversity of exotic pets kept in captivity dictates that the veterinary practitioner be versed with the health and husbandry of a wide array of species commonly kept as pets. Husbandry factors are often at the root of medical conditions seen in these animals. A thorough history, including husbandry information, is necessary for appropriate case management. Training of veterinary support staff will enhance the veterinarian’s ability to accommodate these species in a busy schedule. Providing adequate time for acquisition of a thorough history requires a minimum appointment time of 30 minutes. Longer appointment times minimize stress to the patient and allow necessary time for client education. Knowledgeable front office staffs are also critical in facilitating exotic pet care. Staff should be familiar with the species being seen and should demonstrate their interest to your clients. Statements that are derogatory regarding ownership of certain species or the decisions of owners to spend money on pets that are deemed “inexpensive” should be avoided.

Natural history and husbandry
It is important to educate yourself regarding species identification and the natural history of the species you’re seeing. Certainly years can be spent learning all there is to know about every species kept as a pet, but a general working knowledge of those commonly kept will lend confidence to your client interactions. In addition, knowing the natural history of the species presented to you will help you identify the husbandry requirements of that animal when kept in captivity. The goal in keeping any exotic pet is to maintain it in an environment that is as close as possible to that it would be living in if it were free-ranging. Often the problem that an exotic pet presents for is related to deficiencies or excesses of environmental conditions or their diet. Being familiar with the husbandry requirements of a species will help you identify potential problems and give insight in implementing treatment recommendations. It will also allow you to help your clients determine if that particular species would make a good pet for them based on their circumstances.

The internet provides a readily available means to search for information helpful in identifying an unknown species. While this is not often a concern when working with exotic mammals, or even birds, reptiles kept as pets range from the common bearded dragon to those unfamiliar to the practitioner such as the uromastyx. Unfortunately, in some situations, the owner may not even be aware of what species they own. Internet sources range in the accuracy of the information they provide but many zoos, nature organizations, and species interest groups maintain sites that provide images and basic natural history information that provide clues to the species. Additionally, field guides or other texts are useful when seeking this information. Once the species is known, determine where that animal is found in nature and how it utilizes its environment. Species natural history includes information regarding the geography, diet, and behavior associated with that species. Geography provides important details about the animal’s climate, including temperature range, humidity, light cycles and seasonality. It also provides details regarding the terrain. Behavioral characteristics are important for determining the social nature of the species, social hierarchies, diurnal or nocturnal activity, and whether seasonal variation in activity should be expected.

The details gained from reviewing a species natural history allow practitioners to critically evaluate their patient’s husbandry, even if the practitioner is relatively unfamiliar with the species. Husbandry refers to the conditions under which the animal is maintained in captivity. It includes information pertaining to caging, including the substrate that is used, the size and ventilation of the cage, temperature and humidity of the environment, exercise opportunities that are provided including furniture or other structures within the cage, what lighting is provided and by what means, and how often is cage cleaning performed. Husbandry also pertains to the diet consumed by the animal including treat foods. Exposure to conspecifics, other animals, and the source of the animal are also important aspects of husbandry. An animal that is normally found free-living in a desert habitat eating an invertebrate diet may likely develop clinical disease if kept in manner that is not reflective of that natural history.
History
A complete history is of paramount importance in providing you with all the information needed to adequately assess the health of the exotic patient. Developing a pre-printed history form will facilitate this process by identifying problems in husbandry to help focus your exam and make specific care recommendations. Additionally, if concerns regarding the patient’s husbandry are addressed prior to the onset of problems, you have provided your client with excellent preventative health care for their pet. Sample history questions should include:

- How long have you owned this pet?
- Where did you acquire this pet?
- What is this pet’s diet (be specific)?
- Are vitamin supplements or medications used for this pet?
- Is water provided for this pet? In what manner?
- How is this pet housed? Describe any cage furnishing the pet has access to.
- What substrate is used in the pet’s enclosure?
- How often is the cage/enclosure cleaned?
- What is the temperature & humidity of the pet’s environment?
- What light is provided for the pet (be specific)?
- What other animals have your pet been exposed to?
- Have you recently added any new pets to your household (within the last year)?
- What problem is your pet currently experiencing?

Physical exam considerations
The key to performing a thorough physical examination on any species, traditional or exotic, is doing it the same way every time. Thorough physical examinations can be done in any number of ways, including proceeding as you would for a dog or cat patient. Consistency is the key. Always be sure to record an accurate weight. Remember that most exotic species that are kept as pets have not been domesticated as a whole and have retained the instincts that protect them in the wild. A sick animal in its natural setting would most often be predated so most exotic species hide their illness. Having an idea of what is normal for a specific species will help you detect the subtle changes that could indicate a problem. Try to plan ahead and have all needed supplies available so as to complete your exam efficiently and safely.

Case plan
The basic principles of case management are applicable to the management of exotic animal patients. Often a physical examination reveals the underlying presenting problems, giving clues to the veterinarian to develop an informed diagnostic or treatment plan. At times, however, the cause of the clinical presentation is masked and diagnostic testing becomes even more critical. When ancillary testing is not available or non-diagnostic, understanding disease prevalence can help the practitioner direct treatment of the patient to the most likely etiology. Begin by noting the problems which may be described by the owner, identified in the history, or assessed during physical examination. Review the problems and develop a differential diagnosis list to include basic diseases. For instance, if a 3 year old male castrated ferret is presented for a 2 day history of diarrhea and the history included appropriate husbandry, free time play, and the addition of a new cage mate, it is not necessary to have extensive knowledge about ferret diseases to proceed. The problem list might include: 1. Diarrhea, 2. New cage mate, 3. Access to potential foreign body. From this problem list, you could create a differential list of enteritis (inflammatory diseases including IBD, or infectious causes including clostridial, corona virus, and helicobacter enteritis), foreign body ingestion, and neoplasia. Once you have created your differential diagnosis list, you can choose the diagnostic tests that will help you hone in on the underlying etiology and reference specific information to help develop a treatment plan for that patient. Armed with your complete case data, you will be able to discuss treatment options with your clients confidently and implement therapy efficiently.

Equipment
Most of the equipment needed for exotic animal patients is the same that you use to see dogs and cats. A few supplies, however, are essential and will make the transition to seeing exotic pets much smoother. Most importantly, an accurate gram scale is necessary to weigh these small animals to help determine their health status as well as to determine appropriate dosing of medication. Small plastic containers can be used to restrain the animals while positioning them on the scale. Towels of varying sizes are useful while restraining exotic patients. Avoid towels with fringe or strings that could entangle an animal’s appendage or nail. General anesthesia is beneficial at times to thoroughly examine a patient or to perform procedures. Isoflurane or sevoflurane gas anesthesia administered via mask or induction chamber is ideal for this purpose. Supplemental heat is often required by compromised exotic patients and may be provided using an incubator or heating pad. Finally, appropriately sized needles (25-27 gauge) and syringes (TB or insulin) will lend to efficient drug administration and venipuncture. Once a practice is committed to caring for exotic animal patients, additional supplies will be
identified which will expedite the care of these species. Developing a good working relationship with a compounding pharmacy will help to avoid the stocking of an excessive drug inventory. Additionally, identifying a laboratory experienced in exotic animal hematology and diagnostic testing or establishing that ability in house is critical to case management.

**Basic skills**
The best time to practice your skills pertaining to exotic animal species is when you are presented with a healthy animal. By learning what is normal for the species, you will more efficiently identify abnormalities allowing you to direct your diagnostic and therapeutic efforts accordingly. Often healthy animals are presented for routine examinations and grooming procedures. Take this opportunity to speak to the owner regarding the importance of periodic health checks. Many small exotic species have relatively short life spans making bi-annual exams ideal. Grooming procedures, such as nail trims and wing trims, provide an excellent opportunity to practice handling techniques and to become familiar with what is normal. Care must be taken to perform these techniques correctly so as to not stress the animal unduly and to avoid causing injury.

**References**
Providing supplemental enteral nutrition to avian and exotic patients suffering from illness or injury minimizes catabolism and gastrointestinal morbidity associated with anorexia while optimizing immune system and organ function and providing nutritional precursors necessary for healing. When determining the nutritional needs of an animal, it is important to account for its metabolic rate and energy requirements. Providing enteral nutrition via assisted feedings or feeding tube utilizes the functional gastrointestinal tract in patients that cannot or will not eat.

**Importance of enteral nutrition**
Nutritional support of zoological pet species is an important component of providing quality care to the sick or injured patient but is often a secondary consideration after providing medical or surgical intervention. Small body mass and high metabolic rates predispose these animals to significant weight loss and decompensation when they demonstrate a reduction in dietary intake. In certain species, such as those exhibiting hind-gut fermentative digestion, decreased intake of dietary fiber results in gastrointestinal motility disturbances including gastric stasis and ileus. Providing enteric nutrition, by means of either assisted feedings or administration of food through a feeding tube, minimizes catabolism in animal patients while optimizing immune system and organ function, and providing nutritional precursors necessary for healing. When deprived of food, enterocytes lose nutritional support for their high rate of replacement and the intestine atrophies. Intestinal atrophy is also seen in animals receiving parenteral nutrition, supporting the importance of enteral nutrition in anorexic patients. Ideally, nutritional support would be provided within 24 hours of the onset of injury or illness, however refeeding should only be instituted after the patient’s fluid, electrolyte, and acid-base abnormalities have been corrected, and normothermia established.

**Developing a feeding plan**
When determining the nutritional needs of an animal it is important account for its metabolic rate and energy requirements. Implementing a feeding plan is indicated when the patient is hyporexic, has a loss of >10% body weight, is malnourished, has expected on-going losses, has an increased energy demand, or is hypoproteinemic. A reasonable expectation is that energy requirements of an individual patient will change over time with the course of disease and recovery. Assessing the patient often and knowing its caloric requirements is critical in providing total nutrition to the recovering patient. An accurate record of the patient’s food consumption, including volume, diet, and time of feeding should be maintained in addition to recording the patient’s weight. Minimally, the weight should be measured daily, prior to feeding.

Feeding plans must be individualized for the patient taking into account the patient’s needs, species and diet, age, reproductive status, and physical condition. Attitude and mentation are also important as this will affect the route with which supplemental nutrition may be provided. Patients that are obtunded or mentally inappropriate are unlikely to self-feed and may be at risk for regurgitation and/or aspiration. Self-feeding is ideal if the patient is able to maintain appropriate nutritional parameters through this route. Attempts to encourage patients to self-feed include such strategies as offering naturally preferred foods such as fresh greens for herbivores or seeds for parrots. While these food items do not offer total nutrition when fed solely, they do stimulate the patient’s appetite which may promote self-feeding. Additionally, understanding the patient’s natural feeding strategies and offering foods in a way that optimize these natural behaviors will also encourage self-feeding behavior. Once a patient has started to self-feed, the composition of the food offered should be altered to achieve balanced nutrition.

When a patient will not self-feed, or is unable to do so, feeding plans must be altered to ensure necessary nutritional intake. Hand-feeding, or coax feeding, is time intensive as it entails frequently offering preferred foods to the patient to encourage them to eat. Often, this strategy is utilized by owners caring for patients at home but is not practical for hospitalized patients due to the manpower required. Assisted feeding, or syringe feeding, is an effective means of providing supplemental nutrition in the form of a gruel or slurry introduced into the oral cavity of the patient. Syringe feeding works well in small mammal patients as well as for some reptiles but does require that that patient be able and willing to swallow. Gavage tube feeding bypasses the patient’s oral cavity, negating their willingness to swallow, and introduces the diet directly into the esophagus or crop. Again, gruel diets are necessary for this feeding strategy as the diet must be accommodated by the diameter of the feeding instrument. For patients that will require long-term assisted feeding, placement of an indwelling feeding tube is warranted.

Providing enteral nutrition via feeding tube utilizes the functional gastrointestinal tract in patients that cannot or will not eat. Additionally, administration of food through a feeding tube is significantly less stressful for uncooperative patients than restraint and
metabolic rate at complete rest, equals the patient’s weight measure in kilograms multiplied to the 0.75 power times a species coefficient (K). The species coefficient takes into account the type of animal’s metabolic rate. The K value for placental mammals is

The caloric requirements of the patients must be calculated and those requirements met on a daily basis through self or supplemental feeding tube placement. Feeding tube placement should be considered in any animal where the stress associated with syringe feeding is likely to result in significant morbidity, or when the need for assisted feeding is protracted. Patients with feeding tubes can often be sent home to be cared for by owners who have been trained in the administration of food, fluids, and/or medications through the tube. Placement of feeding tubes is useful when a diseased portion of the gastrointestinal tract needs to be bypassed in order to allow adequate time for healing. Feeding tubes are named based on the location of placement in the gastrointestinal tract. For instance, a nasogastric tube is placed in the nasal passage and extends to the stomach. Factors specific to the patient must be considered when selecting the most appropriate tube option including nutritional status of the patient, condition of the gastrointestinal tract, the animal’s ability to tolerate anesthesia, and the suspected duration that the animal will require nutritional support through assisted feeding. Additionally, financial considerations, facility constraints, and practitioner skill may limit available options.

Red rubber feeding tubes offer an inexpensive and readily available option for indwelling enteric feeding (Sovereign Feeding Tube, Tyco Healthcare, Covidien, Mansfield, MA). Silicone feeding tubes, such as those developed specifically for exotic animals (Silicon Feeding Tubes, 3.0 mm-6.0 mm, Veterinary Specialty Products, Mission, KS) or those used in small animal practice (Feline Esophagostomy Tube, SurgiVet, Smiths Medical, Dublin, OH), as well as polyurethane feeding tubes (Mila International, Inc., Erlanger, KY) are also available and are softer and more durable than red rubber tubes which tend to stiffen with age. The size of indwelling feeding tubes ranges from 3.5 French to 18 French and the appropriate choice is dependent on the location of placement and the species being treated. French is a unit of measurement equaling 0.33 mm. Nasoesophageal (NE) and nasogastric (NG) tubes, used most commonly in rabbits, are typically 3.5-8 Fr. due to the small size of the naris and delicate tissue in the nasal passage, while tubes used for esophageal feeding tend to be larger.

In extremely calm or depressed animals, topical anesthesia using ophthalmic proparacaine solution or light sedation may be sufficient to place NE or NG tubes. However, general anesthesia is necessary for placement of other tube types and would also be indicated in fractious or stressed patients undergoing NE or NG tube placement. NE tubes terminate in the distal third of the esophagus, proximal to the lower esophageal sphincter, unlike NG tubes which reach the stomach. One reported concern of NG tubes is that the tube’s interference with the lower esophageal sphincter predisposes the animal to gastric reflux and subsequent esophagitis, however this concern is currently unproven. Typically, esophagostomy (E) tubes terminate in the distal third of the esophagus when placed in mammals but are extended into the gastric lumen in birds and reptiles. Tube placement should always be confirmed radiographically prior to initial use. A permanent marker can be used to mark the location on the tube where it exits the body so that tube migration is more easily detected. Only liquid or finely ground diets should be introduced through small diameter tubes (<8 Fr.) and administration of crushed medications should be avoided. The tube should be flushed with 5 ml of sterile water before and after feeding to prevent tube clogging and to confirm appropriate tube placement. If coughing or a change in respiratory character is noted after the water flush, tube position should be confirmed. Feeding tubes do not prevent animals from self-feeding and fresh food and water should be made available to encourage this behavior.

**Determining energy needs**

The caloric requirements of the patients must be calculated and those requirements met on a daily basis through self or supplemental feeding. As in domestic species, metabolic rates are measured in kilocalories per day (kcal/day). The basal metabolic rate (BMR), or metabolic rate at complete rest, equals the patient’s weight measure in kilograms multiplied to the 0.75 power times a species coefficient (K). The species coefficient takes into account the type of animal’s metabolic rate. The K value for placental mammals is 70, whereas for marsupials the K value is 48. K values for parrots, passerines, and reptiles are 78, 129, and 10 respectively. Once the BMR is calculated, the maintenance energy requirement (MER), the metabolic rate based on activity), can be determined by multiplying the BMR by the activity level of the patient (1-2, 1.5 for convalescing animals, 2 for growth). Once the number of kilocalories needed for MER are determined, the amount of food needed to meet that requirement can be calculated and divided over the day’s feedings. The best way to determine if a patient is receiving appropriate nutrition is to weigh the patient daily on an accurate gram scale. Supplemental feeding can then be adjusted as appropriate. Oral administration of a gruel diet will also help hydrate the patient. Several commercial diets are available. Patients should be fed approximately 50 ml/kg/day divided into feedings every 4-6 hours.

**Enhancing gastrointestinal function**

Optimizing a patient’s nutritional support is dependent on optimizing gastro-intestinal function. Gastro-intestinal pathology should be bypassed whenever possible and therapies designed to promote GI function implemented. Probiotics may be useful in re-establishing the predominant microflora of the gastro-intestinal environment, effectively out-competing opportunistic pathogens that can compromise patient status. Endotoxin binders, such as cholestyramine, may be incorporated into feedings if endotoxic shock is a concern. H2 blockers, GI protectants, anti-gas therapies, and prokinetics may also be useful in optimizing GI function.
Patient monitoring and transitioning to self-feeding

Patient that are receiving supplemental nutrition must be weighed daily. This will be the best indicator of whether or not the feeding plan is being successful in meeting the patient’s needs. However, patients that are receiving chronic care may also lose body condition due to inactivity or disease processes. Therefore, it is prudent for the care provider to routinely assess body condition as well as gastrointestinal output volume, consistency, and appearance. Whenever a feeding plan is in place, regardless of the route of administration, the patient should be offered food to have the option for self-feeding if medically appropriate to do so. When transitioning a patient to self-feeding, supplemental feeding should be decreased gradually, however care should be taken to not decrease nutritional quality. Skipping a feeding may be preferred to decreasing volume of any given feeding. Implement strategies to stimulate feeding behavior and ensure optimal environmental conditions. Healthy exercise has also been shown to stimulate appetite in convalescing animals.

References
Let’s start with what we all have in common regardless of the path we take as veterinarians. It’s our Veterinarian’s Oath, and it reads: *I solemnly swear to use my scientific knowledge and skills for the benefit of society through the protection of animal health and welfare, the prevention and relief of animal suffering, the conservation of animal resources, the promotion of public health, and the advancement of medical knowledge. I will practice my profession conscientiously, with dignity, and in keeping with the principles of veterinary medical ethics. I accept as a lifelong obligation the continual improvement of my professional knowledge and competence.*

The Oath guides us to use science and medical ethics in our veterinary practice. The Oath does not define differences in veterinary practices for food animal and companion animal medicine. The responsibility of all veterinarians is to do what is right for the animal first. For food animal veterinarians like me, who practice herd health medicine, what is best for the animal is often based on what’s best for the health of the herd, while the practice of companion animal medicine is largely based on what’s best for each individual pet. With these differences in mind, how do we decide what’s best for a food animal versus a pet? First let’s take a look at what swine veterinarians do to take care of pigs that are raised for food.

A swine veterinarian’s job is to protect the health and welfare of pigs, as well as food safety and public health. It’s a job that keeps evolving as farming methods continue to change. With the help of swine veterinarians, pig farming and pigs have changed more than any other sector of animal agriculture during the last 50 years. Veterinarians have had a significant influence on how pigs are raised, from the design and construction of housing, to nutrition management, to the training of farm employees, and the list goes on. We’re focused on preventive medicine, animal welfare, herd health and public health. We provide advice on disease prevention and treatment, biosecurity, production management, genetics and nutrition.

Swine veterinarians play a big role in pig farming, and have an important role in the discussion about the science and ethics of animal care and welfare on today’s farms. The discussion about modern pig farming practices related to animal welfare focus primarily on animal housing, health management tools, pain management, and euthanasia. Every environment and every animal housing system has its benefits and challenges, and there are multiple factors for farmers to consider. The primary housing systems available to pork producers are pasture, hoop barns, and specialized barns. Raising pigs outdoors appears to have clear advantages for the animal. The pigs have room to roam and the ability to root and forage. However, being outdoors creates several challenges for the pigs and the farmer that include bad weather, parasite control, disease spread by wild animals, and keeping predators away. Hoop barns are tent-like structures that have concrete sidewalls. The housing system relies on deep bedding to absorb manure and natural airflow for cooling pigs in the summer. They offer pigs some exposure to the outdoors while providing them with protection from predators. They also have some drawbacks that include cooling pigs in hot weather, keeping manure away from the pigs, identifying and treating sick pigs, cleaning and disinfecting the barn to control disease, and exposure to parasites. Specialized barns give farmers a lot of control over managing each pig’s health and the health of the herd. The barns can be designed to manage pigs through each phase of their growth cycle, and include breeding and birthing units, nurseries, and finishing barns where pigs grow to market weight before they are sold. The barns are climate controlled in the winter and summer, provide protection from parasites, keep manure away from pigs with slatted floors, and are easy to clean and disinfect for disease control. The barns, however, do not give pigs a lot exposure to the outdoors and hundreds of pigs can be housed in the same barn, so biosecurity measures to prevent disease require constant attention. Many farmers moved their pigs into specialized barns during the last 30 years as agriculture science and technologies pushed forward to address animal health. Because pigs overall are healthier, sows are giving birth to more piglets per litter. And because the survival rate continues to improve for piglets, more of them get a stronger start. That means they grow bigger and healthier, and produce more and better meat than ever before. That’s why, since the 1960s, annual meat production per sow has more than doubled. (Source: USDA/NASS - Ron Plain, University of Missouri, 2009). The combination of modern housing systems, along with better nutrition, breeding practices and disease prevention, have resulted in healthier, more productive pigs.

All of the housing systems previously mentioned allow pigs to move about to a large extent in groups.

Housing options for pregnant sows can differ somewhat. The AVMA and AASV (American Association of Swine Veterinarians) say that regardless of the type of housing system in use, all sow housing systems should minimize aggression and competition among sows; protect sows from detrimental effects associated with environmental extremes, particularly temperature extremes; reduce exposure to hazards that result in injuries, pain, or disease; provide every animal with daily access to appropriate food and water; facilitate observation of individual sow appetite, respiratory rate, urination and defecation and reproductive status by caregivers; promote good air quality and proper sanitation; and allow sows to express most normal patterns of behavior. The AVMA also adds that adequate quality and quantity of space should be provided. The primary options pork producers have available for pregnant sows include group pens and individual stalls. Each system has unique advantages and disadvantages for the sow.

In group pens, sows have the benefits of social interaction, and room to move around the pen. At the same time, those benefits create challenges for the sow including injuries from scratching and biting, lameness from fighting, and competition for food and
water. In groups, pigs, just like many other animals, develop and establish a dominant social order, which can be aggressive at times. Studies published by the Council of Agriculture Science and Technology (CAST) show that the amount of fighting among sows and the resulting injury and social stress are substantially greater in sows kept in group pens rather than individual stalls. There are also benefits and challenges for gestating sows in individual stalls. The stalls are widely used by farmers to ensure proper nutrition, monitor individual health, and manage sow aggression. The challenges for sows in gestation stalls include restricted movement and limited social interaction. Individual farrowing stalls are also used to protect sows and their piglets immediately after birth and during the weaning process. The highest losses in the pig lifecycle occur within 3 to 4 days of birth. Farrowing stalls are widely used to protect newborn pigs from being crushed by sows that sometimes accidently lay on them, manage sow aggression, and ensure proper nutrition for the sow and her piglets through the weaning process. There is broad consensus in the veterinary community that the key to ensuring animal welfare for pigs rests primarily with the caretaker regardless of the housing used. The executive director of the AASV noted in the Journal of the American Veterinary Medical Association (JAVMA) that: “Gestation stalls and pens each have advantages and disadvantages. Each can provide for sows’ welfare when properly managed, and caretakers’ skills are the most important factor.” (Source: Dr. Tom Burkgren, AASV, JAVMA News: Vol. 241, No. 3, August 1, 2012).

Farmers have made a commitment to animal welfare training. During the last 20 years, the National Pork Board has implemented caretaker training and certification programs to address animal welfare. Pork Quality Assurance Plus® (PQA Plus®) is a program that helps farmers measure, track and provide training for things like food safety, animal care and disease prevention. Nearly 62,000 pork workers have been trained and certified through the program. (NPB, November 2014). Transport Quality Assurance® is a program that focuses on teaching the proper handling and transport of pigs to ensure their well-being at every stage of life. Almost 30,000 farm workers have been trained and certified through the program. (NPB, November 2014).

Pork producers also rely on science and technology to help them optimize pig health, and reduce and prevent disease. Their health management tools include genetics, with the focus on consistently breeding animals with the optimum traits such as female lines for productivity and terminal cross boars for growth and carcass; nutrition, with the focus on feeding herds a prescribed diet, controlling nutrients and optimizing proteins, and carefully managing pig diets at every stage of growth; and strategic medicine, with the focus on disease prevention and control, vaccinations and medications, most of which are used under the supervision of a veterinarian.

Strategic medicine also helps pork producers raise healthy pigs. Farmers work closely with veterinarians to develop a comprehensive herd health program, which may include the use of antibiotics. When treatment is needed, it’s based on the recommendations from veterinarians and in strict compliance with FDA rules. Pork producers, with input from veterinarians, have long used FDA approved antibiotics as part of an overall herd health management program. In recent years, public perception about these practices has called into question what’s best for the animal and for our food supply. Farmers are also adapting to changing FDA regulations that are significantly impacting their access to some antibiotics and how they can be used. When pigs are treated with antibiotics, there are safeguards in place to protect the food supply. The FDA, with its rigorous scientific processes, approves antibiotics and regulates how antibiotics are used in food animals on the farm to ensure safe food. To obtain FDA approval for any drug, the drug’s sponsor must demonstrate the drug is safe for animals, humans, and the environment. Producers must follow label directions and utilize appropriate withdrawal times. The USDA’s Food Safety and Inspection Service (FSIS) tests meat to ensure there are no harmful residues entering the food supply.

Veterinarians and farmers have had long-standing FDA approval to use antibiotics in food animals, as described on drug labels, for disease treatment, prevention, and control, and for nutritional efficiency or growth promotion. Antibiotics may be injected and/or used in feed or water for controlling disease when pigs are most susceptible to illness, and for treating sick pigs. Certain antibiotics are also used in feed for nutritional efficiency and as a group are called antibiotic growth promotants (AGPs). Used in this way, they keep the animals healthy, and healthy animals need less feed per pound of weight gain. This is the area where FDA rules are changing for certain antibiotics that are deemed medically important for human health – effective January 1st, 2017, AGP use will go away for these medications and veterinary oversight will increase. Affected feed medications will only be allowed to be fed under a Veterinary Feed Directive or VFD. Affected water medications will be available by veterinary prescription only. Over the counter sales of medically important feed/water antibiotics will be discontinued. While these changing FDA regulations will limit how farmers gain access to some antibiotics for their food animals, the FDA is clear that farmers need these medicines for their animals. The FDA states the following on its website: “FDA also believes strongly that sick animals need treatment, and that these antimicrobial drugs should remain available for the purposes of treating, controlling or preventing disease in food-producing animals.” (Source: http://www.fda.gov/animalveterinary/guidancecomplianceenforcement/guidanceforindustry/ucm216939.htm)

The National Pork Board’s PQA Plus program already educates producers about good on-farm production practices, including responsible antibiotic use, and will focus more resources on training farmers how to comply with the new FDA rules. A core component of PQA Plus training involves these guiding principles for Responsible Antibiotic use: Principle I: Take appropriate steps to decrease the need for the application of antibiotics; Principle II: Assess the advantages and disadvantages of all uses of antibiotics; Principle III: Use antibiotics only when they provide measurable benefits; Principle IV: Fully implement the management practices
The pork industry is supporting the FDA’s changes in antibiotic use on the farm through a number of programs that will help educate pig farmers and fund research to find better ways to use antibiotics for disease prevention and treatment. The National Pork Board 1) has created a Blue Ribbon Panel On Antibiotics that includes veterinarians and professors, representatives from McDonald’s and Walmart, and the former Center for Disease Control (CDC) Director of the Office of Antimicrobial Resistance; 2) is funding $750,000 in new research to look at how farmers can improve their use of antibiotics and for alternatives for disease prevention and treatment (that’s in addition to the $5.3 million that has been spent on research since 2000); 3) is stepping up education and evolving certification programs like PQA Plus with an emphasis on responsible antibiotic use; and 4) is increasing industry communication to keep pig producers up-to-date on FDA rules so they are in compliance with antibiotic use.

What about hormones and pork? Bottom line, there are no hormone-containing products approved for U.S. pork producers to use in nursery, grower or finisher pigs. The USDA's Food Safety and Inspection Service is responsible for ensuring the truthfulness and accuracy in labeling of meat and poultry products. Its policy regarding meat labels promoting “no hormones” states the following: Hormones are not allowed in raising hogs or poultry. Therefore, the claim "no hormones added" cannot be used on the labels of pork or poultry unless it is followed by a statement that says, "Federal regulations prohibit the use of hormones." (Source: http://www.fsis.usda.gov/FactSheets/Meat_&_Poultry_Labeling_Terms/index.asp).

Keeping pigs isolated from disease is another important herd health tool that pork producers use.

Access to barns is strictly controlled to keep people from exposing pigs to disease. Some producers require that people who work with their pigs first shower and put on clean coveralls and boots. Additional disease prevention measures include disinfecting any tools that are brought into the barns from outside. Barns are also kept clean with regular high-pressure washing and are disinfected and dried prior to restocking with a new herd to prevent disease from spreading among the pigs. Farmers also use modern herd management practices to prevent the introduction of disease. In all-in/all-out (AIAO) management systems, pigs are moved into and out of barns in distinct groups. By preventing the commingling of pig groups, farmers reduce the spread of disease. Facilities are cleaned and disinfected thoroughly between groups of pigs.

Unlike companion animal medicine, there are no FDA-approved drugs labeled for pain management in swine. Any product used for this purpose in pigs can only be used under provisions of the FDA’s Animal Medicinal Drug Use Clarification Act (AMDUCA). The act says FDA-approved drugs can be used in food-producing animals only when the health of the animal is threatened or suffering or death may result from failure to treat the animal. These products can only be prescribed by a veterinarian with a valid VCPR and use of these products must not result in any harmful residues. (Source: https://www.avma.org).

What about euthanasia considerations in swine, another area that differs from companion animal medicine? When a pig becomes sick or injured, farmers are faced with two courses of action: treatment or euthanasia. In some cases, euthanasia may be the best option for the well-being of the animal and the food supply. National organizations representing pig farmers, in partnership with the AAVS, have issued guidelines to educate animal caretakers on humane euthanasia methods. These guidelines provide clear, detailed instructions on how to perform euthanasia.

What about pig farming and the environment? Modern pig farming practices have not only helped farmers raise healthier pigs, but the pigs grow bigger and provide more and better meat than ever before. In the last 40+ years, pork producers have doubled the amount of pork they produce. At the same time, farmers have also significantly reduced the amount of natural resources they use to raise pigs.

Compared with hog farms in 1959, farmers today are using 78% less land, 41% less water, and have reduced their overall carbon footprint by 35. Reducing the pork industry’s impact on the environment is also the focus of the National Pork Board’s Sustainability Effort. This multi-year research program has been studying the four elements of pork production sustainability: 1) carbon footprint (greenhouse gases); 2) water footprint; 3) air emissions footprint; and 4) land use footprint. These studies have already resulted in production management tools for farmers that factor in all industry inputs and outputs for all stages of pork production. We also know that pig farming is actually one of the smallest sources of emissions. The Environmental Protection Agency (EPA) estimates that the pork industry contributes approximately one third of one percent of the total U.S. emissions. (Source: Data from EPA GHG Draft Inventory 2013). Farmers are determined to reduce that figure even more by using the latest technology and modern farming practices.

Another outcome of the science behind today’s pig farming can be found in the meat case. Pork tenderloin is now as lean as skinless chicken breast. The seven most common pork cuts are, on average, 16 percent leaner than 25 years ago, and saturated fat has dropped 27 percent. (Study conducted by USDA in conjunction with the University of Wisconsin-Madison and the University of Maryland). In 2012, the American Heart Association (AHA) certified pork tenderloin as a heart-healthy food and gave it the trusted Heart-Check mark, designating it an extra lean meat. This certification means that pork tenderloin has been screened and verified by the AHA and the Heart-Check mark can be displayed on pork tenderloin package labels to help consumers make heart-healthy food choices in their grocery store.
When you hear about animal welfare initiatives, remember the science and the Veterinarian’s Oath that underpins all of our work as veterinarians. The responsibility of all veterinarians is to do what is right for the animal and protect herd health. As an industry, pig farmers and the veterinarians who work with them do not tolerate animal abuse. It just makes sense, because the animals with the best care are the most productive. The pork industry has established its own guiding principles called WE CARE® to promote responsible and ethical farming practices, and continuous improvement. Through this program, pork producers pledge to put the highest ethical standards into practice every day to produce safe and nutritious food, protect and promote the well-being of their animals, protect the public health through all of their production practices, provide a safe workplace for their employees, make their community a better place in which to live, and safeguard their environment.
The first step in evaluating the role that environment can play in therapy and recovery is to assess the patient. Watching how they behave with an awareness of normal and “abnormal” behavior is key. Although behaviors associated with good health are reasonably consistent across cats, those that reflect poor health can vary widely. More importantly, the contrast between how this particular cat behaves when all is well and how the cat is behaving in the present circumstances will illuminate more than just “knowing” a list of normal and abnormal behaviors. For example, a cat that ordinarily enjoys being petted or handled now avoids being picked up, held, or petted even to the point of withdrawing. The ubiquitous presence of “smart devices” in homes capable of video and still photography is a powerful tool in understanding environmental challenges and the behavior of the patient in the home.

Tony Buffington and his team at The Ohio State published a powerful example of the effects of environment on sickness behaviors in JAVMA in 2011. In it they demonstrated significant increases in sickness behaviors, even in healthy cats, in response to unusual external events. Much of this effect was found in the response of cats to changes in their expected routine. These sickness behaviors included hypo- or anorexia, decreased eliminations over time, eliminating outside the litterbox, vomiting and diarrhea.

Having evaluated the patient, three aspects of the environment should be evaluated in order to make recommendations that will improve the health and well being of the feline patient: the macro environment (the house, apartment, neighborhood), the microenvironment (light, sound, temperature, odor, and the social environment (humans and other creatures in the macro environment).

Cats housed solely indoors present a special challenge to owners in that they assume responsibility of the cat’s entire life experience. Free-living cats are opportunistic predators of small prey. Their day is composed of inspecting the territory, interacting with other occupants, finding high places from which to observe, resting in safe places, sleeping, hunting, and eating. A large portion of the day is devoted to the acquisition of food. These activities, if a cat is deprived of the ability to act them out, can cause the stress of a barren, boring life. Thus, their human guardians must provide the indoor-housed cat with the ability to act like a normal cat.

The microenvironment has variable importance depending upon how much control the cat has over his ability to move away from an aversive stimulus. Light should reflect the normal day/night cycle and be composed of natural light spectrum, composed of the colors and intensity associated with normal daylight. Consistent day/night patterns are the least stressful.

Sound can have a significant impact based upon intensity and/or unpredictability. The auditory frequency of cats exceeds that of humans, making evaluation of this as a stressor difficult. In general sounds pressures indoors are above those in the natural setting. Loud music, verbal combat, multiple video devices or machinery are examples of sound pressures that may affect health.

Cats generally prefer a warmer environment than human occupants, around 880 degrees (F). The provision of warm bedding and multiple reliable, safe sleeping places, enough that there will be no conflict over their use among cats in multi-cat households. Strong odors can vary from aversive to toxic for feline patients. Chemical cleaners, perfume, dog odor, alcohol, smoking materials (including vapes), laundry detergent are some examples. These can, not only be aversive, but form toxic particulate matter that is subject to inhalation. Having evaluated the patient, three aspects of the environment should be evaluated in order to make recommendations that will improve the health and well being of the feline patient: the macro environment (the house, apartment, neighborhood), the microenvironment (light, sound, temperature, odor, and the social environment (humans and other creatures in the macro environment).

Cats are solitary hunters who consume multiple small meals a day after a burst of energy during the hunt and kill. Small frequent meals that encourage or mimic hunting behavior can have a very positive impact. Ad lib feeding is a significant predictor of both boredom and obesity. Food puzzles, active feeding, treats as a training reward all can bring the cat closer to its normal behavior repertoire. Bradshaw’s group has shown that while cats that are hungry hunt more vigorously and often than cats that are not, the hunting instinct is separate from the satiety center, so the hunting desire persists regardless of satiety.

Some cats develop a decreased preference for foods that have formed a large part of their diet in the past, the “monotony” effect. Some cats are described also as finicky eaters, though evidence suggests that food refusal is a common response to environmental threat. Water should always be provided at some distance from the food bowl. Cats seem to drink more water, a highly desirable behavior given the desiccated food many cats eat, when water is provided in multiple locations. There should be abundant enough locations to prevent competition. Some cats also have preferences for the method water is provided (fountain v. still), freshness, and bowl shape. Wide bowls filled to the brim and changed regularly may encourage some cats, as the rim of the bowl doesn’t touch their vibrissae.

Elimination is a matter of litter box, litter substrate, and location. Social issues around elimination will be addressed later. Cats seem to prefer unscented, clumping litter though in one study, scented litter was not aversive. The sensation of the litter on the bottom of cats’ feet may have significance. Spherical litter that can catch between toes or litter with edges such as wood chips may be unacceptable to some cats. Cats prefer large litterboxes, as their choice for an elimination site in the wild would be. Under the bed
clothing storage boxes about 29-30 quarts with wall height about 5-6” is ideal for healthy adult cats. Litter boxes are generally made of plastic and, as such, absorb particulate matter and odors. These are cumulative over time so inexpensive boxes are ideal as they should be replaced about every 6 months. Covered litterbox may be aversive to some cats as they trap odors within, as in the human experience of the PortaPotty. Social relationships may make a covered litterbox undesirable by allowing for conflict or stalking.

As solitary territorial animals, cats prefer predictability in all aspects of their environment. Predictability is safety. An environment that is changed in any way from small (rearranging furniture) to dramatic (changing residence) can be tolerable or not depending upon the scope of upheaval and the way in which it is implemented. Species specific behavior should be encouraged with the provision of scratching places, resting places, high spots for observation and safety, and perches with window access (variably desirable).

The quality of relationships comprises the social environment. Repeated interactions between cat and human allow each to make predications about the other. Allowing cats to define the type of social interactions they prefer help create a sense of control for them. Predictability lowers stress as confidence in interactions ensue. Unpredictability leads to fear reactions and the physiological consequences of that fear. Avoiding punishment behaviors may be more important in cats than other species. Because of their heritage as a relatively solitary species, cats have not developed coping behaviors found in more social species. Soft voices, indirect eye contact, slow blinks and movements, allowing the cat to initiate and control the manner of contact reduce the probability of a perceived threat.

Free-living domestic cats live in small groups consisting of related females and their offspring. Females will share parenting duties, pooling their kittens for feeding and safety. By contrast, males are more solitary, living on the margins of groups or ranging as widely as is necessary to find acceptable mates. Males and females hunt by themselves. Only females share their food and then only with offspring who are not old enough to hunt on their own. This independent nature may be negatively impacted by conditions in a multi-cat household.

Cats also do not have coping strategies to resolve conflict. Agonistic interactions may be simply avoided by reducing normal behaviors or decreasing encounters.

The adequacy of resources then becomes a paramount consideration. Unrelated cats do not share them, including feeding in separate locations, multiple resting places, litterboxes, water sources, and climbing access. When cats’ perception of safety diminishes, they respond by trying to restore control. During these responses, some cats become aggressive, some become withdrawn and some become ill. Conflict among cats can develop because of perceived threats from other animals.

Experience tells us (and the cats) that humans are not keen observers of cats. Cats have adapted to this by vocalizing more around humans than with each other. Utilizing questionnaires and pictures, encouraging home video and photos, veterinarians can gently guide cat guardians to better understand the needs of cats, the health consequences of not meeting them, the behaviors their individual cats perform that indicate a satisfactory life or not, and how to create a rich and satisfying life for their beloved family members.

Resources
Lower urinary tract signs (LUTS) – dysuria, peruria, pollakiuria and stranguria – are a common reason pet cats are brought to veterinary practices. When presented with a cat with these signs clinicians need to know whether this is the first episode or whether it is a chronic, recurrent disease as well as what other health problems the cat may have. Armed with this information an appropriate diagnostic plan can be made.

Cats may have multiple reasons for their clinical signs as well as other medical conditions and environmental requirements that need to be addressed. For example, Buffington et al. have presented evidence that some cats with severe, chronic LUTS seem to have a functional rather than a structural lower urinary tract disorder and that peruria can occur in apparently healthy cats exposed to stressful circumstances. There is significant overlap at the present time among treatment recommendations for some LUT disorders particularly with regard to ensuring that the patient’s environmental needs are met.

Severe chronic idiopathic LUTS has been described as a naturally occurring model of interstitial cystitis in women. Interstitial cystitis (IC) has been defined as a disease of chronic irritative voiding signs, sterile and cytologically negative urine and cystoscopic observation of submucosal petechial hemorrhages. The same description in which cystoscopy was not performed in cats but in which other appropriate diagnostic procedures did not identify a cause became defined as Feline Interstitial Cystitis (FIC)

In addition to epithelial abnormalities identified in the bladder of cats with FIC, investigators found significant alterations in components of acetylcholine synthesis and release in the esophageal mucosa from cats with FIC. This suggested that changes in the nonneuronal cholinergic system may contribute to alterations in cell-to-cell contacts and possibly communication with underlying cells that may, in turn, contribute to changes in sensory function and visceral hyperalgesia. Differences in sensory neuron anatomy and physiology also are present in cats with FIC suggesting a more widespread abnormality of sensory neuron function. The acoustic startle response is a reflex motor protective response to a perceived threat. It is a brainstem reflex response to unexpected auditory stimuli and is increased in cats with FIC.

Differences in sympathetic nervous system function have also been identified in cats with FIC. Among them are changes in the brain stem in the region associated with the most important source of norepinephrine in cats and humans. It is involved in such brain functions as vigilance, arousal and analgesia and mediates the visceral response to stress. Other changes in brainstem help to explain the waxing and waning course of symptoms and the aggravation of signs by environment stressors.

Some cats with FIC appear to have abnormalities in the hypothalamic-pituitary-adrenal axis such that there is a decrease in serum cortisol secretion compared with healthy cats. Adrenal glands in these cats were grossly smaller in cats with FIC when compared to healthy cats.

Cats with FIC often have variable combinations of comorbid disorders such as behavioral, endocrine, cardiovascular and GI problems. External stressors appear to exacerbate clinical signs of these disorders. Many human beings with IC suffer from variable combinations of comorbid disorders as well. These appear to have no consistent pattern of onset and so cannot be attributed to LUTS but rather may be some common disorder affecting more than one organ which then responds in its own way.

Ongoing research in both humans and cats with chronic LUTS has begun to include a more comprehensive evaluation of the entire patient. Nosology is defined as the classification of diseases. Until a better understanding of the larger picture of cats presenting with LUTS, naming this constellation of symptoms and organs systems involved should remain vague and not reflect only LUTS. Dr. Buffington has suggested “Pandora’s Syndrome” He and his colleagues, Drs. Westropp and Chew propose tentative criteria for diagnosis of Pandora syndrome:

1. Presence of clinical signs referable to other organ systems in addition to chronic idiopathic signs for which the patient is being evaluated
2. Evidence of early adverse experience (e.g. abandonment, orphaning) and which may differ by individual
3. Waxing and waning of severity of clinical signs with events that (presumably) activate the central stress response system
4. Resolution of signs with effective multimodal environmental modification

Whatever the eventual name, restricting the description of these patients to their LUTS does not capture all of the currently recognized features of the syndrome. A more comprehensive evaluation of cats with these and other chronic idiopathic signs may result in a more complete diagnosis and lead to additional treatment approaches that may improve outcomes. For example, the relationship between the environment and health is quadratic rather than linear, with both deficient and threatening environment increasing the risk of poor health outcomes.
Individual patients presenting with chronic LUTS benefit by a more comprehensive evaluation to elucidate the effect on risk for Pandora syndrome. Included in this history should be:

- Where the cat was obtained
- Any other health or behavior problems that may be present
- Structure of the cat’s environment – amount of time indoors, activity level, availability and management of resources, other cats in the home, people living with the cat.
- Presences of signs referable to other organ systems
- Perceived allergic responses to skin, lung or GI tract
- Any unusual or problematic behaviors

The physical exam should be performed with evaluation of the lower urinary tract last to avoid being distracted and missing other abnormalities such as over-grooming, obesity, acne, cardiac abnormalities or GI tract issues.

For an initial episode in an apparently healthy, young unobstructed patient, the most likely explanation is either a sickness behavior in an otherwise healthy cat or acute idiopathic LUTS. After ruling out other causes of LUTS, the client should be counseled regarding individually tailored multimodal environmental modification (MEMO) to make sure the cat’s environmental needs are being met. The client can also be taught to look for other signs of sickness behaviors and to evaluate response to MEMO for adequacy of accommodation.

Table 1
Forms used as part of the evaluation of cats presented the Ohio State University Veterinary Medical Center for evaluation of chronic lower urinary tract signs. These forms have not been formally validated beyond their face validity for cases in the authors’ practice area. They are offered as an example of an instrument that could be developed and validated for broader use

Cat and client history form
Cat’s name__________________ Owner name_____________________  Date_____________
Contact information: Telephone: ☐ ___________  E-mail: ☐ ___________
☐ Please check preferred method of contact

Cat Information: Breed__________ Color________ Date of Birth _______ Weight ☐ lb ☐ kg
Owned for? _____ years _____ months; ☐ M ☐ F ☐ Neutered? If yes, date: _______  (month/year)
Declawed? ☐ N ☐ Y If yes, Front only ☐ All four paws ☐

Body Condition (please check box that looks most like your cat):

☐Skinny ☐Lean ☐Moderate ☐Stout ☐Obese

Please check the boxes that best apply to your cat:

Diet: (please be as specific as you can, eg, Buckeye Best (company) Adult Chicken and Rice (flavor)

Wet food: name________________________ ☐None ☐25% ☐50% ☐75% ☐100%
Dry food: name________________________ ☐None ☐25% ☐50% ☐75% ☐100%

How many hours each day, on average: does your cat spend indoors?
☐Indoor only ☐18-24 ☐12-18 ☐6-12
☐0-6 Is time outside supervised? ☐Yes ☐No

If you have more than one cat, what is their relationship? ☐Not related
☐Littermate ☐Sibling ☐Parent-Offspring ☐Other (____________________)
Where did you obtain your cat (source)?
- Shelter
- Offspring from a pet I already own(ed)
- Purchased from a friend
- Gift
- Purchased from a breeder
- Purchased from a pet shop
- Stray/orphan
- Other ____________________________

Does your cat frequently (please check all that apply):
- Try to escape
- Pace at outside doors
- Cry at outside doors
- Hide
- Act fearful
- Act friendly
- Follow owners around the home
- Destroy things when left alone
- Act ‘depressed’ (little interest in feeding, grooming, environment, etc.)

Housing (______):
- Apartment: □ studio □ 1-2 bedrooms □ 3 or more bedrooms, □ Other ____________
- House: □ attached/twin duplex □ attached, 3 or more units, □ single □ Other ____________

Total Cats _____ Total Dogs _____ Other Pets ____________

Other People ____________

Please help us understand what your cat does around the house by placing a check (✓) in the box next to each behavior that best describes how commonly your cat does each of the behaviors described below

<table>
<thead>
<tr>
<th>Does your cat:</th>
<th>All of the time</th>
<th>Most of the time</th>
<th>A good Bit of the Time</th>
<th>Some of the time</th>
<th>A little bit of the time</th>
<th>None of the Time</th>
<th>Does Not apply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leave household articles (furniture, drapes, clothing, plants, etc) alone</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Eat small amounts calmly at intervals throughout the day</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>Drink small amounts calmly at intervals throughout the day</td>
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<td>Use the litterbox</td>
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<td>Get along with people in the home</td>
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<tr>
<td>Get along with other pets in the home</td>
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<tr>
<td>Remain calm when left alone</td>
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<td>Stay relaxed during normal, everyday handling (grooming, petting)</td>
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<td>Calm down quickly if startled or excited</td>
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<td>React calmly to everyday events (telephone or doorbell ringing)</td>
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<td>Play well with people</td>
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<tr>
<td>Play well with other family cats</td>
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<td>Show affection without acting clingy or annoying</td>
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<td>Tolerate confinement in a carrier (including travel)</td>
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<tr>
<td>Groom entire body calmly</td>
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<td>Use scratching posts</td>
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<tr>
<td>Play with toys</td>
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</table>

Comments; anything else your cat regularly does or does not do that you think might be helpful for us to know about?
Health history
The cat’s condition today is _____________________________________________________________
Previous illnesses or surgeries _________________________________________________________
Current medications _________________________________________________________________

**Directions:** For items below, please use the following choices to describe how many times you have seen your pet experience the symptom, adding comments/explanation as appropriate.

**Score:**
0 = I have NEVER seen it
1 = I have seen it at least ONCE
2 = I see it at least ONCE per YEAR
3 = I see it at least ONCE per MONTH
4 = I see it at least ONCE per WEEK
5 = I see it DAILY

<table>
<thead>
<tr>
<th>Score</th>
<th>How often does your cat:</th>
<th>Comments/explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cough</td>
<td></td>
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<tr>
<td></td>
<td>Sneeze</td>
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<tr>
<td></td>
<td>Have difficulty breathing</td>
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<tr>
<td></td>
<td>Stop eating</td>
<td></td>
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<tr>
<td></td>
<td>Vomit □ food □ hair □ bile □ other</td>
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</tr>
<tr>
<td></td>
<td>Have hairballs</td>
<td></td>
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<tr>
<td></td>
<td>Have diarrhea</td>
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<td></td>
<td>Have constipation</td>
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<td></td>
<td>Defecate outside the litter box</td>
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<tr>
<td></td>
<td>Strain to urinate</td>
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<tr>
<td></td>
<td>Have frequent attempts to urinate</td>
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<tr>
<td></td>
<td>Urinate outside the litter box</td>
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<tr>
<td></td>
<td>Have blood in the urine</td>
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<td></td>
<td>Spray urine</td>
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<tr>
<td></td>
<td>Groom more than cats usually do</td>
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<tr>
<td></td>
<td>Shed more than cats usually do</td>
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<tr>
<td></td>
<td>Scratch him/herself more than cats usually do</td>
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<tr>
<td></td>
<td>Have discharge from eyes</td>
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<tr>
<td></td>
<td>Seem fearful</td>
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<tr>
<td></td>
<td>Seem to need a great deal of contact or attention</td>
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<tr>
<td></td>
<td>Destroy things when left alone</td>
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</tr>
</tbody>
</table>

Please check any of the following diseases your cat has been diagnosed with:

- □ Periodontal (dental) disease
- □ Asthma
- □ Inflammatory bowel disease
- □ Skin disease
- □ Allergies
- □ Diabetes mellitus
- □ Cardiomyopathy (heart problems)
- □ Obesity
- □ Other

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Household resource checklist

The following questions ask about your cat’s resources so we can learn more about the environment your cat(s) live in. Please select DK if you don’t know, NA if it does not apply, or Yes or No after each question. If you have more than one cat, please answer for all cats. Resources (food, water, litter and resting areas) for each cat are assumed to be out of (cat) sight of each other, such as around a corner or in another room. If they are in sight of each other, please answer No.

### Space

<table>
<thead>
<tr>
<th>Question</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Each cat has its own resting area in a convenient location that provides some privacy</td>
<td></td>
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<tr>
<td>2. Resting areas are located such that another animal cannot sneak up on the cat while it rests</td>
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<tr>
<td>3. Perches are provided so each cat can look down on its surroundings</td>
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<tr>
<td>4. Each cat can move about freely, explore, climb, stretch, and play if it chooses to</td>
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<tr>
<td>5. Each cat has the opportunity to move to a warmer or cooler area if it chooses</td>
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<tr>
<td>6. A radio or TV is left playing when the cat is home alone</td>
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</table>

### Food and water

<table>
<thead>
<tr>
<th>Question</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>8. Each cat has its own food bowl</td>
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<tr>
<td>9. Each cat has its own water bowl</td>
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<tr>
<td>10. Bowls are located in a convenient location to provide privacy while the cat eats or drinks</td>
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<tr>
<td>11. Bowls are located such that other animals cannot sneak up on the cat while it eats or drinks</td>
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<tr>
<td>12. Bowls are washed regularly (at least weekly) with a mild detergent</td>
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<tr>
<td>13. Bowls are located away from machinery</td>
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</table>

### Litter boxes

<table>
<thead>
<tr>
<th>Question</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
<th>No</th>
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<tbody>
<tr>
<td>14. Each cat has its own box (one box per cat, plus one)</td>
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<tr>
<td>15. Boxes are located in convenient, well-ventilated locations that still give each cat some privacy while using it</td>
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<tr>
<td>16. Boxes are located on more than one level in multi-level houses</td>
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<tr>
<td>17. Boxes are located such that another animal cannot sneak up on the cat during use</td>
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<tr>
<td>18. Boxes are located away from machinery that could come on unexpectedly during use</td>
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<td>19. The litter is scooped daily</td>
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<tr>
<td>20. The litter is completely replaced weekly</td>
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<tr>
<td>21. Boxes are washed regularly (at least monthly) with a mild detergent (like dishwashing liquid), rather than strongly scented cleaners</td>
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### Litter boxes (continued)

<table>
<thead>
<tr>
<th>Question</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
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<tbody>
<tr>
<td>22. Unscented clumping litter is used</td>
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<tr>
<td>23. A different brand or type of litter is purchased infrequently (less than monthly)</td>
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<tr>
<td>24. If a different type of litter is provided, it is put in a separate box so the cat can choose to use it (or not) if it wants to</td>
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### Social contact

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<tr>
<th>Question</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
<th>No</th>
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<tbody>
<tr>
<td>25. Each cat has the opportunity to play with other animals or the owner if it chooses to on a daily basis</td>
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<tr>
<td>26. Each cat has the option to disengage from other animals or people in the household at all times</td>
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<tr>
<td>27. Do any cats interact with outdoor cats through windows?</td>
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### Body care and activity

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<tr>
<th>Question</th>
<th>DK</th>
<th>NA</th>
<th>Yes</th>
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<tbody>
<tr>
<td>28. Horizontal scratching posts are provided</td>
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<tr>
<td>29. Vertical scratching posts are provided</td>
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<tr>
<td>30. Chew items (eg, cat-safe grasses) are provided</td>
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<tr>
<td>31. Toys to chase that mimic quickly moving prey are provided</td>
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<tr>
<td>32. Toys that can be picked up, carried, and tossed in the air are provided</td>
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<tr>
<td>33. Toys are rotated on a regular basis (at least weekly) to provide novelty</td>
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If you have additional comments on any of the questions, please write them below, including the question #.

By submitting this form, you agree that anonymous information from it may be used for cat health-related research.
Cytauxzoon felis is a protozoan blood parasite transmitted via the bite of an infected tick. Bobcats (Lynx rufus) are the reservoir host; infected bobcats are believed to develop short-lived illness followed by clinical recovery and a persistent carrier state. When a tick feeds on an infected bobcat, the tick acquires the pathogen. If that tick subsequently feeds on a bobcat, the sylvatic cycle continues. However, if the tick feeds on a domestic cat instead, infection typically leads to severe illness (ie, cytauxzoonosis) and often death. All Felidae are susceptible to infection; infection has not been reported in any non-felid animal.

Bobcats (Lynx rufus) are traditionally considered the main reservoir host of C. felis although other wild felids and domestic cats are also infected and may contribute to infection prevalence in ticks.

Amblyomma americanum, the lone star tick, is considered the predominant vector of C. felis based on experimental infection studies and epidemiologic data. Historical studies indicate Dermacentor variabilis, the American dog tick, can also transmit C. felis to cats. Observed differences in vectorial capacity of different tick species may be due to pathogen strain differences, tick strain differences, or immune status of host.

During feeding, the tick inoculates sporozoites that enter host mononuclear cells and multiply. Infected mononuclear cells distend with organisms known as schizonts and these cells can act as thrombi, resulting in vascular occlusion with resultant multi-organ failure. It is during the schizogenous stage of parasite replication, usually about 2 weeks after the bite of an infected tick, that infected cats develop clinical illness.

The multinucleated schizonts divide into merozoites. Within days, merozoites rupture from the mononuclear cells and are taken up by red blood cells (RBCs) where they appear as piroplasms. Widespread dissemination of schizonts results in parasitic thrombosis, circulatory impairment, tissue infection and a severe systemic inflammatory response, which can lead to multi-organ dysfunction and failure and death within 3 weeks of infection. Many cats die within 24 h of presentation to a veterinary clinic for treatment. When schizonts rupture in the circulation, large numbers of merozoites are released, infecting red blood cells and additional mononuclear cells. This is late-stage disease, with erythroparasitaemia (piroplasm structures within red blood cells) which can be readily observed in blood smears, and may lead to haemolytic anaemia and erythro phagocytosis. In cats that survive initial infection, piroplasms persist for months, years, or even for life, although schizonts can no longer be found. While clinically relevant hemolysis may occur in the acute phase of infection, chronic erythroparasitaemia is relatively benign. The parasite life cycle is perpetuated when a tick takes a blood meal containing merozoite infected RBCs. It has now been documented that recovered domestic cats are competent to transmit the pathogen to feeding ticks.

C felis cannot be transmitted through physical contact between infected cats. Although experimental inoculation of naive cats with schizonts results in cytauxzoonosis and death, inoculation of RBCs containing piroplasms does not lead to schizogony or illness. Vertical transmission, common in other apicomplexan infections, has not been documented.

Since the first reported case of cytauxzoonosis was recognized in Missouri in 1976, there has been a progressive expansion in the geographic range in which disease has been identified. To date, cytauxzoonosis has been confirmed in domestic cats from 17 US states, and the pathogen has been documented in bobcats from Pennsylvania and North Dakota, states where ill domestic cats have not yet been reported. The increase in geographic range is likely due to expansion in the range of the vector Amblyomma americanum (lone star tick) population, hypothesized to be linked to an expanding white-tailed deer population. The prevalence of C felis in bobcats is higher in geographic regions with established A. americanum populations. Veterinarians in areas where lone star ticks are found but cytauxzoonosis has not been reported can be vigilant for the disease.

In 2015, the first report surfaced of C felis infection in Illinois. Veterinary practitioners need to be aware that cytauxzoonosis occurs in southern Illinois so that they can advise owners about disease prevention, including keeping cats indoors and using tick-control products that are approved in cats. In most cats that are affected by cytauxzoonosis, initial disease signs begin within a few weeks following infection via tick bite by and include fever, anorexia and lethargy. Tachypnea and tachycardia are commonly observed. Within days, clinical signs can progress to severe weakness, icterus, respiratory distress and/or neurologic dullness. Diagnosis can be made by identification of piroplasms within erythrocytes during microscopic examination of a peripheral blood smear. PCR assays have been developed to confirm the presence of C felis and other Cytauxzoon species, but so far they are not useful as a quick diagnostic tool in practice. It is recommended that samples from suspected cats are submitted to appropriate laboratories to further confirm the infection. Low levels of parasitaemia can only be detected by PCR assay. In one clinical trial, parasitaemia was determined by qPCR and at significantly lower levels in surviving cats versus non-surviving cats, so qPCR results might be of prognostic value.
An open-label randomized prospective clinical trial demonstrated better survival rates (60% vs 26%) with the combination of atovaquone (15 mg/kg PO q8h) and azithromycin (10 mg/kg PO q 24h) compared with imidocarb (3.5 mg/kg IM once) in 80 cats with acute disease. Mortality was high (41/80 cats). Most cats died during the first 3 days after presentation, only three cats dying after the third day of treatment. Supportive treatment was the same in all cats, comprising fluid therapy and heparin. This study suggests that this antiparasitic combination plus supportive treatment is the current approach of choice. In some cat nasoendoscopy or esophagostomy tube may be needed to administer drugs and enteral feeding.

There is currently no vaccine against C felis, although preliminary studies are being conducted. Prevention is based on living indoors or use of effective tick treatment in cats with outdoor access. Efficacy of an acaricide collar (imidacloprid 10% plus flumethrin 4.5%) for prevention of C felis transmission has been proven in a controlled prospective clinical trial. Two groups of cats (with and without a collar) were exposed to ticks (A americanum) infected with C felis. No cats with a collar, vs 90% of the cats with no collar, were infected.

### Heartworm disease

In endemic areas, indoor and outdoor cats are at similar risk for infection. The infective L3 stage larva enters the cat through a bite wound, moults to L4 and L5 stages and migrates to the pulmonary arteries as immature adults 70-90 days after infection. Once infected, the cat’s natural resistance results in a short period of microfilaremia. The clinical worm burden is also lower in cats, (from 1 to 9 worms) than in dogs. The average time for infective larvae to develop into circulating microfilariae in experimental feline infections is about 8 months. Thus, microfilaremia is uncommon (<20%), inconsistent, and transient in cats, and very low numbers are usually produced. The comparable development period in dogs is 5-6.5 months.

There is high mortality of L5 as they arrive in the distal pulmonary arteries in the cat. High mortality of immature adult heartworms is associated with intense pulmonary bronchial and parenchymal response called Heartworm-Associated Respiratory Disease (HARD). Residual pulmonary pathology related to HARD persists even after immature heartworms die. Thus many heartworm infections may be misdiagnosed as feline asthma.

Heartworm disease in cats is characterized by pulmonary eosinophilic bronchial and interstitial reaction associated with immature adults (3-6 months after infection) chronic lung changes associated with mature adult heartworms (6 months – 4 years) and acute respiratory distress associated with the death of worms at any age.

Lesions associated with HARD are initiated by immature larvae as early as 70 to 90 days after infection. The lesions in HARD are characterized by peribronchial fibrosis, interstitial myofibroblasts and fibrosis of alveolar struts. Muscular hypertrophy, villous endarteritis and adventitial cellular infiltrates are common findings in all pulmonary arteries, although caudal arteries are most commonly seen radiographically. Infiltrative interstitial lung disease, reduced clearance of mucous and inflammatory debris are the hallmark of this lung disease as opposed to increased bronchial wall reactivity as proposed in asthma models.

Wolbachia are gram-negative bacteria belonging to the order Rickettsia that reside within the body of D. immitis and appear within 2 months of exposure to infective larvae. The release of bacteria following worm death has shown to cause upregulation of proinflammatory cytokines, neutrophil recruitment and an increase in specific immunoglobulins, although the role of this intracellular bacteria alone in the pathogenesis of feline heartworm is unclear.

Clinical signs often include coughing or vomiting most commonly associated with immature heartworms arriving in the lungs or death of adult heartworms. The initial arrival of L5 in the distal pulmonary arteries induces diffuse pulmonary infiltrates and often eosinophilic pneumonia. Clinical signs associated with acute phase subside as the worms mature but lesions remain even in cats who clear infection.

Most infected cats will be asymptomatic during most of infection. Adult heartworm death, even as little as one, may induce acute, potentially fatal disease with acute circulatory collapse or severe respiratory distress. Anorexia and lethargy may be the only presenting complaints. Coughing or intermittent vomiting may occur. The vomiting appears unrelated to eating. Inflammatory mediators and stimulation of the chemoreceptor trigger zone are postulated as the cause.

Positive antibody result indicates infection with L3 which has moulted to L4 and lived at least 2-3 months. Adult heartworms may or may not develop from this infection. ELISA antigen testing is specific for glycoproteins associated mainly with reproductive tract of fully mature female worms, making false-negative results common. Cats presenting with HARD from immature adults will be antigen negative as will those with low worm numbers. Eosinophilic cytology from BAL will be most intense 3-6 months after migration and is intermittent. Thoracic radiology is helpful but not specific. Aelurostrongylus and roundworm infection are the most common pulmonary infections to mimic heartworm radiologic signs.

Year round heartworm prevention prevents both patent infection and HARD. When cats were infected with L3 heartworms experimentally and treated with selamectin monthly commencing 28 days later did not develop adult worms but did seroconvert to antibody-positive status. Another study demonstrated that cats pretreated with selamectin 32 and 2 days before L3 infection did not develop HARD.
In a study of mosquitoes in an endemic heartworm region, 30% of mosquitoes had evidence of blood meals from both dogs and cats lending credence to the transmission likelihood between the two species.

Anaplasma phagocytophilum is a rickettsial organism that causes granulocytic anaplasmosis in cats, dogs, horses, ruminants and humans. The organism was first described in 1932 in Scottish sheep, and then reported to affect horses, dogs, cats, cattle, camelids and humans. The worldwide distribution of A phagocytophilum follows the geographic distribution of the primary vector, Ixodes species. In North America, the organism is transmitted by Ixodes scapularis in the Northeast and Midwest, and by Ixodes pacificus in the West. Infections are highest in the late spring and autumn when both nymph and adult ticks are most mobile. Transmission to mammals occurs within 24-48 h of tick attachment.

Once transmitted, A phagocytophilum infects circulating neutrophils forming intracellular inclusions (morulae), which can be observed via light microscopy on a Romanowsky or Wright-Giemsa-stained blood smear. In North America, morulae have been identified in the neutrophils of naturally exposed dogs, horses, humans and experimentally infected cats. In Europe, morulae of A phagocytophilum have been described in ruminants, cats, horses and dogs.

In a recent retrospective study, sixteen cases of feline anaplasmosis were identified. The objectives of this study were to collect retrospectively and describe the clinical and historical findings in cats that were positive by PCR for A phagocytophilum DNA in their blood, and to describe treatment and response. They were also able to characterize intracellular morulae identified in neutrophils on microscopic examination of peripheral blood smears.

Of the 16 cats, all were lethargic and 15 had a fever. 14 were anorexic. Other signs were variable and may not have been specific for Anaplasma infection. All had access to the outdoors and lived in Ixodes endemic regions. Treatment information was available in 15 cats. Another antibiotic had been prescribed prior to PCR amplification of A phagocytophilum in 8 cats. Once PCR assay results confirming the presence of A phagocytophilum DNA were available, all 15 cats were administered doxycycline orally. Clinical abnormalities resolved after initiating doxycycline therapy in the 14 cats with a known response to treatment. The duration of doxycycline administration varied from 21–45 days, with most cats treated for 21 days. All 16 of the cats identified in this study were positive for A phagocytophilum DNA in blood, were from an endemic area, had potential exposure to Ixodes species, and the majority had clinical and laboratory evidence of anaplasmosis, as well as apparent responses to doxycycline.

Morulae were identified in neutrophils in 3/11 (27%) cases in this report, with inclusions in approximately 4–20% of neutrophils. Identification of these inclusions prompted submission of blood for the commercial PCR. test, confirming the diagnosis of A phagocytophilum infection. Morulae appear as basophilic intracytoplasmic inclusions that must be carefully differentiated from Döhle bodies.

As a retrospective study it is a limitation that an explanation for the duration of treatment is unknown. The majority of cats were treated for 21 days; those that were treated for 45 days were all seen at the same practice. Previous reports in cats have recommended treatment with 5 mg/kg doxycycline PO q12h for 28 days. The ideal duration of treatment with doxycycline in cats is unknown and warrants additional study.

A phagocytophilum infection should be included on a differential diagnoses list for any cat that lives in an Ixodes species endemic area with potential tick exposure and presents with acute or intermittent, vague clinical signs of lethargy, fever and anorexia. A phagocytophilum infection can be identified by documenting DNA in peripheral blood by PCR prior to antibiotic administration or by identifying morulae within neutrophils on a direct blood smear examination. Exposure can be documented by demonstrating the presence of antibodies with ELISA and indirect fluorescent antibody serology; a four-fold change in convalescent titers after 14 days indicates an active infection. Year round tick prevention should be included to preventive care for cats living in Ixodes endemic areas.

References
Association of Wolbachia with heartworm disease in cats and dogs Veterinary Parasitology (2010)
Anaplasma phagocytophilum infection of domestic cats: 16 cases from the northeastern USA Journal of Feline Medicine and Surgery (2016)
In both people and companion animals, cachexia and sarcopenia are 2 important syndromes that occur in a variety of chronic diseases and aging, respectively. Although cachexia has been recognized in people for over 2,000 years, only recently has it become acknowledged as a common and detrimental finding that is associated with increased morbidity and mortality, and with this observation has come rapidly expanding interest and research. Both of these syndromes are becoming increasingly important in human and veterinary medicine because of their high prevalence and adverse clinical effects, and a better understanding of the mechanisms underlying these syndromes is critical for optimal patient care, whether human or veterinary.

Cachexia is defined as loss of weight and muscle mass secondary to chronic inflammation or disease. Sarcopenia, “poverty of flesh”, is an age-related loss of lean body mass. Sarcopenia is not caused by disease, is a gradual process and progresses with age. Loss of muscle can occur without fat loss or a decrease in Body Condition Score (BCS). Individual cats, particularly those with long coats or a history of obesity may appear to have a high BCS and yet be under muscled.

One of the keys to the management of cachexia and sarcopenia in dogs and cats is recognizing it in its earliest stages. To achieve this, BCS and Muscle Condition Score (MCS) must be consistently assessed. The goal for BCS in a healthy cat is 4–5 on a 9-point BCS scale. However, in certain diseases (eg, CHF, CKD), a slightly higher BCS may be desirable (ie, a BCS of 6–7/9), although further research is required to make specific recommendations. Even in animals with these diseases, obesity (BCS > 7/9) should be avoided.

The MCS differs from the BCS in that it specifically evaluates muscle mass. Evaluation of muscle mass includes visual examination and palpation of the head, scapulae, epaxial muscles over the thoracic and lumbar vertebrae, and pelvic bones.

In people, the loss of LBM has direct and deleterious effects on strength, immune function, wound healing, and survival. In fact, cachexia is an independent predictor of survival in people. The specific deleterious effects of muscle loss have not been as well studied in dogs and cats although there are studies associating thin body condition with decreased survival.

The weight loss that occurs in cachexia is unlike that seen in a healthy animal that loses weight. In a healthy animal that is receiving insufficient calories to meet requirements, metabolic adaptations allow fat to be used as the primary fuel source, thus preserving LBM. Conversely, acute and chronic diseases alter concentrations of a variety of mediators (eg, inflammatory cytokines, catecholamines, cortisol, insulin, glucagon), which then decrease the ability to make metabolic adaptations required to switch to fat utilization, and amino acids continue to be used as a primary source of energy. Therefore, muscle and LBM quickly are catabolized.

Numerous other factors can contribute to muscle and weight loss. Maintenance energy requirements vary with age, genetics, health status and gender (intact or altered). In presence of some disease states, maintenance energy requirements increase significantly. Decreased nutrient absorption is another possible mechanism for muscle loss in cachexia and sarcopenia. Studies in cats have shown decreased digestive ability. One investigator showed a reduced ability to digest protein in 20% of geriatric cats with about 33% having a significant reduction in ability to digest dietary fat. Micronutrient absorption, potassium, phosphorus, sodium, choline, B vitamins and Vitamin E, is also decreased.

Cats derive most of their energy requirements from protein and are metabolically less able to handle decreased amounts of protein and increased amounts of carbohydrates to maintain their energy requirements. Omnivores adapt to lower dietary protein by down regulation of their protein metabolism (protein sparing) but cats have been proven to be unable to make this physiologic adaptation. This preferential use of protein for energy can have clinical effects when cats are ill or anorectic as protein malnutrition can occur.

An important problem in cardiac and other forms of cachexia is a decreased calorie intake. The anorexia may be secondary to fatigue, dyspnea, or may be because of medication toxicity or alterations in appetite that often accompany CHF, cancer, and CKD in cats. Absolute food intake may decrease in animals with these diseases, but there also may be altered food preferences, cyclical appetite, and other issues that negatively affect overall food intake. Anorexia, for example, is present in 34–84% of dogs and cats with heart disease.

Increased energy requirements, alterations in nutrient absorption, and decreased energy intake all likely play important roles in the pathogenesis of cachexia by causing a net calorie deficit. However, a healthy animal that has a calorie deficit, either as a consequence of decreased food intake or increased energy requirements, would primarily lose fat. Therefore, these factors are not sufficient to explain the muscle and LBM loss and relative sparing of fat that are the hallmarks of cachexia and sarcopenia. This discrepancy suggests that metabolic alterations also are present.

Because of the important implications of cachexia and sarcopenia on morbidity and mortality in people, there is now extensive research into the prevention, diagnosis, and treatment of these syndromes. There are exciting opportunities for new and effective...
targets to decrease energy requirements, enhance energy intake, improve nutrient absorption, and modify metabolic alterations to prevent and even reverse the effects of both cachexia and sarcopenia.

A 2008 study on longevity in aging cats studied in a controlled environment for 5 years showed that all cats lost weight over time. However, cats supplemented with dietary antioxidants, prebiotic chicory root and a blend of Omega 3 and 6 fatty acids had a beneficial effect over a commercially fed diet alone or one supplemented only with antioxidants (Vitamin E and beta carotene). Cats in the fully supplemented group lost less weight, lived longer, had better LBM scores, improved fecal flora and fewer diseases. In many cases, practical methods to help owners manage their animal’s appetite are critical to success. This is particularly important because anorexia is one of the most common contributing causes to an owner’s decision to euthanize his or her pet.

Any issues that potentially can affect food intake should be addressed, whether physical or environmental. Dental disease, for example, can substantially impair food intake in an otherwise healthy or sick animal. Pain (eg, back or joint) can decrease an animal’s mobility and make it more difficult to secure adequate food intake. Environmental issues also can negatively impact food intake. Multipet households may impede the ability of an individual animal to gain access to food (eg, a more frail or timid animal may be crowded out from the food bowl). Stress often can increase for animals after diagnosis of any illness because of lifestyle changes (eg, medication administration, new foods), as well as increased stress on the part of the owner, which may be detected by the animal.

Once environmental issues are ruled out as a cause of weight loss, a nutritional screening is crucial. Older cats may need 5-6 g of protein/kg to prevent protein catabolism. Reduced digestive ability indicate that a high energy, highly digestible diet may be needed. Some kitten formulas may be more appropriate. Folate and cobalamin supplementation may be useful. Commercial cat foods vary quite widely in caloric density. Specific formulas should be investigated for adequacy.

Cachexia should be anticipated in animals with chronic diseases such as CHF, CKD, cancer, and others. Consistently evaluating MCS in all patients will help identify muscle loss at an early, mild stage in aging or ill animals, rather than waiting until muscle loss is moderate or severe, when it may be more difficult to successfully manage. Similarly, as animals age, muscle loss is likely to occur, even in healthy individuals. Therefore, muscle mass should be thoroughly evaluated in geriatric cats and dogs.

References
V. Paul Doria-Rose, DVM, and Janet M. Scarlett, DVM, PhD. Mortality rates and causes of death among emaciated cats. JAVMA Feb 2000, Vol. 216, No. 3, Pages 347-351
http://www.wsava.org/nutrition-toolkit
One thing that I can't stress enough is to entertain and inform more than you sell. Even though every business wants to make money, that shouldn't be the focus of the vast majority of your social media posts. Keep the 80/20 rule in mind: 80 percent of the content you post or share should be entertaining or informative, while no more than 20 percent of your social media communication should directly relate to the services you provide. Even when you are selling you should still take the opportunity to educate and show the benefits of the product/service.

For example, rather than simply posting
"Saturday is cheap spay & neuter day! Call us for an appointment."
Say something like:
"Did you know all of the health benefits of spaying or neutering a cat? Spaying females prior to their first heat cycle nearly eliminates the risk of breast cancer and totally prevents uterine infections and uterine cancer. While neutering males prevents testicular cancer and enlargement of the prostate gland and greatly reduces their risk for perianal tumors.

This Saturday only we are offering a free 20% discount on all spay/neuters. Call us now to make an appointment: 1 (123) 555-6789."

This educates the client and shows how he/she can help their cat. The second part gives him/her a sense of urgency (Oh, no, I can't miss this deal) with a value-added statement (a free discount - everyone loves free, especially if it's a discount) and you make it convenient for them by giving your phone number (remove road blocks, not giving your phone number means they have to look for it and therefore less likely to follow through since it's another task).

Have you ever come across a company's social media presence that seemed too personal, or too robotic? We've all seen it once or twice - or a few dozen times. All too often, these examples stand out much more than brands that are doing everything right. Here are some of the most common mistakes brands make, as well as tactics everyone should adopt:

**Do's**

**Know your customers**
Your social media pages should be tailored to your consumer base, not based on your own personal interests. To be honest, you consumers don't care about YOU. They care about what you can do for THEM. Post stories that they can relate to. If you work at a bakery, post recipes and tips and tricks of the trade. Follow the 80/20 rule. 80% of your content (or more) should be relevant to THEM, while 20% or less can be specifically about your business, products or services.

**Be active but don't over-do it**
Be active on your social media, but don't post so often that you overwhelm or annoy people. This could lead to two problems: 1) Too much information can cause your followers to stop following your posts and 2) Your posts can get lost within their newsfeeds and they won't see the posts that could be really important to them.

**Time does matter**
Pay attention to analytics. Find out when your followers are most active on social media and post your information during those time frames.

**One voice**
Social media is great for sharing, but make sure your message is the same across all channels: website, public, social media, etc.

**Share**
We've been taught to share since we were young kids and some things never change. This is one of those things. Share information!

**Checklists**
Checklists are great for many aspects in life. Social media works well with checklists. Sure, it sounds simple. Just get on Twitter or Facebook, type a post and hit share, right? Unfortunately, it's not quite that simple. Make a checklist to ensure that your message will be understood, check for any grammar issues, make sure the information your sharing can be shared, check the time you send it to ensure your readers will see it. Make sure all your links are valid. These are just a few examples of things to pay attention to your tweets and posts. Attention to this detail could save you time and trouble later.

**Be original**
Take a chance and be creative with your posts. Show your personality. Help people see what makes you different from the competition. Especially when your main goal is to bring in customers, stand out and make people remember you!
Great customer service can make or break a company. Happy customers are more likely to come back and establish a loyalty to your brand. Not only will these customers be loyal to you, they will also be your best brand ambassadors by word-of-mouth advertising, which is the best advocacy you can ask for!

Having a personality
Dull and boring posts lead to dull and boring results. You have to be excited about your company, your product and the services you can provide your customers. If you aren't excited and don't love what you stand for, neither will your clients.

Understanding which social media platform(s) are best for their business
Just because everyone seems to be on doesn't mean you need to be. Social media is not one size fits all. If you want to improve your SEO, then Google+ or YouTube might be your best choice. If you want to drive traffic to your site, or improve your customer engagement then Facebook, Twitter and Pinterest might be a better fit, according to Social Media Today.

The don't do's

Liking your own posts
Of course, you like your post, you posted it! Don't "like" or "favorite" your own material. Encourage your employees, however, to share and like the material that is posted. Your employees can be the brand's greatest ambassadors.

Neglect
Use your social media! Just because you have a Facebook page or Twitter account doesn't mean you're doing enough. You have to use the accounts to make them work.

Share too much
Be careful of the things you share, don't share too much information. This one goes back to having a checklist. If you are premiering a product and post hints or pictures too early it could destroy the whole product launch that others worked so hard to build.

Connect with everyone
Just because someone follows you, doesn't mean you have to follow them back. Think of it at choosing who you surround yourself in real-life. Before you follow back think of how it will reflect on you, is this someone that would represent you and your business in a good way if consumers saw that you were associated with them?

Forget to network
Just as you would in real-life, always work to make connections and grow your brand by networking and building quality relationships on social media. If your impression is good enough you might be able to work in a few word-of-mouth shoutouts.

Privacy settings
Don't forget about privacy settings. You use them on your personal profiles, do the same with your business. Remember once you share something on the internet, it doesn't go away. Sure you can delete the post, but you don't know who may have seen the post and saved the information before you deleted it. Taking care of the privacy settings also includes protecting your passwords. Only give passwords to a select few and choose a password that is extremely secure. You don't want your social accounts to fall into the wrong hands!

Don't be spammy
Nobody likes spam, whether it be email or social networking. Just don't do it. If you are constantly posting the same information over and over or inundating your followers, you could become an annoyance and could lose some of the followers you've worked so hard to attract.

Deleting negative comments
Acknowledging the problem can not only make an upset customer happy, but it can also prevent the company from a PR issue later. Acknowledging negative comments shows that your company is proactive in resolving issues and that you aim to serve your customers.

Not responding at all
It is vital to engage with your customers, with no engagement they may feel as if you don't care about their question, comment or concern. It all comes back to customer service. Don't ignore relevant comments. Use them as an opportunity to interact with your followers and show them how you address positive and negative situations.

Too much automation
You lose the personal touch with customers if it sounds like a computer is speaking to them. "Humanize" your brand to make the engagement experience for the customer more personable.

What is search engine optimization and why should I care?
So how do you know if you're committing a cardinal sin of SEO and optimization? How do you know if your website or content is repellent to Google’s algorithm?

We’ve got three big things about Google’s algorithm (and how it assesses your content and site) that you might not know.
Pogo-sticking is the bounce rate’s evil cousin
Also known in the SEO world as “Return-to-SERP,” “pogo-sticking” is one of the biggest indicators of searcher dissatisfaction.

So what is pogo-sticking, you ask?
According to Neil Patel it’s the act of pogoing back and forth between an SERP and the content it presents:

“Have you ever searched for a particular topic in Google, clicking on the first result in the top 10 only to be completely dissatisfied with what you found there? You probably quickly clicked the “back arrow” and went back to either search for another term or to click on a different web page.

That’s pogosticking — where the user is jumping back and forth between the SERPs and content pages, because their needs haven’t been met.”

Pogo-sticking is slightly different from the bounce rate in that it signifies a clear dissatisfaction with a webpage’s content; the user isn’t spending time on the page and then clicking away. They are actively choosing another website from the SERP to get the information they’re looking for.

A high rate of pogo-sticking is bad news bears when it comes to ranking in Google’s algorithm, where user satisfaction is the king of the hill. If users are consistently finding and clicking your website, but immediately returning to the SERP and choosing another result, you’re either not presenting information relevant to their search term or your page’s design and layout is bad for user experience. Or both.

You need to address the issue
Analyze navigation paths through your analytics tools, and tweak your badly-performing pages so that they offer clearer, more concise information, above the fold and in an accessible design.

Guest posting: you must proceed with caution
Since that time, brands have been forced to address (and often change) their own guest blogging practices.

In 2016, here’s what you should know:

Don’t accept guest bloggers who request backlinks to toxic sites
If someone approaches your company with a strong piece of content, in search of a guest blogging opportunity, it’s important to ask what they expect in return for their content. Most of the time, a simple backlink is all they want. However, if that backlink is to a toxic, low-quality, irrelevant site, you’re doing yourself no favors (and are actually hurting your credibility) by featuring them.

Check DA (Domain Authority) on all the websites you’re pursuing for backlinks. Do the same for the websites of guest bloggers pursuing you for backlinks
If a website has a domain authority of 20 or less, they offer no value to your website and probably have serious on-page SEO and user satisfaction issues.

Don’t accept guest posts that feature content that is irrelevant to your niche
Google crawls and indexes your entire website and its content. Content and keywords that are unrelated to your niche will weaken your site’s authority within queries related to that niche. from Moz https://moz.com/researchtools/ose/ how to check a website’s domain authority before including them in your link building profile.

Are you over-optimizing? Stop...right now

• “But optimizing is good!” you might say.
• And yes, it is good.
• But, much like pizza, exercise or drunken online shopping, too much of it can be bad.
• Over-optimization can be as simple as bad linking techniques or as annoying as keyword stuffing. No matter what form it takes, it’s best to avoid it altogether.

Some big examples of over-optimizing?
According to Kissmetrics’ blog some of the biggest culprits are keyword stuffing, keyword-rich anchors for internal links, and using multiple H1s on a page.

Remember: Google values natural, informative writing. And now that they have their most sophisticated algorithm ever, simply loading a page with keywords you want to rank for doesn’t cut it. You have to actually offer value in your content. If you don’t, Google will know. And they won’t let your pages show up in SERPs.

Consider these fascinating facts about SEO next time you write a blog post, optimize your web pages or pursue another website for backlinks. You might just escape some of Google’s wrath.

How to Avoid Over-Optimizing Your Website
Too much of a good thing is a bad thing. This is true in life, and in SEO. SEO is awesome, but too much SEO can cause over-optimization.

Search engine over-optimization is the practice of creating too many SEO improvements, to the point that the improvements begin to ruin the website’s ability to rank. You’re doing all the typical SEO good stuff, but then you jack it up too far. Things go downhill.
First, here’s everything you need to know about the history of over-optimization.

1. **Over-optimization used to work.**

Back in the Wild West days of SEO, over-optimization was the way that sites gained rank. Stuffing a site with tons of keywords, or sending thousands of spammy links to a site was the quick-and-easy way to boost a site in the SERPs.

2. **Over-optimization doesn’t work anymore.**

It definitely doesn’t work that way anymore. Today’s SEO’s know that keyword stuffing and linkspamming is SEO suicide. In April of 2012, Google leveled the over-optimization penalty, which completely deindexed sites that were still playing the linkstuffing and linkspamming game. A bunch of SEO techniques went black hat, and SEOs became wiser about the ways of Google.

3. **Over-optimization isn’t what it used to be.**

Today, many SEOs glibly assure themselves, “I’m not over-optimizing!”

Why do they say this?

Because they’re not keyword stuffing.

But today, over-optimization is more than just keyword stuffing. SEO is in a constant state of evolution. Google the search engine gets smarter, searches get smarter, SEOs get smarter, and then Google’s webspam team gets smart, too — and the playing field gets disrupted yet again.

Enough SEOs know and heed the over-optimization perils of 2012. But that was years ago. Today, we need a new guide to over-optimization that will keep us from doing too much of a good thing — and bringing our SEO to a tragic end.

7 Signs that you might be over-optimizing your site

1. **Keyword-rich anchors for internal links.**

Internal linking is good. Internal linking by using keyword-rich anchor text is bad.

   If I had to pick the single biggest oversight in over-optimization, this would be it. Here’s an example:
   
   - Check out our awesome new blue widget page for more information:

   - (Links to: example.com/awesome-new-blue-widget.htm)

   - Here is another example:

   - We sell top-rated cheap blue widgets.

   - (Links to: example.com/awesome-new-blue-widget.htm)

   Anchors that use the exact URL of the destination or anchors that use keywords are bad.

   I know what you’re thinking: “But those kinds of links are good!” Remember, though, we’re talking about over-optimization.

   Sure, the occasional anchor that matches the URL exactly could contribute to positive SEO. But if you start doing this too much, you’re setting yourself up for penalization.

   Using keyword-rich anchors too much begins to ruin your link profile. Your link rich profile is key. Don’t ruin it.

   The healthy alternative to using keyword-rich or overoptimized anchors is to spread your anchor text across a sentence fragment like I did in the above link. See how the anchor covers ten words? By creating a long anchor, I have diluted the keywords within the anchor (“link profile…SEO”) and created a softer and risk-free link to Quicksprout.com.

   The one notable exception to keyword-dense anchor is using anchors that are the same as the root domain URL.

2. **Non-relevant keywords.**

Don’t try to gain traffic for keywords that are not relevant to your site.

In the old days of SEO, some webmaster would place adult-themed keywords in their content in order to gain some of the search traffic for those queries.

Obviously, anyone with a penchant for adult-themed content is probably not going to convert on a page that is not adult-themed.

Don’t expect the conversion rate to go up.

This is an example of over-optimizing off-niche with non-relevant keywords. If you have a conversion optimization website, then don’t write articles on the healthiest ingredients to feed your pet parakeet.

As Google indexes your site, it will take into consideration all the keywords that you use across the entire domain, then rank your site for relevant queries. Too much content or keywords that are unrelated will detract from the overall strength of the site in the SERPs.

I recommend having a laser-like focus on your niche, and your niche only. Just stay within your niche.

3. **Pointing all internal or external links to top-level navigation pages.**

A healthy link profile has links pointing to deep internal pages as well as the home page. Most of the time, however, the majority of a site’s links point to the homepage or to top-level navigational pages.

For these sites, the ratio of home page links to internal links is pretty high. Here’s a typical example, which isn’t too bad:

Notice how the majority of the links (dark orange) point to homepage. It’s normal to have most links point to the start page, but it tends to weaken the link profile. The strongest link profile has links pointing to deep internal pages. A healthy ratio is 1:1, or 50% of the links pointing to deep internal links.
A strong content marketing campaign will draw deep internal links. People love your content, and they want to link to it. So, they create a link to your article (not to your homepage). Voila! You just gained another deep internal link.

The over-optimization problem occurs when webmasters themselves create a ton of links to their homepage or to main navigation pages like “Contact Us,” “About Us,” or “Our Services.” You want to create internal links, but you don’t need to point the links to these pages. Those pages get plenty of links as it is. Rather, strengthen your link profile by pointing to deep internal links.

4. Using multiple H1s on a page.

An H1 header is for a page’s main heading. Unfortunately, some webmasters think that awesome SEO means using a bunch of H1 text. This is simply not true. Using more than one H1 tag on a page is over-optimization.

Remember this rule of thumb: Just 1 H1.

It’s fine to use several H2s, 3s, and 4s, but there should only be one H1.

5. Linking to toxic sites.

The sites that you link to are almost as important as the sites that link to you.

Many webmasters are ignorant of the peril of linking to toxic, low-DA, or spammy sites. Link juice flows both ways.

If your site links to a toxic spam site, then your site may receive negative SEO repercussions.

If you’re trying to win reciprocal linkbacks by linking to other sites, be careful. The more outbound links you have to low-DA sites, the greater likelihood you have of placing your own site in this low-DA neighborhood.

Sites that languish in a sub-20 DA range are usually in that range for a reason. Maybe they have over-optimization issues, or an algo penalty, or some other barrier to growth. Don’t waste your time sending your outbound links to such sites. Instead, focus on associating with or linking to healthy sites in your niche — sites that have strong DAs and a strong reputation.


Ah, the footer! How many over-optimization sins have been committed in website footers.

Over-optimizing your website footer is a great way to shoot your SEO in the foot. The only type of footer optimization that you should do is not to do it.

Evidence shows that Google devalues footer links. Besides, due to their position at the bottom of a page’s content, they receive minimal crawler recognition. Their CTR is a bunch of zeros followed by some abysmally low number, and they simply don’t add any SEO value to a page. If you insist on adding a bunch of keywords to your footer, you are risking over-optimization.

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For pain in animals, pain is what WE (humans) say it is. There is tremendous individual variability among both patients and observers. There is not one “right” answer. Pain is complex and scientifically intriguing, clinically challenging, and easy to overlook (especially in cats). To improve the treatment of pain, no matter the patient, means making changes.

The fundamentals of acute pain management mean understanding that treating pain is good medicine, that providing peri-operative analgesia means before, during, and after the surgery, and that multi-modal analgesia is the strategy of choice. Compassionate care, of which CRI is a quintessential example, means giving each client your best effort every day.

Our moral imperative is to advocate on behalf of a being that cannot advocate for itself...

“Medicine should be practiced as a form of friendship.” - - Leon Bernard

CRIIs are EASY - - MUCH easier than you think it is if you listen to many of the “experts” who seem to do their best to make CRI seem mysterious and achievable only by huge practices or only those with specialists on staff. This is pure bunk!

You do NOT need a syringe pump to do CRI. You do NOT need special IV tubing to do CRI.

You DO need a precision IV infusion pump to do CRI correctly. You DO need to have IV fluids flowing with the CRI drug (in its own standard dilution bag with its own IV line and its own precision IV fluid pump) “piggy-backed” in order to overcome the hydrostatic pressure within the vein (22ga x 1 ½” needle into IV line port). This is especially true during the post-op period when flow rates are exceptionally low.

STANDARD DILUTIONS of CRI drugs are the KEY. Make life easy with Excel spread sheets with all the calculations made in 0.2 # (or 0.1 kg) increments of patient body weight.

Examples from real life
CRI fentanyl

Use this drug pre-op, intra-op, and post-op. The standard concentration is 0.05 mg/ml (50 μg/ml). The pre-op bolus is drawn from the standard concentration vial.

**Pre-op bolus dose**

Dogs & cats – 0.005mg/kg (5 μg/kg) IV

For intra-op and post-op CRI, create a STANDARD DILUTION in small bags of saline (0.9%), and then simply vary the flow rate according to the patient’s size and need.

**Intra-op dosing**

Dogs & cats – 20 - 40 μg/kg/hr

May go as high as 60 μg/kg/hr or more depending upon the patient’s need. Remember that pure mu opiates are given to effect.

**Post-op dosing**

Dogs & cats – 2 - 4 μg/kg/hr using the same standard dilution solution

Adjust the dose up if needed (we have had patients who needed 15 – 20 μg/kg/hr post-op - - remember to effect)

Create a STANDARD DILUTION of fentanyl (and other drugs) for CRI use and then simply vary the flow rate as needed.

- Fentanyl standard dilution:
  - mg/ml

**Case example for fentanyl CRI**

- 6.6# cat = 3 kg
- Pre-op bolus:
  - 5 μg/kg X 3 kg = 15 μg
  - 15 μg / 50 μg/ml = 0.3 ml of standard concentration fentanyl

**Intra-op**

20 μg/kg/hr X 3 kg = 60 μg/hr

Using the standard dilution of 0.01 mg/kg, a 100ml bag contains 10 μg/ml (the bigger the patient - - dog or cat - - the bigger the bag you will need), so 60 μg/hr / 10 μg/ml = 6 ml/hr. Increase the flow rate if the cat needs more fentanyl to achieve the desired analgesic result.

**Post-op**

2 μg/kg/hr X 3 kg = 6 μg/hr
We use the standard dilution, 100 ml bag which contains 10 μg/ml, so 6 μg/hr / 10 μg/ml = 0.6 ml/hr. Again, adjust the flow rate as needed.

**Case example for ketamine CRI**
The standard concentration is 100 mg/ml. Create a **STANDARD DILUTION** of 0.5 mg/ml.
The pre-op bolus dose is generally taken from the DILUTED ketamine.
- **Bolus dose:**
  - Dogs & cats – 0.5 mg/kg
- **Intra-op dose:**
  - Dogs & cats – 10 μg/kg/minute
- **Post-op dose:**
  - Dogs & cats – 2 μg/kg/minute

**Case example for ketamine CRI**
- 13.2# cat = 6 kg
- Use the 0.5 mg/ml **STANDARD DILUTION** (1 mg = 1000 μg)

**Pre-op bolus**
- 0.5 mg/kg X 6 kg = 3 mg
- Standard dilution bag contains 0.5 mg/ml ketamine, therefore 3mg = 6 ml bolus

**Intra-op**
- 10 μg/kg/min X 6kg = 60 μg/min; 60 μg/min X 60 min/hr = 3600 μg/hr;
- 3600 μg/hr / 1000 μg/mg = 3.6 mg/hr;
- 3.6 mg/hr / 0.5 mg/ml = 7.2 ml/hr

**Post-op**
- 2 μg/kg/min X 6kg = 12 μg/min; 12 μg/min X 60 min/hr = 720 μg/hr;
- 720 μg/hr / 1000 μg/mg = 0.72 mg/hr;
- 0.72 mg/hr / 0.5 mg/ml = 1.44 ml/hr
- Simply adjust flow rate up or down as needed to modify dose

**CRI lidocaine (dogs, NOT cats)**
The standard concentration is 20 mg/ml. Create a standard dilution of 4 mg/ml. There is no bolus dose, and the intra-op and post-op doses are the same:
- 50 μg/kg/min
- This is ½ the CRI dose to control cardiac dysrhythmias

We use lidocaine CRI during any abdominal procedure in dogs as it seems to be especially useful for visceral pain. Combine it with fentanyl (or morphine) CRI and ketamine CRI

**Practice tips**
1. Create spreadsheets in Excel to lower the risk for miscalculations of flow rates
2. Use increments of 0.2# (or kg), use the standard dilutions described above, and have Excel calculate flow rates based on the appropriate intra-op and post-op dosing
3. Simply multiply flow rates to increase the delivered dose if needed

**CRI standard bag dilutions**
**Fentanyl 0.01 mg/mL**

<table>
<thead>
<tr>
<th>Bag Size</th>
<th>Dilution</th>
<th>Formula</th>
<th>Calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>25mL Bag</td>
<td>0.25mg</td>
<td>25mL X (0.01mg/mL) = 0.25mg</td>
<td>25mL X (0.01mg/mL) = 0.25mg / (0.05mg/mL) = 5.0mL</td>
</tr>
<tr>
<td>50mL Bag</td>
<td>0.5mg</td>
<td>50mL X (0.01mg/mL) = 0.50mg</td>
<td>50mL X (0.01mg/mL) = 0.50mg / (0.05mg/mL) = 10.0mL</td>
</tr>
<tr>
<td>100mL Bag</td>
<td>1.0mg</td>
<td>100mL X (0.01mg/mL) = 1.0mg</td>
<td>100mL X (0.01mg/mL) = 1.0mg / (0.05mg/mL) = 20.0mL</td>
</tr>
<tr>
<td>250mL Bag</td>
<td>2.5mg</td>
<td>250mL X (0.01mg/mL) = 2.5mg</td>
<td>250mL X (0.01mg/mL) = 2.5mg / (0.05mg/mL) = 50.0mL</td>
</tr>
<tr>
<td>500mL Bag</td>
<td>5.0mg</td>
<td>500mL X (0.01mg/mL) = 5.0mg</td>
<td>500mL X (0.01mg/mL) = 5.0mg / (0.05mg/mL) = 100.0mL</td>
</tr>
<tr>
<td>1000mL Bag</td>
<td>10.0mg</td>
<td>1000mL X (0.01mg/mL) = 10.0mg</td>
<td>1000mL X (0.01mg/mL) = 10.0mg / (0.05mg/mL) = 200.0mL</td>
</tr>
</tbody>
</table>

Remove the corresponding volume of saline and replace with the calculated amount of fentanyl.
Ketamine 0.5 mg/mL

25mL Bag

25mL X (0.5mg/mL) = 12.5mg / (100mg/mL) = 0.125mL
Remove 0.12mL of saline and replace with 0.12mL Ketamine.

50mL Bag

50mL X (0.5mg/mL) = 25.0mg / (100mg/mL) = 0.25mL
Remove 0.25mL of saline and replace with 0.25mL Ketamine.

100mL Bag

100mL X (0.5mg/mL) = 50.0mg / (100mg/mL) = 0.50mL
Remove 0.50mL of saline and replace with 0.50mL Ketamine.

250mL Bag

250mL X (0.5mg/mL) = 125mg / (100mg/mL) = 1.25mL
Remove 1.25mL of saline and replace with 1.25mL Ketamine.

500mL Bag

500mL X (0.5mg/mL) = 250mg / (100mg/mL) = 2.50mL
Remove 2.50mL of saline and replace with 2.50mL Ketamine.

1000mL Bag

1000mL X (0.5mg/mL) = 500mg / (100mg/mL) = 5.00mL
Remove 5.00mL of saline and replace with 5.00mL Ketamine.

Lidocaine 4 mg/mL

25mL Bag

25mL X (4mg/mL) = 100mg / (20mg/mL) = 5.0mL
Remove 5.0mL of saline and replace with 5.0mL Lidocaine.

50mL Bag

50mL X (4mg/mL) = 200mg / (20mg/mL) = 10.0mL
Remove 10.0mL of saline and replace with 10.0mL Lidocaine.

100mL Bag

100mL X (4mg/mL) = 400mg / (20mg/mL) = 20.0mL
Remove 20.0mL of saline and replace with 20.0mL Lidocaine.

250mL Bag

250mL X (4mg/mL) = 1000mg / (20mg/mL) = 50.0mL
Remove 50.0mL of saline and replace with 50.0mL Lidocaine.

500mL Bag

500mL X (4mg/mL) = 2000mg / (20mg/mL) = 100.0mL
Remove 100.0mL of saline and replace with 100.0mL Lidocaine.

1000mL Bag

1000mL X (4mg/mL) = 4000mg / (20mg/mL) = 200.0mL
Remove 200.0mL of saline and replace with 200.0mL Lidocaine.

Morphine 0.9 mg/mL

25mL Bag

25mL X (0.9mg/mL) = 22.5mg / (15mg/mL) = 1.5mL
Remove 1.5mL of saline and replace with 1.5mL Morphine.

50mL Bag

50mL X (0.9mg/mL) = 45.0mg / (15mg/mL) = 3.0mL
Remove 3.0mL of saline and replace with 3.0mL Morphine.

100mL Bag

100mL X (0.9mg/mL) = 90.0mg / (15mg/mL) = 6.0mL
Remove 6.0mL of saline and replace with 6.0mL Morphine.

250mL Bag

250mL X (0.9mg/mL) = 225mg / (15mg/mL) = 15mL
Remove 15mL of saline and replace with 15mL Morphine.

500mL Bag

500mL X (0.9mg/mL) = 450mg / (15mg/mL) = 30mL
Remove 30mL of saline and replace with 30mL Morphine.

1000mL Bag

1000mL X (0.9mg/mL) = 900mg / (15mg/mL) = 60mL
Remove 60mL of saline and replace with 60mL Morphine.
Study after study consistently reports that more than 80% of pet owners think of their pets as children. There are more cats than dogs as pets now, and this means job security for us! Our moral imperative is to advocate on behalf of a being that cannot advocate for itself.

Pain, according to the IASP definition, is an unpleasant and emotional experience associated with actual or potential tissue damage. The definition goes on to state that the inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain relieving treatment. Pain is a complex and multifaceted experience. There is a sensory-informational component, an emotional dimension (suffering aspect), and a cognitive-evaluative component (attention, previous experience, perceived threat to individual). In people, pain is what THE PATIENT says it is because humans can self-report. As for pain in animals, pain is what WE say it is - - we assess our patients by proxy. There is tremendous individual variability both among patients and observers. There is no one “right” answer.

Nociceptive pain is transient pain in response to a noxious stimulus. Inflammatory pain is spontaneous pain and hypersensitivity to pain in response to tissue damage and inflammation. These are both considered “adaptive” pain. “Maladaptive pain”, on the other hand, is represented by neuropathic pain - - spontaneous pain and hypersensitivity to pain in association with damage to or a lesion of the nervous system - - and functional pain - - hypersensitivity to pain resulting from abnormal central processing of normal input. It was Clifford Woolf in the Annals of Internal Medicine who articulated these two overarching categories of pain. It is important to understand that pain is a spectrum and that pain can (and will) morph from adaptive to maladaptive if it is not appropriately managed.

Wind-up involves sensitization of nociceptors, and peripheral and central pain pathways, in response to a barrage of afferent nociceptive impulses resulting in expanded receptive fields and an increased rate of discharge. This is what we need to try to avoid.

Patient pain assessment is highly individual and variable for both the patient and for the observer. In our assessment, we must consider the reason and context for the patient’s pain. A good pain scoring system must be based on behavior, species specific, and must be influenced by the type and intensity of pain the animal is experiencing. The ideal pain scoring system would have the following characteristics:

- Clearly defined assessment criteria
- Suitable for all observers
- Simple and quick to use
- Sensitive
- Useful tools for intervention
- Identified strengths and weaknesses
- Validated in the cat (because cats are NOT small dogs!!!)

For the record, we still do not have an ideal pain assessment system…

Pain induced behavioral changes include:

- Loss of normal behaviors
- Lack of grooming
- Decreased appetite
- Decreased water intake
- Lack of movement
- Decreased interactive behaviors

Are you dealing with an aggressive cat? Think about pain!

Data suggest that @20% of cats across all ages are dealing with OA, yet 20% of cats in veterinary practices are not being treated for OA. You can’t treat what you don’t see, so we must look for pain, keep an open mind, localize it, identify the source if possible, and make a treatment plan.

This pain palpation examination is adapted from the human pain arena, referenced and refereed, and is part of the training of human practitioners as the technique for identifying tender points in a diagnosis of fibromyalgia. It appears in the veterinary literature in an article titled, “Managing chronic maladaptive pain” in Clinician’s Brief, August 2011 (Downing). This is a technique we have used at The Downing Center for nearly 10 years with excellent reproducibility.

Perform your pain palpation systematically and the same way each time. Use 4kg of pressure at each palpation site. Use the fleshy part of P3 (NOT the fingertip). Begin in the paraspinals at the base of the occiput and proceed to the base of the sacrum. Palpate the paraspinals at approximately each spinal segment, and don’t forget the base of the tail. Palpate the circumference at base of neck by
the hands of the clock - - 10 & 2, 9 & 3, 8 & 4. Palpate caudal to scapulae and at the T/L junction the same way. Next palpate the lateral lumbar muscles segment by segment, and then the iliopsoas muscle bundle starting at the T/L and proceeding to the L/S. Squeeze the proximal quadriceps between the flat of the thumb and the lateral surface of the index finger (P2 & P3). Perform joint ROM from toes to torso, and then finish with the head and face. Return to the areas generating a reaction and evaluate them with additional palpation and/or ROM to better identify and characterize the presence and the nature of the pain.

Teach, drill, and practice, practice, practice and include your entire staff. It IS possible to create consistency of assessment within your practice!

Resources

ISFM & AAFP Consensus Guidelines: Long-term Use of NSAIDs in Cats
Task Force Members:
Andrew H Sparkes, BVetMed, PhD, DECVIM, MRCVS
Reidun Heine, DVM, PhD, MRCVS
B Duncan X Lascelles, BSc, BVSc, PhD, MRCVS, CertVA, DSAS(ST), DECVS, DACVS
Richard Malik, DVSc, DipVetAn, MVetClinStud, PhD, FACVS, FASM
Llibertat Real Sampietro, DVM
Sheilah Robertson, BVMS (Hons), PhD, CVA, DACVA, DECVAA, MRCVS
Margie Scherk, DVM, DABVP (Feline Practice)
Polly Taylor, MA, VetMB, PhD, DVA, MRCVS

Download at: http://jfm.sagepub.com/content/12/7/521.full.pdf+html

AAHA/AAFP Pain Management Guidelines for Dogs & Cats
Download at: www.aahanet.org
Pain and Fear in Felines: Linked More Often Than You Think
Robin Downing, DVM, DAAPM, DACVSMR, CVPP, CCRP
The Downing Center for Animal Pain Management
Windsor, CO

Divinum Est Opus Sedare Dolorem (Divine is the Work to Subdue Pain) - Galen

Always approach feline pain from a comprehensive perspective. Begin at the beginning with a thorough examination that includes a neurologic exam, soft tissue palpation, joint ROMs, and gait assessment. Include a metabolic profile so as not to miss important co-morbidities.

Treat the treatable - and treat all the treatable. Make a plan and work the plan, and recognize that chronic pain is best addressed from a MULTIMODAL approach, and it is no longer appropriate simply to throw an NSAID at the patient. Break the pain cycle as quickly and effectively as possible before initiating physiotherapy/tissue manipulation. Multimodal management of chronic pain means multi-tasking, and the client is an absolutely essential partner in the process, or the process is doomed to fail.

Creating a fear-free experience for cats is a huge part of our obligation to them!

Pre-exam medication options
Buprenorphine
- 0.05mg/kg
- Delivered by owner at home @ 1 hour before the visit
- Be sure to explain HOW to deliver it into the cheek pouch
- It will NOT mask chronic cat pain!

Gabapentin
- 100mg PO @ 1 – 2 hours pre-exam
- It will NOT mask chronic cat pain!

Some cats do best with BOTH gabapentin AND buprenorphine.
Acepromazine (1mg/ml) – 0.01mg/kg + hydromorphone (2mg/ml) – 0.05mg/kg - - same syringe & delivered SQ
Deliver as cat arrives, wait 20 minutes, will NOT mask chronic pain!

So, what is the relationship among pain, stress, and distress? While animals do not anticipate or fear their own DEATH, they most certainly DO anticipate and fear PAIN, rightly attempting to avoid pain whenever possible. The pain cats may choose to avoid may include the following:
- IBD
- OA
- Periodontal disease

Pain, anxiety, stress, and distress can become a self-perpetuating cycle.

Let me share some cases
Coco Chanel
- Ragdoll, FS, 5kg, 10 yrs
- Reclusive, impossible to pet, non-interactive with other cats
- Never seen when company came to the house
- Veterinary visits under general anesthesia
- Biopsy confirmed IBD
- Liquid, projectile diarrhea
- Occasional vomiting
- Easily angered

Sybil
- DMH, FS, 3.2kg, 15 yrs
- Chronic herpes
- HATED car travel
- Poop, pee, and puke in the carrier with every veterinary visit

Ganache
- Siamese cross, MC, 6kg, 5 yrs
- Should weigh 5kg
- Aggressive with other cats in household
• Anxious/aggressive at veterinary visits

**Flower**
• At rescue:
  • DSH, FS, 7.3 kg (16# !!!), 9 yr
  • Could not be touched
  • Attacked visitors to home
  • Mats over 80% of body
  • Excoriated perianal/perivulval areas
• Now:
  • 3.4 kg (7.9#) (Hills Metabolic Advanced Wt Sol®)
  • Gregarious, interactive, playful

**Rooney**
• DMH, MC, 2.8kg
• CRD
• OA – lowback, L/S, SI joints
• **21½ YEARS OLD!**
• Pain, Anxiety, & Fear (oh my!)

With unacceptable behaviors (“bad actors”), ALWAYS think about pain FIRST! Remember that anxiety exacerbates pain, and pain exacerbates anxiety. Look at the WHOLE patient. Ask LOTS of questions and LISTEN carefully to clients for clues. Ask for pictures and videos. Adequate resources are paramount, but comfort is critical. It is not enough to offer one or two fear-free techniques. Reducing fear may require reducing pain.
**Pharma and Feline Pain: Making the Best Choices**
Robin Downing, DVM, DAAPM, DACVSMR, CVPP, CCRP
The Downing Center for Animal Pain Management
Windsor, CO

Our Moral Imperative: To advocate on behalf of a being that cannot advocate for itself...

**Acute pain**
Acute pain may be planned (surgery) or unplanned (trauma), varies in severity (which dictates the intensity of our pain management strategy), may ALSO involve dealing with chronic/maladaptive pain, and we MUST recognize that a multi-modal approach provides us the opportunity to create a rational plan based on specific tissue targets.

**Surgery patients**
These patients provide the greatest opportunity for us to make a difference, and we now know that the best pain management strategy begins BEFORE the surgical insult. This decreases the need for induction agents, inhalant agents, and post-op pain medications. This provides greater comfort for the patient, sets the stage for optimal healing, and using targeted therapy means making rational choices about the areas of the nervous system we need to address and how to get there. We need to prevent the unrelenting afferent barrage of nociceptive signals which leads to peripheral and central sensitization, and “windup”. General anaesthesia ONLY causes unconsciousness and does NOTHING to move us toward the goal of analgesia. Without proper intervention (e.g. pre-emptive analgesia), the pain experience upon awakening, will be WORSE than before becoming unconsciousness.

**Preventing windup**
Local anesthetics prevent windup by blocking pain signals completely. In addition, opioids obtund windup and N-methyl-D-aspartic acid (NMDA) receptor antagonists block central sensitization e.g. ketamine as a CRI

**Pre-op**
Combining a sedative and opioid (hydromorphone & acepromazine) potentiates the effects of the opioid (but ace is NOT analgesic -- this is VERY important!). Consider carefully the use of anticholinergics (we use ONLY PRN). Keep your feline patient warm!

---

**Butorphanol has NO PLACE in a perioperative protocol!**
- Pre-med, the analgesic effects are GONE just in time for the first stroke of the scalpel
- Diminishes effectiveness of any subsequent delivery of pure mu opiate
- Expensive (by comparison) to “real” analgesics
- Still in use due to complacency

**Immediately pre-op:**
Position your patient to have an optimal surgical experience, with minimal pain and optimal healing, so at induction, take 5 – 10 minutes to help your patient by pre-oxygenating @ 4 – 6 L/min via mask while you are placing the catheter & then during induction. Create a SMOOTH induction with propofol + midazolam. It is PAST time to retire ketamine/valium! It creates a ghastly experience for cats!

**Last step pre-op - - local anaesthesia**
Place your local anaesthetic BEFORE the surgery begins! Infusing local anaesthesia AFTER a surgery/after an incision is made, is precisely like locking the barn door after the horse is gone. Depending upon the nature of the procedure, the local may be best administered via epidural, intra-articular injection, specific nerve block, etc.

**Continuous rate infusion (CRI)**
This is “continuous” rather than “constant”, and in fact, it changes with the patient’s needs as well. It is an extremely effective way to manage pain intra- and post-op. CRI dramatically reduces the inhalant concentration needed for maintenance. CRI is really the only place for ketamine any longer (not for induction)

**Intra-op**
Inhalant anaesthesia DOES NOT provide analgesia. Our pre-meds need a long enough duration to cover our surgery time, but for longer procedures, consider an epidural and/or CRI. Good surgical techniques assist with pain management by minimizing the trauma to tissues. Whatever you do, don’t forget to keep your patient warm!

**Post-op**
Continue CRI’s if they are in place, and titrate the dose over time as the cat recovers and regains strength. Do not stop CRIs abruptly. Begin your NSAID at this time if appropriate for the patient. There is NO compelling evidence to support giving the NSAID BEFORE the surgical event. Regularly assess patients and revise the pain plan accordingly. Opioids post-op can be pulse-dosed for patients who do not need or receive CRIs.

Until now – regular injectable buprenorphine was used IV or TM, and the doses quoted in the literature are:
- 0.03mg/kg – 0.05mg/kg
The dosing interval depends in large measure upon level of pain, and this formulation requires a fairly frequent dosing interval. Simbadol® (Zoetis) is really a game-changer. It is an excellent complement to an NSAID.

- **Dose:**
  - 0.24mg/kg SQ ONCE daily for 24 hours of pain relief
  - Can administer the first dose 1 hour pre-op (label)
  - We deliver it at the end of our procedures
  - For use once daily for up to 3 days

- **Be careful:**
  - Do not get it on your mucous membranes
  - Do not use in moribund cats
  - Be careful in cats with hepatic disease
  - If cats are sensitive to buprenorphine or other opioids, they may be sensitive to Simabol®

- Do NOT dispense for client administration at home
- Give the patient the benefit of the doubt when assessing for pain.
- Pain is easier to prevent than to reverse.
- An NSAID paired with an opioid better than an NSAID alone.
- The therapeutic & side-effects of opioids are dose-dependent.
- Don’t be afraid to let the patient sleep post-op provided they have stable vital signs.
- Reduce any potential adverse side effects by choosing drugs and doses to meet the patient’s needs.
- Transition to oral meds for going home.

**NSAIDs**

- Choose according to the best fit for the patient
- Determine how long the patient will benefit from their use
- If using meloxicam in cats be sure to PRECISION dose
- There is NOT compelling evidence to demonstrate that NSAID should be given BEFORE the anaesthetic event.
- NSAIDS can increase the risk of complications.

**Tramadol**

NO SAFETY OR EFFICACY DATA IN CATS! Do not use this drug!

**Gabapentin**


Sedation is the dose-limiting side effect - - non-linear pharmacokinetics. So far there is only one post-operative study about gabapentin in veterinary medicine (canine amputees), and the drug failure was predictable due to the very low and the very short time it was used… opioids will also fail if the dose is too small. Clinically, in “big pain” cases, gabapentin improves outcomes in pain scores and function – both short and mid-term. The dose range is quite wide - - 5 – 20 mg/kg PO BID – TID. There will be effects within 24 hours – consistent effects within 3 – 5 days – post-op for 14 – 21 days.

Remember to use physical medicine options to enhance pain management post-op. You can use these techniques immediately post-op as well as at home when appropriate. Don’t forget general nursing care - - keep the patient warm, turn regularly, keep bladder empty, keep clean of soiling. Use cryotherapy over the surgery site to decrease pain & swelling. Build your “pain management pyramid” based on several factors:

- The anticipated pain resulting from the procedure
- Routine castration < routine OHE or cryptorchid castration < toe amputation < large tumor removal < total ear ablation < limb amputation or other orthopedic procedure
- Give the patient the benefit of the doubt
- Layer your modalities to meet the patient’s needs both immediately post-op and as recovery proceeds

**Resources**

Veterinary Anaesthesia Support Group
www.vasg.org
American Academy of Pain Management

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When considering pain sensation, we MUST remember that all pain is not created equal!!!

Nociception is where it all begins. The reception, conduction, and central nervous system processing of nerve signals is generated by the stimulation of nociceptors. This is the first step in the physiological process that leads to the perception of pain. We are taught to consider “types” of pain.

**Transient/physiologic pain**
This is pain that is elicited by activation of nociceptive transducers in the absence of any tissue damage. Transient/physiologic pain plays a protective role, occurs on a daily basis, and does not require medical attention. It actually protects us and preserves us. It [plays an important evolutionary role.

**Acute pain**
This is pain that is elicited by substantial injury and includes surgery, trauma, chemical insult, radiation. This is the normal, predicted physiological response to an adverse chemical, thermal or mechanical stimulus, and is the initiation phase of an extensive, persistent nociceptive and behavioral cascade triggered by tissue injury. If either unmitigated or UNDER=managed, it can set the stage for a perpetual state of self-sustaining pain. This is pain that demands our careful attention.

**Chronic pain**
This is pain that persists for longer than the expected time frame for healing or pain associated with progressive, non-malignant disease. We have been taught to consider it to be a distinct category of pain.

**Finally, we consider maladaptive pain**
This is pathologic pain - - pain that has exceeded its protective usefulness. It can be an overwhelming experience and is often associated with tissue injury incurred at the time of a surgery or injury that is either untreated or UNDER-treated. The results can include hyperalgesia and allodynia. Another type of specific pathologic pain can occur in the cancer patient, and may be the result of a primary tumor, metastatic disease, or the toxic effects of chemotherapy and radiation.

It is time to consider the fact that pain is a spectrum from adaptive to maladaptive pain - - “good” pain vs “bad” pain - - pain that helps the patient survive vs pain as disease. (Clifford J Woolf, Annals Intern Med 2004) Maladaptive pain includes neuropathic pain (spontaneous pain and hypersensitivity to pain in association with damage to or a lesion of the nervous system) and functional pain (hypersensitivity to pain resulting from abnormal central processing of normal input). Maladaptive pain is associated with “wind-up”, the sensitization of nociceptors, and peripheral and central pain pathways, in response to a barrage of afferent nociceptive impulses resulting in expanded receptive fields and an increased rate of discharge.

Patient pain assessment is highly individual/variable both for the patient and for the observer. We must consider the reason and context for the patient’s pain. AND, of course we must remember that cats are NOT small dogs!

One important way to make pain a priority in your practice, educate your team, educate your clients, become members of the IVAPM. The next step is to pursue pain management certification as a Certified Veterinary Pain Practitioner.
For pain in animals, pain is what WE (humans) say it is. There is tremendous individual variability among both patients and observers. There is not one “right” answer. Pain is complex and scientifically intriguing, clinically challenging, and easy to overlook (especially in cats). To improve the treatment of feline pain, we MUST make changes - - never an easy task.

The fundamentals of acute pain management mean understanding that treating pain is good medicine, that providing peri-operative analgesia means before, during, and after the surgery, and that multi-modal analgesia is the strategy of choice.

We need to update our previous training about different “types” of pain - - acute vs. chronic, cancer pain, acute on chronic, etc. We now understand that pain is a spectrum that transitions and transforms from “adaptive” pain to “maladaptive” pain. If we deal with pain aggressively early on, we prevent the transformation to maladaptive pain. Maladaptive pain gives us more targets to treat. Wind-up involves the sensitization of nociceptors, and peripheral and central pain pathways, in response to a barrage of afferent nociceptive impulses resulting in expanded receptive fields and an increased rate of discharge. While general anaesthesia causes unconsciousness, thus preventing the cortical experience of pain, it does not prevent wind-up. Without proper intervention (e.g. pre-emptive analgesia), the pain experience upon recovery from anaesthesia will be worse upon awakening.

Local anesthetics prevent windup by blocking pain signals. Opioids obtund windup. N-methyl-D-aspartic acid (NMDA) receptor antagonists block central sensitization (e.g. ketamine CRI). Part of the therapeutic approach means to minimize tissue damage when possible. The pharmacological approach means blending medications:

- Opioids
- (+/-)Alpha-2 agonists
- Local anaesthetics
- NSAIDs
- Ketamine (CRI)
- Sedation (acepromazine is NOT analgesic)

Other strategies include good nursing care, interaction with care-givers, ice packs, supportive bandages, lubricating eyes, emptying the bladder, removing dried blood and soap, providing plastic sheets with absorbent liners, supporting head and limbs, turning regularly. Complementary techniques may also help (acupuncture, therapeutic laser, etc.). Keep your patient warm before during and after the procedure, provide an IV fluid bolus before surgery, provide IV fluids during every anaesthetic event, and pre-oxygenation before beginning.

Multimodal therapy utilizes multiple actions on the CNS to prevent and treat pain. Sedatives and opioid analgesics are the most common combination of drugs used prior to general anaesthesia.

- Acepromazine as a pre-med:
  - Excellent tranquilizer
  - NO analgesic effects
  - Potentiates opioid effects (synergistic) in the pre-med
  - Use very low doses
  - Cats – 0.01 mg/kg SQ
  - Dilute to 1 mg/ml solution

- Benzodiazepines (e.g. diazepam, midazolam):
  - Few cardiopulmonary effects
  - Not a good anxiolytic when used alone
  - Can cause paradoxical excitement (diazepam)
  - We use midazolam for INDUCTION, and NOT as part of the pre-op protocol
  - Induction dose – 0.25 mg/kg IV (BOTH dogs & cats)

- α2-agonists (e.g. dexmedetomidine):
  - Dose-dependent sedation, cardiopulmonary depression, and analgesia
  - Pick your patients carefully!!!

- Opioids:
  - μ-agonists: morphine, hydromorphone, fentanyl
Cornerstone of perioperative pain management
- Given to effect
- Possible bradycardia, respiratory depression, nausea, but ONLY when the need for pain relief is exceeded by the dose
- Good for moderate to severe pain

Morphine is not as effective an analgesic for cats as dogs, so it will not be discussed here.

- **Hydromorphone:**
  - Moderate to severe pain
  - Similar in action and duration to morphine w/ no histamine release
  - Less vomiting than morphine in non-painful patients
  - Duration 4 hr IV, IM, SQ
  - Possibly better efficacy in cats than morphine, but hyperthermia at analgesic doses in many cats
  - Pre-op with low-dose (1mg/ml) acepromazine:
    - 0.05 mg/kg SQ
  - Post-op:
    - 0.05 mg/kg SQ or IV

- **Fentanyl:**
  - Moderate to severe pain (similar to morphine)
  - Rapid onset (minutes), VERY short duration (minutes)
  - IV, IM, SQ, epidurally (transdermal – no good)
  - Best as IV CRI for balanced anesthesia or for unremitting pain
  - Loading dose, then infusion
  - Pre-op bolus:
    - 5 µg/kg
  - Intra-op CRI:
    - 20 µg/kg/hr
  - Post-op CRI:
    - 2 – 10 µg/kg/hr

- **Buprenorphine:**
  - Partial µ-agonist, mild to moderate pain, drowsiness (little sedation), duration 6 to 12hr
  - NOT absorbed via SQ
  - Use IV or TM (can use IM if needed)
  - NOW we have Simbadol® from Zoetic (1.8mg/ml)
    - VERY DIFFERENT dosing than conventional, human formulation, buprenorphine
    - This formulation MUST NOT be sent home with clients!!!

- **Butorphanol:**
  - Mixed µ-agonist/antagonist with a ceiling effect and an EXTREMELY short duration (@20 min analgesia)
  - Attenuates the efficacy of subsequently administered µ-agonists (long blockage of µ-receptors) to PREVENT effective analgesia
  - *NOT AN EFFECTIVE ANALGESIC!*
  - ONLY good for smoothing out dexmedetomidine

- **Ketamine:**
  - NMDA receptor antagonist
  - No longer has a place for anaesthesia induction
    - Dissociative (induction) dose merely creates dissociation, no long-lasting pain relief
    - Rough to sleep, rough to consciousness
  - Best in CRI application for balanced anaesthesia (prevents wind-up)
  - Can also use as CRI to “ramp-down”/”break cycle” of chronic pain
  - Pre-op bolus:
    - 0.5 mg/kg
  - Intra-op CRI:
    - 10 µg/kg/minute
  - Post-op CRI:
• 2 µg/kg/minute

• Creating the acute pain plan/general anaesthesia:
  o Pre-med (Hydro+ace, no anticholinergic) + EMLA
  o Pre-oxygenation during induction
  o Induction (propofol + midazolam) followed by loco-regional blocks
  o Inhalant maintenance
  o +/- adjuncts (CRI)

• Post-op pain management:
  o Opioid
  o Hydromorphone (careful about feline hyperthermia)
  o Buprenorphine
    ▪ IV or TM
    ▪ Mild – moderate pain
    ▪ Don’t forget Simbadol®
  o NSAID (if appropriate for the patient)
  o +/- Adjuncts - - CRI, cryotherapy, therapeutic laser
  o Other consideration:
    ▪ Gabapentin

• Gabapentin:
  o Neuromodulator at the α-2-δ ligand of Ca channel in dorsal horn of spinal cord
  o Alters calcium permeability & raises firing threshold
  o Like resetting a pain thermostat
  o Standard for chronic maladaptive pain – new in perioperative setting – in C-section & knee arthroplasty, decreased need for opioid (human studies)
  o No good studies yet in dogs or cats – anecdotally, great results
  o Some good response within 24 – 48 hrs, max effects within
  o 5 – 7 days
  o 5 – 15mg/kg PO BID for 10 – 14 days, q 24hr for 7 – 10 days, then discontinue

Pain summary
• LOOK for pain and you will find it
• Remember the risk factors (age, breed, size, history of injury, etc.)
• Learn how to “ask” your patients if they hurt
• Learn how to respond when they say “yes”

Resources
Veterinary Anaesthesia Support Group www.vasg.org
American Academy of Pain Management www.aapainmanage.org
American Society of Pain Educators www.paineducators.org
Cats do not see or experience the world the way we do. In our busy days at the clinic, it is imperative that we take pause and assess how our feline patients interpret their veterinary experience. From the moment the client schedules the appointment to the moment they return home, the cat’s experience can be traumatic and frightening. Understanding natural cat instincts and planning their visit to accommodate for this will improve the visit experience for everyone, especially the cat and client.

In a natural environment, cats are predators AND prey. We frequently think of cats as hunters but forget that they are also hunted. This means that during their veterinary visit, they frequently feel threatened. This is the baseline instinct that can drive many cats’ negative reactions to a visit to the veterinarian.

The cat’s unique senses
The unique senses of the cat impact how they interact with their world. Cats communicate through olfactory, visual, tactile and auditory means. A cat’s sense of smell is significantly more sensitive than a human. They perceive their world in overlapping clouds of smell. This in itself can lead to a heightened sense of awareness in the examination room. Although we believe we thoroughly clean our hospitals, many scents remain behind to arouse our feline patients. This can lead to redirected aggression or fear in the examination room. Vision at night for cats may be good, thanks to the retinal tapetal reflective tissue. Since they primarily hunt at night, our feline friends have little need for colour vision. The feline range of vision is best at 2-6 metres. Close up, feline vision is less than ideal, thus impacting their stress levels when foreign items are close by (this includes cucumbers, which can completely traumatize the unsuspecting feline). The feline binocular vision which has a 98 degree overlap allows for accurate assessment and judgement of distance. Cats have amazing hearing, using their pinna to rotate and collect as many surrounding sounds as possible. The pinna can swivel almost 180 degrees and move independently of one another. This helps them to track and locate prey, but also to detect predators. Remote sounds from outside of the examination room can be frightening to the feline patient.

Tactile senses permit communication with fellow felines and other species, including the veterinarian. Their responses can include affiliative communication like rubbing, head hunting, nose-touching, kneading, treading and allogrooming. Negative or agonistic communication can include biting and scratching.

Cats are easily threatened. Their response to threats is to flee, freeze or fight. As veterinarians we have all experienced this range of reaction in our feline patients. Our patients communicate with us by many visual cues. Understanding these is critical to improving feline visits. We need to monitor their posture, examine their facial expressions and respond accordingly.

Routine patterns of behavior
As obligate carnivores and solitary hunters, cats tend to be territorial and find safety in predictability of their surrounding environment. As household members, most clients understand that their cats are schedule-oriented. Cats appreciate consistency, know when mealtime has arrived, and are stressed by disruptions in their regular routines. A visit to the veterinarian is a definitive disruption in this routine. When one considers how stressed clients can become in anticipation of a trip to the vet with the cat, the cat is more likely to anticipate the changes and experience stress well in advance of departure. Assisting our clients in preparation for the veterinary visit can go a long way to reducing the initial visit stress.

Goals for a good veterinary visit
Our goals for a good veterinary visit should include reducing anxiety in clients, patients and staff. Veterinary staff should seek ways for the client and patient to have a positive, productive visit with zero injury to patient, client and staff.

A stress free visit starts at home. The client should be provided with emotional support in person and by telephone or email. The client should be encouraged to stay calm. Provision of the best type of carrier, and instruction to use this correctly is important. Treats, rewards, facial pheromones and necessary medications should be discussed and utilized as needed.

Avoid anticipating behavior in a negative way. It is critical to understand and remember that a patient may have a history of negative behavior in our clinic, but labeling patients as fractious, for example, is not productive in making change to the patient experience. Making positive steps to address this are better than just preparing for the worst. The initial minutes after a ‘bad’ visit should be used to assess what went wrong, understanding how the patient was feeling and how that dictated the patient response. In addition to providing telephone support, the author utilizes medications such as gabapentin and/or buprenorphine or alprazolam for anxious patients. These are prescribed and administered in advance of the veterinary visit.
Address pain prior to the visit
Pain is a major consideration in many of our patients. In a study by Lascelles et al (2010), cats between 6 months and 20 years of age were randomly selected for radiographic assessment for the purpose of detecting lesions consistent with arthritis. In this study, 91% of the cats were shown radiographic evidence of DJD. This was show to exist with equal frequency in all age groups. This study highlights the need for practitioners to ANTICIPATE pain. A proactive effort to reduce or eliminate pain prior to the visit and handling will make a significant difference in the patient’s experience. The author frequently utilizes gabapentin, and sometimes buprenorphine, in advance of visits in patients with known or suspected arthritis pain. This applies to patients with other types of pain as well.

Restraint: Lightest touch is the strongest hold
During handling of feline patients for any reason, the least amount of handling is best. Most patients do better when they have some control over the situation and are not restrained in any aggressive way. Enhancing the patient environment with dim lighting, quiet and a warm, fuzzy blanket can improve the patient demeanor dramatically. Sitting on the floor with the patient can be helpful, if the examination table is too stressful. Some practitioners do not have examination tables in their feline consultation rooms. Avoiding eye contact is beneficial. Reading patient body language and responding accordingly will ease anxiety. The practitioner should avoid touching the patient if the cat is engaged in sniffing items of particular interest. In this type of environment, cats are more amenable to handling for physical examinations, blood pressure assessments, injections and tissue sampling (blood draw and urine sampling).

For blood and urine sampling, patient pain should be addressed well in advance. Patient positioning should depend on patient comfort levels. Reduced restraint levels produce better results. Aggressive restraint methods are not ideal. The author does not support the use of scruffing or so-called ‘clipnosis’.

In cases where patients are simply not cooperative for handling, chemical restraint in the form of sedation is best. The alternative is physical restraint. The latter hurts the patient, reduces the success of data collection and puts all involved at risk of injury. The latter also impacts future veterinary visits for the now traumatized patient and client.

Feline friendly practice
Certification with the AAFP Feline Friendly Practice program will assist the veterinary staff and practitioners in understanding and being prepared to interact with the feline species. The certifications are valuable in clinic promotion to existing and potential clients as well.

At the end of each feline visit, the goal is for everyone involved to feel like the experience was positive and productive for feline, client and staff inclusive. Objectives for care should be met without the need for brute force, with an aim to understanding the unique feline senses and how these affect the feline veterinary experience.

References
As cats reach their senior and geriatric years, our focus on their health needs to intensify. By now we have established a working relationship with our client over the years of the patient’s life. They recognize that their pet is getting old. However, they do not always understand exactly what changes are going to occur. Above all, the goal of the veterinarian and the client should be to avoid patient suffering.

Changes can occur rapidly. Feline patients over the age of 7 or 8 years of age should be seen for a healthcare assessment every 4-6 months. For the healthy, this can be every 6 months, but for those with chronic health conditions, a move to healthcare assessments every 4 months will be beneficial.

**Recording normal behaviors**

Clients should be advised to start a journal or notebook to highlight the normal patterns of behavior of their particular cat. Timing of eating, elimination behaviors, sleep, and play, when documented, will act as an excellent resource when attempting to identify changes. This type of recording should begin as early as kitten hood. For the clinician, this journal can also assist when end-of-life decisions have to be made. Quality of life discussions are difficult at best. Knowing the level of changes that have occurred in behavior patterns over time help the client to come to terms with end-of-life decisions.

**Pain**

Many senior or geriatric patients will be experiencing one or more health conditions. A variety of diseases and conditions will predispose the patient to pain. Even if the patient is apparently healthy, the consideration of pain secondary to arthritis is critical. In a study by Lascelles et al (2010), cats between the ages of 6 months and 20 years of age were randomly selected for radiograph study. Ninety-one percent of the cats had radiographic evidence of degenerative joint disease (DJD), with equal frequency in all age groups. This type of study provides convincing evidence that DJD should not be ignored in cats, and that pain related to DJD can be a significant concern. Clients may actually be observing mobility changes and possibly even lameness, further convincing the clinician that pain needs to be addressed. There are many pain management options available to the clinician. These should be selected based on the patient history, health status and the actual source of pain. The author frequently prescribes gabapentin as a pain medication for cats with arthritis, debilitating disease and moderate to severe dental disease. While chronic use of non-steroidal anti-inflammatory drugs are frequently off-label in the feline species, these can be beneficial as an adjunct therapy. The author frequently uses meloxicam (Metacam) and rodenacoxib (Onsior) for arthritic patients. Other pain medications that the practitioner may consider for various types of pain include buprenorphine, tramadol, or amantadine. Patients with windup pain may benefit from 2-3 days of intravenous fluid therapy with constant rate infusion (CRI) therapy with ketamine, buprenorphine and/or other drugs. Many neutraceuticals are available for feline arthritis. These include omega fatty acid supplements, glucosamine and chondroitin, in liquid, pill or food form. The author frequently utilizes injectable polysulfated glycosaminoglycans (Adequan, Cartrophen) as a successful adjunct to arthritis therapy and subsequent pain relief.

“One of the psychological curiosities of therapeutic decision making is the withholding of analgesic drugs because the clinician is not absolutely certain that the animal is experiencing pain. Yet the same individual will administer antibiotics without documenting the presence of a bacterial infection.

Pain and suffering constitute the only situation in which I believe that, if in doubt, one should go ahead and treat.” **LE Davis, Clinical Pharmacologist, University of Illinois.**

**Condition scoring: Body weight, body condition & muscle condition scoring**

Senior and geriatric patients should be assessed on a regular basis. Practices should develop a recording system for body weight, body condition score (BCS) and muscle conditioning score (MCS). Trends in these values should be observed, with individual patient assessment scheduled at regular intervals throughout the year. While the patient may be seeing the clinician every 4-6 months, the patient should visit with a registered veterinary technicians every 2-4 months for assessment of body weight, BCS and MCS. Trends in these values can be the first indicators of disease or failure to successfully control known diseases. Meeting with a technician for this assessment every 2-4 months keeps the client informed of the patient status, and facilitates the client’s own understanding of body condition as a reflection of health or disease. These visits will also encourage dialogue about dietary concerns. The client may have changed the patient diet, or the patient may be experiencing previously unobserved periods of Inappetence, vomiting or anorexia. These changes will alert the technician to potential problems. The technician can in turn alert the veterinarian, and appropriate, timely follow up can be pursued.
Nutritional needs
The clinician should ensure that the dietary intake of the patient is quantified in detail by the client. Metabolic energy requirements (MERs) should be calculated frequently by the clinician or veterinary technician. Biannual visits with the veterinarian will help detect body condition or body weight changes, but regular visits in between these times should also be considered. A visit every 2-4 months with the veterinary technician for a weigh-in, BCS, MCS and nutritional assessment will go a long way to detecting problems early as well as ensuring the patient is taking in sufficient calories. Veterinary technicians take charge of these types of programs in a most efficient manner. They can easily learn and set up standard protocols for determining MERs. They will quickly become familiar with nutritional guidelines for various foods, both those sold at veterinary clinics and those available over the counter. Ensuring adequate intake is critical in senior and geriatric patients to promote health and proper immune function, as well as to avoid situations of muscle wasting and cachexia due to insufficient protein intake. In addition, when patients are showing signs of weight loss, the MERs can be evaluated to determine if reduced intake is a part of the weight loss dilemma. Quantifying daily intake for cats is a critical piece of knowledge for clients with senior and geriatric cats.

Disease concerns
Increasing age comes with an increased risk of disease. Regular monitoring for evidence of problems includes a regular, thorough physical examination and collection of a minimum data base (MDB). As noted previously, part of every consultation should be a weigh in, as well as BCS and MCS assessment. These values should be compared to previous values in order to identify trends. The physical examination should be meticulous and thorough, as should the discussion of the patient status and history with the client. Even very subtle changes in behavior patterns may be significant of a declining health status. Thorough evaluation of the patient’s eyes, and in particular the retinas, is something that is often overlooked. The retinas can provide early indication of hypertension, and thus their status should be assessed and recorded with every senior feline visit. Blood pressure testing is a critical part of every feline senior or geriatric visit. The clinician should evaluate a MDB for every senior patient. The MDB includes a total thyroid, clinical chemistry, complete blood count and urinalysis. Urinalysis may results may indicate the need for further testing such as urine culture and/or urine protein creatinine ratio.

Where renal disease is identified, thorough IRIS staging will target therapies and improve prognosis as well as quality of life.

Unique environmental needs
Senior and geriatric cats have unique environmental needs. This mainly stems from their likely reduced mobility secondary to arthritis, but can also be related to cognitive dysfunction either as a primary or secondary (to other disease) problem. The senior patient may no longer feel comfortable running to the basement to use the litter box. Placement of boxes throughout the house is recommended. A high walled litter box may be viewed as a painful challenge to be avoided. Use of low entry or low walled, uncovered litter boxes is recommended. Assistance with access to higher furniture such as beds and window sills can be accomplished with steps or platforms to reduce necessary jumping heights.

Cognitive changes
There are no specific diagnostic criteria for the diagnosis of cognitive dysfunction (CD) in cats. In many instances, it is a diagnosis of exclusion. The mnemonic DISHA is used as a means of diagnosing CD in dogs (Disorientation, Interactions, Sleep-wake, House-training, Activity). This may be of some use in the diagnosis of feline CD. Therapies for CD are not well-studied in the feline species. Selegiline (Anipryl) has been utilized for the treatment of CD in cats. Nicergoline (Fitergol), Propentofulline, (Vivitonin), oxazepam/orazepam/clonazepam, buspirone and fluoxetine have also been used. Omega fatty acid supplements and other antioxidants may be beneficial. Environmental adjustments should be made to accommodate patient needs. Pain management is a mainstay component of care in senior and geriatric cases of CD.

References
Cats age rapidly. The domestic cat reaches its prime by 3 to 6 years of age. At 7, the domestic cat can be classified as mature. A 10-year-old mature cat is the human equivalent of a 56 year old. From 11 to 14 years, the cat is classified as a ‘senior’, the human equivalent of 60-72 years of age. After 15 years, the cat is considered geriatric, the equivalent to a human 76-years of age or older.

It follows that if age category changes occur so rapidly, so will health. While in the junior and prime years a cat might get away with seeing the veterinarian once yearly. The senior cat needs more frequent visits. In the space of less than 6 months, a geriatric feline can change from perfectly healthy to diseased and debilitated. Biannual visits are best and should be discussed, recommended, and scheduled with the client. The clinically ill should be seen more frequently as medical needs dictate.

Feline aging
Although the risks of certain diseases increases with age, age in and of itself is not a disease. Changes in the patient as noted by the client or the clinician should be investigated thoroughly as they are likely to pertain to disease or poor health. Many conditions are treatable and manageable, particularly if caught early. Avoiding pursuit of diagnostics and care simply because the cat is ‘old’ is not recommended. The value and quality of life at the patient may be impacted positively for years if conditions are diagnosed and treated appropriately.

Pain
If cats are masters at hiding illness, they are geniuses at hiding pain. A client may not perceive changes suggestive of pain in their feline friend. Day to day changes may be subtle in their progression, eluding the client’s observations. Some clients may excuse away the changes, citing age as a factor, but not recognizing that the main impetus behind behavior changes is likely to be pain. Monitoring normal patterns of behavior will help detect changes that may be occurring as a result of pain. Having the client note the cat’s mobility pattern, including willingness to jump up or down, as well as litter box usage is key. Clients should be encouraged to make notes about their cat’s activities and behaviors, especially as they age. This way, subtle and gradual changes will not be missed.

The subtle signs of sickness
Many practitioners are aware of the ‘subtle signs of sickness’ in cats. We understand that cats are masters at hiding illness. We understand and seek to help our clients recognize what subtle changes can mean with regard to feline health. In addition to being a sign of disease, we also have to recognize these subtle changes as evidence of possible pain.

1. Inappropriate Elimination Behavior or Litter Box Use
2. Changes in Interaction
3. Changes in Activity
4. Changes in Sleeping Habits
5. Changes in Food and Water Consumption
6. Unexplained Weight Loss or Gain
7. Changes in Grooming
8. Signs of Stress
9. Changes in Vocalization
10. Bad Breath

Condition scoring: Body & muscle
As cats age, changes in body weight, body condition and/or muscle condition can be the earliest signs noted that disease is present. Assessment and recording of body weight at every single veterinary visit is necessary to detect subtle changes early. Each patient will need to have body condition scoring as well as muscle condition scoring. As cats age, body muscling naturally changes. Cats will undergo decreases in muscling, a natural process referred to as sarcopenia. This needs to be distinguished from the more negative change known as cachexia. Cachexia can indicate the presence of disease, insufficient dietary needs and in particular, insufficient dietary protein.
Disease concerns
Senior and geriatric patients are at increased risk of disease in general. Risks of conditions such as chronic renal disease and hyperthyroidism are known to increase with age. Older patients are also at increased risk of neoplasia, hypertension, cardiac issues and of course, arthritis/degenerative joint disease. Dental disease and pain are common. Observations of unexplained changes in body weight, behavior, appetite, drinking, elimination behavior and grooming need to be addressed by the client and clinician in a timely fashion.

Nutritional needs
As cats age, their caloric and nutritional needs change. Early on in the aging process, up to 11 years of age, a cat’s energy needs will decrease by 3% per year. However, at the age of 12 and up, the energy needs actually increase. As cats age, they become less efficient at digesting food. In particular, the digestion of fats and proteins may be impaired. Senior & geriatric feline patients can be susceptible to weight loss. Dietary palatability is a major concern in this age, including ensuring that the patient is consuming sufficient calories to meet their metabolic energy requirements (MERs).

Each clinic should develop its own program for monitoring and supporting the individual nutritional needs of patients. Registered veterinary technicians are excellent resources for this type of program, and can often handle managing these programs, reporting to the clinician as concerns arise. The senior and geriatric feline should have regular weigh in visits to determine body condition and muscle condition scores. These visits should be conducted at least every 3-4 months, and more often in the clinically ill senior or geriatric feline.

Unique environmental needs
The senior and geriatric feline will have changes associated with their five senses, as well as strength and mobility. These changes will impact environmental needs. Changes in play items and structures, sleeping areas and litter boxes will need to be considered. Litter box locations and wall heights will need to be addressed. As arthritis needs are addressed with pain management and other care, environment changes will reduce the stresses on the musculoskeletal system.

Cognitive changes
Behavioral problems in the geriatric cat may be explained by the presence of disease and pain. Treatment of the disease, and/or treatment of pain will often resolve behavioral changes. Howling may be observed in some cases of hyperthyroidism, as well as patients with hypertension. Changes in elimination, including soiling outside of the litter box can occur with conditions such as diabetes mellitus, renal disease, lower urinary tract disease, hyperthyroidism and neoplasia. Pain can lead to many changes in behavior including, but not limited to, elimination issues, irritability, increased sleeping, howling, decreased grooming and decreased mobility. Regular clinical testing as well as pain management will help identify disease and pain-related causes of behavior changes. In some cases, cognitive dysfunction (CD) may be the primary source behind the behavior changes noted. Although there are no specific diagnostic criteria for CD in cats, ruling out other causes and treating for pain will help the clinician form a presumptive diagnosis. Cognitive dysfunction signs in cats can include disorientation (time or space), altered learning and memory, house soiling, altered interactions with the client, activity changes, sleep pattern changes, alterations in appetite, and/or decreased grooming. Vocalization may also occur.

References
Vomiting in the Cat: How much is too much?
Clients and veterinarians often accept that vomiting in cats is a regular occurrence not significant of underlying health problems. This is a particularly common assumption with regard to vomit containing hairballs. Cats spend approximately 25% of their waking hours grooming (Panaman et al, 1981). Ingestion of hair is obviously a natural side effect of this process. The majority of ingested hair passes through the cat’s digestive tract into the feces with no negative side effects (Panaman et al, 1981). Given the large commercial industry built around the ‘hairball’ phenomenon, clients may view hairballs as a normal process. When questioning clients about occurrence and frequency of vomiting during routine examination, veterinarians should inquire separately about the occurrence and frequency of hairball vomit. The answer is sometimes surprising, and may be key to identifying the presence of gastrointestinal disease (GID) in the patient (Cannon, 2013)

Cats that vomit once or twice a year may not be considered to have any specific underlying GID. However, cats that are vomiting more often than twice monthly are significantly more likely to have some baseline underlying GID (Norsworthy et al, 2015). In a study by Norsworthy et al (2015), 100 cats that presented with vomiting more frequently than twice monthly were subjected to abdominal ultrasound, exploratory surgery and intestinal biopsy. Of the 100 cats biopsied, 99 were found to have histologic evidence of intestinal disease. Only 1 patient had normal intestinal biopsies. For many of these cats, the clients had not been concerned about the frequency of vomiting, citing it as a normal result of hairballs, as well as behaviors such as eating too fast.

Where to start: Identifying abnormal vomiting or diarrhea
During routine preventive care examinations, detailed questioning about diet, diet changes, vomiting and hairballs is essential. As noted above, many clients will not consider hairballs noteworthy or mentionable. As a result, without proper questioning by the veterinarian, a patient vomiting frequent hairballs may not be identified. Where clients are not sure about vomiting and/or hairball frequency, a calendar recording system should be recommended. Simple recording of the vomit or hairball on the kitchen calendar, noting frequency, will help the client and the veterinarian document the vomiting frequency. This type of recording is helpful in evaluating response to treatment as well.

A history of abnormal bowel movements should also be investigated. Diarrhea can occur in conjunction with upper GID, or as a manifestation of lower GID. Documentation of stool character, volume and frequency will help elucidate the source of the diarrhea (small intestine versus large intestine versus mixed). Conditions such as inflammatory bowel disease (IBD) can exist as a problem within the upper gastrointestinal, small intestinal, combined small intestinal/large intestinal or solely large intestinal tract. Identification of which intestinal segment is causing the diarrhea is important in identifying which portion of the GI tract is potentially diseased.

Small bowel versus large bowel diarrhea
- **Small**: large volumes, variable frequency, flatulence, borborygmus, melena, steatorrhea, vomiting
- **Large**: small, frequent volumes, tenesmus, urgency, dyschezia, mucus, frank/fresh blood, vomiting

The veterinarian should also carefully question the client to identify evidence of constipation. Difficulty with passage of bowel movements may also be a cause of nausea. The client may have noticed small, hard feces in the litter box. Discussion with regard to what is normal fecal matter in the cat will help elucidate whether the patient is experiencing abnormal feces. During the last two decades, when cats have been fed primarily dry food, clients have come to have an expectation of small, firm feces. In recent years, more cats are being moved to canned diets. As cats consume more water in their diet, the definition of normal’ fecal consistency will need to be changed.

In addition to regular vomiting, the patient may be showing signs of nausea at home that are not obvious to the owner. Cats that have a tendency to be fussy about the food they eat, moving from one type to another for short periods followed by food refusal, are likely experiencing nausea or GI upset. While ‘flavor fatigue’ is an understood observation in the domestic feline species, refusals of new diets may not simply be due to boredom (Little, 2011). Clients may describe cats that go off food entirely for 1-2 days followed by a perfectly normal appetite. This should not be considered to be normal. Clients and veterinarians may notice lip licking, gagging or excessive swallowing at home or during the abdominal palpation. Coughing can be caused by upper respiratory, lower respiratory or GID. Careful history taking will elicit clues about GID and nausea. A video of apparent coughing episodes is helpful.
The subtle signs of nausea
- Finicky appetite
- Occasional loss of appetite or anorexia
- Licking of lips
- Gagging/swallowing
- Ingestion of grass to stimulate vomiting

A thorough physical examination of the vomiting cat is also beneficial when characterizing GID. The patient should be observed for signs of lip licking and frequent swallowing. A thorough oral health examination, a critical part of every feline examination, may reveal foreign objects looped under the tongue, oral ulceration or other oral or dental disease that may impact appetite and vomiting.

Feline patient weights should be recorded on every visit to the clinic, as subtle weight loss can be one of the first signs of disease. The documentation of weight loss in a cat with frequent vomiting may be the only physical examination change noted. Regardless, this change can be a hallmark of mild to significant GID.

Evidence of low-grade dehydration in the presence of a skin tent, tacky mucus membranes or sunken eyes may be identified. Dehydration may be directly related to frequent vomiting.

The subtle signs of gastrointestinal disease
- Unexplained weight loss
- Evidence of vomiting and nausea
- Evidence of dehydration
- Abdominal pain
- Licking of lips during abdominal palpation
- Small, hard feces in the colon
- Large, firm, feces distending the colon
- Gas in the intestine/flatulence during examination

Careful abdominal palpation is important in the vomiting cat. The abdomen should be examined in quadrants and the patient carefully observed for evidence of nausea or pain during palpation of each quadrant. Cats noted to lick their lips or swallow during abdominal palpation are likely exhibiting signs of nausea. Evidence of pain during abdominal palpation may include very subtle changes. The patient’s face should be monitored closely for evidence of wincing, blinking or other facial expression changes that could indicate pain. The patient may growl or hiss, although this is rare. Guarding of the abdomen during palpation of the painful quadrant(s) may also be observed.

Abnormal findings during the palpation may include evidence of an enlarged liver, distended stomach, thickened/ropy intestines, free-fluid in the abdomen, masses and/or enlarged mesenteric/intestinal lymph nodes.

**Formulating a differential list**

Establishing a good history in the vomiting patient, as well as performing a thorough physical examination will help refine the list of differential diagnoses. The patient signalment can also provide clues, as can the duration of the vomiting. Acute vomiting in a kitten or young cat can be fairly straight forward, but the cat that has been vomiting intermittently for as long as weeks, months or even years, can be more puzzling.

Adult cats are less likely than kittens to ingest toxins or inappropriate objects, but these still need to be ruled out, particularly in the acute patient.

Obstructive disease causing vomiting in cats can occur as a result of physical obstruction, but may also be a functional issue. Gut motility changes associated with inflammation can, for example, predispose to vomiting.

If the patient is vomiting hairballs, the potential for excess ingestion of hair secondary to a skin condition needs to be addressed. If skin disease is not a contributing factor, then the presence of hair within the vomitus is most likely to be incidental secondary to vomiting for another cause. In rare occasions, hairballs can become obstructive or impeding, and may act as a foreign body causing vomiting (Cannon, 2013).

**Differential diagnoses in the vomiting adult and senior cat**
- Dietary indiscretion: toxic substance, foreign body
- Skin disease: excessive grooming
- Intestinal accident-intussusception
- GIT stasis/altered motility-diet, stress, pain, dehydration, obesity
- Intestinal parasitism
-renal disease
- hyperthyroidism
- pain
• constipation/obstipation
• diabetes mellitus

Within any age group, intestinal parasitism needs to be considered. An apparent lack of exposure based simply on an indoor lifestyle does not rule out intestinal parasitism. The presence of GI parasites can complicate the clinical picture, so the possibility of infection should be eliminated using appropriate empirical therapy.

In the vomiting patient over 7 years of age, diagnoses of renal disease, hyperthyroidism, or diabetes mellitus should be ruled out. Neoplasia will also be higher on the differential list in the vomiting senior cat. Any of these diseases can lead include vomiting as a clinical sign. Appropriate testing will rule these conditions in or out.

References
Symptomatic, targeted and empirical therapies

**Dietary**
The role that dietary allergens may play in the development of gastrointestinal disease is poorly understood (Jergens, 2012). It makes sense that dietary sensitivity or food allergy could play a role in the development of gastrointestinal inflammation. At the very least, the inflamed gut will have unique challenges digesting complex diets. Dietary changes may be beneficial to the patient with gastrointestinal disease. Changing dietary format, such as dry to canned formulations may improve digestion. The use of veterinary formulations that are easy to digest such as Royal Canin Gastrointestinal formulas, Hill’s i/d or PVD EN gastroenteric may reduce or in some cases eliminate active gastrointestinal signs. This does not preclude the need for diagnostic testing, but is beneficial in stabilizing the patient and reducing morbidity.

The selection of true hypoallergenic diets may be indicated when dietary allergens are suspected to play a role in the patient’s disease. The decision to pursue a hypoallergenic diet may be based on diagnostic testing, failure to respond to gastrointestinal diets or evidence of concurrent clinical signs known to be associated with food allergy. Large bowel signs are more often observed in patients with food allergy (Jergens et al, 2012). In addition to gastrointestinal signs, cutaneous disease can be a concurrent clinical sign of food responsive enteropathy (Jergens et al, 2012).

**Parasiticides**
Intestinal parasites need to be considered as a differential diagnosis in any vomiting case. While intestinal parasites may not be causing vomiting, their presence in the gastrointestinal tract may impact digestion and contribute to a worsening of clinical signs. A thorough review of the patient’s deworming history is wise. Empirical deworming, even where fecal testing is unremarkable, is strongly recommended. The clinician should select a broad-spectrum deworming agent to treat the patient for common roundworm, hookworm and tapeworm. These medications should be utilized in a series of treatments in order to eliminate all stages of the parasite life cycle. Recommendations can be found in the Cat healthy protocols http://www.cathealthy.ca/protocols/ and/or Companion Animal Parasite Council (CAPC) website http://www.capcvet.org/capc-recommendations/

**Nausea**
Anti-emetics may be beneficial to the vomiting patient. Drugs with prokinetic effects should be used with caution in cases where intestinal obstruction has not been ruled out. Gastric acid blockers such as ranitidine and omeprazole are less likely to play a beneficial role in feline patients with gastrointestinal disease.

**Appetite stimulants**
Appetite stimulants for loss of appetite or anorexia may be beneficial in improving intake, but in the presence of nausea and gastrointestinal inflammation, these drugs are likely to be of little utility until underlying disease is addressed. Mirtazapine is the author’s drug of choice for stimulating appetite. In some cases, cyproheptadine may also have utility in appetite stimulation.

**Fluid support**
Loss of appetite, vomiting, and/or diarrhea all lead to fluid dehydration. Provision of rehydrating fluids and electrolyte stabilization will reduce morbidity.

**Pain management**
Patients with gastrointestinal disease (GID) are likely to be experiencing pain as a result of or concurrent to their GID. Cats older than 7-9 years of age are likely to have some degree of arthritis, which may impact morbidity. As the signs of pain in the feline patient can be subtle at best, any conditions identified as potentially painful should be treated as such. Gabapentin, buprenorphine, non-steroidal anti-inflammatories (where steroids will not be employed) may all be beneficial in pain management.

**Cobalamin**
It has been recommended that all cats with gastrointestinal disease and a serum cobalamin of <300ng/L should receive parenteral supplementation of cobalamin (Ruaux et al, 2005). The current supplementation dosage recommendations from Texas A&M University (TAMU) are 250 micrograms cobalamin SQ weekly for 6 weeks, then one dose after 30 days, and retesting 30 days after the last dose. Evaluation of the clinical signs and cobalamin levels at this stage in treatment will provide some indication of prognosis with regard to GID. Regularly updated recommendations for the usage and monitoring of cobalamin can be found at http://vetmed.tamu.edu/gilab/research/cobalamin-information

**Antibiotics**
The empirical use of metronidazole or tylosin is sometimes considered (Cook et al, 2008). The success of this approach is hypothesized to occur as a result of either the eradication of an unknown pathogenic bacteria or the alteration of the host response to endogenous bacteria (Cook et al, 2008). Empirical use of antibiotics should generally be avoided, in order to continue responsible use
of these drugs. Where specific infections have been identified, culture and sensitivity patterns should dictate the choice of antibiotic therapy and duration. Follow-up cultures to ensure eradication of infection, should be conducted whenever possible.

**Steroids**

The empirical use of steroids is generally not recommended in any situation in feline medicine. Despite this, it is a frequent occurrence in feline GID patients. Limitations of finances and client willingness to pursue diagnostic biopsy may impact the treatment selection process. Steroid use can be directed as for IBD, but in the absence of a diagnosis, ensuring client understanding of risks and rewards becomes critically important. Steroid usage where a potential diagnosis of small cell or large cell lymphoma have not been ruled out, may decrease patient longevity and reduce choices for therapy should a diagnosis be made later in the course of care. Any steroid usage precludes or limits usefulness of ultrasound or biopsy, as the drugs will change the local inflammatory pattern, thus confounding diagnosis. Clients need to be made aware of this prior to embarking on steroid usage. Where steroids are to be employed, urine culture should be considered prior to drug initiation, in order to rule out occult urinary tract infection.

**Cyclosporine**

Empirical use of cyclosporine is not recommended, but may be considered in cases as noted below.

**Chlorambucil**

Empirical use of chlorambucil is not recommended. In some cases of severe GID where a diagnosis has not been obtained, clinicians may elect to use this chemotherapeutic agent in conjunction with steroid therapy. This is an empirical use of the drug and clients should be made aware of this as well as the potential side effects noted below.

**Inflammatory bowel disease and lymphoma**

**Inflammatory bowel disease**

The non-specific treatments noted above often play a role in therapy for presumed or diagnosed inflammatory bowel disease (IBD) patients. In many instances, some or all of these treatment modalities may have been tried prior to diagnosis with some degree of response.

**Steroids**

Where a diagnosis of inflammatory bowel disease has been made, steroids may be used to in an attempt control, reduce or (rarely) eliminate inflammation. The selection of steroids should be based on the targeted cell type (based on histopathologic diagnosis) and client/patient needs. Dexamethasone may provide a more targeted control of pro-inflammatory T cells and allow reduced dosing frequency. Long-term daily use of this drug is not recommended as the diabetogenic effects may be significant. Prednisolone is the biologically active metabolite of prednisone. In cats, direct administration of prednisolone increases the bioavailable dose, as the feline species is inefficient at converting prednisone to prednisolone. The author does not recommend the use of depomedrol in cats. Budesonide may play a role in some patients, but reduced levels in the circulation will limit the drug’s benefit to patients with concurrent pancreatitis. Cyclosporine may have benefit in some patients, particularly those experiencing notable steroid side effects include a pre-diabetic or diabetic state, as well as steroid-induced hair loss (rare).

**Small cell lymphoma (low grade lymphoma)**

A variety of protocols have been published for the treatment of low-grade alimentary lymphoma (LGL). The majority of these protocols consist of a combination of glucocorticoids and the alkylating agent chlorambucil, usually established on a long term, maintenance schedule of administration. Glucocorticoid treatment can be administered in oral or parenteral forms of prednisolone or dexamethasone. Regardless of the form selected, the initial dosage of glucocorticoid is usually immunosuppressive in nature. For example, one group used 3 mg/kg prednisolone orally every 24 hours initially in treatment of low grade alimentary lymphoma patients (Lingard et al, 2009). The subsequent dose of steroid was tapered down to 1-2 mg/kg body weight every 24-48 hours. Well-established protocols include the concurrent use of chlorambucil at a dose of 15-20 mg/m² orally. The drug may be administered every 1-3 days initially or long-term. After an initial period of frequent administration, the administration frequency may be decreased to 15-20 mg/m² orally every 2 weeks (Stein et al, 2010). High dose, pulse therapy with chlorambucil may also be recommended (Lingard et al, 2009). In this study, cats were treated with 15 mg/m² oral chlorambucil every 24 hours for 4 days every 3 weeks. The use of oral prednisolone and high dose pulse chlorambucil is thought to be effective in producing durable remissions in feline low-grade alimentary lymphoma patients (Lingard et al, 2009). Side effects observed secondary to steroid use may include increased drinking, urination and increased appetite. The main potential side effect of chlorambucil is myelosuppression. Anemia, leukopenia, thrombocytopenia, vomiting and diarrhea may be observed (Plumb, 2011). Withdrawal of the drug is recommended in these situations.

Cyclosporine may play a role in treatment of patients experiencing significant side effects from steroid usage. However, it is currently not recommended in the literature, nor has it been evaluated, as a primary line of treatment for LGL.

**Large cell lymphoma (high grade lymphoma)**

The diagnosis of lymphoma by biopsy is most critical in order to rule out this more aggressive, lymphoblastic form of the disease. Empirical treatment of the cat for IBD or LGL may lead to incorrect therapy of a very aggressive form of the disease. This reduces prognosis significantly. Treatment of high-grade lymphoma (HGL) in cats requires the use of chemotherapeutic protocols including...
drugs such as doxorubicin. Several protocols have been published. In some cases, referral is the best option for treatment of HGL, as the use of chemotherapeutics is dangerous and requires special handling as well as aggressive patient monitoring. Treatment of feline HGL with multi-agent chemotherapy protocols has been reported to generate a median remission of 140 to 213 days (Mahoney et al, 1995; Zwahlen et al 1998).

References
The list of differential diagnoses in the vomiting adult and senior feline patient is long and complex (See Lecture 1: The Vomiting Cat. A quirky feline trait or a sign of disease). Identifying the cause(s) of vomiting as early as possible will reduce long-term negative effects. Following physical examination, a decision needs to be made about the pursuit of diagnostic testing. Depending on the severity and duration of disease, the client and veterinarian may elect to either first pursue a minimum database (MDB) or they may elect to pursue more extensive tests.

The clinical chemistry will be beneficial in identifying the presence of primary hepatic disease, renal disease or diabetes mellitus. The results will also provide indication of changes secondary to vomiting, such as electrolyte disturbances.

A complete blood count (CBC) may reveal shifts in white cell parameters suggestive of infection, inflammation, parasitism or allergy. Anemia may be present as a result of gastrointestinal blood loss. Hematology changes should always be investigated further by the careful examination of a blood smear. The blood smear should be evaluated by an experienced veterinarian or veterinary technician. Microscopic cellular changes can provide significant information about the patient’s disease process above and beyond what the sheer numbers may reveal.

A urinalysis is a critical component of the minimum database. It should not be overlooked. Despite the apparent lack of connection with gastrointestinal disease (GID) signs, the urine may reveal a number of changes that are relevant to the therapeutic plan. The urine may reveal evidence of bilirubin, which can indicate liver and/or gall bladder disease. The presence of glucose and/or ketones in the sample may assist in a diagnosis of diabetes mellitus. Even in the absence of GID-related changes in the urine, a concurrent problem with urinary tract issues such as crystals or infection will impact the therapeutic plans.

The patient’s Feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) status should be determined. Consideration for risk factors associated with acquiring these viruses, as well as previous tests, will determine whether previous negative testing is adequate. Feline leukemia virus is a known cause of lymphoma in the feline patient, and used to account for a significant portion of these diagnoses (Cotter et al, 2011). However, with the introduction of vaccination against FeLV, there has been a shift in the type of intestinal lymphoma most common in cats (Cotter et al, 2011; Louwerens et al, 2005). This shift does not change the value of knowing the patient’s retroviral status in this or any other disease state, as disease management will be impacted by the presence of retrovirus infection.

Fecal testing for common parasites can be revealing, but the lack of a positive fecal test does not rule out infection. Fecals tests are notoriously insensitive, making the argument for empirical deworming in most (all) cases of GID.

Beyond the minimum database
Gastrointestinal profiles are an important part of the vomiting cat ‘workup’. In addition to the minimum database, diagnostic testing will be enhanced by blood testing for cobalamin (B12), folate, feline specific pancreatic lipase (sFPL) and trypsin-like immunoreactivity (TLI)

**Cobalamin (B12)**
Cobalamin is released from food in the stomach, and bound to haptocorrin (Non specific binding, low pH environment). Cobalamin is released from haptocorrin in the duodenum and bound to intrinsic factor (IF; specific binding, neutral pH environment). In the feline species, IF is a product of the pancreas. This complex binds to specific receptors in the ileal enterocytes and enters the portal circulation. Without IF, cobalamin cannot enter the bloodstream and passes out in the feces.

Low serum cobalamin is a common finding in cats with chronic intestinal diseases, pancreatitic and liver diseases (Ruaux et al; 2005, Simpson et al, 2001; Xenoulis et al, 2008). Cobalamin has an important role in the metabolism of sulfur containing amino acids, and is a factor in lipid and DNA synthesis (Ruaux et al, 2005; Reed et al, 2007). When cobalamin is deficient, actively dividing tissues such as bone marrow and enterocytes, are negatively affected (Xenoulis et al, 2005). Cobalamin deficiencies associated with GID can lead to further health compromise (Xenoulis et al, 2008).

In a study by Simpson et al (2001), cats found to have low cobalamin had been presented with clinical signs such as weight loss, diarrhea, vomiting, anorexia and thickened intestines. Definitive diagnoses in these patients included IBD, intestinal lymphoma, cholangihepatitis, cholangitis and pancreatic inflammation. Cats with intestinal lymphoma were found to have particularly low levels of cobalamin. In total 49/80 cats with GID had subnormal serum concentrations of cobalamin. Many cats with GID and low cobalamin had simultaneous presence of disease in the intestine, pancreas or hepatobiliary system, making identification of the cause of low cobalamin difficult.
Folate
Subnormal concentrations of folate were found in 3 of 5 of the cats with intestinal lymphoma and subnormal cobalamin levels (Simpson et al, 2001). In general, subnormal concentrations of folate can be considered to be indicative of chronic GID. Elevations in folate may be detected. These changes may be related to disturbances in intestinal bacterial populations, although the phenomenon of small intestinal bacterial overgrowth (SIBO) has not been described in cats (Hall et al, 2011).

Feline specific pancreatic lipase (sPL)
Assessment of sPL levels may facilitate the diagnosis of pancreatitis in GID patients. Diagnosis of pancreatitis is helpful in case management. Feline pancreatic lipase immunoreactivity has a reported sensitivity of 54-100% and a specificity of 67-91% (Cosford et al, 2010). In a study evaluating FPLI in comparison to conventional tests, overall sensitivity was 67% and the overall specificity was 91% (Forman et al, 2004). This latter test is now considered a useful serologic marker for the diagnosis of pancreatic disease in cats (Cosford et al, 2010, Xenoulis et al, 2008). The variable sensitivity and subsequent difficulty in ruling out pancreatitis with a normal pancreatic lipase requires consideration when interpreting test results. A normal sPL/FPLI does not rule out pancreatitis.

Trypsin-like immunoreactivity
(TLI) may have some benefit in the diagnosis of pancreatitis in GID cats, as well as the diagnosis of exocrine pancreatic insufficiency (EPI). Trypsin-like immunoreactivity has a reported sensitivity of 33-86% and a specificity of 75% (Cosford et al, 2010). Studies have failed to demonstrate a positive correlation of the concentration of this marker with a diagnosis of feline pancreatitis (Forman et al, 2004). Elevations in TLI may be associated with pancreatitis. Decreases in TLI, concurrent with suggestive clinical signs, are likely to be associated with a diagnosis of EPI.

Imaging
Radiography
Radiography is beneficial in elimination of some differential diagnoses in the vomiting cat. Thoracic radiographs should not be ruled out as necessary. In older cats, the presence of neoplastic lesions within the thorax may be the only identifiable source of vomiting. Abdominal radiographs will be beneficial in identifying some foreign bodies, masses, intestinal accidents, and other gastrointestinal tract changes. Abdominal radiography also offers the opportunity to identify urinary tract disease as a source of pain and discomfort. Close evaluation of skeletal structures may also provide information about the presence of painful arthritic lesions and/or spondylosis.

Ultrasound
Ultrasoneography is quickly becoming a common diagnostic tool in general practice. Ultrasound machines are now more affordable than ever. Successful ultrasound does require training, skill and experience. Use in general practice should be guided by the availability of a practitioner skilled in performing ultrasounds.

Endoscopy and exploratory laparotomy: Diagnostic biopsy
Where clinical signs and laboratory studies are strongly indicative of disease such as IBD, lymphoma (diffuse neoplasia), discrete neoplasia, hepatitis/cholangitis/cholangiohepatitis and/or pancreatitis, biopsy is warranted. The decision to pursue endoscopy versus full abdominal exploratory may be impacted by the findings, the relative invasiveness of each procedure and cost. The clinician will need to carefully evaluate what biopsies are needed. Endoscopy limits biopsy of the upper GI tract to the stomach, duodenum, jejunum and proximal ileum (at best). As intramural lymphoma is often found in the ileum, a diagnosis via endoscopy may not be possible. Endoscopy limits the biopsy to primarily mucosal layers and does not provide full thickness biopsy. Lower bowel endoscopy may permit biopsy of the distal ileum via the ileoceccolic junction (ICCJ), but this can be difficult to pass with subsequent difficulties in obtaining quality biopsies. Exploratory surgery permits full visual assessment of all intra abdominal organs, biopsy of extraintestinal tissues (liver, pancreas, lymph nodes etc) and full thickness intestinal biopsy (Kleinschmidt et al, 2010).

References


Beyond the Bloodwork: Diagnostics for Liver Disease
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The liver is a vital organ necessary for many functions in the body, including nutrient metabolism and detoxification of various substances. As a result, liver dysfunction due to many different etiologies can be potentially life threatening. The powerful regenerative capability of the liver, however, makes early disease detection critical when considering a favorable long term prognosis.

Liver diseases can be divided into two broad categories: hepatocellular injury and hepatocellular dysfunction / failure. Clinical signs may overlap between these two groupings, and in general will typically be non-specific with liver disease. These categories can frequently be differentiated based on initial laboratory work, including a serum chemistry panel, complete blood count, and urinalysis. More often, however, baseline blood work will be suggestive of generalized liver disease but more information will be required to make informed decisions on treatment and prognosis.

Serum biochemistry profile
ALT, AST, ALP, and GGT are all liver enzymes that can be elevated with liver injury or dysfunction. The pattern of elevation can be helpful in determining the source of the injury. For example, if ALT and AST are substantially higher than ALP then damage to hepatobiliary cells should be suspected. If, however, GGT and ALP are more elevated compared to ALT then biliary obstruction or cell membrane damage should be suspected. Elevated total bilirubin would also be expected with the latter (Center SA 2007). With severe liver disease (i.e. chronic active hepatitis, fibrosis, cirrhosis, toxicity-induced failure, etc.) all four of the previously mentioned enzymes may be markedly elevated due to multifactorial cellular injury. Caution should be taken when evaluating a case where ALT and AST are the only elevated liver enzymes, as these enzymes are found in myocytes and muscle injury will cause their release.

In addition to liver injury and membrane damage, late-stage hepatocellular dysfunction may also be recognized on a biochemistry profile, characterized by a deficiency in products made by the liver. This may include hypoglycemia, hypcholesterolemia, and hypoalbuminemia. These results remain non-specific, as various gastrointestinal and endocrine diseases will have similar results. Normal concentrations of liver-specific enzymes cannot be used to rule out liver disease in these cases either, as with severe liver failure there may be a deficiency of hepatocytes to even synthesize and release the enzymes. An elevation in serum total bilirubin can also be non-specific, as this can be seen with hepatocellular dysfunction (intra-hepatic cholestasis), extrahepatic biliary obstruction, or even hemolysis unrelated to liver disease.

Complete blood count
Red blood cell microcytosis can be seen in some dogs with liver disease, most frequently being seen with a portosystemic shunt. With acute cholangiohepatitis, neutrophilia with or without a left shift may be present. Thrombocytopenia has been reported with late stage liver failure due to a diminished concentration of thrombopoetin (Webster CRL and Cooper JC 2014).

Urinalysis
Isosthenuria may be present due to medullary washout with liver failure secondary to decreased urea nitrogen production. Overflow of bilirubin will also be reflected in the urine, characterized by bilirubinuria. This can be a normal finding in low concentrations, especially in male dogs.

If initial blood work is suggestive of liver disease, additional diagnostics should be pursued in a logical and step-wise fashion.

Blood tests for liver function
When liver function is greatly diminished, many toxins that are ordinarily filtered remain in circulation. The most well documented and easily measured is ammonia. A portosystemic shunt is the most common cause of hyperammonemia, however this can be seen with other diseases that lead to diminished liver function including cirrhosis and fibrosis as well. Measurement of ammonia has historically been challenging due to instability in serum over time, however recent advancements in laboratory technology now allow in-house testing. This now makes serum ammonia concentration testing a reasonable non-invasive first step in the evaluation of liver function. Ammonia testing should be considered especially when the patient has clinical signs suggestive of hepatic encephalopathy.

When diagnosing a portosystemic shunt, fasting serum ammonia concentration has a sensitivity of 85% in dogs. Serum bile acid testing has even higher sensitivity at 93% (Ruland K et al 2010). These results from 2010 contrast a 2006 study where fasting ammonia concentration was found to be both more sensitive (100% vs. 92%) and specific (89% vs. 68%) than serum bile acids for detecting a portovascular anomaly (Gerritzen-Bruning MJ et al 2006). There are many cases where liver function is compromised but not to the extent of hepatic encephalopathy and resulting hyperammonemia, such as with mid-stage chronic active hepatitis. In these cases serum bile acid testing is the preferred first-choice. This test consists of a fasting blood sample followed by feeding the patient
and then a 2-hour post-meal blood draw. Be aware that mild elevations may be considered normal, especially in some breeds (ie. Maltese) (Tisdall PL et al 1995).

Other laboratory tests
Most coagulation factors are synthesized in the liver, making the measurement of prothrombin time and partial thromboplastin time useful cage-side tests to evaluate liver function. Since clotting times are frequently elevated in dogs with liver dysfunction, this is useful information prior to obtaining liver aspirates or biopsies as well.

Plasma protein C can be used to help differentiate portosystemic shunts from microvascular dysplasia, when these are the top differentials for liver dysfunction (protein C was <70% in 88% of dogs with a portosystemic shunt) (Toulza O et al 2006).

Diagnostic imaging
Abdominal radiographs can be a sensitive test for evaluating liver size in dogs, with microhepatica being associated with a negative gastric axis on the right lateral view while hepatomegaly presents with a near horizontal gastric axis on the same view.

Abdominal ultrasonography by an experienced ultrasonographer is a useful test for evaluating liver shape and size and investigating echotexture. Size and number of visible intrahepatic vessels, intra- and extra-hepatic bile duct size and shape, presence of single or multifocal nodules or masses, etc. are just a few of the potential abnormalities that can be identified using ultrasound. Surgical planning can be accomplished using ultrasound, especially when finding a single mass versus identifying diffuse infiltrative disease. Mass location, however, can be challenging to definitively identify. A recent study showed only 52% success in correctly locating the lobe affected by a single hepatic mass (Wormser C et al 2016). This should be taken in to account when planning a surgery for a liver lobectomy. Ultrasound is the test of choice for some clinicians for identifying a portosystemic shunt, however sensitivity can be as low as 75% (Berent A and Weiss C 2010). If an anomalous vessel is not directly identified on ultrasound, ancillary findings including bilateral renomegaly and urolithiasis as well as microhepatica and inadequate intrahepatic vasculature can help increase the degree of suspicion for a shunt (d’Anjou MA et al 2004).

There are some liver diseases that have characteristic findings on ultrasound, including a confirmed anomalous vessel. Many diseases, however, have non-specific and sometimes even minimal to no abnormalities seen on ultrasound. A recent study found that 64% of livers that had no ultrasonographic abnormalities had some degree of pathology when biopsies were taken, including moderate to severe fibrosis (Kemp SD et al 2013).

Nuclear scintigraphy can be used as a highly sensitive test for portovascular anomalies, however availability is limited for most practitioners. Computed tomography (CT) is becoming more prevalent and is available in most specialty practices. This test is sensitive for even small liver masses (ie. primary or metastastic) and with angiography can be diagnostic for portosystemic shunts.

Liver sampling
A fine needle aspiration is the least invasive method of sampling the liver. This can frequently be done with an awake or lightly sedated patient. Use of a 22-gauge needle with a 6cc syringe is the author’s preference. Ultrasound-guidance can help target specific lesions and avoid important vasculature. Cytology results should be interpreted carefully, however, as diagnostic accuracy is marginal. Correlation between cytology and histopathology of the liver is reported to occur only 30% of the time (17/56 dogs) (Wang KY et al 2004). Vascular hepatopatholysis was the diagnosis with the highest degree of accuracy, however this was also the most commonly misdiagnosed disease with cytology. Hepatocellular inflammation was incorrectly identified 75% of the time.

While comparisons can be made, as above, to cytology and histopathology, not all biopsy samples are equal either. There are many ways to obtain a liver biopsy, including some of the following: ultrasound-guided tru-cut biopsy, laparoscopic biopsy, punch biopsy, guillotine method, liver lobectomy, etc. Clinician preference and confidence in various techniques plays a significant role in which method is used, as well as the suspected underlying disease. When a single mass lesion is detected a liver lobectomy accomplishing an excisional biopsy may be recommended, whereas a collection of laparoscopic or surgical punch biopsies may be indicated with diffuse infiltrative disease. Equipment availability will also play a role, as laparoscopic capabilities are not available in all practices.

When an abdominal ultrasound identifies diffuse liver disease it is preferable to biopsy multiple liver lobes if possible. Even though the external appearance of the liver may be similar diffusely the histopathology may vary between lobes; odds of obtaining the correct diagnosis increases with each additional liver lobe that is sampled (Kemp SD et al 2015). Method of biopsy has not correlated well with an increased odds of diagnosing the disease, provided at least 3 portal triads are sampled from each lobe (Kemp SD et al 2015).

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Colorectal Disease in Dogs
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The large bowel is an uncommon site of disease in the dog, but when present the clinical signs can be severe and often challenging to manage. Constipation and even obstipation may be present with some forms of colonic disease (i.e. megacolon), but large bowel diarrhea is more commonly found when the colon is affected. Clinical signs of large bowel diarrhea are often distinctive and include hematochezia, tenesmus, increased frequency of defecation, and pain during defecation. While these signs are characteristic of disease isolated to the colon, frequently colitis will be seen in conjunction with small bowel disease and clinical signs will be less specific, including weight loss, abdominal pain, large volume of stool, etc.

<table>
<thead>
<tr>
<th>Clinical signs</th>
<th>Small bowel</th>
<th>Large bowel</th>
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<tbody>
<tr>
<td>Weight loss</td>
<td>Possible</td>
<td>Rarely</td>
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<tr>
<td>Frequency of stools</td>
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<td>Increased</td>
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<tr>
<td>Hematochezia</td>
<td>Rare</td>
<td>Possible</td>
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<tr>
<td>Tenesmus</td>
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<td>Likely</td>
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<tr>
<td>Melena</td>
<td>Possible</td>
<td>Rare</td>
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**Diagnostics for colorectal disease**

A detailed history and physical examination will often lead to a high suspicion of colon disease, especially when following the above table if diarrhea is present. Once the pathology has been isolated to the colon, diagnostics should be performed in a step-wise fashion to try and identify the underlying cause. A serum biochemistry profile and complete blood count should be performed but are rarely abnormal with primary colonic disease. One exception is chronic Histoplasmosis, which may present with hypoalbuminemia and hyperglobulinemia. Dogs with chronic colitis, especially older females, will be at an increased risk for an ascending urinary tract infection so a urinalysis should also be included.

A fecal flotation and Giardia ELISA should be tested on all dogs with suspected colitis. Giardia and Trichuris vulpis are two widespread parasitic diseases that can be associated with large bowel diarrhea. A fecal cytology can be helpful especially when trying to identify overgrowth of a single population of bacteria, particularly clostridium spores. If there is clinical suspicion for Histoplasma capsulatum, a rectal cytology should be performed. This is a simple test that can be performed in-house during the physical examination. It is important, however, to not mistake this test for a fecal cytology. Diagnostic accuracy of the rectal scrape depends on penetrating deep to the mucosal layer using either a finger or cytology brush; the organism is unlikely to appear in just mucus or stool. When examining a rectal scrape cytology, neutrophils or large mononuclear cells should be seen indicating that an adequate depth has been achieved. The organisms will be found in clusters within macrophages in most cases. If this test is unrewarding but Histoplasmosis is still suspected then a urine Histoplasma antigen titer can be tested.

Abdominal radiographs are often non-specific and are not sensitive for colonic disease; however mucosal irregularities may be present with a severe nodular colitis. Sublumbar lymphadenomegaly may be present with severe Histoplasmosis or neoplasia. Abdominal radiographs can be helpful identifying megacolon or severe obstipation.

Abdominal ultrasound is more sensitive for detecting colonic wall thickening as well as evaluating the wall layering of the colon, however this modality also has its limitations with the colon. There is a section of colon and rectum within the pelvic canal that cannot be imagined with ultrasound. Additionally, the colon is frequently stool or gas distented, both of which are obstacles to ultrasound penetration. An MRI or CT scan may be alternative methods of advanced imaging that can better evaluate the entire length of the colon and do not suffer from the effect of gas or fecal artifact.

If chronic large bowel diarrhea is present secondary to ulcerative colitis, inflammatory bowel disease, etc. then it is possible all of the above diagnostic tests are within normal limits. Surgical full thickness colon biopsies carry increased inherent risks due to the thin wall and high intraluminal bacterial load, making this a less desirable method of obtaining samples. As a result, colonoscopy is the preferred method of biopsy procurement in dogs with chronic large bowel diarrhea. Adequate colon preparation is vital for thorough investigation of the entire colonic mucosa. This can be accomplished using oral solutions such as GoLytely (50mL/kg PO once to twice the day prior to the procedure) and / or a series of warm water enemas given the day of the procedure. A 36 hour minimum fast is usually recommended. Colonoscopy allows thorough investigation of the entire colonic mucosa, including segments unavailable to ultrasound, as well as allowing a safe method of taking multiple representative biopsy samples. Entry in to the ileum and cecum may also be possible, depending on the level of experience of the endoscopist.
Chronic lymphoplasmacytic colitis
Commonly referred to as inflammatory bowel disease or steroid-responsive diarrhea, lymphoplasmacytic colitis may be an isolated disease or may be one component of diffuse gastrointestinal inflammation. This condition will often contribute to mixed-bowel diarrhea. An underlying cause is infrequently identified, however many theories exist including food allergies. If the patient is clinically stable besides the diarrhea, a diagnosis of exclusion should be initiated. A food trial using a hypoallergenic diet (novel protein, hydrolyzed protein, etc.) should be fed exclusively for a minimum of 2-3 weeks. If there is no clinical improvement then food allergy is considered less likely. An antibiotic trial should then be instituted (metronidazole or tylosin generally). If clinical signs persist, then biopsies should be obtained. In dogs with suspicion of mixed bowel disease, an upper GI endoscopy is often performed just prior to the colonoscopy in order to obtain a wide selection of biopsies. Diagnostic accuracy will be maximized with more good quality biopsy samples representing all aspect of the gastrointestinal tract (Willard MD et al 2001).

If histopathology confirms IBD, then localization of the disease will dictate the therapeutic plan. When inflammation is severe and widespread including small and large intestine, immunosuppression may be indicated. Prednisone is typically the first-line medication for this, with secondary drugs including Cyclosporine A, azathioprine, and more recently chlorambucil. Sulfasalazine as monotherapy is indicated if inflammation is restricted to the colon.

Canine ulcerative colitis
The Boxer is the most common breed affected by this disease. It has been categorized as a form of aggressive IBD and occurs more often in young dogs. Unlike traditional IBD, or steroid-responsive diarrhea, ulcerative colitis is typically non-responsive to corticosteroid use. Since this disease affects the aforementioned breeds almost exclusively, there is a presumed genetic predisposition. Until 10-15 years ago this disease was considered to be a highly refractory form of severe IBD and patient morbidity and mortality were high. In recent years, however, a connection with intracellular E.coli has been identified (Mansfield CS et al 2009). Now that a bacterial component has been proven, both morbidity and mortality have decreased significantly. Oral enrofloxacin has been successful in treating many such cases, however recently fluoroquinolone resistance has been determined (Craven M et al 2010). In addition to the Boxer dog, a series of cases with a similar presentation including E.coli identification has been reported in the French Bulldog (Manchester AC et al 2013).

Colorectal neoplasia
Hematochezia, tenesmus, and constipation are some of the more commonly reported clinical signs associated with neoplasia in the colon. The most common tumors seen include adenocarcinoma, lymphoma, and GI stromal tumors (ie. leiomyosarcoma). Positive contrast enema with radiographs, abdominal ultrasound, digital palpation, and endoscopy are all ways that colon and rectal masses can be identified. Histopathology is the only way to definitively determine the diagnosis, which makes colonoscopy an essential tool to reduce morbidity and still make a diagnosis. Prognosis for benign adenomas and stromal tumors is favorable with surgical or endoscopic excision, while malignant neoplastic diseases including lymphosarcoma and adenocarcinoma carry a guarded prognosis even with excision.

Location of a focal mass will dictate the surgical approach and morbidity. A mass in the ascending or transverse colon, provided it is not immediately adjacent to the ileocolic junction, makes surgical excision more routine. A mass in the distal descending colon or the rectum may be much more challenging, especially if it is located within the pelvic canal. A distal rectal mass may be amenable to a rectal pull through surgical approach. This procedure carries a favorable outcome for benign rectal polyps, however there is a high complication rate (78%) with malignant rectal masses, with fecal incontinence being the most common (Nucci DJ et al 2014).

Constipation / obstipation
This is a clinical phenomenon that is more commonly seen in cats than dogs, secondary to colonic hypomotility. When a dog is presented with severe constipation or obstipation, a primary underlying disease should be investigated including outflow obstruction (rectal or colonic mass, prostatomegaly, etc.) or sources of pain while defecating (lumbosacral disease, perianal fistulas, etc.). Inflammatory disorders are more likely to contribute to decreased water absorption and subsequent diarrhea as opposed to constipation.

If idiopathic constipation is identified, one or more warm water enemas may be all that is needed to help relieve the obstruction. If this becomes a recurring problem then special attention should be paid to the patient’s metabolic status, level of hydration, and diet. A stool softener such as lactulose or a moderate fiber diet may help to control the problem. If colonic hypomotility is suspected, using a prokinetic agent such as cisapride may be beneficial.

Rectal prolapse
A rectal prolapse is an easily identifiable abnormality found on physical examination. In most cases there is a history of tenesmus or other large bowel signs that over time will weaken the anal sphincter making prolapse more likely. Rectal or colonic intussusception
will look nearly identical to prolapse, and can be differentiated by probing the lateral aspect of the tissue. If the probe advances easily beyond the anal sphincter then an intussusception is present and not a rectal prolapse.

Identification and treatment of the underlying disease (frequently intestinal parasites) should be done prior to surgical correction. A purse string suture can be placed after reduction of the prolapse along with a stool softener, however if the dog continues to have tenesmus there is a high likelihood of prolapse recurrence. If the rectal prolapse persists once the underlying pathology has resolved surgical intervention may be indicated. A colopexy (securing the serosal surface of the colon to the left caudal aspect of the abdomen, helps to provide tension to the descending colon and rectum and will help to prevent further prolapse.

References
Pancreatitis is a commonly diagnosed condition that affects dogs of all ages. Clinical signs can vary greatly depending on both the chronicity and severity of disease. While in some cases pancreatitis is a straightforward diagnosis, the presenting complaints are often vague or non-specific, diagnostic tests may be misleading, and concurrent illnesses may complicate the clinical picture. This is made even more difficult by the lack of a single gold standard test.

**Diagnosing pancreatitis**

- **Clinical signs**
  - Mild pancreatitis: Decreased appetite, lethargy, loose stools, etc.
  - Severe pancreatitis: Vomiting, diarrhea, abdominal pain, lethargy, fever, hypovolemic shock

- The initial diagnostic testing should help to begin ruling out other illnesses with similar presenting complaints, including gastroenteritis, acute renal failure, gastrointestinal obstruction, cholangiohepatitis, etc.
  - A thorough medical history may be the most important step in making a diagnosis. Questions should focus on whether there have been any changes in diet, has the dog eaten anything unusual lately, is he taking any medications, are there any concurrent illnesses, etc.
  - Physical examination: Is the patient clinically dehydrated, is there abdominal pain (focal vs. non-specific), is nausea present, how do stools look on rectal examination.
    - Will guide the clinician towards a working diagnosis as well as help to start formulating a treatment plan.
  - Baseline blood work: When a dog is presented for evaluation of vague, non-specific clinical signs initial lab work should include a minimum of a serum chemistry panel, complete blood count, and urinalysis. Abnormalities that may be seen directly related to or secondary to pancreatitis may include:
    - Inflammatory leukogram (mild to marked)
    - Non-regenerative anemia
    - Thrombocytopenia (with severe necrotizing pancreatitis, leading to DIC)
    - Azotemia (pre-renal or renal)
    - Cholestasis (secondary to post-hepatic biliary obstruction from inflamed pancreas)
    - Hypoalbuminemia (negative acute phase protein)
    - Elevated amylase and lipase (variable)
    - Metabolic acidosis secondary to azotemia, poor perfusion, etc.
    - Isosthenuria
  - If vomiting and abdominal pain are presenting complaints, then 2-view abdominal radiographs should be included in the initial diagnostic testing. While radiographic changes are often non-specific for pancreatitis this can be a helpful measure to rule out other differentials including intestinal obstruction.

If the above test results remain suggestive of pancreatitis, then more specific testing should be pursued. Since amylase and lipase have poor sensitivity for canine pancreatitis (32-73% and 42-69%, respectively) (Hess RS et al 1998) as well as low specificity (~50%) (Steiner J 2008), additional testing is often necessary to confirm or to rule out the diagnosis. The SPEC cPL (pancreatic lipase assay) is the most accurate confirmatory test for pancreatitis, with a sensitivity of 87-94% and specificity of 81-88% (McCord K et al 2012). A newer test (Precision PSL) has similar accuracy to the SPEC cPL (Kook PH et al 2014). The SNAP cPL has similar sensitivity and specificity to the SPEC cPL and Precision PSL, but has the advantage of being cage-side. Specificity of the SPEC cPL will increase to as high as 88% when a more stringent cut-off of 400ug/L is used, which makes it a preferred test when clinical signs are suggestive of pancreatitis (McCord K et al 2012). When used in union, the SNAP cPL can be an effective and rapid screening tool, however for a more definitive diagnosis (and to obtain a quantitative result) the SPEC cPL should be the follow-up test.

Abdominal ultrasonography is a useful diagnostic test for pancreatitis in the hands of an experienced ultrasonographer. Possibly more so than with any other test for pancreatitis, this is a great deal of user variability with ultrasound which makes results difficult to interpret. Challenges in imaging the pancreas are multifactorial, including:

- Some animals can be challenging to image due to body shape (deep-chested dogs), making even identification of the pancreas.
- Patients with severe pancreatitis will frequently have severe abdominal pain which makes accurate imaging difficult without adequate pain management and/or sedation.
Ultrasound machines vary greatly in quality. Older machines will deliver grainy images and make detailed imaging nearly impossible, especially when trying to evaluate the pancreas.

User inexperience is the biggest road block to obtaining an image of the pancreas and being able to interpret the finding. Even in the hands of an experienced radiologist making a diagnosis of pancreatitis can be demanding. In some cases ultrasonographic changes lag behind clinical signs, and vice versa. Sensitivity of making a diagnosis with ultrasound has at best been shown to be 70% (Steiner J 2010).

The gold standard diagnostic test to confirm pancreatitis remains histopathology, but this is widely considered an unnecessary test that can lead to increased morbidity and mortality. Placing a hemodynamically compromised patient under general anesthesia and manipulating the pancreas may be indicated if there is acute bile duct obstruction or neoplasia is suspected, but a presumptive diagnosis can often be made prior to going to surgery and rarely does a patient benefit clinically from this procedure.

**Treatment of pancreatitis**

Fluid therapy is the most important management strategy in dogs with pancreatitis. Early and aggressive use of IV fluids can be the difference between a patient surviving or not, however caution should be taken to avoid over-use of crystalloids leading to fluid overload. While dogs with mild pancreatitis may thrive on crystalloid therapy alone, patients with more severe pancreatitis often require a more diverse plan. Hypoalbuminemia, vasculitis, severe pain, and hypotension can all be components of pancreatitis requiring a tailored fluid plan including the following:

- **Colloid therapy** (ie. Vetstarch) in the form of boluses initially to raise BP as well as a continuous infusion.
- **Vasopressor therapy** such as a dopamine CRI, to help raise blood pressure (once rehydration has been completed).
- **Continuous infusion of pain management** (ie. fentanyl CRI) either administered separately through a syringe pump or combined in a bag of crystalloids.
- **Ongoing anti-emetic therapy** in the form of a CRI (ie. metoclopramide).
- **Other targeted colloids**, including human albumin and fresh frozen plasma. The success / failure of these products with acute pancreatitis has not been confirmed, and there are risks especially with albumin, but severely critical cases may require this level of aggressive management.

Additional medical therapy is dictated by the patient’s ongoing clinical signs and severity of clinical illness, including the use of other antiemetics, intravenous antacids, alternative pain control, supplemental oxygen therapy, etc.

Management of chronic pancreatitis can be frustrating, especially if the only abnormality is in the blood work. In asymptomatic dogs with persistently elevated cPL, for example, a low fat diet may be all that is indicated. If the dog is symptomatic (including inappetance, mild chronic abdominal pain, intermittent vomiting, etc.) then supportive care including antiemetics, appetite stimulants, antacid therapy, etc. may be necessary during supposed flare-ups. If these therapies are not effective, an alternative diagnosis should be suspected and more testing may be indicated (ie. intestinal or liver biopsies, gall bladder culture, etc.).

Pancreatitis can be a challenging condition to both diagnose and manage, especially when 24 hour care is not available. Learning how to interpret the available diagnostic tests (including having a solid understanding of their pitfalls and inaccuracies) and implementing early and, if necessary, aggressive therapy will help to improve the outcome of your patients with pancreatitis.

**References**


Diagnostic Approach to Canine Chronic Enteropathy
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A “chronic” enteropathy (CE) is typically defined as gastrointestinal disease that has been present for at least two weeks. This may be in the form of two solid weeks of persistent diarrhea, or intermittent loose stools for many months that fluctuates between normal and liquid. A chronic enteropathy is a description of a symptom, and as such there are many underlying illnesses that can be responsible for these clinical signs. The diagnostic approach will have some fundamental differences compared to a dog who is presented with more acute clinical signs.

A detailed history is a crucial first step towards trying to identify an underlying disease process. In many cases a great deal of information can be gathered simply by taking a complete medical history. The differential list can be narrowed and appropriate tests can be pursued, while other tests may be considered unnecessary, following a thorough history on a patient and getting a better understanding of how long the signs have been occurring and to what degree the patient is affected (vomiting, regurgitation, diarrhea, weight loss, inappetance, etc.).

The value of a thorough physical examination cannot be overstated in dogs with CE. While most causes of CE will have non-specific changes seen on the physical examination (including normal to decreased body condition score, possible abdominal pain, nausea, etc.), there are some exam findings more suggestive of certain GI diseases. Dogs with weight loss and a thickened colonic mucosa may be more likely to have an infectious cause of diarrhea such as Histoplasmosis. Thickened small intestines on abdominal palpation, or suspected mesenteric lymphadenomegaly, may be indicative of GI lymphoma. While rare, a sliding intussusception can occur as a consequence of a severe CE; this can also be detected on PE. While these specific changes are less common, there is still a substantial amount of information that can be gathered from a physical examination. Urgency of diagnostic tests is another important take-away from the physical examination. A dog with a protein losing enteropathy that has muffled lung sounds and ascites and is dehydrated is going to need more intensive medical management and more aggressive diagnostic testing to find an underlying cause compared to a dog with mild intermittent diarrhea and no other clinical signs.

Baseline blood work will generally follow the history and physical examination. Many underlying metabolic diseases that can cause chronic gastrointestinal disease can be identified by reviewing these basic tests, including chronic renal failure, pancreatitis, liver failure, chronic hepatitis, pyelonephritis, and others. In the absence of abnormalities on the initial lab work, further investigation will be needed.

If the signalment and history are suggestive of an infectious cause (young dog, history of shelter experience, extensive travel history) then infectious disease testing should be prioritized. While an uncommon cause of chronic diarrhea, a viral disease such as Parvovirus is easy to rule out with ELISA testing. PCR testing is available for other viral diseases such as canine coronavirus and distemper virus. In addition to these viral diseases, PCR testing is also available for other infectious diseases that may be more likely to be associated with chronic gastrointestinal diseases, such as Salmonella, Clostridium, Giardia, and Campylobacter. A routine fecal evaluation for ova and parasites should also not be forgotten in the initial work-up for all dogs with chronic diarrhea. Depending on the geographical location of the patient, Histoplasmosis should also be considered for dogs with chronic enteropathy, especially with predominantly lower GI clinical signs. A rectal scrape for cytology can be performed (different from a fecal cytology) to look for organisms, as can a fine needle aspirate of enlarged lymph nodes or other abnormal abdominal organs. If these tests are negative and Histoplasmosis is still suspected, confirmation can be made via an antigen detection test on urine.

There are many common non-infectious causes of CE in dogs, and thus a diagnostic plan should developed beyond the investigation for infectious diseases. In dogs with waxing and waning GI signs including vomiting, diarrhea, and inappetance, signalment and disease severity should help dictate the prioritization of tests. In young to middle aged dogs, I suggest ruling out hypoadrenocorticism (even with normal electrolytes) by checking the dog’s resting cortisol. A value of >2.0ug/dL has a 100% sensitivity of detecting Addison’s disease, thus making this a rapid and highly sensitive screening test for this disease. If liver disease is suggested on base line blood work, now is the time that pre- and post- prandial bile acid testing should be pursued to test liver function. If chronic small intestinal diarrhea is present as well as weight loss, serum TLI should be tested to rule out exocrine pancreatic insufficiency. While a rare disease outside of the German Shepherd Dog breed, making this diagnosis prior to more invasive testing will save the owner a great deal of expense and the patient decreased morbidity associated with general anesthesia.

Abdominal imaging should next be pursued, with or without abnormalities detected on abdominal palpation. It is generally advised to take abdominal radiographs prior to performing an ultrasound, as complementary information can be obtained with both tests. Radiographs will give a good overall impression of the abdominal organs including gas distention of the intestines. Intestinal wall thickness is often over-interpreted on radiographs and caution should taken when making this determination. Fluid in the bowels can easily be interpreted as a thickened wall. Following radiographs, a wealth of information may be gathered from an abdominal ultrasound performed by an experienced ultrasonographer. Most importantly in cases of presumptive primary GI disease, the integrity

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of intestinal wall layering and overall wall thickness of the entire visualized GI tract (from stomach to colon) should be evaluated. Mucosa, submucosa, muscularis, and serosal layers should all be discretely identified in all segments of the intestinal tract. The colon is the thinnest layer, measuring up to 0.2cm on average. The small intestine can range up to 0.3-0.4cm, while the normal stomach may be as thick as 0.4-0.5cm. These figures are averages, so there may be some normal variation in some dogs, but subtle changes can also represent diffuse infiltrative disease. It is important to keep in mind that normal wall layering and thickness does not rule out inflammatory disease. Complete loss of layering in the intestinal wall, especially if focally observed, is highly suggestive of neoplasia.

If the clinical signs and initial diagnostic testing suggests chronic intestinal disease, a malabsorptive blood panel should next be considered. Serum folate and especially cobalamin concentration are commonly decreased with inflammatory bowel disease as well as other causes of chronic malabsorption. Similar to intestinal wall thickening, the presence of normal serum cobalamin does not rule out a chronic enteropathy including inflammatory bowel disease (ie. the test is not as sensitive as it is specific for IBD).

In dogs with mild clinical signs or diarrhea with no other systemic signs of illness, a hypoallergenic diet trial should be pursued prior to more advanced testing. There are two options to consider with a diet trial; a novel protein or a hydrolyzed protein. I will frequently make my decision dependent on the patient’s diet history; if the dog has been exposed to many dietary proteins in the past I will generally choose either a hydrolyzed diet or an anallergenic diet such as Royal Canin Ultamino. The owner must be made aware that this is a strict exclusive diet trial that should last a minimum of three weeks with no treats or other food made available to the dog.

If the patient has had chronic diarrhea and the diet trial is unsuccessful (ruling out food-responsive diarrhea), an antibiotic trial should next be pursued. Tylosin dosed at 20mg/kg by mouth every 12 hours is my antibiotic of choice. In many cases, if antibiotic-responsive diarrhea is present there will be a resolution of clinical signs within a few days when starting this new medication. If diet and antibiotics are unsuccessful at resolving clinical signs and no primary disease has been identified, intestinal biopsies should be taken. Endoscopy is the least invasive method of obtaining samples for histopathology, with the ability to reach the stomach, duodenum, colon, and possibly ileum. Disadvantages of this technique include equipment availability and experience of the endoscopist, ability to only take mucosal/submucosal biopsies, and inability to visualize the entire gastrointestinal tract. Benefits include it being an out-patient procedure with minimal complications. Alternatively, a laparotomy with full thickness intestinal biopsies can be pursued. This approach allows for full evaluation of the gastrointestinal tract. If no foreign bodies or masses are identified, multiple full-thickness biopsies can be obtained representing various segments, including stomach, duodenum, jejunum, and ileum. A negative exploratory should not be looked upon as a waste of time or an inappropriate test, but an opportunity to obtain biopsies.

Histopathology of the GI tract should help to better define the underlying disease process. The diagnostic approach to chronic enteropathies must be methodical and step-wise, as biopsy results showing lymphoplasmacytic enteritis is not diagnostic for inflammatory bowel disease, which is a diagnosis of exclusion. For example, a patient with food-responsive diarrhea may also have similar pathology results.

Dogs being presented for clinical signs consistent with a chronic enteropathy can be a diagnostic challenged, but if a standard approach is taken, including using therapeutic trials at some stages, an definitive diagnosis can often be made allowing for targeted therapy.

References available upon request
The gallbladder is a storage vessel for bile, located between the right medial and quadrate lobes of the liver, and is a major component of the extrahepatic biliary tract (EHBT). Bile is synthesized in the hepatocytes before being secreted into the canaliculi. From there it flows through the interlobular ducts, lobar ducts, the left and right hepatic ducts, and then either in to the cystic duct to the gallbladder or down the common bile duct to the duodenum. Bile enters the duodenum through the sphincter of Oddi after the common bile duct joins the minor pancreatic duct.

There are two broad categories of disease involving the gallbladder and the extrahepatic biliary tract: obstructive and non-obstructive. Obstructive diseases almost always will require corrective surgery, while non-obstructive diseases may be able to be managed medically.

Clinical signs and presenting complaints of dogs with gallbladder disease are often vague and non-specific. Vomiting, loss of appetite, diarrhea, and abdominal pain may be reported. Since there is a wide variation of severity of gallbladder disease, these signs can vary greatly. Clinical signs associated with obstructive gallbladder disease are often worse and may present more acutely, but this is not always the case.

**Cholecystitis**

Bacterial cholecystitis is a rarely reported condition in dogs that can cause a variety of clinical signs. Definitive diagnosis of this condition requires documentation of bactobilia, ideally with a subsequent positive bacterial culture. Gram-negative rods are the most common bacteria isolated from the gallbladder of dogs, with E. coli being the most commonly reported (Lawrence YA et al 2015). Procurement of a bile sample for culture and cytology can be via a cholecystocentesis or at the time of surgery (typically performed at the time of a cholecystectomy). Indications for this testing may include laboratory abnormalities including elevated liver enzymes indicative of hepatocellular injury or cholestasis, ongoing inflammation, or ultrasonographic evidence of gallbladder disease including a thickened wall, static sludge, persistently dilated bile ducts, etc. (Lawrence YA et al 2015).

Bacterial contamination of the gallbladder and subsequently the biliary tree and liver is most commonly thought to occur due to pancreatic or intestinal disease causing an ascending infection. The bile duct anatomy of the cat makes this species more likely to have bacterial cholangitis. Hematogenous spread of bacteria via the portal vein is another possible avenue for infection.

If bile cytology and/or culture is indicated and surgery is not necessary, a cholecystocentesis may be performed ultrasonographically with the patient heavily sedated using either a 22 or 25 gauge needle, angling through the right medial liver lobe if possible to help prevent bile leakage. Even when clinically and clinicopathologically indicated, bile cytology and culture can be unrewarding. A recently published study evaluating 140 bile samples from dogs and cats with suspected bacterial cholecystitis found bactobilia in only 24% of cases, with bacterial culture yielding a positive result in only 21% (Peters LM et al 2016).

Treatment for mild to moderate bacterial cholangitis / cholangiohepatitis should include hepatic support medication (SAM-e, milk thistle), a choloretic agent such as ursodiol, and broad-spectrum antibiotics (a combination of metronidazole, amoxicillin, and enrofloxacin may be indicated unless a positive bacterial culture and sensitivity panel is available for review). In more severe cases, especially if the gallbladder wall appears compromised on ultrasound, a cholecystectomy should be performed. Special focus after removing the gallbladder should be on the common bile duct to ensure patency, and a stent placed if unsure (Richter KP and Pike FS 2014).

**Cholelithiasis**

The presence of choleliths in a dog may warrant immediate surgery for removal, however in most cases these are an incidental finding and are unlikely to be associated with the patient’s presenting clinical signs. Stones typically form in the gallbladder and this is where they are most often incidentally found. Due to their composition, they are frequently radiolucent and thus are found only on ultrasound (14/27) (Kirpensteijn J et al 1993). Despite the infrequency of extrahepatic biliary obstruction secondary to cholelithiasis, cholecystitis can be seen concurrently with stones and thus medical management may be indicated. In the above referenced study, 15/20 cases that were taken to surgery for cholecystectomy had a positive bacterial culture (primarily gram negative rods, as in cholecystitis). Additionally, many of these cases had histopathological evidence of cholecystitis and mucosal hyperplasia. Thus, while immediate obstruction may not be present and surgical removal not indicated if the stones are found incidentally, the patient should be monitored for evidence of bacterial cholecystitis and medical management pursued if clinical or clinicopathologic evidence arises.
Gallbladder mucocele

A gallbladder mucocele has been defined as an accumulation of a green-black, bile-laden, semisolid to immobile mucoid mass within the fundus of the gallbladder (Aguirre AL et al 2007). The mucocele is widely considered to be the most common cause of extrahepatic biliary obstruction in dogs. Mucoceles are being diagnosed with an increasing frequency, which is likely due to the increasing availability of ultrasound in clinical practice. As with any test, however, false positives are a possibility and over-interpretation of a gallbladder image can lead to unnecessary surgical intervention. The appearance of a gallbladder mucocele on ultrasound is frequently compared to that of a kiwi fruit, with the impression of spokes around the periphery. What this represents is non-gravity dependent thick sludge within the lumen that has adhered to the walls of the gallbladder. Gravity-dependent echogenic sludge that is non-obstructive, however, may be easy to mislabel as a mucocele, especially when only still images are being evaluated.

The underlying cause of gallbladder mucocele formation is still unknown, however there are some diseases and breeds that seem to predispose dogs. Shetland Sheepdogs, Miniature Schnauzers, and Cocker Spaniels are prone to gallbladder disease, particularly mucoceles (Aguirre AL et al 2007). Some endocrinopathies put dogs at an increased risk for mucoceles, in particular hyperadrenocorticism and hypothyroidism. Dogs with hyperadrenocorticism have a 29 times higher risk of developing a mucocele compared to dogs with normal cortisol (Mesich MLL et al 2009). Diabetes mellitus has not been associated with mucocele formation.

Dogs do not always present with clinical signs of cholecystitis or other gallbladder disease when a mucocele is identified; in fact 11/38 dogs in the Aguirre study referenced above had their mucoceles identified incidentally during an abdominal ultrasound. Another study showed 7/30 dogs with a confirmed gallbladder mucocele had no adverse clinical signs (Pike FS et al 2004). The two most important questions when evaluating a dog for a possible mucocele are: is the dog clinical for gallbladder disease, and is there evidence of obstruction / post-hepatic cholestasis. An answer of “no” to each of these questions does not rule out a mucocele, but it should make the clinician at least consider medical management prior to going to surgery. Medical management of presumed mucoceles often involves hepatic support therapy (ie. Ursodiol, SAM-e, milk thistle) with or without antibiotics (ie. metronidazole, amoxicillin). Use of a choloretic in cases of suspected extrahepatic biliary obstruction should be discouraged; in these cases surgical intervention is indicated.

Perioperative mortality rate with cholecystectomy due to a mucocele has been reported as high as 21% (Pike FS et al 2004). This study included gallbladders that were both ruptured and intact, however there was no significant difference between these two types of cases. While this study did not show a higher mortality rate when the gallbladder was ruptured, from a clinical perspective post-operative management is more intense and hospitalization is often longer with a higher total bill if the gallbladder has ruptured prior to surgery. It is the author’s preference, therefore, to recommend cholecystectomy if a mature mucocele is identified. In the absence of clinical pathologic evidence of cholestasis in a patient with no clinical signs medical management may be pursued, but close observation of the patient along with routine follow-up visits including blood work are highly recommended.

References


Protein losing enteropathy (PLE) is defined as greater than normal loss of protein through the gastrointestinal tract (GIT) (Hall EJ 2010, Moore LE 2009). As much as 10% of daily protein catabolism occurs in the GIT, therefore losses greater than this are considered pathologic (Greenwald DA 2006). Clinically apparent PLE, however, involves much greater protein loss, as the liver is capable of increasing albumin production by greater than 100% to compensate for the deficiency (Peterson PB 2003). Quiescent disease, therefore, may be present long before clinical detection of hypoalbuminemia is made. There is a short list of differentials for hypoalbuminemia, including severe protein losing nephropathy/nephrotic syndrome, liver dysfunction, and protein losing enteropathy (PLE). One key difference that separates the three is the panhypoproteinemia that is seen with PLE. The loss of proteins is non-discriminatory and thus results in loss of small proteins (ie. albumin) as well as larger protein (globulins).

In dogs, PLE is frequently associated with lymphoplasmacytic enteritis and lymphangiectasia (Kull PA 2001), but any disorder resulting in disruption of the intestinal mucosa or increased intestinal lymphatic hydrostatic pressure may result in protein loss. Additionally, protein loss may occur in the face of normal intestinal mucosa secondary to leakage through enterocyte tight junctions (Bode L et al 2008). Some of the less common causes of PLE include acute or chronic infectious diseases (Parvovirus, Histoplasmosis), neoplasia (diffuse round cell neoplasia such as lymphoma, discrete mass such as adenocarcinoma, etc.), acute or chronic GI bleeding (neoplasia, ulcerations), severe dysbiosis, etc.

When hypoalbuminemia is identified on initial blood work in a sick dog, even in the face of severe chronic diarrhea, certain additional laboratory tests should be performed to help rule out liver and kidney involvement. At a minimum, a urinalysis and bile acids (pre- and post- prandial) should be checked; finding normal results in each of these tests will help to direct a diagnostic plan geared towards the gastrointestinal tract. A detailed medical history including patient signalment is equally as important to the aforementioned lab tests. There are a few well known breed-specific enteropathies that contribute to hypoalbuminemia, including the following:

- Yorkshire Terrier: Commonly seen with primary and secondary lymphangiectasia which can lead to severe PLE, hypomagnesemia, hypocalcemia, etc. (Kimmel SE et al 2000)
- Soft Coated Wheaten Terrier: Genetic predisposition to presumed severe food allergy that can contribute to PLE and protein losing nephropathy, with a poor long term prognosis (Littman MP et al 2000).
- Norwegian Lundehun: Nearly 100% of the breed is affected to some extent by a severe chronic enteropathy leading to PLE (Berghoff N 2007).
- Many breeds seem more sensitive to the effects of chronic enteropathies, leading to PLE, including Rottweiler, German Shepherd Dog, and Chinese Shar-Pei.

As previously mentioned, the most common causes of PLE in dogs are lymphoplasmacytic enteritis and lymphangiectasia. Diagnosis of these conditions can be made via intestinal biopsies, taken either endoscopically or surgically. The author’s preference in dogs with severe PLE is for endoscopic biopsies, as intestinal edema, ascites, and hypoalbuminemia can contribute to an increased risk of dehiscence secondary to prolonged healing time of the enterotomy site. An additional advantage is that immunosuppressive therapy, including corticosteroids, can be initiated immediately following an endoscopic procedure. If lymphangiectasia is a suspected differential diagnosis, administering 5-10mL of corn oil per os 2-4 hours prior to the endoscopy can make dilated lacteals more prominent, aiding in a visual diagnosis.

The quality and size of the endoscopic biopsies has been proven to be correlated to ability to make an accurate diagnosis, which makes experience of the endoscopist and quality of the equipment used of great importance when consider options for biopsies (Washabau RJ et al 2010). Additionally, endoscopy equipment is not readily available to all veterinarians, making surgery the only way to obtain tissue samples.

Once a cause has been identified treatment should begin as soon as possible, as acute decompensation can occur when hypoalbuminemia progresses. A combination of diet therapy, gastroprotectants, antibiotics, and immunosuppressive medications should be considered. If primary lymphangiectasia is suspected (or confirmed), an ultra low fat diet is indicated (Okanishi H et al 2014). In moderate cases a prescription low fat diet can be used.

Gastroprotectants can be used when gastric ulceration has been documented, or if vomiting is an accompanying clinical sign that may result in esophagitis. Omeprazole and famotidine are first-line antacids that should be used.

Antibiotics may be indicated in severe cases for treatment of bacterial translocation across the wall of the diseased intestines. Broad spectrum coverage with an antibiotic such as amoxicillin/clavulanic acid would be indicated in this instance. More commonly, however, there may be a component of dysbiosis, or bacterial overgrowth, which can contribute to a non-specific condition known as
antibiotic-responsive diarrhea. While this is not generally the only pathology associated with PLE, it can often be one part of the disease process. Tylosin 10-20mg/kg PO BID) is the drug of choice for this condition (Kilpinen S et al 2014).

Immunosuppressive therapy, first and foremost corticosteroids, will be indicated in most cases of chronic enteropathy that are severe enough to cause hypoalbuminemia. Prednisone is the most commonly used first line therapy, with starting doses up to 3mg/kg/day for severe cases. Larger dogs generally require a lower starting dose, in some cases only 1mg/kg/day. Alternatively, budesonide has been shown to be as effective (Dye TL et al 2013). In severe cases or if prednisone therapy has failed, additional immunosuppressive medications can be used. The use of chlorambucil (4.4mg/m²/day) has recently been shown to be more effective than prednisone alone in a subset of dogs with severe refractory PLE (Dandrieux JR et al 2013). Cyclosporine (Atopica) is another drug that has shown effectiveness in dogs with inflammatory bowel disease refractory to prednisone alone (Allenspach K et al 2006).

Long-term survival in dogs with chronic enteropathy is shorter in dogs with hypoalbuminemia compared to those with normal albumin at the time of presentation (Owens SL et al 2011). If an underlying diagnosis can be made and appropriate medical management started early, however, a favorable prognosis can be achieved. Recognizing breed predispositions, obtaining a thorough medical history, and ruling out other cause of hypoalbuminemia will help allow earlier medical intervention, contributing to an increased chance for survival.

References
Pancreatic Diseases in the Cat: The Common and not so Common
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The pancreas consists of both an exocrine and an endocrine component, each with its own very different purpose in normal daily homeostasis. The endocrine pancreas consists of alpha, beta, and delta cells that produce glucagon, insulin, and somatostatin, respectively. The most common disease involving the endocrine pancreas is under-production of insulin by the Beta cells, leading to diabetes mellitus. The much larger (anatomically) portion of the pancreas, the exocrine pancreas, is responsible for producing and secreting digestive zymogens that, when mixed with proteases in the intestinal lumen, become digestive enzymes such as amylase and lipase. Pancreatitis is the most commonly diagnosed disease affecting the exocrine pancreas; however other diseases including exocrine pancreatic insufficiency and pancreatic neoplasia are also reported.

Unlike in dogs, most cats that develop diabetes mellitus retain some normal functioning ability of the beta cells in the pancreas. This equivalent to “Type 2” diabetes in humans more often occurs due to insulin resistance systemically (due to obesity, systemic inflammation, etc.) and less often due primary pancreatic dysfunction. Since the remaining beta cells retain their ability to produce and secrete insulin, early management should be focused on trying to diagnose and eliminate the underlying cause of insulin resistance, and an attempt should be made to achieve diabetic remission. In some studies the ability to achieve remission is as high as 40-50%.

A connection between exocrine and endocrine pancreatic disease has been suspected, with the belief that cats with pancreatitis are at higher risk for diabetes mellitus. A recent study, however, has shown that there was no significant difference in pancreatic histopathology between cats with diabetes mellitus and healthy control cats. Additionally, the presence of ketoacidosis did not increase the risk of inflammation. In a separate study in dogs, up to 40% of diabetic cases were associated with pancreatitis.

The primary focus of this talk will be on diseases of the exocrine pancreas. Pancreatitis is by far the most common of these diseases, with neoplasia and EPI being seen much less frequently. Literature has shown that pancreatitis is a highly under-diagnosed disease, as necropsy studies have shown up to 67% of cats have evidence of pancreatic inflammation, including 45% of cats with no clinical signs of GI disease. Relevance of this finding, however, should be questioned, as many of these cats had no prior clinical signs associated with pancreatic disease. While histopathology is considered the gold standard for diagnosis, this is also an impractical test for most cats. This lack of a highly available gold standard test makes the definitive diagnosis even more challenging, and makes a thorough physical examination and history even more important when determining the cause of illness and how to develop a treatment plan.

There is a wide range of disease severity when discussing pancreatitis in cats, from very mild to life-threatening. There are also many suspected triggers for this disease, although in most cases the underlying cause goes unidentified. Recent dietary fat intake and obesity / hypertriglyceridemia is not a common cause of pancreatitis in cats, unlike what is seen in dogs. In up to 2/3 of cases of cats with pancreatitis, however, a concurrent disease is present including inflammatory bowel disease, cholangiohepatitis, diabetes mellitus, etc. Ultimately the most important reason to identify this trigger is to be able to avoid it in the future, as case management will be similar regardless of cause. The most common clinical signs in cats with pancreatitis are lethargy and anorexia, with vomiting only seen in ~40% of cats and diarrhea even less commonly occurring at 11-38%. Other physical examination findings are often vague and non-specific, including depression, dehydration, and cranial abdominal pain.

When a cat is presented with clinical signs consistent with gastrointestinal disease, baseline blood work including chemistry, complete blood count, and urinalysis should be performed. Unfortunately the standard lab variables on these tests (specifically amylase and lipase) are highly insensitive and non-specific for pancreatitis. As a result, interpretation of these two tests should only be done as one component of all other lab and clinical abnormalities. There are, however, many other diseases that will present with similar clinical signs and history that will have detectable abnormalities on these tests. These might include acute cholangiohepatitis, acute renal failure, pyelonephritis, etc. Cats are even less likely to have changes seen with the leukogram compared to dogs.

When pancreatitis is suspected based on clinical history, physical examination, and baseline blood work, additional testing should involve testing pancreatic lipase (SNAP test or SPEC IPl) and an abdominal ultrasound. While pancreatic lipase testing is more sensitive and results are more accurate compared to serum lipase, there are still great deficiencies with these tests. The cage-side SNAP IPl is designed to be highly sensitive and acts as a screening test for pancreatitis; sensitivity ranges up to 92% depending on the severity of the pancreatitis. The SPEC IPl lacks the sensitivity of the SNAP test (54% for mild pancreatitis), but is a more specific test (82%) for pancreatitis. The interpretation of ultrasonographic changes is dependent upon skill and experience of the ultrasonographer. There are no specific guidelines for the diagnosis of pancreatitis, but typical changes seen with the disease include pancreatic enlargement, peri-pancreatic inflammation or edema, cystic changes, hyperechoic echotexture, and a dilated pancreatic duct.
Ultimately the diagnosis of pancreatitis in cats is made based on a combination of clinical history, physical examination findings, laboratory abnormalities, and ultrasound results. The gold-standard diagnosis is made via histopathology, although this is rarely necessary to make the diagnosis and this is not without a high risk of morbidity.

Treatment of pancreatitis should be dictated based on the severity of the patient’s clinical presentation. Regardless of severity, fluid therapy is the most important component of treatment. Rehydration with crystalloid fluids followed by correction of ongoing losses (if vomiting and diarrhea is present) will help maintain adequate blood flow to the pancreas and aid in healing. Further supportive care is dictated by the patient’s clinical signs (anti-emetics, anti-diarrheal medication, etc.). Pain management can be a very important aspect of therapy as well. Overt abdominal pain is not always clearly evident, but is frequently present. As such, pain management should always be considered in the treatment plan. My top recommendations for the abdominal pain associated with pancreatitis include intermittent buprenorphine injections or, preferably, a CRI of fentanyl. Fentanyl is very fast acting and has a short half-life, which means the dose can easily be manipulated to meet the patient’s needs. Abdominal pain associated with pancreatitis can often be deceiving and lead to lethargy, depression, inappetance, and vomiting, even in cats that don’t outwardly object to abdominal palpation. In these cats a trial of pain management should be instituted to see if clinical signs begin to resolve.

Exocrine pancreatic insufficiency (EPI) is an uncommon pancreatic disease in cats. The presumed cause of this condition is severe end-stage chronic pancreatitis, resulting in loss of the acinar cells; however there are many cats that have no clinical history of chronic pancreatitis that are diagnosed with this disease. Regardless of the cause, clinical disease is typically not detectable until ~95% of pancreatic function is lost. Unlike in dogs with EPI who are frequently presented with chronic history of a ravenous appetite and voluminous soft stools, the most common presenting complaint in cats is weight loss. Diagnosis of EPI in cats is made by confirming a low serum TLI (trypsin like immunoreactivity). Management of this disease is simple in most cats, as they will frequently respond quickly to supplemental pancreatic enzymes added to the diet. When adding the enzyme, it is suggested to moisten the food (if feeding kibble), sprinkle the powder over the food, and then allow it to sit for 10-15 minutes prior to feeding.

Hypocobalaminemia is a common concurrent finding in cats with EPI, so serum cobalamin should be measured with subsequent supplementation if necessary. If weight gain and clinical improvement are not noticed within a few weeks of starting the pancreatic enzyme then evaluation for other underlying diseases such as inflammatory bowel disease should be considered.

Pancreatic adenocarcinoma is the most common neoplasia found in the cat pancreas. In the largest case series available, 34 cats were reviewed. Of these 34 cats, 11 had evidence of metastatic disease at the time of presentation. Prognosis is guarded with pancreatic adenocarcinoma, especially if the tumor has already metastasized at the time of diagnosis. 10% of cats survived over 1 year, but overall median survival was just 97 days. Median survival was slightly longer if surgery or chemotherapy was pursued (165 days).

There are other less common pancreatic diseases in cats, including pancreatic abscesses (which can be an adverse effect of severe pancreatitis), pancreatic cysts, and parasitic pancreatic disease.

References
Diarrhea is one of the most common reasons for presentation of a cat to their veterinarian. There are many underlying causes of diarrhea in cats, including both acute and chronic disease. Acute gastroenteritis characterized by diarrhea seems to occur less frequently in cats compared to dogs, possibly because cats are less likely to experience dietary indiscretion after getting in to the trash, eating human food, etc. Chronic diarrhea is a more common occurrence, however this can be difficult to detect for some cat owners as certain types of cat litter can help the stool clump and appear more solid than it actually is. Additionally, many pet owners have more than one cat and defecation is rarely observed so it may take longer to make the diagnosis.

Characterize the diarrhea

There are some distinct differences between large and small bowel diarrhea that must be determined prior to pursuing appropriate diagnostic tests. Classic signs of large bowel diarrhea include tenesmus, production of excessive mucous, frequent defecation (up to 5-6 times per day), and frank blood in the stools. In cats exclusively large bowel diarrhea is rare and usually accompanies an infectious disease such as Tritrichomonas or Giardia. Small bowel diarrhea includes weight loss, normal frequency of defecation, large voluminous stool, and normal urgency. In many cases there is some degree of overlap between these two types of diarrhea, however certain diseases are more likely to be associated with either large or small bowel diarrhea so localization can be helpful especially if considering histopathology.

Determining the cause

Most cases of feline diarrhea can be characterized as either infectious or non-infectious. Infectious diarrhea is more common in younger cats, especially cats who have originated from a cattery or a shelter environment. Differentials for infectious diarrhea include feline panleukopenia, Giardia, Tritrichomonas, Campylobacter, multiple intestinal parasites, Histoplasmosis, and Salmonella. Non-infectious causes of diarrhea include food allergy, antibiotic-responsive diarrhea, inflammatory bowel disease (IBD), hyperthyroidism, intestinal neoplasia, pancreatitis, and idiopathic gastroenteritis.

Clinical history is crucial to helping to differentiate infectious versus non-infectious causes of diarrhea. In many cases a detailed medical history will help to prioritize the differential diagnosis list which will help guide further diagnostic tests. Important questions to ask include the following:

- Has the cat spent any time recently in a cattery or cat shelter, or been exposed to other cats that have?
- Does the cat spend any time outside, or indoors only?
- Has there been any change in diet recently?
- Is the diarrhea acute and progressive, or chronic?
- If chronic, has the cat been losing weight?
- Are there any concurrent clinical signs, such as vomiting, loss of appetite, lethargy, etc.?

Knowing the answers to these few questions will go a long way towards determining the first tests that need to be performed. A thorough physical examination should also be performed and can be helpful for similar reasons. While most physical examination findings are unlikely to be pathognomonic for any one disease, there are some classic findings that will help shape your diagnostic plan. Diffusely thickened, or “ropey” intestines is more likely to be associated with chronic infiltrative disease. This finding in addition to enlarged mesenteric lymph nodes is more suggestive of GI lymphoma (although there are other causes including severe IBD and Histoplasmosis). Muffled lung sounds and labored breathing accompanying diarrhea is suggestive of either a protein losing enteropathy (less common in cats compared to dogs) or a diffuse systemic disease such as lymphoma.

Initial diagnostic testing should be prioritized based on the history and physical examination. In all cases of feline diarrhea a routine fecal ova and parasite test should be completed, as even older cats with chronic diarrhea may have a compromised GI immune system making them more at risk for intestinal parasites. I will routinely add Giardia testing on to that as well. If the cat is young and very ill with acute and severe diarrhea, a canine Parvo virus SNAP test can be performed to quickly rule out Feline Panleukopenia. If the cat is young and otherwise healthy with a negative ova and parasite test, and if infectious diarrhea is still considered likely due to the history, then a fecal PCR panel should be considered. This test is available through outside reference laboratories and will test for the following infectious causes of feline diarrhea:

- Campylobacter
- Clostridium
- Salmonella
- Giardia
- Cryptosporidium
- Coronavirus
- Feline panleukopenia virus
- Toxoplasmosis
- Tritrichomonas

Results from this test should be regarded with some skepticism depending on the patient’s clinical signs. For example, many cats may be Clostridium perfringens positive but have diarrhea due to another cause that has compromised the normal flora of the GI tract. In this case using targeted therapy against the Clostridium may help in the short term but would be unlikely to cure the patient’s diarrhea entirely, and it may come back just as bad once treatment is completed. Regardless of the results of fecal testing, there are some intestinal parasites that are not shed regularly and thus may not be seen on routine testing, so a trial treatment with a broad spectrum de-wormer such as fenbendazole should be considered.

Routine chemistry, complete blood count, and urinalysis should be tested primarily to rule out non-intestinal causes of diarrhea. In older cats a T4 should also be checked, especially if weight loss is also occurring. Causes of diarrhea that may be found on routine lab work include kidney disease (acute vs. chronic), pancreatitis (may be suspicious for based on these tests but serum amylase and lipase are highly inaccurate for diagnosis pancreatitis in cats), liver disease (bacterial cholangiohepatitis, liver failure, etc.), and hyperthyroidism.

**Advanced testing**

If the above diagnostics do not provide a diagnosis for the patient’s diarrhea, there are many options on how to proceed. If the diarrhea is chronic, non-life threatening, and no other clinical signs are present, then a diet trial is suggested. For cats my first option is a hydrolyzed diet such as Hill’s z/d, followed by a novel protein diet. The new food should be transitioned slowly over the course of a few days, and then fed exclusively for a minimum of 3 weeks.

If the food trial is unsuccessful but the patient remains stable with only diarrhea (and/or mild vomiting) then an antibiotic trial may next be pursued to rule out antibiotic-responsive diarrhea, such as metronidazole or tylosin. If the patient’s clinical signs are too severe to warrant 4-5 weeks of therapeutic trials, or these both fail, then an abdominal ultrasound should be considered. This test is unlikely to provide a definitive diagnosis for the diarrhea, but can provide valuable information to help determine additional testing. The most common abnormalities seen when investigating chronic diarrhea include overall intestinal wall thickness, loss of normal wall layering, and thickening of the muscularis layer of the small intestine. Additional findings may include enlarged and irregular mesenteric lymph nodes, and enlarged or irregular pancreas, dilated common bile duct, etc. Thickening of the muscularis layer, when coupled with enlarged mesenteric lymph nodes and clinical signs consistent with GI disease, is highly suggestive (although not pathognomonic for) GI small cell lymphoma. Inflammatory bowel disease (most commonly associated with lymphoplasmacytic enteritis / colitis) with no documented underlying etiology has no consistent, reliable changes seen on ultrasound.

Measurement of serum cobalamin and folate should be performed especially in cases of weight loss accompanying the diarrhea. These B vitamins help determine the presence of malabsorption with chronic intestinal disease. The value of this test is low in acute cases and should only be considered if a chronic enteropathy is suspected. Serum TLI should be tested especially if weight loss is present along with the diarrhea, as this is the most common presenting complaint in cats with exocrine pancreatic insufficiency.

If the diagnosis remains elusive with all of the above testing completed, intestinal biopsies should be taken. With presumed diffuse infiltrative disease, gastrointestinal endoscopy is an appropriate option to obtain biopsies. Evidence has shown a difference in histopathologic diagnosis (especially with severity of inflammation) in different segments of the bowel so when possible the stomach, duodenum, colon, and ileum should be sampled. Alternatively, full-thickness surgical biopsies can be taken of these same areas (although full thickness colon biopsies are not typically recommended unless a mass must be removed).

If results of the biopsies do not correspond with the clinical picture, or if neoplasia is suspected but not confirmed, additional testing may be performed. Immunohistochemistry and PARR are two different tests that can be performed on formalin-fixed tissue that will help to confirm or deny monoclonal lymphocyte populations, suggestive of lymphoma.

Treatment of feline diarrhea is entirely dependent on the underlying cause. Some pharmaceuticals such as metronidazole or even prednisolone will be effective for multiple underlying etiologies but will be simply masking the disease and not curing it. Alternative therapies including probiotics may be helpful in conjunction with more targeted therapy.

**References available upon request**
Vomiting is a common yet non-specific presenting complaint in dogs, which can be defined as forceful, active expulsion of gastric contents from the body (Twedt D 2010). In some cases, vomiting is a necessary response to expel toxic contents from the body. In the majority of cases, however, a non-gastric disorder will stimulate the emesis center leading to the act of vomiting.

There are two central locations that respond to hormonal influence to inducing vomiting: the emetic center and the chemoreceptor trigger zone. Various factors are capable of stimulating these areas, which makes vomiting such a non-specific clinical finding. These include gastric over-distention, pancreatic inflammation, pain, intestinal stretch receptors, uremic toxins, vestibular imbalance, and other factors. Hormone receptors that are capable of inducing vomiting include serotonin (5-HT3), alpha adrenergic receptors, and neurokinergic (NK-1). This information is clinically important when considering anti-emetic therapy.

The single most important first step when evaluating a patient for vomiting is a detailed medical history. Differentiating vomiting from regurgitation is a vital first step, since the diagnostic approach for each is very different. Identification of the three stages of vomiting (nausea, retching, and actual emesis) is crucial to differentiating it from the more reflexive act of regurgitation. Once vomiting has been established, the rest of the patient’s history will help dictate what additional testing may be indicated to most quickly determine an underlying cause. The following are some pertinent questions to be asked during the history:

- What is the duration and frequency of vomiting
- Is the patient on any current medications
- Has there been a recent diet change or unusual food eaten / table scraps, etc.
- Have any remedies been tried and failed
- What other clinical signs are the patient exhibiting (diarrhea, inappetance, chronic weight loss, etc.)
- Are there any known concurrent diseases
- Has there been recent travel or exposure to infectious diseases

Once the medical history is complete, a thorough physical examination is performed. Baseline lab work can then be completed (chemistry, CBC, urinalysis). Many underlying metabolic diseases that cause acute or chronic vomiting can be identified by reviewing these basic tests, including acute or chronic renal failure, pancreatitis, liver failure, chronic hepatitis, pyelonephritis, and others. In the absence of abnormalities on the initial lab work, further investigation will be needed:

- **Abdominal radiographs**
  - Evaluate the stomach for over-distention, foreign material, and marked thickness.
  - Inspect the small intestines for two separate populations (one normal, one markedly distended) indicating an obstruction.
  - Evaluate overall serosal detail for suggestion of peritoneal effusion.
  - If there is still suspicion of regurgitation, remember to take a right lateral thoracic radiograph to look for megaesophagus.

- **Barium study**
  - With increased availability of ultrasound, this is becoming a less desirable test. Barium in the GI tract prevents the ability to perform endoscopy until it has all passed, and if a bowel perforation has occurred barium peritonitis can aggravate the septic process further (Ko JJ 2014).
  - Interpretation can be difficult, especially when trying to determine gastric outflow and speed of transit through the gastrointestinal tract. Gastric and intestinal mobility may be delayed due to an underlying metabolic disease, hypoperfusion, or medications the patient is receiving, leading to a possible false positive for intestinal obstruction.

- **Abdominal ultrasound**
  - This can be a useful test to determine a cause of vomiting, however be cautious to avoid over-interpreting results. Severe intestinal ileus from pancreatitis, for example, can lead to dilated, fluid-filled loops of intestine. This finding can also be suggestive of an obstruction.
  - While abdominal ultrasound can be a sensitive and specific test when performed by an experienced ultrasonographer evaluating a case of acute vomiting for bowel obstruction, it has a lower utility for cases of chronic vomiting. A recent study showed that abdominal ultrasonography helped establish a diagnosis in only 23% of cases of chronic vomiting (Leib MS 2010). In the majority of cases the results did not change the clinical course.
• Additional lab work
  o Resting cortisol
    • Atypical Addison’s disease is an uncommon cause of chronic vomiting, but should not be overlooked (Sadek D 1996). Failure to recognize this disease prior to anesthetizing a patient for more invasive diagnostics can lead to a possible crisis with increased risk of morbidity. A resting serum cortisol >2.0ug/dL should be sufficient to rule out this condition. Perform a full ACTH stimulation test if the resting cortisol is <2.0.
  o Bile acids
    • Decreased liver function can be present in the absence of marked elevations in liver enzymes; conversely primary gastrointestinal disease can contribute to elevated liver enzymes. Bile acid testing will help to differentiate these disorders.
  o Leptospirosis antibody titer
  o GI Panel
    • Cobalamin/folate
    • SPEC cPL
    • TLI

When a definitive diagnosis cannot be obtained using the above diagnostics, consider the clinical history to help dictate the course of action. When the vomiting is acute and gastritis / acute gastroenteritis are suspected, symptomatic therapy including supportive care and anti-emetics should be pursued. If abdominal pain is initially present and persists, or if it develops after a therapeutic trial has begun, repeat abdominal radiographs may be indicated to recheck intestinal gas distention.

Anti-emetic therapy:
• Serotonin antagonists (5-HT3 receptor inhibitor) (Plumb D 2015)
  o The 5-HT3 receptors are found both centrally and peripherally.
  o Receptors are stimulated by serotonin when intestinal mucosa is disturbed
  o Ondansetron
• Substitued benzamides (Plumb D 2015)
  o Dopamine antagonist (and 5-HT3 receptor blocker at higher doses)
  o 5-HT4 agonists
  o Also include prokinetic properties (caution if obstruction is suspected)
  o Metoclopramide, cisapride
• Neurokinin-1 antagonist (Benchauoi HA 2007)
  o Acts as a ligand for Substance P in the brain (Substance P-Neurokinin receptor complex is thought to be the final pathway in the vomiting reflex).
  o Effective with both central and peripheral causes of vomiting.
  o Maropitant

Prior to more invasive diagnostics, and in a patient with chronic vomiting, consider prescribing a hypoallergenic diet as an elimination diet trial. If the patient continues to vomit after 3 weeks on an exclusion diet, a food allergy can be ruled out. If a novel protein diet is chosen instead of a hydrolyzed diet, two or three diet trials may be indicated if the patient’s complete diet history is not well known.

If vomiting continues in the face of symptomatic therapy, and a definitive diagnosis has yet to be reached, consider obtaining gastrointestinal biopsies. Gastroendoscopy is the least invasive method of obtaining samples for histopathology, with the ability to reach the stomach, duodenum, colon, and possibly ileum. Disadvantages of this technique include availability and experience of the endoscopist, ability to only take mucosal biopsies, and inability to visualize the entire gastrointestinal tract. Benefits include it being an out-patient procedure with minimal complications. Alternatively, a laparotomy with full thickness intestinal biopsies can be pursued. This approach allows for full evaluation of the gastrointestinal tract. If no foreign bodies or masses are identified, multiple full-thickness biopsies can be obtained representing various segments, including stomach, duodenum, jejunum, and ileum. A negative exploratory should not be looked upon as a waste of time or an inappropriate test, but an opportunity to obtain biopsies.

Once histopathology results have been evaluated, any further therapy that may be indicated should be started. If full thickness biopsies are taken surgically and corticosteroid therapy is warranted, I recommend waiting at least 5 days after surgery before starting, to allow adequate healing time.

References
Plumb D. Veterinary Drug Handbook 8th Ed. 2015
The conclusive diagnosis of inflammatory disease of the pancreas in dogs and cats is difficult. While the presence of compatible clinical signs such as vomiting, abdominal pain, dehydration, and pyrexia increase our index of suspicion for the disease, no specific findings on clinical examination are pathognomonic for pancreatitis.

As there are no specific findings on clinical examination or in an animal’s history that point conclusively to pancreatitis, confirmation of this clinical suspicion is based on clinical pathology results, imaging studies and the use of more specialized tests. The purpose of this presentation is to overview the various methods now available to the clinician that are used for the diagnosis of pancreatitis and their clinical utility in both diagnosis and management of dogs and cats with pancreatic inflammatory disease. With the availability of semi-quantitative ‘cage-side’ diagnostic tests measuring pancreatic lipase immunoreactivity, increasing numbers of animals are diagnosed with pancreatic inflammatory disease in practice. With this increased ability to specifically diagnose exocrine pancreatic disease, we are realizing that pancreatic disease may be more common, and more variable, than previously appreciated.

Markers of pancreatic disease
The most commonly used markers of pancreatic disease, and more specifically pancreatitis, are digestive enzymes. Serum amylase and lipase activities are the “traditional” markers used to support a clinical diagnosis of pancreatitis, in the presence of compatible history and findings on examination.

Low or normal serum lipase activity has been said to “almost always rule out the possibility of pancreatitis” in dogs, but increased serum lipase activity is not pathognomonic for pancreatic disease.1 While serum lipase activity is often empirically felt to be of higher sensitivity and specificity for the diagnosis of pancreatitis in dogs, controlled studies to demonstrate this are lacking. In cats, the performance of amylase and lipase as diagnostic markers of acute pancreatitis is even worse than in dogs. Experimentally, lipase and amylase activities are increased in induced pancreatitis in cats, but in spontaneous clinical cases these enzymes have no value in making the diagnosis. This likely relates to the typical timing of presentation of cats with pancreatitis, and the underlying pathological process in most cats, as discussed later.

While amylase and lipase activities may be the easiest biochemical markers of pancreatic inflammation to measure in dogs, the pancreas is a source of many other potential marker compounds. Of the digestive enzymes other than amylase and lipase, the enzymes most studied with respect to pancreatic disease are trypsin-like-immunoreactivity (cTLI and fTLI) and specific pancreatic lipase (Spec-cPL™ and Spec-fPL™).

Experimentally, serum TLI concentrations increase rapidly following induction of pancreatitis or ligation of the pancreatic duct in both dogs and cats. Serum TLI concentrations then drop rapidly, and are often back to normal or slightly sub-normal concentrations within 48 to 72 hours after disease induction. This is probably due to a protective down-regulation of trypsin synthesis by the pancreatic acinar cells, without further production of trypsinogen the serum concentrations decline rapidly.

Lipase produced in the pancreas is structurally distinct from the other lipases in circulation, although it shares the same substrate specificity. This means that, while activity assays are relatively non-specific for pancreatic disease, immunoassays for pancreatic lipase have a much higher specificity.

Canine pancreatic lipase immunoreactivity (PLI) has been measured via several differing immunoassay methods in recent years. The original studies using immunoassays to measure this protein were carried out at the GI Laboratory at Texas A&M University, using an ELISA method. Subsequently, the assay was commercialized as Spec cPL™ by IDEXX Laboratories, using differing antibodies and a recombinant protein for standards.2,3 Finally, IDEXX has produced “cage-side” diagnostic tests, SNAP cPL and Snap fPL, that measures the same protein in a semi-quantitative manner, defining a patient as normal or abnormal by reference to the intensity of the color spot developed. These assays are absolutely species specific, feline pancreatic lipase immunoreactivity cannot be determined using Spec cPL or the SNAP cPL tests, and vice versa.

As pancreatic lipase is found only within pancreatic acinar cells, an increase in the serum concentration of PLI is consistent with increased release of this enzyme from the acinar cells. The most common mechanism whereby pancreatic lipase is likely to be released is through increased “leakiness” of the acinar cells, this may occur as a result of mishandling of enzyme granules by the acinar cells, or due to compromised acinar cellular membrane permeability secondary to inflammation.

While the pancreatic lipase protein (and trypsinogen/TLI) are pancreas-specific, and thus elevated concentrations of these compounds in a serum sample are strongly suggestive of pancreatic pathology, the actual serum concentration of these compounds is altered by several factors other than just the presence and degree of pancreatitis. In particular, differences in the routes and speed of clearance of these proteins from the circulation can have a great bearing on the concentration measured.
Changes in the serum concentrations of TLI and PLI with pancreatitis

Trypsinogen, the precursor to active trypsin and the major protein measured in the TLI assay, is a relatively small protein with an overall negative charge. These factors both favor clearance by renal mechanisms, and the clearance half-life of trypsinogen is relatively short. Active trypsin is also detected by the TLI assay, however this protein is very rapidly complexed to scavenger proteins in the circulation and cleared from the circulation within minutes, thus active trypsin is rarely a significant contributor to the serum TLI concentration. This rapid clearance of trypsinogen/TLI, in combination with the dramatic down-regulation of pancreatic enzyme synthesis discussed above, results in serum TLI concentrations within the reference range, or in some cases below the reference range, in many animals with pancreatic inflammatory disease. These rapid changes in TLI concentration following onset of the disease contribute to the relatively low sensitivity of serum TLI concentrations for the diagnosis of acute pancreatitis in both dogs and cats.

In comparison to trypsinogen/TLI, the PLI protein in both dogs and cats is much larger (approximately twice as large) and has an overall positive charge. These features both prevent renal clearance of the PLI protein. The actual mode of clearance of PLI from the circulation is unknown at this time, but it is presumed to be via the hepatic reticuloendothelial system. The actual clearance half life of pancreatic lipase in the dog has been reported to be about 90 minutes, however the duration of elevation of PLI in dogs with pancreatitis is often a week or longer. This slower return to baseline/cessation of pancreatic lipase release increases the sensitivity of the test for detection of pancreatitis, as the clinician is more likely to be sampling a patient when the concentration is increased. In the cat, with experimentally induced pancreatitis, the degree of elevation of PLI is greater than that of TLI, and the serum PLI concentration remains elevated for an average of 10 days.

One of the many factors influencing pancreatic enzyme synthesis and release is feedback regulation from the small intestine. When active proteolytic enzymes enter the distal small intestine, a negative feedback signal that cuts off pancreatic enzyme synthesis and release is generated by the small intestinal mucosa. In the cat (but, interestingly, not the dog), small intestinal disease is often associated with a mildly but persistently increased serum TLI concentration. This most probably results from the mucosal disease and the loss of this negative feedback signal. This effect often leads to confusion amongst clinicians, as the clinical signs of marked small intestinal disease in the cat (steatorrhea, marked small intestinal diarrhea, weight loss, ± polyphagia) are often remarkably similar to exocrine pancreatic insufficiency. In many cats with significant small intestinal disease the serum TLI concentration is elevated, while the PLI concentration is within normal.

Test selection and interpretation in animals with suspected pancreatic disease

When selecting TLI or PLI tests for use in clinical patients, appropriate test selection will depend upon the clinically suspected diagnosis and the duration of clinical signs. In most cases where the clinician suspects the presence of pancreatitis, either acute or chronic, the PLI test is the best choice. Care must be taken with interpretation when using the “Snap” tests. These test are interpreted on the basis of the degree of color development seen, not as positive or negative (i.e. color development is expected in most cases, a very dark spot indicates high serum PLI). A positive Snap test should be confirmed by running the quantitative version of the test at a reference laboratory.

One consequence of the increasing availability of the PLI tests has been the recognition that chronic pancreatic disease, particularly chronic pancreatitis, is both much more common and in many cases more subtle than we originally thought. Particularly in the cat, most cases of pancreatic disease appear to be chronic and often clinically silent. In a case with chronic pancreatitis, the expectation is that the serum concentrations of PLI will remain persistently elevated. As the average time for decline to below the cut off value for a diagnosis of pancreatitis is around ten days, documentation of persistent elevation of PLI in serum samples taken at least 14 days apart can support the clinician’s suspicion of chronic pancreatitis. If the PLI returns to within the normal range at 14 days, this supports a retrospective diagnosis of an isolated bout of acute pancreatitis. These distinctions can be important, as the therapeutic approach to chronic pancreatitis is in many respects different to our approach to a patient who has had a single bout of acute pancreatitis and subsequently recovered.

Do TLI or PLI concentrations have prognostic value?

The degree of elevation of TLI or PLI in an animal with pancreatic inflammatory disease, as discussed above, is influenced by many factors. The amount of tissue compromised, the time from the beginning of the disease process, factors such as fluid losses that influence clearance; all of these factors may influence the final concentration measured in a patient. To date there is a limited amount of well-controlled data assessing the prognostic importance of varying degrees of abnormality in serum PLI concentrations. In one recent study, cats hospitalized for pancreatitis with serum Spec-fPL concentrations >20µg/L on the day of hospitalization were more likely to die or be euthanized. 4

Within an individual, resolution of elevated PLI concentrations, or a trend towards normality, appear logically to indicate progress towards a more “normal” state. Certainly the persistence of elevated PLI concentrations in a patient who continues to show clinical signs of pancreatic disease is consistent with ongoing inflammation in the pancreas, and an increasing concentration in this patient would be consistent with worsening or deterioration of the patient’s condition. The absolute degree of abnormality, however, does not seem to correlate with the symptoms seen or the duration of illness in many spontaneous pancreatitis cases.
Some observations on therapy for acute pancreatitis

Acute pancreatitis is an unusual disease process as total loss of exocrine pancreatic function is not immediately life threatening. Most general practitioners will be familiar with dogs that have pancreatic acinar atrophy leading to exocrine pancreatic insufficiency. These dogs may have virtually no functional exocrine pancreatic tissue, yet they do not suffer immediate life-threatening biological derangement because of this lack of pancreatic tissue. By comparison, the complete loss of hepatic, renal, cardiovascular, or respiratory function is associated with immediate, life threatening metabolic disturbances. While loss of pancreatic exocrine function is not typically life threatening, inflammation of this gland will often induce systemic metabolic derangement and organ failures distant from the pancreas itself. These distant organ failures, rather than the failure of the pancreas itself, are commonly the causes of death in acute pancreatitis.

In animals with complicated pancreatitis of high severity, the replacement and maintenance of circulating fluid volumes, attention to plasma colloid oncotic pressure, and the promotion of oxygen delivery to the tissues are all critical to successful therapy. Dogs with severe acute pancreatitis have a form of circulatory shock with many similarities to septic shock, and the clinical approach to these two forms of shock is essentially identical. Animals with pre-existing severe inflammatory disease, hypoalbuminaemia and multiple organ failure as a result of acute pancreatitis are beyond the therapeutic and management capabilities of most veterinary hospitals, and typically require referral for intensive care if treatment is desired. The prognosis for dogs with acute pancreatitis once they have developed this extent of metabolic derangement is guarded to grave, with mortality rates greater than 75% in some studies.

‘Feeding through’ bouts of acute pancreatitis, multimodal antiemetics

The idea that the pancreas should be “rested” during the treatment and recovery period from acute pancreatitis has long been held in the veterinary world. The theoretical basis for this recommendation was to decrease pancreatic enzyme synthesis and secretion, under the assumption that release of enzymes into the pancreatic interstitium and circulation was responsible for many of the clinical signs and complications of pancreatitis. While this approach still has some currency in the veterinary community, it flies in the face of our current understanding of the best management of critically ill patients. Animals with acute abdomen presentations are typically in a catabolic state, have additional metabolic demands due to the inflammatory process, and the development of functional ileus can result in substantial additional morbidity.

One of the main reasons for the nil per os approach to treatment of acute pancreatitis was in an attempt to reduce frequency and severity of vomiting in these patients. In recent years we have had a dramatic increase in the number of effective antiemetic medications available to us, most notably the neurokinin-1 receptor antagonist maropitant citrate (Cernia®, 1 mg/kg q24 hr), and the 5-HT3 receptor antagonists such as dolasteron (Anzemet®) or ondansetron (Zofran®), 0.3-0.5 mg/kg IV q24 hr). These medications are highly effective at controlling vomiting and nausea in our patients, and have the advantage that they can be administered by injection or orally, and need only once daily dosing. They act via different mechanisms, and there does not appear to be any meaningful interaction between these drug classes. Maropitant also has some benefit as a visceral analgesic, which is of significant benefit in acute pancreatitis cases.

With the use of a multimodal antiemetic regime, vomiting and nausea are controlled rapidly in most cases of acute pancreatitis. This allows consideration of an early return to feeding. While controlled studies of early enteral nutrition in severe pancreatitis in veterinary species are relatively few, the data available so far suggest that early enteral nutrition in dogs with acute pancreatitis is associated with less incidents of vomiting, lower incidences of complications than parenteral nutrition, and no difference in outcomes.

The author’s approach to dogs with pancreatitis is to reintroduce feeding as soon as possible, often within hours of ICU admission. Early enteral nutrition of cats with pancreatitis is arguably even more important, due to the risk of hepatic lipidosis in this species.

Dietary manipulations in the management of chronic pancreatitis

In the dog, use of a fat-restricted diet in the post-recovery period from a bout of acute pancreatitis is commonly accepted, and most authors will recommend fat restriction in dogs with chronic pancreatitis. The use of fat restricted diets is not recommended in cats, however, as this species has an obligate requirement for relatively high intake of essential fatty acids. Additionally, most fat-restricted commercial diets substitute carbohydrates for fat to maintain an iso-caloric formulation, this represents a problem for cats as their obligate carnivore nature means that they are less able to adapt to carbohydrate rich diets, and have a tendency towards protein catabolism if dietary fat is restricted. In many cats pancreatitis accompanies enteritis and cholangitis or cholangiohepatitis, so-called “triaditis” or “feline inflammatory syndrome”. While the underlying pathology of triaditis is not fully understood, overall it appears that the presence of inflammatory disease in the small intestine may be a common precipitating factor. Given the relatively low sensitivity of non-invasive tests for small intestinal disease in cats, and the low frequency with which biopsies are obtained, it is reasonable to assume that many cats with chronic pancreatitis will actually also have intestinal inflammatory disease that goes undiagnosed. Many cats with these diseases respond to dietary protein-source modification, using either a novel protein source or a modified antigen type diet. The author’s typical approach to a cat with a diagnosis of chronic pancreatitis is identical to the approach for inflammatory disease of the intestine, with carefully managed dietary elimination trials and screening for comorbid deficiencies in
water soluble vitamins such as cobalamin and folate. The protein type of the diet, rather than dietary fat content, has a much greater influence on diet selection in feline chronic pancreatitis cases.

References
Exocrine pancreatic insufficiency (EPI) is a syndrome characterized by maldigestion, malassimilation and marked, large volume small intestinal diarrhea. In the dog, this condition is usually the result of complete loss of pancreatic acinar tissue secondary to Pancreatic Acinar Atrophy (PAA). This condition is well known in the German Shepherd Dog, and is usually easy to recognize. In the cat, the underlying pathology leading to loss of exocrine pancreatic tissue mass is more likely to be chronic pancreatitis. This presentation will review the general features of EPI in both cats and dogs, including pathophysiology and diagnostic testing. Therapy of dogs with EPI is usually straightforward, and will be reviewed. In cats, therapy can be more challenging and other issues, including chronic cobalamin deficiency, must be addressed to ensure a good outcome. While most cases of EPI are the consequence of complete loss of exocrine pancreatic tissue, apparent selective pancreatic enzyme deficiencies have recently been recognized in several dogs, resulting in atypical presentations of exocrine insufficiency that can be diagnostically challenging.

Pathophysiology of diarrhea in exocrine pancreatic insufficiency
A lack of digestive enzyme synthesis and release into the small intestine results in a lack of digestion of dietary substrates. The osmotic draw produced by the unabsorbed, partially degraded nutrients within the small intestine will then produce an osmotic diarrhea. Fats are particularly strong inducers of diarrhea, as bacterial degradation of the fat produces large quantities of free fatty acids. These fatty acids can not be absorbed, and thus are potent osmotic agents, and in many cases the fatty acid products of bacterial fat degradation are toxic to the enterocytes, setting up secondary inflammation and motility disorders.

The pancreatic duct system, which is the source of pancreatic bicarbonate secretion, is spared in most dogs with EPI, and thus pancreatic bicarbonate secretion is normal. Trophic factors for the intestinal mucosa are lost, resulting in secondary abnormalities in structure and surface area of the villi and loss of brush-border enzymes. The exocrine pancreas has a sizable reserve capacity, and clinical signs are usually not seen until there has been a loss of >90% of the acinar tissue.

Pancreatic acinar atrophy vs. chronic pancreatitis
In the dog, the most common cause of primary pancreatic exocrine insufficiency is pancreatic acinar atrophy (PAA). This condition is associated with progressive loss of pancreatic acinar tissue, in at least some groups of dogs PAA appears to be associated with or preceded by a lymphocytic/plasmacytic pancreatic infiltrate. In both German Shepherd dogs and the Rough-coated Collie there is evidence for heritability of this disease, most likely as an autosomal recessive.

In the cat, EPI is most commonly due to loss of exocrine tissue due to ongoing chronic pancreatitis, the end-point of chronic pancreatitis being fibrosis and scarring. Juvenile onset disease, similar to PAA in dogs, has not been described in the cat to date. Chronic pancreatitis as a cause of primary exocrine insufficiency has been described in dogs and is the most common cause of late-onset exocrine insufficiency (i.e. in dogs >4 years of age).

Signalment and breed factors
Pancreatic acinar atrophy is usually diagnosed in young dogs, with a peak time of diagnosis at around 18 months of age. Development of clinical signs consistent with EPI in dogs older than three years should prompt the clinician to search for small intestinal disease. The German shepherd breed is most commonly recognized, as previously discussed this breed and the Rough-coated collie have a heritable predisposition for PAA in some family groups. PAA may be diagnosed in any breed of dog, although overall large breed dogs are diagnosed more commonly.

In the cat, EPI is usually diagnosed in middle-aged to older cats, reflecting the occurrence of EPI as an end result of chronic pancreatitis in this species. The time for development of EPI in cats with chronic pancreatitis is unknown, but given that this is a relatively rare (but increasingly recognized) diagnosis in the cat, and chronic pancreatitis appears to be remarkably common (See “Finicky feline: Pancreatitis in cats”), development of EPI is probably a relatively slow process in the cat.

Making the diagnosis of exocrine pancreatic insufficiency
Measurement of the serum concentration of trypsinogen (TLI) is the diagnostic test of choice to rule in/rule out exocrine pancreatic insufficiency due to a loss of pancreatic acinar tissue. As loss of acinar tissue is the most common cause of exocrine insufficiency, assessment of serum TLI should be carried out early in the diagnostic assessment of animals with compatible clinical signs.

In the dog, a serum TLI concentration of 2.5 µg/L or less is highly sensitive and specific for pancreatic acinar atrophy. In the cat, exocrine insufficiency is diagnosed when the serum TLI concentration is less than 8 µg/L. Detection of > 5 µg/L serum TLI in the
dog or > 12 μg/L serum TLI in the cat effectively rules out a loss or absence of pancreatic acinar tissue, and thus makes the diagnosis of primary pancreatic exocrine insufficiency much less likely.

The major differential diagnosis for EPI is small intestinal disease. A failure of the small intestinal mucosa to absorb digested nutrients will result in osmotic diarrhea and steatorrhea, with large volume diarrhea and weight loss. Animals with small intestinal disease may also present with a ravenous appetite and failure to thrive, a result of the decreased efficiency of utilization of dietary nutrients. Empirically, many animals with small intestinal disease will show mild improvement in their clinical signs with digestive enzyme supplementation, but this is an expensive and usually only mildly beneficial therapy for these cases. For this reason, digestive enzyme supplementation (see therapy below) should typically be reserved for cases where EPI/PAA has been confirmed by measurement of serum TLI concentrations.

In the cat, small intestinal disease is much more common than EPI. Most cats in which serum TLI is measured due to a suspicion of EPI actually have normal or mildly elevated serum TLI concentrations. This finding rules out loss of pancreatic acinar tissue, and should prompt the clinician to investigate more thoroughly for small intestinal disease.

As small intestinal disease is a major differential for EPI, measurement of serum concentrations of cobalamin and folate is often helpful. In both cat and dog, but particularly in the cat, serum concentrations of cobalamin are often low in EPI patients due to lack of pancreatic intrinsic factor. The presence of low serum cobalamin with a normal TLI is a highly specific indicator of small intestinal disease. Even in patients with confirmed EPI, cobalamin malabsorption and subsequent deficiency may lead to poor response to therapy.10 11

Several other methods for assessing pancreatic exocrine function have been described. Before the development of the TLI assay, determination of fecal proteolytic activity, measurement of the fecal fat content and microscopic examination of fecal smears for undigested muscle fibers have both been used in the past. Recently, measurement of canine fecal elastase activity has been promoted as an alternative to the TLI assay, with the benefit of an ELISA methodology that can be run in-house (the canine TLI assay is a radioimmunoassay, limiting its availability to specialty laboratories). Without exception, these other tests show a lower sensitivity and specificity for diagnosis of EPI than the TLI assays. Fecal proteolytic activity assays are still occasionally used in exotic species (ferrets, meerkats), but their use is strongly deprecated in dogs and cats.

**Therapeutic considerations in the dog**
The mainstay of therapy for EPI in the dog is replacement of pancreatic enzymes with any of a variety of porcine-pancreas derived products. Powdered forms are generally preferred; enteric-coated tablet forms have poorer bioavailability in the dog and are often associated with treatment failure.

Using a powdered form, a typical starting dose is 2 teaspoons/20 kg of dog, given with every meal. There are no benefits to pre-incubation of the meal with the enzymes. A standard maintenance diet is usually adequate for initial treatment, although some dogs will show additional benefit from the lower fat diet to reduce the osmotic load from fatty acids. Higher fiber diets should be avoided, as these may bind to the digestive enzyme supplement and reduce its availability. Fat absorption is particularly problematic for dogs with EPI, and development of fat-soluble vitamin deficiencies has been documented.12 Parenteral supplementation with vitamin K should be provided in affected individuals. Serum cobalamin concentrations should be monitored every 3-6 months, and supplementation provided if the serum cobalamin concentration is decreased.

Two meals a day of a balanced canine maintenance diet are usually adequate for weight gain and normalization of the nutritional state. Diarrhea usually resolves within 4-5 days, however up to 20% of dogs in one study showed poor response to therapy. 13

**Therapeutic considerations in the cat**
In common with the dog, effective treatment of exocrine pancreatic insufficiency in the cat relies on the effective replacement of digestive enzymes with powdered porcine pancreas extracts. A reasonable starting dose for the cat is approximately 1/4 teaspoon of extract per meal. Pre-incubation of the meal with the enzymes should be avoided as this may lead to food aversion in the cat. Compounding of the enzyme powder into gelatin capsules can be used in cats with severe food aversion, however this relies on the ability of the owner to administer the capsules to the cat. Gelatin-encapsulated enzyme powder capsules must be kept scrupulously dry or the capsule will be degraded.

Cats with EPI are almost invariably cobalamin deficient, the exocrine pancreas is the only source of intrinsic factor in the cat. Parenteral cobalamin supplementation (250 μg/cat by subcutaneous injection, once weekly to every second week) is necessary in most cats with EPI, response to treatment is often poor if cobalamin is not supplemented.

**“Subclinical” EPI**
Occasionally dogs are encountered with mild clinical signs of small intestinal malabsorption and serum concentrations of TLI that are lower then the bottom end of the reference range (5.7 μg/L), but not at or below 2.5 μg/L. If the dog is a young, large breed dog with a known predisposition for PAA, this may represent a subclinical state of PAA and warrants monitoring for progression to full-blown PAA and EPI. This “subclinical” state may persist for extended periods in some dogs, and if clinical signs are not seen, specific
therapy is not indicated. Dogs with this gray zone TLI concentration and mild or only sporadic clinical signs often respond well to diet change, preferably to a lower fat diet.\textsuperscript{14} Digestive enzyme supplements benefit some of the dogs in this group, but the efficacy of this treatment is usually no greater than that achieved with fat restriction, and enzyme therapy is significantly more expensive.

**Comorbidities and complications**
Lymphocytic/plasmacytic enteritis and intestinal dysbiosis are common complicating conditions in dogs with EPI, thus in those cases with poor responses to therapy the use of glucocorticoids (prednisone/prednisolone at \(\sim 1\)mg/kg SID) and broad-spectrum antibiotic therapy (the author’s preference is tylosin (Tylan Powder\textsuperscript{r}, Elanco) at 25 mg/kg BID) may be indicated.

Gastric acid degradation is not usually a significant problem, but in some cases where appropriate doses of enzymes, antibiotics and glucocorticoids are being administered yet response to therapy is poor, additional benefit may be seen from treatment with a proton pump inhibitor such as omeprazole.

Both dogs and cats developing EPI as an end result of chronic pancreatitis may also be at increased risk for the development of insulin-dependent diabetes mellitus.\textsuperscript{15,16} Dogs developing EPI due to pancreatic acinar atrophy, however, are at no greater risk for development of diabetes as the islet tissue is spared.

**Selective pancreatic enzyme deficiencies**
A limited number of dogs have recently been described with a clinical syndrome that appears to reflect a selective deficiency in pancreatic enzyme synthesis, rather than a complete loss of acinar cellular mass.\textsuperscript{17,18} These dogs presented at a comparatively young age (4 months to 1.5 years) with ravenous appetites, long histories of small intestinal diarrhea, poor body condition and failure to thrive. All of but one of these dogs showed normal serum TLI concentrations, with only one dog having TLI below the reference range, but greater than 2.5µg/L. All other diagnostic testing on these dogs was relatively unremarkable. In several of these dogs Specific-PL values were below the lower limit of detection of the assay, but this is also commonly observed in normal dogs and is not considered diagnostic for exocrine insufficiency or pancreatic acinar atrophy.\textsuperscript{19}

Interestingly, after failure of all other diagnostic tests to yield a diagnosis, and only limited response to dietary modification trials, all dogs showed marked clinical response to pancreatic enzyme supplementation, supporting the hypothesis that clinical signs in these dogs were due to absence of at least one of the pancreatic digestive enzymes.

While these recent case reports indicate that at least some dogs can present with a condition that requires pancreatic enzyme replacement therapy while serum TLI concentrations were within the normal range, it is important to note that these dogs had all undergone rigorous diagnostic work ups to exclude all other potential differential diagnoses. Small intestinal disease due to other etiologies, such as dietary intolerance or chronic parasitism, are far more likely causes for dogs to present with these clinical signs, and should be rigorously excluded before therapeutic trials of digestive enzymes are considered.

**Prognosis**
The prognosis for dogs with EPI due to PAA is fair to good for recovery of normal intestinal function and weight gain with appropriate therapy. While this is often a straightforward condition to manage, it can become expensive. Particularly in larger breed dogs, where this diagnosis is made most often, the cost of enzyme replacement therapy for the life of the dog can be substantial, and may represent a hardship for some owners. For this reason, accurate diagnosis and differentiation of PAA/EPI from other small intestinal diseases is very important.

Overall the prognosis for cats with EPI is more guarded than for the dog, due to the greater tendency to food aversion, more difficult administration of enzyme supplements and the existence of comorbid conditions such as chronic pancreatitis, enteritis and other age-related diseases.

**References**


In recent years, with greater availability of high-level diagnostic imaging and minimally invasive methods for the determination of pancreas-specific marker proteins in the serum, we have undergone a paradigm shift with respect to pancreatic disease in the cat. Once thought to be uncommon, we now know that a very large proportion of feline patients have chronic pancreatitis. In one remarkable study, the overall prevalence rate for chronic pancreatitis was 67% in ill cats, and even more remarkably, 45% in normal cats, based on histopathologic examination of 115 feline pancreata. Far from being uncommon, it is now apparent that pancreatic pathology, and particularly chronic pancreatitis, is common in the domestic cat. This disease likely represents a large proportion of feline cases presenting with reduced appetite, lethargy or chronic vomiting disorders, hopefully further large-scale epidemiologic studies will help to confirm or deny this hypothesis.

Chronic pancreatitis in the cat is distinctly different from chronic pancreatitis in most dogs, and shares more features with chronic pancreatitis in humans. In particular, marked lymphocytic infiltration and the presence of profound fibrosis are common in feline chronic pancreatitis.

Clinical signs of pancreatitis in the cat

One of the great challenges in managing the cat with pancreatitis is the vague nature of clinical signs typically manifested in these cats. Based on the aggregation of data from three studies, involving a variety of underlying histological diagnoses and apparent disease severities, the most common clinical signs of pancreatitis in the cat are reduced appetite, lethargy, dehydration and vomiting (Table 1, below).

Table 1

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Stockhaus²</th>
<th>Ferreri³ (ANP)</th>
<th>Ferreri³ (CP)</th>
<th>Hill⁴</th>
<th>Total</th>
<th>Overall Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Cats</td>
<td>33</td>
<td>30</td>
<td>33</td>
<td>40</td>
<td>136</td>
<td></td>
</tr>
<tr>
<td>Inappetance</td>
<td>(97%)</td>
<td>(63%)</td>
<td>(70%)</td>
<td>(98%)</td>
<td>113</td>
<td>83%</td>
</tr>
<tr>
<td>Lethargy</td>
<td>(100%)</td>
<td>(50%)</td>
<td>(52%)</td>
<td>(100%)</td>
<td>105</td>
<td>77%</td>
</tr>
<tr>
<td>Dehydration</td>
<td>(73%)</td>
<td>(33%)</td>
<td>(51%)</td>
<td>(93%)</td>
<td>88</td>
<td>65%</td>
</tr>
<tr>
<td>Vomiting</td>
<td>(55%)</td>
<td>(43%)</td>
<td>(39%)</td>
<td>(35%)</td>
<td>58</td>
<td>43%</td>
</tr>
<tr>
<td>Icterus</td>
<td>(18%)</td>
<td>(16%)</td>
<td>(24%)</td>
<td>(53%)</td>
<td>40</td>
<td>29%</td>
</tr>
<tr>
<td>Weight Loss</td>
<td>(9%)</td>
<td>(40%)</td>
<td>(21%)</td>
<td>NS</td>
<td>22</td>
<td>16%</td>
</tr>
<tr>
<td>Abdominal Pain</td>
<td>(52%)</td>
<td>NS</td>
<td>NS</td>
<td>10(25%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abdominal pain, a very common clinical sign of pancreatitis in the dog, is much less frequently recognized in the cat. Accurate assessment of abdominal pain in the cat can be quite difficult, and thus the true frequency of this problem in cats with pancreatitis may be underestimated, however the central observation that abdominal pain is rarely appreciated by clinicians assessing cats with pancreatitis remains true. Given the vague nature of clinical signs of pancreatitis in the cat, this disease should be considered in the differential diagnosis of any cat with vomiting, anorexia/hyporexia or lethargy where another, more proximate cause has not been identified.

An interesting observation from the aggregated retrospective studies is that these signs were the same regardless of underlying type of pancreatitis in the cats, with both severe necrotizing disease and more chronic, fibrotic disease having the same general signs. Based on these observations, it is not possible to distinguish acute from chronic pancreatitis in cats based on clinical presentation, duration of clinical signs or apparent severity of the disease. While chronic pancreatic disease is commonly thought to be less severe than acute pancreatitis in the cat, either disease can present with complications or comorbidities that are potentially life-threatening, and attempting to draw a distinction between these two conditions is not particularly clinically helpful.

Diagnostic approach to pancreatitis in the cat

In order to make the diagnosis of pancreatitis in the cat, obviously a clinical suspicion is necessary. Given the highly vague and variable nature of pancreatic disease signs in the cat, essentially any sick cat should have pancreatitis on their differential diagnosis list.

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at first assessment. Routine chemistry panels are very useful for screening for other significant diseases that can cause lethargy and a poor appetite, such as renal insufficiency. Routine chemistry panels may also provide evidence of hepatobiliary disease, which is a common comorbidity with pancreatitis in the cat.

In many patients routine chemistry panels are unremarkable, reflecting the fact that there are no diagnostic tests on a typical screening chemistry panel that are sensitive and specific for pancreatic disease. This includes amylase and lipase activities, which are generally thought to have no diagnostic utility for detection of pancreatitis in the cat.1,3 Further assessment of these patients typically involves both abdominal ultrasound examination and the use of more specialized blood tests, particularly the feline specific pancreatic lipase assay (Spec-fPL).

At the time of writing Spec-fPL has the highest sensitivity and specificity for the diagnosis of pancreatitis of any diagnostic test in the cat, exceeding ultrasound, plain radiography and computed tomography scanning.3,9 This test also has the advantage of being minimally invasive and relatively inexpensive.

Spec-fPL values increase dramatically early in the development of pancreatic inflammatory disease, and then are cleared from the circulation relatively slowly, taking up to 14 days to return to the baseline value after the onset of acute pancreatitis. When the clinician suspects that chronic pancreatitis is present, determination of Spec-fPL concentrations repeatedly at 2-3 week intervals can bolster this diagnosis. The expectation is that serum Spec-fPL will remain elevated above the reference range throughout this period, even if the cat is showing few or no clinical signs.

The low sensitivity and specificity of traditional amylase and lipase activities for the diagnosis of pancreatitis, in all species, may be partly explained by low substrate specificity for most of the catalytic assays. The substrates used in these assays vary in terms of selectivity for pancreatic lipase, with some substrates showing much higher selectivity for pancreatic-origin lipases in the circulation.

1-2-o-Dilauryl-rac-glycero-3-glutaric acid-(6′-methylresorufin) ester (DGGR) is a lipase substrate with relatively high substrate specificity for pancreatic lipases. In a recently published study of 251 client owned cats with a clinical suspicion of pancreatitis, DGGR-lipase activity >26 U/L showed a sensitivity of 48% with a specificity of 63%, while Spec-fPL >5.3 μg/L showed a sensitivity of 57% and specificity of 63%.10 This study suggests that DGGR-lipase activity may have some clinical utility in the assessment of cats, however this would be reliant on the use of this specific substrate in whichever analytical system is being used. Information regarding the actual substrates used by the various reference laboratories and in-house chemistry systems commonly found in veterinary practice is not readily available at this time.

Therapeutic approaches to the outpatient case
Given the very high frequency of dietary intolerance recognized in some studies of cats with chronic gastrointestinal disease, a rational initial step in the approach to a cat with a diagnosis of pancreatitis is an elimination trial using a hypoallergenic diet. The author’s preference is to use a novel protein source, selected based on a thorough dietary history, rather than the modified/partially hydrolyzed protein diets, however in some cases these modified diets are effective and well received. Dietary modification to a novel protein source often seems to be helpful in cats with chronic pancreatitis as well as in those with gastritis. Fat restriction, the mainstay of therapy for chronic pancreatitis in the dog, is less beneficial in most cats with chronic pancreatitis.

The patient is started on the elimination diet exclusively for a minimum period of 14 days. In most patients with diet-responsive disease a notable improvement in clinical signs will have occurred at 14 days, and those that have failed to show a good response are unlikely to show much benefit from a longer period on the diet. If the cat responds to the diet change, the diagnosis becomes one of food-responsive gastritis or dietary intolerance. “Dietary allergy” implies demonstration of a hypersensitivity response to a dietary component, as this is not achieved in most veterinary patients this term is not appropriate to most cases. Reintroduction of the previous diet or dietary components (protein sources etc) can be attempted, and if clinical signs recur the diagnosis of food intolerance is confirmed, subsequent therapy emphasizes the avoidance of the offending food component. In many cases owners are unwilling to reintroduce the original diet if clinical signs are abated and the new diet is continued empirically.

If the patient shows no response to the first diet change at 14 days, another new diet can be trialed. Most owners are unwilling to persist beyond two dietary trials, and additional therapy is needed. Failure to respond to dietary modification in a cat with infiltrative gastric mucosal disease allows the diagnosis of idiopathic chronic gastritis to be made. Therapy for this condition usually requires anti-inflammatory therapy, typically using glucocorticoids. Many cats with chronic pancreatitis also show satisfactory responses to glucocorticoid therapy, typically starting with prednisone/prednisolone at 1-1.5 mg/kg SID for approximately 14 days. There is no evidence to date that glucocorticoid therapy is associated with increased risk of worsening pancreatitis in the cat. If good control of clinical signs has been achieved, a gradual taper of the glucocorticoid to the minimum effective dose is started.

Pain control, maintenance of adequate nutritional intake in cats with inappetance, and maintenance of hydration are all critical to success. Most cats with pancreatitis presenting to companion animal practices are able to be managed on an outpatient basis, but the owner should be counseled on the need for close monitoring of food intake and the possibility of worsening of the disease which may require hospitalization for fluid therapy and assisted nutrition (see below).
A common empirical therapy is the use of pancreatic enzyme supplements to attempt to “down regulate” the pancreatic synthesis of digestive enzymes, the theory being that this will reduce ongoing pancreatic pathology. There is no evidence that this is efficacious. Given that the main pancreatic pathology in the cat is a chronic lymphocytic and fibrotic process, rather than an autotoxic degradative/necrotic process, we now know that there is little physiological rationale for this therapy, and it is not recommended.

**Therapeutic approach to the cat with severe disease**

Cats with suspected pancreatitis presenting with marked abdominal pain, tachypnea, tachycardia, significant fever, collapse or other evidence of systemic inflammatory syndrome or circulatory shock are considered to have severe disease, and require immediate and aggressive, hospital-based care. The existence of multiple abnormalities in screening clinical chemistries, particularly hypalbuminemia and hypocalcemia, is a strong indicator of severe and potentially life threatening disease.5

As with dogs, cats presenting with severe pancreatic inflammatory disease require aggressive therapy, including fluid therapy, effective analgesia, and early planning for nutritional support given the risk of hepatic lipidosis as a comorbidity. The aims of therapy are to replace circulating fluid volume, restore and maintain end organ perfusion (particularly of the pancreas, as pancreatic ischemia is a significant contributor to the development of necrotizing pancreatitis6), restore and maintain plasma colloid oncotic pressure. Colloid fluids, such as synthetic hydroxyethyl starches, are often highly beneficial in the initial resuscitation of these cases. Fresh-frozen feline plasma can also be considered, and likely provides oncotic support while replenishing coagulation cascade proteins, however there is little information in the veterinary literature regarding use of plasma in severe feline pancreatitis cases. We typically use a combination of synthetic colloid and crystalloid fluids for initial resuscitation and volume maintenance in these cats in our clinic. Substantial electrolyte abnormalities, particularly hypokalemia and hypocalcemia, should be anticipated in these cats.5,7 Supplemental potassium is administered in combination with crystalloid fluids following routine guidelines for concentrations based on serial determination of serum potassium.

Effective analgesia and control of vomiting are important aspects of management of severe pancreatitis in all species, including the cat. Narcotic pain control is typically indicated in cats with sufficiently severe pancreatitis to warrant hospitalization. Transdermal fentanyl patches (25µg/hr) can be very effective for longer term (up to 72 hrs) analgesia without the need for frequent handling and injection in these patients, but initial therapy with an injectable or sublingual agent (commonly buprenorphine) is necessary as it can take up to 12 hours for therapeutic fentanyl concentrations to be reached.7 Maropitant, a neurokinin-1 receptor antagonist, is both an effective antiemetic and has antinociceptive effects in the viscera.11 The combination of maropitant with a 5-HT3-receptor antagonist, such as ondansetron or dolasetron, provides an effective control for vomiting and nausea in these patients with minimal need for repeated handling during the day.

**The special case of the diabetic cat**

Cats with diabetes mellitus and chronic pancreatitis represent a significant challenge, particularly if they are poorly or minimally improved by rigorous use of an elimination diet. The use of glucocorticoids in these cats risks the loss of glycemic regulation, increased insulin requirements or the development of insulin resistance. When faced with this particular quandary, the author’s personal preference is to emphasize dietary modification and weight loss to attempt to improve the glycemic state of the cat, rather than use of glucocorticoids to control gastric or pancreatic inflammation. Many cats will show an improvement in their chronic vomiting as they enter a euglycemic state, and anecdotally many cats with chronic pancreatitis show an improvement in their clinical signs and a normalization of serum fPLI concentrations after they are switched to the higher protein, low carbohydrate dietary regimes currently recommended for management of diabetic cats.12

**References**

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Chronic gastrointestinal disease is one of the most common reasons for companion animal owners to seek veterinary care. Clinical signs such as diarrhea, vomiting and inappetance are common in dogs and cats with chronic gastrointestinal disease, these signs are distressing to owners. Many underlying diagnoses can lead to chronic gastrointestinal signs, and conclusive diagnoses are often difficult to achieve. Successful management of these cases relies on a multi-pronged approach, involving dietary manipulations, identification and elimination of parasites, assessment of gastrointestinal function and, in some cases, pharmacological manipulation to mitigate inflammation.

A common feature of many of the diseases leading to chronic gastrointestinal signs is the presence of gastrointestinal inflammation, this inflammation may be the result of dietary intolerance (so called “Food-Responsive Diarrhea”), intestinal dysbioses or chronic colonization by bacterial pathogens (so called “Small Intestinal Bacterial Overgrowth” or “Antibiotic-Responsive Diarrhea”), or may be idiopathic. As classically understood, canine idiopathic inflammatory bowel disease (IBD) is characterized by persistent or recurrent clinical signs of GI disease of unidentified cause, associated with histologic evidence of inflammatory infiltration of the intestinal mucosa. The final diagnosis of an individual patient as food-responsive, antibiotic-responsive or idiopathic inflammatory bowel disease depends upon rigorous completion of therapeutic trials to rule out the food- and antibiotic-responsive diseases. In many cases a definitive diagnosis cannot be made due to imprecise or non-specific findings from diagnostic tests and the difficulty of accurately assessing the GI tract in an non-invasive manner. Thus many authors prefer the less restrictive term “chronic enteropathy” to refer to patients with these signs. This term throughout these notes in recognition that in many patients a true diagnosis of idiopathic inflammatory disease cannot be made with a high degree of certainty.

**Historical and clinical findings**

The small intestine is the main site of digestion and absorption of dietary nutrients. Disease in the small intestine may result in decreased ability to digest dietary nutrients (maldigestion syndromes) or decreased ability to absorb the products of digestion (malassimilation). In most small intestinal diseases both of these processes, i.e. maldigestion and malassimilation are present to varying degrees. Normal digestive function in the small intestine relies on the maintenance of a normal luminal pH, presence and function of normal brush border enzymes on the microvilli and the maintenance of normal tight-junction function, amongst other critical factors. Any disease process affecting the small intestine may potentially interfere with one or more of these processes, leading to clinical signs of small intestinal disease.

Typically small intestinal disease leads to clinical signs of diarrhea and weight loss. Reduced absorptive surface area in the small intestine, decreased brush border enzyme activities and compromise of the epithelial tight junctions may all decrease the ability of the small intestine to absorb the products of digestion. The products of digestion are typically small molecules, and with the loss of the ability to absorb these molecules effectively there is an increased osmotic pull into the small intestinal lumen, leading to increased fluidity of the small intestinal content. This is manifested as diarrhea in most cases. As there is a failure of small intestinal absorption and increased net water losses from the small intestine, small intestinal diarrhea typically manifests with large volume bowel movements, the total volume of feces passed in the day is increased. In animals where the disease is isolated to the small intestine, the large intestine may be able to increase its water absorptive function and produce feces with only mildly increased water content. Clinical signs such as tenesmus, urge incontinence and excessively frequent defecation are more suggestive of large intestinal disease. In many patients, however, both the small and large intestine are involved in the disease process and a mixture of small and large intestinal diarrheal signs may be seen.

If the large intestinal water absorptive capacity is able to cope with the increased fluid input from the small intestine, diarrhea may not manifest and the major clinical sign may be weight loss. Vomiting and inappetance are also common complaints in animals presenting with small intestinal disease. In many animals there is an increased appetite and polyphagia, a consequence of the decreased efficiency of utilization of nutritional input. Clinical signs of weight loss or failure to thrive, polyphagia and large volume diarrhea are also typical of exocrine pancreatic insufficiency, and it is important to conclusively rule this condition in or out during the diagnostic assessment of an animal with a chronic enteropathy.

**Approach to the assessment of a chronic enteropathy patient**

Routine biochemistry and complete blood count panels are indicated in the initial approach to a chronic enteropathy patient. As there are no specific tests that directly assess the small intestine in either of these panels, it is not unusual for a patient with a chronic enteropathy to have a complete lack of abnormal findings. This step is important, however, to assess the physiology of the patient,
screen for signs of other disease processes that may result in weight loss and diarrhea, and as a preliminary step before anesthesia in many patients for the collection of biopsy samples. Identification of significant abnormalities in other organ systems (i.e. azotemia/uremia, elevated liver enzyme activities, abnormal Na+ : K+, elevated cholesterol or triglycerides) may prompt the clinician to work up the patient for other diseases.

In addition to routine clinical chemistry and complete blood count, a group of more specialized tests should be considered during the work up of a gastrointestinal disease case. For companion animals with small intestinal diarrhea, the author recommends measurement of serum TLI, cobalamin and folate concentrations. These tests are described in more detail below. Unless pancreatic inflammatory disease is suspected, the addition of a pancreatic lipase immunoreactivity assay (Spec cPL/Snap cPL or Spec fPL/Snap fPL) rarely advances the diagnosis and is not recommended for most chronic enteropathy patients as a first line diagnostic test.

Direct, specific assessment of the small intestine is complicated by the difficulty of accessing the tract for collection of samples. Ideally, a marker for small intestinal disease should be sensitive (i.e., detect most diseased patients) and specific (able to rule out disease in normal patients). At this time there are no non-invasive diagnostic tests for small intestinal disease that are both highly sensitive and highly specific. The most commonly used, minimally invasive tests currently available involve the measurement of serum concentrations of specific water-soluble vitamins to establish a lack of mucosal absorption. Specifically, the serum concentrations of cobalamin and folate can be measured, and abnormalities in these vitamins may indicate the presence of mucosal disease. The practical assessment of “The GI Panel” in dogs and cats with chronic enteropathies is discussed in more detail in separate proceedings for this meeting.

**“Inflammatory bowel disease”**

Inflammatory bowel disease is one of the most commonly diagnosed, and likely one of the most commonly missed, small intestinal diseases in the dog. Ironically, this condition is also likely over-diagnosed, or diagnosed inappropriately in animals that have not had a sufficiently thorough work up. In essence, inflammatory bowel disease is an idiopathic diagnosis based on histological findings, which means that we really should not be making the diagnosis of idiopathic IBD without intestinal biopsies and a complete, quite stringent work up to exclude other diseases (such as food responsive and antibiotic responsive enteropathies) that have similar histopathologic appearances. Intestinal inflammation falls into a variety of histological categories. These types are summarized in Table 1 below.

<table>
<thead>
<tr>
<th>Histological Category</th>
<th>Frequency</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytic/Plasmacytic</td>
<td>~60%</td>
<td>The most common form of inflammatory bowel disease diagnosed in dogs nationwide. Often idiopathic, but the same histological changes are seen with dietary intolerance/allergy.</td>
</tr>
<tr>
<td>Eosinophilic</td>
<td>~15%</td>
<td>As with LP disease, may be idiopathic or associated with dietary intolerance/allergy. Often associated with GI parasitism. Relatively common in the Rottweiler. Anecdotally, more common in the Pacific Northwest than elsewhere.</td>
</tr>
<tr>
<td>Neutrophilic</td>
<td>&lt;5%</td>
<td>Common in humans, often antibiotic- or probiotic-responsive.</td>
</tr>
<tr>
<td>Granulomatous</td>
<td>Rare</td>
<td>In veterinary medicine, granulomatous colitis of boxers is the most common manifestation of granulomatous intestinal inflammatory disease encountered.</td>
</tr>
</tbody>
</table>

Inflammatory bowel disease is, by definition, a histological diagnosis. The majority of cases diagnosed with inflammatory enteropathies via intestinal biopsy are lympho-plasmacytic or eosinophilic, with some regional variations in prevalence. Unfortunately there is a lack on consensus on histological descriptions and definitions of the severity of disease amongst veterinary histopathologists. Histopathology scores, as currently assigned, correlate poorly with the clinical severity of inflammatory bowel disease in canine patients and suffer from poor inter- and intra-observer consistency. Histopathology remains useful for definitive diagnosis of an inflammatory disease process and identification of differential diagnoses such as lymphoma or lymph drainage abnormalities such as lymphangectasia.

**Therapeutic planning in patients with chronic enteropathies**

On diagnosis of an inflammatory enteropathy, therapeutic planning can take place. A remarkably large proportion of dogs with a diagnosis of lymphocytic/plasmacytic enteritis will show at least partial food responsiveness. A dietary exclusion trial is indicated in most cases, the author’s preference is for use of a novel protein source diet selected on the basis of a thorough dietary history. Partially hydrolyzed and modified antigen diets may also be beneficial. The patient is started on the elimination diet exclusively for a minimum period of 14 days. In most patients with diet-responsive disease a notable improvement in clinical signs will have occurred at 14 days, and those that have failed to show a good response are unlikely to show much benefit from a longer period on the diet. If the dog responds to the diet change, the diagnosis becomes one of food-responsive diarrhea or dietary intolerance. “Dietary allergy” implies...
Dogs failing to respond to dietary modification, particularly when a rigorously controlled elimination diet trial has been carried, out are diagnosed with idiopathic inflammatory bowel disease. The mainstay of therapy for idiopathic inflammatory bowel disease is anti-inflammatory to immune suppressive drug therapy, typically with glucocorticoids. Prednisone or prednisolone is started at doses of approximately 2mg/kg per os SID, typically in the morning, for at least 14 days. If clinical signs are well controlled at this time, a gradual taper to the minimum effective dose is begun, with dose reductions of approximately 25% per week. In some animals, more aggressive immune suppressive therapy may be required. Azathioprine (2mg/kg SID to every other day) has traditionally been the next step for immune suppression, this drug requires several weeks to manifest full effect. Recent publications have examined the utility of cyclosporine-A in therapy of dogs with refractory IBD and saw benefit in many patients, however expense may limit the use of this drug in some patients.7 Drugs such as mycophenolate mofetil and leflunomide have been investigated for use in these patients, but to date limited information is available regarding their efficacy.

Most patients respond well to dietary change and judicious use of anti-inflammatory or immune-suppressive medical therapy and have a fair to good prognosis. Some animals, unfortunately, show more refractory disease and the prognosis is more guarded. In a review of risk factors for adverse outcomes with inflammatory bowel disease in dogs, 18% of patients were eventually euthanized due to intractable disease.8 High clinical severity scores, severe changes visible on endoscopy, low serum cobalamin and hypoproteinemia were all associated with a higher likelihood of an adverse outcome.8 Where low serum cobalamin or folate concentrations are detected, supplementation of these vitamins is recommended.

“Ecological” diseases – “SIBO”, ARD and tylosin-responsive diarrhea

The small intestine is home to a large, diverse population of bacteria and other microflora. The microflora is essential to the normal anatomy and physiological function of the gastrointestinal tract, and abnormalities in this microflora are commonly associated with clinical signs of disease. The total number of organisms present in the canine GI tract has been a point of controversy. The term “Small Intestinal Bacterial Overgrowth” (SIBO) was originally defined in the context of culture studies that defined the upper limit of normal for the bacterial population of the duodenum as 10^5 colony-forming units/ml.2 We now know from more recent culture-based and genetic diversity studies of the canine intestinal microflora that these original culture studies likely underestimated the numbers and diversity of bacteria present.9 The term “antibiotic-responsive diarrhea” is gaining currency in the recent literature, recognizing the antibiotic responsive nature of this condition without applying arbitrary criteria regarding expected bacterial numbers.

Disturbances in the gastrointestinal flora are likely common in dogs with other chronic enteropathies, such as idiopathic IBD or dietary intolerances. The maintenance of the normal flora relies on normal mucosal function, secretory function of the stomach and pancreas and gastrointestinal motility. Any of these functions may be abnormal in dogs with small intestinal disease, leading to an abnormality in the gastrointestinal flora.

In many dogs with chronic enteropathies, therapy directed against the bacterial flora may be advantageous. As the disturbed flora is typically secondary to some other disease process, and these primary disease processes are usually managed rather than cured, long durations of therapy are necessary. Given the need for repeated or chronic therapy, alternative approaches via dietary modification or supplementation are desirable. Potential modalities include supplementation with probiotic compounds, such as fructooligosaccharides or inulin, that are preferentially fermented by “beneficial” organisms (typically Lactobacillus or Bifidobacterium spp). Empirically, diets containing these compounds (for instance, many of the “intestinal health” diets) are often helpful in managing dogs with chronic enteropathies. Probiotic organisms may also act via a displacement mechanism, and in some cases they are of benefit. There is a plethora of probiotic products on the market with very little data from well-controlled studies. A more traditional approach would be to use antibiotic medications. The author’s preference is to use tylosin (Tylan Powder™, Elanco), at a dose of 20-25 mg/kg per os BID. Treatment is given for a minimum of 6 weeks, however it is not unusual for clinical signs to recur within weeks of antibiotic withdrawal. In some patients a pulse therapy approach, with 2 weeks on and 1 week off tylosin, is able to control clinical signs adequately. Some patients require constant, lifelong tylosin therapy.

References
It is well known that clinical chemistry analytes vary naturally over time, both as a result of disease processes and due to intrinsic biological variation within the individual. Variation occurs at the level of the individual, as a result of analytical imprecision, and within groups of individuals. The degree of within individual variation is itself quite variable, some analytes showing marked changes over time, while other analytes are under more rigid homeostatic regulation and thus vary less within an individual.

In both human and veterinary medicine the diagnosis and management of chronic disease conditions is becoming increasingly important. As a profession we promote the idea of “screening blood tests” performed on a regular basis. The American Animal Hospital Association (AAHA) Senior Care Guidelines for Dogs and Cats, for instance, recommend regular clinical chemistry panels for “senior” dogs, defined as dogs in the last 25% of their expected lifespan. Similarly, the American Association of Feline Practitioners-AAHA Feline Life Stage Guidelines recommend a panel of clinical chemistry parameters that are considered part of the “minimum database” for regular assessment of mature and elderly cats. An implicit assumption in these recommendations is that clinically meaningful changes will be detectable, and that early, beneficial interventions will be prompted by the detection of changes. In order for the clinician to accurately gauge the presence and importance of changes in these “screening” biochemistry panels, we need a reasonable understanding of just how much these tests change over time in an individual, and just how great a change needs to be documented to most likely reflect a change in the patient’s physiology.

Concepts and terminology of biological variation

When considering biological variability and how it impacts the use of serial blood work, there are two important concepts that need to be understood: the Index of Individuality and the Reference Change Value. The index of individuality of any particular test is derived from the ratio of variation over time in an individual patient to variation within the population as a whole. If a test has a high degree of individuality, results from a patient will tend to cluster together over time, and the results of tests from one patient will often not overlap with results from another patient. If a test has a high degree of individuality, important changes for an individual may be missed if comparing to a broader, population based reference range. This concept is illustrated in Figure 1 (right). In this figure, each dog’s results are tightly grouped within the individual, with little overlap between individuals. The broken lines represent a theoretical reference range based on the 95th percentile of all the data. Note that quite large deviations from Dog B’s “normal”, in either direction, would still fall within the reference range and thus meaningful change may be missed.

Tests with a high degree of individuality are best used by taking serial measurements over time and comparing the individual’s test results to prior values, rather than taking a single measurement and comparing it to a population based reference range.

Most diagnostic tests in veterinary medicine are traditionally compared to population-based reference ranges derived from large populations of “normal” individuals. This means we make an underlying assumption that most diagnostic tests we use have a low individuality and comparison to a reference range is appropriate. There is actually relatively little information regarding the individuality of tests commonly used in dogs and cats in the veterinary literature, but what is available would suggest that a substantial majority of the tests that we use regularly actually have quite high individuality, leading to the unsettling thought that we miss important changes in our patients in spite of regular biochemistry screenings.

If we are monitoring a value over time, we need to have some idea of the magnitude of change that most likely represents a change in the patient’s physiology (either a worsening or an improvement, depending on the context of the testing). This is the Reference Change Value (RCV), which is derived from measurements of the within patient variability and the variability of the measurement technique itself. In most cases, the reference change value is stated as a percentage change, statistically associated with a P value of <0.05; in other words a change of this size has a ~1:20 chance of being random, and thus is more likely due to a change in the patient’s physiology. A change in a test result less than this value, regardless of whether it is “better” or “worse”, is statistically unlikely to represent a real change, and therapeutic decisions should be made with caution if less than this degree of change is seen.
Factors that influence biological variability

As previously indicated most chemistry parameters that we measure in our patients have high individuality, and many have high reference change values. Generally speaking analytes that are actively regulated by some form of physiological homeostatic process will have lower individualities and relatively low reference change values. Examples of substances with low individualities and comparatively low reference change values include serum electrolytes (particularly potassium and calcium) and blood glucose concentrations. This intuitively makes sense; these electrolytes are rigorously regulated by the renin-angiotensin-aldosterone system and parathyroid/calcitonin hormone production respectively, while under normal circumstances glucose is regulated via the insulin/glucagon system within a relatively tight range of values.

Release of “leakage” enzymes, such as alanine transaminase (ALT) and the specific pancreatic lipases is not under any form of homeostatic control, their release into the circulation varies with their rate of synthesis (which may vary with disease), rate of loss from the cells (which may also vary with disease), and may also vary with changes in the clearance mechanisms of these enzymes from the circulation. Consequently, these enzymes often feature a very high degree of within-individual variation, resulting in high reference change values. This has been reported for both pancreatic lipase immunoreactivity and liver enzyme activities in dogs,5,6 and for liver enzyme activities in cats.4

The effect of disease states on biological variability

Disease states can also influence the degree of biological variability, and thus the reference change values for individuals who are already diseased may actually be markedly different from healthy individuals. This has been reported for the cardiac biomarker NT-proBNP in dogs,7 where the reference change value for dogs with mitral regurgitation was estimated at approximately 50%, while the RCV for healthy dogs was nearly 100%, the lower RCV in the dogs with mitral regurgitation was mainly due to lower within-individual variation in that group.

Data on biological variability and reference change values for other enzymes and other disease states are currently lacking for many important diseases and for many enzymes that are routinely measured in practice. While data for biological variation in specific canine pancreatic lipase (Spec-cPL) has been reported for healthy dogs (where a very high RCV of approximately 450% was reported),8 data from dogs with chronic pancreatitis or on the feline specific pancreatic lipase assay (Spec-fPL) in any group of cats (healthy or diseased) are not available at the time of writing.

The importance of biological variability depends upon magnitude of change with disease

While many analytes used in clinical practice have high individuality, suggesting that the application of population based reference ranges is of limited utility, the degree of deviation from normal is often sufficiently high that, when used as a screening test, diseased individuals are still readily distinguished from the normal range. An example would be cardiac troponin-I in individuals with myocardial ischemia, where several hundred-fold elevations in cardiac troponin-I concentrations are regularly documented, a vastly greater change than the estimated RCV (approximately 110% for healthy dogs) for this marker. Similarly, even though the RCV for canine specific pancreatic lipase is approximately 450% (4.5 fold), the cut off value considered consistent with pancreatic disease (> 400µg/L) is actually greater than 4.5-fold higher than the average healthy dog’s Spec-cPL value (which is about 63µg/L). Because of the very large deviations from normal seen with these tests, the implication of their high individual variability is mitigated when using these tests in a clinically appropriate manner to establish a diagnosis. The application of these tests to ongoing monitoring of the disease state post-diagnosis should still be approached with caution, however, until better data regarding RCV’s for these markers in animals with chronic disease is available.

For some tests, the analyzer is the limiting factor

To this point the discussion has mainly been about understanding why remaining aware of biological variation and reference change values are important, and less about how we actually go about getting the values that we are monitoring. This does not mean that how the sample is obtained and how the instruments doing the test are performing is not important, but in most cases we assume that samples are handled correctly and analyzed on machines that are well maintained and calibrated appropriately.

When we are interested in changes that are occurring in substances that show very little intra-individual variation, however, the performance of the analyzer doing the test can actually become very important. The criteria for acceptable analyzer variability when calculating and using reference change values are that the analyzer’s contribution to the variability seen must be less than half of the biological variation (in our case, this is the intra-individual variation).

In one study of biological variation carried out by the author, the performance of three different levels of clinical chemistry instruments were compared using the same set of samples. The instruments tested were a Beckman Coulter AU480 (a very high end-machine used in large clinical pathology practices), a Sirrus chemistry system (middle-range, would be used in a busy human urgent care facility) and the IDEXX VetTest 8008, a common system used in veterinary practices. All of the analytical systems used were precise enough that they could be used to derive reference change values and monitor for changes in all of the analytes measured. The AU 480, interestingly, was insufficiently precise to derive a reference change value for serum cholesterol in the dog, and none of the
three instruments showed sufficient precision to derive reference change values for total calcium.\textsuperscript{5} The implications of these findings are not entirely clear, but they do illustrate that even under the best of conditions and using rigorously maintained and calibrated instruments, our actual ability to actually detect meaningful changes in blood chemistry values is sometimes a lot lower than we would think.

References
Protein loss through the GI tract (protein-losing enteropathy, PLE) presents in a spectrum of severities, ranging from cases with insidious weight loss and mild diarrhea through to highly challenging cases complicated by the development of ascites, peripheral edema and coagulopathies. Recognition of PLE can be quite challenging, particularly in cases where overt hypoproteinemia is not yet present. The presence of PLE in dogs with chronic inflammatory enteropathies has been shown to be a negative prognostic factor in these cases, and warrants more aggressive therapy. While cases with PLE secondary to inflammatory enteropathies tend to be the most challenging to manage, a variety of other mechanisms can result in significant intestinal protein losses. Selection of the appropriate therapy for a PLE case depends upon accurate identification of the pathology present, as differing modes of therapy are indicated for some of the non-inflammatory, structural diseases.

Clinical signs in PLE
Depending on the degree of severity and duration of the disease, clinical signs of PLE can range from the development of ascites, peripheral edema or acute severe respiratory distress due to pulmonary thromboembolism, through to vague signs such as weight loss and poor appetite. If an inflammatory enteropathy is present diarrhea may be noted, but many cases with PLE present with normal stools. Consequently, PLE should be considered as a differential diagnosis for any dog presenting with hypoalbuminemia, even if gastrointestinal signs are lacking.

Mechanisms of protein loss in PLE cases
With some specific breed-related exceptions, the major mechanisms of protein loss in most cases of PLE in dogs (and cats, but this condition is recognized infrequently in that species) can be divided into two major groups: primary lymphatic drainage abnormalities such as lymphangectasia, lipogranulomas or mesenteric lymphatic obstructions, and secondary to mucosal inflammatory disease. While these major categories account for the majority of PLE cases encountered in clinical practice, gastrointestinal ulcers, significant gastrointestinal parasitism and gastrointestinal neoplasms (both lymphosarcoma and carcinoma) can also result in marked GI protein loss. Intestinal motility accidents, such as intussusception, partially obstructing foreign bodies, and diverticulae can also result in quite marked protein loss, however their clinical signs and initial examination findings are usually sufficiently distinct from the more typical PLE cases to allow rapid identification.

Breed predispositions
While any dog can present with PLE, particularly as a complication of severe inflammatory enteropathies, some breeds are recognized to have particularly marked breed predispositions. Often the manifestations of PLE in these breeds are quite severe.

Lymphatic drainage abnormalities such as lymphangectasia have been reported in the Rottweiler, Yorkshire terrier, Shar-pei and the Maltese terrier. In the Rottweiler lymphangectasia commonly accompanies eosinophilic inflammatory disease of the intestine, but a direct relationship between these two conditions has not been established and both diseases are commonly diagnosed in this breed in isolation.

As well as primary lymphangectasia, the Yorkshire Terrier is commonly diagnosed with significant inflammatory lesions of the intestinal crypts. These crypt lesions are commonly associated with severe gastrointestinal protein loss. The mechanism of crypt abscess formation in these dogs has not been well defined, but there does not appear to be any association with bacterial invasion.

The Norwegian Lundehund, a relatively rare dog breed in the USA, has been reported to have 50% or more of individuals affected with intestinal lymphangectasia, with associated PLE.

The Soft-coated Wheaten terrier presents with a breed specific PLE and protein-losing nephropathy that can be quite challenging to manage. This disease has been linked to food hypersensitivities in this breed, and is apparently worsened by high level exposure to gluten and other dietary allergens, but does not appear to be a true gluten hypersensitivity. Soft-coated Wheaten terriers may present with protein-losing enteropathy alone, protein-losing nephropathy alone, or may have both conditions simultaneously.

Diagnostic approaches to a suspect PLE case
In most dogs with PLE, the suspicion that this disease is present first arises when a low serum albumin concentration is detected. This may be noted as part of the work up for chronic gastrointestinal disease, but as noted above many dogs with significant PLE may first present with ascites or edema and with little in the way of gastrointestinal signs, thus the presence of a low albumin should always prompt the consideration that PLE may be present.
When presented with a case with low albumin (typically <2.0g/dL), we have a relatively limited number of ways in which this could have come about. Major routes for protein loss include via the kidneys (protein-losing nephropathies), through significant skin lesions or open wounds, through the gastrointestinal tract, and as a result of hepatic synthetic failure/hepatic insufficiency.

A rational approach to a suspect PLE case with hypoalbuminemia, then, is to screen for and rule out as many of these conditions as possible. Protein losses due to cutaneous lesions or open wounds can be readily ruled out via physical examination, as the extent and severity of these lesions necessary to cause this volume of protein loss is quite dramatic. Protein losses due to protein-losing nephropathies can be ruled out by the detection of a normal protein-creatinine ratio. It is important to remember that lower urinary tract infections, or any other disorder leading to an active urinary sediment, can also cause an elevation in urinary protein:creatinine, screen for and if necessary treat urinary tract infections first before assessing this test for evidence of a protein-losing nephropathy.

Detection of hepatic insufficiency and synthetic failure can be more complicated, as liver enzyme activity elevations and hyperbilirubinemia are not present in many animals with end stage liver disease. Other common clinical chemistry findings with hepatic failure, such as low cholesterol and blood urea nitrogen, are also common findings with PLE.\(^2\)

The best method for non-invasively assessing hepatic function is to perform a pre- and post-prandial bile acids tests. Normal pre- and post-prandial bile acid concentrations rule out hepatic insufficiency as a cause of hypoalbuminemia with a high degree of certainty. Some clinicians will measure only the resting, pre-prandial bile acid concentration, but this reduces both the sensitivity and specificity of this test. Some animals with synthetic failure are still able to clear bile acids to a normal pre-prandial value while fasting, and evidence of loss of hepatic function is only seen after the bile acid challenge. Alternatively, animals with gastrointestinal disease can show mildly to moderately elevated pre-prandial bile acids due to the reduced efficiency of clearance of bile acids from the portal circulation that have undergone bacterial deconjugation.\(^1\)

In a suspected PLE case with hypoalbuminemia, where skin disease and protein-losing nephropathy have been ruled out and pre-/post-prandial bile acid tests are normal, the diagnosis of PLE can be made with a high degree of certainty through simple exclusion.

The diagnosis of PLE in dogs that have not reached a state of overt hypoalbuminemia is more challenging. This can be quite important in dogs from the breeds previously listed with predispositions for PLE, as some of the adverse outcomes of PLE such as hypercoagulability and thromboembolic potential can manifest before the albumin is markedly low, and thus in these breeds early diagnosis and management is important.

Unfortunately, albumin itself cannot be detected in stool samples as it undergoes bacterial degradation. A surrogate for albumin losses, α-1 Proteinase Inhibitor (α-1PI), is able to survive transit through the GI tract and can be detected in fecal samples.\(^13,14\) This protein has a very similar molecular mass and charge to albumin, thus elevated α-1PI in stool samples is suggestive of increased albumin loss into the GI tract. This test is somewhat complicated, requiring multiple stool samples to be collected and stored frozen, and is only available from one laboratory (The GI Lab at Texas A&M). The author typically only uses this test in the previously mentioned, predisposed breeds, particularly if they present with weight loss and mild GI signs without hypoalbuminemia. The majority of PLE cases seen in our clinic are diagnosed by exclusion, as detailed above, and a combination of diagnostic imaging and endoscopic biopsy findings.

**Diagnostic imaging findings with lymphangectasia of the intestine.**

Abdominal ultrasound examination is a valuable modality for the assessment of dogs with potential PLE. As well as giving some idea of the hepatic size and potentially revealing the presence of low volume ascites, occasional dogs will show characteristic hyperechoic striations in the intestinal mucosa, often referred to as a “tiger stripe pattern”. This finding is strongly suggestive of intestinal lymphatic dilation, either due to lymphangectasia or distal lymphatic obstruction.\(^13\)

**Therapy for PLE: “Uncomplicated” cases**

As the mechanism of protein loss in many “uncomplicated” cases of PLE revolves around loss of intestinal lymph, strategies to reduce lymph loss are useful in the management of PLE cases. The major driver of intestinal lymph production is the intake of dietary fat. (Recall that the intestinal lymphatics are called “lacteals” due to the presence of high concentrations of fat in chylomicrons). Thus the use of extremely low fat diets is recommended in most dogs with PLE, and is the mainstay of treatment for most dogs with primary lymphangectasia.\(^2\) The use of ultra-low fat diets has been shown to be effective in dogs with lymphangectasia that had failed to respond to glucocorticoid therapy or showed a relapse as glucocorticoid doses were reduced.\(^16\)

The author’s first choice of diet for management of relatively uncomplicated PLE cases is typically one of the commercially manufactured, ultra-low fat diets such as Royal-Canin’s LF or Hill’s I/D-LF (NB: the I/D low fat formulations. I/D GI health is too high in fat). Alternatively, home cooked diets have been described for dogs that are also extremely low in fat, and with careful attention to vitamin and mineral supplementation can be used long-term.\(^2\)

Many dogs with PLE due to lymphatic drainage abnormalities within the mucosal will also develop lipogranulomas or other inflammatory lesions in the mesenteric lymphatics. Patients showing only partial response to ultra-low fat diets after 2-3 weeks of
therapy will often benefit from the addition of prednisone at 1-2 mg/kg/day. This will also assist in management of secondary inflammatory disease in these patients.

Therapy for PLE: “Difficult” cases

“Difficult” PLE cases fall into two main groups: severe disease with marked hypoalbuminemia, ascites and/or edema, and cases where the PLE is a complicating factor for other diseases, such as severe inflammatory enteropathies.

Cases with markedly severe hypoalbuminemia represent a significant therapeutic challenge. Ideally, colloid oncotic support should be given before any invasive diagnostic intervention, to reduce the risk of wound dehiscence and anesthetic complications due to embolic events. Fresh frozen plasma transfusions can be very useful, as they replace clotting factors as well as albumin, but the volumes of plasma necessary to replenish albumin in many dogs can become cost prohibitive. If available, 20% human albumin solutions can provide rapid oncotic support at relatively low cost and risk in dogs, but this product is often difficult to obtain. Synthetic colloids may also be used, often in combination with fresh frozen plasma.

Ascites fluid is usually not drained, except if the volume of fluid is sufficient to cause respiratory compromise. Only sufficient fluid should be drained to relieve respiratory compromise. Removal of large volumes induces a large body-wide protein deficit in the patient that will promote a catabolic state and cachexia, and also activates the renin-angiotensin-aldosterone system to normalize blood pressure following substantial volume loss, this can increase blood pressure and increases the rate of further ascites accumulation. Animals who are recurrently effusive may benefit from diuretic therapy.

Animals with this degree of PLE are usually assumed to be hypercoagulable. This hypercoagulable state persists in many dogs after therapy that increases albumin, and thus these dogs should be considered at long-term risk for thromboembolic complications.

The presence of significant PLE in dogs with inflammatory enteropathies is a poor prognostic sign, and the early use of more aggressive immune suppression therapy is indicated. Interestingly, in a recent study of dogs with inflammatory enteropathies and PLE, patients treated with chlorambucil-prednisolone showed a better outcome than those treated with azathioprine-prednisolone. The reason for this difference is not clear, but at least in this one study the differences in outcome were quite dramatic (azathioprine-pred group had a median survival of 30 days, while chlorambucil-pred group did not reach a median survival as 10/14 were still alive at the end of the study).

References


Recognition and management of gastrointestinal protozoal infection can be challenging for the companion animal practitioner. These organisms are often present in relatively low numbers, may show episodic shedding, asymptomatic carrier states are quite common, and the organisms are quite fragile and may be damaged or destroyed when using routine fecal flotation solutions. Clinical signs of disease with these organisms are also remarkably variable, and may depend upon the presence of other comorbidities or immune compromise in the host. To add to the difficulty of managing these cases, the enteric protozoa are eukaryotes and often depend upon the host to supply nutrients, thus they are resistant to many commonly used antimicrobials. Marked strain and geographical variances in apparent pathogenicity are seen. In animals with symptomatic infections an integrated approach to treatment of the affected animal, other in-contact animals and the housing environment are necessary to achieve the best chance at eradication, but even with diligent treatment of both animal and environment reinfection is common. The purpose of this presentation is to review the diagnostic and clinical approach to two common and important gastrointestinal protozoal groups, the Trichomonads and Giardia. Additionally, this presentation will discuss evidence for emerging protozoan infection in humans, Blastocystis, as either a pathogen or zoonotic threat in companion animals.

Feline intestinal trichomonosis
Trichomonosis is a rapidly emerging feline disease in cats that is caused by the protozoal pathogen, Tritrichomonas foetus. T. foetus was definitively identified as a cause of waxing and waning large bowel diarrhea in cats in 2003 and since then its prevalence has been recognized worldwide. The prevalence of T. foetus can be quite high (>30%) especially in young purebred cats that are maintained in high density housing environments (e.g. shelters, catteries). However, older, mixed breed cats have also been identified with the infection. No breed of cat is known to be resistant to infection. The pathophysiology of feline trichomonosis is largely unknown. The route of infection is presumed to be fecal-oral (e.g. sharing of litter boxes and mutual grooming). Following transmission, the trichomonads can be cleared by the feline host or can persist in the ileum and large intestine. Infected cats can be subclinical carriers or can develop signs of large bowel diarrhea.

Clinical presentation
The most characteristic clinical sign of feline trichomonosis is chronic waxing and waning large bowel diarrhea that is frequently malodorous with the consistency of cow patty feces. Occasionally, frank blood and/or mucus may be observed in the diarrheic feces. Young, severely affected cats may also have signs of proctitis, fecal incontinence and/or rectal prolapse. Although exclusively large bowel diarrhea is a more common finding, infected cats may also exhibit small bowel signs including weight loss, anorexia and/or vomiting. T. foetus-induced diarrhea may resolve with antibiotic administration but returns following discontinuation of antimicrobial therapy. Untreated cats may develop new onset signs (or demonstrate relapse) following stressful events (diet change, new cat introduced into household, etc). Infected cats generally present in good body condition unless they are immunocompromised (young, FELV/FIV, etc) or have a comorbidity.

Testing for T. foetus is recommended for any cat with large bowel diarrhea, particularly young purebred cats, cats raised in a high-density housing facility, and/or cats that have developed diarrhea following introduction of a new cat into the household. There are several diagnostic tests that are useful in detection of T. foetus infection. The best sample to evaluate for the presence of T. foetus is one obtained by a colonic flush (video available at: http://www.youtube.com/watch?v=JMfZ9M80V8E) however, fresh fecal samples obtained by fecal loop or immediately after voiding are acceptable. Feces should be diarrheic and antibiotics should be discontinued a minimum of 14 days prior to testing.

Cats can be evaluated for T. foetus in several ways. Direct smear examinations of freshly voided, diarrheic feces may reveal mobile trophozoites. The motility of the trophozoites may be used to distinguish T. foetus from Giardia (T. foetus has a “tumbling” motion, Giardia looks like a falling leaf). Fecal culture in a pouch system, originally developed for diagnosis of venereal trichomonosis in bulls, can increase the sensitivity of diagnosis, but may take several days to a week for clear results, and are susceptible to bacterial overgrowth that results in loss of the trichomonads and a false negative finding. PCR examination of fecal samples is the current gold-standard test for this parasite. Samples are stable for prolonged periods at room temperature when preserved with isopropyl alcohol at 1:1 volume.

No diagnostic test has 100% sensitivity. Negative test results should be interpreted with caution. Repeated testing is advised if a strong clinical suspicion exists in the face of a negative test result. Giardia is often confused for T. foetus and vice versa. Cats that do not respond to appropriate anti-giardial therapy or that have exclusively large bowel signs should be tested for T. foetus. Co-infections are common in T. foetus-infected cats. Thus, additional enteric infectious disease testing (e.g. Giardia spp, coccidia) is recommended in cats testing positive for T. foetus. Other chronic causes of large bowel diarrhea (antibiotic-responsive diarrhea, inflammatory bowel...
disease, enteric parasitic and fungal infections such as histoplasmosis if appropriate differential for the area, colonic neoplasia, etc.) should also be included among the differentials.

**Therapeutic plan**

Infected cats can often be successfully treated with ronidazole (30mg/kg PO q24hr for 14 days). Neurotoxicity can be a side effect of treatment however most reported cases of ronidazole toxicity occurred when cats were given higher than recommended daily doses of drug. Nevertheless, cats should be carefully monitored during treatment for signs of neurotoxicity. Doses above 30mg/kg SID are not recommended.

If diarrhea continues well-beyond ronidazole treatment, cats should be retested for T. foetus infection. A positive result may be attributed to resistant infection, poor owner compliance, re-infection and/or improper drug compounding or ineffective dose. Untreated cats may experience resolution of clinical signs however they often remain infected and can spread the organism to previously uninfected cats. Therefore, if concern for transmission to uninfected cats exists, the author recommends that infected cats be treated or be isolated from uninfected cats until shedding ceases (based on multiple negative PCR results).

**Giardiasis**

*Giardia duodenalis* infections are highly prevalent in dogs and cats. Reported prevalence rates are dependent on the sampling method used but are likely around 12-15% in cats and dogs. The route of *Giardia* transmission is fecal-oral usually via ingestion of environmentally stable cysts present in contaminated water and/or food. Once transmitted, trophozoites colonize the small intestine. Infected animals can become subclinical carriers or can develop small intestinal diarrhea. The prolonged survival of the cyst form in the environment represents a particular challenge for long-term management, as many patients will be living in highly contaminated environments, or may be exposed to reinfection by visiting community parks and other public spaces.

**Clinical presentation**

Clinical signs of *Giardia* spp. infection vary widely among infected animals. Many animals are subclinical carriers and do not exhibit signs of disease. Susceptible animals (e.g. young, immunocompromised, shelter-housed) may develop severe and/or chronic signs of disease. Clinical signs may include abdominal pain, acute self-limiting diarrhea, chronic diarrhea and/or weight loss. In human beings a “post-*Giardia*” irritable bowel syndrome has been recognized. This syndrome is associated with increased intestinal motility, functional dyspepsia, gassiness and abdominal pain and may persist for several months beyond the original diagnosis of giardiasis.

**Diagnostic plan**

There are a variety of *Giardia* diagnostic tests available. The most widely available diagnostic tests for the private practitioner are direct fecal smear, fecal flotation, *Giardia* SNAP ELISA and IFA.

As mentioned with the intestinal trichomonads, co-infection with *Giardia* is common. In animals with “resistant infection”, underlying diseases such as inflammatory bowel disease may be present. Thus, infected animals should be evaluated for the presence of mixed infection and/or concurrent intestinal disease.

**Treatment**

Many animals will have self-limiting diarrhea, which may not necessitate treatment. If treatment is required, fenbendazole (50 mg/kg q 24 h for 5 days) may be effective especially when concurrent infection with nematodes or cestodes is present. In patients with documented recurrent, a second course of fenbendazole 14-21 days after initial treatment and diligent attention to environmental control are crucial. Metronidazole (15-25 mg/kg q 12 h for 5-7 days) may also be used unless concern for neurotoxicity exists. Bathing the animal and changing the animal’s environment on the last day of each round of treatment should be performed if possible.

**Blastocystis**

*Blastocystis* spp is a highly diverse group of enteric protozoa. These organisms are the most common enteric parasite identified in human stool samples within the USA, with marked regional and seasonal variations in prevalence noted. The pathogenicity of *Blastocystis* is an area of some controversy in the human literature, however infection with this organism is commonly noted in human patients with IBS symptoms. Initial descriptions of *Blastocystis* gave species names to the organisms reflecting the species from which they were isolated (i.e. *B. hominis, B. ratti*), however it is now recognized that genetic diversity within these species is very high, and thus consensus has developed around categorization of these organisms via numbered subtype (analogous to the concept of infectious assemblages in *Giardia*). At the time of writing at least 14 distinct subtypes have been reported, with new additional new subtypes being regularly identified.

**Diagnosis**

In human beings the most commonly used method for diagnosis of *Blastocystis* carriage is via direct light microscopy of either unstained, wet mount or stained fecal preparations. Enrichment culture and fecal PCR are also commonly used, with fecal PCR methods having the additional advantage of allowing subtype identification via sequencing of the small subunit ribosomal RNA gene. Fecal PCR has greater sensitivity than direct light microscopy.
Prevalance of blastocystis in companion animals, zoonotic potential?

Within the veterinary and parasitology literature there is a remarkable dearth of data regarding any link between Blastocystis carriage and clinical signs of disease in companion animals. A solitary case report describes a significant parasite burden in a mixed-breed dog with vomiting, diarrhea, and weight loss. This animal was definitively diagnosed with exocrine pancreatic insufficiency and marked hypocobalaminemia, which is sufficient to explain the observed clinical signs.13

The true prevalence of Blastocystis carriage in companion animals is unclear at this time, with most of the parasitology literature in this area focused on the domestic dog. Early data from subtropical and tropical environments suggested quite high carriage rates for Blastocystis in dogs, with rates as high as 70% reported for carriage in shelter-resident dogs in a subtropical environment, although this finding has been thrown into some doubt by recent PCR-based studies from the same environment. In a study of asymptomatic individuals and pet animals living in the same household as humans symptomatic for Blastocystosis (n=11), 8/8 in-contact animals (5 dogs, 3 cats) were positive for Blastocystis via fecal PCR, with 7/8 of the in-contact animals carrying the same strain as the symptomatic in-contact human. Interestingly, none of the in-contact animals or in-contact humans (n=17) in this, admittedly small, study showed clinical signs consistent with Blastocystosis. These data suggest that transmission between humans and companion animals is possible, however this is most likely a transient phenomenon and it appears unlikely that domestic dogs and cats represent a significant risk for zoonotic transmission.

Selected references (additional references provided on request)
The components of the “GI panel”

Most clinicians, when referring to a GI panel, are referring to the measurement of serum concentrations of specific pancreatic lipase immunoreactivity (Spec-cPL in the dog, Spec-fPL in the cat), serum trypsin-like immunoreactivity (cTLI in the dog, fTLI in the cat), and the serum concentrations of two water soluble, B-group vitamins, cobalamin (Vitamin B₁₂) and folate (Vitamin B₉). Together, these compounds can provide valuable information regarding the presence and localization of disease in the pancreas and small intestine, and they may also suggest the need for therapeutic supplementation. The normal physiology and significance of abnormalities of these compounds are discussed individually below. While most clinicians will use the full panel of all four compounds, particularly in cases where clinical signs are vague or inconsistent, in some situations it can be cost effective to measure only one of the pancreas markers. For instance, if the clinical suspicion is of exocrine pancreatic insufficiency in a dog, due to the presence of compatible clinical signs, little additional value is obtained from measuring Spec-cPL. cTLI is the test of choice. Equally, in a dog with a strong suspicion of pancreatitis there is usually little additional value in measuring cTLI and Spec-cPL is the test of choice. In the cat, however, the clinical signs and histories of both exocrine pancreatic insufficiency and pancreatitis are sufficiently vague and non-specific that it is generally advisable to at least initially measure both fTLI and Spec-fPL in this species.

Trypsin-like immunoreactivity (cTLI, fTLI)

The serum concentration of trypsin-like immunoreactivity represents the presence of (mainly) trypsinogen and (rarely) active trypsin in the circulation. Trypsinogen, the zymogen precursor to active trypsin, is essentially exclusively synthesized in the pancreatic acinar cells, where it is packaged in secretory granules before excretion in to the pancreatic duct system. Pancreatic acinar cellular damage, for instance with pancreatitis, can result in the loss of trypsinojeg into the pancreatic interstitium and circulation, resulting in a higher than normal concentration. Loss of acinar cell mass, as occurs in both pancreatic acinar atrophy in dogs and as an end stage of chronic pancreatitis in dogs and cats, can result in subnormal concentrations of TLI. Detection of a serum TLI concentration ≤2.5µg/L is highly sensitive and specific for the diagnosis of exocrine pancreatic insufficiency in the dog. In the cat, a serum fTLI concentration ≤8 µg/L is suggestive of exocrine insufficiency. Values within the reference range, even if “low normal”, rule out exocrine insufficiency due to reduced functional acinar cell mass.

Elevations in serum TLI will be seen in some animals with acute pancreatitis. Serum TLI concentrations rise rapidly early in the course of acute inflammatory disease of the pancreas, but also return to baseline relatively rapidly, and are typically at or slightly below baseline values within 48 to 72 hours after the onset of a bout. Consequently, a normal serum TLI concentration does not reliably rule out the presence of inflammatory pancreatic disease.¹ In the context of the GI panel, the greatest utility of the serum TLI concentration lies in the diagnosis or ruling out of exocrine pancreatic insufficiency as a cause of small intestinal diarrhea.

In some cats the serum fTLI concentration is mildly to moderately elevated, even though the clinical signs reported (diarrhea, weight loss) are more consistent with small intestinal disease. In many of these cats, serum Spec-fPL concentrations are normal. While the mechanism underlying this pattern of results is uncertain, it likely relates to a loss of normal negative feedback from the small intestine to the pancreas. This particular pattern of results (high TLI, normal PLI) in the cat is strongly suspicious of small intestinal disease, and warrants assessment of the serum cobalamin and folate concentrations.

Specific pancreatic lipase (Spec-cPL™, Spec-fPL™)

As with trypsin/trypsinogen, specific pancreatic lipase is synthesized only in the exocrine pancreas. Release of enzymes into the circulation is via leakage, and increased release is generally held to be consistent with acinar cellular damage occurring during pancreatitis. Generally speaking, serum concentrations of PLI show greater magnitudes of increase and longer durations of elevation above baseline than TLI in the same patient.

Detection of elevated serum concentrations of specific pancreatic lipase (fPLI or Spec-fPL) has a higher reported sensitivity and specificity than fTLI for diagnosis of pancreatitis in the cat. In one study, where fTLI achieved overall sensitivity and specificity of 28% and 82%, respectively, fPLI achieved overall sensitivity and specificity of 67% and 67%, respectively.² In the same study, sensitivity of fPLI for the diagnosis of “moderate to severe” pancreatitis was 100%. A larger study (n=182 cats) of the Spec fPL assay reported an overall sensitivity for this test of 79%, with a specificity of 82% for detection of pancreatitis in this group.³ Overall, the Spec-fPL assay has the highest currently reported sensitivity and specificity of any diagnostic modality for the detection of pancreatitis in the cat.¹

While pancreatic lipases are highly specific for the exocrine pancreas, the normal range of these assays in both dogs and cats includes values close to or equal to zero. Consequently, the Spec-c/fPL assays cannot be used to diagnose exocrine pancreatic
Insufficient. The main utility of the pancreatic lipase concentrations lies in the detection of exocrine pancreatic inflammation in both species, this is particularly valuable in the cat as clinical signs of pancreatitis in this species are often subtle or vague.

**Serum folate**

Folate is a water-soluble, B-group vitamin (Vitamin B₉) that is abundant in most small animal diets. As dietary deficiency of this vitamin is highly unlikely, the serum concentration of folate is an indicator of the small intestinal absorptive capacity for this vitamin. Folate monohydrate, the major form of folate absorbed from the small intestine, is absorbed exclusively via a receptor mediated process in the duodenum, thus a low serum folate concentration suggests a lack of duodenal receptors, and implies duodenal mucosal disease with a very high specificity.

Folate availability from the GI tract can be increased in some disease states. Many intestinal bacteria, including some *Lactobacillus* spp and representative flora from the large intestine, are net synthesizers of folate and release significant quantities of folate into their environment. In the dog an increased serum concentration of folate has traditionally been considered suggestive of bacterial overgrowth (see below), based on the assumption that a more “large intestinal” flora has migrated up into the small intestine. However, as mentioned above, some *Lactobacillus* organisms are net folate synthesizers as well as being “desirable” flora. With increasing use of partially fermentable fiber sources such as fructose-oligosaccharides in pet diets, there has been a population wide increase in serum folate concentrations.

Relatively recent studies of dogs with chronic enteropathy and suspected small intestinal bacterial overgrowth have found no difference is serum folate concentrations between dogs that responded to antibiotic therapy and those that did not. In the author’s experience at least, elevated folate concentrations are common in many animals with minimal to no evidence of typical “bacterial overgrowth”, and this finding is of little impact to the management of clinical cases. The obverse of this observation, though, is that a low serum folate is highly meaningful, and a strong indicator of significant small intestinal disease of some form.

**Serum cobalamin**

Cobalamin is also a water-soluble, B-group vitamin (Vitamin B₁₂). In common with folate, this vitamin is abundant in small animal diets and it is extremely difficult to induce cobalamin deficiency in companion animals via dietary means. Also in common with folate, the serum concentration of cobalamin reflects the small intestinal absorptive capacity for this vitamin. Cobalamin undergoes a complex receptor-mediated absorptive process that occurs exclusively in the ileum in all species studied to date, including both dogs and cats. As the absorption of cobalamin occurs exclusively in the ileum, a low serum concentration of this vitamin strongly suggests ileal mucosal dysfunction.

Absorption of cobalamin relies on the formation of complexes between cobalamin and a binding protein called intrinsic factor, this protein is synthesized in the pancreas and gastric mucosa in dogs, and exclusively in the pancreas in the cat. Thus exocrine pancreatic insufficiency is almost invariably associated with low cobalamin concentrations in cats. As the clinical signs of exocrine insufficiency in many cats are vague and often dominated by weight loss and poor appetite, it is important to measure serum fTLI in cats with low cobalamin to help rule in/rule out this disease. While exocrine insufficiency is certainly a potential cause of low cobalamin in cats, it is not the primary cause. Infiltrative disease of the ileum, either inflammatory enteropathies or lymphoma, remain the most common cause of low serum cobalamin in cats and dogs.

Some enteric bacteria, particularly some species of *Clostridium*, are able to degrade the cobalamin/intrinsic factor complexes and then utilize the cobalamin for their own needs, thus patients with the conditions referred to as “bacterial overgrowth” may present with low serum cobalamin due to bacterial competition. Decreased serum cobalamin concentration was identified in 16/29 dogs with chronic enteropathies, however there was no differences noted in dogs with differing definitive diagnoses.

Cobalamin malabsorption can lead to a state of body-wide cobalamin deficiency, with deleterious effects on many cell types in the body, including enterocytes. Recognition of low serum cobalamin and parenteral supplementation to address this is an important part of the management of dogs with chronic enteropathies. Interestingly, low serum cobalamin concentration has been identified as a negative prognostic factor for dogs with chronic enteropathies, and cats with gastrointestinal lymphoma.

The combination of low serum cobalamin and folate concentrations is a very specific indicator of diffuse small intestinal mucosal pathology of some form. Any infiltrative disease, including the various forms of inflammatory bowel disease and intestinal lymphoma, may lead to this combination of abnormalities. Documentation of this combination of abnormalities in a dog with clinical signs of a chronic enteropathy warrants further, more invasive diagnostic testing, such as endoscopy with mucosal biopsy or exploratory laparotomy with biopsy.

Folate and cobalamin are intrinsically linked biochemically, with most enzyme systems that rely on cobalamin as a co-factor also utilizing folate as a methyl group donor. This means that animals that are cobalamin deficient are often not utilizing folate particularly efficiently, which can result in accumulation of folate in the circulation. When the low cobalamin is detected and supplementation begins, it is common for serum folate concentrations to drop quite markedly, in some cases folate drops low enough to suggest the presence of duodenal mucosal disease.
The clinical significance of elevated cobalamin concentrations remains unclear. At least one publication in the veterinary literature has associated high cobalamin concentrations with some hepatic and neoplastic diseases in cats, similar data are lacking for dogs.

Common patterns of results and interpretation for cobalamin and folate

The table below summarizes the common patterns of results that may be detected when measuring serum cobalamin and folate concentrations in dogs and cats with gastrointestinal disease. It is important to remember that these tests have high specificities due to the very localized absorption sites, but they have relatively low sensitivities and thus these tests cannot be used to rule out the presence of small intestinal mucosal disease.

<table>
<thead>
<tr>
<th>Cobalamin</th>
<th>Folate</th>
<th>Potential DDx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>Low</td>
<td>Diffuse SI mucosal diseases: Infiltrative (IBD, LSA) Structural (lymphangectasia, Short Bowel Syndrome)</td>
</tr>
<tr>
<td>Low</td>
<td>High</td>
<td>Disturbed intestinal flora: “SIBO”. Diffuse SI mucosal Exocrine Pancreatic Insufficiency (particularly in cats), check [TLI]</td>
</tr>
<tr>
<td>Low</td>
<td>Normal</td>
<td>Distal SI disease (infiltrative or structural) MOST LIKELY Abnormal bacterial population/dysbiosis Exocrine Pancreatic Insufficiency (particularly in cats), check [TLI]</td>
</tr>
<tr>
<td>High</td>
<td>Normal</td>
<td>Possible association with hepatic and neoplastic disease in cats, consider iatrogenic sources, coprophagia</td>
</tr>
<tr>
<td>Normal</td>
<td>High</td>
<td>Intestinal dysbiosis if compatible signs Possibly no significance, consider iatrogenic sources, coprophagia</td>
</tr>
</tbody>
</table>

Indications for supplementation

As well as acting as a marker for intestinal mucosal disease, there is an increasing body of evidence that cobalamin deficiency, which can manifest with serum cobalamin concentrations in low end of the normal range for both dogs and cats, is associated with poorer response to therapy and poorer prognosis in a variety of diseases. While a full discussion of cobalamin supplementation dosing and schedules is outside the scope of these notes, a substantial amount of information is available from the GI Lab at Texas A&M website, at: http://vetmed.tamu.edu/gilab/research/cobalamin-information

Low serum folate concentrations will also often prompt supplementation, and anecdotally there does appear to be a link between low serum folate and poorer response to therapy, but objective data regarding thresholds for supplementation and doses required are lacking at this time. The author typically recommends folic acid supplementation, 5-10 µg/kg per os daily for dogs and cats with serum folate concentrations <4.5µg/L. Additionally, animals with low normal serum folate and subnormal cobalamin concentrations receive folate supplementation preemptively, due to the common occurrence of low folate following cobalamin supplementation.

References

Focus on Anemia
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Gulfcoast Veterinary Oncology and Internal Medicine
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Mary Anna Thrall, DVM, MS, DACVP
Ross University School of Veterinary Medicine
St. Kitts, West Indies

Anemia is defined as the absolute decrease in the PCV, Hgb or RBC count. Anemia is a condition and not a diagnosis. The initial evaluation of anemia should include measurement of the PCV or HCT, quantification of the total protein, a CBC with examination of a blood film and reticulocyte determination. The differential diagnosis of anemia is best classified based upon the severity of the anemia, bone marrow response (reticulocyte count) and size (MCV) and hemoglobin content (MCHC) of erythrocytes.

Packed cell volume/total protein

Packed Cell Volume (PCV) is directly measured via centrifuging blood in a microhematocrit tube for 3 minutes. It is the quickest measure of the red blood cell component of the blood and is affected by plasma trapping and how the red cells pack within the column. Examination of the HCT tube also yields valuable information about the color and clarity of the plasma (icterus, lipemia and hemolysis) as well as the size of the buffy coat (contains WBC and platelets). Total Protein should always be evaluated simultaneously with the PCV. The total protein concentration may help distinguish between blood loss (low PCV and TP) and hemolysis (low PCV with normal TP).

Complete blood count

Hematocrit (HCT) is a calculated value obtained from the CBC analyzer. It is the product of the mean cell volume (MCV) and the red blood cell count (RBC). Anything that falsely increases or decreases the RBC or MCV will affect the HCT. Hemolysis will cause a decrease in the RBCs and thus falsely lower the HCT. Storage of blood will cause the RBCs to swell, increasing the MCV and falsely increasing the HCT.

MCV: Mean cell volume
- Cats 40-50 fL  Dogs 62-71 fL
- MCV above reference interval: Macrocytic
- MCV within the reference range: Normocytic
- MCV below the reference range: Microcytic

MCHC: Mean cell hemoglobin concentration
- Cats 32-36 g/dl  Dogs 33.7-36.5 g/dl
- MCHC above the reference interval: Hyperchromic
- MCHC within the reference interval: Normochromic
- MCHC below the reference interval: Hypochromic

RDW Index
The red cell distribution width (RDW) is the variation in RBC volume and is the electronic equivalent of anisocytosis on a blood film; high RDW indicates more variation than normal.

The blood film
- Polychromatophils: Immature RBCs that stain blue-purple on blood smears (evidence of regeneration)
- Spherocytes: Smaller and darker than normal RBCs. Moderate to marked numbers are highly suggestive for IHA, but can also be seen with Heinz body anemia, zinc toxicity, vasculitis, post transfusion, neoplasia and ADIC
- Autoagglutination: Grape like aggregation of RBCs (immune component to the anemia)
- Heinz bodies: Clumps of denatured hemoglobin that appear as small circular projections off the RBC membrane; associated with oxidative damage (acetaminophen, garlic and onion, propofol, zinc). In cats they can also be seen with hyperthyroidism, lymphoma and diabetes
- Howell Jolly bodies: Basophilic nuclear remnants; associated with regenerative anemia or hyposplenism
- Basophilic stippling: Basophilic dots within cytoplasm; associated with regenerative anemia or lead poisoning
- Schistocytes: Fragmented RBCs; indicative of DIC, hemangiosarcoma, vasculitis, heartworm and liver disease
- Erythroparasites: Mycoplasma spp., Cytotauxzoon felis, Babesia gibsoni and canis
- Nucleated RBCs: Metarubricytes, rubricytes and earlier stages of RBC development; appropriate/expected response to anemia. Inappropriately released with any bone marrow injury, lead toxicity; can be normal in Miniature Schnauzers
- Other Cytopenias or Abnormal Cells
Reticulocyte count

Reticulocytes are immature red blood cells that are released from the bone marrow before they fully extrude their RNA and cellular organelles. New methylene blue is a supravital stain that precipitates the RNA and stains it blue. There are two types of reticulocytes including aggregate (clumps of reticulum, larger and less mature) and punctate reticulocytes (single dots of reticulum). In cats, aggregate reticulocytes are considered evidence of current regeneration while punctate reticulocytes have a long half-life and their presence may indicate an insult that occurred days to weeks prior to testing. Both types are counted together in dogs. Reticulocytes are not seen for 2-4 days after an acute episode of blood loss or hemolysis and their response peaks between 4-7 days.

Procedure for in-house reticulocyte count

1. Mix equal volumes (2-3 drops) of blood and NMB stain in a test tube and incubate the mixture for at least 10 minutes, then gently remix sample and remove a drop to make a blood film; air dry
2. Count number of reticulocytes per 1,000 RBCs
3. Report as a percentage by dividing the number of reticulocytes by 10
4. To get an absolute number, multiply the above percentage by analyzer generated RBC count

Uncorrected reticulocyte percentage = Number of reticulocytes/10

Corrected reticulocyte percentage = Reticulocyte % X Patients HCT /normal HCT (45 for dogs or 35 for cats)

Absolute reticulocyte% X RBC count (in million/uL)

<table>
<thead>
<tr>
<th>Degree of Regeneration</th>
<th>Dogs</th>
<th>% Reticulocytes</th>
<th>Cats</th>
<th>Dogs</th>
<th>% Reticulocytes</th>
<th>Cats</th>
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<tbody>
<tr>
<td>None</td>
<td></td>
<td>&lt;60,000</td>
<td></td>
<td>&lt;15,000</td>
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<tr>
<td>Slight</td>
<td>1-4</td>
<td>0.5-2</td>
<td>60,000-15,000</td>
<td>150,000</td>
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<tr>
<td>Moderate</td>
<td>5-20</td>
<td>150,000-300,000</td>
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<tr>
<td>Marked</td>
<td>&gt;20</td>
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<td>&gt;500,000</td>
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<table>
<thead>
<tr>
<th>Grade of anemia</th>
<th>Dog</th>
<th>Cat</th>
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<tr>
<td>Reference normal</td>
<td>41-58</td>
<td>31-48</td>
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<tr>
<td>Mild</td>
<td>30-40</td>
<td>25-35</td>
</tr>
<tr>
<td>Moderate</td>
<td>20-30</td>
<td>15-25</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;20</td>
<td>&lt;15</td>
</tr>
</tbody>
</table>

3 major mechanisms of anemia

- Hemorrhage
- Usually regenerative
- Hemolysis
- Usually regenerative
- Decreased Production
- Usually non-regenerative

Interpretation based on erythrocyte indices

Macrocytic, hypochromic, mild to severeregenerative anemia
Hemorrhage or hemolysis

Macrocytic, normochromic, mild to severe non-regenerative anemia
Vitamin B12, Folate or Cobalamin deficiency, FeLV, myelodysplasia, drugs, non-regenerative IHA, hereditary stomatocytosis

Normocytic, normochromic, mild to moderate non-regenerative anemia
Anemia of inflammatory disease, chronic renal disease, endocrine disease, infectious agents, drugs, lymphoma, histiocytic sarcoma, severe nutritional deficiencies

Microcytic, normochromic to hypochromic, mild to severe non-regenerative anemia
Iron or copper deficiency, Portosystemic shunts
Microcytic, hypochromic, mild to severe regenerative anemia
Iron deficiency

Regenerative vs. pre-regenerative vs. non-regenerative anemia

Regenerative anemia
The hallmark of regenerative anemia is the presence of a reticulocytosis, which indicates an appropriate compensatory response to anemia. It is characterized by erythroid hyperplasia in the bone marrow and increased release of red cells into the circulation before they are fully mature (polychromatophilic red cells). Causes of regenerative anemias include hemorrhage and hemolysis.

Acute hemorrhage
Common causes of acute hemorrhage include trauma, coagulopathies, platelet disorders, splenic rupture and gastrointestinal via neoplasia and ulcerative disease. The clinical signs of blood loss are dependent upon the amount of blood lost, time and site. Clinical signs of blood loss may be apparent before the onset of a decreased PCV due to the simultaneous loss of both plasma and RBCs as well as splenic contraction. Splenic contraction delivers a high PCV blood (80%) to the circulation. Polychromasia, anisocytosis, Howell Jolly bodies, increased nRBCs and reticulocytes become evident by 48-72 hours and reach a maximum about 5-7 days after hemorrhage. The hemogram returns to normal 1-2 weeks following a single acute hemorrhagic episode.

Hemolysis
Hemolytic anemia is often associated with a stronger regenerative response than hemorrhagic anemia. The total protein is usually normal. Hemolysis can be intravascular, extravascular or a combination of both. Both types can result in icterus. Intravascular hemolysis is the result of RBC destruction/rupture within the circulation and thus can result in hemoglobinemia, hemoglobinuria and bilirubinuria. Extravascular hemolysis occurs when RBCs are phagocytized by macrophages in the spleen, liver and bone marrow; hemoglobinemia and hemoglobinuria are not present. Causes of hemolysis include:

- Immune Mediated
  - Primary: Idiopathic IMHA, transfusion reaction
  - Secondary: Infectious, neoplasia, drugs, vaccine adverse reaction
- Infectious
  - Hemotropic mycoplasmosis, ehrlichiosis, babesiosis, cytauxzoonosis, FeLV, septicemia
- Fragmentation/physical damage to RBC membrane
  - DIC, cavals syndrome, glomerulonephritis, hemolytic uremic syndrome, hemangiosarcoma, vasculitis, heatstroke, splenic torsion, hepatic disease, severe burns
- Drugs, toxins, chemical damage
  - Zinc (pennies minted after 1983), snake envenomation, hypophosphatemia, bee stings, spider bites, oxidants
  - (onions, garlic, mothballs (naphthalene,) acetaminophen)
- Congenital red cell abnormalities
  - Phosphofructokinase deficiency (English Springer Spaniels, Cocker Spaniels), Pyruvate Kinase deficiency
  - (Abyssinian, Somali and DSH, Beagles and Basenjis), hereditary stomatocytosis

Pre-regenerative anemia
An anemia with a regenerative response that is impending, but not yet apparent on the CBC, is termed pre-regenerative.

It takes 3-4 days after an acute blood loss or hemolytic event until reticulocytes are evident on the CBC and several more days until the regenerative response peaks.

Non-regenerative anemia
The hallmark of non-regenerative anemia is the absence of reticulocytosis, which indicates erythropoiesis is being inhibited in some way. Most non-regenerative anemias are normocytic and normochromic other than secondary to FeLV (macrocytic) and iron deficiency (microcytic). Causes of non-regenerative anemia include anemia of inflammation, iron deficiency anemia, chronic renal failure, neoplasia, endocrinopathies, non-regenerative IMHA, liver disease, FeLV, toxic insults (chemotherapy, estrogen, etc.), space occupying disease in the bone marrow and nutritional deficiencies.
Ultrasound imaging has become widely accepted as an important diagnostic tool for imaging the gastrointestinal tract in animals. Anticipated artifacts created by gas and fecal material were initially thought to significantly limit or exclude examination of the gastrointestinal tract by ultrasound. In actuality, the artifacts, though present and at times obstructive, rarely significantly interfere with the entire examination. There has been marked improvement in technology with higher resolution (12-18 MHz and higher) transducers and more affordable equipment currently available. This combined with endosonography and Doppler ultrasound has resulted in more thorough imaging of the intestinal tract with improved critical evaluation and understanding of changes from diseases affecting the gastrointestinal tract.

Ultrasound uses sound to reflect from the tissue boundaries within the body to form an image that we can recognize and interpret. It is noninvasive and has no known significant biological risk at diagnostic frequencies (1-25 MHz). It is particularly useful in evaluating the intestinal wall, the luminal contents, function evidenced by peristalsis, and in interrogating the surrounding organs such as the peritoneum, pancreas, liver and lymph nodes. Ultrasound is also useful in guiding needle placement for either an aspirate or biopsy of a detected abnormality. Intestinal ultrasound provides additional and complementary information to other diagnostic modalities (survey radiographs, computerized tomography, nuclear medicine, magnetic resonance imaging, contrast radiography). With recent improvement in technology and knowledge, there has been advancement in the diagnosis of diseases affecting the intestinal tract of cats. But with the improvement in technology and knowledge, there are new questions that arise as to the meaning of the findings and there remains a great deal to be learned regarding the intestinal tract of the cat. It is especially important to correlate the ultrasound findings with the history, clinical findings, laboratory results and ultimately with cytopathology and histopathology. Ultrasound imaging has now become such an integral part of the diagnostic work-up of small animals with gastrointestinal signs that there has been marked reduction in radiographic contrast procedures such as upper GI studies. It is faster, more cost effective and often provides as much or more information than the other diagnostic modalities.

**Method of evaluation**

To achieve the best image quality, hair should be removed from skin of the imaging area in most cats via a surgical clipping blade (40). The area clipped usually encompasses a rectangular area extending from the epaxial muscles in the dorsocranial abdomen (usually 8-9th ribs) to the caudal abdomen just in front of the pelvic limbs and ventrally to the mid line on both sides of the animal. A concerted attempt is made to keep the margins of the clipped area neat and straight as this appearance is often noticed and important to the owner. Mammary papilla may be obscured by the surrounding hair and may be at risk for trauma from the clipper blades from those less experienced in clipping. The area clipped will be more extensive when imaging from a lateral position compared to the dorsal recumbent position. Commercially available ultrasound water soluble gel is used to act as an interface between the skin and the transducer. If the animal has thin hair, wetting the hair with water and alcohol may be done in lieu of cutting the hair. Alcohol may also be used to clean the skin of animals with dry, dirty or scaly skin and this application of alcohol often results in reduction in the amount of gel used and an improved image quality.

**Normal anatomy**

Examination of the entire intestinal tract is typically a part of each sonographic study of the abdomen. The examination includes interrogation of the specific sections of the intestine (esophagus, stomach, duodenum, jejunum, ileum and colon) noting the wall thickness, distinction of the wall layers, the luminal contents in each section, motility and vascular integrity.

The wall layers of the intestine include 5 layers with an alternating hyperechoic and hyperechoic appearance. The 3 hyperechoic layers (the “S” layers are serosal, submucosa and surface of the mucosa) and 2 hypoechoic layers (the “M” layers are the muscularis and mucosal layers). The wall thickness is in part related to degree of distinction of the loop of intestine. It is mildly thinner the more distended it becomes. A contraction during normal peristaltic activity does mildly affect the width of the intestinal wall and the width of specific layers may momentarily change with contractions. The intestinal tract is dynamic and contractions cause a shortening of the length of the loop of intestine with an increased width of the muscular and mucosal layers in the contracted area compared to adjacent dilated segment. It is important to be careful and consistent in placing the cursors for wall measurement. The stomach has characteristic rugal folds that radiate towards the center and is often empty. The stomach wall varies from 1.7 to 2.8 mm in width. It is important to make sure that a rugal fold or an oblique cut is not being measured and giving an erroneous thickened measurement especially on an empty stomach. It is easiest and most accurate to measure the wall when a small amount of fluid is present within the stomach lumen. This may require orally administering water if there is a question about the wall thickness. The duodenum and jejunum are usually 2.3 to 2.8 mm in width. The ileum is more difficult to accurately measure the wall due to the contracted “wagon

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**Gastrointestinal Ultrasonography: What a Gas!**

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wheel appearance that is usually seen. The ileum has prominent hyperechoic submucosa and hypoechoic muscularis layers. The wall may measure from 2.4-2.8 mm in width. The colon wall is thinner (usually 1.4 to 2.3 mm) and the walls are less distinct. The far wall is often obscured by shadowing artifact due to absorption of sound by feces. In general, the intestinal wall in a cat is fairly consistent at 2.3-2.8 mm. It is regarded as abnormal when the wall is greater than 3 mm or there is a change in the appearance of the wall layers. The colon is thinner at approximately 2.1 mm.

The luminal appearance varies depending on the ingested contents. Gas, fluid, mucous, food and feces are normal contents observed. Ring down, comet tail and sometimes attenuation and shadowing artifacts are frequently observed due to the sound interacting with the luminal contents. The luminal contents often contain mucous mixed with tiny gas bubbles (chyme), fluid and/or ingesta. Fluid (with minimal gas bubbles) may be given per os to accentuate imaging the wall and lumen of the proximal GI tract. Normal motility is observed with real time ultrasound. Four to five contractions per minute is normal in the stomach and duodenum and one to three in the remainder of the small intestine. It is not common to see contractions in the colon. Identification of specific segments of the GI tract may be determined by using topographic landmarks and characteristic appearances of different segments. The stomach (fundus to pylorus), the duodenum, jejunum, ileum and colon can all be separately identified. The stomach has a characteristic pattern with its size, shape, and prominent rugal folds. The empty stomach of a cat has a rosette appearance. It is located in the left cranial abdomen just caudal to the liver, cranial medial to the head of the spleen and cranial to the left kidney. The pylorus extends to the midline or slightly to the right of midline. The duodenum courses in a straight superficial path from the pylorus along the right body wall for approximately 2/3 the length of the abdomen. It then curves medially at the caudal flexure and then courses craniomedially. The sphincter of Odi is the entrance of the conjoined bile duct and pancreatic duct located in the proximal flexure of the duodenum just distal to the pylorus. The jejunum is identified as the longer intestine between the duodenum and the shorter ileum. The ileum is a small section of intestine that is recognized because of the characteristic appearance and the entrance into the colon. The ileum has a prominent hyperechoic submucosal layer and corresponding undulating mucosal layer. It is often contracted and has a reported “wagon wheel” appearance. The cecum and/or the ileum may be seen connecting to the larger colon. The colon is recognized by its larger size, thinner wall, its location extending dorsal to the bladder through the pelvic inlet and the attenuation and shadowing caused by luminal contents (feces) or reverberations by a gas distended lumen. The ascending, transverse and descending colon may be followed. It is easiest to trace the short ascending segment from the right side and the descending from the left side of the abdomen. If there is a question regarding if a loop of intestine is an abnormal segment of small intestine or colon, it is most helpful to follow the loop to see if it courses through the pelvic canal or to trace what is known colon to the area in question. Small intestine has a thicker wall and does not have the dilated lumen with shadowing contents seen in the colon.

Gastrointestinal disorders
Disorders involving the gastrointestinal tract may be divided into those involving the lumen, the wall or those arising outside the intestinal wall. A final diagnosis or differential diagnosis involves correlating the abnormal sonographic findings to the history and laboratory results and may require an aspirate or histopathology or cytology.

Luminal changes
The luminal abnormalities typically are due to abnormal contents such as a foreign body or an intussusception or abnormal distention of a segment of intestine that may indicate an obstruction. This dilation may be combined with specific mural changes that may be due to a linear foreign body, or an infiltrative disease such as neoplasia that extends into the lumen and affects passage of contents.

Obstruction
The stomach and small intestine are often empty of contents and it is atypical for the normal feline intestinal tract to contain much fluid or gas. If distension of the lumen is seen, then questioning when a meal was ingested or if gastrointestinal signs are present will often help to determine the significance of the finding. If there remains a question of the significance, and the patient is stable, fasting the patient for 12 hours and repeating the study to ascertain if the material is still present may be warranted. If the stomach is distended, this may be due to a recent meal, a motility problem or possibly due to an outflow obstruction. Careful evaluation of the antrum and pylorus and proximal duodenum for the cause is warranted. When luminal obstruction occurs, the intestinal tract will often be distended and filled with motile hypoechoic fluid and possibly gas oral to the obstruction. The cause of the obstruction may be intraluminal, mural or less often extraluminal. Just because the distended intestinal tract contains gas evidenced on the radiographs does not preclude the utility of abdominal ultrasound in identifying the abnormality. If there is a high obstruction within the duodenum, vomiting may but not always result in a decrease in the distension of the lumen. Segments of small intestine distal to the obstruction are usually of a normal size. It is useful to locate the dilated segments and follow them to the point of the obstruction. Try to determine the location of the bowel segment obstructed and evaluate the lumen for an intraluminal foreign body, mass with wall thickening or an extra luminal component. If it is the ileum, the entire small intestine may be affected and dilated. If it is the jejunum or duodenum, the segments distal should be normal in size. Locating the ileum is useful to make sure that it is due to an obstructive process and not due to a metabolic cause. With pancreatic insufficiency, malabsorption syndrome, or some inflammatory diseases, the intestine may be markedly distended throughout without normal-sized small intestine detected. If the entire small intestine is dilated
including the ileum and there is no mass or intussusception at the ileocolic junction, then the luminal dilation is unlikely to be due to a mechanical obstruction.

**Foreign bodies** are a frequent cause of luminal obstruction. The foreign body will vary in appearance depending on their physical make up. The shape and appearance of the foreign body may allow identification of the actual foreign body. Fluid in the lumen surrounding and outlining a margin the foreign body may facilitate its identification due to a characteristic recognizable geometric shape

Some foreign bodies are very characteristic in appearance and make identification simple. Other foreign bodies such as cloth are highly attenuating and may have a hyperechoic surface. Not all foreign bodies are inciting a clinical problem in the animal. Grass, rocks, and other materials may have been ingested especially if the animal has an intestinal problem. They may not result in clinical signs but be identified on the ultrasound exam. Parasites such as ascarids may be identified as mobile, tubular, undulating, often tangled foreign bodies within the GI tract. Determining if foreign objects are incidental will be important in the management of the patient. This may be accomplished by examining the relevant historical signs of intestinal disease, determining if the foreign object is causing an obstruction or resulting in a plication of the intestine. Foreign bodies such as a hair ball (gastric or enteric trichobezoars) often have an irregular poorly defined margin, are heterogeneous, have a hyperechoic surface and have significant attenuation and shadowing. This may be clinically relevant if the history supports accurate historic fasting for at least 12 hours. Correlating the ultrasound findings with abdominal radiographs may also assist in identifying the significance of specific foreign bodies such as an eroding penny resulting in zinc toxicity. Determining the kind of foreign body present is often a delightful mental challenge that is quickly rewarded by the removal and identification of the object.

**Linear foreign body**

A foreign body that is linear in shape may be found in the lumen of the gastrointestinal tract and may be due to ingested material such as hair or grass. This material has a linear appearance but is not embedded in the wall, the ends may be located (not anchored in the stomach), and the foreign object does not have a taunt appearance and is not on the mesenteric side of the small intestine with a placation or gathered appearance to the adjacent segment of intestine. The appearance of a true linear foreign body will depend on the type of material present. Linear foreign bodies have a place of secured attachment such as around the tongue or trapped within the stomach. The remainder progresses aborally downstream and becomes embedded in the wall of the intestine. With peristalsis and segmental contractions, gathering of the intestine on the string as it embeds in the wall occurs. Typically, the foreign body is eccentrically located on the mesenteric side of the bowel and is taunt as it is anchored with the ends not free in the lumen. It is often partially imbedded in the wall. The bowel may be plicated and thickened as the string erodes into the wall. Peritoneal fluid may be present. Free air in the peritoneal cavity often indicates perforation. Panty hose, cloth-like material, plastic or rubber products will attenuate the beam and often have shadowing similar to feces in the colon. This can be more challenging to identify compared to string (like dental floss) as it may be incorrectly assumed to represent feces in the colon. Making sure that the segment of intestine is the small intestine and not the colon is needed. The colon should have a thinner wall, have ileum entering it, and exit through the pelvic canal. If necessary, the colon can be identified as it courses through the pelvic canal. It is then followed cranially to determine if it communicates with the questionable segment of intestine. When a linear foreign body is identified, the wall should be closely examined for thickening, ulceration or perforation. Intussusceptions may occur in conjunction with a linear foreign object due to the abnormal motility and constriction afforded by the plicated segment of intestine.

**Intussusception**

An intussusception occurs when one segment invaginates into an adjacent segment. The intussusception is made of the double walls of the outer intussuscipiens and the inner loop of invaginated intestine, the intussusceptum. The most frequent type is an ileocolic intussusception. Other types include enterenteric or less commonly, cecocolic, gastroesophageal or duodeno gastric. The classic sonographic image is a transverse plane through the intussusception. The double concentric hyperechoic rings that form the thicker outer wall surrounding a central hyperechoic core with a cross section of an eccentrically located loop of small intestine within the lumen is classic for the diagnosis of an intussusception. The intussuscipiens is often thickened, and hyperechoic. The intussusceptum located within the cross sectional view is the transverse segment of intestine eccentrically located within the lumen of the intussuscipiens. This usually appears as a normal empty loop of intestine. The eccentrically positioned hyperechoic material is the mesentery attached to the mesenteric border of the intussuscepted segment of intestine and it acts as a mass resulting in displacement of that segment of intestine to the opposite wall. The longitudinal plane shows multiple layers of the two bowel segments telescoped within each other. The thicker layered outer wall is the combined wall of both the intussusceptum and intussuscipiens. The leading edge of the intussusceptum may be identified at the distal end of the intussusception. In cats, identification of enlarged lymph nodes or thickened GI wall may be seen and indicate the initiating cause of the intussusception such as an adjacent tumor of the intestine or enlarged lymph nodes. The mesentery should be uniform except for small vessels. Enlarged lymph nodes or masses may be seen within the mesentery intussuscepted and may give an indication of the cause. There may be fluid present that may be due to hemorrhage or a chronic intussusception and lymphatic obstruction and cystic formations or vascular compromise. The
intussusceptum should be empty of internal contents. A hyperechoic internal structure may indicate a linear foreign body was the inciting cause. This may be followed beyond the intussusception where plicated intestine may be found.

**Mural changes**

This is an area that is complicated and challenging with many diseases and overlapping appearances. A lot of work to learn more is currently underway in this area and a great deal still needs to be done to increase our knowledge base. We know the normal size of the wall of the intestine and the 5 distinct wall layers. We know that an infiltrative disease of what ever kind may affect the wall and cause thickening of one or all of the layers and that there may be disruption of the layers with an indistinct margin between the layers. We use this information combined with the history and abnormalities affecting other organs to attempt to determine the underlying disease process. Some diseases are unique and specific and easier to diagnose. The ultrasound appearance does not provide a histologic diagnosis but it does give hints. It does provide morphologic information. It may indicate the location and extent of involvement. An aspiration or biopsy is often necessary for confirmation of the final etiology. Wall thickening is the most consistent sonographic finding of mural diseases. Thickening of the wall, presence or absence of retention of the wall layers and echogenicity are useful aids in evaluating the GI wall. A clear distinction between diffuse change vs focal change is frequently helpful in ranking the differential diagnosis. Generalized thickening is often seen with inflammatory disorders and localized and asymmetrical thickening with disruption of wall layers is an indication of a potentially serious infiltrative process. Unfortunately, the severity of thickening is not always a parameter that can distinguish inflammatory disease from neoplastic disease.

Sonographic appearance of inflammation varies with the cause, duration, extent of involvement and secondary involvement of other organs. It can be accompanied by normal or increased wall thickness.

A general rule to apply is that generalized, symmetric wall thickening without loss of the wall layers is often associated with inflammation possibly from IBD, mycobacterium, FIP or Histoplasmosis. A challenge is sometimes differentiating between lymphosarcoma (small cell) and IBD or other causes for an inflammatory process. Asymmetric wall thickening with loss of the layers is typical of a more aggressive process such as neoplasia. A change in the echogenicity of the wall layers may occur with both but is seen more with neoplasia. Spasticity may accompany an irritated segment of intestine or one with vascular compromise. Most neoplasms create large mass effects, are hypoechoic and there is disruption of the wall layers. Disruption of the mucosal surface often denotes an aggressive process. The focal infiltrative disease may distort the wall and protrude into and compromise the lumen and may also protrude beyond the serosal surface. The appearance will vary depending on the stage of advancement of disease and tumor type. Rigidity may be evaluated by the peristaltic waves that progress through a specific segment. Concomitant obstruction may be present. Abscesses, granulomas or tumors may result in this appearance. Wall thickness either symmetric or asymmetric and localized or generalized may help but often a biopsy is needed to ascertain cell type. Success of aspiration of the GI wall may be unrewarding. It is more successful in exfoliating lymphosarcoma and malignant diseases especially masses >2 cm. Aspiration of a regional enlarged lymph node may be more helpful. The more destructive the process and the more other organs are involved, the more likely that neoplasia is the root problem. Ulcers identified as gas filled craters surrounding thickened wall are often associated with tumors but may be seen secondary to non-inflammatory drugs.
Musculoskeletal Ultrasound:
A Sturdy Foundation
Kathy Spaulding, DVM, DACVR
Texas A&M University
College Station, TX

Musculoskeletal imaging with ultrasound has been used extensively in the equine. It is often used in the diagnosis of human musculoskeletal diseases. It has started to become very useful in the dogs and cats.

This session will focus on specific joints and diseases affecting the musculoskeletal system. The shoulder including the appearance of OCD affecting the humeral head and the normal and abnormal appearance of the tendons and ligaments around the shoulder joint including the bicipital groove will be discussed. The calcaneal tendon and also the stifle joint including specific changes from abnormalities including trauma affecting each area will be discussed. In addition, the iliopsoas muscle, other muscles and body wall masses will be presented with instruction on how to find and identify the structures and how disease will secondarily affect this normal appearance. Examples of the appearance of neoplasia affecting bones, joints and the soft tissue will be presented. The unique appearance of different kinds will be discussed. Examples of normal nerves and the appearance of nerve sheath tumors will be shown.

Tips for aspirating cells from some of these challenging bony lesions will be given.

This diagnostic tool is a useful technique to add to your radiographic imaging.
Radiographic Imaging of the Thorax
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A radiographic interpretation may state “compatible with the age of the patient”. There are changes within the thorax that are commonly identified in our older canine population. Some of these represent changes within the structure of the lung, or skeleton. They may not be related to any clinical evidence for significance. They mimic significant disease or mask disease. This paper will look first at some of the changes commonly seen that are considered not significant. The last part of the paper will include diseases more commonly identified in the older population. The aged patient usually would include patients 6-7 years or greater.

The lung is often an area that changes with age. It is an especially challenging area to interpret. As a study in 2000 elderly human patients described the “senile lung” as a spongy or lacy pattern of the lung fields associated with increased peripheral lung markings and an increased contrast of pulmonary markings”. Changes within the lungs may involve the distal airways, the interstitial space, the pleura, and the cardiovascular structures. When an alveolar pattern is present, this is more commonly associated with a specific disease and less often associated with simply the aging of the lung.

Bronchial pattern
Thickening and mineralization of the trachea and distal airways is often seen to increase the visibility of the walls of these structures. This is presumed to be due to calcification within the walls. There are more tramlines and “donuts” seen within the lung especially near the hilus. The tracheal rings are mineralized and can be traced into the main stem bronchi and into the parenchyma. This appearance may be also associated with diseases such as Cushing’s disease. The patients usually are not coughing. Dogs especially the small breed dogs such as the Pomeranian or Poodle may have collapsing of the trachea and main stem bronchi. There is often coughing with this disease process. Fluoroscopic evaluation is often warranted. With chronic bronchial disease there may be bronchiectasis with focal widening of the bronchial lumen. There may be distal alveolar disease due to the ineffective mucociliary apparatus. Dogs chronically exposed to noxious material including smoke may have thickened bronchial walls and an increased unstructured interstitial pattern.

Vascular pattern
Mineralization of the walls of vessels (atherosclerosis) may be secondary to hypothyroidism, vascular injury (possibly from heart worms or other PTE causes including Cushing’s disease). There may be mineralization of the aortic valves. The primary cause for a change in the appearance of the pulmonary vessels is due to cardiac disease often observed in the older population. This includes heart worms (enlarged pulmonary arteries), chronic valvular disease (enlarged pulmonary veins), and cardiomyopathy (enlarged pulmonary veins and sometimes the arteries). End-on views of the vessels may mimic pulmonary metastatic disease. A decreased size to the vessels often is due to hypovolemia (blood loss, dehydration, Addison’s disease). It may be seen in the aged population from other diseases but is not generally a true aging finding.

Interstitial pattern
This pattern is the category most often affected by aging changes. This pattern can be divided into the unstructured appearance and the structured appearance (nodules, masses). The unstructured pattern is most often seen. Often the older patient has an overabundance of body wall fat. This adds to the overall opacity to the lung. The patient may not take deep inspiratory breaths and thus the lungs are less aerated and this adds to the overall opacity to the lungs. In addition there are fine, nonvascular linear markings that are associated with pleural thickening. This is especially evidenced between the right middle and accessory /caudal lung lobes. These do not branch as would occur with the bronchial or vascular structures. They are often seen in specific locations. Pleural fibrosis may occur and may be seen as pleural scars (plate atelectasis) or more diffuse areas of pleural thickening. There is more fibrous connective tissue present. The alveolar walls and ducts may thicken with fibrous connective tissue. This may also be seen sub pleural. There may be an increased amount of cuboidal and squamous epithelium and hypertrophied alveolar lining cells. Dogs with chronic mitral insufficiency had prominent and thickened alveolar walls.

A structured interstitial pattern may be found in the older patients. This is often described based on size and opacity of the nodules. They range from reticular nodular, military nodular to nodular and then to masses. The opacity may be soft tissue or mineralized. They may be solid or cavitarded. Usually with benign age related changes the structured changes are small nodules. This may occur in approximately 10% of the older dog population examined in one report. If mineralized they usually represent metaplasia or heterotopic bone. They are usually not clinically important but if extensive then it could be clinically relevant. Alveolar microlithiasis of the lungs from Cushing’s disease has been reported. The lungs are noncompliant and do not collapse when removed from the patient. Discrete foci of bone have been described histologically with heterotopic bone. This has been hypothesized to involve transformation of fibroblasts into osteoblasts with the formation of dystrophic mineralization. This is often at the periphery of the lung.
especially in Collies. They are often small measuring 1-3 mm in width. If smaller they may not be discerned as a nodule but merely increase the overall opacity to the lungs. They are separated from vessels because of the tail of the vessel and the size of the structure based on where it is located in the lung. The closer to the hilus the larger it is. It should be the same size as the adjacent vessels in that part of the lung if it is an end on vessel. The changes in the lungs over time may be associated with subclinical disease, exposure to pollutants or noxious material inhaled.

**Specific diseases**
Diseases that occur more frequently in the older patient population is varied.

**Neoplasia** is a frequent disease in the older population is a common reason to image the thorax. Typically, neoplasia affects the interstitial space and is manifested by a structured pattern. Metastatic disease is often soft tissue in opacity and multiple. Some types of metastatic tumor may have cavitated centers. These are usually from tumors that are glandular. Mineralized metastatic lesions occur but are uncommon. Primary lung tumors are often singular and large. They may be mineralized and cavitated. Lymphoma may have more of a reticular nodular pattern as the lymphatics are distended with the neoplastic infiltrate. The lymph nodes (sternal, cranial mediastinal and hilar) are often enlarged. There may be rib tumors (primary or metastatic) overlying the lungs that may have an extra pleural sign, pleural effusion and metastasis.

**Heart disease** is a common abnormality in the canine population. Chronic valvular, heart worms and cardiomyopathy are the most frequently encountered. There are specific changes identified in the size of the heart and the specific chamber affected. Noting if it is right sided vs. left sided is important in determining the underlying disease. It is important to determine if heart failure (right or left) is present.

**Pleural disease** is usually manifested by either air fluid or a mass in the pleural space. The lungs usually respond by decreasing in size and increasing in opacity. This in part is due to less air in the lungs and the overlying soft tissue opacity overlying the lungs. Fluid in the pleural space may be categorized as blood, pus or water. It may be due to hemorrhage from a clotting disorder, trauma, and rupture of a mass. It may be due to purulent material from trauma, foreign body, and septic pleuritis. Water is often due to heart failure or chyle from lymphatic disruption. Fluid or air will cause separation of the lungs from the thoracic wall and a change in opacity. The lungs contain vessels that go to the periphery. When a pneumothorax or pleural effusion causes the lungs to not extend to the periphery, then the vessels also do not go the full extent of the thoracic cavity.  

**Mediastinal disease** is usually manifested by a change in size, shape and opacity of the mediastinal area. The esophagus if dilated will cause the trachea to be displaced ventrally. It is often dilated with fluid and gas. Masses in the cranial mediastinum often cause a widening of the mediastinum. This may displace the trachea and heart and lungs. Structures within the mediastinum may be affected. This included the lymph nodes, the thymus, vessels. Cysts may also cause a mass effect. A heart based mass usually dorsally displaces the distal trachea and silhouettes with the heart. A thymoma often is more ventral and close to the heart. Lymphoma often affects multiple lymphocenters such as the sternal, cranial mediastinal and hilar. A pneumomediastinum may come from a patient with interstitial disease or trauma to the esophagus, neck or trachea. The structures within the mediastinum are better imaged than normal.
Ultrasound Imaging of the Pancreas in the Dog and Cat
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Imaging of the pancreas by radiography is limited by the effect pancreatic disease has on the size of the pancreas, the production of fluid and impact on surrounding structures. The changes seen are often subtle and nonspecific and thus a diagnosis from a radiograph is often challenging. The advent of real-time ultrasound has made visualization of the pancreas a reality even in normal patients. However, even though the pancreas may be seen with ultrasound, the differential can still be challenging especially with cats. The pancreas is typically a challenging organ to image. As imaging equipment has improved, the ability to recognize the pancreas has markedly improved. It is very important to know normal landmarks to find the pancreas. Things that interfere with good examination include; a postprandial exam, excessive gas, and an uncooperative patient with a tense abdomen. Lack of abnormal sonographic findings does not totally eliminate pancreatic disease as a cause of clinical signs. The major challenges are to distinguish the normal pancreas; to distinguish this from abnormal; to differentiate between pancreatitis and malignant neoplasms and to separate pseudocysts from abscesses.

Disorders involving the feline pancreas in the past have been considered uncommon. But recent information suggests that pancreatitis is more common. This is a challenging disease ante mortem. Clinical signs may be mild or nonspecific. Blood work including chemistry, pancreatic enzymes are non-sensitive. Radiography is insensitive. This leaves one with ultrasound imaging. It is currently the imaging modality of choice in the cat and the dog.

Normal anatomic location
The pancreas may be located by identifying its parenchymal architecture and anatomic landmarks that surround the organ. The normal pancreas is hard to see and may not be seen if using a transducer lower than a 7.5 MHz. Ideally the transducer should be a 10-13 MHz or higher. The echogenicity of the pancreas is isoechoic to slightly hyperechoic to the caudate lobe of the liver and slightly hypoechoic to the spleen and hypoechoic to the surrounding mesenteric fat. The mean thickness of the pancreas in the cat was as follows: right pancreatic lobe (4.5 mm range 2.8-5.9), body (6.6 range 4.7-9.5mm) and left side (5.4 range 3.4-9.0 mm). The pancreatic duct had a mean thickness of 0.8 mm (range 0.5-1.3 mm). The gastric lymph node was identified in 6/20 cats. 10 mm-6.0 mm. They found not significant difference based on age, sex weight or body condition. (Etue, Penninck et al)

Different authors have described different methods to find the pancreas. The pancreas consists of the left limb, body and right limb. To locate the left pancreas, place the patient in a right lateral decubital position and image the left dorsocranial quadrant of the abdomen. A triangle of organs is useful in finding the left lobe. Locate the spleen in the near field, the greater curvature of the stomach cranially and cranial pole of the left kidney caudally. The left lobe will extend laterally to just medial to the spleen. The left lobe of the pancreas is located deep or medial to the spleen and caudal to the splenic vein. You may need to fan the region to see this structure. The tip of the left limb of the pancreas is larger than the rest of the pancreas and this is often an area that is affected by pancreatitis. In cats the entire pancreas can be imaged from this side. A long gray scale and high resolution are needed to see the subtle pancreas. The normal pancreas is only slightly different in echogenicity from the surrounding fat. It is best seen in small, young patients that image well. If the animal is fat or aged, then the increased amount of fat within the gland will increase the echogenicity and it will be difficult to distinguish from the surrounding fat. Often, the vessels within the pancreas and the pancreatic duct are seen. The right lobe and the body of the pancreas are best seen from the right side of the dog but are well delineated in the cat from either side. The landmarks for the body and right limb include the portal vein, the duodenum, the right kidney and stomach. The body of the pancreas is best seen from the right side with the transducer positioned parallel to the ~10-11 ribs just behind the liver. To help to locate the pancreas several techniques may be used. Find the mid abdominal aorta and caudal vena cava; slide the transducer ventrally while looking for a branching vessel (the portal vein confluence from the jejunal veins). Follow the portal vein cranially. A branch that comes toward the transducer (laterocranially) is the pancreaticoduodenal vein. Follow this vessel. It will point to the pancreas. While in the mid abdomen, look for a segment of intestine coursing in a craniocaudal direction. It should be straight and then in the mid to caudal abdomen curve medially. If you follow this intestine cranially, it will join the stomach. This is the duodenum. Between the pancreaticoduodenal vein and the duodenum lives the pancreas. If you look at the duodenum in cross section, the pancreas will be located on the mesenteric side or medial and dorsal to the duodenum. The caudal vena cava is dorsal and medial and is not usually seen from this scanning plane. The right kidney is dorsal and lateral. Remember that the right lobe lies in the meso-duodenum dorsomedial to the descending duodenum, ventral to the right kidney and ventrolateral to the portal vein. The body of the pancreas is located between the right lobe and the larger left lobe (in the cat). It is found caudal to the pylorus of the stomach, craniomedial to the right kidney, ventral to the portal vein. The vessels within the right lobe include the cranial and caudal pancreaticoduodenal veins. The cranial vessel empties into the gastroduodenal vein which courses into the portal vein. The caudal vessel joins the cranial mesenteric vein. The vein in the left lobe empties into the splenic vein. Using these landmarks and relationships, the pancreas may be imaged
either from the ventrodorsal position or the recumbent lateral position. Usually, an animal with pancreatitis is less painful when viewed in a lateral position. In addition, if the animal vomits readily when pressure is applied to the abdomen, then the lateral position is safer than a dorsal recumbent position. The proximal flexure of the duodenum may be traced into the pylorus of the stomach. The bile duct may be traced to the papilla where it empties into the duodenum. In the cat the bile duct and the pancreatic duct join in the cistern of Vader before entering the duodenum at the sphincter of Odi. The pancreas is located medial and dorsal to the duodenum. The right kidney and duodenum in a transverse view is a useful landmark to locate the right limb of the pancreas. The gastroduodenal and cranioepancreaticoduodenal veins are useful landmarks to help locate the pancreas. The normal pancreas is mildly hypoechoic relative to the surrounding fat. In younger animals, it is frequently more hypoechoic and easier to image. In older animals, it may become more difficult to see as more atrophy has occurred.

Pancreatic diseases
Typical diseases involving the pancreas are usually related to inflammation or neoplasia.

Pancreatitis
This can be challenging in the cat as there may be no, minimal or significant sonographic changes apparent and the animal still have pancreatitis. The sonographic appearance will vary depending on the severity and duration of disease. The entire pancreas, the adjacent duodenum, the stomach, the biliary tract and peripancreatic tissue all should be included in the exam of the pancreas. Ultrasound findings may be negative in the milder forms of acute pancreatitis. Mild forms may have only an interstitial edema without involvement of the peripancreatic inflammation or fat necrosis. Severe cases have fat necrosis, parenchymal necrosis, hemorrhage and peripancreatic inflammatory changes. These typically sonographically will have varying degrees of hypoechogeticity. The pancreas will become increasingly more hypoechoic relative to the liver and increase in size. The pancreas may appear inhomogeneous. Fluid filled pseudocysts or abscesses may be present. The pancreatic duct may be enlarged. But this has been found to be larger in our older population of cats normally. Focal peritoneal fluid and hypechoic peripancreatic fat are features seen with pancreatitis. If multiple incidences occur, fibrosis and calcification may result and the pancreas will increase in echogenicity. Pseudocysts and abscesses both may form as a consequence of the pancreatitis. The term pseudocysts should be reserved for an accumulation of fluid that persists for several weeks. Often with pancreatitis, the duodenal wall will be thickened and the segment of bowel will appear spastic. The bile duct may enlarge due to an obstruction or partial obstruction. Beware that an enlarged duct does not necessarily mean it is obstructed. In cats with pancreatitis, they may also have hepatic lipidosis and inflammatory bowel disease. The bile duct may be mildly dilated and tortuous in cats with hepatic lipidosis and not be obstructed. With pancreatitis, the duct may become obstructed due to the surrounding inflammation. However, the obstruction may resolve and the duct remains dilated. Compare the ultrasound findings with the clinical picture. A sonographically observed dilated duct along with the appropriate clinical picture is indicative of an obstructed duct. Serial sonograms are important to follow pancreatic lesions. The surrounding fat is hypechoic due to saponification of fat. The animals frequently have been vomiting and often the stomach is empty. Peritoneal effusion is frequently present in more severe cases but may not be as evident in cats with interstitial changes and milder pancreatitis. Chronic pancreatitis is often more of a sonographic challenge. Chronic pancreatitis is more difficult to image as atrophy, scarring with possible mineralization in some areas and inflammation in others may make diagnosis more challenging. In the cat, hepatic lipidosis may accompany pancreatitis. It is often more difficult to recognize pancreatitis in the cat. An abscess within the pancreas is important to try to ascertain as this may be a surgical emergency. An abscess may be difficult to tell from a pseudocyst. The clinical history, the laboratory data and course of disease are often used to help sort this out. Usually the wall of an abscess is thick and irregular. The echogenicity of the fluid contents may vary. Gas may be present. Fibrous connective tissue proliferates. The sonographic findings may include the inability to see the pancreas because the scar tissue resulted in a similar echogenicity as the surrounding fat. The echotexture may be mixed with some areas hypechoic and other areas hyperechoic. The hypechoic areas are most likely due to local inflammation of the remaining pancreas. The hyperechoic areas are likely due to fibrosis and mineralization of the scarred pancreas. The pancreatic duct may be dilated. With acute pancreatitis, thrombi have been seen in the portal vein. These potentially may cause acute severe problems if the portal vein is completely obstructed.

Pancreatic neoplasia
Pancreatic neoplasia may be difficult to tell from pancreatitis in some instances. The history, lab data and thoracic radiographic films evaluating for the presence of metastatic lesions are useful. Two major types of pancreatic tumors are seen. The insulinomas are small and may not be seen. In people, 70% are solitary adenomas, 10% are multiple adenomas and 10% are malignant. The remaining 10% are diffuse hyperplasia or extra pancreatic. The size may vary from minute lesion to huge masses. 90% are less than 2 cm in diameter. If seen they are typically hypechoic and singular but may be multiple. Regional lymph node and liver metastasis may be present and may be all that is detected. The history and lab data supporting hypoglycemia is especially important in the diagnosis. Gastrinomas, a G cell tumor, producing the Zollinger-Ellison syndrome are relatively uncommon. They may be small masses within the pancreas that result in abnormally high gastrin levels with subsequent gastric wall hyperplasia and ulcers. Pancreatic adenocarcinomas are often quite extensive by the time we see the patients. Adenocarcinoma: In people, approximately 70% of the pancreatic cancers arise in the
region of the head, 14-20% in the body and 5% in the tail. In 20% of cases the tumor is distributed diffusely throughout the gland. Metastases to the liver Mesentery, omental and portohepatic nodes are characteristic. There may be marked disruption and enlargement of the pancreatic tissue Complex hyperechoic tissue and mineralization may be present. Obstruction of the biliary tract and metastasis is often evident. Carcinomatosis may be identified as hypoechoic nodules on the peritoneal surface. Evidence of metastasis may be detected in other visceral organs especially the liver.
Imaging of the thorax is an important and useful technique. The heart is typically the organ imaged in the thorax. However, the lungs, the pleural space, mediastinum may be diseased and ultrasound helpful in diagnosing abnormalities. This talk will center on those non-cardiac structures and how disease may appear on ultrasound and be diagnosed.

The cranial mediastinum should be evaluated for suspected masses, thrombi within major vessels or the possibility of perforation of the esophagus. The periphery of the lung, the pleural space, diaphragm, and pericardial sac and superficially of the heart should all be included in the examination of the cranial mediastinum.

Cranial mediastinum
The front leg is extended cranially. The heart is evaluated for cardiac or pericardial disease. Fluid and type of fluid (echogenic or anechoic) is noted either within the pericardial space or pleural cavity. The cranial mediastinum is sometimes challenging to image. The fat in this region and the small window between the lung lobes sometimes influences the complete examination. Normal lymph nodes are not typically seen unless enlarged. Masses in this area especially cysts or enlarged lymph nodes are imaged as hypoechoic or anechoic masses. Often they are well defined. Lymph node masses are not typically highly vascular. They may displace the major vessels within the mediastinum. Highly vascular masses are more likely tumors such as a thyroid carcinoma or heart based masses. These tumors have a propensity to invade local vessels and possibly obstruct them.

Lung
Typically, the lung is air filled and the ultrasound does not penetrate the surface and reverberation artifacts are generated. Disease in the lungs usually interferes with aeration of the lung. The usual reverberation artifact seen at the surface of the lung is affected. Nodules such as from metastasis may have sharp round margins. Pneumonia often has consolidation of the lung with poorly defined borders. Fluid or air may be seen in the bronchi in the consolidated lung—air bronchograms seen on the radiographs. Pulmonary edema (cardiac or non-cardiac) may increase the number of reverberation artifacts at the surface of the lung but not be as consolidated with the lung devoid of air as with other lung diseases. Lung lobe torsion and lung tumors appear as hypoechoic masses. If there is air remaining in a bronchus, then linear hyperechoic lines may be apparent. The consolidated lung looks like the liver ("drowned lung"). Tracing vessels or the bronchus or identifying the caudal vena cava and hepatic veins at the level of the diaphragm will be helpful in distinguishing a diaphragmatic hernia from a lung consolidation.

Pleural space
Pleural diseases such as pleural effusion from chylothorax, pyothorax, heart failure etc. may be documented and the character of the fluid helpful in determining if this is a transudate or exudate. A pneumothorax may be seen as air causing artifacts that do not change in position with breathing. Ultrasound may help in the placement of a needle for aspiration or biopsy of fluid or a mass. A diaphragmatic hernia may be diagnosed with ultrasound. The appearance will vary depending on the contents and the location. Bowel loops may contain gas and produce reverberation artifacts. Usually they are easy to identify because of their shape, location crossing the diaphragm and their wall. Other abdominal contents are also usually easy to identify. The pericardial-peritoneal diaphragmatic hernias may have liver or GI present within the pericardial sac. It is important to try to assess viability of the different structures. Looking at blood flow and the echogenicity of the organs is beneficial. A liver that has vascular compromise may look like an infarcted spleen. It will be hypoechoic and may have thrombi within vessels. The gall bladder and the pancreas may also be within the thoracic cavity and be secondarily affected by the trauma.
Lymph nodes within the abdomen are important indicators of extent or presence of disease. There is a multitude of different lymphocenters present within the abdomen. Specific lymph nodes to be evaluated in each examination include the medial iliac lymph nodes and the jejunal lymph nodes (mesenteric). The medial iliac lymph nodes located at the bifurcation of the aorta in the caudal abdomen. The mesenteric lymph nodes are located in the mid abdomen adjacent to the confluence of the jejunal veins into the portal vein. The normal glands are large enough to be seen in the dog and cat. Additional lymph nodes occasionally seen when they are infiltrated are varied. They are usually located adjacent to the organ they drain and close to vessels. They include: hepatic, gastric, pancreaticoduodenal, splenic jejunal, colic, aortic, renal hypogastric and sacral. The adjacent organs and vessels are used to name the lymph nodes. When a single lymphocenter is found to be abnormal, the region or organ that node drains should be closely examined for disease. Multiple lymphocenters involvement often indicates a more systemic involvement.

Normal lymph nodes may be seen and included each abdominal examination. Normal lymph nodes or reactive nodes may be uniformly slightly hypoechoic relative to surrounding fat and may have a hyperechoic strip in the center (the fat-strip sign). Older patients or patients on chemotherapy or steroids may be small and have an isoechoic or hyperechoic appearance. Normal glands have a fusiform shape and are relatively thin and uniform in echogenicity. The contour is smooth and often there is a thin hyperechoic capsule surrounding the gland. The medial iliac lymph nodes are often smaller cranially and slightly fuller caudally. The deep circumflex iliac vessels (artery and veins) cross over the isthmus at the cranial margin of the glands just ventral to L5-6. The jejunal lymph nodes are long and uniformly thin. There are a group of these lymph nodes that are gathered adjacent to these vessels. The jejunal (cranial mesenteric) arteries and veins are sandwiched centrally between two jejunal lymph nodes at the base of the mesentery. Vessels are very helpful in locating different lymphocenters. Lymph nodes in other locations may be more rounded or unique in appearance. Anatomic size references have been reported in dogs to have a maximum size of 5 mm-) (Bezuedenhout 1993). However, glands 3-6 mm are within normal limits in the author’s experience depending on the size or the patient. When measured, it is usually the width that is recorded. The length is easier to incompletely have in the plane when measuring and more error can be encountered. There are variable sizes depending on the age, breed, size and species. Young dogs especially less than one year have mildly enlarged jejunal lymph nodes likely due to a reactive process associated with the intestinal exposure to multiple new antigens and inflammatory process. The glands should keep their normal shape. The shape is often helpful in determining an infiltrative process. A reactive process usually results in mild enlargement but retention of the normal shape. A ratio comparing the short and long axes should be less than 0.5 in normal nodes (Llabres-Diaz 2004; Nyman eta al 2004). The medial retropharyngeal lymph nodes (1cm width/.5cm height / 2.5 cm in length)

10 Normal cats were imaged. MILN - 100%
Jejunallymph nodes 90%
Hepatic lymph nodes 70%
Aortic lumbar, Splenic Pancreticoduodenal - 60%
Ileocecal and Colic 50%
Renal 40%
Gastric, 30%
Sacral 20%
Caudal mesenteric 10%

Abnormal lymph nodes change in size, shape, margin, echogenicity, and shape. They may have the entire gland affected or part of the gland involved. Depending on the disease and stage of disease, the glands become enlarged, loose the fusiform shape and become rounded.

Often lymph nodes with lymphosarcoma are large, have a rounded shape and are very hypoechoic.

Ratios that compare the long and short axes increase significantly with an infiltrative disease especially neoplasia. A Short/Long axis ratio > .5 has been used to predict neoplasia. Neoplasia often affects the internal appearance of the gland. This may include mineral (often from prostate gland or anal carcinomas). In people, the hyperechoic fat stripe is sometimes used to denote a benignancy and the loss of the stripe is used to indicate a malignancy. An infiltrated gland often becomes more hypoechoic and heterogeneous in appearance. The outside contour of the gland may become irregular. There may be extension of the tumor (especially lymphoma) to the root of the mesentery. It may become extensive and result in obstruction of the vessels at the root of the mesentery and subsequent devitalization of the intestine. The enlarged glands (other than systemic disease) are located adjacent to the affected organ. Exceptions include the sternal lymph nodes that drain the body wall and the peritoneal cavity. It is helpful to know what a specific lymphocenters drains and then to carefully examine that area. The medial iliac lymph nodes drain the pelvic limbs, the caudal abdominal cavity, and
the perineal region. It is important to note if both sides are affected or only one side. If only one side is affected it is useful to carefully scrutinize that side including the anal sac on that side. The glands may become cystic due to degeneration, abscess formation or associated with neoplasia. Granulomatous or inflammatory diseases such as Histoplasmosis, Pythiosis, Mycobacterium, FIP or others may mimic infiltration by neoplasia and may result in enlarged infiltrated glands. Following therapy especially for lymphosarcoma, the center of the lymph nodes often become hyperechoic as hemorrhage and necrosis occurs. Mineralization may be seen as bright hyperechoic areas. If they are large enough, then shadowing may occur. This feature is often seen with prostatic tumor or perineal carcinomas. Ultrasound is much more sensitive than radiology in detecting lymph node enlargement. Aspiration of lymph nodes is often useful in determining the extent of disease or the underlying cause for disease. It is most productive the larger the glands. Aspiration of only mildly enlarged glands especially the mesenteric lymph nodes is often unrewarding. The glands have a tendency to move away and few cells may be harvested. The vessels are close by and may be penetrated if the patient or gland moves. Even small cell lymphoma may not provide enough unique cells for the cytopathologist to make a diagnosis. When evaluating the lymph nodes, the appearance of surrounding structures is important in the overall interpretation. Other lymph nodes such as the medial retropharyngeal, the cranial mediastinal, axillary and other nodes are often examined when disease is suspected in the region.

The evaluation of lymph nodes should be a part of every abdominal exam especially when attempting to diagnose an abnormality as cancer or to stage the cancer or for progression of disease as a response to therapy.

References
A Elke Schreurs, et al. Ultrasonographic Anatomy Of Abdominal Lymph Nodes In The Normal Cat. Veterinary Radiology & Ultrasound. 200849 (1);68-72
Breathing is Fundamental:
Keep an Eagle Eye on CO\textsubscript{2} to Make Life in Surgery Easier
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Mississippi State University
Mississippi State, MS
Jennifer Wardlaw, DVM, MS, DACVS
Gateway Veterinary Surgery
St. Louis, MO

Breathing and anesthesia involves a lot more than oxygen, it’s all about carbon dioxide. Monitoring it, equipment options and goals for the patient will be discussed. Surgical cases where this is especially important will be discussed and well as suggestions for pre and postop care of “at risk” cases.

- Several patient considerations make it more difficult or less likely for a patient to ventilate well enough on their own
  - Mucous membrane color and SPO\textsubscript{2} are not sensitive enough to indicate issues until they are critical
  - Respiratory rate gives no indication for the quality of breath (depth of breath) they are taking
  - Gas exchange is the critical component for breathing
- Obese patients are less likely to breath well on their own
  - Extra weight on rib cage
  - Weaker muscle strength, even before they are asleep
- Dorsal recumbency – most common position for surgery (electives, exploratory, knees etc)
  - Unnatural breathing position
  - Puts more weight on diaphragm
- Pregnant animals or laparoscopic procedures
  - Extra pressure from babies or insufflation on diaphragm
  - Relies more heavily on intercostal muscles to breath – negligible when anesthetized
    - C-section
- Geriatric patients have weaker muscles, more sensitive to drugs and can have secondary lung disease making ventilation more troublesome
  - “old dog lungs”
- “At risk breeds”
  - Cavalier king Charles – syringomyelia, CSF flow issues, Chiari Malformation
    - Increasing intracranial pressure can result in herniation of the brain
  - Brachycephalic breeds
    - Increased oropharynx soft tissues
    - Obesity
    - Increased vagal tone
    - Hypoplastic trachea 1” compromise to airflow
    - Postoperative breathing is also vital during recovery period in these patients
- Trauma case
  - Need to keep CO\textsubscript{2} low to assure cerebral pressure does not climb
  - Pain from trauma can further decrease thoracic compliance to breathing
    - Pain with ribs or intercostal muscles
    - Pulmonary contusion pain or lack of lung compliance for air movement
- Preoperative bloodwork should be performed for general anesthesia health
- Spo\textsubscript{2} and oxygen used for preoperative prepping
  - Many will preoxygenated BAOS patients and pregnant animals 5-10 minutes before induction
- Have capnography hooked up in the prep area
  - One of the most critical times for anesthesia risks
  - Drugs in full effect and most common time to be “too deep” and have breathing compromise
- Under normal physiological conditions the primary indication for mechanical ventilation during general anesthesia is patient PCO\textsubscript{2}. Patient oxygenation does NOT define ventilation.
- There are two ways to monitor patient PCO\textsubscript{2}: arterial blood gas analysis and/or end-tidal PCO\textsubscript{2} (PETCO\textsubscript{2}, capnography).
- Although arterial blood gas analysis is more accurate, it is also expensive and impractical. Capnography provides a useful, and practical, means to monitor patient PCO\textsubscript{2}, and is recommended for all anesthetized patients undergoing mechanical ventilation under general anesthesia. There are two categories of capnographs: main-stream, which analyzes the patient’s
exhaled breath adjacent to the endotracheal tube, and side-stream, which removes a sample of the patient’s breath and delivers it to an analyzer away from the patient.

- Capnography is based on the principle that end-tidal exhaled PCO2 (PACO2) is roughly equal to pulmonary arterial PCO2 (PaCO2)
- Graphical illustration of the PETCO2 over time is called a capnogram. Capnograms are useful for visually monitoring an anesthetized patient’s PCO2 and other problems that can develop, such as a leak in the breathing system.
- Several capnograms will be demonstrated with discussion regarding their interpretation and how to mitigate associated issues.
In this seminar Dr. Landsberg will collaborate with a colleague to discuss real cases of canine aggression cases and the selection and use of drugs and natural supplements for these cases. Therefore the summary below provides a brief overview of drug selection and use.

**Pharmacotherapy and canine aggression**

When a dog is excessively aroused, fearful, anxious, overly reactive lacking impulse control or “behaviorally abnormal”, psychotropic medications are indicated to improve the problem as well as address the dog’s well-being. However, drugs do not change the relationship with the stimulus; therefore, concurrent behavior modification is needed to desensitize, countercondition and train desirable.

Selective serotonin reuptake inhibitors might be most effective for hyperactivity, aggression, social anxiety, generalized fear and anxiety and panic disorders. Four weeks or longer is generally required to achieve full therapeutic effects. Starting the medication at the time of the consultation allows time for the drug to reach optimal therapeutic effect when the exposure program begins. Medication might not be required for dogs that can be effectively kept away from fear-evoking situations, provided the dog is sufficiently settled and relaxed. Adjunctive medication to further reduce anxiety especially prior to stimulus exposure might include benzodiazepines, trazodone, clonidine or propranolol, alone or in combination. If effective these drugs might be used several times a day.

**Psychotropic drugs**

Selective serotonin reuptake inhibitors (SSRI) are most commonly used in dogs that are behaviorally abnormal, to control reactivity and impulsivity, reduce fear and anxiety and improve trainability as well as address the dog’s behavioral well-being. SSRI’s are selective in blocking the reuptake of 5HT1A into the presynaptic neurons. Fluoxetine and paroxetine might be useful for general anxiety disorders, stabilizing mood, reducing impulsivity and behaviorally pathologic aggression. Fluvoxamine and sertraline are other options for social and irritability.

The primary mechanism of action of TCA’s is to block the reuptake of serotonin and to a lesser extent noradrenaline. They also have anticholinergic and antihistaminic effects which may contribute to varying levels of sedation, urine and stool retention. Clomipramine and amitriptyline may be useful in controlling underlying anxiety and impulsivity in aggressive dogs. However, studies have shown no effect of amitriptyline or clomipramine on canine aggression.

While antidepressants reach peak plasma levels within hours, reuptake inhibition may induce down-regulation of postsynaptic receptors that are responsible for clinical effects. Therefore, 4 weeks or longer is generally recommended to fully assess therapeutic effects.

Buspirone is a serotonin (5HT1A) receptor agonist and a dopamine (D2) agonist. It is used for mild fear and anxiety. It is non-sedating, does not stimulate appetite, and does not inhibit memory. It takes a week or more to reach effect. Adding buspirone to an SSRI might add to the serotonin pool.

Benzodiazepines potentiate the effects of (GABA), an inhibitory neurotransmitter. They cause a decrease in anxiety, hyperphagia, and muscle relaxation. Most have a rapid onset and short duration in dogs. They can be used alone or adjunctively primarily on an as needed basis but may be considered in select cases on an ongoing basis with multiple daily dosing. They may cause paradoxical excitability, increased activity, and an amnesic effect. Buspirone and benzodiazepines can disinhibit fearful and inhibited pets which may result in aggression.

Beta blockers such as propranolol reduce physiologic signs of anxiety (heart rate, respiratory rate, trembling). Therefore they might be most useful if combined with drugs that reduce behavioral anxiety. Clonidine a selective alpha-2 agonist that blocks noradrenaline, might be used together with SSRI’s for situational use in fear or territorial aggression, separation anxiety, or noise phobias.

Trazodone, a serotonin 2A antagonist-reuptake inhibitor, may be useful in dogs for generalized anxiety, separation anxiety, storm phobias, and some forms of aggression including interdog aggression and impulse control disorders. Trazodone can be used on as needed basis alone or in conjunction with a TCA or SSRI or 2 to 3 times daily.

Focal seizures of the temporal lobe may present with mood alterations or hallucinatory and self-traumatic behaviors. Generalized seizures may be associated with aggression e.g. in the post-ictal phase. Therefore anticonvulsants may be a consideration in diagnosis and treatment. Levetiracetam may be effective for focal seizures, and for anxiety, panic, and mood disorders which may have comorbidity with epilepsy. Gabapentin might be combined with SSRI’s for the treatment of impulse control disorders, noise phobias.
and to reduce reactivity. Carbamazepine is also a mood stabilizer that may be a useful adjunct to SSRI’s for irritable and impulsive aggression.

Neuroleptics decrease motor function at the level of the basal ganglia in the brain, elevate prolactin levels and may reduce aggression as dopamine antagonists. Phenothiazines such as acepromazine are sedatives but do not reduce anxiety.

Selegiline is an MAOB inhibitor licensed for CDS in North America, and emotional disorders in Europe. Chronic stress associated with stereotypic and displacement behaviors, fear aggression, and autonomic signs, may have elevated prolactin levels, which might improve with selegiline, while lower prolactin levels are seen with acute onset fears and phobias which might improve with fluoxetine.

Complementary and alternative medications are another option; however, few have been assessed in evidence based studies. Products that might be useful in reducing anxiety and improving trainability include Adaptil, alpha-caseozepine, l-theanine, melatonin, Harmonese and aromatherapy. Each of these might be used concurrently with drug therapy. Aggression might be reduced by supplementing tryptophan to a reduced protein diet (to optimize entry through the blood brain barrier). In addition, adding tryptophan to an SSRI or TCA may increase the available serotonin pool. Royal Canin Calm diet contains both alpha-caseozepine and l-tryptophan. There have been no studies to demonstrate efficacy of other natural products including Bach flower remedies or homeopathy.

**Abnormal aggressive dogs**

For most cases of behaviorally abnormal dogs an SSRI such as fluoxetine or paroxetine would be the first choice for managing underlying anxiety and impulsivity. Immediate acting medications might be needed concurrently prior to specific events including benzodiazepines (e.g. alprazolam, lorazepam, diazepam), trazodone, clonidine, or propranolol. Drug combinations may be a consideration but safety and potential for reaefficacy must be weighed against potential adverse effects. Natural products might also be used concurrently. In some cases drug combinations will need to be considered such as a combination of SSRI with carbamazepine, gabapentin, clonidine, trazodone, buspirone or even a TCA (with cautious monitoring for serotonin syndrome).

**Drug doses for behavior therapy**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
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<tbody>
<tr>
<td>Alprazolam</td>
<td>0.02-0.1 mg/kg bid to qid</td>
</tr>
<tr>
<td>Clonazepam</td>
<td>0.1-1.0 mg/kg bid to prn</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.5-2 mg/kg prn to q6h</td>
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<tr>
<td>Lorazepam</td>
<td>0.25-0.2 mg/kg sid to prn</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>2.0-4.0mg/kg bid</td>
</tr>
<tr>
<td>Citalopram</td>
<td>1-3 mg/kg bid</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>1.0 – 2.0 mg/kg bid</td>
</tr>
<tr>
<td>Fluvoxamine</td>
<td>1.0 -2.0 mg/kg sid – bid</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>0.5-2.0 mg/kg sid</td>
</tr>
<tr>
<td>Sertraline</td>
<td>1.5 mg/kg sid or divided bid</td>
</tr>
<tr>
<td>Clonidine</td>
<td>0.01-0.05mg/kg prn to tid</td>
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<tr>
<td>Propranolol</td>
<td>0.5-3.0 mg/kg bid or prn</td>
</tr>
<tr>
<td>Buspirone</td>
<td>0.5-2.0 mg/kg sid-tid</td>
</tr>
<tr>
<td>Trazodone</td>
<td>2 to 8 mg/kg prn to tid (up to 15 mg/kg prn)</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>10-30 mg/kg bid to tid</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>4-8 mg/kg bid to tid</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td>20 mg/kg tid</td>
</tr>
<tr>
<td>Selegiline</td>
<td>0.5-1 mg/kg sid in am</td>
</tr>
</tbody>
</table>

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Apples and Oranges: Significant Differences Between Fear Free and Fearful Practices
Marty Becker, DVM
North Idaho Animal Hospital
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There are three things I can pretty much predict someone will say every time these situations arise:

1. **Cute baby in sight.** I’ve always loved children and, like everyone else who sees a baby, I always say in a syrupy sweet voice, “Ahhh….she’s so cute!” The baby could be wrinkled, bald, and spewing pablum, and we still say the same thing.

2. **Pets lower blood pressure.** Since veterinary school I’ve been privy and partner to the human-animal health connection and wrote an award winning book on the subject called *The Healing Power of Pets*. When doing the initial book tour in 2001 and every time since then, when the book’s subject title comes up, 7 out of 10 people say, “I hear pets lower blood pressure.” It’s like that’s the only benefit of pet companionship anyone has ever heard of.

3. **Fear Free veterinary practice.** I’ve yet to speak on this subject without at least several members of the audience assuring me, “We’ve been doing Fear Free for years.” In some ways, yes they have, but in most of them, no, they haven’t – and here’s why.

Starting in the late 1980s, working with famed veterinary behaviorist and inventor (Gentle Leader) Dr. R.K. Anderson, we tried to become “All Treat Veterinary Hospital” vs. “All Pain Veterinary Hospital” by deploying treats at every moment of truth in a veterinary exam or procedure. Yes, this helped reduce anxiety and stress for pets, and is a component of a less-stress visit, but it wasn’t Fear Free.

Fear Free is a multimodal approach to removing or reducing anxiety/stress/fear triggers, mitigating these same factors when they arise, and always having the pet’s emotional wellbeing in our hands (along with physical wellbeing).

Let me contrast a Fear Free (FF) practice with one that remains fearful for pets, pet owners, and the hospital team:

1. **Primary focus**
The North Star for a FF practice is the emotional wellbeing of all stakeholders (pet, pet owner, practitioner, practice team). In a fearful practice, the focus can be medicine, money, or both.

2. ** Hiring**
FF practices hire for attitude and genuine care and compassion for pets and people. They’re looking for that special 1 out of 10. Fearful practices hire for performance above all, and pay little or no attention to attitudes of compassion in their team members.

3. **Harmony**
Fearful practices care more about getting the job done than the emotional environment of the business, out front and in back. Fear Free practices know both pets and people can easily detect, and are put off or made anxious by, stressful interactions among members of the hospital team.

4. **Design**
In the fearful hospital, design is all about curb appeal, efficiency, durability, safety and cost. The FF practice has colors that please pets and people, use high coefficient of friction floor coverings, use a lot of soundproofing, have species-specific exam rooms (some have species-specific treatment areas), and have the ability to isolate pets who are hospitalized for procedures or treatment from pets who are currently being treated or examined to minimize the viral spread of fear.

5. **Where pets are parked**
In the fearful hospital, appointments are booked without thought, and in doing so aggressive pets can be side-by-side with fearful ones, dogs with cats, or new puppies with hyperkinetic, emotionally damaged pets. Fearful hospitals pack the schedule and the waiting area with nervous pets and people. Fear Free practices utilize the exam rooms (with enough rooms and the technology) for checking pets in-and-out, bypassing that stewpot of stress called reception. If there aren’t enough rooms or the technology, FF practices have pet owners check in (leaving the pet in the vehicle) and then go back outside and wait with their pet in the vehicle or outdoor area until it’s their turn to be seen.

6. **Pace**
Fearful hospitals are all about auguring the pets through the system to help as many animals medically as possible. More pets = more money. FF facilities are all about smart scheduling (looking how to minimize stress for individual pets and people), being more relaxed, working to remove or reduce excessive noise, and not being afraid to delay or stop a procedure if fear flares.
7. Exam
In a fearful facility, almost every pet is put up on the exam room table for exams and procedures. In FF, the pet is given the choice of where to be examined and treated (on the table, floor or lap).

8. Hold still
To examine ears, eyes, wounds, or to draw blood or give vaccinations, fearful practices using restraint, which is designed to protect the healthcare provider. FF practices use Gentle Control or Stress Less handling, which are designed to protect both the pet and the person.

9. Sedation
In fearful practices, sedation is rarely used and is thought of as a last resort. Sedation is a resort reached for early and often in a Fear Free practice. FF practices have a saying, “If you can’t abate (as in anxiety/stress/fear)…then you must abate (with sedation).”

10. Records
It’s all about the medical record in a fearful practice, whereas in the FF practice, there’s a great medical record but it’s bookend is a great emotional record. It includes things like the anxiety/stress/fear triggers for this unique pet and pet owner; where the pet likes to be examined; what Gentle Control method works best; what treat the pet likes best, what tricks the pet knows, with cue words; what products (compression garments, chill pills, pheromones, sedatives) work best with this pet.

So just as not every baby really is the cutest one every born, and pets do so much more than lower blood pressure, Fear Free is not about adding treats and a warm bedside manner to your “get ‘er done” veterinary strategy. And you can take that to the bank, fearlessly – guaranteed!
Fear Free Practice:
With it You’ll Thrive- Without it, You May Not Survive
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I’ve been a practicing veterinarian for over 35-years and I’ve seen three previous transformations of significance:

1. Feline Medicine – I grew up on a family farm/ranch in rural Southern Idaho. In that environment, cat and veterinarian didn’t belong in the same sentence (would be as odd as smoking and healthy). By and large the vast majority of cats with barn cats, lived in the haystacks or were outside cats. Very few ever went to a veterinary hospital for treatment and even fewer for preventive care. Eventually veterinary medicine realized that cats, too, were beloved pets, weren’t just small dogs, that they were a separate species that needed their own meds, dosages and treatment protocols.

2. Dentistry – Circa my graduation in 1980, dentistry consisted of the occasional ultrasonic teeth cleaning and a lot of pulling rotten teeth. Talk of prevention was rare. Then came veterinary dentists and the blossoming of dental care with a focus on prevention (daily oral care), digital dental radiographs, dental suites and advanced therapy.

3. Pain management – Have pity on this profession that for so long, we ignored the existence of and consequences from pain in pets. Thankfully, we started to focus on pain, developed multimodal pain management, and made the prevention or relief of animal pain or suffering a priority.

Of course, feline medicine is limited to cats that receive veterinary care (still far to few). And although 80% of companion animals have dental disease by the age of three, the majority of pets suffer in silence with a mouth full of inflammation and infection. Pain management is widely offered, but this only helps with pets that have pain (trauma, joint or disc disease, post surgery). The most important transformation in the history of companion animal medicine is here now. And rather than just affecting a species or a condition, this initiative involves every pet, every veterinary visit, every practice, every day, forever. This make or break transformation is creating Fear Free veterinary visits.

The polar opposite of a fad, Fear Free has become a practice imperative if you’re going to thrive, or even survive. Too strong of a statement? Far from it. You should fear not being a Fear Free veterinarian, technician, hospital team member or facility. Why? Because pet owners can see, hear, feel if you’re Fear Free and can measure success.

Before Fear Free, companion animal practices largely competed on location, convenience, curb appeal, quality of medicine and price. Of these, the most subjective is quality of medicine. You’ve heard it said a dozen ways, hundreds of times, but it’s literally impossible for a pet owner to judge the quality of medicine. How do they know inside of the incision that did a better job of surgery than someone else. Or that another veterinarian could have removed that carnassial tooth with much less trauma, or done a much better job of cleaning out infected ears. Fear Free is different and a differentiator, because pet owners can easily tell at every moment-of-truth (outside the hospital front door, in reception area, exam room, interaction with technician and veterinarian, getting preventive care) whether their pet is calm or anxious/stressed/fearful.

Our profession, and increasingly pet owners, are coming to understand that maladaptive fear is the worst thing a social species can experience and that it causes permanent damage to the brain. And that going to a veterinary practice (or grooming or boarding) where the pet will experience fear, is causing repeat, severe psychological damage to the pet. Conversely, taking a pet to a facility that looks after both the pet’s physical and emotional wellbeing, is better for both the pet and the pet owner.

The Bayer Veterinary Healthcare Usage study showed conclusively that the #1 reason people weren’t taking their pets to the vet as often was because of stress to the pet. And #3 was stress to the pet owner taking the stressed pet to the veterinarian (#2 was money btw). So in providing a Fear Free experience for the pet, you can eliminate two of the top three reasons people aren’t taking pets to the veterinarian. More pet owners visiting the veterinary for their pet’s accidents, illnesses and preventive care means everybody wins.

For individuals and facilities that remain fearful, many will wither and some will die. For those that embrace the necessity of looking after the physical and emotional wellbeing of pets (plus pet owners and team members), they are going to thrive.
The Top 10 Ways to Get Started with Fear Free Practice
Marty Becker, DVM
North Idaho Animal Hospital
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Are you, your hospital team members or clients some of the millions of pet owners who’ve seen recent Internet slide shows featuring frightened pets at the vet? Meant to be funny --- with images of huge dogs in people’s laps, cats inside the exam room garbage can, canines trying to hide in a tiny toy box, felines hunkered down behind canisters of Q-Tips and gauze on the counter – are actually so sad they make me, as a veteran veterinarian and lifetime pet lover, want to cry. Why?

Pets that go to the veterinarian in a fearful state often end up with repeat, severe psychological damage. Like PTSD, these pets don’t even have to suffer the stressful circumstances, such as being hoisted on the exam table, poked, pricked or restrained, to have fear. Just the sight of the carrier coming out or being loaded in the car can be enough to bring back the traumatic memories of a previous veterinary visit. Imagine the added stress of a pet that’s been languishing in the shelter or just recently moved from the cage to your couch. And interestingly enough, once you know the signs of anxiety and fear, you will see it in most animals, even the ones pet owners think like going to the vet.

But just as dentists changed from focusing just on oral health to looking after children’s dental and emotional health (I hated to go to the dentist as a child and now my Granddaughter looks forward to it!), as a profession we’re now embracing our obligation to look after both a pet’s physical and emotional wellbeing.

Can you imagine an environment where most dogs want to go to the vet and remain calm throughout the process of moving from the living room to the exam room and back home? A process through which cats find going to the veterinarian less stressful than having a stray cat wander through their yard, taking a bath or oral medications? You don’t have to imagine because veterinarians, technicians and practices across the country are learning the techniques and protocols to provide for Fear Free veterinary visits for all pets. Stress less. Fear Free. Whatever you call it, it just means that pets won’t hate going to the vet and pet owners won’t cringe at the thought of taking them.

Fear Free requires an extra effort, not just on the part of the veterinary healthcare team, but also the pet owner (compared to past veterinary appointments). Here are my Top 10 pet owner tactics and tips for a Fear Free veterinary visit:

1. Remove or reduce anxiety triggers
Does your cat or small dog cringe when the carrier comes out? Then take it out several days before the wellness visit and feed high value food or treats in it. But don’t force your pet- let it find the goodies on its own. Do you have pets that hate to be separated? Take them both to the vet. The one will get examined and treatment but both will get treats.

2. Make the carrier a little slice of heaven
If you’re one of those people whose carrier only comes out when it’s time to go to the vet, you can make it more like a condo than a cage by a) leaving it out all the time, b) treat it like a safe room for the pet, c) frequently put toys and treats inside of it. If you don’t have the space or desire to leave it out, then at least take it out three days before the scheduled visit.

3. Magic carpet ride of pheromones
“Ask your veterinarian for a recommendation of a synthetic version of naturally occurring dog or cat pheromones,” recommends Gary Landsberg, a boarded veterinary behaviorist and the author of “Behavior Problems of the Dog & Cat.” Landsberg warns that pet owners need a product that’s clinically proven to work not just has “Veterinary Recommended” on the label which means nothing. By spritzing or wiping the pheromones onto surfaces like the pet’s bedding, the door and walls of the carrier, the upholstery of the car you deliver a magic carpet ride that extends from carrier to car to clinic.

4. Just chill out
Many pets require some chill pills, focusing on calming wraps, compression (calming) wraps, ear covers that reduce auditory stimuli, some calming music (classical not heavy metal) or all of them. I recommend starting out with products you can get from your veterinarian that work in a natural way. One of my favorites is a green tea extract in a chewable tablet (has amazing palatability in both dogs and cats), another is a capsule containing milk proteins. The compression wraps act like the deep hugs you get when someone wants to comfort you, swaddling a baby or the tightening some autistic people find so relaxing. And if you know from previous experience that the visit will be stressful discuss in advance with your veterinarian whether anxiety reducing medications might be right for your pet. “There are medications which in combination with supplements (such as melatonin or sun-theanine) that are generally safe and may help decrease anxiety and fear in dogs and cats,” says Dr. Alicia Karas, who teaches and practices anesthesia and pain medicine at Tufts University’s Cummings School of Veterinary Medicine. “These can be prescribed by your vet and given an hour or so prior to leaving for the clinic. Very strong sedatives are not suitable for pet owners to give, but if
coordinated with the veterinary staff, can be given immediately upon arrival to permit examination and minor procedures such as radiographs or ultrasound or even suture removal.” Realize that being sleepy or groggy is often preferable to recovering from a severely stressful experience and just let them safely “sleep it off”. Talk to your vet about this – especially if you have had tough visits in the past.

5. Hungry will mean happy
Withhold the bulk of your pet’s food within 12 hours of the veterinary visit so that they enter the clinic very hungry and more likely to accept food rewards from the veterinary team. Landsberg warns, “If your pet is a puppy, kitten, a miniature breed or has any health issues, check with your veterinarian on how long to withhold.” A great thing to do is to bring your pet’s most favorite treats to the clinic so that they are delighted when they flow copiously.

6. Ready, steady, go
Pet owners typically put the carrier containing the cat or small dog on the car seat. Problem is the cant of the seat bottom makes the carrier tilted which if discomforting for the pet. There are commercial products available for keeping pizzas flat on the car seat that can be used or you can just use rolled up towels. A seat belt can provide further restraint for the carrier.

7. See no evil
Face the level carrier towards the front of the vehicle. Cover the carrier with a light sheet, leaving only the front door uncovered. This greatly reduces visual stimuli (such as dogs being walked, vehicles whizzing by) and increases chances for a state of calm.

8. Forget the baby talking
We put GoPro cameras in pet owner’s vehicles and watched and listened to what happened. What a hot mess! The problem was pet owners saw their pet’s stressed or were stressed themselves, they baby talked the pet, the pet got more anxious, the pet owner got stressed and they kept feeding upon each other until both pet and pet owner were ready to boil-over by the time they pulled into the veterinary clinic parking lot. In short, what you say and do, will affect how your pet feels. Don’t force the issue, raise your voice or get angry or upset. A better thing to do is to remain calm and quiet or upbeat (pretend it’s a trip to the dog park or the pet store). Bringing along a favored toy may also help to keep your pet occupied and happy.

9. Don’t just check in as usual
For decades pet owners have followed the well-rehearsed route of stopping in the veterinary hospital parking lot, grabbing the carrier or the leash and taking the pet inside to check in at the receptionist desk, waiting in the reception area with other pets and pet owners for your turn, and then going into the exam room. The reception area is nothing short of a stewpot of stress. Ask your veterinarian if you can either a) be taken straight into an exam room to be checked in, or b) park the vehicle, call or text the front desk to let them know you have arrived, and wait with your pet until an exam room clears and it’s your turn to be seen. NOTE: It worth reminding to keep AC on if it’s hot outside.

10. Building on success
Having a stress free, positive veterinary visit puts your pet on the road to success. After a negative experience future visits become increasingly more traumatic for the pet. But following positive visits, future visits become increasingly easier and more enjoyable for all.
Use client education to show your community how your practice is a behavior- and training-friendly clinic that enhances pets' well-being.

**Teamwork approach**

Outside resources to address behavior issues alongside: Veterinary Behaviorist, Associate Certified Applied Animal Behaviorist, Certified Applied Animal Behaviorist, Veterinary Behavior Technician, trainers certified, insured and reward based methods. Look for team members and resources who will use reward based training without the use of punishment to avoid various pitfalls of coercion based training that includes increased rates of aggression and potential for a bite. The Humane Hierarchy defines the use of methods helpful for changing behavior that are effective with less risk.

Addressing fear early by doing preventive training and addressing fear at the early stages for puppies and kittens gives the best chance of success. For this reason, puppy classes and kitten classes are one possible resource for clients that ultimately make the care easier and less upsetting for animal and veterinary team members. Early handling of puppies and kittens as well as a variety of socialization experiences done in a positive, gradual manner are protective.

The time period between 6 months to 3 years of age is the prime time when behaviors issues develop or worsen. For this reason watching over the animal’s emotional welfare and behavior during this time allow for addressing issues early. Consider classes or workshops that may help dogs during this transition time in particular in addition to regular veterinary checks. Specialty classes like Cautious to Confident Canine or Rowdy Rover Classes allow for specific triggers like barking and lunging at other dogs or fear of new people and situations to be addressed. Some dogs are better suited to private training and a behavior modification plan as directed by the veterinarian or in some cases working in conjunction with a veterinary behaviorist.

Poor behavior in animals is unfortunately associated with a higher rate of surrender by pet owners. Thus, improving behavior ultimately keeps pets in the home longer. Giving proper chewing outlets and providing appropriate exercise are two ways to help dogs settle into their homes and show less chewing and hyperactive behaviors that are a main complaint of many dog owners. In addition, look for sources of barking, such as underlying anxiety or fear that may be causing the problem behavior. Food puzzles, regular play sessions, enrichment and an appropriate environment with ample resources are helpful strategies for reducing stress.
There are two types of veterinary hospitals – busy ones, and barely surviving ones. What do they both have in common? Both need a commitment from all staff to create a culture and environment that focuses on the pet’s physical well-being. Both need to address their patient’s fear, anxiety and stress in order to provide optimal healthcare, and in order to provide the best experience imaginable for pets, pet owners, and veterinary staff. Both benefit from embracing the paradigm shift in healthcare – the essence of Fear Free™.

The ideal Fear Free™ exam starts with the hospital partnering with the pet owner in effort to create a fantastic experience. The appointment is scheduled having given consideration to the needs of the pet and other activities in the hospital. We work with the pet owner to then properly prepare the pet for transport to the hospital. We email clients clear instructions, reminding them of their appointment, and reminding them what they can do to prepare their pet for transport. Instructions specifically address how to introduce the carrier (when appropriate), how to prepare the pet, and how to prepare the car. Pets often arrive hungry so we can tempt them with food rewards at every step along the way.

Upon arrival at the hospital, pets are behaviourally conditioned to enter the exam room willingly and happily based on past positive experiences. Pets are examined in an area chosen by them. Approach techniques are slow, calm and sometimes intentionally delayed. The exam room is purposefully prepared to both draw attention to our Fear Free efforts, and to create a calming environment. My exam room is like a spa!

The exam process is carefully planned in advance. Each pet is treated as if they are the most important pet in my practice. A concerted effort is made to ensure that every pet is calm, relaxed and comfortable prior to starting the exam. The exam includes slow purposeful movements when touching the pet. Careful consideration involves the selection of vaccines, and preparing the pet for painless blood and urine sample collections where indicated.

When the pet is hospitalized, staff are empowered with the knowledge and resources to insert IV catheters using pain-free, stress-free techniques. The pet is then transferred to an appropriate enclosure. Animal housing requires careful and individual attention giving consideration to the individual’s preference for lighting, sensory stimulation, and caging preferences. Pets requiring radiography are properly prepared in advance. Various protocols are offered to create varying degrees of sedation. With appropriate sedation, staff efficiency is increased, patient positioning and subsequent x-ray quality is superior, staff are never exposed to radiation, and pets recover comfortably and uneventfully after the procedure is complete, at which time, they are returned to their comfortable enclosure. Careful and aggressive attention to pain relief is provided throughout the hospital stay where indicated.

But what happens when two patients in your hospital do not receive the same level of Fear Free care? Two very different experiences start to emerge. The negative experience of one pet starts to have impact on the other. Staff become torn when their efforts start to become undone. The individual pet’s healthcare can be both directly and indirectly compromised due to neglect. Consequences can be disastrous for both pet care, staff morale, and your business. It is of utmost importance to have consistency across your practice, with all staff on board.

When the entire team works together through every aspect of your patient’s care, pet healthcare is optimized, and staff feel both rewarded and accomplished. Pet owners have always been in search of something better, but didn’t know how to ask. Now your hospital is well positioned to “make every visit to the veterinary hospital the best experience imaginable”. With this approach, you start to differentiate your business from your competition, you rebrand, and your business prospers.

References
Reversing the Decline in Veterinary Care Utilization: Progress made, challenges remain. AAHA-AVMA white paper 2014
Wong T. Chew-Toy preferences in kennelled dogs. UC Davis, 2007 Web
Stress and anxiety are perhaps the most common reasons that pet owners cite for not wanting to visit the veterinary hospital. The Veterinary Care Usage Study and subsequent update indicate that over 50% of pet owners feel that their pet dislikes going to the veterinarian, and that greater than one third of pet owners get stressed just thinking about taking their pet to the veterinarian. Experience and careful observation reveals that those sentiments are not restricted to just the outpatient visit, but also apply and contribute to the pet owner’s reluctance to hospitalize their pet. In addition, pet owners are reluctant to approve necessary routine procedures such as blood collections and x-ray for much the same reason. Implementing strategies to maximize patient comfort is the most prudent way to create successful experiences while visiting the hospital. Pet owners visiting my practice have been overwhelmingly accepting and appreciative of any effort to ease their pet’s fear and anxiety. My staff are also eager to accept the Fear Free™ concepts because it leads a more enjoyable work environment including calmer pets, reduced bites and scratches, a quieter work environment, and better quality medicine.

While in the hospital, healthcare workers need to pay attention to the signs of pet fear, anxiety and stress, including hiding at the back of the cage, hiding under bedding, trembling, vocalizing, panting, retraction when approached, pacing, and hypervigilance. When we take action to prevent fear, anxiety and stress, or take action to intervene when signs are present, then we create better in hospital experience. The results are more comfortable pets, and safer sample collection. Blood pressure results, blood test parameters, and vital signs are also more accurate and reflective of the pet’s true condition.

The pet’s surroundings and housing
Common belief has long been that dogs are colour blind. Dogs can however see many of the same colours that humans can see. Fear Free™ has developed a colour palette that was selected to be both positive and visually comfortable. Bright lighting can also be uncomfortable for pets. Dimmable lights are ideal. Cages are believed to best suited for housing when there is opportunity for looking outward with few obstructions, or have the option to retract to an area when less sensory stimulation is present is also ideal. This can be achieved by providing boxes, tents, or partial covers so pets can choose their level of stimulation.

Sample collection
Attention should be paid to commonly performed procedures such as blood collection, urine collection, IV catheter placement and removal, treatment of skin wounds etc. Procedures used to make these more comfortable experiences often include the use of compression wraps, topical anesthetics such as Emla cream, pharmaceuticals, and environmental control.

Radiology
For years, pets have been forced into dark X-ray rooms, stretched out onto hard table tops, placed on their backs with their legs being squeezed by the vice like grip of lead lined gloves, while their limbs are pulled in 4 different directions. There is nothing natural or enjoyable about this for pets. Retakes are numerous, and X-rays are often of compromised quality. Several options including compression wraps, pinch induced behavioural inhibition, or pharmaceuticals are often used to create faster, better quality, more comfortable x-ray experiences for pets.

Post-operative care
Pets often experience stress and anxiety due to the direct result of pain. Careful attention must be paid to regularly assessing and addressing pain relief in our hospitalized pets. Environmental control is also critical in ensuring a smooth, comfortable post-op recovery including consideration given to noise levels, music, pheromones, body positioning etc.

A comfortable hospital stay is not only influenced by the care provided to your patient, but is also influenced by the condition of other pets in the hospital. Imagine waking up from surgery when boarders are barking in the dog ward, or the impact that a feisty cat having its blood collected may have on the pet receiving oxygen therapy nearby. Fear Free™ care involves global consideration of the whole hospital environment and all hospital procedures.

When the hospital culture and environment become low stress and Fear Free™, pets are afforded better quality care, and staff work in quieter, safer, more enjoyable workspaces. Staff become more efficient, more productive, and report much higher staff morale.

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Reversing the Decline in Veterinary Care Utilization: Progress made, challenges remain. AAHA-AVMA white paper 2014
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The Big Goal:
Reducing Fear, Pain, and Stress in Veterinary Patients
Ralph Harvey, DVM, MS, DACVAA
University of Tennessee
Knoxville, TN

- Reduce Fear – Pain – Stress
- A “Big Idea”
- Emphasis across our industry
- Benefits for All Involved
- Client’s and Patient’s fear and stress is real
- Sedation can allow for less stress to patient
- Pet owner appreciate compassionate treatment
  - Less stress to pet and pet owner
  - Better overall experience

Multi-Modal approach to fear and stress, just as with pain management or anesthesia
- Gentle Handling Refined AAFP and ISFM Feline-Friendly Handling Guidelines
- Fear - Stress - Aggression
- Separated Areas for Cats or Feline Only Facilities
- Pheromone spray
- Towel wraps
- Remove the top and work in the cat carrier
- Avoid / Minimize “scruffing” or excessive stretching
  - Gentle technique, Less is More…
- Avoid chamber induction of anesthesia

Goals: Sedation is not anesthesia. They can arouse
- Relieve patient anxiety
- Muscle relaxation
- Provide analgesia if required
- Patient is unaware of surroundings, but can respond to noxious stimuli

Avoid chamber induction of anesthesia
- Increase the stress of the patient
- What is the actual Inhalant dose – % MAC
- Delay ability to gain airway control, monitor
- Increases personnel exposure to inhalant gas

Sedation is preferred rather than manual restraint
  *Excessive manual restraint can be stressful to the patient
  *Struggling causes stress hyperglycemia, elevated lactate and norepinephrine
- The patient may injure itself or staff

What if the patient has undiagnosed HCM or Heartworm disease?
  *Prevalence of subclinical cardiomyopathy is 16%
- Number of cats with asymptomatic heartworm disease is?
- Would you put this cat in a chamber?
- Would you manually restrain this case?
- How can you reduce the stress?

Stress reduction is preferred for all critically ill patients
- Deleterious stress and stress-associated responses, particularly disadvantageous for compromised patients.
- Blunting stress response is increasingly recognized as a desirable medical goal.
• Sympatholytic contribution of dexmedetomidine (lower doses) credited with beneficial effects.
• Reduction in catecholamine release and stress responses benefits “brittle” or “fragile” patients.

Many benefits for sedation accrue to all participants
• Reducing the potential for injury and worker’s compensation events:
  • 53% of bite and 82% of scratch wounds by cats
  • Average loss per claim: $1,770.00
  • Preclude injury: appropriate training and use of chemical restraint
  • Reduce Personnel Exposure Radiation

Updated and expanded 2015 AAHA AAFP pain management guidelines
• Pharmacologic and non-pharmacologic modalities
• Evidence-based or consensus of expert opinion
• Behavioral changes – Validated pain scales
• Post-surgical pain is predictable
  • Strategies mitigate adaptive & maladaptive pain
• Chronic pain – DJD predominates
  • Prevalent, Under-diagnosed, Ubiquitous, Progressive Evidence-based strategies emerging in cats

Managing routine surgical pain
• Preoperative opioid plus a tranquilizer/sedative
• (acepromazine, midazolam, diazepam, dexmedetomidine)
• NSAID either pre- or postop - patient risk factors & clinician preference
• Local anesthetics are integral to surgical plan

Managing higher-level pain
• For more severe, protracted or maladaptive postoperative pain:
  • Cold compression – (Not during anesthesia)
  • Alpha-2 Adrenergic Receptor Agonist - Dexmedetomidine
  • Ketamine CRI
  • Lidocaine CRI (dogs but usually not for cats)
  • Gabapentin
  • Epidural analgesia / anesthesia
  • “Less is More” - For everyone involved

Best use of opioids
• Morphine
• Oxymophone
• Hydromorphone
• Fentanyl (Duragesic, Recuvyra)
• Remifentanil (Ultiva)
• Butorphanol
  • (Torbutrol, Torbugsie, Stadol)
• Buprenorphine
  • (Buprenex, Temgesic, Simbadol, Buprenorphine-SR)
• Tramadol (Mu agonist plus SNRI - inhibits reuptake of serotonin and norepinephrine)
• Common adjuvants:
  • Maropitant - eliminates vomiting, returns spontaneous feeding
  • Metoclopramide - gastro prokinetic, antiemetic, tighten LES
  • Famotidine or ranitidine - protect the gut

Better use of opioids
• Buprenorphine - Buprenex
  • Oral Trans Mucosal
  • Nasal Trans Mucosal
• Simbadol – a concentrated buprenorphine
  • FDA Approved for 24 hour analgesia in cats, repeat for 72hr total
o Effective and safe analgesic for cats
o Our first 24 hour duration opioid analgesic for cats
o Plateau or “ceiling effect” as buprenorphine dose increases:

**Maropitant (Cerenia)**
- An antiemetic therapy for the prevention and treatment of acute vomiting in dogs, and for the prevention of vomiting due to motion sickness in dogs
  - Greatly improves return to spontaneous feeding following opioids
- Opioids reduce feed intake.

**NSAID therapy in perioperative patients**
- Perioperative NSAID for inflammatory pain
- Multi-Modal or Balanced Analgesia
- Patient Selection
- Changing paradigm for NSAIDs

**Chronic non-steroidal anti-inflammatory drug therapy in cats**
- Feline ignored for too long
- Incidence of DJD is high in older cats
- Behavioral and Post Mortem evidence is clear
- Extra-Label Use in United States
- Use with Caution in DJD / Chronic use
- Dose to Lean Body Weight
- Regular laboratory testing for chronic therapy
- Regular Consultation / Adjust Therapy Plan

**Robenacoxib (Onsior)**
- Tablets approved (US) three day course of periop therapy
  - Rapid onset, short plasma T1/2, pKa 4, Freed from albumin at areas with low pH - sites of injury or inflammation
- Limited toxicity
- Dose 1 mg/kg (1-2.4 mg/kg) up to 3 days approval in US
  - avoid combinations with other NSAIDS, avoid with renal disease,
  - injectable form USFDA approved and available in 2015
- Paradigm shift in pre-op use: Oral! and NSAID!
- Highly palatable (Compliance improved)

**Value of sedation - A “one medicine: concept”**
- Newer generation agents include dexametomidine, the dextrorotary S-enantiomer of medetomidine
  - Detomidine - horses
  - Romifidine – dogs
  - Dexmedetomidine – humans, dogs, cats, etc.
- DOSE DEXDOMITOR BY BODY SURFACE AREA IN DOGS. DOSE BY WEIGHT IN CATS.
- Benefits of relative bradycardia are being recognized.
- Avoid anticholinergics - But if Atropine is Used: Administer At Least 10 Minutes Before Dexdomitor
- Expanding (extra-label) roles for Alpha-2 Agonists:
- The complex and dose-dependent effects of -2 AR agonists supported caution in clinical application
- Expanded application – strongly influenced by the accumulation of additional evidence and experience
- Patient selection - always the key to success in clinical application
- Medetomidine improves cardiac function in a subset of cats with hypertrophic cardiomyopathy
- In HCM patients – Complete elimination of dynamic LVOT obstruction and systolic anterior motion of mitral valve after medetomidine
- Peak LVOT velocity decreased
- Return to normal flow
- Loss of dagger-shaped concavity
- Sedation to prevent or treat post-operative delirium or agitation
Out-patient anesthesia techniques in human and veterinary anesthesia have improved the quality of care and are usually credited with improved patient outcomes as well as reduced medical costs.

As rapid recovery from anesthetic agents has become possible, sedation to prevent or treat post-operative delirium or agitation has become increasingly important.

Sedation for the transition from anesthesia to recovery

Lessons from equine anesthesia strongly support the role of -2 AR agonists (xylazine, etc.) to improve the transition from inhalant anesthesia to standing recovery.

Lower doses of dexmedetomidine, 0.5 to 1.0 mcg/kg, have been similarly useful to reduce agitation and delirium in dogs and in humans recovering from general anesthesia.

Suggested reading and references

AAHA/AAFP Pain Management Guidelines for Dogs and Cats
ISFM and AAFP Consensus Guidelines: Long-Term use of NSAIDs in Cats Journal of Feline Medicine and Surgery July 2010 vol. 12 no. 7 521-538. doi: 10.1016/j.jfms.2010.05.004
Simbadol (buprenorphine injection) Freedom of Information Summary, NADA 141-434; July 18, 2014
Approaches to modifying fear and anxiety
While preventive, proactive measures are essential to prevent fear and anxiety and turning the veterinary experience into a “happy one”, far too many pets will arrive fearful, anxious or painful or become fearful, anxious or stressed at some point during the visit or procedures. From the history, the reason for the visit, what the owner is communicating verbally and emotionally and what the pet is communicating, if there is any indication that the pet is or will become fearful or aggressive the visit should cease to reassess and rethink how best to proceed. Depending on the pet’s behavior and health, the reason for the visit, and the owners, the options are a) to find an alternative way to successfully and positively succeed b) to put off the visit or the procedure in favor of a strategy that will best manage or treat the problem at a future visit with modifications to management, desensitization and counterconditioning, behavior products and pre-visit drugs or c) to use intramuscular sedation.

a) Look, listen and stop to revise and proceed; Management and low stress handling
Consider other options for approach, handling and restraint that might allow the visit to continue without further fear or if the pet or owner is too aroused and stressed, what modifications might be made if the visit is to be rescheduled.
If the pet is sufficiently settled, an alteration in approach or handling may be sufficient i.e. calm, and slow, avoiding threatening stimuli, minimizing restraint and positive distractions. Determine if the odors, sounds, visual stimuli or surface are fear evoking and how they might be modified in order to proceed. Approaching slowly, avoiding direct eye contact, reaching, or sudden movements, minimizing or avoiding restraint, distracting with motivating food or treats and the use of appropriate handling aids such as a towel or blanket may be sufficient. Determine where and on what surface the pet would be most calm: on the exam table, on the floor, or sitting on your lap – on a blanket, towel or rubber mat. Consider pain control or sedation for procedures that might be uncomfortable or fear evoking.

For cats avoid reaching into the carrier or grasping by the scruff. Instead remove the top of the cage and lift the cat out, with the aid of a towel if necessary or examine the cat in the bottom half of the carrier, perhaps covered or partially wrapped in a towel for comfort, security and safety. For small dogs and cats a towel or blanket can be used to wrap or swaddle the pet. Covering the head or eyes (e.g. with a blanket or thunderscap) may further reduce the threat. The use of a head collar or muzzle for dogs may allow the procedures to be completed safely with a minimum of fear and anxiety provided the pet is already adapted to the use of these products or the veterinary team and owner have sufficient time and patience to calmly and positively apply.

Other behavior management products that might help in achieving calm and successful outcome are pheromones sprayed onto a blanket or towel for examination and procedures and the thundershirt or anxiety wrap. The application of pressure clips to the scruff of the cat’s neck, may cause behavioral inhibition in some cats, where the cat reaches a calm, semi-immobile trance like state. The procedure known as pinch induced behavioral inhibition or “clipnosis” may produce stress free restraint in cats.1 (www.clipnosis.com) Not all cats are responsive including those that are stressed or fractious when handling the back of their necks. In addition, there is ongoing review to determine whether clipnosis may be causing inhibition without decreasing fear. Therefore, the pet’s response should be carefully observed for signs of stress and distress, should these products be used.

b) Desensitization and counterconditioning – making veterinary visits positive
If the procedures are not immediately necessary, the visit should be postponed, and strategies implemented to make further visits fear free including desensitization and counterconditioning and how the pet might be safely and positive managed. The pet will need to be exposed to each component of the veterinary visit to make positive associations using high valued rewards to the carrier or restraint device (seat belt, head halter), car ride, reception, exam room, and procedures and multiple happy visits for exploration, play and treats. The pre-training program can include mat, touch and chin down training, counterconditioning to touch, handling, lifting, instruments, eye and ear procedures and mock injections which can then be gradually implemented with greater distractions at home before progressing to the veterinary clinic. Training should start with a calm pet in a comfortable environment, with minimal distractions and highest motivating rewards (treats, play). Clicker training can be most effective for rewarding and shaping and adapting the pet to a head halter can provide additional time and head and muzzle control to reorient, and maintain focus and safety.

Effective implementation, requires a dedicated and committed owner, good guidance and oversight (and/or resource material) by technicians or trainers and time to achieve effect, beginning in the home and then with multiple fear free visits to the veterinary clinic, building gradually on success. However, this may not be a practical solution for some owners, some pets and some procedures. For owners that are willing and committed to to minimize fear, anxiety and stress and improve the veterinary experience, the needs of the client and pet can best be addressed by members of the veterinary team who are trained in reinforcement based training, behavior modification and clicker training. While having a veterinary behavioral technician in each practice would be the ideal, staff and trainers can be educated to learn the skills necessary to offer this service to your clients. For examples of what can be done and what pets can achieve see the videos by veterinary technician specialist in behavior and karen pryor academy certified trainer (KPA-CTP),
Alicia Howell, at http://www.hillviewvets.com and Laura Torelli (KPA-CTP) and her program “Ready Set for groomer and vet” at abtconcepts.com

Another option is to administer anxiolytic medication in advance of the visit and to condition the pet to behavior management products that might be required including a carrier, head halter or muzzle.

c) Sedation

If a delay is impractical or the owners are unable to implement effective behavior strategies, the most prudent option for all is the use of intramuscular sedation to proceed safely with minimal further stress for the pet. Towel and blanket techniques, a head cover, soft sided carrier or a leash and halter or muzzle for dogs, should provide a means for safely injecting with a minimum of stress. Other options for management tools that might be considered if used humanely and knowledgeably include an E-collar, safety gloves, a capture net or E-Z Nabber for cats wild child squeeze chute, a squeeze cage for dogs or pulling the leash through a cage or door for restraint. An anesthetic induction chamber for cats or mask induction for dogs can be effective but anesthetic levels are more difficult to gauge and environmental exposure of anesthetic gas to personnel is an issue.

Pre-visit medications

For mild fear and anxiety in dogs, the use of trazodone, benzodiazepines (such as alprazolam, diazepam or lorazepam) or gabapentin might be effective prior to the visit. Clonidine might also be considered. In cats, gabapentin, trazodone and benzodiazepines such as lorazepam or alprazolam might be effective. When using gabapentin an additional dose the night before the visit might also be considered. When additional sedation is needed phenobarbital or acepromazine might be combined with the benzodiazepine, trazodone or gabapentin. A home trial in advance of the visit is recommended and combinations may be necessary. Natural products such as pheromones, alpha-casozepine, l-theanine and a magnolia/phellondendron herbal combination might also be used if started sufficiently in advance.

Sedative effects have also been demonstrated in cats with oral transmucosal medications including buprenorphine (.03 mg/kg) plus dexmedetomidine (.02 mg). Also in dogs, transmucosal dexmedetomidine might be used at 0.01-0.04 mg/kg. As the route of administration is transmucosal, slow administration into the buccal pouch mixed with a viscous syrup such as honey or maple syrup may improve absorption. Pets that require transmucosal medication should be counterconditioned in advance by administering flavored syrups or canned food by syringe into the buccal pouch. Most recently an oromucosal gel has received FDA approval for noise phobias, which offers a prepared licensed product for at home administration. Drugs should be administered approximately an hour in advance of the visit. For dogs another option is telazol (teletamine / zolazepam) at 9.6 mg/kg plus 1-2 mg/kg acepromazine orally with ½ given transmucosally before the car ride and ½ given transmucosally in the parking lot.

While these medications might provide sufficient calming or sedation to proceed with procedures when the pet is admitted into the veterinary hospital, they can also serve to facilitate the use of intramuscular sedation where needed. Therefore consider what medications the pet has already received when selecting and dosing intramuscular drugs.

Drug selection and use for fearful and anxious pets

When using injectable sedation, consider that the greater the level of fear, anxiety or arousal at the time of administration, the sedative response may be more variable and potentially less effective. Therefore prompt and calm injections when the pet is minimally stressed are safest and most effective.

Optimal and balanced sedation can be achieved with intramuscular injections of low dose dexmedetomidine, butorphanol (or an alternate narcotic), and midazolam (anxiolytic, muscle relaxant and amnesic effects). In more fractious patients the dexmedetomidine can be increased or ketamine might be added. Telazol at 5 mg/kg is another option for fractious dogs with a quick onset but longer duration of action. As ketamine and telazol are dissociative anesthetics, patients given these drugs require anesthesia monitoring. In addition duration of action may be prolonged, the drugs are not reversible and as ketamine causes pain on administration, it is best reserved for add-on therapy if the initial combinations are not sufficiently effective. In place of butorphanol, buprenorphine might provide more analgesia but less sedation and mu agonists such as morphine and hydromorphone offer greater pain control, greater sedation and are reversible. Opioids often cause emesis.

Aflaxalone is an anesthetic that can be administered intramuscularly for sedation, particularly in cats. It has less cardiovascular effects than the dissociatives with longer duration and no reversal agent. Because of its volume it is not practical for larger dogs and should be combined with other agents at a dose of .5-1 mg/kg such as with an opioid and midazolam for a smoother recovery.

Dexmedetomidine is an alpha-2 agonist, which provides fairly rapid analgesia and sedation (e.g. 20 minutes) and can be reversed with an intramuscular injection of atipamazole if a more rapid recovery is indicated. It should be avoided in pets with cardiovascular compromise as it causes vasoconstriction and hypertension leading to increased cardiac work. Level of sedation may vary between individuals and pets that appear sedated may be suddenly responsive to stimuli including pain. Therefore they are most commonly used in combination as described above.

Acepromazine (.01-0.05 mg/kg) might be substituted into the combination in lieu of dexmedetomidine. Acepromazine has a longer duration of action, may provide less reliable and less profound sedation, has no anxiolytic or analgesic effect, and is not

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reversible. Reversal agents for midazolam (flumazenil at .001-.01 mg/kg iv) and for oxymorphone, morphine and hydromorphone (naloxone at .01 mg/kg) or partial reversal with butorphanol are also available if safety is a concern and faster recovery is required.

### Doses for oral pre-medication

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trazodone</td>
<td>4-12 mg/kg</td>
<td>50-100 mg / cat (for travel)</td>
</tr>
<tr>
<td>Clonidine</td>
<td>0.01-.05 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Gabapentin</td>
<td>20 mg/kg</td>
<td>20 mg/kg (100 mg/cat)</td>
</tr>
<tr>
<td>Alprazolam</td>
<td>0.02-0.1 mg/kg</td>
<td>.125 mg - .25 mg per cat</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.5-2.2 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>.05-0.5 mg/kg</td>
<td>.05-.25 mg/kg</td>
</tr>
<tr>
<td>Acepromazine</td>
<td>.5-2.2 mg/kg (combine with anxiolytic)</td>
<td>0.5-2.2 mg/kg (with anxiolytic)</td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>5-10 mg/kg (combined with anxiolytic)</td>
<td>5 mg/kg (with anxiolytic)</td>
</tr>
</tbody>
</table>

### Doses for intramuscular sedation

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butorphanol</td>
<td>0.2 -0.4 mg/kg</td>
<td>0.2 - 0.4 mg/kg</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>0.003-.01 mg/kg</td>
<td>0.005-.01 mg/kg</td>
</tr>
<tr>
<td>Ketamine (if needed)</td>
<td>3 mg/kg</td>
<td>2 to 5 mg/kg</td>
</tr>
<tr>
<td>Midazolam</td>
<td>.05-0.2 mg/kg</td>
<td>.05-.2 mg/kg</td>
</tr>
</tbody>
</table>

1 Can substitute buprenorphine at 0.02-0.03 mg/kg, morphine at 0.2-1 mg/kg, or hydromorphone at .05-.2 mg/kg or oxymorphone .1 mg/kg for superior pain management and reversible

2 Increase to .02 mg/kg in dogs and .03 mg/kg in cats if greater sedation required

3 May provide anxiolytic, muscle relaxation and amnesic effect but may cause paradoxical excitation

**Dog:** Geriatric or ill: Butorphanol .2 -.4 mg/kg + midazolam .2 mg/kg

**Cat:** Geriatric or ill: Butorphanol .2 -.4 mg/kg + midazolam .2 mg/kg

### Reading and resources

The use of pre-visit sedation and natural products as well as in clinic sedation protocols are covered in the Fear Free on line course and certification modules. For details visit fearfreepets.com

- American Association of Feline Practitioners – www.catvets.com
- See guidelines for Cat Friendly Practice, Feline Friendly Handling, Feline Friendly Nursing Guideline
- Catalyst Council – catalystcouncil.org – Cat Resources and videos
- Fear Free Initiative – dvm360.com/fearfree
- International Cat Care – www.icatcare.org – Cat Friendly Practice – Resources
- Veterinary Anesthesia and Analgesia Support Group – vasg.org
- For veterinary technicians: see Society of Veterinary Behavioral Technicians (svbt.org) and for details on behavioral certification see Academy of Veterinary Behavioral Technicians at avbt.net.
- For clicker training see clickertraining.com and for Karen Pryor certification (KPA-CPT) see karenpryoracademy.com clicker

### References

It’s not really surprising that the veterinary clinic would be a scary place. Unfamiliar and fear evoking sights, sounds, and odors; examination of the eyes, ears and perhaps even prostate; taking temperatures and giving injections; nail trimming and anal sac expression; and visits for illness and injury will all contribute to negative experiences and outcomes.

Making the veterinary clinic a happy place takes requires a proactive, positive, preventive approach to turn potentially unpleasant experiences into positive ones. In addition, the use of both pre-visit pharmaceuticals and in clinic sedation protocols can further help manage situations in which fear, anxiety, and pain cannot be prevented or avoided (see lecture notes on behavioral management).

Stress associated with veterinary visits has been demonstrated to increase Urine Cortisol Creatinine Ratios in dogs, increased blood pressure, temperature, panting and pulse in dogs, a significant increase in blood pressure, heart rate and respiratory rate and an increased temperature in cats, and increased glucose in cats in hospital visits compared to home visits.

Preventive strategies
Since the puppy’s sensitive period begins to wane by 12 weeks and the kitten’s by 7 to 9 weeks, the first few visits will shape how the pet views the veterinary clinic. Every effort should be made to insure a pet friendly experience, using treats, toys, and force free handling to build positive associations, while minimizing or preventing unpleasant experiences. Problems can be prevented by considering each step in the visit: crating, travel, procedures, the hospital, and the actions of veterinarians, staff, and owners.

Canine and feline communication
Learn to recognize body postures, facial expressions and vocalizations that indicate a relaxed state, a desire to positively interact as well as signs of fear and anxiety.5-11 Monitor closely since signs of fear can be subtle and change quickly. At the first signs of fear and anxiety, unless safety is an issue, back off from further interactions and determine the best way to proceed, or discontinue the procedures and schedule for a future date.

Why are pets aggressive in the veterinary hospital?
Pets that display aggression in the veterinary clinic are fearful and defensive. Even pets that freeze or flee, may become aggressive if approached, cornered or restrained. If the pet learns that aggression is successful at removing the threat, the behavior is negatively reinforced. Fearful responses by the owner, veterinarians or staff, cause the pet’s fear and aggression to further escalate. In addition, physical restraint or confrontation can lead to fear and aggression. Even if the pet can be subdued to allow completion of the procedure, it increases fear and the potential for aggression at future visits.

Minimizing fear from travel through procedures
a) Positive travel experience
Address management of the pet during travel including positively conditioning the pet to the carrier or restraint device in advance. A pheromone spray or wipes (Feliway, Adaptil) might increase comfort with crating and travel. Crates with top entry or a removable top are more practical for handling cats. Once adapted, begin to accustom the pet to the car gradually while pairing with high value rewards. A treat and train can also help to reward and countercondition remotely during car travel. Also see http://drsohpiayin.com/videos/entry/how-do-you-use-your-treattrain-calm-behavior-in-the-car and www.catvets.com/public/PDFs/ClientBrochures/Cat-to-Vet-HandoutPrint.pdf

(b) Pet friendly veterinary environment – facility, scheduling and flow of activity.
Consider separate cat and dog waiting areas, minimal waiting times, and separate times of day for cat appointments. One study found that dogs may be more settled if given time to adapt, and that the weigh scale was the most stressful procedure. Therefore positive associations with weighing, or weighing pets while in their carrier or a towel wrap, with perhaps a short wait to allow the dog to adapt (while offering rewards) may be advisable. Be prepared to direct the pet into an open exam room; however with some dogs social interactions with other calm, friendly, dogs may have a positive effect on the veterinary experience. Book fearful pets when the clinic is least likely to be busy, and when staff can properly prepare for the visit. Keep records of how the pet’s behavior on previous visits to use what works and avoid what doesn’t. While pets with a recent veterinary visit are more likely to be stressed, pets with previous positive veterinary experiences will be less fearful.

Sensory input will strongly influence the outcome of the veterinary visit. Tone of voice, environmental sounds, visual stimuli, odors and tactile stimuli can either aid in calming or increase anxiety. In addition, the emotional state of owners, staff and other pets can either help to calm or evoke fear. A soft, calm tone of voice can help to reduce fear while punishment and corrections will increase
fear. Every effort should be made to have the owner present for examination and procedures, to help calm the pet. Removing the pet from the owners may result in a pet that is more inhibited but emotionally more fearful. However, if the owner is emotionally anxious, fearful or confrontational this will add to the pet’s anxiety.

For auditory stimuli, a cover over the cat carrier, sound muting of exam rooms and playing soft calming music, or white noise or a water fountain can help minimize auditory cues. In fact classical music or species specific background music may also help to calm. A soft, pleasant tone of voice can also prove to be calming while loud and boisterous voices, telephones, clippers, and other novel auditory stimuli can evoke fear.

Dogs and cats can detect pheromones as well as odors that communicate alarm or distress. Odors should be removed by cleaning thoroughly between patients and the use of animal odor eliminators. Adaptil and Feliway may also help to calm in the air, on the surfaces or perhaps on clothes. A lavender or chamomile aroma may also be calming for some pets.

Avoid sudden movements, direct eye contact, or direct approaches. Visual barriers, a blanket over the pet’s cage or a covering over the eyes can minimize visual stimuli. Approaching from the side and avoiding any type of reaching or grabbing for the pet can reduce anxiety. Consider natural daylight and pastel colors rather than bright white which might appear fluorescent to pets.

A water fountain can help minimize auditory cues. In fact classical music or species specific background music may also help to improve the owner experience and encourage them to naturally move toward where the pet will need to go.

Also consider the surfaces to insure that they are comfortable, secure and perhaps warmed and a perching area for cats in the examination room. Cold surfaces, insecure footing and unsteady scales or table tops can negatively affect behavior.

Active engagement owners in working to make veterinary experiences happy, by accentuating what is positive and eliminating what is negative. While a neutral outcome is acceptable it does not achieve the positive experience on which to build for future visits, and can quickly spiral downward if a future outcome is negative. Both owners and staff members can influence behavior positively by their attitude, and by offering motivating highly motivating treats or play toys. Fasting the pet for a portion of the day, so that the pet arrives a little hungry can increase motivation for treats. In addition, begin offering treats (multiple small treats) or engaging the pet in play before beginning interactions to start with a positive baseline.

Where practical discuss with owners how to prepare the pet for veterinary visits by positively conditioning the pet to touch and body handling, lifting onto a table (if indicated), and even to the instrumentation used in the visit (lights, stethoscopes). Training to touch on cue (targeting), to settle on a portable mat or bed, and to rest the chin on cue can help to maintain focus and achieve positive outcomes throughout the visit. In fact, these can be valuable components of puppy classes and dog training.

(c) Pet friendly handling

Train staff on how to physically manage pets with low stress handling and a minimum of restraint to insure a calm, positive outcome. Offer high value food treats or play toys to encourage and maintain a positive experience. Food tubes and wooden spoons can be used to offer viscous foods such as cheese spread or peanut butter (or a can of Kong stuffing or lick sticks) to maintain enjoyment of the experience. Proceed slowly and monitor the pet’s body language at all times to determine optimum handling and preferred location, when to proceed and when to stop. For cats, while greeting the client and taking the history, open the carrier and allow the cat to come out on its own onto the floor, a chair or exam table. Approach slowly and avoid reaching, staring, or sudden movements. If the cat will not come out voluntarily try leaving treats in front of the carrier or enticing with treats or a toy. Avoid reaching in or grasping by the scruff. Instead remove the top of the cage and lift the cat out, with the aid of a towel if necessary. Some cats will be more comfortable remaining in the bottom of their carrier. Feliway may facilitate handling.

When performing procedures maintain the least amount of restraint necessary to achieve success, and allow the pet to select a position in which it would prefer to stand, sit or lie down. Use handling techniques that keep the pet safely and securely controlled. For cats, minimize hands on control by keeping a hand or towel in front of the cat to keep it from moving forward. Some pets will be more secure in the owner’s presence while others will be less fearful if the pet is removed from the room, or the pet is kept in the room with the owner asked to leave. For small dogs and cats a towel or blanket can be used to wrap or swaddle the pet or cover its head to more secure in the owner’s presence while others will be less fearful if the pet is removed from the room, or the pet is kept in the room

(d) Hospitalization

When pets are hospitalized consider the type of confinement that best suits the pet. Bedding for warmth and comfort, classical music, pheromone diffusers or sprays and aromatherapy may help to calm. Consider whether drugs are indicated to reduce anxiety, manage pain or sedate prior to the visit or prior to caging. For dogs depending on their sociability, the reason for hospitalization, the level of fear and noise levels, the best option might be to be housed in a ward with other sociable dogs. On the other hand, dogs should be isolated or a blanket or towel placed over the door of the cage, if they are stressed by the sights or sounds of other dogs. Food can be placed in manipulation toys or stuffed into chew toys to increase enjoyment and enrichment. An Adaptil diffuser in the dog wards may help to calm. Cats that are hospitalized may do best if kept in their own carrier sprayed with Feliway inside a hospital cage.
Cages should have a perching area or level separate from the litter, a place to hide, bedding, litter and good or toys. Cat wards should be separate from dog wards to minimize sounds and odors. Classical music in the wards may further help to calm. Fear free management of each component of the veterinary visit is covered in detail in the fear free on line course and certification modules. For details visit fearfreepets.com

Reading and resources
American Association of Feline Practitioners – www.catvets.com
Cat Friendly Practice, Feline Friendly Handling, Feline Friendly Nursing Guideline
Catalyst Council – catalystcouncil.org – Cat Resources and videos
Fear Free™ Initiative – dvm360.com/fearfree
International Cat Care – www.icatcare.org – Cat Friendly Practice – Resources

Body language resources
Learn to speak dog and teach your kids: doggonesafe.com
Zoom Room Guide to Body Language https://www.youtube.com/watch?v=00_9PltXHI

References
8. Learn to speak dog and teach your kids: doggonesafe.com
10. Zoom Room Guide to Body Language https://www.youtube.com/watch?v=00_9PltXHI
11. How to read cat body language – Cat Channel http://www.youtube.com/watch?v=rilHuk9Xr1E, accessed February 2014
Cervical pain is a common presentation to our neurology service. The term “hyperesthesia” is more appropriate as pain is a symptom and therefore an interpretation vs. the defined noxious response to a non-noxious stimulus. Cervical hyperesthesia is without other neurologic deficits are frequently encountered mostly because of the large vertebral canal: spinal cord ratio when compared to the thoracolumbar spine. Causes of cervical hyperesthesia run the gamut of the VITAMIN D list, stressing the importance of a thorough history and physical examination. Signalment, Acuteness of clinical signs, progression of disease and presence of spinal hyperesthesia will be essential in prioritizing the differential list. For example, vascular diseases will be acute in non-chondrodystrophic breeds, non-painful after 24 hours and non-progressive as opposed to atlantoaxial luxation, which will be subacute, progressive and painful in young toy breed dogs. The following is a list of potential causes of spinal hyperesthesia.

**Intervertebral disc disease (IVDD)**

IVDD presents as an acute-chronic, progressive disease seen in mostly chondrodystrophic breeds. Cervical IVDD typically affects dogs >greater than three years of age. Survey radiographs have a reported overall accuracy of only 35% for the diagnosis of the site of disc extrusion in dogs with cervical IVDD.\(^1\) Conservative therapy should consist of appropriate analgesia and strict cage confinement. The success rate associated with conservative management of dogs with neck pain only from cervical IVDD is 50–90%, but nearly half of conservatively managed dogs will have a recurrence of clinical signs. The authors experience is that these conclusions are high and cervical hyperesthesia cases of IVV responded quicker and more completely when treated surgically. Surgical correction with ventral slot is the procedure of choice in most cases with cervical hemilaminectomy and alternative in lateralized discs inaccessible by the slot due to venous sinus borders.

**Atlantoaxial subluxation (AAS)**

Congenital AAS typically causes clinical signs of C1–C5 myelopathy in immature small and toy breeds of dogs, although it can occur in any age or breed. It should be a primary differential consideration in any young small breed dog with neck pain or cervical myelopathic signs. Developmental abnormalities of the dens (hypoplasia or aplasia) or malformations of or trauma to the supporting ligaments are responsible for the development of clinical signs, which results in instability of the atlantoaxial joint and contusion and/or compression of the spinal cord. During the examination, extreme care should be exercised when manipulating the neck of AAS patients as even normal motion, especially flexion, can exacerbate clinical signs.

Survey radiographs will be diagnostic for AAS in the majority of cases, but accurate positioning is crucial. On the lateral projection, an increase in the space between the dorsal arch of C1 and the spinous process of C2 (> 4 mm) is diagnostic for AAS. Congenital absence or hypoplasia of the dens can be seen on the ventrodorsal or rostrocaudal open mouth views. Occasionally a flexed lateral view may be required to demonstrate instability.

AAS may be managed conservatively (cage rest and external coaptation) or surgically. Conservative management can be associated with very good outcomes in some cases, but is only chosen because of financial limitations or if the patient is too young for implant fixation. Often, the finances of conservative management approach those of surgical therapy once the multiple bandage changes have been done. The approach and choice of surgical fixation is primarily based on surgeon personal preference, but ventral approaches using transarticular fixation are the most common, and are associated with lower complication rates compared to dorsal techniques.

**Cervical spondylomyelopathy (wobblers disease)**

In contrast to cervical IVDD, this is predominantly a condition of large and giant breeds of dogs. Wobblers is a generic term used to describe a syndrome of compressive cervical myelopathy that may result from one or more of the following pathological abnormalities: disc protrusion, congenital vertebral canal malformation or acquired osseous stenosis, vertebral instability / subluxation, ligamentous hypertrophy, and joint capsule proliferation / synovial cyst formation. Also in contrast to cervical IVDD, giant breeds of dogs (Great Dane, Mastiff) with congenital vertebral malformations or joint capsule proliferations may show clinical signs at a young age (< 1 year), although many dogs will not develop signs until middle age. The clinical signs of CSM are usually associated with gait dysfunction, with our without signs of neck pain. Signs of neck pain may not be readily apparent in these breeds, but they may carry their heads low, or be reluctant to bend down to eat. Definitive diagnosis of CSM requires advanced imaging, but survey radiographs are useful to rule out other differential diagnoses, and may reveal changes suggestive of CSM, such as proliferative articular facets and vertebral subluxation.
**Diskospondylitis**
This is an inflammatory condition involving the intervertebral disc and end-plates. The infection usually results from hematogenous spread of infection from remote sites in the body, which commonly include the urogenital tract and skin. The most commonly implicated bacterial pathogens include *Staphylococcus intermedius*, *E. coli*, *Strep* spp, and *Brucella canis*. Fungal pathogens, particularly aspergilliosis, may also cause diskospondylitis. Large breed dogs are most often affected, and the disease most often manifests at the highly mobile regions of the vertebral column (cervicothoracic, thoracolumbar, and lumbosacral junctions). The initial clinical sign is almost always spinal hyperesthesia, which may be associated with systemic signs of illness such as fever, anorexia. Neurologic deficits may develop secondary to epidural or meningeal spread of infection, disc extrusion, or pathologic vertebral fracture.

Survey radiographs are often diagnostic for diskospondylitis, although lesions may be radiographically occult within the first few weeks of the onset of clinical signs. In animal with radiographic evidence of diskospondylitis, a urinalysis and urine culture and *Brucella* screening test should be performed. Blood cultures have been reportedly positive in up to 66% of diskospondylitis cases. Initial treatment is systemic antibiotic therapy, ideally based on culture and sensitivity results, for 6 weeks. Dogs with neurological deficits associated with diskospondylitis may require advanced imaging and surgical therapy.

**Syrinxomyelia**
Syrinxomyelia (SM) is characterized by the development of fluid-filled cavities within the spinal cord parenchyma, and can be caused by numerous congenital (caudal occipital malformation syndrome [COMS]) or acquired (trauma, meningitis, neoplasia) causes of disturbed CSF flow dynamics. COMS is the most common condition associated with the development of SM in dogs. The prevalence of COMS-SM is high in several small and toy breeds of dogs, but most notably the Cavalier King Charles Spaniel. The clinical signs associated with COMS-SM can be quite variable and reflect dysfunction of the vestibular system, cerebellum, cervical spinal cord, or can suggest a multifocal lesion. Dogs with COMS-SM also commonly manifest with signs of maladaptive neuropathic pain, which may include allodynia, such as intolerance to petting or grooming the head and neck, or dysesthesia, which often appears as "phantom" scratching the head, neck, or shoulders without making contact with the skin.

These pain behaviors are theorized to arise from damage to the spinal cord dorsal horn or ascending nociceptive pathways from SM. Clinical signs are more common in dogs with SM associated with COMS, and there appears to be some correlation between the severity of signs and syrinx size. COMS with or without SM may also be an asymptomatic, incidental finding in at risk breeds. MRI is the preferred modality for the definitive diagnosis of COMS-SM.

The primary goals of multimodal medical therapy are to slow the progression of disease by decreasing CSF production and palliate neuropathic pain. Since COMS-SM is a mechanically obstructive problem, corrective surgery, which typically involves a foramen magnum decompression and variably extensive cervical dorsal laminectomy, offers a better chance for long-term control of clinical signs.

**Meningitis / meningomyelitis**
Although there are numerous infectious diseases that can meningomyelitis, the most common etiologies of inflammatory spinal cord disease in dogs that cause cervical pain appear to be variants of immune-mediated diseases such as steroid-responsive meningitis arteritis and granulomatous meningoencephalomyelitis (GME). The index of suspicion for meningitis should be high when a dog presents with cervical pain and fever. Survey radiographs are usually unremarkable in these cases, and the diagnosis requires MRI of the cervical area, CSF analysis, and serology.

**Extradural neoplasia**
Spinal neoplasia is typically a disease of older dogs, with extradural neoplasms accounting for half of all spinal tumors. Primary bone tumors arising from the vertebral bodies, such as osteosarcoma and fibrosarcoma, are the most common, although numerous other histologic subtypes of primary tumors as well as metastatic tumors have been reported. Most dogs with extradural vertebral body tumors will have cervical pain, and it can often be intense and unrelenting. Neurologic deficits, when present, can be acute onset and catastrophic when associated with pathologic fracture. Survey radiographs are valuable in that they will often demonstrate characteristic osteolytic lesions involving the vertebral. All dogs with lytic lesions involving the vertebrae should be evaluated systemically for tumor staging purposes. The treatment of choice for most primary tumors is cytoreductive surgery with consideration given to adjunctive or primary radiotherapy. Although obtaining wide margins is not possible in most vertebral tumors, surgical debulking can often be done in a fashion that allows for temporary improvement or resolution of clinical signs.
Head trauma or traumatic brain injury (TBI) is a common emergent presentation in both humans and animals. In human medicine the recommendations are largely based on the guidelines from the Brain Trauma Foundation that provide meta-analysis of the large body of current literature for diagnosis and treatment of TBI. In veterinary medicine, the number of studies evaluating TBI is scant. Many assessments of human TBI patients involve cognitive testing and do not translate to the dog or cat. Most of the tests that evaluate basal neurologic function demonstrate the severity of the condition at a point beyond which intervention would prove successful. Rapid and aggressive therapy is therefore essential. Despite the lack of recent advance in TBI cases, most veterinary patients can have a reasonable prognosis with supportive systemic treatment.

Intracranial physiology
The Monro-Kellie hypothesis describes the intracranial space as a rigid volume with three main compartments: The brain, blood and CSF. As the total volume is fixed, enlargement of one compartment or addition of a new compartment (ie.hematoma or tumor) necessitates the decreasing volume of another.

The brain requires a fixed perfusion for the neuronal components to function adequately. The total cerebral perfusion pressure (CPP) is a function of the mean arterial blood pressure (the pressure of blood entering the intracranial space) and the intracranial pressure (ICP), which opposes this inflow of blood.

\[
CPP = MABP - ICP
\]

In the normal patient, this equation is regulated by a function known as auto regulation. Two types of autoregulation are mechanical and chemical. In mechanical autoregulation, baroreceptors of the intracranial vessels detect an increase pressure and consequently constrict. Chemical auto-regulation controls vessel size by detecting proton (CO2) levels. Vasoconstriction occurs when the patient becomes hypocapnic. This effect decreases intracranial blood volume and consequently increases perfusion, but may have negative consequences on tissue vascular exchange.

When intracranial pressure is low (<25mmHG), there is adequate compliance in the system where these autoregulatory changes can have volumes fluctuate without affecting pressure significantly. However, should ICP raise up 25mmHG (such as in the TBI patient), these autoregulatory changes are ineffectual, because the system has reached the limits of its compliance and small volume changes lead to major pressure changes.

The other consequence of an elevated intracranial pressure is the secondary effects that they exert on the brain compartment, eventually leading to brain shifting or herniation. These shifts lead to neurologic dysfunction and death soon thereafter.

Assessment of the patient
The Modified Glasgow Coma scale (MCGS) was created to emulate the validated human correlate that grades TBI as mild, moderate or severe. The test evaluates the animal’s mentation, motor function and cranial nerve reflexes and assigns a numeric value that correlates with prognosis. Clinical signs of elevating intracranial pressure include postural changes, pupillary size and the Cushing’s reflex. Miosis often represents forebrain dysfunction and mydriasis indicates CNIII dysfunction following brain herniation. The Cushing’s reflex also demonstrates a systemic effort to correct acute ICP changes. With elevated ICP, the body will increase MABP to maintain CPP. Peripheral baroreceptors detect this pressure change and accordingly decrease the heart rate. It is important to recognize that once these clinical signs emerge, the patients are considered severely affected.

Imaging
Cross sectional imaging is being used as a prognostic indicator with more frequency in human medicine. In veterinary medicine, its prognostic value is somewhat unknown, but it can lead to changes in therapeutic direction, specifically the need for surgery.

<table>
<thead>
<tr>
<th></th>
<th>CT</th>
<th>MR</th>
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<tr>
<td>PRO</td>
<td>Speed</td>
<td><em>Soft tissue resolution</em></td>
</tr>
<tr>
<td></td>
<td>Availability/affordability</td>
<td>Identify herniation</td>
</tr>
<tr>
<td></td>
<td>Less susceptible to motion artifact</td>
<td>Identifies hemorrhage</td>
</tr>
<tr>
<td></td>
<td>Less susceptible to metal artifacts</td>
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</tr>
<tr>
<td></td>
<td>Resolution of bone and blood</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3D reconstructive ability</td>
<td></td>
</tr>
<tr>
<td>CON</td>
<td>Ionizing radiation</td>
<td>Time consuming</td>
</tr>
<tr>
<td></td>
<td>Poor Soft tissue resolution</td>
<td>Expensive</td>
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</table>
Treatment
Therapy is geared at maintaining CPP by decreasing ICP and maintaining CPP. More advanced therapy targets secondary brain injury, the downstream effects following the initial insult that can cause further injury (ie. Free radical formation, excitatory neurotransmitters, and inflammatory metabolites).

To this point, two therapies have been definitively proven to improve patient outcome in the human field: fluid therapy and osmotic diuretics.

Intravenous fluids
Hypertonic saline administration (4–5 ml/kg over 3–5 minutes) draws fluid from the interstitial and intracellular spaces into the intravascular space which improves blood pressure and cerebral blood pressure and flow, with a subsequent decrease in ICP. Often colloid therapy is used in conjunction to maintain intravascular volume once the hypertonic solution has extravasated. Balanced isotonic solution administration to maintain normovolemia follows this treatment to maintains MABP and CPP.

Osmotic diuretics
Osmotic diuretics such as mannitol are very useful in the treatment of intracranial hypertension. Mannitol has an immediate plasma-expanding effect that reduces blood viscosity, and increases cerebral blood flow and oxygen delivery. This results in vasoconstriction within a few minutes causing an almost immediate decrease in ICP. The better known osmotic effect of mannitol reverses the blood-brain osmotic gradient, thereby reducing extracellular fluid volume in both normal and damaged brain. Mannitol should be administered as a bolus over a 15-minute period, rather than as an infusion, in order to obtain the plasma-expanding effect; its effect on decreasing brain oedema takes approximately 15–30 minutes to establish and lasts between 2 and 5 hours. Repeated administration of mannitol can cause an accompanying diuresis, which may result in volume contraction, intracranial dehydration and the concomitant risk of hypotension and ischaemia. There has been no clinical evidence to prove the theory that mannitol is contraindicated in the presence of intracranial haemorrhage. There is contradictory evidence that the combination of mannitol with furosemide (0.7 mg/kg) may lower ICP in a synergistic fashion, especially if furosemide is given first.

Oxygenation and ventilation
Hyperoxygenation is recommended for most acutely brain-injured animals. Partial pressure of oxygen in the arterial blood (PaO₂) should be maintained as close to normal as possible (at or above 80 mm Hg). Supplemental oxygen should be administered initially via face-mask, as oxygen cages are usually ineffective since constant monitoring of the patient does not allow for a closed system. As soon as possible, nasal oxygen catheters or transtracheal oxygen catheters should be used to supply a 40% inspired oxygen concentration with flow rates of 100 ml/ kg/min and 50 ml/kg/min respectively. If the patient is in a coma, immediate intubation and ventilation may be needed if indicated by blood gas evaluations. A tracheostomy tube may be warranted in some patients for assisted ventilation. Hyperventilation has traditionally been known as a means of lowering abnormally high ICP through a hypocapnic cerebral vasoconstrictive effect. However, hyperventilation is a double-edged sword. Besides reducing the ICP, it induces potentially detrimental reductions in the cerebral circulations if the pCO₂ level is less than 30–35 mm Hg. The major difficulty with hyperventilation is our present inability to monitor the presence and effects of ischaemia on the brain. It is important that animals do not hypoventilate, and such animals should be ventilated to maintain a PaCO₂ of 30–40 mm Hg. Aggressive hyperventilation can be used for short periods in deteriorating or critical animals.

Hyperventilation, head elevation and hypothermia
Though its efficacy is unknown, head elevation between 15-30 degrees is recommended to promote venous drainage, as it is a benign treatment. Hyperventilation to reduce CO₂ levels and promote intracranial vasoconstriction has little evidence support. However, preventing hypoventilation is important and emphasizes the importance of providing the patients with a patent airway. Intubating the patient allows for a capnography and airway protection in the mentally debilitated patient. The BTF suggests maintaining normocapnea. Similarly, hypothermia was suggested but not adequately substantiated as a treatment with the goal of decreasing neuronal activity and subsequent excitatory neurotoxicity. While hypothermia is preferable to hyperthermia, maintaining normothermia is ideal. Other techniques to decrease neuronal activity, such as barbiturate coma are fraught with possible complications including decreasing MABP. This technique is not recommended unless advanced monitoring with electroencephalography and continuous mean arterial blood pressure are available.

Glucocorticoids
1. Do not lower ICP
2. Do not improve outcome in head-injured patients (human studies)
3. Not recommended in animals with brain trauma
4. Often associated with adverse effects
Surgery
Decompressive surgery for the sake of improving ICP elevations has poor evidence based support and should only be pursued in cases of progressive neurologic deterioration despite medical intervention. However, ongoing compressive lesions, such as fractures or hematomas, can be removed to restore normal anatomy.

Despite the limited effective therapies available to the practitioners many dogs severely debilitated with neurologic dysfunction following TBI have a good prognosis with supportive care. If animals
Function of the vestibular system
The vestibular apparatus is a sensory system essential for the maintaining posture and balance relative to gravity and movement. The system has its sensory receptors in the inner ear, its processing center in the brainstem and its output caudally through the spinal cord and rostrally towards the eyes. It receives regulatory input/feedback from the cerebellum. It is clinically divided into peripheral and central components, both because of the ability to separate neurolocalization based on clinical signs and because of the implications these localizations have on differential diagnosis.

Peripheral vestibular anatomy
The membranous labyrinth deep within the inner ear and the vestibular portion of the vestibulocochlear nerve make up the peripheral vestibular system. Within these labyrinths are cavities that contain endolymph fluid. The receptors (crista ampularis) detect the movement of this fluid and in turn detect angular acceleration. Angular acceleration is essentially rotational movement of the head from its resting state. Other receptors (the utricle and saccule; The maculae) have small rocks (otoliths) which give the receptors mass and make them capable of detecting the exerted force of gravity. This is known as linear acceleration. These mechanoreceptors give afferent information along with auditory inputs to the vestibular ganglion and then to the brainstem. The proximity of CN VIII to CNVII is highest within the middle ear and can have clinical consequences. The sympathetic innervation to the face also runs adjacent to these nerves.

Central vestibular anatomy
The four vestibular nuclei are in the most dorsal portion of the medulla, directly under the cerebellum. This proximity manifests clinically in that diseases causing herniation of the cerebellum through the foramen magnum can compress these cell bodies. There is a direct connection between these nuclei and the “vestibulocerebellum” made up of the floccular-nodular lobe. The primary outputs of the vestibular system are to the extraocular muscles of the eyes and caudally through the spinal cord. The medial longitudinal fasciculus sends outputs to CN III, IV and VI, maintaining the globe’s position in the orbit and the ability to maintain gaze in the face of head rotation. The outputs to the spinal cord are through the vestibulospinal system, essential an anti-gravity tract facilitating mostly extensor muscles to the neck, trunk and limbs.

Clinical signs of vestibular disease
Certain clinical signs are inherent to any form of vestibular disease and are in some cases pathognomonic for the neurolocalization. They include
1. Head tilt
2. Nystagmus (resting or positional)
3. Vestibular Ataxia
4. Ventrolateral strabismus
These clinical signs emerge because of imbalance of vestibular input. In the normal patient, the vestibular system is bilateral providing input that when processed together yields the precision system of balance. When one side is diseased and fails to provide normal input, the non-diseased side produces unilateral information and thus clinical signs often reflect the non-diseased side rather than the diseased side. Many of the clinical signs of vestibular disease would be absent in the animal affected bilaterally. The precedent for this is seen mostly in cats with bilateral otitis media/interna. They have no nystagmus, head tilt or ataxia. Clinical signs in these patients include snake like movements of the head and a lack of normal vestibular-ocular function best assessed by evaluating the physiologic nystagmus (vestibulo-ocular reflex).

Differentiating central vs. vestibular disease
As previously stated, differentiating these diseases are essential for differential diagnosis creation and prognostication. The differences are inherent to the adjacent structures that would be affected. The peripheral component is in close proximity to CNVII and the sympathetic nerve to the face. Thus facial paresis and Horner’s syndrome are commonly seen with this neurolocalization. Central disease is in proximity to the sensory proprioceptive systems, the arousal centers and other cranial nerves. Therefore, proprioceptive deficits, mental dullness and cranial nerve dysfunction (other than CNVII) are common with this localization. Although simple in theory, distinction of central vs. peripheral disease can be difficult in practice as the clinical signs are rarely clear and completely present. Should the vestibular cerebellum become diseased, these signs would point to a central vestibular
Localization, but often with conflicting sign sidedness. This is known as paradoxical vestibular disease. Postural reaction deficits are used to determine sidedness, as they are consistently ipsilateral to the lesion.

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>Peripheral</th>
<th>Central</th>
<th>Paradoxical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head tilt or circling</td>
<td>Toward lesion</td>
<td>Toward lesion</td>
<td>Away from lesion</td>
</tr>
<tr>
<td>Nystagmus</td>
<td>Horizontal or Rotary, Non-positional</td>
<td>Horizontal, rotary or vertical, +/- positional</td>
<td>Horizontal, rotary or vertical, +/- positional</td>
</tr>
<tr>
<td>Mentation</td>
<td>Alert</td>
<td>+/- Mentally inappropriate.</td>
<td>+/- Mentally inappropriate.</td>
</tr>
<tr>
<td>CP Deficits</td>
<td>No</td>
<td>Ipsilateral</td>
<td>Ipsilateral</td>
</tr>
<tr>
<td>CN deficits</td>
<td>+/- CN VII</td>
<td>+/- CNs V-XII</td>
<td>+/- CNs V-XII</td>
</tr>
<tr>
<td>Horner's syndrome</td>
<td>+/- Ipsilateral</td>
<td>No</td>
<td>No</td>
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**Differential diagnosis of vestibular disease**

Creation of a differential diagnosis list is best approached by using the VITAMIN D scheme. Common differentials for peripheral disease include infectious (otitis media/interna) and idiopathic, commonly seen in the geriatric patient. More common central vestibular differentials include neoplasia, infectious/inflammatory (meningoencephalitis), vascular, and toxic (metronidazole toxicosis).

Cross sectional imaging is a mainstay of diagnosing the cause of vestibular disease. MRI has supplanted CT as the diagnostic modality of choice. CT can adequately identify disease of the peripheral system and most specifically tympanic bulla changes. However, beam hardening artifact limits the modality in evaluating the caudal fossa (central vestibular system). MRI can image the entire system with superior contrast resolving ability. Other ancillary diagnostics may include myringotomy/culture and brain auditory evoked potentials to evaluated the auditory component of CN VIII. Treatment and prognosis are largely contingent on the etiology itself.
Neurolocalization is the ultimate goal of the neurologic examination, piecing the puzzle of reflexes and responses together to an anatomic segment. Once accomplished, the practitioner is able to assign a rank list of differential diagnosis for this neuroanatomic site based on the patient’s age, breed, clinical onset/progression and the presence of spinal hyperesthesia. Whereas this list can easily be found in any neurology text, the ability to interpret findings and combine them to fit a single lesion site requires practice.

A concept integral in localization is that of the Upper Motor Neuron (UMN) and Lower Motor Neuron (LMN). The Upper Motor Neurons are essentially the long tracts from the brain that instruct the lower motor neurons what to do. The LMN consists of the motor neuron cell body within the spinal cord, the nerve itself, the neuromuscular junction and the muscle itself. In cases of UMN disease, the lower motor neuron works without instruction, known as a loss of inhibition. It is classically hypertonic, with normal to increased reflexes and minimal atrophy. In cases of LMN disease, the upper motor neuron is irrelevant, because the LMN cannot respond to its instruction. Thus hypotonicity, decreased reflexes and severe neurogenic atrophy are noted.

The spinal cord can be divided into four areas. The LMN of the limbs are located in swellings of the spinal cord known as intumescences. When these intumescences are the sight of disease, LMN signs are notable. When the lesion blocks the long tracts between the limb the brain and the LMN, UMN signs emerge. Thus the spinal cord can be divided into the following segments.

<table>
<thead>
<tr>
<th>Lesion location</th>
<th>Thoracic limbs</th>
<th>Pelvic limbs</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1-C5</td>
<td>UMN</td>
<td>UMN</td>
</tr>
<tr>
<td>C6-T2</td>
<td>LMN</td>
<td>UMN</td>
</tr>
<tr>
<td>T3-L3</td>
<td>Normal</td>
<td>UMN</td>
</tr>
<tr>
<td>L4-S3</td>
<td>Normal</td>
<td>LMN</td>
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The neurologic examination is comprised of multiple parts for accurate localization.

Gait
Ataxia is defined as an uncoordinated gait and is frequently classified as “the drunken sailor walk”. It is often classified as
1. Proprioceptive/spinal-a.k.a The true drunken sailor walk, often coupled with a spastic/long strided gait suggesting UMN dysfunction. Patients are often thought of as overstepping or “floating”
2. Vestibular-Characterized by imbalance often manifest as “wall walking, coupled with a head tilt and nystagmus
3. Cerebellar lesions –Characterized by hyper/dysmetria (Goose stepping), truncal swaying, and intention tremors.

Paresis is weakness of the gait, reduced voluntary movement, whereas paralysis is complete loss of voluntary movement. Both paresis and paralysis (-plegia) can be used to describe the deficits in only one limb (monoparesis/plegia), in the pelvic limbs (paraparesis/plegia), in all four limbs (tetraparesis/plegia) or on one side of the body (hemiparesis/plegia).

Proprioception
The ability of a patient to identify the location of its limbs in space. A subjective but integral component to the examination, these tests confirm the presence of a neurological disorder and can detect subtle dysfunction, helping identify which limbs are affected. This includes paw position, hopping and placing responses.

Spinal reflexes
Reflexes are quite different than responses, such as proprioception, in that they do not require forebrain input. This is an important distinction

The easiest and most reliable are the patellar reflex in the pelvic limbs and the withdrawals in both the pelvic and the thoracic limbs. Don't forget to examine the tail and anus (perineal reflex). Reduced reflexes in a limb identify a LMN lesion in that limb, whilst normal or increased reflexes localize the lesion to the UMN

- Cutaneous trunci reflex (panniculus)--helps narrow down lesion localization in the thoracolumbar region. After pinching the skin, the sensory information enters the spinal cord approximately two vertebral spaces cranially, ascends the spinal cord to the level of C8-T1 where bilateral synapse occurs with the motor neurons of the lateral thoracic nerve, which then course through the brachial plexus and innervate the cutaneous trunci muscle, resulting in bilateral contraction of these muscles. Normally, this reflex is present from T2 to about L4-L5 and a cut-off in this region suggests a spinal cord lesion just cranial to the cut-off level. Loss of the cutaneous
trunci reflex can also be due to a brachial plexus lesion, in which case it will be completely absent on the side of the lesion and normal on the contralateral side.

**Pain sensation**
The spinal pathways that carry pain sensation are located deep in the spinal cord so only a severe lesion will impair pain perception (making this an important prognostic factor). For conscious perception of pain, manifested by vocalization and/or turning the head and trying to bite, the information needs to be recognized by the sensory nerve, travel up the entire spinal cord cranial to that area and be interpreted by the brain. It is important to differentiate a pain response from a local withdrawal reflex (which should be present if both the peripheral nerve and spinal cord segment of the stimulated peripheral nerve are intact), in which case the limb will be retracted but no signs of conscious awareness of the pain will be evident. Pain sensation is tested by applying heavy pressure with haemostats to the bones of the digits (don't forget to test different digits) or to the long bones of the limbs.

**Spinal palpation**
Looking for areas of hyperesthesia or deformities. Pressure is applied to the spinous and transverse processes of the vertebrae in all spinal segments. Manipulation of the cervical spine in all directions is performed.
Seizure Disorders are extremely common in dogs with an incidence estimated as high as one percent of the pet population. The cause of these seizures is generally classified as structural, metabolic or idiopathic/heritable. Idiopathic epilepsy is by far and away the most common cause, representing 30% of all epileptics. Despite large advances in anatomic imaging, genetic characterization and surgical care, the understanding of the cause of idiopathic epilepsy is still in its infancy. However, many novel anti-seizure medications are entering the clinical arena with potential use in patients unresponsive to traditional medications such as phenobarbital and bromide.

Managing seizure disorders presents a major challenge to the veterinarian, especially when a dog does not respond to standard (i.e., phenobarbital, bromide) therapy. Such refractory cases account for between 25–30% of all epileptics. It is very important for the clinician to inform the pet owner that most epileptic dogs do not reach seizure-free status; success is typically considered a reduction in the frequency and duration of seizures. Nonetheless, the goal of anticonvulsant therapy should be to eliminate seizure activity in the patient, or come as close to this goal as possible, without subjecting the patient to unacceptable side effects of drug therapy or the client to unreasonable financial burden.

**Phenobarbital**

**MOA**
- Primary mechanism of action is by decreasing seizure onset via enhanced GABA activated chloride conductance
- Secondary mechanism of action is by decreasing seizure spread via reduced current through calcium channels and reduce glutamate-mediated excitation

T ½
- 24-40 hours

**Metabolism/excretion**
- Majority metabolized by the liver, with 1/3 excreted unchanged in the
- Phenobarbital will induce hepatic microsomal enzymes (p450 enzymes) and it can be expected that elimination half-lives will decrease with time with concomitant reductions in serum levels

**Side effects**
- Behavioral: hyperexcitability, restlessness, sedation. Normally this is seen for the first few weeks of treatment
- Immune mediated neutropenia or thrombocytopenia or anemia (these reversible blood dyscrasia occurs within the first 6 months of dosing)
- Idiosyncratic hepatic reactions: RARE. Evidenced by a rapid elevation in ALT and abnormal bile acids – phenobarbital should be stopped immediately and another AED should be loaded and started
- With chronic dosing, PU/PD is common and psychogenic polydipsia may develop. The most common serum biochemical changes include elevated alkaline phosphatase

**Dose**
- 2.5mg/kg PO BID as a starting dose, with all future adjustments based on serum drug concentrations in conjunction with clinical assessment

**Blood levels**
- Serial serum phenobarbital concentrations should be evaluated at 14, 45, 90, 180 and 360 days, and 60 days thereafter
- Therapeutic range: 20-40mg/dl

**Bromide**

**MOA**
- Primary mechanism of action is by decreasing seizure onset via enhanced GABA activated chloride conductance

T ½
- 20-46 days

**Metabolism/excretion**
- Bromides are principally excreted by the kidneys

**Side effects**
- PU/PD, lethargy and mild ataxia
- Pancreatitis and gastrointestinal intolerance have been reported but are infrequent

**Dose**
- 40-60mg/kg PO SID
Blood levels
- Measured 8-12 weeks after initiating treatment
- Monotherapy: 1000-3000mg/l
- With Phenobarbital: 1500-2500mg/l

Zonisamide
**MOA**
- Primary mechanism of action is by decreasing seizure spread via reduced current through calcium channels
- Secondary mechanism of action is by decreasing the seizure onset via enhanced sodium channel inactivation

**T1/2**
- 15-20 hours

**Metabolism/excretion**
- Most of the drug is excreted via the kidneys into the urine, but about 20% is metabolized, primarily in the liver

**Side effects**
- Sedation, dry eye, ataxia, inappetence and vomiting – patients with a history of sulfa drug hypersensitivity should NOT be prescribed this medication
- Metabolic acidosis and liver dysfunction has been reported in dogs

**Dose**
- 5-10mg/kg PO BID

**Blood levels**
- Currently, zonisamide levels are able to be evaluated at Auburn University. This is a new medication and at this time we aim to obtain a blood level close to 20ug/ml (10-40ug/ml). It has been reported that stable plasma concentrations are achieved within 3-4 days with oral administration of zonisamide

**Levetiracetam (Keppra)**
**MOA**
- Binding with a specific synaptic vesicle protein (SV2A) in the brain.
- No directly affect common neurotransmitter pathways (e.g., GABA, NMDA) or ion channels (e.g., sodium, T-type calcium).

**T ½**
- 3–4 hours

**Metabolism/excretion**
- 70% excreted in urine
- no hepatic metabolism

**Side effects**
- Lethal dose is 100 times the recommended dose. Dosing can be increased several fold in attempts to increase efficacy.
- No side effects of note

**Dose**
- 20 mg/kg PO TID

**Blood levels**
- Not typically performed because dose-efficacy ratio not direct.

**Other notes**
- neuroprotective properties, and may ameliorate seizure-induced brain damage.
- Prevents further seizures (anti-kindling)
- Injectable-Possible emergency drug for SE
- Honeymoon effect
- More effective in cats- with limited side effects and no noted honeymoon effect

**Felbamate**
**MOA**
- Positive modulator of GABA<sub>A</sub> receptors
- Possible NMDA antagonosit of the MR2B subunit

**T ½**
- 5-6 hours

**Metabolism/excretion**
- 70% excreted in urine
- Remainder hepatic metabolism
Side effects
- No sedation
- Possible hepatotoxicity
- Blood dyscrasia and KCS

Dose
- 15-20 mg/kg PO TID with dose escalation permissible

Blood levels
- The author has not performed drug levels of Felbamate

Other notes
- Expensive

Gabapentin

MOA
- Binding to the α2δ subunit of voltage-gated neuronal calcium channels. This binding decreases intracellular calcium influx, leading to decreased synaptic release of excitatory neurotransmitters.

T ½-
- 3-4 hours

Metabolism/excretion
- Urine excretion
- Hepatic metabolism: 30-40% of the orally administered dose of gabapentin undergoes hepatic metabolism to N-methyl-gabapentin

Dose
- 15-60 mg/kg PO TID or QID

Blood levels
- Suspected blood levels are 4-16 mg/L. As this drug has questionable AED efficacy, I rarely have blood levels checked.
- In recent years, the efficacy of gabapentin as an anti-convulsant has come into question, particularly with the other previously mentioned tertiary agents.

Pregabalin (Lyrica)

MOA: Same as gabapentin

T1/2
- 7 hours (11 hours in cats)
- The half-life of elimination of pregabalin in dogs is about

Dose
- 2-4mg/kg PO BID

Topiramate

MOA
- Mechanisms of action include
  - Decreasing seizure onset via both enhanced sodium channel inactivation and enhanced GABA activated chloride conductance
  - Decreasing seizure spread via reduced glutamate-mediated excitation

T1/2
- 2-4 hours

Metabolism/excretion
- Low rate of hepatic metabolism
- Both unchanged topiramate and its metabolites are excreted mainly by the kidneys

Side effects
- Sedation and weight loss
- In recent studies elevations in liver enzymes were appreciated, however, these patients were also receiving phenobarbital (in conjunction with the topiramate) so it is unknown if the liver elevations were secondary to the phenobarbital or the topiramate

Dose
- 5-10mg/kg PO BID

Blood levels
- Not established at this time
Formulating an initial treatment plan for gastrointestinal disease

A nutritional plan must be formulated for nearly all patients with primary gastrointestinal (GI) disease. Location of the major clinical problem (i.e. esophageal, gastric, small bowel, large bowel) and chronicity can help a clinician determine the most appropriate diet selection. A dietary plan will vary based on the primary problem however the underlying condition may not always be identified. Therapeutic GI diets can be divided into three main categories: highly digestible, novel/hydrolyzed protein, and high fiber. Further, some of these diets may be low in fat which may also benefit patients with GI disease. In general, first line therapy in patients with upper GI diseases (i.e. esophageal to small intestinal) will involve a highly digestible diet and with chronicity may move to a novel/hydrolyzed protein diet. In patients with lower GI disease (i.e. large bowel), a high fiber diet may be the first line therapy followed by novel/hydrolyzed protein diet.

Improvement of GI signs is usually seen rapidly in patients on an optimal diet for their GI disease. If clinical signs persist for > 2 weeks or worsen during this time, an alternative dietary strategy should be considered. GI disease is considered chronic when clinical signs persist for more than 3 weeks or are intermittently present for more than 3 weeks.1 The most common types of chronic enteropathies can be classified based on their response to therapy: food-responsive (including both food intolerance and hypersensitivity), antibiotic responsive, or steroid responsive. Approximately 50% of chronic enteropathy cases will be food responsive.2 In a more recent retrospective study, 64% of dogs with chronic enteropathy were food responsive, 33% antibiotic responsive, and 39% steroid responsive.3 Dietary options, particularly in patients with inflammatory bowel disease (IBD), can include the following strategies: global modification (switching to a different diet all together), optimizing assimilation (feeding a reduced fat, reduced fiber, highly digestible diet), antigenic modification (hydrolyzed/novel protein diets), or immunomodulation (i.e. omega-3 fatty acids, prebiotic fibers).2 Further, hypcobalaminemia has been reported in 36% of dogs and cats with chronic GI disease and should be supplemented in these cases or empirically.4,5

Highly digestible diets

Digestibility is defined as the percentage of a food stuff taken into the digestive tract that is absorbed into the body.6 The goal of a highly digestible diet is to maximize nutrient absorption and minimize stool production. Additionally, a highly digestible diet may decrease the volume of food required to meet energy requirements. The digestibility of a pet food can be determined using specific protocols found in the Association of American Feed Control Official (AAFCO) Official Publication. The terminology “highly digestible” is not defined by AAFCO, however products with a protein digestibility of ≥ 87% and fat and carbohydrate digestibility ≥ 90% may be considered highly digestible.7

Several factors can influence the digestibility of a diet including ingredient selection, heating and processing methods, and animal related factors. As the fiber content of a pet food increases, typically the protein and fat digestibility will decrease. The digestibility of starchy carbohydrates will improve with heating. Animal related factors include both breed and size for dogs. Large breed dogs tended to have higher fecal water contents, decreased stool quality and increased number of defections than small breed dogs while differences were noted in certain breeds (i.e. Irish wolfhounds had lower fecal water content than Labrador retrievers).8 Age has also been shown to be a significant factor in macronutrient digestibility with increasing digestibility from 11 – 60 weeks of age in dogs.9 In this same study, large breed dogs had increased fecal moisture content and lower fecal scores than small breed dogs, but their nutrient digestibility was significantly higher demonstrating that decreased fecal quality was not related to lower nutrient utilization in these otherwise healthy dogs. Reduced protein digestibility is reported to occur in some geriatric cats10 Cats can efficiently digest and absorb carbohydrates. Seventy one percent of cats with chronic diarrhea responded with improvements in fecal scores to both a highly digestible, moderate carbohydrate and highly digestible, high-protein, low-carbohydrate diet with no difference between groups.11 Highly digestible diets are typically a first line therapy for dogs and cats with acute gastroenteritis and may also be useful in patients with GI diseases including chronic small bowel diarrhea, short bowel syndrome, megaesophagus, and post-operative GI surgery.

Low fat diets

Low fat diets can be found in all categories of GI diets, however most therapeutic GI low fat diets are highly digestible. While there is no standard cut off for “low fat” in cat or dog diets, typically the author regards diets containing <25%ME fat for dogs and < 35%ME fat for cats as low fat. Diets containing <20% ME fat are considered ultra-low fat for dogs. AAFCO defines “low fat” on an as fed basis using crude fat for both cats and dogs for dry, semi-moist, and canned diets. Fat reduction may also be relative to what the animal is currently consuming. Diets with a high fat content may be less ideal for some patients with GI disease as they can reduce the tone of the lower esophageal sphincter through gastrin inhibition and increased secretin and cholecystokinin (CCK) release, decrease gastric emptying through increased CCK, and may contribute to steatorrhea if excess fat passes into the colon.12–15 Low fat diets can
be important for the management of dogs with chronic vomiting, regurgitation, or protein losing enteropathies. Dietary fat restriction is efficacious in treating dogs with intestinal lymphangiectasia, particularly those unresponsive to prednisone treatment or with recurrent clinical signs and hypoalbuminemia when prednisone is tapered. Low fat diets may be less important for the management of chronic GI disease in cats. Cats with chronic diarrhea had no difference in their response of fecal scores when placed on either a high fat (45.1% ME) or a low fat (23.8% ME) highly digestible diet with 78.2% of all cats improving in their fecal scores and 36% of cats developing normal stools.

**Novel protein/hydrolyzed protein diets**

Novel or hydrolyzed protein diets are typically used in patients with chronic GI disease to avoid food antigens that may lead to an immune response, lessen clinical signs of GI disease, and prevent relapse. A novel carbohydrate is also typically recommended for these patients as carbohydrate ingredient sources will also contain varying amounts of protein. Diagnosis of food hypersensitivity can only be determined at this time by food elimination trial with challenge. Salivary allergen-specific IgA tests are not recommended as validation studies are not available to determine their efficacy. Serum allergen-specific IgE tests are unreliable with one study reporting a 15.4% positive predictive value. Twenty nine percent of cats with chronic GI signs were determined to be food sensitive through the use of a novel protein diet with dietary challenge finding 55% of affected cats sensitive to more than 1 food ingredient. In dogs with food responsive chronic enteropathy, 55% were on an elimination diet, 44% on a hydrolyzed protein diet, and 1% on a home cooked diet with no significant difference in outcome between dogs on an elimination diet or hydrolyzed protein diet. In another study evaluating long term efficacy of a hydrolyzed protein diet, 88% of dogs with chronic small bowel enteropathy on both a hydrolyzed protein and highly digestible diet responded favorable in the first 3 months, however long term control was significantly better in the hydrolyzed protein group at 6-12 months and 3 years later. No prospective studies comparing the response to a novel protein diet versus a hydrolyzed protein diet are currently available in the dog or cat.

Therapeutic hydrolyzed protein diets may be advantageous for clinicians given the wide variety of ingredients available in over-the-counter (OTC) pet diets making selection of a novel protein diet difficult. Further, cross contamination of food antigens not listed on the label of OTC diets renders these diets inappropriate for food elimination trials and increases the potential number of antigen exposure for a pet. This cross contamination likely occurs at some point during the manufacturing process of the diet. Three of 4 OTC venison dry dog foods tested positive for beef or soy antigens with no beef or soy products in the ingredient lists. Another study found soy protein in 3 of 4 OTC diets claiming to contain no soy. Interestingly this same study found soy protein (>2.5ppm) in 4 of 7 therapeutic diets for food elimination trials with 2 containing a soy product on the label (soy protein hydrolysate). Soy-sensitized dogs have been shown to tolerate hydrolyzed soy protein with no clinical response after an oral challenge making hydrolyzed soy protein diets still a good option. When choosing a novel protein diet, one must have a thorough knowledge of dietary protein exposure and that the diet chosen only contains the protein listed. Even with home prepared diets, cross contamination can occur at the level of the butcher, supplier, or in the home of the owner preparing the food therefore careful selection of protein source, supplier, and preparation should be emphasized for these owners.

**High fiber diets**

While dogs and cats do not have a nutritional requirement for fiber, fiber can be added to the diet for maintenance and promotion of GI health. A high fiber diet is recommended as a first line therapy for cats and dogs with large bowel diarrhea or constipation. Fiber is a poor source of energy, therefore its inclusion in pet food will decrease energy density and digestibility. Fiber is expressed on pet food labels as a maximum percentage of crude fiber which reflects the percentage of cellulose (insoluble fiber) in the diet. Total dietary fiber more accurately reflects the fiber content of a pet food by including both soluble and insoluble fibers with the exception of certain types of fiber such as oligosaccharides. Fibers are classified based on their solubility (increased water-binding capacity to make a viscous solution), fermentation (ability of colonic microbes to produce short chain fatty acids), and prebiotic ability to support the growth and activity of health-promoting bacteria in the GI tract. Soluble fibers, which form a gel in the upper GI tract, will increase GI transit time, slow nutrient absorption, and increase fecal water content. Insoluble fibers increase fecal bulk, are moderately to slowly fermentable in the colon, and have no effect on transit time. Sixty three percent of dog with a history of chronic idiopathic large bowel diarrhea with limited success to other dietary strategies had an excellent response when started on a highly digestible diet with added soluble fiber (psyllium husk). A moderate fiber, psyllium-enriched, dry extruded diet was shown to be efficacious in cats with a history of recurrent feline constipation with 85% improvement in all cats and a significant decrease in medical management. Prebiotic fibers, such as inulin, fructooligosaccharides, manno-oligosaccharides, and resistant starch, are selectively fermented by commensal bacteria in the GI tract to produce short chain fatty acids that have beneficial effects on colonic health. Dietary fiber has also been shown to alter the microbiome of both dogs and cats. Cats and dogs that may benefit from a high fiber diet can be placed on a therapeutic commercial diet option or supplemental dietary fiber can be added to an existing diet.
References

Feeding Patients that have Chronic Kidney Disease: The Controversy and Lessons from the Literature

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Nutritional assessment of patients with chronic kidney disease
A complete nutritional assessment and staging of a patient’s chronic kidney disease (CKD) is required prior to making nutritional recommendations. The World Small Animal Veterinary Associations has guidelines to complete a nutrition assessment.1 Guidelines for staging and sub-staging recommendations are provided by the International Renal Interest Society (IRIS).2 When taking a diet and general history, the owner of a patient with CKD should be questioned about appetite, weight loss and time frame for weight loss, current diet, evaluation of current protein intake, treats or table foods, foods used for medication administration, and supplements. Evaluation of the patient should include body weight, body condition score (BCS), evaluation of muscle mass, and systolic blood pressure. Important laboratory parameters include creatinine, blood urea nitrogen (BUN), potassium, albumin, bicarbonate, urine specific gravity, and urine protein-to-creatinine ratio.

Adult maintenance diets versus therapeutic kidney diets
Kidney diets are amongst the oldest therapeutic diets for dogs and cats. The IRIS recommends nutritional intervention at IRIS stage 2 to control serum phosphate levels, however a diet change may be recommended for patients with IRIS stage 1 following a complete nutritional assessment. Double-blinded, randomized, controlled clinical trials have compared the efficacy of therapeutic kidney diets (TKD) to adult maintenance diets (AMD) in patients with ≥ IRIS stage 2 chronic kidney disease (CKD):

- Ross et al. fed cats with spontaneous IRIS 2 and 3 CKD a TKD (n = 22) or an AMD (n = 23) for 24 months.3 No cats in the TKD group experienced a uremic crisis and no kidney-related deaths occurred. Twenty six percent of cats in the AMD group had a uremic crisis and 21.7% of cats experienced a kidney-related death.
- Elliott et al. fed client-owned cats with spontaneous CKD a TKD (n = 29) or an AMD (n = 21).4 Cats fed a TKD had a median survival time of 633 days (1.7 years) compared to 264 days (0.7 years) for cats fed an AMD. Feeding a TKD also helped reduce plasma phosphate, blood urea nitrogen, and parathyroid hormone concentrations.
- Jacob et al. fed dogs with ≥ IRIS stage 2 CKD a TKD (n = 21) or an AMD (n = 17) for 24 months.5 Dogs fed a TKD has a median time to uremic crisis of 615 days and median survival time of 594 days. Dogs fed an AMD had a median time to uremic crisis of 252 days and median survival time of 188 days. Kidney related deaths accounted for 33% and 65% of deaths in the TKD and AMD groups, respectively.

These studies demonstrate that TKDs can be used to improve quality of life by controlling signs of uremia and increasing life span by altering disease progression.

Protein intake and chronic kidney disease
Protein restriction is commonly recommended for the management of CKD to reduce nitrogenous wastes and glomerular proteinuria. The timing and necessity of protein restriction to avoid protein malnutrition and loss of lean body mass (LBM) is controversial. Nitrogenous wastes can contribute to clinical signs associated with uremia, polyuria, polydipsia, and anemia. Loss of LBM related to chronic illness, also referred to as cachexia, occurs in patients with CKD. Cachexia is associated with altered strength, immune function, wound healing, and overall survival although more specific research in cats and dogs is needed.6 Dogs with a body condition score (BCS) of 1 – 3/9 had a reduced survival compared to dogs with a BCS of ≥ 4/9.7 The optimal protein intake for patients with CKD is likely multifactorial depending on the stage, sub-stage, and complete nutritional assessment of the patient.

Protein requirements
Protein requirements for dogs and cats have traditionally be determined through the use of nitrogen balanced studies, wherein nitrogen loss is equivalent to nitrogen intake. More recently, a study by Laflamme & Hannah evaluated the protein requirement of healthy adult male cats based on the maintenance of LBM.8 To achieve nitrogen balance, 56g protein/1000kcal of diet was needed while 95g protein/1000kcal was needed to maintain LBM. Regression analysis suggested that while 1.5g protein/kg body weight was required for nitrogen balance, 5.2 grams of protein/kg body weight is recommended to maintain LBM. This demonstrates that nitrogen balance can be achieved on a low protein diet, but it may be at the expense of LBM through the use of endogenous proteins. Suboptimal protein intake in the face of CKD can increase the production of uremic toxins through protein catabolism. It should be noted however that suboptimal protein intake can also result from hyporexia or anorexia which occurs commonly in patients with CKD.

Proteinuria and protein restriction
The American College of Veterinary Internal Medicine recommends a reduced protein intake in dogs with proteinuria secondary to glomerular disease.9 In rats and humans, protein directly injures the tubulointerstitium through release of vasoactive and inflammatory substances that trigger renal scarring and loss of function.10 Feeding a TKD may improve proteinuria.
In dogs with hereditary nephritis, feeding a protein restricted TKD reduced structural damage to glomeruli by decreasing glomerular basement splitting and delayed the progression of kidney failure.\textsuperscript{11}

Dogs with proteinuria treated with benazepril fed a TKD had a reduced urine protein to creatinine ratio (UPC) over 60 days (UPC 3.16 to 1.2) compared to dogs fed an AMD (UPC 3.62 to 2.14).\textsuperscript{12} The dose of benazepril did not differ between groups.

Proteinuria was reduced significantly in dogs with glomerulonephropathy when they were switched from a diet containing 72g protein/1000kcal to a diet containing 33g protein/1000kcal.\textsuperscript{13}

A reduction in protein intake by 25 – 50\% is recommended based on the severity of proteinuria and patient assessment. This reduction may be relative to the patient’s current intake.

**Dietary protein in TKDs**

At this time, there is no definitive conclusion in the veterinary literature of which is better: a diet with unrestricted protein with the remaining features of a TKD versus a protein restricted TKD. The answer to this question is likely dependent on the species, IRIS stage and sub-stage. Studies demonstrating improved survival of dogs and cats fed a TKD compared to an AMD are limited to those with confounding dietary variables such as phosphorus restriction. The amount of protein needed to achieve protein restriction is not clearly defined.

- National Research Council (NRC) Minimum Requirement for crude protein is 40 grams and 20 grams of protein per 1000kcal for cats and dogs, respectively
- Association of American Feed Control Officials (AAFCO) minimum crude protein is 65 grams and 45 grams of protein per 1000kcal for cats and dogs, respectively

At this time, TKDs for CKD range from 25 – 55 grams protein in dogs and 58 – 82 grams protein per 1000kcal in cats. A highly digestible protein source is recommended. Reduced protein digestibility is reported to occur in some geriatric cats.\textsuperscript{14}

**Phosphorus restriction**

Phosphorus restriction, independent of other dietary factors, delays progression of CKD in both dogs and cats. Dogs with induced CKD fed a high phosphorus diet had significantly lower glomerular filtration rates and decreased survival compared to dogs fed a phosphorus restricted diet.\textsuperscript{15} Cats with induced CKD fed a normal phosphorus diet had evidence of renal mineralization, fibrosis and mononuclear cell infiltrates compared to cats fed a low phosphorus diet which has no histologic changes. AMDs often contain added phosphorus (usually >1.5g per 1000kcal) to avoid phosphorus deficiency and maintain a 1:1 – 2:1 calcium to phosphorus ratio. AAFCO minimum for adult cats is 1.25g/1000kcal and adult dogs 1g/1000kcal. TKDs range from 0.4 – 1.2 g/1000kcal and 0.8 – 1.35 g/1000kcal for dogs and cats, respectively.

**Omega-3 fatty acids**

Supplementation with polyunsaturated omega-3 fatty acids (EPA and DHA) can have renoprotective effects. Dogs fed a diet supplemented with a high dose of fish oil had reduced proteinuria, creatinine, and histopathologic lesions compared to dogs fed a diet supplemented with safflower oil or beef tallow.\textsuperscript{17} A standard dose of 40mg/kg EPA + 25 mg/kg DHA once daily is recommended for both cats and dogs. Recently, a specific dose for dogs with CKD was recommended: 140 mg EPA + DHA / (kg body weight)\textsuperscript{0.75}. Some companies may add alpha-linoleic acid to TKDs, however this omega-3 fatty acid is insufficiently converted to EPA and DHA in both cats and dogs. Additional supplementation with EPA and DHA is recommended if the diet does not provide these nutrients.

**Dietary potassium**

Hypokalemia is common in cats with CKD and therapeutic diets may provide supplemental potassium beyond that added to a typical AMD. While hyperkalemia is typically associated with acute kidney failure, it may be a complication in some dogs with CKD. Hyperkalemia has been corrected by feeding a home prepared diet with reduced potassium (0.91 ± 0.14 g/1000kcal) in some dogs with CKD.\textsuperscript{18} Some TKDs contain potassium levels around this concentration which may be useful in these patients. Referral to a board-certified veterinary nutritionists for a home-prepared diet formulation may also be considered for a potassium restricted diet.

**Other dietary features of TKDs**

- Reduced sodium content to avoid the potential for sodium retention and contribution to systemic arterial hypertension
- Calorie dense with moderate to high levels of dietary fat to increase caloric intake and enhance palatability
- Alkalining to help correct metabolic acidosis
- Added antioxidants such as vitamin C and E to decrease oxidative stress
- Added soluble fiber to promote colonic bacterial growth and utilization of nitrogen and urea; beneficial for constipation
**Tips for feeding patients with CKD**

Introduce a TKD before clinical signs of uremia occur if possible. Diets are available in a variety of forms, flavors and textures. Provide clients with various samples to establish patient preferences. Be cautious when introducing a TKD to a hospitalized patient. This may lead to a food aversion in a diet best utilized for long-term feeding.

Feeding tubes are useful when managing patients with CKD. Liquid enteral diets containing < 1.5g phosphorus/1000kcal (both human and veterinary) are available for use with nasogastric and nasoesophageal feeding tubes. When an esophageal or gastric feeding tube is in place, a slurry of a canned TKD is recommended. Caloric density of a slurry can be improved when using a liquid enteral diet rather than water.

Home prepared diets are useful in patients with a poor or selective appetite. Referral to a board certified veterinary nutritionists is recommended. Home prepared diets found in books and on websites have numerous inadequacies and are not recommended. Before referral, updated IRIS staging including systolic blood pressure and UPC is recommended.

Overall, nutritional intervention in a cat or dog with CKD can greatly affect patient morbidity and mortality. The optimal diet for a patient with CKD relies on a variety of factors including the stage and sub-stage of disease, nutritional assessment, and patient preferences.

**References**

Formulating a Plan for Feeding Hospitalized Patients
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Nutritional assessment of a hospitalized patients
Malnutrition in hospitalized small animals can contribute to both patient morbidity and mortality. Caloric intake is positively associated with hospital discharge and outcome in small animal patients. The nutritional assessment of a hospitalized patient should take place daily. Historical information that can indicate malnutrition and impact the feeding plan includes:

- Weight loss and the time frame for weight loss
- Dietary intake and the length of anorexia/hyporexia
- Nutritional adequacy of the diet consumed (e.g. unbalanced home cooked diet versus complete and balanced commercial diet)
- Persistent gastrointestinal signs such as vomiting or diarrhea
- Other disease states that may impact metabolic needs (i.e. diabetes mellitus)

Physical exam should include evaluation the patient’s body weight, body condition score (BCS) and muscle condition. Muscle mass score, while not a validated scale, can be useful in assessing lean body mass (LBM). It is important to note the BCS does not account for LBM therefore it is possible to have an overweight/obese patient with poor muscle condition as a result of cachexia. Cachexia is the loss of LBM due to a disease state that decrease the body’s ability to preserve protein from catabolism which can directly impact strength, immune function, wound healing, and survival. Additional physical exam parameters as part of a nutritional assessment includes mentation, presence of ascites or edema, and hair coat quality. Laboratory parameters that may directly impact the nutritional plan include albumin, blood glucose, electrolyte status (potassium, magnesium, phosphorus), triglycerides, or presence of renal azotemia. A daily nutritional assessment should also assess feeding orders and previous day’s intake to determine if sufficient or if nutritional intervention is needed.

In practice, the author commonly encounters hospitalized patients with gastrointestinal dysmotility. Clinical signs include persistent vomiting or regurgitation, abdominal pain, constipation, or intolerance to enteral feeding. The primary types of gastrointestinal motility disorders in small animal patients include esophageal dysmotility, delayed gastric emptying, functional intestinal obstruction (ileus), and colonic motility abnormalities. Assessment of gastrointestinal dysmotility is limited for many clinicians to clinical signs, abdominal imaging, and gastric residual volumes (GRVs) if a nasogastric or gastric feeding tube is in place. Currently there is no clearly defined rule on how to adjust feeding when accounting for GRV. Clinical decisions regarding feeding after a GRV is recorded should take into account current pharmacological interventions and clinical signs. Dogs with nasoesophageal or nasogastric feeding tubes were randomized to receive either continuous or bolus feeding enteral nutrition. There was no difference in GRV between the continuous or bolus fed groups of dogs with nasogastric feeding tubes. Additionally the researchers were unable to find a significant correlation between the GRV and occurrence of vomiting or regurgitation. This suggests that termination of enteral feedings due to high GRV may not be warranted. If gastrointestinal dysmotility is diagnosed or suspected, therapy should be multimodal to include fluid therapy, early ambulation, early nutritional support, pharmacological intervention to stimulate motility and control pain as well as treatment of the underlying condition, metabolic derangements, and establishing normothermia.

Writing appropriate feeding orders
Feeding orders should include the following information: Resting energy requirement (RER), type(s) and amount of food to be offered, and frequency of feeding. Veterinary nurses and students were given a hypothetical voluntarily-eating canine patient with vague (frequency only), intermediate (diet and frequency), and specific (diet, amount, and frequency) feeding orders and asked to feed the patient. Significant differences in kilocalories fed per day were found between specific orders when compared to vague and intermediate, with no differences between vague and intermediate. Results of this study highlight that the amount of kilocalories fed is impacted by the manner in which the patient feeding orders are written. In a study evaluating caloric intake of hospitalized dogs, dogs were in a negative energy balance (<95% RER achieved) 73% of days evaluated (601/821 days). This was due to anorexia/hyporexia (44%), food withheld (34%), and poorly written feeding orders (22%). Evaluation of the patient’s intake over the last 24 hours during the morning assessment is also an important step before determining a patient’s nutrition plan and feeding orders for the day.

Resting energy requirements (RER)
RER is the number of calories required to maintain homeostasis at rest in a thermoneutral environment while the animal is in a fed state. While disease states can alter energy requirements, it is recommended to start with a patient’s RER as the initial goal to avoid overfeeding. Predictive equations are used to estimate energy requirements in small animal patients. While use of indirect calorimetry
Food typically offered to hospitalized patients includes therapeutic recovery type diets or therapeutic highly digestible gastrointestinal type diets. When creating an in-hospital stock of food for feeding hospitalized patients, recommend the following general considerations:

- Choose a highly digestible gastrointestinal diet approved for both growth and adult maintenance.
- Have available a low-fat (<25% metabolizable energy (ME)), highly digestible gastrointestinal diet for dogs with fat intolerance (i.e. pancreatitis).
- Recovery type diets are typically higher in protein (>25%ME) and fat (>45%ME). These diets are highly digestible and palatable, however are not appropriate for every patient especially dogs with fat intolerance. Also ensure these diets are approved for growth if fed to growing animals.
- Have both dry and canned options available for cats including canned foods with a variety of textures and flavors.
- If stocking alternative food choices like meat baby food or home prepared foods like chicken and rice, know the general nutrient profile and possible contraindications for feeding (e.g. meat baby food is typically high fat (>50%ME), 50:50 boiled boneless, skinless chicken breast and cooked white rice is highly digestible and ultra-low fat (<20%ME)).

Amount of food to be offered

The amount of food should be recorded as a standard measurements such as cup, can, or tablespoon. The use of non-specific feeding amounts such as “meatball” should be avoided without some type of quantification. If using dry extruded kibble, ensure an 8 ounce measuring cup is used. However, measuring cups are imprecise. Over 12 studies, variable accuracy when measuring dry extruded kibble ranged from an 18% under-estimate to an 80% over-estimate in portion size. Weighing both canned and dry food with a kitchen scale in grams is more precise and is preferred by the author in practice. Weighing the food in grams prior to and after feeding is an objective way to determine a patient’s caloric intake and help a clinician determine if assisted feeding is necessary. In the author’s experience, subjectively recording a patient’s intake by percentage is limited and can be further complicated if the person offering the food is different from the one recording intake. Hospitalized patients can be started on a fraction (25 – 33%) of RER initially gradually increasing in increments every 12 – 24 hours to full RER. This method useful in patients with anorexia or hyporexia to avoid food waste, and helps to minimize complications such as gastrointestinal signs or refeeding syndrome.

Frequency of feeding

The number of times per day a patient is fed should also be recorded on the treatment sheet. Typically the author advocates twice daily feeding for patients following routine procedures such as spay, neutering, dental, or orthopedic procedures or patients with an excellent appetite that are otherwise feeling well. Increased frequency of feeding should be considered in other patients including the critically ill, very young (<6 months in age), hypoglycemic, or those with gastrointestinal disease, pancreatitis, or post-operative gastrointestinal surgery.

Assisted feedings for hospitalized patients

Nutritional intervention should be considered in any patient on admission with anorexia ≥ 3 days, a patient where anorexia is anticipated for ≥ 3 days, long term hyporexia, and sooner in some patients following nutritional assessment. This includes and is not limited to overweight or obese cats, septic peritonitis, pancreatitis, or parvovirus. Nutritional support initiated <24 hours postoperatively in dogs with septic peritonitis was associated with a significantly shorter hospitalization length (1.6 days) when compared to dogs receiving nutritional support after 24 hours. The concept of “bowel rest” is no longer recommended as several studies have indicated that early enteral nutrition is well tolerated with few complications. Early enteral nutrition through an esophagostomy tube in dogs with severe acute pancreatitis was well tolerated with fewer complication that dogs receiving parenteral nutrition. Early enteral nutrition instituted at 12 hours after admission through a nasogastric tube in dogs with parvovirus resulted in earlier clinical improvement and possibly improved gut barrier function compared to dogs receiving enteral nutrition after vomiting.
had ceased for 12 hours (approximately 50 hours after admission).12 Prior to initiating nutritional support, a patient should be cardiovascularly stable, recovered from anesthesia, and electrolyte and acid-based abnormalities addressed.

The use of nasoesophageal (NE) and nasogastric (NG) feeding tubes are utilized frequently in practice by the author. NE and NG feeding tubes are very useful for temporary feeding support (3 – 5 days). Use of an esophageal of gastric feeding tube is recommended in patients needing long term assisted feeding (>1 week). The placement of NE and NG feeding tubes does not require specialized equipment or general anesthesia. Some animals may require minimal to heavy sedation during NE or NG tube placement. A liquid enteral diet is required with nasoenteric feeding tubes to avoid clogging of the tube. Nasojejunal (NJ) feeding tube placement has been described in dogs using flurosocopy, endoscopy, and have also been placed in the author’s experience during abdominal surgery with guidance of the tube into the jejunum by the surgeon.13,14 NJ feeding tubes are indicated in patients where post pyloric feeding is ideal including acute pancreatitis, pyloric disease, or patients where gastric feeding is poorly tolerated. The use of NJ tubes typically requires the use of specialized equipment and therefore can limit their use in general practice. Complications from NE and NG tube placement can include vomiting, diarrhea, tube blockage, tube removal, rhinitis, epistaxis, and esophagitis. The most severe complication may be tracheal intubation with iatrogenic pneumothorax, abscess, tracheal perforation, fistula formation, or death.15 The technique of measuring and aspirating the tube at the level of the thoracic inlet may help to reduce tracheal intubation and associated complications.16 Retrospective review in dogs with NE or NG tube placement revealed no significant difference in recorded complications.17 There was also no significant difference in gastrointestinal complications or nutrition delivered between continuous versus intermittent feeding through nasoenteric feeding tubes in both cats and dogs.18 The use of 1/4 teaspoon pancreatic enzymes and 325 mg sodium bicarbonate in 5 mL of water has been used successfully by the author to unclog nasoenteric feeding tubes.19 When using esophageal or gastric feeding tubes, a slurry of the diet with a liquid enteral diet instead of water can improve caloric density. Patients requiring other types of assisted feeding such as jejunostomy feeding tube or parenteral nutrition likely required 24 hour care and referral to such a hospital is recommended.

References
In 2013, Calcium oxalate (CaOx) uroliths submitted to the Minnesota Urolith Center for analysis represented 41-42% of submissions in cats and dogs. CaOx uroliths cannot be dissolved and must be removed physically from the patient. Unfortunately, recurrence rates of up to 50% of dogs within 3 years and 33% of cats in two years of removal have been reported. Risk factors for the formation of CaOx urolithiasis in cats include males, neutering, increasing age, and breed such as Burmese, Persian, and Himalayan. Risk factors for the formation of CaOx urolithiasis in dogs include <7yr age, males, neutering, toy-small sized breeds, acidic urine pH and history of cystitis. In a recent retrospective study of 135 dogs with CaOx urolithiasis, Miniature Schnauzers were 3 x at risk for recurrence than other breeds. This study also evaluated the effect of two therapeutic CaOx preventative diets versus any other type of diet on the recurrence of CaOx uroliths finding no significant difference between groups. Prospective controlled clinical trials are needed to further evaluate the long term effects of therapeutic CaOx preventative diets given that the time to recurrence can be several years. Given the inability to medically dissolve CaOx uroliths and the relatively high recurrence rate, preventative strategies should be instituted. Urolith prevention includes the following principals regardless of stone type: increasing water intake, altering urine pH, decreasing precursors, and increasing urinary inhibitors.

CaOx form in urine oversaturated with calcium and oxalate. Relative supersaturation (RSS) is a validated method for measuring the pH and saturation of mineral ions in the urine of cats and dogs to determine their risk for urolith formation. This method is commonly used by pet food manufactures to determine the efficacy of their diet for CaOx prevention. When urine is oversaturated with mineral precursors, crystals will form which will then grow and aggregate to form a calculus. Crystals will dissolve in urine when the RSS is <1 (undersaturated). When urine has an RSS of >1, it is supersaturated. Supersaturated metastable urine will prevent spontaneous crystallization while labile or unstable supersaturation will allow crystallization to occur. Since CaOx uroliths cannot be dissolved, target RSS is metastable to prevent crystallization.

Increasing water intake
The purpose of increasing water intake is to decrease the urinary concentration of urolith precursors and increase urine volume to increase voiding frequency. Target urine specific gravity (USG) in dogs is <1.030, while a USG <1.040 is appropriate for cats. Switching to a canned diet is the simplest and most practical way to accomplish this goal. High moisture content diets increase the daily fluid intake, resulting in lower USG and lower RSS for CaOx versus diets with a lower moisture content. The use of water fountains has also been suggested as a way to increase water intake in cats. Two studies reported water intake of cats using still or flowing water sources. Neither study was able to find statically significant differences in water intake. A recent abstract by the author and colleagues investigated the differences in water intake and urinary parameters between free-falling, circulating, and still water bowls in a randomized, cross-over design with 14 laboratory cats. No significant differences were found between groups in water intake, USG, urine osmolality, and RSS for CaOx and struvite. Three individual cats in our study did have statically significant differences in water intake between bowls, but these were different for each cat suggesting that some cats will have an individual preference for water source.

Sodium is added to some therapeutic urinary diets to increase water intake. Dietary supplementation with NaCl in dogs increased urine volume and urine calcium excretion while urine oxalate concentrations and RSS for CaOx were significantly decreased. Some evidence has suggested that cats with early renal disease (IRIS Stage 1) will have progression of their renal parameters on a high sodium diet. Alternatively, healthy aged cats (median age 10.4yrs) had no alterations in glomerular filtration rate, blood pressure, or routine clinical pathological parameters in a 2 year period randomized to either a high sodium (3.1g/1000kcal) versus a low sodium (1g/1000kcal) diet. The exact appropriate level of sodium for a therapeutic CaOx preventative diet in cats and dogs is unclear as commercial therapeutic diets have a wide range from sodium contents.

Altering urine pH
Acidic urine pH is a reported risk factor for the formation of CaOx urolith formation in dogs and cats by increasing urine calcium excretion.5,17 The role of urine pH in CaOx prevention unclear. A recent abstract demonstrated that while calcium excretion increased with decreasing urine pH (mean 6.37 – 5.93), CaOx RSS was not affected by urine pH. Another study demonstrated that urinary saturation of CaOx was decreased in cats consuming an alkalinizing diet with a urine pH >7.2 compared to cats consuming the same diet but acidifying with an increased in CaOx urinary saturation at a pH <6.5. Multifunction urolith preventative diets typically target urine pH slightly acidic as struvite urolith solubility is pH dependent and dissolution can be achieved. Supplementation with potassium citrate at 75mg/kg PO q12hr is recommended to alkalinize the urine. Target urine pH for CaOx urolith prevention is 6.6-7.5.
Decreasing precursors
Oxalate is primarily derived from dietary intake. High oxalate containing foods include certain vegetables (e.g. spinach), legumes, and fermentable fibers (e.g. beet pulp). The endogenous hepatic metabolism of glycine and vitamin C will also contribute to oxalate formation. 

Decreased dietary intake of calcium is not recommended without also decreasing dietary oxalate in normocalcemic patients with CaOx urolithiasis as this may increase their risk of formation. Dietary calcium can bind oxalate in the lumen of the gastrointestinal tract thus preventing absorption.

Thirty five percent of cats (7/20) with idiopathic hypercalcemia were diagnosed with urinary or renal calculi. Two of these cats underwent cystotomy and analysis revealed 100% CaOx uroliths. Dietary and medical strategies should be aimed at treating the hypercalcemia in these cats. Lower calcium intake may be beneficial for idiopathic hypercalcemic patients. Two of 3 hypercalcemic cats with a history of CaOx urolithiasis fed a calcium oxalate-prevention diet achieved normocalcemia while the third cat had a significant reduction in its hypercalcemia. These three cats also had significant reductions in urinary calcium excretion. Hypermagnesemia was reported in 33% of cats undergoing a prospective randomized clinical trial investigating the recurrence rate of CaOx urolithiasis in cats on a test diet for CaOx prevention versus a regular maintenance diet.

Cats on the prevention diet had fewer episodes of hypercalcemia and the RSS for CaOx and urinary calcium concentration were lower in cats fed the prevention diet. CaOx preventative diets may be useful in these cases if reductions in hypercalcemia or normocalcemia can be achieved although additional research is needed.

Increasing urinary inhibitors
Citrate is a urinary inhibitor that forms a soluble salt with calcium to reduce CaOx precipitation. When urine pH is low, urinary concentration of citrate decreases due to increased absorption of citrate by the renal proximal tubules. Supplemental potassium citrate can also be used to alkalize the urine which can increased renal citrate excretion. Recommended dosing is 75mg/kg PO q12hr. Large molecular weight proteins in the urine, such as Tamm-Horsfall glycoprotein, nephrocalcin, uropontin, and glycosaminoglycans, are recognized as inhibitors of CaOx formation by influencing crystal formation, aggregation, and growth.

Cats with a history of CaOx urolithiasis fed a therapeutic CaOx prevention diet has significantly higher concentrations of glycosaminoglycans than when they were fed the diet they consumed prior to diagnosis. No differences in Tamm-Horsfall glycoprotein and nephrocalcin were found between diets.

Monitoring tips
Recommended monitoring includes urinalysis with evaluation of a fresh urine sediment in-house. Persistent calcium oxalate crystalluria, particularly in a predisposed breed, represents an increased risk for CaOx urolith formation. CaOx crystalluria is not a reliable indicator of CaOx urolithiasis as uroliths can be present with or without the presence of crystalluria. Follow-up urinalysis is recommended every 3-4 months until target urinary parameters are achieved. Bi-annual monitoring of urinalysis and abdominal imaging is recommended for long term monitoring.
References


Obesity is the most common form of malnutrition in cats and dogs. Animals are considered obese when they reach a body condition score (BCS) of 8/9 which is approximately 20% over their ideal body weight or ≥35% body fat. Owners and veterinarians must both recognize that a patient is overweight or obese while also acknowledging the health consequences of excess adiposity. Surveys of dogs and cats presenting to veterinarians in primary or referral practices indicated that only 11.4% of overweight/obese dogs and 3.6% overweight/obese cats had overweight or obesity listed as a medical problem. Further, 53% of owners in one study assigned their overweight or obese dog with an appropriate BCS, however 39% of these owners thought their dog’s weight was acceptable or normal.

Life span and quality of life
Overweight/obesity significantly impact life span, quality of life, and the development of chronic diseases in cats and dogs. Labrador retrievers fed 25% less than age-matched pairs had a median life span of 13 years while dogs fed 25% more had a median life span of 11.2 years. Dogs fed more were overweight, not obese, with a mean BCS of 6.7±0.19 compared to the feed restricted dogs with a mean BCS 4.6±0.19. Chronic treatment for osteoarthritis was delayed in the feed restricted group (13.3 vs 10.3 years) and treatment for chronic diseases in general was also delayed (12 vs 9.9 years). Radiographic evidence of osteoarthritis (OA) was also found later in the feed restricted dogs (at 2 years of age, hip OA 4% versus 42%). A similar life time study does not exists at this time in cats. Mobility is often a factor for owners when assessing their pet’s quality of life. Modest weight loss of 6.1% significantly decreased lameness in dogs. Obese cats are 4.9 times more likely to develop lameness requiring veterinary care. Dogs completing a weight loss plan had improved quality of life (increased vitality and decreased emotional disturbance and pain) when compared to dogs that failed to achieve their ideal body weight. Cats undergoing weight loss for 8 weeks had an increase in pre-feeding behaviors such as begging, following, meowing, pacing however owner reported their cats became more affectionate post feeding.

Obesity paradox
The finding that overweight or obesity may be protective in regards to mortality in a variety of disease conditions is termed the obesity paradox. While overweight, obesity, and abdominal adiposity are associated with increased risk of heart failure in people, overweight/obesity are associated with lower all-cause and cardiovascular mortality in people with congestive heart failure (CHF). While the protective mechanism of the obesity paradox is not clear and likely multifactorial, increased reserve of lean body mass (LBM) with obesity and lack of cachexia likely plays a major role. Few studies investigate this phenomenon in veterinary medicine. In a recent abstract, cats below a median body weight of 4.2kgs at the time of diagnosis for chronic kidney disease (CKD) has a significantly shorter survival time compared to cats with a body weight ≥4.2kgs. Underweight dogs with CKD (BCS 1-3/9) had a significantly shorter median survival time (MST) compared to moderate (BCS 4-6/9) and overweight dogs (BCS 7-9/9) with no difference between moderate and overweight. A study of survival in dogs with CHF failed to demonstrate a significant association with BCS, however weight change was significantly associated with survival with dogs gaining weight surviving the longest. A similar study in cats with CHF found a U-shaped relationship between body weight and survival with reduced survival times in cats with the lowest and highest body weights. Given the available literature, maintenance of a BCS of 6-7/9 in patients with chronic diseases such as CHF or CKD is a reasonable recommendation to preserve LBM and prevent cachexia. Additional research is required to investigate this recommendation.

Chronic kidney disease
Obesity is a risk factor for the development and progression of CKD in people. Experimentally-induced obesity in dogs has been shown to increase mean arterial pressure and plasma renin activity, alter renal function (glomerular hyperfiltration), and cause histologic changes including expansion of Bowman’s capsule, increased mesangial matrix, thickening of glomerular and tubular basement membranes, and increased cell proliferation in the glomerulus. Following weight loss, dogs had evidence of improved renal function with increased urine specific gravity (USG), decreased urine protein to creatinine ratio (UPC), and decreased levels of biomarkers of renal injury (homocysteine, cystatin, and clusterin). Creatinine was also decreased after weight loss, but this may be confounded by the loss of LBM in addition to fat mass. In this same study, 8 dogs had UPC > 0.5 prior to weight loss, while only 1 dog had UPC > 0.5 after weight loss. Another study comparing UPC in dogs with BCS 4-5/9 versus ≥6/9 found no statistical difference between groups. The researchers in this study were unable to separate out overweight versus obese dogs for additional statistical analysis due to the limited sample size. Overweight/obese dogs and cats with CKD must be assessed on an individual basis...
to achieve optimal body weight. Generally, the use of a therapeutic kidney diet will take precedence in dogs and cats with later stage CKD. In obese dogs and cats with early stage CKD (IRIS stage 1 ± 2), a weight loss plan may be considered only after a complete nutritional assessment and only if close monitoring is available. If progression of CKD is noted, active weight loss should be suspended and appropriate diet modifications should occur until further evaluation. Consultation with a board-certified veterinary nutritionist is recommended for obese patients with CKD.

**Cancer**

In people, increased body-mass index is associated with an increased risk of development and death from cancers including esophageal, thyroid, colon, kidney, endometrium, gallbladder, breast, and pancreas as well as development of leukemia, multiple myeloma, and non-Hodgkin lymphoma. Limited studies are available in veterinary medicine investigating overweight/obesity with cancer risk and outcomes. Some work suggests an increased risk in the development of mammary cancer and transitional cell carcinoma in dogs. In one study, the prevalence of overweight/obesity was slightly lower in dogs with cancer, although there was a higher prevalence of overweight/obesity in dogs with mammary cancers though this was not statically significant. Out of 100 dogs presenting to an oncology service at a veterinary teaching hospital, 26 were overweight and 29 obese based on BCS. Fewer data is available in cats with one study evaluating BCS and survival time finding feline cancer patients with a BCS <5 having a MST of 3.3 months compared to 16.7 months in cats with a BCS ≥ 5.

Before instituting a weight loss plan for an overweight/obese cancer patient, a complete nutritional assessment including cancer staging, evaluation of clinical signs, and determination of prognosis must be performed. Clinicians must decide if weight loss will provide a clinical benefit in light of the patient’s survival time. Weight loss to improve quality of life rather than achievement of ideal body weight may be appropriate for some animals such as an obese dog with osteosarcoma and OA now ambulating on 3 legs or an obese cat with lymphoma and diabetes mellitus receiving corticosteroids. Typically the author starts with a conservative caloric reduction to achieve slow rate of weight loss (0.5-1% body weight per week). Aggressive caloric restriction in a sick animal may contribute to preferential loss of LBM therefore serial assessment of muscle condition is also recommended. A high protein, high fat, and low carbohydrate diet is typically advocated in veterinary cancer patients to support LBM and protein metabolism as well as decrease the energy supply to neoplastic cells that are inefficient in oxidizing fat for energy. A diet with the above characteristics enriched in omega-3 fatty acids has been shown to prolong survival time is a subset of dogs with lymphoma compared to a similar diet unenriched in omega-3 fatty acids. Additional work is needed to investigate potential clinical benefits of high fat versus low fat diets in dogs and cats with cancer. Weight loss diets for dogs are generally low in fat to avoid increases in caloric density, but high in protein to support LBM. Some weight loss diets are formulated with higher amounts of omega-3 fatty acids which may be desired for reasons other than neoplasia including OA.

**Diabetes mellitus**

Diabetes mellitus (DM) in dogs is due to inadequate insulin production from immune-mediated destruction of beta cells or from pancreatitis. Feline DM occurs commonly due to peripheral insulin resistance as a result of excess adiposity and beta cell dysfunction. Although insulin resistance in dogs does exist, dogs will have an absolute requirement for insulin administration that diet alone will not correct. Insulin sensitivity in lean dogs was 58% greater than overweight dogs. Dogs with a body weight gain of approximately 43% had significantly higher basal insulinemia and insulin resistance. In contrast, cats can have insulin resistance at 10% over their lean body weight. Obese cats are up to 3.9 x as likely to have DM than cats with an ideal BCS. In dogs, the role of obesity in the development of DM is less clear although a relationship likely exists. Twenty percent of obese dogs in one study had criteria for obesity-related metabolic dysfunction. This criteria was modeled from human guidelines for diagnosis of metabolic syndrome, a risk factor in people for the development of Type II DM and cardiovascular disease. The progression of insulin resistance to the development of DM is not documented and further studies are needed to determine what these criteria in dogs mean for disease risk and outcomes.

Goals for managing an obese diabetic patient include eliminating clinical signs associated with hyperglycemia and glycosuria, avoiding hypoglycemia, improving patient body weight and condition, and maintaining the pet and owner’s quality of life. Initial recommendations for overweight and obese dogs should be weight maintenance until glycemic control is achieved. Once this is established, conservative weight management (0.5-1% body weight per week) can be instituted which may help to improve insulin sensitivity. A modest reduction in energy intake (decreasing caloric intake by 20%) with control of treat consumption (≤ 10% caloric intake) can be tried initially. Some dogs may require more or less caloric restriction for weight loss at follow-up. The use of a therapeutic weight loss diet is recommended for obese patients. These diets are typically high in protein (>30% metabolizable energy (ME)) with moderate to high amounts of total dietary fiber (>30g/1000kcal). Added soluble and insoluble fiber to a high carbohydrate diet (>50%ME) has been shown to improve glycemic control in dogs.

Weight loss is an important goal in overweight/obese cats with DM and can help achieve and maintain diabetic remission in conjunction with appropriate medical management. Every 1kg increase in body weight has been associated with a 30% decrease in
insulin sensitivity which was normalized with weight loss. The timing of weight management however should be delayed in an overtly ill cat until stabilized. Diabetic remission typically occurs in cats during the first 3-4 months of therapy. A recent survey of veterinarians in the Southeastern United States revealed that 97% (87/90) of vets always or usually recommended dietary management at the time of DM diagnosis and 93% of respondents recommended diets marked as low carbohydrate (LC). A low carbohydrate (<15% ME) diet may be advantageous for cats with DM. Cats fed a LC-low fiber diet had a significantly higher remission rate of 68% compared to cats fed a moderate carbohydrate-high fiber diet (41%). Cats also appeared to be better regulated on the LC diet. The author typically recommends canned, low carbohydrate diets for weight loss in diabetic cats. In obese cats that only consume dry food, a low calorie density, high fiber weight loss diet is typically preferred to improve owner compliance. Both therapeutic low carbohydrate and high fiber weight loss diets contain moderate to high amounts of protein (>35% ME) to support LBM.

References
Popularity of raw meat based diets
In the United States and Australia, raw food or bones were reported to be fed at least daily as part of the main meal in 9.6% of cats and 16.2% of dogs. Another 0.9% of cats and 7.4% of dogs received raw meat or bones as a treat or snack at least once weekly. Interestingly, these survey results were collected prior to the melamine pet food recall of 2007. It is reasonable to hypothesize that feeding practices amongst pet owners has changed over the last decade. Raw freeze-dried pet food retail sales increased 64% in 2014 from US$25 million to US$40 million while raw frozen pet food increased 32% from US$52 million to US$69 million. Given the increasing popularity of commercial raw pet food products, it is likely veterinary professionals are encountering pets fed a raw meat-based diet (RMBD) or treat commonly in practice.

Nutritional adequacy of raw meat based diets
The nutritional adequacy of commercial RMBDs can be determined based on the Association of American Feed Control Officials (AAFCO) guidelines for formulation or by feed trial. RMBDs labeled for intermittent or supplemental feeding are not nutritionally complete and balanced. The use of whole prey diets may be used by some owners feeding a RMBD. Whole 1-3 day old chicks and adult ground chicken were found to meet macronutrient requirements but were deficient in some mineral requirements, including manganese, copper, potassium and sodium compared to AAFCO requirements for adult cats. These diets were not evaluated for vitamin content. Research kittens consuming a diet solely of whole raw ground rabbit developed dilated cardiomyopathy secondary to taurine deficiency resulting in the death of one kitten. The diet of a prey animals (e.g., chicks, mice, or rabbits) sold for pet consumption may also influence their nutrient profile. Home-prepared diets, raw or cooked, can be obtained by owners through a number of sources including the Internet, pet magazines, and books written by veterinarians and non-veterinarians with varying levels of nutrition training. Several studies have evaluated the nutritional adequacy of home-prepared diets in companion animals finding numerous and significant nutritional imbalances. Evaluation of 200 published home-prepared recipes for adult maintenance in dogs written by veterinarians (64.5%) and non-veterinarians (35.5%) revealed at least one essential nutrient deficiency according to National Research Council or AAFCO guidelines in the majority of diets (95%), and 83.5% of recipes has multiple deficiencies.

Analysis of 77 home prepared bone and raw food rations for dogs in Germany found that 76% had one or more nutritional imbalances. A home prepared raw food diet formulated by a now board-certified veterinary nutritionists and commercial raw food diet were determined to be nutritionally adequate based on a 10 week AAFCO feeding trial in kittens.

Animals may develop clinical signs associated with nutrient deficiency or toxicity when consuming an unbalanced diet. Nutritional secondary hyperparathyroidism has been reported in both cats and dogs consuming unbalanced home prepared diets manifesting as spontaneous fracture, muscle twitching, seizures, and limb deformities. A dog consuming an unbalanced home prepared diet with deficiencies including calcium, phosphorus, and vitamin D developed tetanic seizures and hyperthermia during evaluation of bilateral humeral osteochondritis dissecans. Other reported nutrient imbalances resulting in clinical signs include metabolic osteopathy with extensive new bone formation from hypervitaminosis A in a cat consuming raw pork liver, pansteatitis in cats secondary to vitamin E deficiency while consuming a high poly-unsaturated fat raw diet, and hyperthyroidism in dogs consuming raw beef gullet with thyroid tissue.

Potential benefits of raw meat based diets
Owners may choose to feed a RMBD to their pets due to anecdotal health benefits and to provide a more natural or ancestral diet. Many of these benefits remain unproven, although the body of scientific evidence surrounding RMBDs is limited. Cats and wolves in the wild will consume a variety of prey to support survival and reproduction, and some owners may choose to mimic this diet closely. However, this same type of diet may not be optimal for domestic animals expected to live long and healthy lives primarily indoors and reproductively altered.

Some owners may report a benefit of smaller stool volumes, less fecal odor, and improved gastrointestinal health in cats and dogs fed home-prepared or commercial RMBDs. Several studies have demonstrated a high digestibility of RMBDs fed to both exotic and domestic cats when compared to extruded diets. Digestibility of dry matter, organic matter, crude protein, and gross energy was significantly higher in a commercial and homemade raw food diet compared with a canned heat-processed diet in domestic kittens. Cats consuming whole ground rabbits had significant improvements in stool quality compared to cats consuming a commercial diet. Another study found no difference on total tract energy and macronutrient digestibility in cats fed a commercial raw beef-based diet or the same diet cooked in the microwave to at least 160°F. Significant differences have been noted in the fecal microbiota of cats fed a
raw diet consisting of 1-3 day old chicks compared to a chicken-based extruded diet although these differences could not be only attributed to the raw vs extruded nature of the diets as the nutrient composition differed.21

Heat processing of pet foods can result in Maillard reactions responsible for the browning of foods when heated. This reaction causes decreased bioavailability of lysine and formation of Millard reaction products (MRPs) and advanced glycation end-products (AGEs) which may have harmful biological effects.22 A study found that dog and cats had an average daily intake of one MRP 122 and 38 times higher respectively than the average daily intake for adult humans.23 Further studies are needed to investigate the long-term health implications of this in dogs and cats. Heat processing of meat proteins can also negatively impact the creatine concentration of the dry dog food and meat/meat and bone meal, although the benefits of a creatine-rich diet is dogs is unknown.24 Researchers suggest this may also be due to Maillard reactions.

The infectious potential of raw meat based diets
Commercial and home prepared RMBDs may be contaminated with potentially harmful pathogens. The concern for public and animal health has led organizations such as the American Veterinary Medical Association and the American Animal Hospital Association to discourage the use of RMBDs.25-26 Studies have documented the presence of bacterial contamination in both commercial and home prepared RMBDs. A study by the United States Food and Drug Administration (FDA) in 2011-2012 found that out of 196 commercial RMBDs, 7.7% were positive for Salmonella spp., and 16% positive for Listeria monocytogenes.27 A study examining 25 commercial raw canine and feline RMBDs found contamination with Clostridium perfringens (20%), Escherichia coli (64%), Salmonella spp. (20%), Clostridium difficile (4%), and Staphylococcus aureus (4%) based on culture.28 Twenty-three percent of RMBDs for dogs contained extended spectrum cephalosporin-resistant E.coli.29 Salmonella in raw meats sold for human consumption has reported rates of 44% in chicken and 4% in beef and pork, therefore it is not surprising to find contamination in raw pet food.30 While the presence of Salmonella is allowed on meat for human consumption by the USDA, the FDA maintains a zero-tolerance policy on Salmonella in pet foods.31 The FDA lists 26 dog and cat food recalls from 2015 (expanded recalls are counted as 1 with the original recall), with the vast majority from RMBD.32 Eleven treat products were recalled, 10 of these from Salmonella contamination. Seven of the recalled treats due to Salmonella contamination were beef products including beef gullet, tripe, trachea, jerky, and bone. Twelve of 15 complete and balanced recalled commercial diets were RMBD. Seven RMBDs were recalled for Salmonella, 1 for Listeria monocytogenes, 3 for both Salmonella and Listeria, and 1 for thiamine deficiency.

Once fed a RMBD containing Salmonella, cats and dogs have been shown to shed the bacteria in their feces.10,34,35 Kittens fed a home prepared and commercial raw food diet had significantly higher globulin levels and red blood cell microcytosis compared to kittens consuming canned heat-processed diet.10 Positive fecal Salmonella Heidelberg and Clostridium difficile toxin was noted in the raw diet groups. Lab work changes were purposed to be associated with known enteropathogenic exposure. Seven of 16 dogs fed a known Salmonella-contaminated single meal shed the bacteria in their feces within 7 days of exposure.35 None of the dogs fed the Salmonella-contaminated meal experienced clinical signs. Feeding a commercial or home prepared RMBD is also a risk factor for antimicrobial-resistant Salmonella spp. and E. coli in the feces of dogs.36 In addition to the zoontic potential, animals may be clinical affected. Three of twelve cats fed a raw food diet of whole or ground 1-3 day old chicks developed clinical salmonellosis (anorexia and diarrhea).37 Significant differences in the fecal microbiome were noted in the symptomatic cats including the detection of other potentially pathogenic bacteria and increased proportions of other potentially pathogenic bacteria. Salmonella bacteriuria was reported in a cat with lower-urinary tract signs fed a Salmonella-contaminated uncooked granular diet by a company that manufactures RMBDs.38 Septicemic salmonellosis in two cats resulting in death after being fed a diet containing uncooked beef has also been reported.39 In one of the cases, Salmonella Newport was isolated and found to be identical to the bacteria isolated in the diet.

Clinical recommendations
The potential for human and animal disease with commercial and home-prepared RMBDs is well documented. Food utensils, feeding bowls, litter boxes, the RMBD, feces and animals with bacteria present in their mouths or on their coat are all sources of potential pathogen exposure for people. The risk of pathogen contamination is particularly a concern among elderly, young, pregnant, lactating, or immunocompromised pets and people. Policies to protect veterinary staff and hospitalized animals from pathogens shed in feces are recommended when treating an animal consuming a RMBD. The FDA also has resources on safe handling tips for pet food and treats and recommendations to owners who choose to feed a RMBD.40 Consultation with a board-certified veterinary nutritionists or individual with similar training for owners wanting to home prepared any diet, cooked or raw, is recommended.

References

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Key points

- Lymphoma is a common canine cancer and is a systemic disease that requires chemotherapy in almost all cases.
- The majority of dogs achieve a complete remission with chemotherapy (approximately 80%). Higher remission rates are typical with CHOP multi-agent chemotherapy protocols.
- Early accurate diagnostics and careful staging are keys to proper clinical decision making.
- To determine the best protocol for a patient and owners, it is important to understand efficacy of the various protocols, the potential toxicities, and prognostic factors.
- Dogs treated with chemotherapy live significantly longer than untreated dogs, and chemotherapy is generally well-tolerated in most dogs. Only a minority develop significant toxicity.

Biology of lymphoma

Lymphoma is a collection of cancers arising from the malignant transformation of lymphocytes. Even though lymphoma is clinically a diverse group of neoplasms, the common origin is the lymphoreticular cells. Lymphoma is one of the most common canine cancers, accounting for 7-24% of all canine tumors and 85% of hematopoietic tumors. Dogs of any age, gender, and breed can be affected with lymphoma. Affected dogs are typically middle aged to older dog.

Anatomic classification

Multicentric (PLN) is the most common form, accounting for 80% of lymphomas. Most dogs are typically asymptomatic, and 20-40% are clinical (substage b) with anorexia, lethargy, fever, V/D, weight loss, melena

Gastrointestinal (GI) involvement is less common accounting for only 5-7% of LSA cases. It is more common in males, and Boxers and Shar-peis over-represented. Weight loss, anorexia, panhypoproteinemia, malabsorption are common. It typically involve multifocal & diffuse of submucosa & lamina propria layers of small intestine. Phenotypically, GI LSA is typically T-cell. Histologically, can be challenging to distinguish from lymphoplasmacytic enteritis (LPE). In GI LSA, lymphocytic and plasmacytic inflammation can be adjacent or distant to the neoplastic population of cells. There is also the question of whether LPE a pre-lymphoma change?

Mediastinal forms are also less common, accounting for only 5% of LSA cases. It typically involves the cranial mediastinal LN and/or thymus, but 20% multicentric LSA have cranial mediastinal LN involvement. Hypercalcemia is most common with this form. In one study of 37 hypercalcemic dogs, 16 dogs (43%) had mediastinal lymphoma. Phenotypically, mediastinal LSA is typically T-cell

Cutaneous LSA can be a solitary lesion or generalized lesions, and may have oral mucosa lesions, +/- extracutaneous involvement of LN, liver, spleen, BM. This form is referred to epitheliotrophic form or as mycosis fungoides. It is more common in dogs than cats. The immunophenotype is typically T-cell (CD8+). In contrast, B-cell cutaneous LSA spares the epidermis and papillary dermis and affects the deeper dermal layers.

Clinical appearance

Historic findings

The most common complaint is generalized lymphadenomegaly. Owners commonly report that lymph node size is rapidly increasing – over days to 1 to 3 weeks. In the early stages, dogs appear healthy and are not showing clinical signs. When present, clinical signs tend to be nonspecific and include vomiting, diarrhea, melena, anorexia, fever, and weight loss (substage b).

Common examination findings

Lymphoma can be indolent or aggressive, solitary or multicentric, or node-based or associated with any organ. Non-painful generalized lymphadenomegaly is most common physical exam finding. Multicentric lymphoma involving the peripheral lymph nodes is most common, accounting for 80% of patients.

Most dogs are “healthy” substage a. T-cell dogs tend to be sick (b). In dogs, multicentric LSA is generally the NHL (non-Hodgkin’s LSA) form. Hepatosplenomegaly is common. Diffuse pulmonary infiltration has been reported in 27-34% based on CXR but on BAL, lung involvement may be higher. The lack of generalized lymphadenomegaly does not eliminate the possibility of lymphoma, as some dogs will have internal involvement only (i.e. hepatosplenic form, GI). Another scenario that can lead to confusion is hypercalcemia, often without peripheral lymphadenomegaly so lymphoma is not suspected.
Preliminary diagnosis

Cytology

Confirmation of lymphoma starts with fine needle aspirate of an affected lymph node. Cytology is minimally invasive, less expensive than biopsy, and typically provides rapid results, in 1 to 2 days. Cytology reveals monomorphic abnormal lymphocyte populations. Cytology does not provide complete classification, grading, or phenotype. Avoid reactive LN, such as the mandibular LN.

Diagnostic work up

The minimum tests required for treatment are cytological confirmation (lymph node or affected organ), CBC, chemistry panel and urinalysis. The next diagnostic I encourage owners to submit is phenotyping to determine B vs T-cell subtype. Phenotyping is typically determined with immunocytochemistry from aspirates, immunohistochemistry from biopsy, or flow cytometry or PARR from aspirates. If there is a peripheral lymphocytosis on CBC (stage V), flow cytometry can be submitted on a whole blood sample to determine phenotype. Phenotype is the best independent prognostic factor; prognosis is worse with T-cell than B-cell.

Lymph node biopsy is ideally performed for histologic grading but is often only collected when cytology was inconclusive. Baseline chest radiographs and abdominal ultrasound are recommended for staging purposes to determine extent of disease. While stage is prognostic, I also find it valuable to have these baseline imaging tests to be able to compare treatment response or progression. Bone marrow cytology is also considered part of the basic staging but it is often not done is the majority of cases, factoring in the additional cost and sedation for most cases. Bone marrow cytology is of less clinical utility in most cases. However, if there are cytopenias and/or a lymphocytosis, a bone marrow should be considered to identify bone marrow involvement.

To stage or not to stage?

Complete lymphoma staging includes lymph node cytological confirmation, CBC, chemistry panel, urinalysis, lymph node histology, urinalysis, thoracic radiographs, abdominal ultrasound, bone marrow cytology and phenotyping. These tests are useful and informative, as they provide prognostic factors and a baseline for a patient’s response. These tests can also help determine if there large tumor burden and risk for acute tumor lysis syndrome with induction chemotherapy. Still, we must consider the owner’s financial issues. While it is ideal to perform all the tests, we can also consider each test on a case by case basis and help the owner make an educated decision. We can treat without but review pros and cons with the owner and let owner make educated decision and maybe choose more important tests for that dog.

Histology

NIH WF & Kiel System most useful, and both describe architecture and cell morphology, including mitotic index, cell size, and cell shape.

Why do I care about histology? It’s prognostic! Positive: Low grade LSA, Including mantle-zone, follicular, T-cell. But low grade LSA may only partially respond to chemotherapy and is often incurable. Negative: intermediate and high grade LSA BUT have a high mitotic rate & are more likely to completely respond to chemotherapy.

Phenotype

60-80% of LSA are B-cell, and this is an important positive predictor, associated with higher rate of CR, longer remission, increased ST, and most high grade are B-cell. Breed prevalence with B-cell includes Cocker and Dobies. Goldens have equal B and T-cell. 10-38% of LSA are T-cell, and this is an important negative predictor, associated with lower rate of CR, shorter remission, shorter ST, and tends to be associated with hypercalcemia. Boxers are over-represented.

Flow cytometry

(FCM) involves staining live cells with labeled antibodies that bind to cell surface proteins. These live cells are suspended in liquid (saline, tissue culture media). Different types of lymphocytes express different proteins. Flow cytometer tells us how many cells of each type are present and can determine the lineage of the cells present. Flow could not identify LSA in 30% of newly diagnosed cases

PARR

PCR Antigen Receptor Rearrangement is a polymerase chain reaction (PCR) assay that amplifies DNA with PCR primers in the dog or cat. It tells us if the majority of cells in the sample are clonal: same original clone - most consistent with neoplasia, or from multiple clones/polyclonal - lymphoid proliferation - most consistent with a reactive process, It is useful to determine: whether lymphoid neoplasia, phenotype (B vs. T), and to monitor for MRD in treated patients, It must be interpreted with history, clinical signs, cytology, flow cytometry, IHC.

For sensitivity & specificity, both are ~90% in dogs, and it is more sensitive for circulating cells > blood, bone marrow. In cats, it is better for T cell (89%, 80%) vs B-cells (60%, 70%). FCM and PARR are NOT useful for neutrophilia to r/o chronic myelogenous leukemia, when hypercalcemia is only sign, not helpful on LN, fluid, etc., or as a screening test for healthy dogs and cats without clinical signs.

Prognostic factors

There are many prognostic factors, but the more significant predictors include:

- Phenotype: B-cell is better than T-cell. 60-80% are B-cell and this is associated with higher rated of CR, longer remission rates, and increased ST. Most high grade LSA are B-cell.
- Histologic grade: high grade has better CR rate than low grade, but low grade often has comparable survival times with less intensive chemotherapy protocols.
- Administration of prednisone prior to chemotherapy is a negative predictor
- Substage: clinically healthy dogs tend to do better than sick dogs
- Higher stage (stage IV and V) tend to do worse than lower stage (I to III)
- Hypercalcemia: negative predictor due to association with T-cell phenotype
- Mediastinal mass: negative predictor due to association with T-cell phenotype

Remember, prognostic factors cannot predict an individual’s response, and lymphoma is typically treatable and rewarding to treat for the patient, owner and the veterinarian.

Treatment modalities
Treatment pearls
Chemotherapy is the mainstay of therapy to promote a rapid, durable and complete remission (CR), while maintaining a good to excellent quality of life even during chemotherapy. Complete remission is complete disappearance of all detectable lymphoma and resolution of clinical signs. Lymphoma is typically rewarding to treat with high response rates, and most dogs tolerate chemotherapy quite well.

Treatment: Chemotherapy
The goal of therapy is to achieve a complete remission and a good to excellent quality of life. Dogs that respond and achieve CR are usually free of clinical signs of lymphoma and live longer and live well. Only a minority develops significant toxicity or do not respond to therapy. Most patients are treated on an outpatient basis. Newly diagnosed lymphoma patients that are sick (stage substage b), dehydrated, and have a large tumor burden (advanced stages) are at increased risk for acute tumor lysis syndrome with induction chemotherapy. In such cases, the dogs should be admitted for IV fluid therapy, supportive care, and intensive monitoring prior to chemo and for 24 to 72 hours after.

Combination chemotherapy provides improved remission rates and duration in comparison to single agent protocols. Multi-agent CHOP protocols are the most successful, with complete remission rates of > 80% and remission durations of typically 6-11 months. Median survival times (MST) are 1 year when followed by rescue protocol, and 25% of dogs are long term survivors > 2 years. There are numerous CHOP protocols that vary in drug dosages, scheduling, and dose intensity. The UW-Madison protocol is often recommended for owners choosing a combination protocol for its high complete remission rates, higher remission duration, and lower morbidity and mortality rates. Commonly used UW protocols are the 25 and 19 week protocols

Multi-agent CHOP protocols typically combine vincristine, cyclophosphamide, doxorubicin and prednisone. Recent studies suggest the inclusion of l-asparaginase at induction does not significantly impact remission duration or survival times and can be omitted and saved for the rescue protocol.

Additionally recent studies suggest there is no survival benefit of maintenance phase. Most current protocols are discontinuous without a chronic maintenance phase and provide comparable remission durations. It is thought the period without chemotherapy may lead to greater responsiveness at relapse by lack of selection of resistant cells.

For some clients, alternative protocols are elected over the multi-agent CHOP protocol due to budget, toxicity profile on par with clients’ willingness to assume risks of chemo, and schedule and time commitment. In some cases, it is to avoid drugs that target a patient’s weakness or concurrent illness. For example Lomustine is avoided with liver dysfunction and doxorubicin can cause cardiotoxicity so should be used cautiously in dogs with some pre-existing cardiac disease.

Alternative chemotherapy protocols include COP (vincristine, cyclophosphamide, and prednisone), single agent doxorubicin for B-cell lymphoma, and single agent Lomustine for T-cell lymphoma. These protocols generally have lower response rates ranging from 50-80% and shorter remission durations of 6 to 7 months.

New therapies for lymphoma include monoclonal antibodies and a lymphoma vaccine. It is hopeful these new therapies will increase survival times.

If chemotherapy is declined
If chemotherapy is declined, another option is single agent steroids. Typical response rates are 50% with duration of 2 to 3 months. Prednisone should not be started prior to chemotherapy since it may decrease response rate to chemotherapy started after the steroids. Pre-chemotherapy steroids use is associated with shorter remission and survival times due to induction of multi-drug resistance. If staging tests are done after prednisone is started, higher stage patients may appear to be lower stage (down-stage). Without chemotherapy the prognosis for lymphoma is poor, with MST of 1 month.

Relapse
The majority of lymphoma patients relapse as there is the emergence of tumor clones that are more resistant to chemotherapy, or survival-of-the-fittest lymphoma cells. These MDR (multi-drug-resistance) clones are more likely to express MDR-1 gene that encodes for protein transmembrane pump associated with multidrug resistance. Other reasons for relapse include inadequate
chemotherapy dosing, inadequate chemotherapy frequency, or failure to achieve high chemotherapy concentrations at certain sites, such as the CNS.

When a patient relapses, I recommend reintroducing the initial protocol if it was successful, meaning the expected remission duration was achieved. For example, if a dog relapses one month after completing a CHOP protocol I will not recommend restarting front-line chemotherapy. However if the dog was off chemotherapy for 4-5 months with a 1st remission of 9-10 months, I will recommend restarting the induction protocol as re-induction rates of 90% can be expected. Remember there is a cumulative dose of doxorubicin, so doxorubicin is typically replaced after a total of 6 doses. When a dog no longer responds front-line chemotherapy, rescue protocols are recommended. There is decreased likelihood of response (30-50%) and shorter remission durations, typically half the length of the initial remission. Still some patients experience long-term re-inductions. Some commonly used protocols include MOPP, doxorubicin or mitoxantrone with DTIC, Lomustine/l-asparaginase/prednisone, and single agent Lomustine.

**Other treatment options**

Other treatment options include localized radiation for local disease, such as nasal or CNS lymphoma. Palliative radiation can be used for bulky localized disease such as rectal, bone or mandibular lymph nodes. The addition of half body radiation to multi-agent chemotherapy improved ST and remission duration in some studies, but there is increased costs and toxicity to balance. Whole body radiation is used in combination with bone marrow transplants.

More recently, monoclonal antibodies have been introduced as targeted therapy for both T- and B-cell canine lymphoma, but efficacy and administration schedule are still being worked out. Canine remission times on CHOP have plateaued at about 9 months.

In human monoclonal antibodies are standard of care. Before rituximab, results of CHOP-based chemotherapy plateaued in human medicine. Since its launch in 1997 it is the standard of care for non-Hodgkin’s lymphoma in humans and the addition of rituximab to standard CHOP has increased overall survival by 55%.

**Recheck frequency**

After completion of chemotherapy, I recommend monthly rechecks to evaluate for relapse, especially at time of expected relapse depending on the protocol elected. In addition to physical exam, monitoring with lymph node palpation, cytology, chest radiographs, ultrasound, and advanced diagnostics can be helpful. Recently blood tests have been evaluated to look for molecular markers to detect early relapse before clinically detectable. The Canine Lymphoma Blood Test (cLBT, Avacta) has recently been shown to detect relapse earlier, and the lowest score during treatment was prognostic for ST and TTP. (Alexandrakis, 2014).

**Overall**

Lymphoma is one of the most successfully treated cancers in dogs, and many patients with lymphoma outlive animals with other noncancerous diseases such as kidney, heart, and liver disease. Dogs treated with chemotherapy live significantly longer than untreated dogs, and chemotherapy is generally well-tolerated in most dogs.

**Additional resources**


Lymphoma (LSA) is one of the most commonly occurring cancers in cats. Lymphoma is a systemic disease that requires chemotherapy in almost all cases. Outcomes for treated cats are less predictable than dogs, but cats tend to tolerate chemotherapy better than dogs. Treated cats live longer, and chemotherapy is generally well-tolerated.

**Biology of lymphoma**

Lymphoma is a collection of cancers arising from the malignant transformation of lymphocytes and is a diverse group of neoplasms with the common origin of the lymphoreticular cells. In contrast to dogs, feline lymphoma most commonly affects the gastrointestinal (GI) tract.

Lymphoma is one of the most common feline cancers, reported at 30% of all cancers. In the FeLV era from the 1960-1980s, lymphoma accounted for 50-90% of hematopoietic tumors. However, there was a shift after the 1990s, also called the post FeLV-era. With the aid of FeLV diagnostic assays and elimination regimens in 1970s and 1980s, there was a dramatic decline in FeLV-associated LSA. Still lymphoma prevalence is increasing, especially the alimentary form.

**Etiology**

**Viral**

In the FeLV era of the 1960-1980s, two-thirds of lymphoma was associated with FeLV antigen. FeLV-positive cats had a 62-fold increased risk. This form was predominantly seen in younger cats, was the mediastinal form, T-cell, and the virus had a direct role in tumorigenesis. Being FIV-positive increased lymphoma incidence by 5-6x. In contrast to FeLV, FIV has an indirect role secondary to immunosuppressive effects and is associated with B-cell and the extranodal form. Cats that are both FeLV and FIV positive have an increased risk of 77-fold.

**Immunosuppression**

FIV has an indirect role with lymphoma secondary to immunosuppressive effects. Ten percent of feline renal transplants develop lymphoma following transplant and associated immunosuppressive therapy.

**Environmental**

Environmental tobacco smoke (ETS) has been reported to increase the risk of LSA by 2.5 to 3.2-fold.

**Genetic and molecular factors**

The predisposition of oriental breeds suggests a heritable risk, but this is still being investigated.

**Chronic inflammation**

While definitive proof is lacking, there is growing evidence of the link with chronic inflammation and lymphoma, in particular with and intestinal LSA. This has been as area of interest with IBD and GI LSA.

**Diet and GI LSA**

While definitive proof is lacking, the diet changes over last 20 years in response to diseases such as urinary tract and the increase in GI LSA has led to the suggestion of a link, but more studies are needed.

**Signalment**

Lymphoma can occur in cats of any age, any sex, any breed. The median age is 11 years, and a male predisposition is reported and intact females are at decreased risk, suggesting a protective benefit of sex hormones. Overrepresented breeds include Siamese cats, Manx, and Burmese. Signalment varies with anatomic site and FeLV status.

**Pathology and behavior**

For the alimentary/GI from, the LSA typically involved the intestines alone or intestines, lymph nodes (LN), and liver. In the GI tract, it can be solitary vs diffuse. 55% of GI tumors are LSA. Siamese are at increased risk. The GI form typically occurs in aged cats of 12 to 13 years old. The small intestines are four times more affected than the large intestines. Enteropathy-associated T-cell LSA (EATL) has 2 forms. EATL Type I is intermediate to large B-cells, high grade, lymphoblastic lymphoma. This form often has a palpable mass. EATL Type II is called small cell, low grade, lymphocytic lymphoma. This form is more diffuse throughout the GIT and T-cell is more common.
Clinical appearance
Alimentary/GI
For low grade small cell LSA, clinical signs include weight loss (83-100%), V/D (73-88%), anorexia (66%), and icterus (7%). 70% have abnormal palpation on exam, either thickened GI or a palpable mass 33%. The history is usually chronic over several months, with a median 6 months.
For high grade LSA, the clinical signs are similar but icterus is more common and the onset is more rapid – days to weeks. A palpable mass is common. Rarely the cat will present with acute abdomen due to obstruction or perforation.

Diagnosis and staging
Basic diagnostics include CBC, chemistry panel, and UA. For the GI forms, 23% have panhypoproteinemia and 76% are anemic. Test for FeLV/FIV status. Diagnosis typically made with cytology or histology of a LN or organ. Cytology may be inconclusive and be reported as benign hyperplastic and reactive, and histology will be needed. Other diagnostics may include abdominal ultrasound (AUS) and chest radiographs. Bone marrow cytology may be recommended especially for cases with anemia, leukopenia, or cellular atypia. Phenotype can be determined with PARR 80% sensitive or flow cytometry.
For high grade large cell (EATL type I), the diagnosis is typically more straightforward with GI masses, enlarged mesenteric LN, or liver involvement. The diagnosis is typically made with abdominal ultrasound and cytology/histology. Surgery is less commonly needed.
For low grade small cell (EATL type II), intestinal thickening is often modest or absent and similar to IBD. Cytology alone is often insufficient and will come back as benign hyperplasia. To confirm the diagnosis, AUS and histopathology are typically needed, and may require phenotype and clonality.
It can be challenging to distinguish low grade vs IBD with abdominal ultrasound. With low grade GI LSA, 60-90% have an abnormal AUS with 50-70% diffuse SI thickening, predominantly muscularis propria and submucosa layers. Mesenteric lymph nodes are abnormal in 45-80%. Focal GI masses are uncommon. For IBD, 10-50% have diffuse SI thickening and mucosal thickening more common. The incidence of mesenteric LN lymph nodes is lower at 15-20%, and other abnormal organs are typically normal.
Cytology is rarely useful for distinguishing low grade GI LSA vs IBD. The debate rages on regarding endoscopy vs full thickness biopsy (laparotomy vs laparoscopy). On histopathology, lymphoma typically has lymphoid infiltration beyond mucosal layer, epitheliotrophic, heterogeneity, and lymphocyte nuclear size consistent with malignant. If diagnosis is still equivocal, phenotype or PARR is recommended.

Feline chronic small bowel disease (CSBD)
This study highlights that CSBD often is often considered normal by cat owners. Excuses include: “He just eats fast”, “She is a nervous cat”, “He has a sensitive stomach”, “She gets hairballs”, “He’s always done this.” CSBD includes IBD and enteropathy-associated T-cell LSA (EATL) type 2. EATL type 2 most common infiltrative GI LSA in cats, and treatment is different than IBD.
In this study, the authors looked at the association of clinical signs and disease in 100 cats that had an AUS of small bowel ≥0.28 cm in ≥2 locations. These cats had ≥1: vomiting ≥2x/month for at least 3 months, several weeks of small bowel diarrhea, and weight loss > 0.5 kg in last 6 months. Interestingly, 26 cats were getting wellness exam. 65 cats did not have surgery and were excluded. Clinical signs included weight loss 70%, vomiting ≥2x 61%, diarrhea 11%, and V/D 13%. 92% had at least 1 AUS measurement ≥0.3 cm, 8 cats 0.29-0.29 cm, and 76 cats 1 measurement <0.28 cm. 99 of 100 had cats had IBD or LSA. Only 1 cat had normal histology. 49% had IBD/chronic enteritis. 46% had LSA (n=44 EATL type2). Cats <8 years old had enteritis, and cats > 8 years old enteritis or cancer. The 1 normal cat was 5 years old.
Cats with GI clinical signs are common and should undergo diagnostics. Do not let clients make excuses, and get a good history. Chronic vomiting is often considered normal, but vomiting is not normal! Clinical signs should trigger abdominal ultrasound. One of the common excuses is vomiting hairballs is normal. Is vomiting hairballs is normal? Does chronic small bowel disease slow bowel movement and predispose to formation?

Treatment
Treatment: Dogs vs cats
These are less feline data than for canine LSA. Papers often lump together small number of cases of multiple subtypes of various anatomic, phenotype and histologic grades. Outcomes are less predictable in cate and there is greater variation in histologic type and anatomic location in cats. But cats tolerate chemotherapy well and better than dogs. Febrile neutropenia is rare. Most owners happy they chose to treat and the QOL improves.

Which protocol?
For intermediate and high grade/EATL I, there is an overall response of 50-80%, a median remission of 4 months, and a median survival 6 months. Cats that achieve a complete remission have a MST of 1 year. I typically recommend a CHOP multi-agent protocol
such as the UW 25 week protocol. When using doxorubicin in cats, I use a lower dose (1 mg/kg). Cardiac toxicity is not clinical problem in cats in contrast to dogs, and renal function (BUN, Cr, USG) should be monitored in cats when giving doxorubicin. In dogs, data supports shorter maintenance-free protocol, but there is no data in cats, and some cats may need chronic chemotherapy.

An alternative protocol is the COP protocol with reported complete remissions of 50-70%. This is commonly used in used in Europe with similar results to CHOP in 1 study. While the protocol requires less frequent visits, it is a longer 1 year protocol. Other studies support the addition of doxorubicin to COP for durable responses.

For single agent options, Lomustine can be given at 50-60 mg/m2 every 4-6 weeks, which is given at a lower dose and less frequently than dogs. Single agent doxorubicin is cats is less successful with complete remission rates of <50%.

**For low Grade/EATL type II**, less aggressive chemotherapy protocols are typically used. Oral chlorambucil (Leukeran®) can be dose with pulse dosing (20mg/m² every 2 weeks or 15 mg/m² for 4 days every 3 weeks) or with chronic dose (>4 kg start @ 2 mg PO q 2 day, maintenance q 3 days; <4 kg start @2 mg PO q 3 day, maintenance q 4 days). For cats I prefer prednisolone, typically at 1 - 2 mg/kg orally daily and reduce to 0.5 to 1 mg/kg daily. In some cases, prednisolone may be discontinued.

For relapsed cases, cyclophosphamide, Lomustine, and vinblastine are recommended. For severe or refractory cases, I will used CHOP or COP protocols

**Nutrition for EATL type II**

With evidence of role of inflammation and many have concurrent IBD, there is thought to consider transition to a novel protein diet and add probiotics. I also recommend running B12 levels, and supplementing as indicated

**Prognostic factors**

The prognosis and response in cats is more variable than in canine lymphoma. Prognostic factors include anatomic location, achieving a CR, FeLV status, substage, and a multi-agent protocol (CHOP vs COP?). Factors that are NOT prognostic in cats include stage and immunophenotype, age, weight, gender, and FIV.

For GI forms, the prognosis is overall extremely variable. For EATL type I, response rates are 50-75%, median remission duration is 4-6 months, and expected survival is 6-8 months. 15-25% can live 1-2 years. For EATL type II, remission is generally defined as improvement or resolution of clinical signs,

And 70%-85% will respond for a median survival time of >2 years.

**References**


Why is oncology important?
The statistics are overwhelming. One in 3 dogs of any age will get cancer, and 50% of dogs over 10 years old will be affected by some tumor. Cancer is the number one cause of canine death. Many pets are considered important family members, and owners have increasing expectations. They want the same standard of care, highest quality medical care, compassionate care, and respectful communication. But when the pet is sick and has cancer, the human animal bond becomes stressed and fragile, making communication between the veterinarian and the owner more challenging.

Cancer communication challenges
Veterinarian/client communication is critical to optimal patient care. Yet there is a lack of guidelines and training to help veterinarians and clients broach difficult conversations about prognosis, treatment and palliative care options for pets diagnosed with cancer. As veterinarians and oncologists, we need clinical guidance to help initiate these conversations and better integrate both definitive and palliative therapy into our oncology practice.

Cancer communication training varies with regard to content, duration and methods. There is often a skills gap between veterinary school curriculum content and actual skills to be successful in practice. As a result, many veterinarians feel unprepared for difficult conversations about cancer.

Since the cancer diagnosis is typically made by the primary care veterinarian, The primary care veterinarians often have the more difficult job compared to the oncologist. The primary care veterinarians have the relationship with the client. On the other hand, when the client visits the oncologist, the owner usually knows cancer is the diagnosis, and the focus is treatment options and decision making, but there is no pre-existing relationship, familiarity, or trust.

Many barriers currently prevent veterinarians and clients from engaging in cancer conversations and optimal cancer care planning. To address barriers to advanced cancer care planning, we must first identify the challenges.

For the veterinarian, challenges may include general discomfort in talking about cancer and death, lack of training, shortness of time, practice culture, feelings of responsibility for cancer or a late diagnosis, perception of failure, unease with death and dying, uncertainty of outcome, impact on relationship with client, and the worry about patient quality of life (QOL), about client response, about costs, and about the veterinarian’s own response.

For the client, conversations are challenging as clients are often emotional and dealing with their feelings of self-blame, guilt, anxiety, fear, and frustration. There is the unease with death and dying, anticipatory grief, and concerns about the effect on human-animal bond. The client is also concerned for the pet’s QOL, the costs, the time required to treat the cancer.

How do you give your information?
The first method is called Data Dump, and is often nick-named the Shot-put technique. The oncologist does most of the talking like a monologue, and the client is more passive. The intent is on delivery of information, but it is often too much information for the client to absorb and too challenging to receive the message. One way to improve this is to add open ended questions, so we know we are all on same page with cancer information.

A better method is the collaborative approach, is often nick-named the Frisbee technique. This reciprocal interaction focuses on a dialogue. The delivery is light and airy, and information delivery is given in small pieces. Here the emphasis is on eliciting client feedback.

Core cancer communication skills
Gathering information
It is very helpful to identify the client’s full agenda and help the client identify concerns. Use open ended questions that start with: how, what, and tell me. Examples are “What other questions do you have?” or “Anything else you’d like to discuss?”

Elicit the client’s perspective. Does the client have a previous experience with cancer in people or another pet? It is important to identify misconceptions of cancer and barriers to care. Examples are “What are you goals with treating Bo’s cancer?” “What are your hopes?” and “What are your fears?”

Explaining and planning
Assess the client’s knowledge level and what level of information to give. It is also important to determine what degree the client wants information, and be aware this may change with time. Since many clients are overwhelmed in the beginning, it is often helpful to start with the big picture and ask what they know already and to what additional information they are seeking. “Chunks and check”
is very helpful when having cancer conversations. You give information in small chunks, and then follow with checks for understanding. This is less lecturing and aims to increase recall, understanding and commitment. Use questions like: “What questions do you have?” or “What part of the plan is most difficult?”

**Building relationships**

Offer partnership and use inclusive language like let’s, we, our, us, such as “We’ll work together for Teddy.” Asking permission during the conversation will allow you to assess the client’s readiness to take next step with questions like: Would you like to schedule surgery? Are you ready to start treatment?

Express Empathy: Acknowledge clients emotions and put yourself in their shoes and communicate that you know where they are coming from. Example: I can only imagine how hard this is. Nemo has been part of your family for so long.

Demonstrate appropriate nonverbal behavior: This can be helped with an attentive body posture, sit at same level, sit close, and maintain good eye contact. Use a slow pace, lean forward, reach out to touch.

**Provide structure and summarize**

It is useful to take time to reflect what the client heard, to repeat key aspects of diagnostics and treatment, and to provide a summary at end of appointment. For example, “I recommend these tests and this treatment for Bo’s melanoma but there are options. What questions do you have?”

“I don’t have time for this!” Core communication skills actually save time and allows for more efficient veterinarian-client-patient interaction. If you spend time to build a relationship early, it will pay dividends through diagnosis and treatment.

**Delivering bad news**

I was never taught this in school, my internship, or my residency. Most oncologists learn to break bad news by observing more experienced colleagues in clinical situations, typically during their residencies. Many veterinarians report a lack of confidence in their ability to break bad news.

The specific lack of training opportunities appears to play a major role in leading to this problem. In a human study, almost 40% of respondents not only had no didactic training, but also did not have an opportunity to gain experience from observing other clinicians breaking bad news.

Delivering bad news is a complex communication task that you may have to do thousands of times. It is stressful for clinicians to carry the burden of responsibility for communicating bad news. Complicating factors include our experience (or inexperience) giving bad news, the relationship with the client and their pet, and often the limited treatment options.

For the client, the response is affected by their relationship to their pet, severity of the diagnosis, past experiences with other pets or human family members, other stressors in their life, and their support system.

When saying “Your pet has cancer”, be aware of where and when you deliver the bad news. Too many clients hear those fateful words with less than an appropriate manner in less than an appropriate setting. Common mistakes include having the conversation in a space with no privacy, having a conversation that is too short, and having no treatment plan to discuss. Poor communication can lead to general dissatisfaction and a loss of trust.

Instead, when saying, “Your pet has cancer”, think empathy and respect, go to a private location, have sufficient time and attention, and be sensitive.

**Six step strategy: SPIKES**

**SETTING UP the interview**

- Mental rehearsal
- Privacy
- Involve significant others
- Sit down
- Make connection
- Eye contact
- Touch

**Assessing the client’s PERCEPTION**

- Before you tell, ask
- Open ended questions
- “What have you been told about…”

**Obtaining the client’s INVITATION**

- Some want full information for diagnosis, prognosis and details of the illness
- Some do not
• Some may want more information later

**Giving KNOWLEDGE and information to the client**
• Warning that bad news is coming “Unfortunately I have some bad news”
• Use non-technical terms: Spread vs metastasis, Sample of tissue vs biopsy
• Chunk and check

**Addressing the Client’s EMOTIONS with empathetic responses**
• Observe: silence, disbelief, crying, denial, anger
• Let client express feelings
• Make a connecting statement
• Examples of empathic, exploratory, and validating responses (Baile, 2000)

**STRATEGY and SUMMARY**
• Make a clear plan
• Consider a referral,
• Address pain control and symptom relief

**Discussing prognosis**
There are a few approaches to discussing prognosis. The first is realism. Interestingly, in people, 20% of patients do not want full prognosis information. Second is optimism. If overly optimistic, clients may lose opportunities to fulfill last wishes, prepare themselves and family, and spend quality time with their pet. With avoidance, you may appear evasive or dishonest. In addition, the veterinarian risks the trust and relationship with client, and the client could compromise pet’s care.

Do not make assumptions about what the client wants to know. Ask. “How much would you like to know about course of Myles’ lymphoma?” Some like details or the big picture. This is a good time to use chunk and check.

Balance hope and reality. The median survival time can be helpful. Acknowledge the client's emotional reaction and remember to compose yourself, pace yourself, and allow time to reflect.

**First reaction**
Remember the client’s initial reaction may be to not treat at all. It is okay not to treat after we provide information about the diagnosis, treatment options and prognosis. We must provide accurate information about cancer, and a range of treatment options. We can replace misperceptions and fear with knowledge and hope and educate the client that pets with cancer can live longer, and live well – not only after treatment, but during treatment too.
Cancer is not a death sentence in pets. Chemotherapy is well tolerated in the majority of dogs and cats undergoing treatment. With treatment, many cancer patients are not only living longer, but living well.

**Importance of oncology**
The pet is a family member, and owners often want same standard of care for their dogs and cats as they do themselves. Sadly, cancer is leading cause of death in pets. “Cancer” is a scary word that is often equated with death. There is often a visceral fear of cancer, and people think cancer equals pain and suffering. Owners think cancer treatment will just make the patient sicker. With cancer, there is no hope. I disagree. Cancer is not a death sentence. While we all want a cure for cancer, I encourage thinking about many cancers as chronic conditions that may require chronic therapy, such as kidney or heart disease. As an oncologist, I recommend treatment when the pet is likely to live longer with it than without. Thankfully, most cats feel good, if not great, during treatment. I believe it is important to approach the topic of cancer with knowledge, compassion, and a positive attitude.

**Chemotherapy**

**Conventional chemotherapy**

Conventional chemotherapy is typically given at high dosages, known as maximum tolerated dose, or MTD. The goal is to kill the rapidly dividing cancer cells. But some normal cells that also have high turnover often can be temporarily damaged by MTD chemo. Most commonly, it is the GI tract cells and the neutrophils that are temporarily damaged. As a result there is a break period to allow these cell populations to recover. MTD is typically given weekly to every 3 weeks.

Chemotherapy drugs attack rapidly dividing cells. The normal tissues that typically are most sensitive to chemotherapy are the bone marrow, hair follicles (alopecia), and the gastrointestinal lining. This is often referred to as “BAG”.

Bone marrow suppression most commonly results in a neutropenia but cats seem to be more tolerant than dogs. Neutrophils and platelets are at greatest risk due to the shorter circulating lifespan, and shorter bone marrow transit times. Neutropenia is the dose-limiting toxicity in veterinary oncology.

When giving a potentially myelosuppressive drug like doxorubicin, carboplatin and Lomustine, I personally like to check the expected nadir (low neutrophil count) to see if antibiotics and/or a dose reduction are needed. The nadir typically occurs 7 days after chemo administration. Pay attention to the neutrophil count, not the total white blood cell count. For some chemotherapy drugs the nadir is more variable such as carboplatin and Lomustine. For cats, the nadir is can occur 7 to 28 days after treatment. In dogs the nadir for carboplatin in day 10-14. Chlorambucil tends to cause delayed neutropenias and thrombocytopenias after chronic use.

Alopecia (hair loss) is due to damaging the rapidly dividing hair follicle. In dogs, potentially affected breeds have continuously growing coats and include Poodles, Scottish Terriers, and Westies. In cats, alopecia is rare, but shaved areas tend to grow back more slowly (limb catheters, abdominal ultrasounds). Cats more commonly lose their whiskers. The good news is that hair and whiskers will re-grow once the treatments have completed. Occasionally, hair will grow back a different texture or color. In cats it is typically softer, aka the “chemo coat”. It is important to remember pets do not care about this cosmetic side effect, and it does not impact the quality of life. However, pet owners like to be advised about the whiskers and coat so they are not surprised.

Gastrointestinal (GI) toxicity includes vomiting, diarrhea, decreased appetite, nausea. It typically 1 to 5 days after chemotherapy and is self-limiting – lasting on average 2-3 days. These side effects are less common in feline chemotherapy patients than dogs. I often will use Cerenia or mirtazapine as needed.

**How toxic is chemotherapy?**
The overall toxicity rate is very low in veterinary chemotherapy patients. In my experience, only 15-20% experience side effects, and this is even less common in cats than dogs. The primary goal is to provide the best quality of life possible for as long as possible. As I say, live longer, live well. Most side effects are mild and medically manageable.

If there are side effects, I also typically will add prophylactic medications to prevent side effects like nausea, vomiting or diarrhea as indicated. I recommend Cerenia SQ with the following drugs: vincas, doxorubicin, mitoxantrone, carboplatin, and the MOPP protocol. I always recommend oral Cerenia for 4 days after doxorubicin in dogs to prevent nausea and vomiting. If the GI side effects are more severe in a patient, the drug type or dosage may be adjusted at subsequent treatments to minimize the chance of side effects recurring.

When a chemotherapy drug is used that is known to have a high potential for bone marrow suppression, a complete blood count (CBC) is often checked after the treatment to see if the WBC are low. Antibiotics may be prescribed as a preventive measure. Subsequent doses of chemotherapy are adjusted based on the results of the CBC. Unlike dogs, I do not routinely use prophylactic antibiotics or GI medications unless the cat had issues with a prior treatment.
In my experience, there is less than a 5% chance that a patient will need hospitalization. If this does occur, these patients are usually hospitalized for typically 24-48 hours with supportive care including IV fluids and antibiotics. In my experience most chemotherapy patients can successfully receive that drug again with a dose reduction.

**Metronomic chemotherapy**

In contrast to MTD chemotherapy, metronomic chemotherapy is pulse or low-dose continuous chemotherapy. This is typically administered daily or every other day. The target is endothelial cells in that line tumor blood vessel. The goal may be tumor is stabilized, but this prevents further growth and spread. Common chemotherapy drugs include Palladia, cyclophosphamide, and chlorambucil and also with NSAIDS. There is still much to be learned including best drugs, dose, schedule, tumor types, and toxicity. This can be considered for some dogs and cats with advanced metastatic disease.

**What to do at the nadir visit?**

In addition to running a CBC, it is important to get a good history, TPR (that temperature is so important in chemotherapy patients), and a complete physical examination. I am always interested in knowing how the patient handled chemo – did she eat well, any vomiting/diarrhea, did the owner use any nausea or diarrheal medications? For the exam, did he lose weight, was she febrile? The nadir CBC should not be a technician appointment to just pull the blood sample. The history and exam are very important.

Pay attention to the neutrophil count, not the total white blood cell count. The nadir typically occurs 7 days after chemo administration, but can vary (see above). I recommend antibiotics if the neutrophil count is <1500. If the patient has <1500 neutrophils and is afebrile and feeling well, I recommend managing as an outpatient. However, if the patient has <1500 neutrophils and is febrile and sick, I recommend admitting for supportive care. Remember a febrile neutropenic is an oncologic emergency.

Also, I prefer that we get blood samples from the jugular veins for patients getting IV chemotherapy (unless thrombocytopenic). Save those peripheral veins for treatment please. Finally many times the oncologist has run a recent chemistry panel, so check with the oncologist, and try not to repeat unneeded blood work to keep costs down.

In addition to the chemotherapy targeting rapidly dividing bone marrow stem cells, other mechanisms for neutropenia includes bone marrow infiltration with neoplastic cells (leukemia, advanced stage lymphoma, multiple myeloma) and increased consumption due to infection.

<table>
<thead>
<tr>
<th>Neutrophil count (per uL)</th>
<th>Fever, systemic signs</th>
<th>Plan</th>
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<tr>
<td>1500-2500</td>
<td>No</td>
<td>Monitor +/- treatment delay 2 to 4 days</td>
</tr>
<tr>
<td>&lt;1500</td>
<td>No</td>
<td>Oral antibiotics treatment delay Consider dose change</td>
</tr>
<tr>
<td>&lt;1500</td>
<td>Yes</td>
<td>ATH for IVF &amp; IV antibiotics treatment delay Dose change</td>
</tr>
</tbody>
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**Sepsis**

Sepsis in chemotherapy patients is typically due to patient’s own flora - Gram negative from GI bacteria: *E. coli, Klebsiella, Pseudomonas*. Gram positive from skin bacteria: *Staphylococcus epidermitidis and aureus*, Anaerobes from oral bacteria. Predisposing factors include neutropenia (infection risk well correlated with degree and duration), cellular immune dysfunction, humoral immune dysfunction, prolonged hospitalizations, indwelling catheters, and poor nutrition.

History and clinical signs are typically straightforward - cytotoxic chemotherapy was administered typically 5 to 7 days ago. Remember, the febrile neutropenic patient is an oncologic emergency!!! In addition the patient may have an inability to mount an inflammatory response, so the lack of fever, pyuria, or radiographic changes of pneumonia does not rule out sepsis. Signs of illness are unrelated to absolute neutrophil count, but are related to an increased susceptibility to local and systemic infections when neutropenic. Gastrointestinal, urogenital, and respiratory infections are most common. Shock is also possible.

The sepsis work up includes: CBC, Chemistry panel, UA & UCS (if >50,000 platelets). If respiratory signs are present, chest radiographs are recommended, and TTW should be considered. Blood cultures may be needed, but uncommon in my experience. Culture any catheters suspected as the infection source.

Treatment for sepsis includes: IVF and broad-spectrum IV antibiotics. Neupogen is human recombinant G-CSF. The MOA is stimulation of proliferation & maturation of neutrophil precursors, and monocyte precursors to a lesser extent. It also primes neutrophil for cell killing & neutrophil migration. The benefit for the febrile & febrile neutropenic patient is contradictory, and in my experience, Neupogen is rarely needed. The recommended dose is 5 ug/kg SQ SID until neutrophil >1000.
When should I lower chemotherapy dose?

Dose Intensity is chemotherapy given at MTD & shortest possible interval. It is important to remember than small dose changes can have significant impact on cancer control. Dose reductions as small as 20% can decrease drug efficacy up to 50%. Dose reductions should not be considered lightly.

Vomiting and diarrhea

Acute vomiting is typically associated with cisplatin, doxorubicin (Adriamycin), dacarbazine (DTIC), cyclophosphamide, actinomycin, 5-FU streptozotocin. This can typically be prevented with pre-treatment

Delayed vomiting is more common in our patients. This is due to direct damage to rapidly dividing GIT cells (crypt cells) or via the centrally mediated CRTZ stimulated via gut vagal efferents. Delayed vomiting is most commonly 2 to 5 days post-chemo and seen with doxorubicin and the vinca alkaloids. Clinical signs include vomiting, diarrhea, anorexia, lethargy, weakness, + dehydration.

For work up, I recommend CBC, chemistry panel, UA, +/- fecal floatations and cultures. If abdominal pain is present, consider AXR or AUS to rule out foreign body, obstruction, and intussusception. For patients with GI neoplasia, it can be challenging to differentiate chemotherapy side effects vs. disease, and a good history can be key.

For outpatient treatment, I recommend NPO, food & water trial, bland diet, anti-emetics, antibiotics with severe diarrhea and a probiotic. Do not forget to discontinue oral chemotherapy or delay chemotherapy treatment. In addition, I recommend prophylactic therapy with the next chemotherapy

For inpatient, I add injectable antiemetics, IV fluid therapy, and IV antibiotics. An important note, I strongly encourage owners to NOT EUTHANIZE at this time. It is amazing with 1 to 2 days of good supportive care how quickly these patients improve. And with prophylactic therapy and a dose reduction, these patients can tolerate the same chemotherapy drug.

Don’t treat cats like small dogs when it comes to chemotherapy

Some chemotherapy drugs are dosed differently in cats. In dogs, weight and body surface area are used to determine the carboplatin dose. In cats there is now a more accurate method to dose carboplatin in cats based on glomerular filtration rate, which is determined with an iohexol clearance test.

Side effects in cats are also different. Cardiotoxicity is a well-described adverse effect in dogs treated with doxorubicin, but it has not been reported in cats. Sterile hemorrhagic cystitis (SCH) is a relatively uncommon complication of cyclophosphamide in dogs and ifosfamide therapy in dogs and cats. SCH is typically associated with long-term use, but possible after single dose, and can progress to bladder fibrosis. The incidence with cyclophosphamide has been reported to be 9% in dogs (7-24%), 3% in cats, and 24% in humans. Unlike dogs, concurrent administration of furosemide with cyclophosphamide is not recommended in cats. Mesna, which binds the SCH-inducing acrolein, is recommended for cats and dogs when administering ifosfamide.

Don’t monitor the bump or lump. Do get an aspirate or biopsy. Why wait? Aspirate.®

Visual monitoring of skin and subcutaneous masses is not enough. Even the most experienced veterinarian or oncologist cannot look at or palpate a mass and know whether it is malignant or not. Cancer is a cellular diagnosis! It is always recommended to evaluate masses that are growing, changing in appearance, or irritating to the patient. But these guidelines are not enough.

“See Something Do Something, Why Wait? Aspirate. Dr. Sue Cancer Vet®” (SSDS) provides guidelines for evaluating superficial masses in dogs and cats. These guidelines will increase client awareness and will promote early cancer detection, diagnosis, and early surgical intervention. In veterinary medicine, most skin and subcutaneous tumors can be cured with surgery alone if diagnosed early when tumors are small.

See Something: When a skin mass is the size of a pea (1 cm) and has been present for at least 1 month,

Do Something: Aspirate or biopsy, and treat appropriately!

Why diagnose early? Obtaining a definitive diagnosis with cytology or biopsy early and before excision will lead to improved patient outcomes for superficial masses. When smaller, superficial tumors are detected early, surgery is likely curative – this is especially true for benign lesions and tumors that are only locally invasive with a low probability of metastasis. If tumors are removed with complete surgical margins, the prognosis is often good with no additional treatments needed.

- Pet owners need to be aware of the “pea” size requirement to have masses evaluated.
- Veterinarians must measure and document the size of the mass in order to compare growth.
- If > 1 cm (or size of large pea) and present for a month, the mass should be aspirated or biopsied.
- Knowing the tumor type prior to the FIRST surgery will increase success of a curative-intent surgery

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References

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Cancer, even advanced metastatic disease, is not a death sentence in pets. Chemotherapy is well tolerated in the majority of dogs and cats undergoing treatment. With treatment, many cancer patients with metastasis can live longer and living well.

**Conventional chemotherapy**

Conventional chemotherapy is typically given at high dosages, known as maximum tolerated dose, or MTD. The goal is to kill the rapidly dividing cancer cells. But some normal cells that also have high turnover often can be temporarily damaged by MTD chemo. Most commonly, it is the GI tract cells and the neutrophils that are temporarily damaged. As a result there is a break period to allow these cell populations to recover. MTD is typically given weekly to every 3 weeks.

Chemotherapy drugs given at MTD attack rapidly dividing cells. The normal tissues that typically are most sensitive to MTD chemotherapy are the bone marrow, hair follicles (alopecia), and the gastrointestinal lining. This is often referred to as “BAG”.

Bone marrow suppression most commonly results in a neutropenia. Neutrophils and platelets are at greatest risk due to the shorter circulating lifespan, and shorter bone marrow transit times. Neutropenia is the dose-limiting toxicity in veterinary oncology. Cats seem to be more tolerant than dogs.

Alopecia (hair loss) is due to damaging the rapidly dividing hair follicle. In dogs, potentially affected breeds have continuously growing coats and include Poodles, Scottish Terriers, and Westies. In cats, alopecia is rare, but shaved areas tend to grow back more slowly (limb catheters, abdominal ultrasounds). Cats more commonly lose their whiskers. The good news is that hair and whiskers will re-grow once the treatments have completed. Occasionally, hair will grow back a different texture or color. In cats it is typically softer, aka the “chemo coat”. It is important to remember pets do not care about this cosmetic side effect, and it does not impact the quality of life. However, pet owners like to be advised about the whiskers and hair coat so they are not surprised.

Gastrointestinal (GI) toxicity includes vomiting, diarrhea, decreased appetite, nausea. It typically 1 to 5 days after chemotherapy and is self-limiting – lasting on average 2-3 days. These side effects are less common in feline chemotherapy patients than dogs. I often will use Cerenia or mirtazapine as needed.

The overall toxicity rate is very low in veterinary chemotherapy patients treated at MTD. In my experience, only 15-20% experience side effects, and this is even less common in cats than dogs. The primary goal is to provide the best quality of life possible for as long as possible. As I say, live longer, live well. Most side effects are mild and medically manageable. If there are side effects, I also typically will add prophylactic medications to prevent side effects like nausea, vomiting or diarrhea as indicated. It is important to be proactive and educate clients.

In my experience, there is less than a 5% chance that a patient will need hospitalization. If this does occur, these patients are usually hospitalized for typically 24-48 hours with supportive care including IV fluids and antibiotics. In my experience most chemotherapy patients can successfully receive that drug again with a dose reduction and prophylactic medications.

**Metronomic chemotherapy**

In contrast to MTD (high dose) chemotherapy, metronomic chemotherapy is pulse or low-dose chemotherapy. Metronomic chemotherapy is the uninterrupted administration or low doses of cytotoxic drugs at regular, continuous and frequent intervals without breaks. This is typically administered orally daily or every other day. Elimination of breaks between dosages reduces or eliminates the ability of the tumor cells to repair damage or alter their microenvironment.

With MTD chemotherapy, the goal is to target and kill tumor cells directly. The target of metronomic chemotherapy is the tumor-associated vasculature. These are the endothelial cells in that line tumor blood vessel. In contrast to the quiescent endothelial cells throughout the body, tumor endothelial cells are much more proliferative. In metronomic chemotherapy, the result may be that the tumor is stabilized, but this prevents further growth and spread.

The key to metronomic chemotherapy is the reduction or elimination of breaks between dosages – to prevent repair and repopulation of the endothelial cells. This is also different than MTD chemotherapy in which the break between dosages allows for recovery of the normal cell populations, like neutrophils and GI tract cells. Another important distinction of metronomic chemotherapy is that chemotherapy is given at low dosages to allow for the continuous often daily dosages.

Overall, metronomic chemotherapy protocols are well-tolerated with low toxicity profiles. Depending on the drugs used, some protocols are also lower in cost. Common chemotherapy drugs include low dose cyclophosphamide, chlorambucil, and Lomustine. Toceranib (Palladia) is also used in metronomic protocols. Other drugs included in some protocol are NSAIDS and doxycycline. There is still much to be learned including best drugs, dose, schedule, tumor types, and toxicity.
How does metronomic chemotherapy work?
In the cancer patient, tumor angiogenesis occurs locally in the tumor microenvironment where circulating endothelial cells (CECs) are stimulated and due to systemic effects of circulating endothelia progenitor cells (CEPs) that are derived in the bone marrow. Continuous low dosages of many chemotherapy drugs are cytotoxic to both CECs and CEPs. There seems to be little toxic effects on non-endothelial cells like white blood cells and epithelial cells. Tumor cells are also not effected by metronomic chemotherapy.

Another interesting target is the regulatory T-cell (Treg), a subset of the CD4+ T-lymphocyte population that helps tumor cell survival by contributing the immune suppression. Low dose cyclophosphamide (CYC) has been demonstrated to be selectively toxic to the Treg cells. It is also believed that NSAIDs can also decrease Treg cells with COX inhibition. Many metronomic protocols combine a chemotherapy drug like low dose CYC and a NSAID.

There is concern for the risk of sterile hemorrhagic cystitis (SHC) with cyclophosphamide, and this risk may increase with cumulative CYC administration. Owners should be advised of the risk of SHC and appropriate and regular patient monitoring is highly recommended. Cyclophosphamide should be discontinued.

In some cases when MTD high dose chemo is no longer effective, metronomic chemotherapy may still inhibit tumor growth. This can be considered for some dogs and cats with advanced metastatic disease.

Antiangiogenic chemotherapy with TKI
Most Receptor Tyrosine Kinase Inhibitors (RCKI) target numerous receptors. Toceranib (Palladia) is a RTKI approved for MCT in dogs that targets the mutated c-kit to directly kill tumor cells. In addition, Palladia also inhibits angiogenesis by targeting other receptors like VEGFR and PDGFR. Palladia may be useful in metronomic chemotherapy protocols.

There is evidence that good biologic activity occurs when Palladia dosages are lower than the label dose of 3.25 mg/kg EOD. This was noted in the Phase I study of dogs with a variety of solid tumors where response was noted at 2.5 mg/kg EOD. Additional studies with solid tumors found lower dosages were associated with good clinical activity and reduced side effects. Biologic activity has been observed in anal gland anal sac ACA, thyroid carcinomas, metastatic OSA, nasal carcinoma, and head and neck carcinoma.

I typically recommend 3 times per week dosing with a target dose of 2.5 to 2.8 mg/kg (ie MWF) and will use low dose compounded CYC on TuThSat. I typically use a NSAID on non-Palladia days if included.

Toxicity and supportive medications
In general, metronomic chemotherapy is well tolerated with minimal toxicity. In my experience, side effects are most likely to occur with Palladia and are usually GI-related. so I typically start Palladia first and make sure the patient is tolerating it before adding additional medications such as low dose CYC. I start omeprazole with Palladia. I avoid metronomic chemotherapy in patients presenting with inappetance and/or vomiting and diarrhea.

Gastrointestinal (GI) adverse effects include vomiting, diarrhea, decreased appetite, nausea. I monitor my patients at 2 week intervals for the 1st 4 to 8 weeks. Good patient history and careful monitoring of body weight is critical. All my Palladia patients go home with a “just-in-case: bag including Cerenia, metronidazole and a probiotic, +/- mirtazapine. In some cases experiencing GI issues, I will recommend Cerenia be given 1 hour prior to Palladia, or Palladia dose will be adjusted.

CBC and chemistry panel should be monitored at each visit. Palladia and chlorambucil tend to cause delayed neutropenias and thrombocytopenias after chronic use. I also recommend periodic urinalysis and UPC.

The goal of metronomic chemotherapy is stable disease which requires chronic administration. It is very important our patients are experiencing minimal side effects and a great quality of life on the protocol, so they can stay on the protocol long term.

Doxycycline
Doxycycline has been documented to have some antiangiogenic effects by inhibiting matrix metalloprotineases, so it is thought that the addition of doxycycline metronomic chemotherapy protocols may enhance the antiangiogenic effects. Further studies are needed to confirm its efficacy and best dosing.

Summary
Conventional chemotherapy is typically ineffective for patients with gross metastatic disease. Metronomic chemotherapy is well tolerated and appealing with the low toxicity and use of oral forms. But metronomic chemotherapy is still in its early use in terms of efficacy and potential for toxicity. Stable disease is typically the goal, so therapy is often chronic and stable disease should be expected to maintain a good QOL for the patient. There is still much to be learned including best drugs, dose, schedule, tumor types, and toxicity.

Additional resources

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What is “See something, do something. Why wait? Aspirate. Dr. Sue Cancer Vet” ™?
“See Something, Do Something” (SSDS) is a lumps and bumps cancer awareness program that provides guidelines for evaluating superficial masses in dogs and cats. We hope these guidelines to increase client awareness will promote early cancer detection and diagnosis, as well as early surgical intervention. In veterinary medicine, most skin and subcutaneous tumors can be cured with surgery alone if diagnosed early when tumors are small.

- See Something: If a skin mass is the size of a pea (1 cm) and has been there 1 month,
- Do Something: Aspirate or biopsy, and treat appropriately!

Why do we need SSDS?
It is well documented that cytologic and histologic evaluations are important diagnostic tools in veterinary oncology and that obtaining a preliminary diagnosis optimizes treatment planning. It is also recommended to evaluate masses that are growing, changing in appearance, or irritating to the patient. At this time, no specific guidelines exist for determining when to aspirate or biopsy or when to monitor canine and feline skin and subcutaneous masses.

Without standard of care guidelines, superficial masses may be monitored for too long. This can negatively impact our patient’s prognosis as well as limit their treatment options. Larger tumors that are diagnosed later may require more advanced treatments. Surgical excision of larger masses may result in less than adequate surgical margins (narrow or incomplete), leading to recurrence and additional costly therapy (second more aggressive local surgery, radiation therapy and/or chemotherapy).

With significant time delays and prolonged monitoring, there may be no reasonable surgical treatment options to remove large advanced tumors. These are often the most frustrating and heartbreaking cases.

Why diagnose early?
Obtaining a definitive diagnosis with cytology or biopsy early and before excision will lead to improved patient outcomes for superficial masses. When smaller, superficial tumors are detected early, surgery is likely curative - especially benign lesions and tumors that are only locally invasive with a low probability of metastasis. If tumors are removed with complete surgical margins, the prognosis is often good with no additional treatments needed.

- Visual monitoring is not enough.
- Pet owners need to be aware of the “pea” size requirement to have masses evaluated
- Veterinarians must measure and document the size of the mass in order to compare growth.
- If > 1 cm (or size of large pea) and present for a month, the mass should be aspirated or biopsied.
- Knowing the tumor type prior to the FIRST surgery will increase success of a curative-intent surgery.

What are the most common tumors?
Primary skin and subcutaneous tumors are common in dogs and cats. While the overall incidence in dogs and cats is difficult to determine, approximately 25 to 43% of biopsies submitted in dogs and cats are of the skin. Of submitted samples, 20 to 40% are reported to be malignant.

The most common malignant skin tumors in dogs are mast cell tumors (MCT) (10-17%), soft tissue sarcomas (including fibrosarcomas [2-6%], malignant nerve sheath tumors [4-7%]), and squamous cell carcinomas (2-6%). The most common benign canine skin and subcutaneous benign tumors include lipomas (8%), histiocytes (8-12%), perianal gland adenomas (8-12%), sebaceous gland adenomas/hyperplasia (4-6%), trichoepitheliomas (4%), papillomas (3%), and basal cell tumors (4-5%).

In cats, the most common superficial tumors include basal cell tumors (BCT) (15-26%), mast cell tumors (13-21%), squamous cell carcinomas (10-15%), fibrosarcomas (15-17%). These four tumor types make up about 70% of all skin tumors in cats. Sebaceous gland adenomas are much less common (2-4%). If BCT are excluded, the percentage of malignant skin tumors in cats is higher than dogs, with studies reporting 70 to 80%.

Is visual monitoring acceptable?
Even the most experienced veterinarian or oncologist cannot look at or palpate a mass and know whether it is malignant or not. Cancer is a cellular diagnosis! It is always recommended to evaluate masses that are growing, changing in appearance, or irritating to the patient. But these guidelines are not enough. All skin and SQ masses that are >1 cm and have been present for 1 month should be aspirated for cytologic evaluation. Biopsy is indicated if cytology does not provide a diagnosis.
Methods of diagnosis

Aspirate and cytology

Fine needle aspiration (FNA) and cytology provide a diagnosis for many skin and SQ masses, especially those that exfoliate well. FNA is useful to distinguish neoplasia from inflammation. Cellular morphology may also allow for the determination of benign or malignant phenotype. FNA is useful for identifying benign masses including lipomas and sebaceous adenomas. For malignant tumors, cytology provides information that assists in formulating diagnostic and treatment plans.

The advantages of cytology include: minimally invasive approach, low risk, low cost procedure, and results are available more quickly than biopsy results. The disadvantages of cytology are that it may be non-diagnostic or equivocal. This may be due to a small number of cells in the sample, poor exfoliation of the cells, or poor sample quality. If the sample is non-diagnostic or equivocal, histopathological confirmation may be required for definitive diagnosis.

Unless the sample is comprised exclusively of only fat, clear cystic fluid, or acellular debris, the sample should be submitted to a trained cytopathologist. WHEN IN DOUBT, SEND IT OUT. Including an adequate history helps the pathologist in diagnostic accuracy.

Biopsy

If cytology is non-diagnostic, a pre-treatment biopsy is recommended PRIOR to complete tumor removal. The pre-treatment biopsy will determine the optimal treatment plan.

The role of excisional biopsy is controversial, even among oncologic surgeons. A practical recommendation for non-diagnostic cytology and the lesion fits in an 8 mm punch biopsy, then PUNCH IT OUT. If the mass is larger than an 8 mm punch biopsy, an incisional biopsy (wedge, tru-cut, punch) is required for diagnostic confirmation.

It is tempting to remove the mass right away. An excisional biopsy establishes a diagnosis and removes the tumor at the same time. However it is not recommended for undiagnosed skin and superficial masses. Malignant tumors often require 2 to 3 cm margins. When an excisional biopsy (or debulking surgery) leads to incomplete margins for malignant tumors, more treatment, more morbidity, and more expense ensue. Thus removing the mass entirely is not recommended without a cellular diagnosis prior to definitive excision. Surgical approaches vary with different tumor types. Research confirms that the first surgery is the best chance for a cure.

Staging diagnostics are often indicated prior to curative intent surgery. Consultation with a veterinary oncologist is recommended.

After the aspirate/biopsy

If the mass is benign

Benign tumors may not need to be removed. A variety of factors, including mass location should be considered. Surgery should be recommended when a benign tumor is causing pain, irritation, bleeding, or infection. Surgery should also be recommended if an increase in growth would prevent a surgery in the future.

Alternatively, if removing the tumor requires a complicated surgery (i.e. near a joint, on the distal limb with minimal surrounding tissue for reconstruction) or the pet has other pre-existing issues, you and the pet owner may make an educated decision as to whether proceeding to surgical removal is warranted. PETS WITH MASSES NOT REMOVED SHOULD BE MONITORED (via measurement) BY THE VETERINARIAN EVERY 3 TO 6 MONTHS.

If surgery is performed, most benign masses require smaller surgeries, as wide margins are typically not needed.

If the mass is malignant

If the aspirate/biopsy reveals malignancy, consult with veterinary oncologist for appropriate staging recommendations. For malignant tumors, the first surgery should be a wide excisional surgery.

If wide excisional surgery is not possible due to the size or anatomic location of the mass, consultation with a veterinary oncologist or board-certified surgeon is indicated. Surgeons may be able to perform specialized surgeries such as axial pattern flaps to remove the tumor completely.

Debulking (cytoreductive) surgery may not be recommended, as this will not obtain margins, and additional post-operative treatments such as radiation will be required to prevent recurrence. In some cases, cytoreductive surgery may be performed for palliation, or with an understanding that adjunctive therapy such as radiation therapy will follow the procedure.

After surgery

- Review the histopathology report – tumor type, grade, vascular and lymphatic invasion.
- Consult with a veterinary oncologist for additional therapeutic considerations for malignant tumors.
- Assess the QUANTITY of surgical margins in consideration of tumor type and biologic behavior. (One mm margins for a malignant tumor may be called “clean” on a biopsy report, but size of margins must be considered in light of tumor histology.)
- If margins are inadequate, recommend adjunctive treatment before tumor recurrence for optimum patient outcome. Post-operative options include scar revision (second surgery), radiation to prevent regrowth, or chemotherapy which may slow recurrence in some cases.
• It is important to consult a board certified surgeon before attempting scar revision.
• Monitor for local tumor recurrence and metastasis as indicated by the histologic diagnosis and margin assessment.

Recurrence and monitoring
Patients with reported complete surgical margins can potentially suffer tumor recurrence due to microscopic cancer extension that is not seen in the evaluated sections. Therefore, it is essential to monitor for local regrowth, and to recruit the pet owner to monitor the surgical scar as well, to identify early relapse.

For malignant tumors with wide, clean margins and low metastatic potential, follow-up rechecks are recommended every two to three months after the surgery for as much as one year of follow up. Early detection is key to addressing recurrence and metastasis to ensure the highest possible chance of success.

Owners are encouraged to check their pets regularly at home for new masses
• Owners should check their pet monthly for superficial masses by noting their location and size.
• Create a “body map” with size and location of superficial masses recorded, along with fine needle aspiration cytology results. This body map can serve as an objective medical record document and owner guide to follow masses longitudinally, and to allow for identification of new masses over time.
• All masses should be aspirated and submitted for cytology. Masses that do not need cytologic assessment include lipomas, cysts, and those containing acellular debris.
• If cytology is non-diagnostic, discuss repeating the aspirate, or proceeding to biopsy.
• Know the tumor type prior to surgery. The first surgery is your patient’s best chance for cure.

Surgery may be all that is needed
We all must be proactive to advocate for early cancer detection. Visual monitoring of superficial masses is not enough. Obtaining a definitive diagnosis via either cytology or biopsy early and before excision will lead to improved patient outcomes for superficial masses. Surgery is likely curative for the majority of these cases, especially for benign masses and those locally invasive malignancies that are non-metastatic. If tumors are detected and removed earlier – when they are small and with clean margins, the prognosis is often good and the patient may not require additional therapy.

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• Do Something: Aspirate or biopsy, and treat appropriately!


References/suggested reading
Osteoarthritis (OA) is a chronic, progressive disease that affects both dogs and cats. It has been noted that up to 20% of adult dogs and 60% of adult cats have radiographic evidence of OA. Owners, themselves are becoming increasingly aware that bone and joint problems are and issue with their pet. Much of this increased awareness has come through the use of the Internet and social media. The overall outcome of osteoarthritis is centered on destruction of the articular cartilage and breakdown of the joint. Because of this OA must be thought of as a global disease process rather than an isolated disease entity. There is considerable cross talk among the tissues that make up a joint. For this reason the joint must be thought of as an organ and the final pathway of OA is organ failure of the joint.

OA primarily affects diarthrodial joints. A diarthrodial joint is composed of the joint capsule, synovial lining, articular cartilage, and the surrounding muscles, ligaments, tendons, and bone. The joint capsule is composed of two layers: the outer fibrous layer and the inner subsynovial layer. Both layers have a rich blood and nerve supply. One explanation of pain associated with OA is distention of the joint capsule due to joint effusion. The synovial lining covers ever structure in the joint except for the cartilage/menisci. It provides a low friction lining and is responsible for the production of synovial fluid. Two major cell populations are present in the synovial lining: type A synoviocytes and type B synoviocytes. Type A synoviocytes are macrophage-like cells that are responsible for phagocytosis. The type B synoviocytes have a more fibroblastic-like appearance and are responsible for producing hyaluronic acid (HA) and other enzymes.

Articular cartilage forms a smooth, compressible surface, which has the primary function of transmitting compressive forces onto the underlying subchondral bone. It is important to note that articular cartilage lacks blood flow, lymphatics and nerves. In fact the oxygen tension in articular cartilage is about 6-7% thus making chondrocytes survive in a hypoxic environment. Cellular destruction and apoptosis has been noted if the oxygen concentration drops below 1-2%. Because the lack of blood flow, articular cartilage must receive its nutrition from the synovial fluid. Histologically, articular cartilage is made up mainly of water (about 80%); a smaller portion is composed of the extracellular matrix (around 10-15%), and chondrocytes (around 5-10%). The chondrocytes are the only metabolically active component of articular cartilage, and are responsible for the production of the extracellular matrix (ECM). The ECM is composed of the proteoglycan matrix and type II collagen.

At the very basic level the proteoglycan matrix is composed of an aggrecan. An aggrecan is a core protein with glycosaminoglycans attached (GAGs) such as chondroitin 5 or 6 sulfate. Multiple aggrecan’s are then attached to a hyaluronic acid backbone (produced from type B synoviocytes) to form an aggrecan aggregate. These aggrecan aggregates have a gel-like consistency and are responsible for resisting compression. The proteoglycan matrix is contained within a type II collagen framework. This lattice type framework gives added support by forming interlocking loops. Having the knowledge of phenotypic make up of cartilage is important when evaluating in-vitro studies. When type II collagen from articular cartilage is taken from an in-vivo condition and placed in-vitro it tends to change its phenotypic expression and begins to produce type I collagen. Many studies utilizing OA models for various treatment modalities are in-vitro. It is very important when evaluating these studies that the study authors have proven that the collagen in-vitro has the same phenotypic expression as collagen in-vivo.

Mature cartilage is classified into 3 un-mineralized zones. Zone 1 (superficial or tangential zone) contains the highest concentration of chondrocytes. These chondrocytes are small, flat, and oriented with the long axis parallel to the joint surface. During joint compression such as with weight bearing the chondrocytes in this zone undergo tension parallel to the joint surface. Chondrocytes in Zone 2 (transitional zone) begin to become larger and more rounded. As the zones become deeper the long axis moves from being more parallel to the joint surface to becoming more perpendicular to the joint surface to compensate for the shearing and compressive forces that predominate in this area as the load on the joint increases. Chondrocytes in the deeper zone 3 (radial zone) contain larger chondrocytes that are predominately oriented perpendicular to the joint surface. This zone forms a rigid mesh and can be partially mineralized. The primary force in this area is compression. The tidemark marks the completion of cartilage maturity as it transitions to the underlying subchondral bone. In a nutshell, mature cartilage forms a pre-stressed, wear-resistant protective diaphragm in zone 1 that helps withstand tension in the plane of the articular cartilage. The middle and deeper layers have the fibrils becoming more organized perpendicular to the plane of the cartilage to withstand compressive loading.

The physiology of cartilage is important because damage to chondrocytes will not only lead to death of that particular chondrocyte but also an inflammatory response that creates problems with neighboring chondrocytes. Thus a downward, progressive spiral occurs which leads to destruction of the “work-horse” (chondrocytes) and loss of extracellular matrix production. The loss of ECM production leads to the loss of cartilage’s ability to soften and transfer loads to the underlying subchondral bone.
The pathophysiology of OA is described as a non-infectious disorder of diarthrodial joints. It is categorized by deterioration of articular cartilage, bone formation at synovial margins (osteophytes), peri-articular fibrosis, and a localized inflammatory response. For OA to develop there has to be some insult to the articular cartilage such as hip dysplasia, a cranial cruciate ligament tear, elbow dysplasia, or an articular fracture. Once the chondrocyte is damaged the inflammatory cascade begins and is followed by the release of multiple cytokines. The two main cytokines involved with OA are interleukin 1 beta (IL-1β) and tumor necrosis factor alpha (TNF-α). IL-1β is responsible for the breakdown of the matrix, while TNF-α drives the inflammatory response. Furthermore, prostaglandins are released, particular prostaglandin E2 (PGE2), which increases the release of metalloproteinases (MMPs). MMPs are responsible for the continued breakdown of the ECM.

This brings into mind one fundamental question; if inflammation is such a key driving force with OA then why don’t anti-inflammatories slow down or eliminate the problem? The answer is there must be one piece of the puzzle missing to the pathophysiology. One such puzzle piece that has been investigated is the concept of oxidative stress. Oxidative stress is an early event during the evolution of many diseases. It occurs when reactive oxygen species (ROS) outweigh the antioxidants. On a daily basis cells undergo oxidative stress to help with normal cellular metabolism, which is kept in balance by the cells natural antioxidants. If at any point the ROS outweigh the antioxidants through either excessive ROS production or antioxidant depletion then oxidative stress will occur. This manifest itself as lipid peroxidation, protein, DNA or RNA oxidation. What has been shown in human medicine is that the effects of oxidative stress on chondrocytes along with MMPs are the 2 main mediators in matrix degradation. Furthermore, inducible nitric oxide synthase expression can occur by a single stimulation of IL-1β or TNF-α. This concept has also been proven in the canine where it was shown that the same inflammatory mediators that cause an inflammatory response also cause an oxidative stress response. Furthermore, oxidative stress to the cell causes a reduction to the cells natural antioxidants. With treatment of certain antioxidants the cells natural antioxidants are able to recover and thus minimize the oxidative stress response.5

It is important to remember that OA is usually always secondary to some other disease process so it is important to eliminate the problem if one is able. Technically, any problem that involves or disrupts the joint will lead to OA. Because of this it is important to make owners aware. For example, it is common to explain to owners why their dog may rupture its cranial cruciate ligament and what treatment options exist. Its equally important to also make the owner aware that no matter what treatment option is elected the patient will still possibly develop some degree of OA.

Owners will typically complain about their pets have a reluctance to exercise, stiffness, lameness, inability to jump, or even some behavioral changes. Remember that cats are not small dogs, and they can have fewer signs. The biggest complaint from owners with cats suffering from OA is a reduction in activity, reluctance to jump, an unkempt appearance, and aggression. Orthopedically, dogs may show disuse muscle atrophy (ensure to rule out any neurogenic atrophy), a reduced range of motion, pain or discomfort on range of motion, crepitus, and joint effusion. Cats can be tricky to examine so allowing them performance tests is encouraged to see how the cat moves and interacts with its environment. One true test is to place the cat on exam table with its carrier below. Most cats will easily jump from the exam table to their carrier. Any reluctance to want to do so raises concern about possible joint pain.

Radiographs are key to aiding in the diagnosis of OA. However, just as with any diagnostic modality there are limitations. Radiographs only provide bony information, they are taken in a non-weight bearing position, and osteophytes are useful to diagnose OA but they are not pathognomonic for OA. Furthermore, the value of osteophytosis for staging OA is controversial and does not correlate with OA progression. Probably the biggest issue with radiographs is that they do not correlate with clinical signs. The radiographic key features of OA are: osteophytosis, enthesophytosis, effusion, soft tissue swelling, subchondral sclerosis, intra-articular mineralization (especially in cats), and subchondral cyst (rarely seen).

Other additional diagnostic modalities include CT, MRI, and arthroscopy. Arthroscopy is probably the most valuable means to objectively evaluate the cartilage. However, it is a surgical procedure and can be costly to perform. It does allow the evaluation of the cartilage, which can then be classified by the Modified Outerbridge score. One looming question is if you don’t perform arthroscopy and radiographs are helpful to diagnose but don’t help stage for monitoring for progression of OA is there some type of subjective based assessment? The answer is yes, the Canine Orthopedic Index (COI) was developed and validated in 2014 to provide reliable assessment of dogs with OA in terms of staging as well as response to treatment. It can be downloaded at www.canineorthopedicindex.com.

In regards to treatment there must be a multimodal approach. There is no “cook book” or one size fits all treatment plan. Treatment must be patient centered and patient specific. What works for one dog may not work for another dog. Furthermore, patients may respond initially to a treatment plan then become less responsive. In these cases the treatment plan has to be changed. In some cases it can really be trial and error. When I approach OA patients I break it into 1 of 2 categories. Am I seeing a patient that has a primary problem and has or will develop OA (such as cranial cruciate ligament rupture) or Am I seeing a patient that had a primary problem and now suffers from OA (the typical “OA consult”).

For those patients that have a primary problem and either have OA or will develop OA I give owners clear expectations for the future. If I can correct the primary problem such as fixing an articular fracture, or addressing a ruptured cruciate ligament then that is recommended. Following surgery I give owners my 4 pillars of OA management: Joint supplements, Omega-3 fatty acids (150-175
mg/kg of DHA/EPA), daily exercise and weight management. Furthermore, formal rehabilitation therapy is key for post-operative patients. Hopefully, once the primary problem is corrected and following these 4 pillars, nothing more will need to be done.

For patients that had a primary problem that either was or was not addressed but now they suffer from OA I will initially recommend joint supplements, omega-3 fatty acids, Adequan, weight management, and daily exercise. Furthermore, if the patient is having a flare-up then I will recommend formal rehabilitation the control the inflammatory response, improve range of motion and improve comfort. I only like to initially use NSAIDS at the lowest possible dose as infrequently as possible. Unfortunately, many of these patients will progress to a daily need for NSAIDS. During times of flare-ups patients will also benefit from additional analgesics such as Codeine or Tylenol 3 (both at 1 mg/kg q8-12h) or Tramadol (5 mg/kg q 8h). The biggest benefit in these patients is owner education. It is very important that owners are aware that this will be progressive and we can’t cure it. Flare-ups will occur and management must be stepwise.

If I have patients that don’t respond initially, or have more frequent flare-ups then NSAID use becomes more frequent. I will consider adding in gabapentin (5-10 mg/kg q8-12h) as well as formal rehabilitation. Other potential considerations are given to using amantadine (3-5 mg/kg q24h) with an NSAID, or intra-articular injections.

Potential intra-articular therapies include regenerative medicine (platelet rich plasma with or without stem cell treatment), hyaluronic acid, or steroids. Discussion of regenerative medicine is beyond the scope of this proceeding. HA is a viscosupplementation that restores the physiochemical properties to the joint. From a molecular standpoint it stimulates production of ECM as well as continued production of HA from resident synoviocytes. It will also inhibit inflammatory mediators. It is important to use a product that closely mimics a dog’s HA such as Evervisc from Everost (sold through Patterson). Evervisc is about 2 million Daltons in size and is made from a fermentation process rather than rooster combs. Until further research is completed it is not recommended to combine an HA injection with any other drug as this may decrease the molecular weight of the HA or could lessen its efficacy. What has been shown is that approximately 80% of dogs respond well to HA, 10% respond fair, and 10% don’t respond. The duration of response is about 4-6 months of relief. When compared to regenerative medicine a response of about 9 months is expected following a platelet rich plasma injection and about 12 months or longer following a platelet rich plasma and stem cell injection.

In summary, OA is a chronic progressive disease and the goal of management needs to be to slow and minimize the progression. Owners need to be well educated to know that it will progress and there will be flare-ups. Treatment needs to be multimodal and patient centered.

References
Bone is essentially the frame that supports locomotion. It’s an amazing tissue with complex properties that are a series of lever arms that act to counteract the forces of gravity while constraining and directing the forces of muscle. In general bone follows Wolff’s law in that it adapts to loads under which it is placed. Essentially, bone is shaped for the greatest strength while at the same time minimizing bone mass that would contribute to excessive weight. Bone is considered both viscoelastic and anisotropic. Viscoelastic implies that the strength of bone depends on the rate upon which it is loaded such that a bone is stronger when loaded rapidly versus slowly. Technically, bone becomes stiffer the more rapidly it is loaded; however, if the rate of loading exceeds the yield point a bone will fracture. The anisotropic property of bone says that its strength is dependent on the direction in which it is loaded, and thus bone is stronger when loaded longitudinally versus transversely.

Bone in general is subjected to many forces. A fracture occurs when the sum of the forces is greater than the ultimate strength of the bone. The 5 main forces that bone is subjected to and thus must be overcome are tension, torsion, bending, shearing, and compression. Tensile forces are a type of axial force that acts to lengthen the bone while compressive forces are a type of axial force that acts to shorten the bone. The anisotropic nature of bone suggests that it is stronger when loaded in compression versus tension. Shearing forces are difficult to conceptualize with respect to bone; however, it is a common force present within bone. Shearing forces acts parallel or tangential to the bone. Torsion acts to twist bone about its long axis. This creates a shear stress in the bone (where tension and compression are seen in oblique planes). Bending forces (also referred to as moments) makes bone convex on one side and concave on the other side. The convex side is undergoing tensile forces while the concave side is undergoing compressive forces. Understanding the forces that act on bone is important as these are the very forces that must be overcome when choosing the appropriate fracture fixation method.

Fractures can occur due to trauma and the force exceeds that of normal bone, or it can occur pathologically when the bone is weakened and therefore the force does not have to be as great to allow a fracture (abnormal bone). In a load deformation curve when bone is loaded there will be slight deformation. As long as the load remains in the elastic region then failure will not occur and the shape of the bone will revert back to normal. However, if the load continues past a certain point known as the yield point then bone will cross over to the plastic region, which will result in permanent deformation. If the load continues the breaking point then the bone will fracture.

Once a fracture occurs, the goal is to allow the bone to heal with restoration of normal function with acceptable cosmetics. There are certain factors that must be taken into consideration for a bone to heal such as the biologic factors (blood supply, location of the fracture, and concurrent soft tissue injuries) and the mechanical factors (such as the degree of stability at the fracture site). The afferent blood supply to the bone is supplied through the nutrient artery, where the blood flow is centrifugal in that it progresses from the medullary cavity to the periosteum. Therefore, blood flow is from the nutrient artery to the metaphyseal arteries, and then the periosteal arteries. After a fracture the medullary circulation is disrupted, therefore, we get an enhancement of existing normal blood supply. Temporarily, there is a transient extraosseous supply from the soft tissues. It is very important to preserve this blood supply and be kind to the tissues during surgery. As the bone heals the medullary circulation is reestablished. From a mechanical standpoint the fixation must counteract the forces acting on the bone while preserving the blood supply. Healing will also depend on the fracture gap and the stability.

Bone healing parallels that of most other tissue in the body such as soft tissues. It will progress through the typical inflammatory, reparative, and remodeling phases. For bone to adequately heal there has to be a stable environment in that the interfragmentary strain is <2%. This is the deformation occurring at the fracture site relative to the size of the gap, which influences the type of tissue that will form in the gap. Secondary bone healing is considered the normal course of bone healing and is how all bones healed prior to the advent of open reduction and internal fixation (ORIF). Essentially this occurs through callus formation by progressively stiffer tissue as bone healing moves through the various phases. Initially when the bone is fractured a hematoma develops. This hematoma provides no strength but is very important in that it releases lots of growth factors. The next stage is the formation of granulation tissue, which adds very slight strength. After the formation of granulation tissue, connective tissue develops followed by cartilage formation, cartilage mineralization, and finally woven bone formation. With ORIF primary bone healing can occur which allows “skipping” of the initial secondary phases. For this to occur once again the interfragmentary strain has to be <2% and the interfragmentary gap must be <1 mm. Thus, even with ORIF if the bone ends are not touching it will proceed through secondary bone healing, but in a quicker time since the fracture will be stable. The 2 types of primary bone healing are gap and contact bone healing. Gap bone healing occurs when the gap is <1 mm. Granulation tissue forms first with its blood supply, then lamellar bone follows without the cartilage phase. Initially the lamellar bone is oriented transverse to the to long axis of the bone (think like “caulk” filling in the gap). Haversian remodeling allows new lamellar bone to be oriented longitudinally. Contact bone healing occurs when the fracture fragments are in contact.
direct contact and there is no interfragmentary motion. There is no lamellar phase, but rather Haversian remodeling occurs directly by bridging the fracture with longitudinally oriented osteons known as cutting cones.

Always remember that if there is enough force to cause a bone to break then there is certainly enough force to cause soft tissue damage. It’s important to triage these patients by checking and stabilizing vitals (such as treating hypovolemic shock). A thorough physical/orthopedic exam and a neurologic exam are needed. It does no good to repair a femoral or pelvic fracture if the sciatic nerve is transected. Baseline diagnostics including chest and abdominal radiographs, TFAST, AFAST should be completed. Any life-threatening issues need to be addressed which may mean delaying surgery. I love fixing fractures, but I want the patient to live more. Fractures in and of themselves are not emergencies. Analgesia is imperative as fractured bones hurt, and thus the pain will lead to a systemic cascade so controlling this is important. Pure mu opioids are recommended such as morphine, hydromorphone, or fentanyl. Unfortunately, butorphanol does not typically provide adequate analgesia. Once the patient is stable then go back and obtain a thorough history, it is important to separate traumatic from pathologic fractures. Evaluate PE/Ortho/Neuro findings. Common signs of fractures include pain, swelling, reluctance to bear weight, crepitus, or angulation deformities. And as previously mentioned if the patient is non-ambulatory it is very important to evaluate for neurologic deficits such as with the radial or sciatic nerves.

The traditional AO classification system I have found to be confusing and not many people classify fractures based on this. Each bone has a number, then there are 3 zones, and finally the fracture is classified into the morphology and severity. More commonly fractures are classified by the anatomical location, severity, configuration, displacement, contamination, and if they are a growth plate fracture or not.

Fractures can be classified by the anatomical location such that they are articular which requires complete anatomical reconstruction with rigid internal fixation, epiphyseal, physeal (which have their own Salter Harris classification), metaphyseal, or diaphyseal. Furthermore, in particular areas special terms can be used such as condylar (as seen with distal femoral or distal humeral fractures), supracondylar (meaning above the condylar region), trochanteric (as seen around the greater trochanter), or subtrochanteric. The severity is described as incomplete meaning the fracture is only through one cortex (sometimes called a “greenstick” fracture in immature patients). There is a small fissure noted but the fracture is not complete. A complete fracture involves a fracture through both cortices. Also, please note that the term “compound fracture” is not used to describe any fracture in either human or veterinary medicine. A comminuted fracture is one with multiple fragments. A segmental fracture is one with two or more separate fractures of the same bone. Avulsion fractures are classified as an enthesis fracture, which is one that occurs at the attachment of a joint capsule, or an apophysis fracture, which is one that occurs at the origin or insertion of a tendon or ligament. The configuration of a fracture can be transverse in that it is perpendicular to the axis of the bone and the fracture equals the diameter of the bone. Or the configuration can be considered oblique. A short oblique fracture is one where the fracture is less than two times the diameter of the bone versus a long oblique where the fracture is greater than two times the diameter of the bone. A spiral fracture is a long oblique with a twist. The displacement is based on the degree of displacement of the distal segment in relation to the proximal segment. You have to have orthogonal radiographs to describe this. One can’t simply have only a lateral or only an AP, but must have both. The degree of contamination is used to classify open fractures. Type I open fractures are those with <1 cm puncture wounds where the fragment briefly penetrated the skin. A type II open fracture is one where there is >1 cm puncture wound with evidence of external trauma. A type III open fracture has extensive wounds with significant soft tissue damaged or absent. It is further subclassified into IIIa where there is adequate skin to close the wound, IIIb where there insufficient skin to close (aka degloving injuries), or IIIc where there is compromised vascular supply to the skin.

If you are presented with an open fracture cover it immediately. When any open fracture arrives in our hospital I cover it as soon as they come in the door with a sterile covering. This can be as simple as a sterile huck towel with vet-wrap around it. Trust me, the bacteria in your hospital will be much worse then the environmental bacteria the bone may have come in contact with. Once the dog is stable then remove your dressing and flush the wound with lots of fluid. In severely contaminated wounds I have used tap water, but typically will use either saline or p-lyte. I’m not a fan of combing iodine or chlorhexidine to my flush solutions because if you are not measuring out the specific concentrations correctly you could be killing viable cells. Once I have flushed and debrided the area then I will cover the wound with a more stable covering. We then have to make the decision about fixing the fracture as well as addressing the wound and dealing with any evidence of infection.

Physeal fractures are classified by the Salter Harris (SH) classification scheme. SH I fractures are through the physis itself, while SH II fractures are through physys and into metaphysis. SH III fractures are through physys and into the epiphysis and are considered intra-articular. SH IV fractures are through physys and into metaphysis and epiphysis as well as being considered intra-articular. SH V are compression fractures though the physis, while SH VI are compression fractures though only a portion of the physis, which results in angulation deformities.

To aid in ease of communication amongst veterinarians we need to list the bone involved (remember left or right), the location, configuration, displacement, and contamination if present. This will allow the veterinarian or surgeon on the receiving end to create a visual image of the fracture to begin to decide on how best to fix the fracture. Radiographs are mainstay for diagnosing fractures.
However, one must take orthogonal views to determine and evaluate the extent of the fracture. This includes at least a lateral and AP radiographs to tell the whole story. CT scans can be helpful especially with sacral fractures, spinal fractures, and articular fractures.

Once an understanding of the appropriate classification of fractures is understood then it is important to understand the approach and selection of fixation. Unfortunately, there is no “orthopedic cookbook” in regards to selecting the appropriate fixation for a fracture. Each fracture needs to be addressed to the individual patient by different factors.

One such factor is the patient. Issues such as size, is this a big dog or a little dog. The age as younger dogs may heal quicker and may require implant removal versus older dogs, which may take longer to heal and thus need a more robust type of fixation. Activity level certainly plays a role, as a less active dog may not require as robust of a fixation versus a dog that is very active or is a canine athlete. Client factors play a role, as they are the ones making decision. I always find it nice to give them options, as finances will play a role in what they are able to do versus what they are not able to do. I will also never commit to a certain type of fixation as my plan may change intra-operatively so I will go over all the available options that may be possible so that if something needs to change in surgery there are no surprises for the owner. Their compliance will play a big role in my selection of fixation. For example if the dog is aggressive or the owner is unable to care for an external fixator then a bone plate with screws may be a better option. The fracture itself as discussed in part I of this series is certainly a factor. The configuration will dictate what type of fixation can be used, for example an IM pin and cerclage wire is not the best option for a transverse fracture. Remember the 5 forces that need to be counteracted with fixation. The degree of contamination will dictate as well what type of fixation may be best. For example a severely contaminated fracture may be better suited for an external fixator rather than bone plates and screws. Another large factor is of course your own ability. Having the understanding of biomechanics and healing as discussed in part I is very important. Knowledge of particular implants will help decide what type of implant will be best suited for that particular type of fracture. Experience and skill level should be considered. Always ask yourself “can I fix this fracture, and should I fix this fracture” Meaning if you have experience and skill to fix it along with available implants and also should I fix this fracture meaning if you don’t have the experience should you refer it rather than attempting the unknown. Implant availability will play a role as far as what you have in your clinic to repair a fracture. It is helpful if you do lots of fractures that you have different types of implants available, as an IM pin and cerclage fixation is not an option for every fracture. If you don’t do many fractures then know the limitations of the implants you have.

In the past fractures were approached from the “carpenter” standpoint, which means absolute anatomic reconstruction with rigid internal fixation. This will disrupt the fracture hematoma and blood flow and requires significant tissue dissection. This type of approach is needed for articular fractures and for fractures that require anatomical reconstruction. Recently, a more biologic friendly way to fix fractures has been described at the “gardener” (biologic osteosynthesis) standpoint. This approach uses minimal reconstruction and rigidity to preserve blood flow. This is accomplished by indirect fracture reduction through limited approaches such as the “open but do not touch” method meaning the fracture area is approached but no manipulation of the fracture is performed or a minimally invasive plate osteosynthesis (MIPO) approach. This is accomplished through a few stab incisions and everything is done in a closed manner. When approaching these fractures there should be minimal to no disturbance of the fracture hematoma. Bridging osteosynthesis rather than rigid fixation is typically elected with limited reliance on secondary implants such as k-wires, cerclage wires, etc. In a perfect world we need to try to find the balance between the carpenter and the gardener. The fixation needs to be something that stabilizes the fracture to allow bone healing but that it is not too rigid to delay bone healing. The fixation should preserve the blood supply to the fracture and not disrupt the fracture hematoma. Furthermore, of extreme importance is to maintain joint alignment and allow early return to function.

After consideration has been given to the various factors, we then need to consider the individual factors of the various implants themselves. I have a chart that I run through in my head beginning with the least invasive yet least stable (external coaptation) and proceeding through the more invasive yet more stable (IM pin/cerclage wire, external skeletal fixator, interlocking nail, or bone plate and screws) for every fracture I am presented with. As I run through this chart I begin to go through the pros and cons of each type of fixation until I decide on the one or two best options for that particular patient.
Medial patellar luxation (MPL)

MPL is a common cause of lameness in small breed dogs and must be differentiated from issues of the hip (avascular necrosis of the femoral head) or other stifle issues (such as a CCL rupture). In fact MPL is the most common direction of patella luxation overall, and up to 98% of patella luxations in small breed dogs are medial. In large breed dogs MPL is also the most common direction found in 67-83% of cases (Labradors being the most common). Bilateral luxations are noted in 50-65% of cases.

MPL has been described as congenital; however, it is technically a developmental disorder because the MPL is not present at birth. The skeletal abnormalities that predispose an animal to an MPL are present. In other words the MPL develops after birth as a result of deformities that may be present at birth. Many of the skeletal abnormalities noted are considered inherited; therefore, affected patients should not be bred. The exact etiology is not completely understood; however, coxa vara (a decreased angle of inclination of the femoral neck) and a diminished anteversion angle (relative retroversion) have been described as the beginning underlying skeletal abnormalities. Recently coxa vara has been called into question and along with coxa vara, coxa valga has also been shown to be present in MPL. Other skeletal abnormalities noted with MPL are femoral varus, genu varum, a shallow trochlear groove (with poorly developed or absent medial trochlear ridge), medial displacement of the tibial tuberosity, internal rotation of the tibia relative to the femur, proximal tibial varus and internal rotation of the foot. In short, the thought is that coxa vara and diminished anteversion angle result in displacement of the quadriceps medially. This displacement then results in abnormal forces on the distal physis, which may retard the growth of the medial side resulting in distal femoral varus and internal rotation of the tibia. Furthermore, absence of the patella in the trochlear groove does not result in the typical pressure of the groove during growth, which results in trochlear hypoplasia.

The diagnosis of a MPL is based off of the physical examination, not the degree of skeletal deformities present. Careful palpation is needed to characterize the grade of the luxation and rule out any concurrent diseases. Gait evaluation at both the walk and trot is important. I find that it is easier for my physical examination to have the patient standing so that I can fully assess symmetry between limbs and to help judge the influence of the quadriceps muscle contraction during weight bearing. In some cases locating the patella can be challenging. At times, I will locate the patellar ligament and follow it proximally from its attachment on the tibial tuberosity until I locate the patella. Once located note if the patella is in the groove or out of the groove to begin to establish a grade. After I have located the patella I will hold it hold it between my thumb and index finger while using the other hand to grasp the tibia and lift the foot from the floor. At that point I run the stifle through range of motion including flexion, extension, internal and external rotation. While completing range of motion I will apply manual pressure in a medial and lateral direction to identify the direction and grade of luxation. For example, extending the stifle and applying medially directed pressure to the patella, internally rotating the tibia at the stifle joint, and flexing the stifle can usually cause a patella to luxate medially. When palpating the stifle pay particular attention to any joint effusion. Joint effusion is commonly associated with CCL rupture and less commonly seen with a MPL. The trochlear groove can be palpated in many cases with the patella luxated to get an idea of the trochlear groove depth. The alignment of the quadriceps can be assessed visually with the hip, stifle, and hock in a neutral alignment as the stifle is flexed and extended.

Clinical signs associated with MPL vary with the degree or grade of the luxation. MPL’s are typically based on a scale of 1-4. A grade 1 MPL is usually an incidental finding and is noted by the patella being located in the trochlear groove, it can be luxated medially when the stifle joint is held in full extension, but returns to the groove upon release of the manual pressure. There is no crepitus noted during range of motion and bony deformities are absent. Clinical signs are typically not present.

A grade 2 luxation will cause intermittent lameness when the patella is luxated; however, when palpated the patella is typically located within the trochlear groove. These patients have the typically “skipping” lameness. This is where the dog will suddenly skip and carry the leg without apparent discomfort, flexes and extends the joint several times, and then begins to bear weight again. The
patella can be luxated with combined internal tibial rotation and joint flexion. To reduce the patella extend the stifle and externally rotate. With a grade 2 luxation mild deformities develop usually consisting of internal rotation of the tibia and abduction of the hock. It can progress to a grade 3 luxation with continued cartilage erosion of the patella and trochlear groove or with concurrent CCL rupture. I tend to further classify grade 2 patella luxations into a low grade 2 or high grade 2 depending on how lax the patella feels during examination.

With a grade 3 luxation the patella is permanently luxated but can be reduced. More severe bony deformities are present such as marked internal tibial rotation and an “S-shaped” curve of the distal femur and proximal tibia. Many times a shallow trochlear groove can be palpated. These patients will often exhibit a “crouched” gait rather than an intermittent lameness because the dog often uses the leg in a semiflexed, internally rotated position.

With a grade 4 luxation the patella is permanently luxated and can’t be reduced. Commonly, marked femoral varus, proximal tibia valgus, and internal tibial rotation are noted. The tibia is typically rotated 60-90 degrees relative to the sagittal plan. Affected animals can be debilitated and develop a “crablike” posture necessitating them having to be carried by their owners rather than walking. Acute worsening of the lameness is often associated with rupture of the CCL. Correction of grade 4 patella luxation early in life is ideal as waiting makes surgical correction more challenging.

CCL rupture has been associated with MPL, and the thought is that the CCL is placed under increased stress due to the abnormal quadriceps mechanism. Concurrent rupture of the CCL is noted in 15-20% of middle aged dogs with MPL. It is unclear if this truly occurs secondary to the MPL or whether it is a manifestation of CCL disease. However, any acute lameness in a patient with an MPL needs to raise the concern of a concurrent CCL rupture.

Radiographs are useful to document the luxation and assess the degree of arthritic changes present. It is also important to identify and if possible quantify any skeletal abnormalities. In cases of low grade MPL or with mild skeletal abnormalities orthogonal views of the stifle are sufficient. However, in cases with more severe skeletal abnormalities or higher-grade luxations then orthogonal views of the femur, tibia, and stifle will be needed. Note that deformities such as varus or valgus can be seen on radiographs, but torsion can only be assessed with a CT scan. A CT scan will also permit a 3-D reconstruction to aid in quantifying the deformities present.

The decision for treatment is based on the combination of clinical signs and grade of the luxation. Typically, grade 1 luxations with no clinical signs will not need surgical management. In cases of grade 3 or 4 luxations surgical correction is warranted early in the course of the disease to minimize the progression of skeletal abnormalities and OA. Grade 2 luxations tend to be more a grey zone in that if it is a grade 2 with no clinical signs then continued monitoring is necessary; however, if there is a grade 2 associated with clinical signs then surgical treatment is warranted.

Conservative management consists of daily controlled activities such as leash walking or ball play, avoidance of high impact activities and formal rehabilitation therapy as needed to improve muscle mass and proprioception. I will also recommend a glucosamine/chondroitin sulfate and omega-3 fatty acid knowing that there will be some degree of OA progression. Unfortunately, formal rehabilitation therapy may help during periods of lameness or with increasing muscle mass, it will not typically improve the grade of the luxation. Unlike in people with patella mal-tracking, dogs with patella luxation have underlying skeletal abnormalities. I also always caution owners about any acute worsening of the lameness for fear of a CCL rupture. Also, owners need to be aware that if the lameness or skipping is becoming progressively worse then surgical therapy needs to be the next step.

The approach to surgical management should be approached to each individual patient. For me I start with an arthrotomy to evaluate the intra-articular structures to ensure there is no damage to the CCL. After evaluation I turn my attention to the trochlear groove. In smaller dogs I tend to find there is a very shallow to absent groove and in chronic cases of grade 3 luxations a “pseudo-groove” is forming just medial (or lateral) to the trochlear groove. In larger dogs I feel as though the groove tends to be a little deeper. If there is a shallow groove I will perform a trochleoplasty via a block recession. I find that for me performing a block recession is easier than a wedge; however, if you prefer a wedge recession then that is fine. It was shown that a trochlear block recession resulted in increased proximal patellar depth (where the patella tends to luxate commonly), increased patellar articular contact, and greater resistance to patellar luxation in an extended position when compared to the wedge recession. I do not recommend a trochlear sulcoplasty where the cartilage and the underlying subchondral bone is removed as this removes the entire source of hyaline cartilage and relays on fibrocartilage. It has been shown that dogs that underwent a trochlear sulcoplasty had quadriceps atrophy, palpable crepitus, and severe erosion of the articular cartilage of the patella as early as 4 weeks following surgery when compared to a wedge recession. For a block/wedge recession one should ensure that the trochlear groove is wide enough to accommodate the patella and the goal should be to provide approximately 50% of the depth of the patella. To prevent wobble or displacement of the block/wedge it is important to ensure that the osteotomies are performed parallel to the initial osteotomy.

Following evaluation of the trochlear groove I then focus my attention to the tibial tuberosity to ensure alignment of the quadriceps mechanism. To assess patellar tracking; the hip, stifle, and hock are placed in a neutral position, the patella is centered within the trochlear groove, the pes is directed vertically, and the surgeon stands at the end of the table near the hind feet. The patellar ligament is then traced from the patella to its attachment on the tibial tuberosity. If the line of action of the patellar ligament is not centered on, and parallel, to the trochlear groove then a tibial tuberosity transposition (TTT) is needed. When performed a TTT I used an osteotome
that is as wide as the tibial crest. The osteotomy is continued distally to leave the distal periosteal attachment intact. Once the osteotomy is complete the stifle is extended to decrease the tension on the patellar ligament and the tuberosity is transposed. Once transposed the stifle is then flexed to increase the tension on the patellar ligament, which will stabilize the tuberosity in its new location. Then appropriately sized k-wires are placed. Note that 2 k-wires should be used, as using only one will not stabilize the tuberosity. In larger dogs I will use a tension-band fixation to prevent inadvertent tibial crest fractures and to aid in the stability. Use care not to damage the stifle joint. The height of the fibular head is a useful landmark such that aiming distal to the fibular head will ensure the k-wires are distal to the joint.

After correcting the skeletal abnormalities then I will perform soft tissue reconstruction techniques. Very rarely if ever, will soft tissue reconstruction alone result in a satisfactory outcome in mature dogs. In immature dogs I will perform soft tissue reconstruction with the owners knowing that if a re-luxation occurs then a second surgery will be required once the patient is mature. To complete the soft tissue reconstruction I will release the retinaculum on the side of the luxation (note that I never leave the joint capsule open as part of the “release”). I will then perform an imbrication of the retinaculum opposite the side of the luxation by placing horizontal mattress sutures. Any redundant tissue is removed.

Lateral patellar luxation (LPL)

LPL is most commonly seen in larger breed dogs (albeit still less than MPL’s). About 2% of small breed dogs, 17-19% of medium to large breed, and up to 33% of giant breed dogs have been noted to have LPL. Proposed skeletal deformities associated with LPL are typically opposite of what is seen with an MPL. Changes such as coxa valga and an increased angle of anteversion are noted. The grading scheme for LPL’s is similar to the classification system of MPLs.

Because of the associated skeletal abnormalities noted with LPL’s well-positioned radiographs or a CT of the femur and tibia are necessary to quantify the deformities. Treatment is similar to MPL correction, only in the opposite direction. However, femoral corrective osteotomies may be needed.

Following surgery for either an MPL or LPL I will place patients in a soft padded bandage for 24 hours. Once home I recommend passive range of motion and stretching exercises. Beginning 2 weeks post operatively I will have owners begin slow controlled leash walks along with a formal home exercise plan. At this time point I will also recommend they begin formal rehabilitation therapy. Radiographs are needed at the 4 and 8-week mark to ensure appropriate healing. Grade 2 and 3 luxations have a good prognosis with a fair prognosis for grade 4 luxations. Complications include delayed union or fixation failure of the osteotomy site, re-luxation of the patella, infection, and OA. The overall complication rate for MPL surgery is about 18% with patella re-luxation occurring about 8-48% of the time. Typically, if re-luxation does occur it is to a lower grade than initially and surgical correction is not needed. If a patient has a re-luxation with clinical signs then surgical correction is warranted and the patient may need a corrective femoral osteotomy. LPL complications are similar to that of MPL’s, and in both situations the complication rates have been noted to be higher for dogs weighing greater than 20kg.

References
Hip dysplasia (HD) was originally described in 1935 by Gerry Schnelle and has become one of the most common orthopedic conditions that lead to joint inflammation and secondary osteoarthritis. Unfortunately, even after all of this time the exact etiology is unknown but considered to multi-factorial. One such factor involved in the expression of HD is genetics. It is not a simple Mendelian pattern but rather a complex inheritance. This means there are multiple genes that are combined with environmental influences that lead to the clinical expression of HD. Joint laxity is considered the initiating cause of HD which in turn leads to hip subluxation and poor congruence between the femoral head and acetabulum. Multiple causes of hip laxity have been described such as abnormal hip development, biomechanics, genetic influences, increased joint fluid, pelvic muscle mass, nutrition, weight/growth, and hormonal and environmental factors. It’s probably safe to assume that HD and the subsequent arthritis are the clinical manifestation of all of these.

At the very basic level dogs with HD have normal hips at birth. The hips will remain normal if complete congruity is maintained between the femoral head and acetabulum. However, if one or more of the previously discussed factors that leads to hip laxity is present then the dog will manifest as having HD. Laxity is typically defined by the distraction index (DI) which has been shown as a primary risk factor for the development of osteoarthritis (OA). Passive laxity as measured with various radiographic and diagnostic techniques is an estimation of functional laxity which permits hip subluxation. From a hip development standpoint the earliest dysplastic joint changes can be noted as early as 30 days where there is increased volume of the ligament of the head of the femur, and increased synovial fluid volume. The ligament of the femoral head is the primary stabilizer for the hip for the first 30 days of life. For the first 2 weeks of life the ligament is short so if the hip is forced to luxate the femoral head will fracture at the fovea. After about 2 weeks the ligament will begin to lengthen; in dysplastic dogs this lengthening allows lateral subluxation of the hip. This subluxation allows articular cartilage to become worn and roughened on the dorsal surface of the femoral head at its point of contact with the acetabular rim. The first radiographic signs of HD can be noted at 7 weeks of age where subluxation of the femoral head with under development of the cranio-dorsal acetabular rim may be noted.

From a biomechanics standpoint in a healthy congruent hip the forces are distributed across the entire cartilaginous surface of the acetabulum. The co-contractions of the gluteals, and adductors along with the biceps femoris, semimembranosus and semitendinosus create a force to reduce and stabilize the femoral head into the acetabulum during weight bearing. During the swing phase the primary muscles used to advance the limb are the transarticular muscles of the rectus femoris, sartorius, and ilipsoas, which have long muscle bellies with lines of action more parallel to the axis of the femur. In patients with hip laxity and thus subluxation the transarticular forces must increase to compensate for lateralization of the center of rotation of the joint. Additionally, the cartilage stress is increased because the forces acting on the articular cartilage are spread over a reduced surface area. This ultimately results in two destructive events: forces crossing the joint increase while the area over which the forces are transmitted decreases. What this means is that in patients with HD that the femoral head subluxates during the swing phase of the gate and upon foot strike the larger hip extensors cause catastrophic reduction of the femoral head. Additionally less muscle mass during development is associated with an increase in joint laxity. There has been a disparity noted between the strength of pelvic muscles and rapid weight gain, which leads to joint instability. Also, muscle mass of dysplastic breeds such as German Shepherds is less than that of non-dysplastic breeds such as Greyhounds.

Nutrition is thought to be a large contributor to joint laxity and thus HD; however, no dietary deficiencies cause HD. Dietary excesses on the other hand can contribute to the development of HD. For example, increased calcium and vitamin D lead to alterations in endochondrial ossification, and delayed bone remodeling. Diets high in excessive vitamin C can lead to hypercalcemia and diets with a high anion gap lead to increased synovial fluid production, which in and of itself has been shown to be a risk factor for hip laxity. Feeding diets to promote rapid growth have been shown to have a higher incidence of HD and also cause early fusion of the acetabular growth plates.

Increased body weight is not a cause of HD, but it certainly has very important clinical consequences in susceptible dogs. Therefore, weight reduction is an effective preventative strategy. In the lifespan study of 49 Labradors it was reported that heavier dogs (dogs allowed to eat ad lib) developed radiographic OA on an average of 6 years earlier than the dogs in the restricted fed group. Furthermore, heavier dogs required long-term treatment for OA on average 3 years earlier than their restricted fed littermates.

The diagnosis of HD is made from the signalment, clinical signs, physical exam findings, and radiographs. Affected dogs are typically large breed fast growing dogs such as German Shepherds, Rottweiler’s, Labradors, or Golden Retrievers. The age of presentation is typically biphasic and contributes to the type of treatment that may be recommended. Juvenile dogs will tend to present between 5-12 months of age with an acute onset of unilateral or bilateral hind limb lameness. These clinical signs are thought to be due to joint laxity. Histologically tearing of the joint capsule along with microfracture of the dorsal acetabular rim is seen. As dogs

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become older the long-standing joint laxity causes periarticular fibrosis, which may decrease or lessen the clinical signs. This is why some dogs will tend to have improvement in clinical signs until later in maturity when they present for clinical signs that are consistent with OA.

The severity of clinical signs depends on the stage/severity of the disease. Lameness can be intermittent, progressive, and range from mild to severe. In young patients with severe laxity a “popping” noise may be heard during ambulation. Both young and older patients may exhibit exercise intolerance and difficulty rising from pain and discomfort. Disuse muscle atrophy is a common finding and the gait may be characterized as either “swaying” or hopping. It is very important to remember that a non-weight bearing lameness is rare and thus other problems should be considered such as a cranial cruciate ligament rupture. Orthopedically pain in the hips along with crepitus may be noted. Many of these patients have decreased range of motion in extension and weight shifting to the forelimb. Evidence of joint laxity is determined through the Barlow, Ortolani, and Barden’s test. The Ortolani is performed with the patient in either lateral or dorsal recumbency and sedation is required in most cases. The first part of the ortolani is the Barlow test where a force is directed through the femur through the dorsum to subluxate the hip. The Barlow test is considered a provocative test in that it creates subluxation in a lax hip. The second part of the Ortolani test is the true ortolani maneuver where the limb is abducted and a click or clunk can be heard as reduction of the hip occurs. The clunk is considered a positive ortolani and indicative of coxofemoral laxity. Some surgeons will use the angles measured during an Ortolani test as indications for a triple or double pelvic osteotomy. The Barden’s test is performed with the dog in lateral recumbency; a direct lateral force is applied to the femur without abducting the limb. In the awake dog pressure on the medial thigh can cause discomfort and this should not be mistaken for hip pain. Any movement of the greater trochanter more than ¼ of an inch suggests laxity. Unfortunately, Ortolani and Barden’s only suggest laxity and do not predict later development of clinical signs of OA.

Radiographs are mainstay for the diagnosis of HD along with the characterization of the disease and any presence of OA. There are several ways to evaluate canine hips, which vary from using the hip extended view as what is done with OFA, or developing a distraction index as what is done with PennHip. OFA style radiographs are generally used in daily practice, this involves that the pelvic limbs are fully extended and parallel, the pelvis is symmetrical and the pelvic limbs are internally rotated. Sedation and/or general anesthesia is usually required. Mal-positioned radiographs can lead to false assumptions. The two most notable and early signs with hip OA are the circumferential femoral head osteophyte (CFHO) and the caudo-lateral curvilinear osteophyte (CCO). The CFHO is a white line at the articular margin of the femoral head that may or may not extend completely around the femoral head. It is graded from I to III. The CCO is also sometimes known as a Morgan's line, it is a well-defined linear density on the femoral neck between the greater trochanter and the capital physis in dogs greater than 18 months of age. It is different from a puppy line in that a puppy line is an indistinct radiodense line on the femoral neck in dogs less than 18 months of age, its in a similar location to the CCO but it is more subtle, more diffuse and shorter than the CCO. A puppy line is considered self-limiting and is not clinically significant.

One big debate is between the use of OFA and PennHip for HD screening. OFA is a subjective scoring system based on the hip extended view. The problem is the hip extended view is an unnatural position for dogs and can mask subluxation because the view actually forces the femoral head into the acetabulum. It does identify OA and moderate laxity but is not a sensitive method to detect early or mild laxity. PennHip uses stress radiography to detect joint laxity and it can be predictive for the development of OA. It is a measure of hip laxity, not a certification process. A study in 2010 using the OFA database described a 1.5% increase in OFA excellent films, a 3.3% increase in OFA good films, and a 2.1% decrease in OFA fair films. To complicate matters it was found that in dogs with OFA excellent films 52% had DI >0.3 putting them into the OA susceptible range, 82% of dogs with OFA good had DI greater than 0.3, and 94% of dogs with OFA fair had a DI greater than 0.3. In other words the progress of eliminating HD is moving very slow. In fact at the current progress it will take about 44 years to move Labs from a hip score of 10 where it is currently to a hip score of 5, which is equal to an OFA excellent grade.

Treatment for HD can be broken into prevention and/or laxity improvement utilizing the juvenile pubic symphysiodesis (JPS) or triple/double pelvic osteotomy (DPO or TPO). More definitive treatment can be accomplished with medical management, a femoral head and neck ostectomy (FHNO or FHO) or a total hip replacement (THA). In immature dogs that are still growing with no evidence of OA then medical therapy can be attempted. This includes promoting weight loss, daily activity, and formal rehabilitation therapy to improve muscle mass, range of motion, and comfort. Many of these patients benefit from NSAIDS, chondroproctants, and omega-3 fatty acids. For those that are severely clinically affected or have failed medical therapy then either a JPS or DPO/TPO, FHNO, or THA can be considered. In mature dogs medical management is geared towards OA management. Older dogs that become refractory to medical management would then become candidates for either a FHNO, or THA. Regardless early detection is key, in susceptible breeds hip palpation should begin by 12 weeks of age. If they have a positive Ortolani or have a high DI after 16 weeks of age then JPS should be considered in at risk breeds. A JPS is a minimally invasive way to pre-maturely cause fusion of the pubic symphysis. This causes ventro-lateral rotation of the acetabulum with growth of the animal (resulting in ventroversion and improved femoral head coverage). To procedure is completed with a small incision to the pubic symphysis, electrocautery is then used every 2-3 mm along the symphysis at 40 watts for 12-30 seconds. Best results are achieved in patients before 16 weeks of age (20 weeks in giant breeds) resulting in about 10-15 degrees of ventroversion if done at 16 weeks. No real benefit is gained if completed in animals greater than
22-24 weeks of age. The resultant hip changes are similar to what is seen with a DPO/TPO; however, it is easier and faster with fewer complications and no implants are needed.

A DPO/TPO involves osteotomies of the ischium (only with TPO, not with DPO), pubis, and ilium. It causes reorientation of the acetabulum to increase dorsal coverage of the femoral head (thus resulting in ventroversion). In theory there should be improved joint stability and congruence and hopefully reduction in the formation of OA. A DPO/TPO is reserved for patients that have no evidence of OA. The age restriction has been discussed as being less than 10 months of age (typically 6-9 months is ideal); however, I have performed TPOs in dogs 12-14 months of age with success if there is no OA present. It is said to result in about 92% improvement in lameness and slower progression of OA. However, the complication rate approaches near 50% and includes excessive narrowing of the pelvic canal, temporary constipation, sciatic nerve injury, implant failure, screw loosening, and continued OA development.

A FHNO has typically been reserved for smaller dogs and cats; however, larger dogs can also be candidates. It involves removal of the entire femoral head and neck and relies on the formation of a pseudoarthrosis. Even though owner satisfaction is high it is a salvage procedure with 62-65% return to normal function from a gait analysis standpoint. Probably the biggest complication with a FHNO is leaving femoral neck behind, other complications include shortening of the limb, patellar luxation, muscle atrophy, limited hip extension, recurrent lameness and chronic pain. In my hospital patients are required to undergo formal rehabilitation therapy beginning 3-5 days after surgery and continuing for 6-12 weeks.

A THA or “hip replacement” is considered by most to be the gold standard treatment for severe HD that is refractory to medical management. In the past it has be reserved for larger dogs; however, it can now be completed in smaller dogs and even cats. A THA results in about 95% return to function from a gait analysis standpoint. Often unilateral THA is enough to provide adequate function in bilateral disease. The complication is less than 10% but this is very dependent on surgeon ability. Contraindications for a THA are local or systemic infection, neoplasia, concurrent cruciate disease, or neurologic dysfunction. Potential complications include aseptic loosening, implant failure, infection, femur fracture, coxofemoral luxation, and sciatic nerve damage.

In summary, HD has a complex pathophysiology with the predominant feature being joint laxity. There are many factors that contribute to joint laxity. Clinical signs will vary depending on the stage of disease, but remember an older dog that is acutely non-weight bearing will often times have a cruciate rupture with underlying HD. A thorough physical examination with good quality radiographs is needed. Early detection is key so that way a JPS can be performed.

References
Panosteitis is a self-limiting inflammatory disease of the bone marrow of long bones. It is sometimes referred to as enostosis, eosinophilic panosteitis, and osteochondrosis/osteochondritis dessecans (OCD). While elbow dysplasia is a type of DOD disease, given its complex nature it is beyond the scope of this proceeding to try and describe elbow dysplasia.

**Hypertrophic osteodystrophy (HOD)**

A DOD in young, rapidly growing dogs also referred to as metaphyseal osteopathy, skeletal scurvy, juvenile scurvy, infantile scurvy, Moller Barlow’s disease, and osteodystrophy II. The incidence is roughly 2.8/100,000 with patients presenting between 2 and 6 months of age. HOD is predominantly seen in large and giant breed dogs. Great Danes, Chesapeake Bay Retrievers, Irish Setters, Boxers, German Shepherd, Golden Retrievers, Labrador Retrievers, and Weimaraners are at an increased risk with males 2.3 times more likely to develop HOD than females.

There are numerous proposed causes such as vitamin C deficiency, over nutrition, heritability, infections, and vaccines; however, no single cause has been determined. More recent studies have refuted the vitamin C and over nutrition theories. Heritability has been suggested for at risk breeds and has been shown in a family of Weimaraners. Infection is thought because many of the patients have a history of systemic illness with the addition of leukocytosis, although in most studies an infectious process has not been identified. One study did document an association with HOD and canine distemper virus; however, a large multi-institutional study did not support a link between HOD and canine distemper.

Diagnosis of HOD is based on signalment, history, clinical signs, and radiographs. The distal radius, ulna, and tibia are the most commonly affected bones. Clinical signs include swelling of the metaphyseal region of the bone and the lesions are often bilateral. The swelling may be warm upon palpation with varying degrees of pain and lameness (from mildly lame to a having a reluctance to want to walk. Some patients may exhibit systemic signs of illness such as hyperthermia, depression, inappetence, anorexia, and diarrhea. The pathognomonic radiographic sign is a lucent line in the metaphysis parallel to a narrow zone of increased radiodensity just adjacent to the physis (the so called “double physeal line”). There can be varying degrees of periosteal and endosteal proliferation. Differential diagnosis includes secondary nutritional hyperparathyroidism, septic polyarthritis, retained cartilaginous cores, and hypertrophic osteopathy.

In the majority of cases HOD is self-limiting within days to weeks, but can persist for months. The prognosis is typically good to excellent; however, in very severe cases death has been reported. For mild cases, analgesics along with a balanced diet should be provided. In more severe cases supportive treatment may be needed especially if the patient is reluctant to eat. Furthermore, owners should be warned about the possibly of angular limb deformity in severe cases. In Weimaraners specifically with severe HOD without bacteremia may respond better to corticosteroids than to NSAIDS.1

**Panosteitis**

Panosteitis is a self-limiting inflammatory disease of the bone marrow of long bones. It is sometimes referred to as enostosis, eosinophilic panosteitis, and shifting leg lameness with an incidence of about 2.6/1000 patients. Breeds that are at an increased risk or Airedale Terriers, Irish Setters, German Shorthair Pointers, Doberman Pinschers, Afghans, Great Danes, Saint Bernard’s, Bernese Mountain Dogs, Newfoundland’s, Mastiffs, Bassett Hounds, Rottweiler’s, Cocker Spaniels, Golden Retrievers, Labrador Retrievers, and German Shepherds. Given the list of pre-disposed breeds panosteitis is primarily a disease of large to giant breed dogs; however, it has been reported in small breed dogs. Age at presentation is typically between 5-12 months of age but up to 2 years of age has been noted. Males are affected more than females with a ratio of 4:1.

Histologically, the first changes noted consist of empty spaces in the adipose bone marrow, vascular proliferation with local bone formation around the nutrient foramen. These changes are thought to lead to vascular congestion and secondary increases in intraosseous pressure. The exact origin and etiology is still unknown.

Much like HOD the diagnosis is derived from the signalment, history, and clinical signs along with radiographs. The hallmark clinical sign is a shifting leg lameness with pain on palpation of affected long bones. The degree of the lameness and pain can be variable from mild to inability to walk. Typically the owner will report an acute lameness with no history of trauma. The most commonly affected bone is the ulna (42%), followed by the radius (25%), humerus (14%), femur (11%), and tibia (8%). Radiographs are helpful to differentiate panosteitis from other conditions (such as HOD, OCD, etc.). The appearance on radiographs will depend on the stage of the disease. In the early stage of disease radiographs may be normal or have a decrease in radiodensity in the medullary cavity near the nutrient foramen. As the disease progresses, the increase in medullary opacity will develop a granular pattern with loss
of normal trabecular pattern. Other findings include periosteal bone formation. After 4-6 weeks the densities will regress leaving a trabecular pattern that is coarser than normal.

Treatment consists of rest and analgesics; however, treatment does not influence the outcome. Recurrence is possible, but the severity will decrease over time as the dog matures.

**Craniomandibular osteopathy**

Craniomandibular Osteopathy is also known as craniomandibular osteoarthropathy, craniomandibular osteodystrophy, mandibular periostitis, lion jaw, Westie jaw, and Scotty jaw with an incidence reported as 1.4/100,000 cases. There is no reported sex predisposition; however, puppies less than 6 months of age are at the highest risk. The incidence is reported to decrease with age. West Highland White Terriers and Scottish Terriers are at an increased risk; other breeds reported to be affected are Boxers, Labrador Retrievers, Great Danes, Boston Terriers, Great Danes, and German Shepherd’s. Given the strong breed predisposition in West Highland White Terriers, a heritable etiology has been suggested, and an autosomal recessive mode of inheritance has been demonstrated in this breed.

The disease is characterized by either unilateral or bilateral symmetric irregular osseous proliferations of mainly the mandible, but the tympanic bullae can also be affected. Osteoclastic resorption of lamellar bone occurs, which is followed by the presence of primitive bone that will expand beyond the periosteum. The bone is replaced by a fibrous-type stroma and inflammatory cells invade the border of the lesion destroying adjacent connective tissue and muscle.

Clinical signs will vary from minor difficulty eating and chewing to complete inability to open the mouth and thus the inability to eat or drink. Because of the lack of nutrition additional clinical signs include weight loss, salivation, depression, and pain while eating. Physical examination will reveal enlargement or irregularity of the mandibles. The patient’s mouth may be able to be opened only partially or not at all, and the teeth are unaffected. Often times blood work and urinalysis are unremarkable. Radiographs will demonstrate bony proliferation of the mandible and/or bullae. CT can be useful to identify lesions and to better delineate the areas affected.

It can be self limiting when the dog is 11-13 months of age; however, patients may need varying levels of supportive care such as feeding soft food/gruel, syringe feeding, or placing a feeding tube. Nutrition and hydration are key parameters to monitor. Given the pain involved with trying to open or close the mouth analgesics are indicated. Over time the bony proliferations will regress completely or partially. Surgical excision of the proliferation is not recommended due to the recurrence within 3 weeks. Rostral hemimandibulectomy has been reported for a severe case that facilitated lapping of food. Euthanasia has been performed for patients with uncontrollable discomfort or with lesions that do not resolve and affect quality of life.

**Retained ulnar cartilaginous cores**

Also known as retained endochondrial cartilage cores, this is a cone of growth plate cartilage that projects from the distal ulnar growth plate into the distal metaphysis. Histologically, the retained cartilaginous core consists of viable hypertrophic chondrocytes. It is essentially failure of the growth plate cartilage to convert to metaphyseal bone, while some consider it a growth plate manifestation of osteochondrosis. Like most DOD it occurs predominately in large to giant breed dogs.

If the retained ulnar cartilaginous core is associated with reduced ulnar length then changes similar to premature closure of the distal ulnar growth plate are noted. These changes consist of cranial bowing of the radius, external rotation, and valgus deviation of the paw; additional subluxation of the carpus and elbow may be seen.

Radiographs are mainstay for the diagnosis, where a radiolucent core (typically triangle in shape) of cartilage is noted in the distal ulnar metaphysis. There may be an area of sclerosis surrounding the area. The core may extend up to 3-4 mm into the metaphysis. This must be differentiated from premature closure of the distal ulnar growth plate. No correlation has been noted between the size of the lesion, histopathology and severity of deformity.

Treatment is based off the degree of deformity. If no deformity is noted then no treatment is recommended, and the core may disappear spontaneously. In these cases it is recommended to closely monitor patients for the development of deformities, especially of the carpus and elbow. In cases with moderate to marked deformity the surgical correction of the deformity may be required.

**Legg-calve perthes disease**

Avascular necrosis of the femoral head is noted as a noninflammatory localized ischemia of the femoral head and neck, which results in deformation of the femoral head and neck leading to a pelvic limb lameness. It has also been referred to as aseptic necrosis of the femoral head, coxa plana, osteochondrosis, and osteochondritis coxae juvenilis. Small breed dogs are the most commonly affected with toy breeds and Terriers predisposed. Initially, the disease is histologically characterized by necrosis of the trabeculae of the femoral head, followed next by the fragmentation phase where loading of the affected hip causes collapse of the epiphysis and secondary thickening and cleft formation of the articular surface.
Osteochondrosis/osteochondritis dessecans (OCD)

In short OCD is a disturbance of endochondrial ossification. There is failure of the cartilage matrix calcification and vascular ingrowth, which results in cartilage retention. The cartilage retention results in thickening of the articular epiphyseal cartilage. There are two distinct areas of osteochondrosis: the growth plate-epiphyseal complex (GEC), and the articular-epiphyseal complex (AEC). Proposed causes of osteochondrosis include genetics (especially with large and giant breed dogs), rapid growth, calcium supplementation, hormonal influences, ischemia and trauma. The incidence of AEC osteochondrosis is 8.1/1000 patients with male dogs more affected than female dogs with a typical age at presentation of 4-9 months.

Proposed mechanisms for the pathogenesis of AEC osteochondrosis is it is a result of generalized disease; however, this does not adequately address the species and site-specific nature of osteochondrosis. The other proposed mechanism is it starts as a focal disease from vascular trauma and subsequent necrosis of the subchondral bone or necrosis of the epiphyseal cartilage canals. This necrosis may possibly lead to cartilage ischemia and necrosis. The necrosis may occur at a development stage when the vessels from the perichondrium are being replaced by vessels from the adjacent epiphyseal bone marrow. As this is occurring the vessels are susceptible to damage by conformational forces and/or microtrauma. If the vessels become damaged and thus necrotic then a cartilaginous infarct develops, which prevents endochondrial ossification.

Osteochondrosis latens is used to describe the initial cartilage necrosis, at this stage the disease process can be resolved or progress to osteochondrosis manifesta where larger areas of necrotic cartilage resists vascular invasion. This will then persist during growth and can be detectable. If the overlying articular cartilage fissures or fractures (thus developing a flap) then the commonly known osteochondrosis/osteochondritis dessecans (OCD) lesion develops.

The most commonly affected joint is the shoulder, followed by the elbow, tarsus, and stifle. The caudocentral or caudomedial aspect of the shoulder is affected the most commonly and it is bilateral in 27-68% patients, while the lesions of the medial aspect of the humeral condyle are common areas affected in the humerus; 96% of lesions in the stifle affect the medial femoral condyle, and in the talus the medial or lateral trochlear ridge is affected.

Clinical signs associated with osteochondrosis typically become apparent when a cartilage flap (OCD) develops. One theory is the motion between the flap and the subchondral bone, or the altered loading may provoke pain. If the flap detaches it can become what is known as a “joint mouse”, which may further contribute to synovitis and OA progression. Patients tend to present from 4-9 months of age with lameness or exercise intolerance. Many cases will show signs of a unilateral lameness; however, the disease is commonly noted bilaterally so careful examination of the contralateral joint is warranted. Once a patient presents with clinical signs of a lameness the OCD lesion is considered chronic and a defect in the subchondral bone is well developed. Radiographs are a sensitive diagnostic method that may demonstrate disruption of the subchondral bone with flattening or concavity of the normal contour. Sclerotic margins may be seen around the defect. Contrast arthrograms can be used to demonstrate unmineralized cartilage flaps and joint mice, effusion, and new bone formation. CT is also useful in demonstrating an OCD lesion. Arthroscopy is useful as both a diagnostic and therapeutic modality.

Aims of treatment need to include elimination of pain and lameness, restoration of the cartilage surface with tissue of similar nature to the native tissue, normalization of joint biomechanics, and prevention of further joint degeneration. Conservative management may be recommended with small subchondral lesions and when the patient is mildly lame or asymptomatic. This form of treatment is only recommended for dogs younger than 6 months of age. Conservative management consists of NSAIDS, exercise restriction, chondroproctants, rehabilitation therapy, and weight control. Persistence of clinical signs suggests the patient should be treated with a surgical approach.

Surgical management consists of either an arthrotomy or arthroscopy (the authors preferred method). Surgical treatment consists of flap excision and joint mouse removal. Additional treatment may consist of removing peripheral cartilage that is not firmly adhered and stimulation of fibrocartilage to the underlying subchondral bed.

OCD of the shoulder usually carries a good to excellent prognosis; however, other joints affected with OCD carry a guarded prognosis with continued progression of OA and an intermittent lameness.

References
It’s a Cruciate Rupture, and Surgery is not an Option. Now What?

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Cranial cruciate ligament (CCL) rupture is one of the most common orthopedic conditions encountered in the dog. In fact, over 1 billion US dollars are spent every year in dealing with the canine stifle. When dealing with hind limb lameness many dogs we see have some degree of hip dysplasia or degenerative changes in the hip; however, an acute lameness is typically not due to a hip problem. In fact 32% of dogs referred for hip problems actually have evidence of cruciate disease. About 33-50% of dogs will present with bilateral disease even if they have a unilateral lameness. Severe bilateral cruciate disease can often mimic other conditions such as severe hip dysplasia or neurologic disease. Therefore, a general rule of thumb is a hind limb lameness in a dog is cruciate disease until proven otherwise.

Personally for me, statements that I do not like are:

- All dogs that rupture their CCL must have surgery
- All dogs with CCL ruptures have joint effusion
- All surgical procedures (extra-capsular repair, TPLO, TTA, XYZ) have the same outcome
- A dog can’t return to pre-injury status following a CCL rupture
- Dogs don’t benefit from rehabilitation therapy either with a conservative approach or following surgery

Anatomy
The stifle is considered a complex condylar synovial joint because the articular cartilages are separated by an intra-articular fibrocartilage or the menisci. The primary functions of the stifle are flexion, extension, and rotation. There are lots of structures that work together that make up the anatomy of the stifle such as the femur, tibia, patella, the soft tissue structures, as well as the intra-articular structures. There are 3 bones that make up the stifle. The femur has 3 major articular areas with 2 condyles that are convex, while the proximal tibia has 2 condyles that are convex. The femoral condyles are separated by the intercondylar eminence and also contain the intercondylar area, which serves as the attachment site of the CCL. The patella is the largest sesamoid bone in the body and articulates with the femoral groove. The patellar ligament is the portion of the quadriceps femoris between the patella and the tibial tuberosity, which is sometimes used interchangeably with patellar tendon. The soft tissue structures of the stifle are the medial and lateral meniscus, which are attached to the proximal tibia by paired meniscotibial ligaments. The primary ligamentous support of the stifle comes from the medial and lateral collateral ligaments as well as the cranial and caudal cruciate ligaments (CdCL). The cruciate ligaments are intra-articular but covered in synovium so they are considered extrasynovial.

The menisci are C-shaped disks of fibrocartilage that act as functional extension of the tibia. They are a true example of a specific structure function relationship. The cranial and caudal meniscal horns are attached to the bone through the cranial and caudal meniscotibial ligaments. There are 4 total: a cranial and caudal for each medial and lateral meniscus. What’s important about the anatomy of the meniscus is the difference between the medial and lateral aspects. The medial meniscus is firmly attached to the medial collateral ligament and the joint capsule making it relatively immobile such that its motion is coupled with that of the tibia. On the other hand the lateral meniscus is less firmly attached to the tibia. It also has a meniscofemoral ligament caudally. Its motion is more coupled with the femur and therefore is less likely to be injured compared to the medial meniscus. The meniscus has a wedge shape that causes radial extrusive forces to develop from compressive forces. The primary function of the meniscus is for load bearing, load distribution, shock absorption, and joint stability. Because of its shape it acts as a spacer and bears about 40-70% of the load.

So why does the meniscus matter anyways? As already discussed the meniscus accepts high loads during weight bearing but also absorbs energy. It does this by undergoing elongation as a load is applied. As the joint compresses the wedge shape extrudes peripherally and the circumferentially oriented collagen fibers elongate. This is known as hoop stress. The hoop stress is then transmitted to the tibia. The meniscus also provides a concavity to the convex tibial plateau. Several studies have shown the importance of the meniscus. For example removal of the caudal horn of the medial meniscus leads to a focal area of high pressure in that area. This alteration of the articular cartilage contact may contribute to degenerative changes following a meniscectomy. Furthermore, a meniscal release causes a 140% increase in peak contact pressure and a 50% decrease in contact area.1,2

Physiology
The primary motion of the stifle in the sagittal plane is flexion and extension while secondary motion is rotation. In Labrador Retrievers the normal range of motion is 41 and 161 degrees of flexion and extension.1 During extension of the stifle the medial and
lateral collateral ligaments are taut and therefore act as the primary stabilizers that limit internal and external rotation. During flexion the lateral collateral ligament relaxes while the medial remains somewhat taut. This allows the lateral femoral condyle to displace caudally and results in internal rotation. Then as the joint is extended the lateral collateral tightens up drawing the lateral condyle cranially and resulting in external rotation. In humans this is known as the screw home mechanism. The CCL functions to limit internal rotation, hyperextension, and tibial subluxation. The CCL is made up of two bands: the craniomedial and the caudolateral. The craniomedial band is primarily responsible for preventing the cranial translation of the tibia while the caudolateral band is responsible for secondary prevention of cranial translation of the tibia. The CCL and the CdCL do indeed cross themselves (hence the term cruciate which means to cross) and both the CCL and the CdCL play a partial role in preventing rotation of the stifle.

**Pathophysiology**

CCL rupture is typically considered to be degenerative in nature and often bilateral. In fact 33-50% of dogs that present with a unilateral lameness will have bilateral disease. It was first described in 1926 and to this day we still don’t know the exact mechanism of action. Proposed mechanisms include immune-mediated conditions, age and time of neutering, confirmation, obesity, lack of fitness, increased TPA, chronic stress, and the list goes on. Purely traumatic ruptures can occur but this is rare. It occurs when supraphysiologic loads are placed on the CCL, which results in a mid-substance “mop end” tear. In the CCL deficient stifle the limb function is altered such that the limb is more flexed throughout the gait cycle most likely as a way to minimize pain and weight bearing on the affected limb. From a kinetic standpoint the peak vertical force (PVF) and vertical impulse (VI) is decreased after a CCL tear. For example in a sound limb the PVF was found to be 70% of the static body weight (BW) of the dog. In the CCL deficient stifle the PVF was 25% at 2 weeks, 32% at 6 weeks and 37% at 12 weeks. Furthermore, tibial subluxation has been noted to be 8-12 mm and even up to 5 mm 2 years after injury. Interestingly there are not really any changes in internal rotation following a CCL rupture. There is evidence of increased meniscal damage and joint capsule fibrosis as well as progression of osteoarthritis (OA). Once the CCL is ruptured the caudal pole of the medial meniscus acts as a wedge preventing the tibia from further subluxation. However, the 2-edged sword aspect of this is that this wedge shape coupled with the anatomy of the medial meniscus also increases the risk of a meniscal tear in the untreated stifle.

**Diagnosis**

The diagnosis is typically straightforward and is based off the history, signalment, clinical signs, physical exam, and orthopedic exam. The history may include an acute or chronic hind limb lameness that may be mild to non-weight bearing. Interestingly, owners may report that the lameness has improved from initial injury. This usually corresponds to the timeframe from when the initial inflammatory response is ending. Regarding the signalment any age or breed can be affected. Typically we tend to see medium to large breed dogs that are around 3-8 years of age. The orthopedic exam is mainstay to diagnosing a CCL rupture. Findings may include a positive sit test where the dog will tend to sit with the affected leg projecting out to the side. Pain on hyperextension is usually the forgotten test but is very reliable. Most affected dogs will exhibit some degree of pain. Crepitus may be noted during ROM, and with chronic tears medial buttress formation may be noted. This is the peri-articular fibrosis that occurs. The classic findings for a CCL rupture are joint effusion, the cranial drawer test and the tibial compression test. A simple way to think about it, is that in an adult dog joint effusion will only be caused by a CCL rupture, septic arthritis, tick-borne disease, or immune-mediated arthritis. A medial patella luxation (MPL) will not cause the same degree of joint effusion, so if you have a patient will underlying MPL that develops joint effusion be thinking about a CCL rupture.

The cranial drawer test is testing for laxity in the CCL, but this is more of a passive test and does not mimic weight bearing. To perform the test one hand is placed on the distal femur with the thumb behind the lateral condyle. The other hand is placed on the proximal tibia with the thumb behind the fabella. The goal is to move the proximal tibia cranially in relation to the femur. Always check drawer in flexion and extension. When checking for partial tears the CCL has two bands, the craniomedial which remains taut in both flexion and extension and the caudolateral, which is taut in extension but lax in flexion. For example if the craniomedial band is torn and the caudolateral band is intact cranial drawer is only present in flexion because in extension the caudolateral band is taut. If the caudolateral band is torn and the craniomedial band is intact no cranial drawer is present because the craniomedial band is taut in both flexion and extension. Cranial tibial thrust is a test meant to mimic active weight bearing. The goal is to hold the stifle at a standing angle (approximately 135 degrees) and while holding the stifle still flex the hock. If the CCL is ruptured there should be a cranial displacement of the tibia. As with cranial drawer, tibial thrust should be checked in both flexion and extension.

Radiographic evaluation will help to see evidence of joint effusion with cranial displacement of the intrapatellar fat pad. With chronic CCL ruptures you may see evidence of OA and if you are lucky the stifle is sitting in drawer on the radiographs. Some people have proposed a stable stifle with joint effusion and a hind limb lameness may be evidence of a partial tear.

**Treatment**

When deciding on a treatment plan there is no one treatment fits all, but there are many, many, many options available. The reason there are so many options is because not one procedure or medical management technique is 100% perfect. I think one reason for this
is because what is considered our final outcome, a stable stifle, a patient that returns to activity pain free, elimination of OA, owner satisfaction, etc.? We will never be content on cruciate disease until we figure out the goals we want to achieve for an outcome.

When I approach a dog with cruciate disease I'm going to have the same conversation with each owner; however, depending on each case I may swing my conversation in one particular direction. Factors I consider when deciding on conservative vs. surgical treatment and which procedure are the patient, owner, and veterinarian factors. I look at the breed, the size of the animal, the age, the activity level, and what is that particular animals job. Are they a pet, an athlete, or a service dog? Regarding the owner I talk to them about their perceived outcome, their ability and willingness to follow directions post operatively, as well as finances. And then I look at my abilities such as what equipment I have available, what procedures am I comfortable doing, and what good and bad outcomes have I had with certain procedures.

When I first tell owners that their dog has a torn cruciate I try to cover 3 main options. Option 1 is we do nothing. By do nothing I mean we cage confine for 6 weeks with medical management (analgesia and NSAIDS) and (hopefully) formal rehabilitation therapy. The most important aspect here is confinement. These owners have to be aware the goal of conservative management is to allow periarticular fibrosis to occur. This can’t occur with the dog remaining active. To break it down to them I tell the owners the dog must be kept in an area where he/she can stand up, lie down, and turn around. The dog eats, drinks, and sleeps in the crate. It only goes outside to urinate and defecate on a leash then back into the crate. I also throw the disclaimer in that in my opinion OA is worse with a rapid progression as long as the stifle is unstable and usually if this is a larger dog they wont return to full function. I also really push the fact that the dog will appear to be do “okay”; however, they have a very high chance of developing a meniscal tear. I tend to tell owners its not “if” but more of a matter of “when” they tear their meniscus. Personally, I am not a fan of this approach!

Option 2 is a conservative approach with exercise restriction, formal rehabilitation therapy, and a custom made stifle orthotic. While this approach parallels that of option 1, we can in theory attempt to help stabilize the stifle with a brace. In human medicine, knee braces are commonly used for multiple conditions. Bracing of the human knee has been shown to enhance proprioception/joint position sense, permit the injured limb to relax, reduce fatigue in injured limb, provides some mechanical protection against impact, and slow movement down to allow muscles time to react and control motion. Categories of knee braces in human medicine include the following: prophylactic (prevent or reduce severity of knee injuries in contact sports), functional (provide stability for unstable knee, rehabilitative (allow protected and controlled motion during the rehabilitation of injured knees), and patellofemoral (improve patellar tracking and relieve anterior pain). Only functional knee braces are utilized in veterinary medicine.

In theory the brace should help limit tibial subluxation. At the authors institution (unpublished data) we did find improved objective gait analysis when a custom stifle brace was worn versus when not worn; however, the gait analysis was not improved equal to that of surgery. This data reveals that a brace is not considered equal to or meant to replace surgery; furthermore, it must be worn for the duration of the pet’s life.

**My issues with stifile orthotics are as follows**

1) Tolerability: I cant ask the patient if he/she will tolerate the brace, I have had some dogs that don’t mind it at all, others take time, and some just freeze or try to chew it. The other issue is given the different shapes and sizes of dog stifles the brace MUST be custom made. This means a mold must be made and sent to the orthotist and then sent back about 2 weeks later. It’s a horrible feeling to have owners its not “if” but more of a matter of “when” they tear their meniscus. Personally, I am not a fan of this approach!

2) Arthritic progress: What I can tell an owner is that with surgery we can slow down and minimize arthritic progression. Without surgery we will have continued accelerated and worsening progression OA. Along that scale is a brace; I just don’t know if the scale is closer to that of surgery or that of no-surgery?

3) Meniscal damage: What I can tell an owner is that with surgery we can minimize the chances of a meniscal injury. Without surgery there is a high incidence of meniscal injury. The problem is again along that scale I don’t know where a brace will fall. Will it help protect the meniscus the same as surgery, or will it not make a difference such as doing nothing? This does bring up a good point about meniscal damage. A “meniscal click” will only get you about 30-40% correct at identifying a meniscal injury. If you add in a positive McMurray test and pain on hyperflexion that may improve to about 50%. Personally, I feel as if a dog has a meniscal tear they will not benefit from a brace because it will do nothing to help with the pain and discomfort. The problem is if at best you can diagnose a meniscal injury in 50% of patients then how does one approach determining if there is meniscal injury? A MRI could be considered but is costly and requires general anesthesia, arthroscopy could be considered but personally would be below the standard of care to go to surgery to identify a meniscal injury but not treat the CCL rupture. Therefore, if I have owners that want their dog in a brace then they must undergo a stifle ultrasound. If there is evidence of meniscal damage then that dog will not be a good candidate for a brace, if they don’t appear to have meniscal damage then we can give it a shot knowing that an ultrasound is not 100%.

Option 3 is surgery with various means such as an extracapsular technique, tibial plateau leveling osteotomy (TPLO), tibial tuberosity advancement (TTA), etc., etc., etc. Granted I’m a surgeon, but option 3 to me is still the best option if I have a patient that can tolerate surgery. For me I prefer the TPLO. At our institution following a TPLO our patients have about a 96-98% return to pre-injury status. Granted owners may want to avoid surgery; however, with a TPLO and formal rehabilitation therapy these patients should be back to normal activity in about 12-16 weeks time.
References
Osteoarthritis (OA) is one of the most common chronic musculoskeletal diseases and causes of lameness in the dogs. The osteoarthritic disease process involves the entire synovial joint, encompassing the synovium, cartilage and underlying bone. Joint failure results from an abnormal mechanical strain causing damage to normal tissue or failure of pathologically impaired articular cartilage and bone under the influence of normal physiological strain or a combination of both. In both cases, the end point is cartilage loss and joint impairment. Osteoarthritic chondrocytes show an altered phenotype characterized by an excess production of catabolic factors, including metalloproteinases and reactive oxygen species (NO). These factors constitute potential therapeutic targets and some new drugs and nutraceuticals have been proposed to promote the return to homeostasis.

It is important to remember that the pain of OA is not felt in the articular surfaces, instead the peri-articular structures such as the inflamed synovium, fibrotic joint capsule, or weak tendons, ligaments or muscle. OA is a disease of the entire joint involving synovitis, atrophy and fibrosis causing pain and progressive degenerative disease.

In recent years the human literature has identified OA pain as maladaptive pain that resembles true neuropathic pain. Maladaptive pain is pain as a disease and involves the creation of peripheral and/or central sensitization.

Pharmacological pain relief
The first line drugs for treatment of osteoarthritis are the Non Steroidal Anti-Inflammatory Drugs (NSAIDs). A number of NSAIDs have been approved for use in dogs and fewer in cats. Generally NSAIDs inhibit one or more steps in the metabolism of arachidonic acid. This class of drugs ameliorates the symptoms of osteoarthritis but also has a role in preventing central sensitization through COX inhibition.

Actions of NSAIDs
Stresses on the joint lead to production of inflammatory cytokines released by synovial cells, chondrocytes, macrophages and fibroblasts. These proinflammatory cytokines, including certain interleukins and TNF-alpha, lead to upregulation of COX-2 enzymes and production of eicosanoids such as PGE2, and the upregulation of matrix metalloproteinases. Normally during metabolism PGs are broken down by enzymes matrix metalloproteinases (MMPs) and aggrecanase. In acute inflammation MMPs increase in number and disrupt the balance of production and destruction in the joint. There is shift toward breakdown of articular cartilage resulting from an imbalance between MMPs and their TIMP inhibitors, leading to thinning and destruction of the cartilage tissue and perpetuation of the inflammatory cascade with PGE2 production and subsequent pain.

NSAIDs block PG synthesis by binding to and inhibiting COX. The major therapeutic and toxic effects of NSAIDs result from this action. The major “safe” NSAIDs are said to be COX2 selective although these do have some COX1 effects.

Adverse events
Adverse side effects of NSAIDs can include gastric upset, vomiting, diarrhea, inappetence, gastric bleeding, platelet inhibition, analgesic nephropathy, liver and cardiac problems. Inappetence is the most common side effect in cats.

Most adverse events occur within 2 to 4 weeks of commencement of the NSAID and stop soon after drug is discontinued. NSAIDs can cause gastric erosions but unlikely that these would occur without clinical signs. Perforations are most likely caused by concurrent use of steroids and NSAIDs or by using high doses of NSAIDs.

Nephrotoxicity can be seen in patients with pre-existing renal disease, hypotension, hypovolemia, congestive heart failure or diuretic administration. Hepatic necrosis appears to be due to an inherited sensitivity to the molecule used and not a true toxicosis.

Common NSAIDs

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<th>Trade name</th>
<th>Dose</th>
<th>Species</th>
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<tr>
<td>Carprofen</td>
<td>Rimadyl—Zoetis</td>
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<td>Deracoxib</td>
<td>Deramaxx- Elanco</td>
<td>1-2 mg/kg q 24 hours</td>
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<td>Previcox—Merial</td>
<td>5 mg/kg q 24 hours</td>
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Meloxicam

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<tr>
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<td>0.1mg/kg q 24 hrs-dogs</td>
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<td></td>
<td></td>
<td>0.05 mg/kg q 24 hrs -cats</td>
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Robenacoxib

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<th>Dose</th>
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<tbody>
<tr>
<td>Robenacoxib</td>
<td>Onsior-Elanco</td>
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<td>Dogs and cats</td>
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Long term use and safety in OA patients

- Use a veterinary approved drug at label dose—can be used long term and may show improvement in disease from 6 months to 1 year
- Meloxicam and Robenacoxib are metabolized in cats by oxidation not glucuronidation. Long term oral use has been safely demonstrated.
- No one veterinary approved NSAID has been proven to be safer than another.
- Veterinary approved products are safer than non veterinary approved products.

Nutraceuticals that work as well as drugs (and are proven winners)

Omega 3 fatty acids

There are a number of Randomized Controlled Clinical Trials (RCCT) proving the efficacy of Omega 3 fatty acids—fish oil or Marine Oil (Algae Oil) but not flaxseed oil.

Arachidonic acid is the primary substrate for the lipoxygenase (LOX) and cyclooxygenase (COX) enzymes. This fatty acid is derived dietary sources and stored in phospholipids of the cell membrane until needed. AA is a member of the omega-6 fatty acid family. AA can be partly replaced in cell membranes by the omega-3 fatty acid EICOSAPENTANOIC acid (EPA). EPA can be used by the LOX and COX enzymes to produce eicosanoids. When EPA is used by the COX and LOX enzymes, they produce the eicosanoids PGE3, thromboxane (TX) A3 and LTB5, which are less active and relatively anti-inflammatory compared to their counterparts produced from AA.

Omega 3 Fatty Acids can be supplied by supplemented diets (Hill’s J/D, Purina JM and RC Mobility Support) or directly supplemented from fish oil capsules or liquid. Dose for supplementation varies but most accepted is:

Injectables

Polysulfated glycosaminoglycans

Adequan (PSGAG) and Cartrophen (Sodium pentosan polysulfate) are the 2 products that are available.

Polysulfated glycosaminoglycans (PSGAGs) are a semisynthetic product (derived from bovine trachea) structurally similar to the GAGs found in articular hyaline cartilage. PSGAGs stimulate collagen synthesis and inhibit collagen breakdown as well as decrease pain and inflammation. Several studies have documented positive effects when administering PSGAGs (Adequan) to dogs with hip dysplasia and osteoarthritis. One study found decreased hip laxity in dogs treated with Adequan twice weekly from 6 weeks to 8 months of age compared to age-matched controls. It is recommended to begin treatment as early in the disease process as possible in order to slow the progression of cartilage damage. The strength of evidence for PSGAGs used at the labeled dose is considered high.

Dose: 5mg/kg once weekly x 4 to 6 weeks then once monthly in dogs, cats first 4 weeks is the same but 2nd month every other week then once monthly

Cartrophen

Pentosan polysulphate—this product is used in Canada, Europe and Australia. Similar actions to Adequate. Dose is 1ml/33kg once weekly for 4 weeks then once monthly.

Other drugs for chronic/ maladaptive pain

Tramadol

In humans tramadol is known to exert its pain modifying effect through two metabolites; one enhances inhibitory neurotransmitters (serotonin, norepinephrine), and the other (0-desmethyltramadol, or “M1”) metabolite is a weak opioid (1/100th the mu-receptor affinity of morphine.) Tramadol has a very short half-life (1.7 hours) in the dog, and it appears that dogs produce very little of the M1 opioid metabolite. Evidence for a pain-modifying effect of oral tramadol remains unknown at this time. Plasma levels in dogs are much lower following oral administration than in humans. One study of oral tramadol reports a statistically significant increase of mechanical threshold levels, but only at the 5- and 6- hour time point. One study does find oral tramadol effective as part of a multi-modal analgesic protocol to control cancer pain, but others have found it (not unsurprisingly) inferior as a solo agent to multi-modal pain management.
Gabapentin
Gabapentin is said to be effective in neuropathic pain states such as post-herpetic neuralgia (shingles) in people. Gabapentin binds to the alpha 2 delta subunit of the voltage-gated calcium channel resulting in the decreased release of excitatory neurotransmitters such as glutamate. It also increases brain concentrations of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter. It has also been used in people for fibromyalgia and diabetic neuropathy pain, and restless leg syndrome, and in acute pain states it may reduce the opioid need of some patients.

Gabapentin is used in dogs with neuropathic pain or in dogs who phenotypically appear as if they have neuropathic pain i.e. osteoarthritis. Dosage of this drug is usually 10 mg/kg BI but geriatric dogs may need a decreased dose of 5,g/kg.

Amantadine
Amantadine is an antiviral drug but it also increases concentrations of dopamine in the CNS as well as being an antagonist at the NMDA receptor. It affects central pain sensitization via NMDA receptor and appears to enhance the analgesic effects produce by opioids, NSAIDs and gabapentin. In dogs, one clinical study using 3 to 5 mg/kg once daily in combination with meloxicam showed significant improvement using client-specific outcome measures for activity on day 42 of administration but not on day 7 or 21. This may be a function of dosage frequency as pharmacological data indicate twice daily dosing is more appropriate.

In cats, there is very good oral bioavailability but a short half suggests twice daily dosing in the similar range to dogs. Central sensitization must be present for efficacy to be demonstrated.

Dosage is usually 10 mg/KG BID or in cats 3 to 5 mg/Kg BID to start

Amitriptylline
Amitriptyline and other TCAs are commonly used in neuropathic pain in people. They produce serotonin and norepinephrine reuptake inhibition, some NMDA antagonism, sodium channel blockade and are anti-inflammatory. In the dog suggested dosage is 3-5 mg/kg every 12 hours.

Acetaminophen
Contraindicated in cats! It has been used in dogs for a washout period if switching NSAIDs and may be combined with codeine or tramadol. May be beneficial for dogs with renal dysfunction but should not be used immediately postoperative. Even at recommended doses there is some potential for toxicity. Dose: 10 – 15 mg/kg PO q8h; if using long-term (>5 days) consider giving q12h at the lower end of dosing range.

Oral opioids
Maladaptive pain secondary to peripheral nerve damage shows decreased sensitivity to opioids. Oral opioids have a very low bioavailability due to metabolism in the liver. Codeine has the highest bioavailability and is often combined with acetaminophen in dogs only.

Dose: 1-2 mg/kg q 4 hours

If combined with acetaminophen dose on the acetaminophen fraction and do not exceed 2mg/kg of codeine.

Cortisone
Corticosteroids are usually the last drug used and do not analgesic but do reduce inflammation. Intra articular injections are common in humans and becoming more common in dogs. Intra articular steroids have been shown to protect articular cartilage in experimental canine OA; however, repeated use may also have deleterious effects on joint tissue from suppression of cartilage matrix synthesis. Benefits usually outweigh risks. Strict aseptic techniques are needed for these injections.

On the horizon
A new EP4 receptor blocking drug, grapiprant, will soon be coming to market. It is rumoured to replace NSAIDs in dogs and will have applications in cats as well. The company producing this drug is Aratana. It should be to market in 2016.

References
Nonpharmaceutical Treatment of Osteoarthritis:
Rehabilitation, Acupuncture, and More
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For many years OA was managed with a single pharmaceutical agent if and when the clinician determined the animal was suffering. Recently it has been realized that pain is a very complex process and involves signalling molecules, pathways, substances, receptors and transmitters with different modes of action. It is unrealistic to think only one pharmaceutical could be effective in eliminating chronic pain. It is equally unrealistic to think that drugs alone can manage OA effectively for the life of the animal. A multimodal approach to the treatment of OA is necessary and the best approach.

Weight loss and diet
Prevalence of OA is likely close to 60 % of all dogs and over 50 % of all dogs are overweight. Excess weight causes an excess load on an abnormal joint, creating more pain. In addition the adipose tissue secretes adipokines which are pro-inflammatory and these increase the overall inflammation in the joints and elsewhere in the body.

Purina Longevity study determined that dogs who are normal weight live on average 2 years longer than their obese siblings. Musculoskeletal problems, especially OA were the leading cause of death or euthanasia and doubled the need for pain medications.

To have success with weight loss, you need to be able to calculate the Resting Energy Requirement ( RER) and determine calories needed for weight loss. Generally for weight loss the pet needs 60 to 80 % of RER. A weight loss diet that is higher in protein ( minimum 1 g/lb of body weight) tends to maintain lean body mass. Just having the client cut down the number of calories with their existing diet can promote muscle loss instead of fat loss, so true weight reduction diets are needed. Here is an example of how to calculate amount of food needed.

Determine RER from Body Condition Score on a 9 scale. For every point that the dog or cat is overweight over the ideal 5/9 body condition score, the pet is 10 % obese. For example a 10 kg cat with a body condition score of 7/9 is 2 x 10% or 20% over ideal weight. To determine Lean body weight in kilograms 10/1.20 = 8.3kg. Take this lean body mass of 8.3 and raise it to the 0.75 power. Multiply this result by 70 to give the RER. In this case a cat with a lean body mass of 8.3 kg has a RER of 342 kcal. Multiply this by 0.8 = 272 kcal. This is the number needed for weight loss. Had the example been for a dog I would have used 7. Use this formula and this calculation rather than amount on bag or can for weight loss. If supplementing with Omega 3 fatty acids be sure to take those calories into account. Consider using 1/2 joint diet and 1/2 weight loss diet and then adding additional Omega 3s for cats or using one of the new combination weight loss and joint health diets (Hill’s).

Environmental modification and assistive devices
Simple environmental modifications can have a positive effect on old painful patients. Raising food and water dishes, putting down area rugs or carpet to reduce slipping, installing ramps and using baby gates to limit dangerous areas are all good ideas for household modifications. A foam bed or other soft area to lie on can cushion old joints. Harnesses, slings, booties, power socks, braces and orthotics are all examples of assistive devices that can be used. Sometimes carts and wheel chairs are also necessary.

Acupuncture
Acupuncture can be used to relieve pain, cause an autonomic nerve response, increase the rate of nerve regeneration, and cause surgical analgesia. Studies have found that acupuncture and non-acupuncture points were differentiated by their connection to different pathways in the central nervous system. They found that the pathway connected to the acupuncture point is different from the pathway connected to the non-acupuncture point. In addition, the pathway connected to the non-acupuncture point is inhibited within the lateral periaqueductal gray when the analgesic inhibitory system (AIS) is activated. They also found that analgesia caused by stimulation of the acupuncture point is naloxone reversible, while that caused by stimulation of the non-acupuncture point after a lesion in the AIS is dexamethasone reversible. Stress-induced analgesia caused by low frequency electrical shock was naloxone as well as dexamethasone reversible.

There are multiple theories as to how acupuncture works in humans and animals alike. It is important to understand that no one theory explains all the different effects of acupuncture. Just as research is continuously being done to further develop western medicine, additional research is being done with both human and animal acupuncture to further our understanding of this ancient healing art. The most current theories are: 1) The Gate Theory; 2) Endogenous Opioid Theory; 3) Autonomic Nervous System Input Theory; 4) Humoral Theory; 5) Bioelectric Theory; and 6) Traditional Oriental Medicine Theory.

1. The gate theory
A beta sensory neurons close the gate to larger pain fiber sensations.
2. Endogenous opioid theory
Studies have found that acupuncture analgesia could be reversed by naloxone. It was also determined that a cross-tolerance can
develop between acupuncture and morphine. Levels of the opiate peptide NAGA and beta-endorphins were shown to increase in the
brain and cerebrospinal fluid (CSF) after acupuncture. It has also been shown that levels of met- and leu- enkephalins significantly
increase in the brain after electrical acupuncture. Opiates are also known to have systemic effects that can be produced by
acupuncture. For example, opiate receptors in the gut are responsible for decreasing peristalsis and increasing segmental contractions,
thus effectively controlling diarrhea.

3. Autonomic nervous system input theory
Type A-delta visceral and somatic fibers have a similar distribution in the dorsal gray matter and tract of Lissauer. Inputs from both
converge in the spinothalamic tract. Visceral A-delta fibers form reflex arcs with propriospinal afferents, and can cause muscle
cramping secondary to visceral inflammation. Conditions of somatic pain can also cause visceral manifestations of disease. These
interactions account for the phenomenon of “referred pain.” Stimulation of acupuncture points can cause a reflex arc, resulting in
sympathetically induced segmental superficial and visceral vasodilation. This explains how acupuncture can be effective in the
treatment of internal organ dysfunction.

4. Humoral theory
This theory was first postulated after studies showed that a transfer of blood, CSF, or brain tissue from an animal under acupuncture
analgesia to an animal not receiving acupuncture resulted in analgesia of the recipient. This analgesia was generalized and reversed by
naloxone. The analgesia level required for surgery took twenty to thirty minutes of stimulation to reach its peak and lasted hours after
stimulation of the points had ceased. Acupuncture has also been shown to cause systemic increases in growth hormone, prolactin,
oxytocin, luteinizing hormone, white blood cells, immunoglobulins, antibodies, and interferons depending on which points are
stimulated(66).

5. Bio-electric theory
Becker and Reichmanis, in 1976, proposed a theory that the healing and analgesic properties of acupuncture are based on a DC
current system. In this system, electric signals are generated and propagated by Schwann cells, satellite cells, and glial cells.
Acupuncture points, like amplifiers, would boost the DC signal along the nerve pathways. Insertion of a metal acupuncture needle
would, in effect, short-circuit the system and block pain perception.

6. TCM theory
According to TCM theory, Qi, also known as Chi, energy, or life force, circulates through each of the meridians or channels every
twenty-four hours. Each channel is connected energetically to a TCM organ. The channels derive their names from the organ upon
which they have the greatest influence. A blockage of Qi circulation manifests as dysfunction or disease. By stimulating or sedating
energy levels at acupuncture points, the body is brought into balance and healing is facilitated.

Physical rehabilitation
The goals of rehabilitation include the restoration, maintenance and promotion of optimal function and quality of life as they relate to
movement disorders. The majority of rehabilitation therapeutics involves manual therapies including joint mobilizations, and
therapeutic exercises. Equipment used on a regular basis in veterinary rehabilitation includes physioballs, therapy bands,
rocker/wobble boards, cavaletti poles and land treadmills. Hydrotherapy equipment can include pools, resistance pools and underwater
treadmills. Modalities such as hot and cold therapy, laser, electrical stimulation, shock wave therapy and therapeutic ultrasound can
also be used. Regenerative medicine with platelet rich plasma and stem cells is now also a part of rehabilitation and pain management.

Manual therapies
Joint mobilizations—a manual technique used to assess a joint and improve its movement (arthrokinematics). Joint mobilizations
improve joint lubrication, modulate mechanoreceptors, and decrease sensory input thus relieving pain. Therapeutic glides are ranked
Grade I to V using the Maitland Mobilization Scale.

Massage is soft tissue massage and soft tissue mobilization. Massage can decrease excessive tissue tension by aiding in removal of
chemical substances in soft tissue that activate chemical nociceptors. Soft tissue massage can also, by the Gate Theory, reduce pain by
stimulating large rapidly conduction fibers, selectively closing the gate against smaller pain fiber input.

Thermal therapy
The effects of thermoderapy are vasodilation with secondary increased local circulation, decreased pain, relaxed muscle tone, reduced
muscle spasm, increased tissue extensibility, increased cellular metabolism, and increased local tissue oxygenation. Heat is generally
used to reduce pain from arthritic, trigger points and muscle spasms, and to prepare tissues for exercise or stretching. Precautions of
using heat therapy include impaired thermal sensation, recent hemorrhage, malignancy, and acute inflammation. Heat can be applied
by gel packs, hot towels, or therapeutic ultrasound.
Cryotherapy can be applied via ice bath, ice massage, ice pack, vapocoolant gel, or circulating ice compression units. The beneficial effects of cryotherapy include vasoconstriction; reduced cellular metabolism; decreased nerve conduction velocity, and decreased production of pain mediators, leading to analgesia; reduction of edema and decreased muscle spasm. Metabolism may be decreased by more than 50%, which facilitates oxygen diffusion into the injured tissues. Joint range of motion is improved through suppression of excitatory muscle spindle afferents. Intermittent pneumatic compression, when combined with cryotherapy has been shown to prevent edema formation, increasing blood flow, and stimulation of tissue healing. Although static compression is effective in edema reduction, intermittent compression optimizes lymphatic drainage. Game ready is commonly used for pain reduction post surgery.

Laser
LASER” is Light Amplification by Stimulated Emission of Radiation. By definition, a laser must be collimated and monochromatic. Penetration of laser energy is determined by the wavelength, and many wavelengths are patented. The physiological effects of laser stimulation include accelerated cell division via mitochondrial stimulation, increased leukocyte phagocytosis, stimulation of fibroblast production, enhanced synthesis of ATP, and angiogenesis. Treatment with laser is indicated for pain management, control of inflammation, and tissue healing.

Electrical therapy
Electrical stimulation (ES) can affect the sensory and the motor nerves. Indications for ES include wound healing, pain control/relief, reduction of inflammation, muscle re-education, reversal of atrophy, and strengthening. Electrotherapy works at the cellular level to cause excitation of nerve cells, changes in cell membrane permeability, and stimulation of protein synthesis, osteosynthesis and fibroblast formation. On the tissue level, electrotherapy causes skeletal muscle and smooth muscle contraction. On the segmental level, electrotherapy facilitates muscle-pumping action, which improves joint mobility and circulatory and lymphatic drainage. ES can be TENS or NMES.

Sound therapy
ESWT has been applied to painful OA lesions in veterinary practice, including hip and elbow dysplasia and Supraspinatus tendinopathy with excellent pain relief results being reported. ESWT works by releasing a sudden high-powered shock wave resulting in tissue modulation in a very focused depth of tissue. This modality does require deep sedation or anesthesia as the treatment is uncomfortable for the patient, however the patient experiences pain relief immediately post treatment, which can last for days to weeks. The mechanism behind the pain-relieving function of ESWT is thought to be due to increased serotonin activity in the dorsal horn, and descending inhibition of pain signals.

Therapeutic exercises
Therapeutic exercise contributes to pain management through Exercise Induced Hypoalgesia (EIH) which results from activation of the opioid system with beta-endorphin release from the pituitary. It is also believed that exercise can activate large afferents and that mechanical hypoalgesia is induced by repeated low load exercises regardless of exercise mode.

Exercises are used for stretching, strengthening, balance, proprioception, flexibility, endurance and muscle re-education

Exercises for stretching, front and hind limb and balance and proprioception
PROM, High 5s, Ball Work, Wheelbarrow, Step ups, Sit to Stand, Backwards Walking, Side stepping, Rhythmic Stabilization, Cross legged standing, Crawling, Sit to Be

Regenerative medicine
Stem cell and PRP (Platelet Rich Plasma) can be used for pain management. Progenitor cells present in almost every tissue that are self-renewing, able to become different tissue types and signal other cells to come in and repair tissue. Adipose derived and bone marrow derived mesenchymal stem cells are used. Benefits are more like due to growth factors. PRP contains growth factors as well. This is a wide topic and only gets a brief mention here.

References
Osteoarthritis is the most common chronic musculoskeletal disease in dogs and cats. It is estimated approximately 60% of all dogs and close to 90% of aged adult cats are affected by this disease. Osteoarthritis (OA) is not a single disease entity but rather a disease process. It is a common final pathway for a failing synarthroidal or diarthroidal joint. It must be remembered the joint is an organ therefore OA affects not only cartilage but also involves the synovium, synovial fluid, ligament, fat and underlying bone. The condition may be confined to the joint but the entire patient is affected due to the pain and disability of OA. Patients with OA have decreased activity and performance, decreased ROM, muscle atrophy (often generalized sarcopenia), pain, decreased flexibility, loss of strength and increased joint stiffness.

OA in dogs is always a secondary problem caused either by abnormal stress on a normal joint (such as may happen with trauma) or normal forces on a joint that has an underlying abnormality (e.g., joint laxity, or instability from hip dysplasia, or cruciate disease). Either scenario will result in a gradual loss of articular cartilage (the morphological marker for OA) and joint impairment. OA in cats can be secondary to genetic disease or trauma but often no initiating cause can be identified.

**Tissues in the synovial joint**

To truly understand OA requires a working knowledge of the metabolism of the tissues of the synovial joint. It is important to remember that all the components of a joint adapt their composition and appearance to match functional demands. The demands are usually mechanical but can change with immobilization, injury, training or inactivity.

The tissues have a certain form and characteristic but change due to demands on the tissue. Stress and strain can change the type of collagen, and amounts, cross linking, PG types and architecture of the joint. Wolff’s law tells us about changes in bone due to demand—there are similar changes in tendons and ligaments.

**What makes up a joint?**

- Bone
- Articular Cartilage
- Synovium
- Tendon
- Ligament
- Menisci
- Labra
- Fat Pads
- Bursae
- Synovial Fluid

Most of these structures are made up of connective tissue. Connective tissues are made up of widely dispersed cells in a large volume of extracellular matrix (ECM). The function of the connective tissue is determined by its ECM not the cells in the tissue. Tendons and ligaments are considered dense connective tissues with tendons being more elastic than ligaments. Important cell types found in joints are fibroblasts in tendons, ligaments, menisci, and labra, chondroblasts in articular cartilage and osteoclasts and osteoblasts in bone.

**Extracellular matrix**

The ECM is mostly protein and water and has both interfibrillar (ground substance) and fibrillar components. The fibrillar component is composed of mostly collagen and elastin. Collagen is the most abundant protein in the body, has a tensile strength similar to steel, is responsible for the integrity of the tissues and their resistance to tensile force.

**Interfibrillar component**

This is mostly glycoproteins and proteoglycans (PGs).

**PG characteristics**

- Distinguished by protein core and attached glycosaminoglycans (GAGs)
- Attract water through attached GAGs
- Regulate collagen fibril size
- Attach to Hyaluronan to form large aggregates called aggrecans
• Are increased in tissues subjected to alternating cycles of compression.

Glycosaminoglycans (GAGs) exist mostly in 2 classes:
- Glucosaminoglycans—Heparan sulfate and keratin sulfate—contain D-glucosamine
- Galactosaminoglycans—Chondroitin sulfate and dermatin sulfate—contain D-galactosamine.
- Exception is Hyaluronic acid (hyaluronan) which is non sulfated D-glucosamine and D-glucuronic acid and does not attach to a core protein. In synovial fluid, hyaluronan is produced by type B synoviocytes but in the ECM it is produced by chondrocytes. Synovial hyaluronan acts as a lubricant and molecular barrier.

PG aggregates (aggrecans) are a number of PGs linked together by link proteins and attached to Hyaluron. Along with collagen they are the major weight bearing macromolecule in the articular cartilage. During metabolism PGs are broken down by enzymes matrix metalloproteinases (MMPs) and aggrecanase. In acute inflammation MMPs increase in number and disrupt the balance of production and destruction in the joint.

Etiology of osteoarthritis
1. Genetic predisposition—Hip dysplasia, elbow dysplasia
2. Aging—Chondrocytes synthesize smaller aggrecans, less functional protein and there is an accumulation of advanced glycation end products in the Type II collagen network. Decreased amounts of chondroitin sulfate are produced and increased amounts of keratin sulfate are produced. Keratin sulfate has less ability to imbibe water and therefore cartilage is stiffer and less resistant to deformation.
3. Obesity—increased load on joint mechanically. Adipose tissues is pro-inflammatory and produces increased levels of tumor necrosis factor (TNF), IL-6 and leptin. Obesity causes osteoarthritis through action of adipokines.
4. Early neutering—this appears to be the case for some joints.
5. Exercise, diet, housing—Over exercising at a young age can damage joints. There are no studies validating home made diets vs commercial diets to prevent the development of arthritis.

Pathogenesis of Osteoarthritis
The formation of OA involves all the tissues of the synovial joint. Changes include alterations of the metabolism and morphology of the articular cartilage, and subchondral bone, osteophyte and enthesophyte formation and synovial inflammation and fibrosis. Changes also occur in the soft tissue structures and the ipsilateral musculature due to disuse and inhibition. Changes in the central nervous system occur due to chronic pain leading to pain sensitization.

A normal healthy cartilage looks like the figure below.

Orientation of the cells and relatively little PG in Zone 1, the Tangential Zone, allows the surface of the joint to withstand high tensile stresses resisting deformation and distributing the load across the joint. Loss of this layer as happens in early OA changes the biochemistry within the cartilage. Zone 2 and 3 contain more PG and this allows them to withstand more compressive loads. PGs have a high affinity for water and when the cartilage is loaded slowly this weeps out onto the articular surface to lubricate the joint. In areas of high stress the cartilage is stiffer. In areas of low stress the cartilage is softer. If excess force is put on softer cartilage, OA can result. The subchondral bone has a large area that meshes with the cancellous bone and is very deformable. This allows it to distribute the load. When OA occurs the subchondral bone becomes stiff. The most profound changes are in the major weight bearing areas.

Three stages of OA
Stage one
- Imbalance in the anabolic and catabolic processes in the cartilage
- ECM degrades and water content increases
- Size of aggrecan molecules in matrix decreases
- Structure of collagen network is damaged which leads to increased stiffness of cartilage
- Macrophages in the synovium produce TNF alpha, IL-1Beta, IL-17, IL-18—all pro inflammatory. These affect the chondrocytes and activate the MMPs and aggrecanase which break down the matrix.

Stage two
- Chondrocytes proliferate and increase metabolic activity—produce more MMPs to try repair damage—decreased TIMP. Chondrocytes express COX-2 and produce Prostaglandin E2. This enhances the degradation of aggrecan and Type II collagen
- Cell clusters form to try and repair damage but catabolism eventually takes over.

Stage three
- Repair can not keep up with damage and cartilage is lost. Chondrocytes produces nitric oxide (NO) synthase which cause progressive cartilage loss. NO inhibits matrix synthesis, activates MMPs and apoptosis.
Degradation of the ECM of the articular cartilage and cell death are key processes in osteoarthritis.

**Pain in OA**
Our understanding of joint pain is poor—much comes from human models.

Joint nerves are A beta, A Delta and C fibers. Only cartilage has no nerve endings so stimulation does not produce pain. Silent nociceptors only respond when there is inflammation in the joint.

Synovium—key tissue in the origin of pain of OA. Cytokines and growth factors are produced by the synovial lining cells and inflammatory mediators which sensitize the silent nociceptors. Synovial inflammation, once established, can alter the metabolism of resident synoviocytes, the major biosynthetic source of hyaluronan (HY) in synovial fluid. Inflammatory mediators released from local synovial cells and infiltrating leukocytes can promote increased vascular permeability and the accumulation of plasma in synovial fluid, thereby decreasing HY concentration. This dilution of HY and reduction in its molecular weight due to abnormal synthesis by synoviocytes results in a decrease in the viscoelasticity of synovial fluid and thus its ability to lubricate and protect articular cartilage. This sets up a vicious cycle of cartilage degradation and pain.

Joint pain results in central sensitization which causes increased pain. COX enzymes play a role in central sensitization and COX inhibitors such as NSAIDs prevent establishment of central sensitization. Central sensitization can increase joint pathology while suppressing it can decrease joint pathology.

** A direct effect of NSAIDs at the level of the joint can result in a reduction in disease progression. NO induces cell death. NO is produced by chondrocytes when they are stimulated by pro inflammatory cytokines. COX-2 inhibitors block this by blocking the pro inflammatory cytokines. NSAIDs are still the most important treatment in OA**

References
Emerging Modalities in the Treatment of Pain
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What exists for painful veterinary patients beyond rest and NSAIDs? What can I do for those really painful patients? Where can I find help and answers for my pain questions? What are the standards of care for pain? Are there any non pharmacological modalities that really work for pain?

These are all very common questions for veterinary practitioners to ask in this changing world. We will deal with what’s new and what is on the horizon for pain in veterinary practice.

Standards of care for pain in dogs and cats is now outlined in the new AAHA/AAFP pain guidelines. Here is an excerpt summarizing the guidelines:

The 2015 guidelines summarize and offer a discriminating review of much of this new knowledge. Pain management is central to veterinary practice, alleviating pain, improving patient outcomes, and enhancing both quality of life and the veterinarian-client-patient relationship. The management of pain requires a continuum of care that includes anticipation, early intervention, and evaluation of response on an individual-patient basis. The guidelines include both pharmacologic and nonpharmacologic modalities to manage pain; they are evidence-based insofar as possible and otherwise represent a consensus of expert opinion. Behavioral changes are currently the principal indicator of pain and its course of improvement or progression, and the basis for recently validated pain scores. A team-oriented approach, including the owner, is essential for maximizing the recognition, prevention, and treatment of pain in animals.

Postsurgical pain is eminently predictable but a strong body of evidence exists supporting strategies to mitigate adaptive as well as maladaptive forms. Degenerative joint disease is one of the most significant and under-diagnosed diseases of cats and dogs. Degenerative joint disease is ubiquitous, found in pets of all ages, and inevitably progresses over time; evidence based strategies for management are established in dogs, and emerging in cats. These guidelines support veterinarians in incorporating pain management into practice, improving patient care.

Pain scoring is important and veterinarians need to use pain scales
Although the Glasgow Composite Pain Scale is the only validated Acute pain scale, the Colorado State Acute Pain scales for post surgical pain are easier to use. They rely on pictures and should be scored by the same person as the inter rater scores could vary.

Chronic pain scales include the Helsinki Chronic Pain Scale, the Canine Brief Pain Inventory, the Cincinnati Orthopaedic Disability Index and others. The important thing is to familiarize yourself with one of these scales and use it consistently.

New or new to you techniques to consider
Local anesthetics with every surgery—Line blocks, Ring blocks, testicular and ovarian blocks.

For chronic orthopedic disability regional anesthesia with bupivicaine can be used to relieve pain so muscles can be strengthened—consider blocking femoral or sciatic nerves—may need to use a nerve finder to do this.

Epidurals—not really new for orthopaedic surgery but consider these for blocked cats ( sacral epidural). For dogs with painful lumbosacral disease consider epidurals with methyprednisilone—it will not improve neurological function but will relieve pain.

- Ketamine—Subanesthetic doses as CRIs for painful surgeries—things that are likely to trigger maladaptive pain syndrome.
- Use of Amitriptyline for Chronic pain—much lower doses than needed for anxiety.
- Joint injections with HA and corticosteroids

New information
- Pruritis receptors are a subset of nociceptors that also respond to pain.
- Substance P and Glutamate can cause pain and itch.
- Itch can be inhibited by pain--mild scratching inhibits itch. In order to inhibit the itch signalling pathway you need to have both itch and pain as these overlap, so consider this when dealing with chronically itchy animals—this may be why amitriptyline works for pain.

Glial cell dysregulation
Originally thought that non neuronal cells (glia) had no input into the nervous system. However research has shown that microglia and astrocytes have an effect on the nervous system and how it handles opiates. Glial cells are key in the development of pathologic pain.

In every clinically relevant model of enhanced pain, the glial cells are activated, so if you block the glia cells you block pain. Glial cells monitor the CNS--they are very active cells--when they find danger they actively attack it--they release all kinds of substances.

The important thing to know is they amplify the pain transmission to brain, they up regulate the NMDA receptor numbers and down regulate GABA and glia glutamate transporters.
Glial cells enhance pain and PREVENT opiates from working.

What activates glia cells?—lots of stuff—Opioids, endogenous danger signals (leakage of blood—anything that should not be in the nervous system that causes cell stress and damage).

Most common endogenous danger signals are peripheral nerve injury, overuse of medication, diabetic neuropathy, spinal cord injury, bone cancer, arthritis, and pancreatitis. These conditions cause pro inflammatory cytokines to be released.

Opioids activate the glial cells so this can actually block the pain control from the glial cells—if you block the spinal IL-1 the analgesia comes back. Opioid effects are different on neurons vs glial cells. The glial cell receptor is TLR4 (not me, not right now okay receptor)—it is a major player in identifying endogenous danger signal and recognizes all lipids. Glial TLR4 is a driver of neuropathic pain. If the glial cells are blocked by naltrexone then it might be a stand alone treatment for neuropathic pain. Other cells involved are in blood vessels and these produce IL-1 (pro inflammatory cytokines) and these can be blocked by naltrexone.

When glial cells are active and you give opioid for pain it makes pain worse because the glial cells are induced to produce more IL-1 and block the opioid receptors. Primed state can occur for a period of time after prior activation—the glial cells are not activated they are waiting and the reaction comes back with a vengeance. Primed glial cells can be activated by aging, opioids, stress, trauma and inflammation.

Clinical relevance—prior surgery changes pain to chronic pain—this can be prevented by glial activation inhibitor.

Another interesting point—morphine can worsen post surgical pain. This is mediated by TLR4—so to control this you need to give a TLR4 blocker when you give morphine.

IL-10—anti inflammatory interleukin—this is being developed for interthecal injection.

Grapiprant—EP4 blocker that works in inflammatory cascade with no NSAID side effects.

Theracurmin—biologically active curcumin—water soluble

**Non pharmacological**

- Acupuncture—more common and more accepted.
- PT modalities easily added to general practice—Laser, exercise therapy, hot and cold therapy, TENS
- Myofascial pain—this is something new to veterinary medicine. It is a myalgic condition in which muscle and tendon are the primary cause of pain. The syndrome is centered around the myofascial trigger point (MTrP). A myofascial trigger point is an extended contraction of a few muscle fibers that results in a painful knot. Dry needling is what is done to eliminate them. If you are interested there is an entire lecture on this topic.
- Regenerative Medicine—PRP and Stem cell
Myofascial Pain Syndrome (MPS) was brought to the attention of modern human medicine by Dr. Janet Travell in 1952 although it has been described in literature as long ago as the 16th century. Despite this, it has failed to enter mainstream medicine, especially in veterinary medicine. Many veterinarians do not even know of its existence! However, within the past decade this aspect of pain medicine along with many others has been steadily gaining a foothold in the general veterinary practice.

What is myofascial pain?
The pathophysiology of myofascial pain is a complex syndrome involving in part, motor, sensory and autonomic nerve components. It is a myalgic condition in which muscle and tendon are the primary cause of pain. The syndrome is centered around the myofascial trigger point (MTrP). A myofascial trigger point is an extended contraction of a few muscle fibers that results in a painful knot. Simons, Travell and Simons define it as

“a hyper irritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is tender when pressed and can give rise to characteristic referred pain, motor dysfunction and autonomic phenomena.”

Therefore all MTrPs have a sensory component, a motor component and an autonomic component. When the motor end plate is overstretched which can happen when only a few muscle fibers are activated then myofascial tension is increased in that fiber. An increase of tension of only 1% will evoke a 10% increase in ACh release. With excessive release of ACh there is excessive motor activity. The local muscle contraction compresses local sensory nerves and blood vessels and reduces the supply of oxygen to the area. Decreased oxygen and increased metabolic demands of the contracted muscle fibers result in a depletion of the local ATP. This cause pre and post synaptic changes in the Calcium pump and leads to muscle spasms. Trigger points are formed which are painful and either excite or inhibit activity on motor activity in the muscle or its functionally related group. This inhibition causes poor coordination and muscle imbalances. There are also autonomic phenomena associated with MTrPs.

Etiology of MTrPs
Mechanical issues
Acute trauma may activate MTrPs but does not perpetuate them. Sudden activation can occur with direct trauma, muscle strain, joint sprain or excessive or unusual exercise. Mostly commonly are formed with chronic muscle overload such as occurs with orthopedic injury, neuropathy, joint dysfunction or osteoarthritic pain. It is thought that low level muscle contractions, unaccustomed eccentric contractions or eccentric contractions in unconditioned muscle as well as maximal or sub maximal concentric contractions may lead to MTrP formation.

In OA the joint dysfunction and postural changes can activate and perpetuate MTrPs. With coxofemoral arthritis the muscles that frequently develop trigger points are the sartorius, tensor fascia lata, pectineus, rectus femoris and iliopsoas (hip flexors). Due to the forward weight shift, they also develop in the triceps, infraspinatus and deltoid muscles. Because pelvic movement is compromised, and more lateral flexion of the spine occurs, the iliocostalis lumborum and lateral multifidi are also effected. If a dog is hopping on one back leg, trigger points can develop in the contralateral limb and hopping causes excessive eccentric contraction of the stifle extensors. In this leg we see MTrPs in the sartorius, tensor fascia latae, rectus femoris and vastus group. The lumbar paraspinals are also involved as they assist in ambulation.

Nutritional deficiencies and metabolic issues
It is unknown if nutritional deficiencies or metabolic problems perpetuate trigger points in dogs but in humans they have been linked with certain deficiencies such as cobalamin, folate, iron deficiency, Vitamin D and B12 deficiency and metabolic diseases such as hypothyroidism and diabetes.

Examination techniques
MTrPs are diagnosed by palpation. 3 types of palpation are used: Flat palpation, Pincer Palpation and Snapping palpation. With flat palpation the finger pressure is applied at right angles to the muscle fiber compressing against the bone—this is used for the infraspinatus, supraspinatus and poas muscle. With Pincer Palpation the muscle bands are pinched and rolled between thumb and fingers to detect taut bands. This works for the triceps, sartorial and tensor fascia latae. Snapping palpation is similar to pincer but the fibers are rolled under the finger similar to plucking a guitar string. Taut bands are palpated and usually animal is painful so jumps (Jump sign)
Clinical cases

**Brooklyn the Rottweiler**

F/S 5 year old Rottweiler BCS 6/9, had TPLO LH 1 year ago and still not using leg well. Current pain medications included Meloxicam and Tramadol. On examination she had a large number of MTrPs in her iliopsoas, sartorial, TFL on the left side and Triceps bilaterally. All of her hip flexors were sore to the point she resent extension of her stifle and was vocal and aggressive with the iliopsoas test. Because Brooklyn had spent a lot of time with her leg contracted she had slight muscle contractions of the hip flexors due. The front leg MTrPs were due to compensation from weight shifting. Brooklyn was uncomfortable and her owners were frustrated.

**Treatment:** Sedation and dry needling

After one session Brooklyn was more comfortable and would allow her muscles to be touched. A rehabilitation program including acupuncture, UWTM, stretching, leg and core strengthening was able to proceed. Within 1 month Brooklyn was back to her normal self and was fully weight bearing.

**Regi the wirehaired fox terrier**

Regi, 11 yr old F/S BCS 6/9 former agility dog, pain in sacral area, elbow arthritis, lagging in walks and not wanting to go many places. Owner felt she was depressed. She noticed Regi was walking “funny” in the front end and base wide in the hind end. She had had several rehabilitation sessions for strengthening and gait retraining as well as medication—Gabapentin, Amantadine, Chinese herbs, acupuncture—nothing seemed to be helping. My examination revealed myofascial pain in her iliopsoas, quadriceps, and sartorial and in the triceps and infraspinatus muscles of both front legs.

**Treatment:** Sedation and dry needling

**Result:** Regi continued rehab therapy but this time there was a big improvement. She went back to walking well and was no longer depressed.

Dry needling is the preferred method of treatment in myofascial pain syndrome in dogs. Dry needling involves the act of placing an acupuncture needle directly into the painful trigger point resulting in a complex cascade of events involving in part spinal reflexes, increased blood flow and an increase in the amount of energy available to the muscle. This causes the taut band of muscle containing the trigger point to relax and the pain relief is immediate. When Brooklyn’s owner picked up her dog after the first session, she was misty-eyed with relief when she saw Brooklyn walking normally as she came out to greet her. Dry needling imparts an immediate benefit but it generally requires several sessions to give complete relief. And unless the underlying cause can be found and completely treated, it eventually returns needing additional treatments, especially in the case of chronic conditions like osteoarthritis.

Dry needling is not taught in university settings. The only regular classes that a veterinarian can take is through the Canine Trigger Point Therapy Program given through Myopain Seminars and taught by Drs. Jan Dommerholt and Rick Wall.

References
Pain is a multidimensional sensory experience that is intrinsically unpleasant and associated with hurting and soreness. It may vary in intensity (mild, moderate, or severe), quality (sharp, burning, or dull), duration (transient, intermittent, or persistent), and referral (superficial or deep, localized or diffuse).

Pain is divided into 2 categories: Adaptive and Maladaptive Pain. Adaptive pain is that which serves a purpose—to protect the body from harmful substances or protect the body while healing occurs. Adaptive pain can be either nociceptive or inflammatory. Nociceptive pain is pain that is transient from noxious stimuli. Inflammatory pain is spontaneous in response to tissue inflammation or injury. Both of these could be considered acute pain and will occur naturally post injury or inflammation and are limited in their scope and time frame. These types of pain are pain with a purpose—to protect and allow rest for healing.

Maladaptive pain is pain as a disease and it serves no useful purpose. Such pain may occur in response to damage to the nervous system (neuropathic pain) or result from abnormal operation of the nervous system (functional pain). Maladaptive pain is the expression of abnormal sensory processing and usually is persistent or recurrent. Maladaptive pain can result from peripheral or central sensitization. In peripheral sensitization inflammation and tissue damage produce a variety of nociceptor-sensitizing substances, including prostaglandins, histamine, serotonin, bradykinin, proteases, cytokines (tumor necrosis factor $\alpha$), and nerve growth factor. This "sensitizing soup" lowers the nociceptor threshold to painful stimuli and activates "silent" or "sleeping" nociceptors, resulting in hyperalgesia (exaggerated response to noxious stimuli) and allodynia (painful response to normal stimuli).

Central sensitization occurs when severe (high-intensity) or chronic painful stimuli activate C fibers, causing the release of glutamate, substance P (Sub P), and brain-derived neurotrophic factor (BDNF) at central nerve terminals; this results in the activation of number of receptors producing acute and long-lasting dull, aching, burning pain sensations. Collectively, the activation of these receptors increases the activity of a host of signaling molecules that alter gene expression and change the responsiveness (sensitize) of the central nervous system to subsequent input. Chronic painful stimulation may result in neurochemical changes (neuroplasticity) in the spinal cord such that all stimuli produce pain. (Gaynor 37)

**Pain assessment in cats**

Currently the only validated acute pain scale for cats is the UNESP-Botucatu Multidimensional Composite Pain Scale. A shorter and easier to use pain scale is the Colorado State University Feline Acute Pain Scale. It involves a simple numeric scale with pictures outlining behavioural and psychological indicators of pain and includes response to palpation. This scale is not currently validated.

For chronic pain, specifically related to degenerative joint disease (DJD) in cats, the only validated system is the NCSU Comparative Pain Research Lab’s Feline Musculoskeletal Pain Index. Behaviors evaluated include litterbox use, grooming, fluidity of gait, temperament, appetite, allowing petting and general activity. Use of activity monitors is another possibility to determine a cat’s pain. These have been used in scientific research and will have a place in feline pain determination in the future.

No matter which system is used, results are best if the same person scores the cat’s pain each time to minimize inter rater variability.

**Pain management in cats**

**Nonsteroidal anti-inflammatory drugs**

NSAIDs are one of the most common drug classes used to treat pain, and there is a robust body of information indicating that NSAIDs are effective in treating acute pain in cats. They have antipyretic, analgesic, and anti-inflammatory properties, which make them appealing therapeutic options; however, remember that there is not, and never will be, a completely safe NSAID for use in cats. (Lascelles)

NSAIDs work to block the cyclooxygenase (COX) enzyme pathway to prevent production of eicosanoids and also work to inhibit central perception of pain. Eicosanoids are metabolically active compounds derived from 20-carbon fatty acids, usually arachidonic acid. The lipooxygenase (5-LOX) and cyclooxygenase (COX) enzymes are the rate-limiting steps in the production of leukotriene B4, thromboxane A2 and prostaglandin E2.

The ideal NSAID should:

- Spare COX-1 as much as possible (to prevent GI erosion and renal tubular necrosis)
- Inhibit COX-2 sufficiently for efficacy against pain and inflammation
- Spare enough COX-2 to allow it to function in normal everyday processes
Robenacoxib (Onsior - Elanco)
Robenacoxib is a targeted tissue selective and a unique COX-2 selective NSAID. It has a very short half-life (3 hours) in the blood, yet persists, and is active, for at least 24 hours in inflamed tissue in cats which demonstrates "tissue selectivity." It is available in injectable and tablet form. Dosage: 1mg/kg q 24 hours

Meloxicam (Metacam — Boehringer Ingleheim)
Meloxicam is an example of a preferential COX-2 inhibitor that has greater inhibition of COX-2 than COX-1. It is also tissue selective but has a longer tissue half-life than robenacoxib.

Used for years in Canada, Europe and Japan. Has a black box label in the US because labelled dose is 0.3mg/kg once post surgery — too high a dose.

Canada has a specific Metacam for Cats with a reduced dose — this dose seems to be the safest. Dose is Injectable 0.1mg/kg—once post surgery followed by oral dose of 0.05 mg/kg q 24 hrs for 5 to 11 days. Dose may be further reduced for long term therapy, we find 0.02mg/kg once daily as a good oral dose for cats with chronic pain — this is study supported although the study was not blinded.

Opiates
Buprenorphine
A partial mu-agonist that is used to manage chronic pain in cats and is classified by the Drug Enforcement Administration (DEA) as a Schedule III controlled substance. Buprenorphine is not approved by the Food & Drug Administration (FDA) for use in cats. The drug may be administered SC, IM, IV, or buccally; buccal administration is the preferred route for chronic pain management. Dosage: 0.01–0.02 mg/kg SC, IM, IV, or transmucosal.

In July 2014, a new veterinary formulation of buprenorphine was FDA-approved and introduced into the marketplace (Simbadol, Abbott). At 1.8 mg/mL it is 6X more concentrated than the human commercial product Buprenex (0.3 mg/mL). It is labeled for postsurgical pain in cats, with a 24-hour duration with one injection at 0.24 mcg/kg subcutaneously (SC); it can be given daily for up to 3 days. The labeled dose is 0.24 mg/kg, approximately 10X the dose previously recommended. Shelf life 21 months unopened and 28 days opened.

Tramadol — can be used but not approved, variable results in dogs but cats do make the M1 metabolite so better results in cats. In cats it is a mu agonist and serotonin—norepinephrine reuptake inhibitor. Cats are sensitive to the side effects of this drug and the bitter taste makes it difficult for cats to accept.

Dose: 1-4 mg/kg q 8 to 12 hours

Fentanyl — patch forms — variable efficacy

Analgesic adjuvants
These are used in combination with NSAIDs or Opiates to treat chronic pain

Amantadine
Amantadine is an antiviral compound used in humans that is reported to exert an analgesic effect through NMDA receptor antagonism. Toxicity and kinetic studies have not been performed in cats. It is often effective however but can cause diarrhea in cats. Caution in cats with liver and kidney disease or seizures. Dosage: 3-5 mg/kg q 24 hours

Gabapentin
Gabapentin is an anticonvulsant that is used in cats for chronic pain particularly neuropathic pain. It is often used with amantadine and NSAIDs. It has been used to treat allodynia and hyperesthesia. Caution in cats with kidney disease — can be used at a reduced dose.

Dosage: 5 to 10 mg/kg q 12 hours

Amitriptyline
Amitriptyline, a tricyclic antidepressant, is usually administered in combination with an NSAID for feline chronic pain of neuropathic origin. Avoid in seizing animals or with liver disorders. Do not give along with Tramadol as you may cause serotonin syndrome.

Dosage: 1-2 mg/kg q 12 to 24 hours

Nutraceuticals
Adequan (Polyglycoaminoglycan) and Cartrophen (Pentosan polysulphate)
These injectables have been used in dogs and are also used in cats. The bioavailability and distribution of PSGAGs to inflamed joints in cats has been demonstrated with extralabel subcutaneous administration.

Dosage: Cartrophen 1 ml per 33kg SQ once weekly for 4 weeks then once monthly

Adequan: 5mg/kg once weekly x 4-6 weeks then q14d then once monthly (SQ)

Omega 3 fatty acids
The primary source of omega 3 fatty acids is fish oil. A recent placebo controlled trial done with Royal Canin’s Feline Mobility support diet found significant improvement in indicators of pain and quality of life when comparing the base-line outcome measures to those collected at the end of the 16-week trial. (Lascelles et al). Dose of combined EPA + DHA is maximum 100 mg/kg if using capsules or adding fish oil to the diet (generally 1 tsp of fish oil is the maximum). There is a concern that this level of
supplementation may cause clotting problems in this species. However, research shows that at this level of supplementation, no cats experienced any clotting problems (Joe Wakshlag, Cornell University, Personal communication)

**Green lipped perna mussel (GLM)**

Perna canaliculus is found in the waters around Australia and New Zealand. It contains EPA, DHA, and ETA. It is also a source of glycoproteins and GAGs. The anti-inflammatory effects of GLM may be derived from its omega-3 fatty acids content or the GAGs or the glycoproteins. Suggested dose is 50-70mg/kg/d

**Herbals**

Flexadin Plus (Vetoquinol) contains Devil’s Claw, Omega 3 and Glucosamine/chondroitin—seems to get a good rating but RCCT lacking.

**Avocado soybean unsaponifiables**

Avocado soybean unsaponifiables (ASU) are residues of avocado and soy oils combined in a 1:2 ratio to produce a product that has demonstrated anti-arthritic properties. Theoretically, ASU decrease the production of pro-inflammatory cytokines such as PGE-2 and TNF alpha. There are no published controlled trials in clinical cats with OA examining ASU alone or in combination products although in vitro studies have been conducted on feline chondrocytes.

**Pain management beyond drugs**

**Weight reduction**

Many cats with OA are overweight. As the OA worsens and becomes more painful the cats become less active contributing to an increase in weight. Excess weight causes an excess load on an abnormal joint, creating more pain. In addition the adipose tissue secretes adipokines which are pro-inflammatory and these increase the overall inflammation in the joint of the cat. Secret to weight loss in the cat is canned weight loss diet amount calculated by RER and exercise.

Determine RER from Body Condition Score on a 9 scale. For every point that the cat is overweight over the ideal 5/9 body condition score, the cat is 10% obese. For example a 10 kg cat with a body condition score of 7/9 is 2 x 10% or 20% over ideal weight. To determine Lean body weight in kilograms 10/1.20 = 8.3kg. Take this lean body mass of 8.3 and raise it to the 0.75 power. Multiply this result by 70 to give the RER. In this case a cat with a lean body mass of 8.3 kg has a RER of 342 kcal. Multiply this by 0.8 = 272 kcal. This is the number needed for weight loss. Use this formula and this calculation rather than amount on bag or can for weight loss.

Exercise therapy can involve an obstacle course, playing with a feather, chasing a laser pointer, climbing a tower etc. A hockey rink with cat toys on a smooth surface can help with weight loss. Moving the food bowl during feeding and making the cat move around is helpful. Food balls can work for cats as well. Designing a cat tree so the cat has vertical and horizontal space is helpful. Indoor outdoor cats are thinner—cat gazebo is a possibility. Treadmill exercise can also be used for cats. Cat nip treats may entice cats to exercise.

**Acupuncture**

Acupuncture is a safe and often enjoyable method of pain relief for cats and should be considered as part of a multi modal pain management plan. It is minimally invasive and can be used with other modalities and medications as well. This author has used it in cat with back pain, osteoarthritis, stifle pain, post surgery, persistent pain post declaw, for excess grooming (that was related to back pain) for interstitial cystitis and other conditions. There is a growing body of evidence for its use in veterinary medicine.

**Physical rehabilitation**

Physical rehabilitation is now considered a mainstay for pain relief post surgery and for geriatric animals. Cats are very amenable to all forms of physical therapies. Physical therapy should be considered part of a long term strategy for pain management in the cat. The goals of physical therapy are to restore muscular and joint strength and function, to restore balance and proprioception, to relieve pain, to improve mobility, endurance and flexibility.

Physical rehabilitation can involve manual therapies, massage, laser therapy, hot and cold therapy, exercise therapy, joint mobilizations, ultrasound, electrical stimulation, myofascial release and hydrotherapy. Exercise therapies using balls, treadmills and other devices can be used with cats and often are simply limited by imagination. Passive range of motion (PROM) is a technique easily taught to clients to help relieve pain for stiff cats and one that should be employed for every geriatric cat.

**References**


Leptospirosis is a bacterial zoonosis that is common worldwide, especially in developing countries. Organisms are shed in the urine of infected animals, including rodents and domesticated animals, which may not show signs of disease. Humans usually become ill after contact with infected urine, or through contact with water, soil or food that has been contaminated. Outbreaks have been associated with floodwaters. In animals, the clinical signs of leptospirosis are often related to kidney disease, liver disease or reproductive dysfunction. In humans, many cases are mild or asymptomatic, and go unrecognized. In some patients, however, the illness may progress to kidney or liver failure, aseptic meningitis, life-threatening pulmonary hemorrhage and other syndromes.

### Etiology
Leptospirosis is caused by various species of *Leptospira*, a spirochete in the family Leptospiraceae, order Spirochaetales. Some *Leptospira* are harmless saprophytes that reside in the environment, while others are pathogenic. The basic unit of *Leptospira* taxonomy is the serovar. Serovars consist of closely related isolates based on serological reactions to the organism’s lipopolysaccharide. More than 250 pathogenic serovars, and at least 50 nonpathogenic serovars, have been identified.

#### Species affected

**Maintenance hosts**

*Leptospira* serovars are generally adapted to one or more mammalian or marsupial maintenance hosts, which may or may not develop clinical signs. Dogs are reservoir hosts for serovar Canicola, and pigs for Bratislava and Pomona. Horses may also maintain Bratislava. Cattle are the primary reservoir hosts for Hardjo; however, this serovar is also maintained in farmed red deer (*Cervus elaphus*) and wapiti (*Cervus elaphus nelsoni*) in New Zealand, and sheep and goats may also have a role. Rodents and insectivores are reservoir hosts for a number of *Leptospira* serovars, including members of the serogroups Icterohaemorrhagiae, Grippotyphosa and Sejroe. In particular, rats are important hosts for serovars Icterohaemorrhagiae and Copenhageni in the serogroup Icterohaemorrhagiae. Other domesticated and wild animals (e.g., skunks, raccoons, wild boars) are also thought to maintain pathogenic *Leptospira*, but in many cases, there is less information about their roles.

**Clinically affected species**

Leptospirosis occurs in dogs, cattle, sheep, goats, horses, pigs, South American camelids and farmed cervids, but illness seems to be rare in cats. Disease also seems to be uncommon in camels, although this might result from nomadic husbandry rather than innate resistance. Leptospirosis has been reported occasionally in zoo animals or wildlife. Among marine mammals, clinical cases have occurred most often in California sea lions (*Zalophus californianus*) and northern fur seals (*Callorhinus ursinus*), but they have also been seen in captive harbor seals (*Phoca vitulina*) and northern elephant seals (*Mirounga angustirostris*). The most common *Leptospira* serovars in clinical cases vary with the host species and geographic region, and sometimes with selection pressures from vaccination. Icterohaemorrhagiae and Canicola were once the most common serovars in symptomatic dogs, but the introduction of Icterohaemorrhagiae/Canicola vaccines has resulted in the increasing prominence of other serovars.

**Zoonotic potential**

A variety of *Leptospira* species and serovars can cause disease in humans; however, members of *L. interrogans* and *L. borgpetersenii* are found most often. People are usually considered to be incidental hosts, which do not act as reservoirs for these organisms. One recent study suggested that people might maintain *Leptospira* in certain environments such as the Peruvian Amazon.

**Transmission**

Leptospirosis can be transmitted either directly between hosts or indirectly through the environment. *Leptospira* spp. can be ingested in contaminated food or water, spread in aerosolized urine or water, or transmitted by direct contact with the skin. The organisms usually enter the body through mucous membranes or abraded skin. They might also be able to penetrate intact skin that has been immersed for a long time in water, although this is controversial.

*Leptospira* spp. are excreted in the urine of both acutely and chronically infected animals. Chronic carriers may shed these organisms for months or years. Although humans can also shed *Leptospira* in the urine, prolonged shedding seems to be uncommon; most people excrete these bacteria for 60 days or less. In animals, *Leptospira* can be found in aborted or stillborn fetuses, as well as in normal fetuses or vaginal discharges after giving birth. They can be isolated from the male and female reproductive organs in some species, and these infections may persist for long periods. For example, serovar *Hardjo* may be found in the reproductive tract of both cows and bulls for more than a year. In rare instances, human cases have also been transmitted during sexual intercourse, or by breast feeding. Other uncommon routes of exposure in people include rodent bites and laboratory accidents.
Pathogenic *Leptospira* spp. do not multiply outside the host. In the environment, they require high humidity for survival and are killed by dehydration or temperatures greater than 50°C (122°F). These organisms may remain viable in the environment for several months under optimal conditions, e.g., in water or contaminated soil. They survive best in bodies of water that are slow-moving or stagnant.

**Disinfection**

*Leptospira* can be inactivated by 1% sodium hypochlorite, 70% ethanol, iodine-based disinfectants, quaternary ammonium disinfectants, accelerated hydrogen peroxide, glutaraldehyde, formaldehyde, detergents and acid. This organism is sensitive to moist heat (121°C [249.8°F] for a minimum of 15 min). It is also killed by pasteurization.

**Infections in animals incubation period**

The incubation period can be as short as a few days, with clinical signs appearing after 5 to 15 days in experimentally infected dogs. It may be longer when clinical signs are the result of chronic, low level damage to the kidneys or liver. Rare case reports in cats suggest the possibility of prolonged incubation periods in this species. Abortions usually occur 2 to 12 weeks after infection in cattle, and 1 to 4 weeks after infection in pigs.

**Clinical signs**

**Dogs**

The syndromes and disease severity are highly variable in dogs. The initial clinical signs are usually nonspecific, and may include fever, depression, anorexia, stiffness, myalgia, shivering and weakness. The mucus membranes are often injected. Later, there may be signs of kidney involvement, with polydipsia, polyuria, oliguria, or anuria, and hematuria or dehydration in some cases. Fever may or may not be apparent in dogs that present with kidney signs. Vomiting, diarrhea, abdominal pain, gray stools, weight loss, jaundice, conjunctivitis and abortions can also be seen. Hemorrhagic syndromes occur in some dogs: the mucus membranes may have petechial and ecchymotic hemorrhages, and in later stages of the disease, there may be other signs such as hemorrhagic gastroenteritis and epistaxis. Leptospiral pulmonary hemorrhage syndrome has been seen in dogs, and can cause coughing, tachypnea or dyspnea. Vasculitis may result in peripheral edema and mild pleural or peritoneal effusion. Evidence of pancreatitis or myocardial involvement (ECG alterations) has also been reported. Deaths can occur at any time, including the acute stage. Chronic kidney disease can be a sequela.

Chronic infections may be asymptomatic, or associated with fever of unknown origin. There may also be a link between leptospirosis and some cases of chronic hepatitis.

**Cats**

Clinical leptospirosis appears to be uncommon in cats, but rare cases with kidney signs, icterus, uveitis and/or lameness have been reported. Experimentally infected cats had only mild clinical signs (mild temperature elevation, polyuria/ polydipsia), although some animals had histopathological evidence of inflammation in the kidneys and liver. The clinical signs included depression, dehydration, vomiting, polydipsia and fever, as well as abortions and neonatal deaths. Affected sea lions are typically reluctant to use their hind flippers, and assume a hunched position with the flippers held over the abdomen. A few case reports have documented leptospirosis in other species. The clinical signs in two black rhinoceroses (*Diceros bicornis michaeli*) included acute depression, anorexia, gastrointestinal discomfort with reduced fecal output, rear leg trembling, dysuria and glucosuria. One animal died rapidly, but the other survived with treatment. Illnesses that might have been caused by *Leptospira* were reported in a captive cougar (*Puma concolor*) with fatal interstitial nephritis, and a wild cougar that was concurrently infected with FeLV. Clinical cases, some severe, have also been reported uncommonly in nonhuman primates. Many infections in wildlife, particularly rodents, are thought to be asymptomatic.

**Diagnostic tests**

Leptospirosis can sometimes be diagnosed by detecting the organism, its antigens or nucleic acids in clinical samples such as blood (acute infections), urine and milk, or liver, kidney and other tissue samples collected at necropsy. Shedding of organisms in the urine may be either continuous or intermittent. *Leptospira* is usually identified in clinical samples by immunofluorescence or immunohistochemical staining, or polymerase chain reaction (PCR) techniques. These organisms stain poorly with the Gram stain, and are not observed by microscopy unless special stains or methods are employed. Silver staining or immunogold-silver staining is sometimes useful as an adjunct technique. Darkfield microscopy can also be used to detect *Leptospira*; however, this technique is non-specific and not very sensitive.

Culture is definitive, but the availability of this test is limited. *Leptospira* spp. can be isolated on a variety of media, but they are fastidious and grow slowly on primary isolation. Special transport media may be required for shipment to the laboratory. Depending on the serovar, culture can take up to 13 to 26 weeks. Identification to the species, serogroup and/or serovar level is done by reference laboratories, using genetic and immunological techniques.
Serology is often used to diagnose leptospirosis. Caution must be used in interpreting serological test results, as subclinical infections are common; the vaccination status of the animal must be considered; antibodies may not be present early in the illness; and antibody titers can become low or undetectable in some chronically infected animals. Paired acute and convalescent samples are preferred from most animals, and rising antibody titers are usually seen in acute cases. Single samples with high titers increase the suspicion of leptospirosis, although they are not definitive. However, a single positive sample from an aborted fetus is diagnostic. Herd tests are often used in ruminants. The most commonly used serological tests in animals are the microscopic agglutination test (MAT) and enzyme-linked immunosorbent assays (ELISAs). The MAT evaluates antibody responses to a selection of *Leptospira* serovars (often 5-7 in veterinary assays). This test is serogroup but not serovar specific, although it may suggest a likely serovar. False negatives are possible if the infecting serogroup was not included in the MAT panel. ELISAs, including a bovine milk ELISA, are available for some species of animals.

**Treatment**

Various antibiotics including tetracyclines (e.g., doxycycline), penicillins, dihydrostreptomycin and streptomycin may be used to treat leptospirosis. Fluid therapy, blood transfusions, respiratory support, and other supportive care may also be necessary. The primary treatment for equine recurrent uveitis in horses is anti-inflammatory drugs and medications to decrease discomfort (e.g., mydriatic cycloplegics such as topical atropine). Surgery (pars plana vitrectomy) and other therapies may also be used.

**Prevention**

Leptospirosis vaccines are widely available for pigs, cattle, and dogs. In some countries, vaccines may also be licensed or used in other species such as sheep, goats or farmed deer. Vaccines are usually protective only against the included or closely related serovars, and their content may need to be changed periodically. Most bovine vaccines contain serovar *Hardjo*, and most porcine vaccines contain Pomona, although other serovars may be included depending on the region. Vaccines for dogs have traditionally contained *interohaemorrhagiae* and *Canicola*, but additional serovars are now included in some vaccines, particularly in North America. In addition to routine use, vaccines are sometimes used to help prevent further abortions in livestock herds in the face of an outbreak.

Isolation and treatment of infected animals reduces the risk that they may spread the infection to contacts. Prophylactic treatment with antibiotics can be used to prevent disease in exposed animals.

Environmental control measures and sanitation may also reduce the risk of infection, although their practicality varies with the host species and situation. Such measures include providing safe drinking water, and preventing contact with contaminated environments (e.g., lakes) or infected domesticated animals and wildlife, particularly rodents. Good sanitation can reduce the risk of infection in kennels, and in areas where livestock are bred or give birth. Quarantine and testing of newly acquired livestock, or new equine studs on a farm, can also help prevent the introduction of *Leptospira*.

Large-scale eradication programs have not been widely used for leptospirosis; however, a control program has virtually eliminated serovar *Hardjo* from cattle herds in the Netherlands. This program includes regular testing of bulk tank milk with a Hardjo-specific ELISA, antibiotic treatment of infected cattle, and certification of Hardjo-free farms. Its success depends, in part, on the absence of serovar Hardjo in wildlife hosts.

**Morbidity and mortality**

Leptospirosis is particularly prevalent in warm and humid climates, and marshy or wet areas. This disease is often seasonal: it is most common during the rainy season in the tropics, and in the summer and fall in temperate regions. Morbidity and mortality vary with the species and age of the animal, the host specificity of the serovar, and other factors.

Leptospirosis is often asymptomatic or mild in adult pigs and cattle, and reproductive signs are the main evidence of infection. In newly infected cattle herds, up to 40% or more of the cows may abort. In endemically infected herds, abortions are usually sporadic and occur mainly in younger animals. Young livestock may be more severely affected, and can develop acute disease. In dogs, the risk of infection can vary with lifestyle factors, such as exposure to lakes and ponds. With treatment, 80% of dogs are expected to survive even when the kidneys are involved. However, the prognosis is poorer in cases with severe pulmonary complications. Domesticated cats rarely seem to be affected, although serological surveys report that up to 35% of cats may be exposed in some populations.

There is little information about the impact of leptospirosis, if any, on most wildlife species. In some areas, major outbreaks occur periodically among wild California sea lions.
Internet resources
Centers for Disease Control and Prevention (CDC)
http://www.cdc.gov/leptospirosis/index.html
FAO Manual on Meat Inspection for Developing Countries http://www.fao.org/docrep/003/t0756e/t0756e00.htm
International Veterinary Information Service (IVIS)
http://www.ivis.org
Public Health Agency of Canada. Pathogen Safety Data Sheets
The Merck Manual
http://www.merckmanuals.com/professional/index.html
The Merck Veterinary Manual
http://www.merckmanuals.com/vet/index.html
World Health Organization (WHO). Leptospirosis
http://www.who.int/topics/leptospirosis/en/
WHO Leptospirosis fact sheet http://www.searo.who.int/about/administration_structure/ce/cks/CDS_leptospirosis-Fact_Sheet.pdf
World Organization for Animal Health (OIE)
http://www.oie.int/
Disease and Infection Prevention Practices: Is Your Clinic Up to Snuff?
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This document is designed to provide a complete and readily accessible summary of infection prevention and control best practices for small animal veterinary clinics, and is intended to be understandable to all members of the veterinary practice team.

1. Infection prevention and control strategies are designed to protect patients, owners, veterinary personnel and the community. All veterinary personnel should play an active role in protecting every person and animal associated with the veterinary clinic.

2. Every veterinary clinic, regardless of type or size, should have a formal infection control program, a written infection control manual, and an infection control practitioner (ICP) to coordinate the program.

3. Some form of surveillance (either passive or active) should be practiced by all veterinary facilities. The keys to passive surveillance are to centralize the available data, and to have a designated ICP who compiles and evaluates the data on a regular basis.

4. Routine Practices that are critical to infectious disease prevention and control:
   a. Hand hygiene, including:
      i. Handwashing
      ii. Use of alcohol-based hand sanitizers
   b. Risk reduction strategies, particularly those related to:
      i. Use of personal protective equipment (PPE)
      ii. Cleaning and disinfection
      iii. Laundry
      iv. Waste management
   c. Risk assessment of animals and personnel with regard to:
      i. Disease transmission
      ii. Disease susceptibility
   d. Education
      i. Veterinary personnel
      ii. Animal owners
      iii. Public

5. All surgical procedures cause breaks in the normal defensive barriers of the skin or mucous membranes, and therefore carry an inherent risk of surgical site infection (SSI). Good general infection control practices (e.g. hand hygiene, cleaning and disinfection) are important for prevention of SSIs, but there are also specific infection control measures pertaining to surgery that should be considered.

6. Every veterinary clinic should have an isolation area for caring for and housing animals with potentially contagious infectious diseases.

7. Proper wound care is critical to preventing transmission of bacteria, particularly multidrug-resistant pathogens, between animals, personnel and the environment.

8. Animals from shelters and similar facilities should be considered high risk from an infectious disease standpoint and managed appropriately to prevent transmission of disease.

9. Safety of personnel and animal owners should always be a priority. Personnel should take all necessary precautions to prevent animal-related injuries (e.g. bites, scratches), and all bite wounds should be taken seriously. Proper sharps handling practices should be emphasized to reduce the risk of needle-stick injuries.

10. Education of personnel and clients about zoonotic and infectious disease risks and prevention is crucial.

Guiding principles
1. Infection prevention and control strategies are designed to protect patients, owners, veterinary personnel and the community.

2. A significant percentage of hospital-associated infections (HAIs) in veterinary clinics can likely be prevented with proper compliance to basic, practical infection control practices.
a. Although poorly quantified, HAIs occur in veterinary clinics and can have a significant impact on animal health. While the proportion of preventable HAIs in veterinary clinics is unknown, it has been estimated at 30-70% of HAIs in human hospitals are preventable (Haley et al. 1985).

3. A systematic approach to infection prevention and control requires all veterinary personnel to play an active role in protecting every person and animal associated with the veterinary clinic, patients or veterinary personnel.

4. Veterinary personnel need to follow infection prevention and control protocols at all times and use critical thinking and problem solving in managing clinical situations.

Basic principles of infection prevention and control

General concept

Every veterinary clinic, regardless of size and type, should have a documented infection control program. This may range from simply a written collection of basic infection control practices, to a formal infection control manual with specific training, monitoring, surveillance and compliance programs. Lack of a clearly defined infection control program may lead to unnecessary patient morbidity and mortality, and exposure of veterinarians, staff and owners to zoonotic pathogens. Improved infection control is a necessity as veterinary medicine evolves. Advances in veterinary medicine mean that animals are living longer, and owners are often expecting a higher level of care for their pets that is more comparable to what they themselves may receive. There are also more animals at higher risk for infection in general because of more invasive and immunosuppressive therapies. In addition to the desire to achieve “best practice” standards whenever possible, the increasingly litigious nature of society may be one of the driving forces toward improved infection control in veterinary clinics. While the potential liability associated with morbidity and mortality in individual pets is limited, the potential consequences of zoonotic diseases in owners and staff are significant and warrant careful consideration.

Infection prevention and control measures can be broadly divided into three main categories: those that decrease host exposure, decrease host susceptibility and increase host resistance to infectious pathogens.

1. Decreasing exposure is the most important aspect of disease control in most situations. If a pathogen does not encounter an individual, then disease cannot occur. The number of organisms to which a host is exposed is also an important factor in determining whether or not colonization or infection (disease) will ensue. Depending on the pathogen, decreasing or preventing exposure may be easy, difficult or impossible.

2. There are many factors that interact to determine whether or not infectious disease will develop in a particular host. In most cases, simple exposure of an animal to an infectious agent does not mean that disease will result. The susceptibility of the individual to a particular number of an infectious agent plays an important role. Although difficult to quantify, certain situations may result in increased susceptibility to infection and disease. Many factors causing increased susceptibility are not preventable, but some are, and efforts should be undertaken to address these issues. Factors to consider include judicious use of antimicrobials and other drugs, provision of proper nutrition, adequate pain control, and appropriate management of underlying disease.

3. Measures to actively increase resistance of a host are commonly used in veterinary medicine, but these should be considered only the third line of defense, after those meant to decrease exposure and susceptibility. Vaccination is currently the main technique used to increase resistance of animals or humans to infection. However, no vaccine is 100% effective. Therefore, while vaccination is an important part of infection prevention and control, it must not be the only component of an infection control program if the program is to be successful. In addition, many HAI-infections are caused by opportunistic microorganisms for which vaccines are unavailable.

Transmission

Microorganisms are transmitted in animal health care settings by four main routes: contact, droplet, air-borne and vector-borne transmission. The same microorganism may be transmitted by more than one route.

1. Contact transmission is the most important and frequent mode of transmission of health-care associated infections (HAIs). It can be divided into direct and indirect contact transmission.

a. Direct contact transmission involves direct body surface-to-body surface contact resulting in physical transfer of microorganisms from an infected or colonized animal. For example, two dogs in a waiting room that come into direct contact when they sniff each other may transmit pathogens present in their noses or perineal areas; direct contact of a veterinarian’s hands with a wound on an animal may result in transmission of opportunistic pathogens from the normal microflora of the person’s hands, or infectious organisms present in the animal’s wound, to the patient or the veterinarian, respectively.

b. Indirect contact transmission is the result of physical transfer of microorganisms from the original animal (or human) source to a new host, without direct contact between the two. This typically involves body surface contact with an inanimate object, environmental surface or the integument of another animal or person that has been transiently contaminated by the original animal (or human) source. For
example, handling one animal and then petting another animal without washing one’s hands constitutes indirect contact between the two animals.

2. **Droplet transmission** is theoretically a form of contact transmission. However, the mechanism of transfer of the pathogen from host to host is quite distinct from either direct or indirect contact transmission. Droplets are generated from the source animal primarily during coughing or sneezing, and during the performance of certain procedures such as suctioning. Transmission occurs when droplets containing microorganisms generated from the source animal are propelled a short distance through the air (usually less than one metre) and deposited on the new host’s conjunctiva (i.e. in the eye), nasal mucosa, mouth, or an open wound. For example, a cat with an upper respiratory tract infection can transmit viruses or bacteria to another cat in the waiting room by sneezing on it, particularly if they are face-to-face, even if the animals do not touch each other directly. Because droplets do not remain suspended in the air, special air handling and ventilation are not required to prevent droplet transmission; that is, droplet transmission must not be confused with air-borne transmission. Droplets can also contaminate the surrounding environment and lead to indirect contact transmission.

3. **Airborne transmission** occurs by dissemination of either airborne droplet nuclei (5 μm or smaller, about 2-3 times the size of most bacterial pathogens) from partly-evaporated droplets containing microorganisms, or dust particles containing the infectious agent. Microorganisms carried in this manner remain suspended in the air for long periods of time and can be dispersed widely by air currents. They may be inhaled by another host within the same room, or they may reach hosts over a longer distance from the source, depending on environmental factors. Airborne transmission of pathogens in veterinary clinics is very rare.

4. **Vector-borne transmission** occurs when vectors such as mosquitoes, flies, ticks, fleas, rats, and other vermin transmit microorganisms. Some act as simple mechanical vectors, comparable to indirect contact transmission, whereas others acquire and transmit microorganisms by biting. It is important to have control measures in place to reduce or eliminate the presence of such vectors in veterinary clinics.

**Routine practices**

Routine Practices are a way of thinking and of acting that forms the foundation for limiting the transmission of microorganisms in all health care settings. It is the standard of care for all patients/clients/residents.

– Rick Wray, Hospital for Sick Children, Toronto, Canada

Routine practices include:

- Hand hygiene
- Risk reduction strategies through use of personal protective equipment (PPE), cleaning and disinfection of the environment and equipment, laundry management, waste management, safe sharps handling, patient placement, and healthy workplace practices
- Risk assessment related to animal clinical signs, including screening for syndromes that might indicate the presence of infectious disease (e.g. fever, coughing/sneezing, diarrhea, abnormal excretions/secretions), and use of risk assessment to guide control practices

Personal protective equipment (PPE) is an important routine infection control tool. PPE use is designed to reduce the risk of contamination of personal clothing, reduce exposure of skin and mucous membranes of veterinary personnel to pathogens, and reduce transmission of pathogens between patients by veterinary personnel. Some form of PPE must be worn in all clinical situations, including any contact with animals and their environment.

These recommendations must always be tempered by professional judgment, while still bearing in mind the basic principles of infectious disease control, as every situation is unique in terms of the specific clinic, animal, personnel, procedures and suspected infectious disease.

Use of personal protective equipment does not eliminate the need for appropriate environmental engineering controls, such as hazard removal and separation of patient areas from staff rooms.

Personal protective outerwear is used to protect veterinary personnel and to reduce the risk of pathogen transmission by clothing to patients, owners, veterinary personnel and the public.

The objective of this summary is to review proper infection control measures for the treatment and management of dogs and cats with *Giardia* infections in shared animal facilities. Shared animal facilities include, but are not limited to, day-care, temporary housing, training facilities, parks, playgrounds and any other area animals congregate.

Despite your best efforts, *Giardia* can persist on items and in outdoor spaces. The risk of human infection and pet reinfection is possible, especially in shared animal facilities. [4]

**Giardia survival in the environment**

In cold temperatures, 4°C (39.2°F), *Giardia* survives about 7 weeks (49 days). At room temperature, 25°C (77°F), *Giardia* survives for 1 week. In a dry, warm environment in direct sunlight, *Giardia* survives a few days. [2, 3] In a moist, cool environment, *Giardia* survives up to several weeks. In water temperatures below 10°C (50°F), such as lake or puddle water in the winter and refrigerated water, *Giardia* survives 1–3 months. In water temperatures above 10°C (50°F) such as river water in the fall, tap water and puddles in the summer, *Giardia* survives less time, less than 4 days for water temperatures above 37°C (98.6°F).

**Proposed solutions**

- **a. Frequency of cleaning and disinfecting**
  Clean and disinfect potentially contaminated items daily or when fecal accidents happen for as long as an animal is sick. If an animal is taking medication, clean and disinfect every day until three days after the last dose of medication is given.

- **b. Cleaning and disinfecting**
  For hard surfaces (i.e., cement or tile floors, crates, tables and trash cans) cleaning should include: removing feces and discarding in a plastic bag, scrubbing surfaces with soap and rinsing surfaces until no visible contamination is present. Disinfect surfaces according to manufacturer guidelines, ensuring contact with the surface for the recommended amount of time. Either quaternary ammonium compound products (QATS) [1], found in some household cleaning products (may be listed as alkyl dimethyl ammonium chloride), or bleach with water (3/4 cup of bleach to 1 gallon of water) may be used. [2] After disinfection, rinse the surface with clean water.

  For carpets and upholstered furniture, remove feces with absorbent material (i.e., double layer paper towels) and discard in a plastic bag. Thoroughly clean the contaminated area with regular detergent or a carpet cleaning agent and allow the material to fully dry. To disinfect, either steam clean the area at 158°F for 5 minutes (212°F for 1 minute) or use a cleaning product with QATS. Read the label for specifications and instructions.

  Household items, such as toys, clothing and pet bedding, should be cleaned and disinfected daily while a dog or cat is being treated for *Giardia*. Dishwasher-safe toys and water or food bowls can be disinfected in a dishwasher that has a dry cycle or a final rinse cycle that exceeds one of the following: 113°F for 20 minutes, 122°F for 5 minutes or 162°F for 1 minute. If a dishwasher is not available, submerge dishwasher-safe items in boiling water for at least 1 minute (boil for 3 minutes at elevations >6,500 feet). Clothing, some pet items (i.e., bedding, cloth toys) and linens (i.e., sheets, towels) can be washed in a washing machine and then heat-dried on the highest heat setting for 30 minutes. If a dryer is unavailable, thoroughly air dry clothes in direct sunlight.

  **c. Reduction of giardia in outdoor environment**
  Remove feces promptly and place in a plastic bag, always while wearing gloves. [3] Limit access of animals with diarrhea or *Giardia* infections to common outdoor spaces. Eliminate all sources of standing water (i.e., puddles, containers with water, and fountains not in use) at your facility. Limit access of dogs to untreated surface water, such as creeks, ponds and lakes, to avoid infection and water contamination. Do not use bleach or QATS in your soil or grassy areas; they are ineffective. Keep new animals, especially young ones, from entering any shared outdoor spaces until advised by a veterinarian.

  **d. Reducing the risk of (re)infection during treatment**
  If an animal in your facility is diagnosed with *Giardia*, tell all facility pet owners to visit their veterinarians. Even animals without signs of illness can be infected and shedding *Giardia* into the environment and may need medication. [3] Bathe all animals with pet shampoo, following medical treatment, to remove fecal residue in their coats. [5] Clean your facility daily, as detailed above, making sure to wash your hands regularly with soap and water.

**Treatment**

No drugs are approved for the treatment of giardiasis in dogs and cats in the United States. Metronidazole is the most commonly used extra-label therapy, however efficacies as low as 50% to 60% are reported. Safety concerns for dogs and cats are also reported. Albendazole is effective against *Giardia* but is not safe in dogs and cats and should not be used.

[4http://www.cdc.gov/parasites/giardia/giardia-and-pets.html - four/}
Treatment of dogs

A combination of febantel, pyrantel pamoate, and praziquantel (DrontalPlus) is effective in treating Giardia in dogs when administered daily for 3 days using the dose bands indicated on the DrontalPlus label. Fenbendazole is effective in eliminating Giardia infections in dogs and has been approved for such use in Europe. Available experimental evidence suggests that it is actually more effective than metronidazole in treating Giardia infections in dogs. Fenbendazole should be administered (50 mg/kg SID) for 5 days. Alternatively, fenbendazole (50 mg/kg SID) may be administered in combination with metronidazole (25 mg/kg BID) for 5 days. This combination therapy, fenbendazole and metronidazole together, may result in better resolution of clinical disease and cyst shedding.[http://www.cdc.gov/parasites/giardia/giardia-and-pets.html - four]

Treatment of cats

Data on treatment of cats with Giardia are lacking. However, cats may be treated with either fenbendazole (50 mg/kg SID) for 5 days, metronidazole (25 mg/kg BID) for 5 days, or a combination of the two, as described above for dogs. There is anecdotal evidence that metronidazole benzoate is tolerated better in cats than metronidazole (USP).[http://www.cdc.gov/parasites/giardia/giardia-and-pets.html - four]

f. Refractory treatment

Treatment failures may result from: reinfection, inadequate drug levels, immunosuppression, drug resistance and Giardia sequestration in the gallbladder or pancreatic ducts. The presence of immunosuppression, reinfection, or sequestration can usually be determined in a clinical setting. Certain immunosuppressed patients are abnormally susceptible to giardiasis and their infections are often difficult to cure. Reinfection is common in endemic regions with high environmental contamination. [10]

g. Vaccine recommendation

Vaccines were previously available, but they are no longer being manufactured. Effectiveness of the vaccine was studied with variable results. While the Giardia vaccine was effective in treating dogs with chronic giardiasis, it was ineffective in asymptomatic canines. Many canines had persistent infections, even 6 months after vaccination. [12]

III. Conclusion

The risk of acquiring a Giardia infection from a dog or cat is small. However, there are some steps you can take to minimize your exposure and animal infection or reinfection. Promptly remove and properly dispose of feces, always while wearing gloves. Regularly clean and disinfect surfaces, areas and items in your facility that animals have access to, following appropriate guidelines and using appropriate products. Limit access of infected animals to common outdoor spaces and surface water.

IV. Recommendations

- All animals should be tested for parasites (by Elisa or PCR) every 3-6 months.
- All animals should be treated with parasiticides regularly. If parasiticide treatment is not routinely done, pets should be brought to a clinic every 3-6 months for treatment.
- Animals with any gastrointestinal condition should not attend shared facilities.
- Animals with fecal incontinence need to be diapered or should not attend facilities.

References


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In recognition of the fact that high-volume spay-neuter programs were becoming a critical component of the efforts to address pet overpopulation and reduce euthanasia of healthy dogs and cats in animal shelters in 2006 PetSmart Charities, Inc. and the ASPCA funded a task force of the Association of Shelter Veterinarians (ASV) to explore all aspects of high-volume spay-neuter programs. The task force consisted of 22 veterinarians representing numerous specialties as well as practitioners in high-volume spay-neuter clinics. After two years of work by the task force in July 2008 the Journal of the American Veterinary Medical Association (JAVMA) published “The Association of Shelter Veterinarians veterinary medical care guidelines for spay-neuter programs.” The guidelines were developed to be appropriate and achievable in all high-volume spay-neuter models: stationary clinics, mobile clinics, mash-style operations, shelter programs, feral cat programs, in clinic clinics and programs at veterinary colleges. The intent was to ensure a level of consistency, acceptability and professional in all high-volume spay-neuter programs. The guidelines included recommendations for preoperative, surgical, anesthetic and postoperative practices and were based on: accepted principles of anesthesiology, critical care medicine, microbiology and surgery; extensive review of the scientific literature; and expert opinions.

2016 guidelines
Recognizing that medical science is constantly evolving the intent was that the spay-neuter guidelines would be periodically revised. In 2014, once again, the ASV convened a task force to update the guidelines. In the 2014 task force the same specialties were represented with a membership mixture consisting of several of the original task force members along with several new members to broaden the perspective. With the support of PetSmart Charities, Inc. and the ASPCA this new task force worked for two years and has submitted a revised document to the JAVMA for publication.

With anticipated publication in the summer of 2016 the revised guidelines again are designed to be appropriate and achievable in all models of high-volume spay-neuter programs. In addition to sections addressing the most current knowledge related to preoperative, surgical, anesthetic and postoperative care the document has been expanded to include sections on patient care and clinical procedures as well as on operations management.

Authors note: The 2016 ASV Veterinary Medical Care Guidelines for Spay-Neuter Programs has been submitted for publication. Accordingly, specific content of the anticipated publication cannot be published in these conference proceedings.
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Authors note: The 2016 ASV Veterinary Medical Care Guidelines for Spay-Neuter Programs has been submitted for publication. Accordingly, specific content of the anticipated publication cannot be published in these conference proceedings.
Most veterinary schools teach students how to perform spays and neuters at a point in their education when they are very inexperienced surgeons. Students are taught techniques that are designed to compensate for lack of anatomical knowledge, inexperience and poor surgical skills. Students are taught to double ligate everything because instructors don’t trust their ligatures. Students are taught interrupted patterns because instructors don’t trust their knots. They are taught long incisions and extensive exposure because instructors recognize that students don’t fully understand abdominal anatomy. The techniques taught are safeguards to protect patients from students at that level of their education. However, as veterinarians gain experience in surgery they become much more competent and comfortable, but often fail to abandon those techniques that were simply designed to compensate for lack of experience? Many of those techniques can be replaced by ones that are much more efficient.

Patient positioning
When performing a spay where does the surgeon stand? What factors influence where you stand during a spay? Do you stand with the patient’s head to your right or to your left? Most right-handed veterinarians stand with the patient’s head to their left and most left-handed veterinarians stand with the patient’s head to their right. But why is this? Try standing with the patient’s head to the side of your dominant hand. There is a very valid reason for this. If you strum the suspensory ligament of the ovary this allows you to strum it with your stronger hand. If you cut the suspensory ligament it allows you to cut the ligament easily with your dominant hand. While I am not recommending that you change sides of the table if you have been doing surgery for years I am recommending that you always ask why you are doing a particular technique a particular way and consider if there is a better, more efficient approach.

In a spay, position the patient with the front legs along it’s side rather than pulled forward past it’s head. Pulling the legs forward, as is commonly done, tightens the muscles of the back and tightens the suspensory ligaments of the ovaries. Positioning the limbs along side the patient’s thorax relaxes the suspensory ligaments making exteriorization of the ovaries through a small abdominal incision easier. It also helps prevent the patient from rocking side-to-side. A simple restraint devise allows this positioning of the patient and helps prevent tilting of the patient to one side or the other. An alternative, that accomplishes the same purpose is to use a V-table or V-trough without tying the front legs at all.

Surgical techniques
Minimally invasive approaches
One key to efficient ovariohysterectomies is making appropriately placed small incisions. While most surgery instructors promote long incisions and maximum exposure; lengthy incisions are considerably more time consuming to close. Small incisions, obviously, can be closed much more rapidly than long incisions. The proper location of the incision varies with species and with age of the patient. The determining factor should be which tissues are most difficult to exteriorize. In a cat spay the tissue that is more difficult to exteriorize is the uterine body. In the adult dog it is more difficult to exteriorize the ovaries. Vary the location of your incisions accordingly. Puppies are intermediate. In the cat spay the skin incision should be located on the ventral abdominal midline with the midpoint of the incision being the midpoint between the umbilicus and the anterior brim of the pubis. In the adult dog, the skin incision is on the ventral abdominal midline just caudal to the umbilicus. In the puppy spay (6 months or younger) the skin incision should be located on the ventral abdominal midline just caudal to the umbilicus. In the adult dog the right kidney and the right ovary are located further cranial in the abdomen than the left kidney and left ovary. It is, therefore, more difficult to exteriorize the right ovary than the left ovary through a small incision. To equalize the difficulty of exteriorizing the two ovaries make the entry into the abdomen through a right paramedian incision. Incise the skin on the ventral abdominal midline, undermine only on the right side of the linea alba and, depending on the size of the dog, incise the rectus sheath 1/2 to 2 cm to the right of the linea alba. To prevent hemorrhage incise only the fascia. Enter the abdomen by bluntly separating the fibers of the rectus abdominis muscle and cutting the peritoneum.

Castration incisions in the cat, the puppy and in the adult dog can be made through the scrotum.

Ligation techniques
Most veterinary students are taught to double ligate ovarian pedicles and uterine stumps and to ligate before transecting the tissue. It is, however, much more efficient to transect the ovarian pedicles prior to ligation and to single ligate each pedicle. In the dog the most efficient technique is to place 3 hemostats, the first most proximal on the ovarian pedicle, the second several millimeters distal to the first, but still proximal to the ovary, and the third between the ovary and the uterine horn. Close the first hemostat one click, the
second two clicks and the third three clicks. The purpose of the 1, 2, 3 clicks is to avoid completely crushing the tissue at the most proximal clamp. Completely crushing predisposes the pedicle to tearing. Before ligating, transect the ovarian pedicle just distal to the second hemostat, between the second hemostat and the ovary. Ligate with a square, surgeon’s or Miller’s knot. If you are skilled at hand ties that, too, will improve your efficiency.

**Hand ties**

Becoming skilled at hand ties; square knot, surgeon’s knot and Miller’s knots will improve efficiency in both dog and cat spays. To be efficient this skill must be practiced. But once you are skilled at hand ties they increases your speed significantly.

**Pedicle ties**

The pedicle tie is a method of ligation in which the structure is tied to itself around a hemostat. The pedicle tie can be used in cat castrations, puppy castrations and in ligating the ovarian pedicles in cat spays. There are several variations of the pedicle tie in the cat spay. In the technique I use, deliver the ovary through the abdominal incision, cut the suspensory ligament and tear a hole in the broad ligament just caudal to the ovarian vessels. Hold the ovary in your non-dominant hand and gently pull the ovary towards you. Using the dominant hand a curved hemostat is crossed over the ovarian vessels into the hole in the broad ligament and underneath and behind the vessels. The hemostat should be held closed with the tip of the hemostat facing away from you. The tip of the hemostat is then directed above the vessels as the hemostat is rotated counter-clockwise to end up facing you. The hemostat is opened and used to clamp the ovarian vessels. The vessels are cut or torn between the hemostat and the ovary and the knot is gently pushed off the tip of the hemostat. The knot should be pulled tight before releasing the hemostat. This technique cannot be used in the ligation of ovarian pedicles in dogs or puppies. Canine ovarian pedicles generally contain a significant amount of fat which interferes with making a secure knot.

**Miller’s knot**

The Miller’s knot is a very secure, self-locking knot that can be placed either with an instrument or with a hand tie. The Miller’s knot can be used on spermatic cords, on ovarian pedicles in dogs and uterine bodies of dogs and cats. To place a Miller’s knot pass the suture under the tissue to be ligated, bring the suture back over the tissue and under the tissue one more time. This creates a small loop of suture above the tissue to be ligated. Position the needle holder through that small loop, wrap the long strand once around the needle holder, grasp the short strand of suture with the needle holder and pull the needle holder towards you while pulling the long strand of suture away from you. Gentle upward tension while pulling this knot tight facilitates placement of the ligature. Complete the knot by place three or four more square knot throws.

**Scrotal castrations in adult dogs**

Scrotal castration are rarely ever taught in veterinary school, in fact, for decades veterinarians have been taught to avoid making incisions in the scrotum of dogs. Scrotal castrations appear, however, to offer several advantages over the prescrotal approach including, smaller incisions, less surgical time, and less tendency for scrotal self-trauma. The justification for avoiding scrotal castrations in dogs had been to prevent self-mutilation, but as long as no external skin sutures are placed in the scrotum there is less risk of self-trauma in a scrotal castration than in a prescrotal castration.

Position the patient in dorsal recumbency. Grasp one testicle and position it in a manner that elevates and exposes the median raphe. Make an incision through the skin and subcutaneous tissue along or near the median raphe over the displaced testicle. Continue the incision through the spermatic fascia to exteriorize the testicle. In the closed castration technique care is taken not to incise the parietal vaginal tunic and tunica albuginea. Use gentle traction to exteriorize the testicle and reflect fat and fascia from the parietal tunic of the spermatic cord using a gauze sponge. Place three hemostats on the spermatic cord and transect the cord distal to the third hemostat. In smaller dogs (under 18 kg) a single ligature tied with a Miller’s knot and placed in the crushed area of the most proximal hemostat is sufficient for hemostasis. In larger dogs (18 kg and above) a transfixation ligature is placed in addition to and just distal to the Miller’s knot. The second testicle is exteriorized through the same scrotal incision. A second incision in spermatic fascia is made over the second testicle to allow exteriorization, transection and ligation of the second spermatic cord is accomplished in a manner identical to the first testicle.

The technique for closure is the surgeon’s preference. Incisions can be left open to heal by second intention, can be partially closed with one buried subcutaneous suture of absorbable suture material, or can be closed fully with skin glue. All three of these techniques are considered acceptable. Do not close with external skin sutures.

**Age at which surgery is performed**

As a general rule the larger the animals is (dog or cat), the more obese the animal is, and the older the animal is, the longer it will take to perform a spay or neuter surgery. Even though most of us were taught to wait until a dog or cat is sexually mature (six to nine months) before sterilization surgery there is growing evidence that there is no reason to wait until the animal is an adult. Pediatric spay neuter has been shown to have little or no adverse physiologic effects on the animal and spay/neuter in the pediatric patient is much easier and quicker than that in the sexually mature patient.
Conclusions
Becoming efficient at spays and neuters is a combination of many factors. One of which, of course, is the skill and comfort level of the surgeon. Adoption of specific techniques that are used commonly in high-volume spay neuter clinics can significantly improve surgical efficiency. Being willing to question why you were taught specific manipulations in veterinary school and recognizing that it is acceptable to abandon some of them (such as always double ligating pedicles) will improve surgical efficiency greatly.
Upper Respiratory Infections (URI) can be a frustrating and extremely costly problem in shelters. The financial costs are easy to measure by looking at the cost of drugs to treat the disease and the amount of staff necessary to maintain isolation wards full of ill cats. The other costs are harder the measure. The increase in suffering and loss of life are dramatic. The emotional toll it takes on employees to care for sick cats only to often see them die cannot be measured. The reputation of the shelter suffers in the community, which may lead to fewer adoptions and compound the problem as fewer cats are leaving the shelter. While many shelters are successfully managing their cat populations and keeping them happy and healthy, there are also many that are not. In East Tennessee, we are seeing many shelters with URI rates nearing 100%. We see the employee burnout; we see the cats suffering and getting so ill that the only option left is euthanasia. This is a very preventable problem. The first step is recognizing that this is truly a welfare issue that needs to be addressed immediately and not just a “kitty cold”. While veterinarians recognize this and can see the suffering these cats endure, we need to remember that many shelters workers do not. Education is the key to improving feline welfare.

The pathogenic causes of URI in animal shelters are primarily Feline Herpesvirus type 1, Feline Calicivirus, Bordetella bronchiseptica, Chlamydophila felis, and Mycoplasma felis. It is important for veterinarians to have a basic understanding of each of these pathogens and an understanding of how proper disinfection and cat handling can decrease fomite transmission of illness. It is also vital that the practitioner understand the latency and recrudescence of FHV-1 specifically. In most cases, however, it is not necessary to know which of these pathogens is causing illness in the population. Testing can be prohibitively expensive, and infections are often caused by more than one organism. Also, these organisms are so common that in the short time it takes to receive test results another infectious organism can be introduced to the shelter. If the shelter is experiencing an abnormal or especially virulent outbreak of URI, a combination of an oropharyngeal and either conjunctival or nasal swab can be submitted for PCR testing. PCR testing has been shown to be a sensitive method for detection of the infectious agents in cats.1

One of the first things I recommend doing before implementing any changes is to determine the current disease rate. If good data tracking has been previously implemented, this will be as easy as determining the percentage of cats that have shown symptoms of URI after being admitted to the shelter. Incomplete data sets aren’t necessarily very helpful when monitoring illness, but there is great potential for shelter software to track the frequency and risk factors for URI.2 This data collection will look different for different shelters, based on intake and release statistics as well as which software system is used. This can be difficult with paper records but is possible. At a minimum track the number of cats that develop URI signs and on what day it is first recognized. Also note health status on intake and at final disposition.

Having accurate data is useful for many reasons. Not only can it help determine the true rate of disease in the shelter, but it can be used to determine if a certain age group, housing area, or other defining factor is increasing disease risk. The rate of disease is important to have before you start to implement changes. While we can easily look in a shelter and know that it has a problem with URI, it is difficult for us to assess over time if that problem is improving. Having accurate data can be used to determine if the changes being implemented are effective and if they are causing any secondary benefits or concerns. Employees that are experiencing burnout or compassion fatigue will be reluctant to change, and allowing them to see the numbers and recognize that changes are helping cats may go a long way in convincing them to make further changes. Without data it may be hard to prove the benefit of your work.

In most areas, cat intake is seasonal so a year’s worth of data would be ideal. Watching cats suffer for a year without making changes is not an option, however. Many times we collect as much data as we can while we work on plans to alleviate other immediate concerns, always remembering that the welfare of the animals is our primary focus.

The relationship between overcrowding in shelters and clinical signs of URI has been widely discussed for some time. One of the ways to lower overcrowding is to lower the intake of cats. In many municipalities the stream of cats coming into the shelter seems never-ending and is often more than can possibly be adopted or sent to rescue. The financial and emotional burden of caring for these cats is great. One unfortunate sequela is that employees do not want to have to euthanize these animals so shelters turn into “warehouses” for them to live until they get sick and need to be euthanized. This takes the control of which cats we devote our time and resources to out of our hands. A lot of this can be cured by lowering intake and making appropriate population management decisions at intake.

Effective Shelter-Neuter-Return (SNR) programs can lower shelter intakes, euthanasia rates, and allow for more resources to care for cats that do develop URI while in the shelter.3,4 Setting up SNR programs can be a big undertaking, but many grants exist to help and volunteers are available in nearly every community to assist with programs. A simple internet search can find numerous resources.
available to those wishing to set up SNR programs in their communities. Some communities may find SNR programs to be the more financially sound decision when choosing between SNR and Trap-kill.

Geographic information systems (GIS) mapping is proving to be an excellent tool for determining where intakes are clustered and focusing outreach resources.6-8 Analyzing GIS data to determine where specific groups of animals (i.e. owner relinquished adult cats, kittens, cats that are unhealthy on intake, etc.) can help the organization target specific areas in an effort to decrease those specific types of intakes.

Managed admission is a relatively new concept that a lot of shelters either fear or believe they can’t implement in their organization. The University of Wisconsin Shelter Medicine Program has an excellent online video series that includes a lecture on managed intake.7 I would encourage any shelters considering this to watch the presentation on their website. Carefully managing the flow of animals into the shelter will allow resources to be allocated efficiently and allow the shelter to better stay within their capacity for care.

Many other opportunities for managing URI happen at intake. These include vaccinating all cats over 4 weeks old with a modified live FVRCP vaccine8, performing intake examinations that closely check for signs of infectious disease, making appropriate housing decisions to decrease the amount of times a cat has to be moved, and designating cats as fast track or slow track to help decrease length of stay. Careful handling of all cats during the intake process is also important to decrease their overall stress level and prevent recrudescence of herpesvirus.9 Ideas for writing successful intake protocols can be found in my lecture in these proceedings.

Stress is a primary contributing factor to cats developing URI in the shelter because any source of stress can decrease immune function and increase the susceptibility to illness.7 This stress can be related to inappropriate housing, increased length of stay, poor nutrition, moving cats often, poor air quality, poor cleaning and disinfection practices, excessive noise, a lack of proper enrichment, and many other aspects of day-to-day shelter operations.

Humane housing should be at the forefront of any URI management plan. Recent work at UC Davis has shown that cage size is related to infection rates as well as to euthanasia rates.10 Large cages can help make cats less stressed, less likely to get sick, and less likely to be euthanized. Further research11 looked at multiple risk factors for development of respiratory infections in cats and found that inadequate floor space was one of the primary risk factors. It is recommended that individual cat kennels have 9 square foot of floor space with separation between food, resting, and elimination areas. This research has yet to be published, but is very exciting news for animal shelters because it strongly suggests that if we operate within our capacity for care and provide adequate housing we could significantly lessen the burden of URI.

Elevated space, hiding boxes12, open kennel sides, outdoor space, appropriate litter boxes, and separated eating and elimination areas are all important housing considerations. Careful, planned group housing is also important and should be reserved for cats over 5 months old that are social with other cats.

The longer a cat stays in a shelter, the more likely it is to develop clinical signs of respiratory disease.13,14 Thus, lowering the average length of stay (LOS) should also be at the forefront of any URI management plan. Addressing housing concerns will have the secondary benefit of lowering LOS. In many shelters, larger housing for cats involves making portals in kennels. Portalizing kennels often means that the organization needs to find its adoption driven capacity and find ways to maintain that capacity.15 Finding and maintaining that capacity will lower the LOS. Lowering the LOS will lower URI. The opposite is also true; lowering URI also lowers LOS since cats are not sitting in isolation wards for 1-2 weeks. From personal experience, I have found that cats that are healthy are much more likely to get adopted quickly. This will also lower the LOS. Problems in animal shelters are often said to be never-ending cycles where one problem leads to another, causing a domino effect. Solutions in animal shelters can be seen the same way. One positive change will lead to another, and the cycle will continue. Improving housing, lowering LOS, and lowering URI rates can be viewed in this way.

Rehousing cats often and disinfecting their entire kennel daily causes enough stress to cause a reactivation of latent FHV-1.16 Cat moves can be made less stressful by bringing a hiding box and bed with them from one kennel to the next. Moves can be made less frequent by determining if a cat will be fast track or slow track during the intake exam and moving them to the appropriate area immediately. Open selection is another means of moving healthy, adoptable cats immediately to the adoption area when they are on stray hold.17 Cleaning kennels using spot cleaning protocols decreases rehousing stress for the cat and will decrease the amount of staff time required to clean cat holding areas.18

Air quality is a concern in many shelters, especially those that are older and were not initially designed to be shelters. For many years, we relied on air filters and air turnover to improve air quality. Proper ventilation is important, but we should be looking beyond that. Fresh air should be used whenever possible. This includes outdoor space and open windows. We also need to pay attention to cleaning protocols and ensure that we are not causing poor air quality by spraying chemicals that may irritate upper airways in areas where animals live. Overcrowding can also lead to poor air quality as litter dust and odors are abundant.

Behavioral enrichment should not be forgotten when considering feline health. As veterinarians, we should always remember that an animal is not truly healthy unless it is emotionally and physically healthy. We must meet those emotional needs in order to truly provide humane care. A couple of very interesting studies have been published that have shown that cats that enter the shelter content
are more likely to stay content if provided with daily human interaction.\textsuperscript{19} Also, cats that enter the shelter anxious may experience more contentment if gentled.\textsuperscript{20} More research needs to be completed, but this strongly suggests that positive human interactions may lead to lower incidence of URI. A trend that is becoming popular is to have children read to and pet cats in after school programs. Anecdotal evidence is showing benefits for both the cats and the children.

Further measures that could be taken include dampening all noise, ensuring all cleaning protocols disinfect while causing low amounts of stress, providing a high quality consistent diet that is age-appropriate, and allowing staff and volunteers the time to interact with cats and potential adopters.

Daily rounds to monitor the health and behavior of every cat in the shelter are important to URI management and to shelter health as a whole. These rounds should include checking every cat for signs of infectious disease and also checking to ensure the cat is eating. If it doesn’t cause undue stress, cats should be weighed weekly to monitor for weight loss. Weight loss increases as stress scores increase and may be an indicator that the cat is at risk for URI.\textsuperscript{21}

The first and most important thing to remember about treating URI in the shelter is that your focus is on the population and the health and welfare of every cat in the shelter. Some shelters are fortunate enough to have many resources available to them, including the funding and staffing to run isolation wards. It is important to remember that no matter how well you can manage these wards—-they should be empty.

In order to ensure the most humane care of individuals while maintaining the absolute best welfare for the population, protocols need to be clear and concise to everyone involved in decision-making. At what point a cat is moved to isolation, when antibiotics or other medications are started, when other supportive care is warranted, and at what point euthanasia is considered are just some of the considerations that need to be in this protocol. Staff members will often disagree on these decisions, and even staff veterinarians may have disagreements. Clear, written protocols will ensure fair and equal treatment for all cats, prevent the overuse of antibiotics, and allow for rational decisions when it comes to transfer or euthanasia.

The best example of a treatment protocol I have found was written by Dr. Cindy Karsten and is available at: \url{http://www.sheltermedicine.com/library/uri-sample-treatment-protocol}. This protocol can easily be adjusted to meet the needs of your shelter and your preferences for treatment. For many of our shelters in East Tennessee, advanced treatment is not an option, and a veterinary consult for euthanasia has replaced some of the later treatment options. Where that line is drawn depends on the live release rate at your facility as well as the resources you can allocate to the treatment of cats. Nearly every shelter can use some aspect of this protocol, however, even if they use it only to decide when cats go to isolation and which cats would benefit from antibiotics. For private practice veterinarians that work off-site, you will need to check local and state laws to determine if staff members are allowed to start antibiotics when you have not seen the patient.

Debate has surrounded the use of L-lysine, interferon, cefovecin and antivirals in the prevention and treatment of respiratory infections in shelter cats. L-lysine has been shown to not be effective at controlling URI in shelters.\textsuperscript{22,23} Interferon has been studied as a potential therapeutic option\textsuperscript{24,25}, but more research needs to be done in shelter situations. Cefovecin injections would likely be less stressful than oral medications for both cats and staff, but doxycycline and amoxicillin-clavulanic acid have both been shown to be more effective therapies.\textsuperscript{26} Famiciclovir has been shown to improve outcomes\textsuperscript{27} but controversy surrounds its use in shelters. It has been shown to be safe and well-tolerated, but drug-resistant strains of FHV-1 have been described\textsuperscript{28} so it should be used with discretion. Also, a single dose of famciclovir at intake has been shown to not be effective at preventing respiratory infections.\textsuperscript{29}

It cannot be stressed enough that the goal of URI management is prevention rather than treatment. By maintaining the shelter’s capacity for care and allocating resources to improving the husbandry of all cats, you can maintain a happy, healthy feline population without utilizing resources on extensive treatment and isolation wards.

References
Sheltering Starts When They Enter the Door: How to Implement a Health Intake Protocol

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The first thing shelters tend to ask is if they need a formal intake protocol. The answer to that question is always yes. Written, standardized protocols are vital to shelter operations to ensure the flow is running as smoothly as possible and good decisions are being made by all employees and volunteers. While many gray areas exist in sheltering, the more we can keep things black and white the fewer mistakes will be made and the less we will see decision fatigue in our staff. Many decisions are made for animals as soon as they walk in the door (and sometimes before) and it is vital that these decisions are based on sound practices and not emotional state.

Intake protocols are necessary for maintaining a healthy shelter population. The behavioral and medical needs of the animals in your care need to be assessed immediately to allow for smooth flow through the shelter. Intake protocols help designate those animals that may need more care and those that can move through the shelter quickly. Allowing these decisions to be made immediately will help the shelter avoid bottlenecks in every other area of the shelter. Intake protocols are also necessary to prevent infectious disease, to control parasites in the shelter, to ensure proper nutrition of shelter animals, and to maintain a safe atmosphere for visitors and volunteers.

Many times “intake” starts before the animals enter the shelter. Many welfare organizations struggle with deciding how to schedule proper staffing for intake, which animals will be able to find homes, and how to divert animals when the shelter may not be the best option for them. While these struggles vary shelter-to-shelter based on many factors a balance must be achieved within each organization. Many times this includes utilizing targeted TNR or outreach programs to reduce intakes from certain areas, using diversion protocols to keep animals out of the sheltering system when that is not the best option for them, managing admission so staffing can be adequate to ensure protocols are followed, and offering programs like finder to foster that allow those that find animals to foster them until they are able to successfully move through the shelter. An added bonus of some of these programs is that it allows vaccinations to be given in advance of admission to the shelter, giving the animal the benefit of immunity to some infectious diseases prior to intake.

The first place to start with writing any shelter protocol is to involve all stakeholders in the process. This team should involve at least the director, a minimum of one staff member that will work in intake, and a veterinarian. If the shelter does not have a staff veterinarian, they should consult with an experienced shelter veterinarian before finalizing any protocol. Many online resources exist that can help non-veterinary personnel write medical and behavioral protocols, but it is important that a veterinarian review them and ensure they meet the needs of that particular organization.

The first thing that needs to be done when an animal enters a shelter is to assess for emergent medical needs. If emergency medical care is needed, a veterinarian should be notified before any further work is performed. As long as the animal appears stable, the intake procedure can be followed as written.

Written behavioral and medical history is important for many reasons. A standard questionnaire should be given to owners or “finders” when they turn an animal into the shelter. Studies have shown that information provided by relinquishing owners is sometimes correlated with the post-adoption behavior. Those that have recently found animals may have little knowledge about them, but any information they can provide will be valuable. It is also very important to note where the animal was found and what it was doing when found. This is also a time to discuss the reasons for relinquishment and offer any available diversion programs.

Next a brief physical exam should be performed. This exam needs to include weight with body condition score, age, sex and reproductive status, signs of illness or injury, and noting any tattoos or microchip. In areas where dermatophytosis is a concern a Wood’s lamp examination should be performed on incoming cats, and a protocol should be in place for what to do if that test is positive. Because microchip scanners are not 100% reliable, it is recommended that animals be scanned for a microchip on at least two occasions during processing.

Animals should be vaccinated with age-appropriate vaccinations. It is recommended that all dogs over 4 weeks old are vaccinated with MLV DHPP at intake. All dogs and puppies should also be vaccinated with a modified live intranasal vaccine containing at least Bordatella bronchiseptica and Canine Parainfluenza on intake. This vaccination can be given to puppies as young as 2-3 weeks old. All cats over 4 weeks of age should be given a MLV FVRCP vaccination upon intake. For pregnant animals and other groups where vaccination may create risks, the risk of vaccination needs to weighed against the risk of the animal being exposed to a potentially fatal disease. For dogs, shelters may choose to run serology or foster pregnant bitches outside the shelter. Pregnant queens are often vaccinated and spayed or sent to foster homes where they can avoid exposure. Protocols for such situations are shelter-specific and highly dependent on live release rates and the ability of the shelter to adopt out those animals as well as the risk of exposure to disease in that shelter. Because mishandling of vaccinations is thought to be a primary cause of vaccine failure, a refrigerator needs to be
easily accessible and designated for non-food use and all staff need to be trained on the importance of proper vaccine handling and administration.

Parasite control measures are important for many reasons and should also be written into intake protocols. Many parasites are ubiquitous and some can cause serious illness. Parasites may also reduce disease resistance so other pathogens can cause concomitant disease, and some have zoonotic potential. Preventative measures depend on the geographic location of the shelter and what parasites are likely to be present in that region. At a minimum, all dogs need to be treated for round and hook worms. For many shelters, oral pyrantel is given to all animals as well as topical or oral flea and tick preventatives. Many shelters are also finding it useful to administer ponazuril to all animals or all puppies and kittens on intake. Online resources are available to help with dosing this medication. Shelters that see high numbers of specific parasites may elect to also treat for those parasites at intake.

Venipuncture may or may not be part of the intake exam. In many situations, this is done at the time of the veterinary exam because animals are stressed during intake, and staff may not be trained well enough to master the procedure. Multiple attempts by untrained staff may cause substantially more stress for the employee and the animals. If animals are fast-tracked and placed directly into adoption, the shelter may wish to perform heartworm testing on dogs and FeLV and/or FIV testing on cats during the intake process. If cats are to be moved immediately into group housing, testing will need to be performed before the cat is moved. If these diagnostics are part of intake, ensure that all staff are properly trained to perform venipuncture and perform these diagnostics. Errors that cause false positives or false negatives could be life-threatening.

An ID band or collar with a tag should be placed. A photo of the animal with additional photos of any identifiable marks should be taken so they can be posted to the website and used for the animal’s medical record. These pictures are important to help reunite lost pets with their families. Many municipalities are moving away from shelter workers attempting to breed-identify dogs during this process. Studies have shown that workers are often not able to properly identify breeds, and mislabeling a dog can have disastrous effects for the animal, particularly if breed-specific legislation exists.

Personnel should carefully note the behavior of the animal during handling and examination. Often dogs that are very friendly and not a behavioral concern can be moved directly to the adoption floor without further behavior testing. Behavior testing is being debated in many circles currently and research is suggesting that we urgently need further research and possibly a more holistic approach to behavior testing. Some shelters are choosing to SAFER test dogs at time of intake, which may be problematic if the dog is displaying signs of stress. An ideal time hasn’t been determined for behavior testing yet, but should be avoided in the stressed dog. Many shelters are choosing to perform this testing 2-3 days after the dog arrives at the shelter, and many are electing to forgo behavior testing and instead using the dog’s behavior during examination and handling to determine its eventual outcome. Behavior can be more difficult to determine in cats. To the untrained employee, a frightened pet cat may appear to be feral. One survey found that as low as 15% of shelters have written guidelines for assessing cats. Protocols for assessing feline behavior that include time for stressed cats to acclimate need to be in writing, and all employees need trained to properly and fairly perform these assessments. One assessment that is used in many shelters is the research-based ASPCA Feline-ality. It has been shown to work well in a modified form that can be used as early as 18 hours post-intake. Behavioral assessment and modification is a rapidly emerging and important aspect of shelter medicine that cannot possibly be fully covered in the confines of this lecture. The reader is encouraged to do further research into the subject area before determining which behavior assessments to use in their shelter and at which point in the animal’s stay to perform such testing.

Next the intake team member needs to determine a pathway for the animal and assign housing. Assigning housing immediately allows for less animal movement which lowers the stress the animal experiences while in the shelter and decreases the amount of work the shelter staff have to do later. It also allows for easier flow through the shelter. If the animal is housed in isolation due to signs of infectious illness or has any other medical concerns, the medical team needs to be alerted. Prompt treatment of medical problems can lead to an animal being cleared for adoption faster.

Assigning a pathway and housing is another area where clear and concise protocols are necessary. Repeatedly making life and death decisions is extremely stressful for staff, and defined protocols can help alleviate that burden. Assigning the initial pathway at intake will identify those slow track animals that may need additional resources, including behavioral or medical intervention, or may need promoted and highlighted as soon as possible. It also protects the most vulnerable population (puppies and kittens) by allowing them to move through the shelter quickly. Animals that are likely to have longer stays can be assigned to larger housing with more enrichment, and cats that will likely be at the shelter for longer periods can be moved into group housing if available.

Determining which animals are likely to get adopted quickly is very shelter-specific. This used to be determined by the local community, but with more adopters using the internet and driving long distances to obtain their perfect animal, this is no longer the case. People adopt animals for a number of different reasons including general appearance, social behavior, personality, age, size, breed, sex, coat pattern, color, and in-kennel behavior. One study even showed that people tend to assume “cuter” dogs have better behavioral traits. Overall, it is important for the shelter workers to understand what factors tend to shorten length of stay at their shelters and what factors tend to lengthen it so animals can be properly assigned a pathway. It is also important to understand that adopters do like a variety of animal types so it is a benefit to have a mix of slow and fast track animals in adoption at any given time.
The importance of accurate records and cage cards cannot be stressed enough. It is very important that the staff member take the time to complete a full and accurate record before moving on to the next arrival. If time elapses and the worker sees more animals before going back to the record, it can be assumed that the risk of errors rises. Inaccurate medical records could cause many devastating outcomes, including an owner not being able to find a missing pet or an animal being mistakenly euthanized.

After the intake process is complete, the surfaces in the intake room need to be disinfected, allowing enough time to permit proper contact time. The intake room should be fully disinfected after the arrival of an animal showing signs of infectious disease and at the end of every day.

Most shelter workers can read these recommendations and understand the necessity for each step in the process. Unfortunately at many shelters these steps are not followed. Often this is not the result of people being unwilling to perform intake procedures properly but is simply a result of shelter staff being overburdened with the amount of intakes and the inconsistency with which they arrive. As mentioned previously, there are many ways to improve this and help maintain the shelter’s capacity for care.

Effective Shelter-Neuter-Return (SNR) programs can lower shelter intakes. Geographic information systems (GIS) mapping is proving to be an excellent tool for determining where intakes are clustered and focusing outreach resources. Analyzing GIS data to determine where specific groups of animals are coming from can help the shelter target outreach efforts aimed at those populations in an effort to decrease those types of intakes. For example, if data analysis reveals that most puppies are coming from one area of the city, and those puppies have higher than average rates of parvoviral enteritis, the shelter could focus spay/neuter and low cost vaccination resources in that area. Monitoring that data could determine if those outreach efforts were working to decrease intakes and infectious disease rates in the shelter.

Managed admission is a concept that a lot of shelters either fear or believe they can’t implement in their organization. The University of Wisconsin Shelter Medicine Program has an excellent online video series that includes a lecture on managed intake. I would encourage any shelters considering this to watch the presentation on their website. Carefully managing the flow of animals into the shelter will allow resources to be allocated efficiently and allow the shelter to better stay within their capacity for care.

Managed admission and diversion programs go hand-in-hand. The shelter is not the best option for many animals, especially if admitting those animals will mean the shelter operates at above their capacity for care. People need to be educated on other options that exist for re-homing their animals. Pet food pantries, assistance with medical costs, and maintaining lists of housing that is pet friendly are just a few of the ways to help keep pets with their families. Outreach services that will work vary greatly by community, and organizations may need to be creative to find the best ways to keep animals out of their shelters.

Any written protocol should be based on the individual sheltering organization’s needs with the ultimate goal of preventing compassion fatigue and ensuring the best possible outcomes for the animals in the shelter. Improving welfare while minimizing suffering should be at the heart of all shelter protocols.

References


Canine Parvovirus (CPV), Canine Distemper Virus (CDV), and Feline Panleukopenia Virus (FPV) are all historically devastating pathogens in the shelter setting. Many shelter workers fear the introduction of these viruses into their populations. While they can be devastating to the individual, proper prevention of disease and management of outbreaks can keep the infections from devastating the population. Protocols for daily operations and outbreak management can save not only lives but also the reputation of the shelter.

A full review of each virus is not necessary, but understanding certain characteristics of these pathogens is important to a discussion about infection control and outbreak management.

Canine Parvovirus (CPV) has a relatively short and defined incubation period. Most dogs will develop clinical signs within the first week of infection, but the incubation can be as long as 14 days. Clinical signs of disease are straight-forward and include vomiting, diarrhea, lethargy, and dehydration. Diagnosis is relatively inexpensive and can be performed in-house. A fecal enzyme-linked immunosorbent assay (ELISA) antigen test is often performed in conjunction with a blood smear for more accurate diagnosis. CPV is very sturdy and can survive long periods of time in the environment. Despite the name, cats can be infected with CPV. The virus may cause clinical in this species or they may act as a reservoir and source of environmental contamination.1,2

Feline Panleukopenia Virus (FPV) has a relatively short and defined incubation period. Most cats develop clinical signs within 7-10 days, but the incubation period can be as long as 14 days. Clinical signs of disease include vomiting, diarrhea, dehydration, lethargy, and sudden death. In-house tests for canine parvovirus in feces have been shown to be diagnostic for FPV in cats3, and the IDEXX SNAP Parvo test has been shown to have minimal vaccine interference.4 White blood cell counts and necropsy also aid in diagnosis. FPV is very sturdy and can remain in the environment for long periods of time.

Canine Distemper Virus (CDV) has a long and poorly defined incubation period. Clinical signs may appear in 1-2 weeks or may take more than a month to develop. The signs of disease can mimic “kennel cough” and be missed in a shelter than often sees upper respiratory infections in dogs. Signs vary greatly and can involve multiple organ systems. Simple, accurate in-house diagnostics do not exist, further complicating proper diagnosis. CDV is less sturdy and easier to remove from the environment, but recovered dogs can shed the virus for months and recontaminate a shelter post-outbreak.

Despite the obvious differences, there are many commonalities among these viruses. There is a constant threat of introduction into the shelter and plans must be in place to prevent their spread. All of them increase suffering, death, and euthanasia. They cause morbidity and mortality in otherwise healthy and highly adoptable animals. Antibody titer tests are available for all of them, however, and vaccinations are highly effective and quickly effective if administered properly.

Epidemiology Review

A review of terminology related to epidemiology is also pertinent to this discussion. First, outbreak (or epidemic) refers to a sudden increase in the number of cases above what would normally be expected in a population. An epidemic curve can be used to plot the number of affected animals in a population over time. In shelters with good prevention protocols and high community vaccination rates, many infectious diseases could be labeled as sporadic, meaning that you see them infrequently and irregularly. In shelters with good prevention protocols but low community vaccination rates infectious diseases could be endemic, meaning that they are a constant presence in your shelter in low numbers. In shelters with poor prevention protocols and low community vaccination rates infectious diseases could be hyperendemic, meaning that you see persistent, high levels of disease occurrence. If you think you may have an outbreak but have not tracked your normal disease rates, you have a cluster.5

Another valuable tool is the attack rate. Measuring the attack rate is relatively simple and done by dividing the number of cases by the total population. This is very useful in shelter disease outbreaks because the attack rate can be determined for various risk factors to help determine potential causes. For example, the attack rate could be determined for cohorts of animals transported from different shelters or for all animals in one specific holding area. If vaccine failure is suspect, attack rates for animals vaccinated by different people or on different days could be determined. This is a simplified explanation, and the reader is encouraged to review the courses available at www.cdc.gov for further information.

Outbreak Prevention

The best way to prevent suffering, decrease euthanasia, decrease the financial burden of disease, and prevent compassion fatigue in shelter workers is to prevent infections rather than to treat them. Some shelters may feel that prevention is expensive and time-consuming but will often find that outbreaks prove to be much more expensive and exhaustive for staff. If day-to-day operations include disease prevention protocols, single cases of CPV, CDV, and FPV will remain single cases and not become outbreaks.

In many shelters, especially where vaccination rates in the community are low, all three of these viruses will occasionally be introduced into the facility. Animals may present either displaying clinical signs or in the incubation or pre-clinical phase. With
Chronic stress from overcrowding can lower the animal’s resistance to infection by compromising the immune system. Overburdened shelter workers may be less likely to follow sanitation and disinfection protocols properly. These are just some of the factors that illustrate how important it is to maintain the capacity for care in order to prevent infectious disease spread. Shelters need to determine their capacity for care and stay within that capacity in order to minimize disease risk and improve welfare.

Training intake staff to immediately recognize the signs of infectious disease so these animals can be isolated from the population is vital to any infectious disease prevention protocol. But it is not possible to determine which animals may be incubating disease and may start spreading viruses after admission.

Shelters should assume that all animals are naïve to infection and treat them as such. One study in Florida found 64.5% of dogs admitted to the shelter had insufficient antibody titers for CPV, CDV, or both. A similar study in cats found that most cats were seronegative for antibodies against FPV at the time of admission to an animal shelter. These studies support the current recommendation that all dogs over 4 weeks old should be vaccinated for both CDV and CPV on intake and all cats over 4 weeks old should be vaccinated with a MLV FPV vaccination on intake. In puppies, DHPP should be repeated every 2-3 weeks until 18-20 weeks of age. Adult dogs can be revaccinated 2-3 weeks after intake. For kittens, FVRCP should be repeated every 2-3 weeks until 18-20 weeks of age. Adult cats can be revaccinated 2-3 weeks after intake. For more information on intake and vaccination protocols, see the intake lecture in these proceedings.

Overcrowding increases the stress an animal experiences and increases the dose of pathogens they are exposed to in the shelter. Chronic stress from overcrowding can lower the animal’s resistance to infection by compromising the immune system.

Overburdened shelter workers may be less likely to follow sanitation and disinfection protocols properly. These are just some of the factors that illustrate how important it is to maintain the capacity for care in order to prevent infectious disease spread. Shelters need to determine their capacity for care and stay within that capacity in order to minimize disease risk and improve welfare.

Cleaning protocols need to be written, easy to understand, and accessible by all employees. Proper training should be provided to every employee and follow up should be done routinely to ensure these protocols are being followed. Disinfectants need to be parvocidal, and all contact times need to be followed exactly as written. Understanding the nature of the disinfectant is important and all recommendations need to be followed when writing protocols. Dilution procedures, how well disinfectants work in the face of organic material, and storage instructions all need to be considered when writing protocols. More information on the importance of proper sanitation and a table of disinfectant properties can be found on the Koret Shelter Medicine Program website, www.sheltermedicine.com.

Animal housing and socialization also needs to be considered. Juveniles should be in separate areas than adults, and animals showing clinical signs of disease should be isolated from the rest of the population. Double compartment housing should be used not only to reduce stress but also to allow for cleaning with minimal animal movement and handling. Dogs should not be allowed to intermingle until approved by the veterinary staff. Play groups have become a very popular addition to dog behavioral enrichment but should be reserved for vaccinated adult dogs. Socialization is extremely important for puppies but needs to be done in a manner that does not risk exposing them to disease. All volunteers and staff that enter puppy kennels for socialization periods should be trained in appropriate disease control measures. Also, cats and dogs should never be housed together in the shelter for many reasons including the risk of spreading illness between the species.

Daily medical rounds to monitor the health of all animals in the shelter will also help prevent spread of disease. At least once daily, someone trained in signs of infectious disease should observe every animal in the shelter, looking for any indication of disease or distress. This practice will allow animals displaying clinical signs of illness to be immediately placed in isolation and examined by veterinary staff, which could prevent the spread of disease to others.

Outbreak Management

Outbreak management plans should exist for each infectious disease and need to include a plan to move forward after diagnosis. The diagnosed animal needs to be immediately isolated or euthanized, the area promptly disinfected, and other animals closely monitored for signs of illness. If more cases are detected and it is determined that an outbreak is occurring, the full outbreak management plan should be immediately put into effect. The more rapid an outbreak response, the more potential exists to mitigate damage.

The first thing that is often done is to stop movement until risk can be assessed. This involves stopping intake if possible and closing adoption temporarily. For open intake shelters, strays must still be taken but owner releases can be put on a waiting list. Stray puppies or kittens should be vaccinated and fostered without entering the shelter if possible. If intake must continue, it is important to make a clean break. This will involve either a temporary facility or moving enough animals to clear and disinfect part of the shelter prior to taking more animals in. In order for it to truly be a clean break, there must not be movement of staff, supplies or animals from one area of the shelter to the other.

Risk assessment is an area of confusion for many people. The goals of risk assessment are to allow for movement of animals through the shelter and to decrease euthanasia. It was not long ago that outbreak management for these viruses was to close the shelter, depopulate, clean, and re-open the shelter. Unfortunately, that practice is still in place in many shelters across the country. Modern risk assessment allows shelters to move beyond that outdated practice.

Many factors need to be considered when determining an individual’s risk. The individual’s immune status, vaccination status, likelihood of exposure and proximity to the infected animals should all be taken into consideration as well as the cleanliness of the
environment. The shelter population can be divided into four groups: 1. Those that are infected. 2. Those that have been exposed and are at risk. 3. Those that have been exposed but are not at risk of developing infection. 4. Those not exposed. Infected animals need to be removed from the general population immediately and either isolated and treated (on-site or off-site) or euthanized. Euthanasia of otherwise healthy, young animals is difficult but if proper isolation facilities that can provide adequate care while safeguarding the rest of the population do not exist, euthanasia is the most humane option. Those known to truly not be exposed could be assumed to have the same risk as an animal that enters the facility in a non-outbreak period as long as they continue to be maintained in a manner that prevents exposure. Serology is a useful tool to determine which animals fall into the second and third groups.

In-house antibody titer testing is relatively inexpensive with high diagnostic accuracy for CDV and CPV and is thus a valuable tool for outbreak response. Two point-of-care tests currently available are the Synbiotics TiterCHEK™ and the Vaccicheck ImmunoComb™ test by Biogal. TiterCHEK™ is a non-quantitative well test kit, and the ImmunoComb™ is a semi-quantitative dot ELISA titer test kit. Much of the available research has been performed using the TiterCHEK™, likely due to the more recent release of the Vaccicheck ImmunoComb™.

One test commercially available for CPV testing (Synbiotics TiterCHEK™) was proven to be inappropriate for use in cats due to its low sensitivity for FPV antibodies. The same study found the point-of-care ELISA test available for cats only identified about half of cats with a protective titer for FPV. A later study did show better specificity, which could be due to modifications to the test, but the specificity for detecting antibody titers of 1:20 was still only 89%. Laboratory testing takes longer but is the most accurate way to measure antibody levels in cats.

Serology should be reserved for exposed animals that are not displaying clinical signs of disease. Risk categories for exposed asymptomatic animals can be assigned in the following manner:

- High Risk: Any age animal not displaying clinical signs with a negative titer.
- Intermediate Risk: Puppies and kittens less than 5 months old with no clinical signs and a positive antibody titer.
- Low Risk: Adult animals with no clinical signs and positive titer results.

Animals that are not displaying clinical signs but have a negative titer are high risk and need to be placed in quarantine for 14 days (CPV, FPV) or 4-6 weeks (CDV). Juveniles should be bathed at the beginning and end of the quarantine period. The bath at the beginning of quarantine is particularly important to avoid exposure to pathogens in the feces on their fur as maternal antibody wanes. To help avoid unnecessary long quarantine periods, antibody titer testing for CDV can be combined with PCR testing to further assess risk and potentially move more animals through the shelter, especially if the consulting veterinarian becomes involved later in the course of the outbreak. Because the incubation period for CDV varies so greatly, it is often recommended that these dogs be released with a medical waiver even if they have been quarantined for 6 weeks and have shown no clinical signs of disease.

Intermediate risk puppies and kittens can be bathed and immediately placed in rescue, foster, or go out for adoption with a waiver. It is not possible to tell if the antibody titer is due to maternal antibody or vaccination so it is important to get these animals out of the shelter as soon as possible. Low risk adults can be moved to adoption or rescue.

Decontamination is vital as animals are moved according to risk assessment. A common misconception is that kennels need to remain empty for a period of time before they can be used again. Decontamination should consist of multiple cycles of mechanical cleaning and disinfection followed by completely drying the area rather than letting it sit for a specified period of time. Three cleaning cycles with complete drying between cleanings is commonly recommended to completely remove all pathogens.

Communication is another important part of outbreak management. Recent adopters, fosters, employees, volunteers, local veterinarians and potentially the local media all need to be informed of the outbreak and given accurate information. Outbreaks of disease can lead to rumors and a loss of reputation for the shelter. Not alerting local veterinarians or other animal care professionals may lead to secondary outbreaks and further loss of life. Providing accurate and timely information can help prevent further damage and save the shelter’s reputation.

Following an outbreak, it will be necessary to review procedures and identify what caused the disease to spread. Procedures and protocols should be reviewed to ensure all measures are being taken to prevent a future outbreak. If a widespread vaccination failure is suspected, vaccination protocols should be examined. If recovering from a CDV outbreak, any wildlife handling procedures should be reviewed. Protocols need to be amended as necessary and staff training will need to be completed immediately to prevent another outbreak. As with nearly every aspect of shelter medicine, prevention is the goal of these measures.

References


Evaluating the Thin Small Ruminant
Kelly Still Brooks, DVM, MPH, DACVPM
Iowa State University
Ames, IA

The overly thin sheep or goat is a common presentation for veterinary intervention, either as the client’s chief complaint or a finding during other herd work. Poor body condition is a non-specific sign caused by a wide variety of underlying pathophysiologic processes. Definitive diagnosis and successful treatment or herd interventions will be facilitated by using a step-wise, problem-based approach.

Database: History and physical exam

Begin with a thorough history and physical examination, including verification of the client’s observations. Signalment, production status, and onset/duration of the weight loss should be taken into consideration, and the patient should be evaluated against its herd mates to identify individual vs. systemic problems. Ask the owner about the herd health program – specifically parasite control protocols, purchased animals, and history of caseous lymphadenitis (CLA), caprine arthritis-encephalitis virus (CAE) or ovine progressive pneumonia (OPP), and Johne’s. Inquire about any past health issues, including pneumonia or scouring which may have led to chronic conditions.

Fully evaluate the feeding program, including water sources. What feedstuffs are offered, how much, and how often. Have the forages been tested? Is a mineral offered, and if so, what type. Is there sufficient bunk space or feeder access for the herd? Look at pastures, feeders, hay, and waterers to identify obvious problems with overgrazing, hygiene, quality, or access. Consider the “Four Diets” - the paper ration, the feed as mixed, the feed consumed, and the actual diet digested; where do they vary? How much sorting or waste is occurring? Is the mineral being consumed, and at what rate? Are separate sources of salt or other supplements being offered?

In addition to the history, the minimum database should include a complete physical exam on a representative group of animals, including body condition scoring, and FAMACHA score. Body condition scoring systems for small ruminants typically run on a 1 (emaciated) to 5 (obese) scale and absolutely require a hands-on evaluation for accuracy. Sheep and meat goats are evaluated over their loin; goats store more fat internally than sheep, so a slight allowance may be made especially when scoring leaner breeds. A separate scoring system should be applied to dairy goats.

In addition to the routine physical examination, extra attention should be paid to the oral and pharyngeal cavities, looking for deformities (e.g. malocclusion or cleft palate), dental pathology, or other trauma.

Problem list – the damnit scheme

From the evidence collected in this database, it will be necessary to identify, localize, and develop a pathophysiologic hypothesis for the observed problems. In order to avoid prematurely focusing on any specific etiology, the DAMN-IT scheme can be used to help draft a list of possible processes causing the observed weight loss, categorized by physiologic mechanism. The acronym reminds us to consider:

- D – degenerative, developmental
- A – autoimmune
- M – metabolic, mechanical, mental
- N – nutritional, neoplastic
- I – inflammatory, infectious, ischemic, immune, inherited, iatrogenic, idiopathic
- T – traumatic, toxic

Weight loss may be an expected reaction to the current production stage (breeding male or lactating doe). In the context of emaciation, major mechanisms across all age groups include nutritional, mechanical, and infectious etiologies. Less commonly, cachexia may present secondary to developmental abnormalities, neoplasia, or other chronic inflammatory condition of organ failure.

<table>
<thead>
<tr>
<th>Degenerative</th>
<th>Neonate</th>
<th>Youngstock</th>
<th>Adults and Aged</th>
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<tr>
<td>Developmental (Congenital)</td>
<td>Cleft palate</td>
<td>Malocclusion</td>
<td>Malocclusion</td>
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<tr>
<td></td>
<td>Congenital Heart Defect</td>
<td>Diaphragmatic Hernia</td>
<td>Diaphragmatic Hernia</td>
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<tr>
<td>Autoimmune</td>
<td>Goiter</td>
<td>H-P-T Axis disorder</td>
<td>Stage of Production</td>
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<tr>
<td>Metabolic</td>
<td>Pica</td>
<td>Obstruction</td>
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<tr>
<td></td>
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<td>Incisor loss / Oro-esophageal</td>
</tr>
</tbody>
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### Initial plan and follow-up

**Diagnostic**

At this point, the signalment, history, physical exam, and oral exam findings should be sufficient to support an initial plan for diagnostic, treatment, and client education. Diagnostic modalities can be used to verify the presumptive diagnosis, further localize the disease process, elucidate the pathophysiologic mechanism, and rule-in/out differentials. Depending on the case, clinical pathology including quantitative fecal floatation, complete blood count (including fibrinogen), and/or serum biochemistry (including GGT) may be indicated.

In addition to gastrointestinal parasites, CLA, CAE/OPP, and Johne’s are the big three infectious diseases that commonly present as chronic weight loss in the face of a good appetite. Serology (ELISA or antigen specific AGID) is very useful for diagnosis of CAE and OPP outside of the periparturient period; though there is a significant degree of cross-reaction between the lentiviruses, OPP-based AGID test are less sensitive when used to test for CAE. Serologic testing (SHI) for CLA is typically recommended only as a herd screening tool as it is difficult to interpret at the individual level. Cross-reactions, past infections, and vaccination can all produce titers in individuals without active disease, and occasionally low titers will be observed in animals with active, culture positive abscesses. However, caprine titers \( \geq 1:512 \) are highly (~95%) correlated with internal abscesses and should have a higher positive predictive value in a high-risk population. Johne’s diagnostics, including both serology and fecal PCR, are similarly challenged by low sensitivity, especially early in the infectious course. However, appreciable weight loss is a sign of terminal clinical disease, and either method should perform reliably at this time point.

Imaging, especially abdominal and thoracic ultrasound, can be done to evaluate rumen and GI motility, rule-out pregnancy or advanced uterine tumors, and screen for enlarged mesenteric lymph nodes, and look for evidence of chronic pulmonary consolidation; a more skilled ultrasonographer could also evaluate liver, kidney, and heart for abscesses, parenchymal abnormalities, or valvular insufficiency. Thoracic radiographs may be a better option for assessing pulmonary changes and identifying mediastinal masses. Many conditions, including internal CLA abscesses and tumors, may not be diagnosed until necropsy; scrapie testing should be considered for any remaining open diagnosis of chronic wasting.

**Therapeutic and client education**

Based on the initial database and any timely diagnostics, the initial treatment plan should specifically address the suspected disease process as well as address any additional symptomatic or supportive care requirements. If gross nutritional inadequacies are suspected (or effective access to feedstuffs is limited by overcrowding or lameness), the animal can be challenged with a higher energy or protein diet offered more frequently or with protected access to decrease competition from herd mates. In uncomplicated cases, condition should noticeably improve within two weeks, ideally confirmed by evidence of actual weight gain. Species-appropriate loose mineral should be available ad-lib, other sources of salt (e.g. salt blocks or loose salt) will compete with the mineral mix for consumption. Geriatric patients will benefit from a highly-palatable, easily digestible diet that may need to be chopped or soaked for easier mastication (e.g. complete ration pellets, alfalfa pellets, beet pulp, or Chaffehay). If neonates are failing to thrive, inspect the...
dam’s udder for teat and gland function, mastitis, or trauma. As a rule of thumb, the dam can be expected to raise one lamb/kid per functional half. Although there are many systems that can be made to work for raising orphans, when bottle babies are struggling recommend that the client feed species-appropriate milk or milk replacer at 15-20% of their body weight daily (3 ounces per pound) divided into 3-4 feedings and weigh the kids or lambs daily for a week to assess response.

If parasitemia is present, it is important to investigate the herd deworming protocol and any history of individual deworming in the patient in order to select a suitable drug. Anthelmintic resistance is common, especially in *Haemonchus contortus* populations, and the herd dewormer may not be effective. Fecal egg counts conducted at the time of presentation/deworming and then again 10-14 days later can help identify anthelmintic failures. For animals affected by *Haemonchus*, with a correspondingly low FAMACHA score, the FAMACHA score should improve by one grade within two weeks after deworming. Chronically parasitized animals may benefit from parenteral or oral iron supplementation.

There are no effective treatment options for CAE/OPP, CLA, or Johne’s disease. Chronic pneumonia or chronic coccidiosis cases are also unlikely to respond well to therapy but early intervention or control programs may be indicated for other herd members. Oral or limb pathology should be addressed as appropriate, with a plan for nutritional support during the treatment and recovery period. Rumenotomy is indicated for obstructive disorders in the adult; reported cases of starvation-induced pica in neonates have not responded well to any intervention.

Additional resources

Dewormer Chart for Goats. [http://media.wix.com/urld/aded98_c7a6cc3b624043aeeae8693f9f13c71.pdf](http://media.wix.com/urld/aded98_c7a6cc3b624043aeeae8693f9f13c71.pdf)

Dewormer Chart for Sheep. [http://media.wix.com/urld/aded98_e173a9632a742aa8241ea5d1f3694a2.pdf](http://media.wix.com/urld/aded98_e173a9632a742aa8241ea5d1f3694a2.pdf)

Henning, E. Dairy Goat Body Condition Scoring Video. [www.youtube.com/watch?v=FC0a1f06v5Y](http://www.youtube.com/watch?v=FC0a1f06v5Y)

Langston University. Body Condition Scores in Goats. [www2.luresext.edu/goats/research/BCS_factsheet.pdf](http://www2.luresext.edu/goats/research/BCS_factsheet.pdf)

On-Line FAMACHA Training, University of Rhode Island Northeast Small Ruminant Parasite Control. [http://web.uri.edu/sheepgoat/famacha/](http://web.uri.edu/sheepgoat/famacha/)

Oregon State University Extension. Body Condition Scoring of Sheep. [http://ir.library.oregonstate.edu/xmlui/bitstream/handle/1957/14303/ec1433.pdf](http://ir.library.oregonstate.edu/xmlui/bitstream/handle/1957/14303/ec1433.pdf)

Intestinal coccidia and strongyles, specifically *Haemonchus contortus* (barber-pole worm), are the two major internal parasites of concern on most domestic small ruminant operations. Clients are often confused about these parasites, deworming protocols, and parasite control programs in general. With the threat of anthelmintic resistance, there is no one-size fits all solution. Instead, tailored solutions must be developed that take into account the production goals and sub-populations, historical performance records, exposure risk factors, epidemiology of the organism of interest, and existing drug resistance patterns. The American Consortium for Small Ruminant Parasite Control website contains an extensive listing of current best practices and timely information on the topic (www.wormx.info). A “Top Ten” client education summary specific to *Haemonchus* control is included at the end of this proceedings.

**Coccidiosis**

Coccidiosis is the most common cause of debilitating diarrhea in young ruminants between three weeks and five months of age, with effects ranging from sub-clinical unthriftiness and reduced gains to peracute death. After about five months of age, acquired immune resistance limits the replication and clinical impact of the of the parasite; thus the producer’s major challenge lays in preempting the significant youngstock morbidity and mortality associated with coccidiosis without interfering with the natural acquisition of immunity.

Coccidia are host-species specific, though some species do cross-infest between sheep and goats. Oocysts are regularly shed in the feces at low levels by healthy adult ruminants and at much higher levels by sub-clinically and clinically affected youngstock. Transmission to youngstock is facilitated by high-density housing and manure contamination of feed and water sources; kids and lambs born at the end of a kidding season may be exposed to a very high infectious dose in the first weeks of life. Acute clinical coccidiosis is most common around periods of stress, such as weaning. Stressed or immunocompromised adults are also susceptible to clinical breaks.

In addition to fluid therapy and other ancillary supportive care, treatment options for acute coccidiosis include oral sulfonamides or thiamine agonists such as amprolium. These drugs are coccidiostats which limit replication but do not eliminate the organism. They are primarily effective early in the organism’s reproductive cycle and may appear more effective when used prophylactically for control than in the face of acute disease. Amprolium has been used extra-label in goats at a dose as high as 50 mg/kg, but patients should be monitored closely for signs of polioencephalomalacia. Several triazine coccidiocidal drugs (e.g. diclazuril and toltrazuril) are available overseas for small ruminants but not domestically. Ponazuril (Marquis®) is a related drug which is FDA-approved for treatment of Equine Protozoal Myeloencephalitis in horses and has been used extra-label for treatment of acute coccidiosis in small ruminants (~10 mg/kg orally as a single dose, FARAD recommends a prolonged slaughter withdrawal period of approximately four months).

Control and prevention strategies focus on both reducing avenues for fecal-oral transmission and reducing the number of total oocysts in the environment. Reduce overcrowding, increase cleaning of pens and equipment, and construct feeders that kids cannot climb into. Oocysts are very persistent in the environment and resistant to most disinfectants so it is difficult to reset the clock once a farm has been highly infected. Whitewash, paint, and impervious materials can be used to reduce build-up of oocysts in the facility environment. In herds that struggle with coccidiosis in the youngstock, does and ewes may be started on a coccidiostat in the feed or water from one-month prior to the start of kidding/lambing through weaning and a medicated starter feed should be offered to the youngstock; in the U.S., decoquinate is labeled for both sheep and goats, lasalocid is labeled for sheep, and monensin is labeled for goats. Youngstock may also be treated with amprolium on a 21-day prophylactic cycle.

**Nematodes**

In most of the continental U.S., *Haemonchus contortus* is the most significant nematode parasite of small ruminants. As a rule of thumb – if you can grow corn, you can grow Haemonchus; this includes irrigated pastures in otherwise arid climates. Other clinical-relevant small ruminant nematodes include *Teladorsagia* (formerly *Ostertagia*), *Trichostrongylus*, *Cooperia*, *Oesphagostomum*, *Nematodirus*, and *Trichuris*. Clinical signs of strongyles infestation include varying degrees of anemia, diarrhea, dependent edema (e.g. bottle jaw), production losses, and death. *Haemonchus* is specifically associated with profound anemia but not typically with diarrhea. For this reason, the FAMACHA© test can be applied in locations where *Haemonchus* predominates, while the Five Point Check© system may be more appropriate on operations where other strongyles prevail. FAMACHA© training materials can be found on-line for free through the University of Rhode Island Northeast Small Ruminant Parasite Control website and the citation for the Five Point Check© is included with the references. These nematodes are transmitted in a fecal-oral pattern and larval development and transmission requires access to forage; for the most part, strongyle transmission is interrupted in true dry-lot or indoor environments.
However, larval hypobiosis is common during the winter in more northern locations when pasture conditions are not favorable for larval survival in the environment.

In conjunction with clinical signs, quantitative fecal egg counts (FEC) are useful for estimating the total strongyle parasite load. The different species of strongyle eggs cannot be differentiated on a routine fecal; if needed, speciation can be performed via larviculture, peanut lectin staining, or PCR. Fecal egg counts commonly rise during the periparturient period. Fecal egg count reduction tests (FECRTS), which consist of serial individual FECs taken 10-14 days apart can be used on a herd level to evaluate dewormer efficacy; <95% reduction indicates some degree of resistance is present and ~50% reduction or less is commonly associated with perceived lack of clinical efficacy. The downside to the FECRT is that only one dewormer class can be evaluated per cycle, it takes approximately two weeks to complete a cycle, and serial testing of recommended numbers of animals can become expensive over time. If clients are reluctant to pursue FECRTs, have them record FAMACHA© scores at deworming and two weeks later – scores should improve by at least one grade if the dewormer is having any effect. If no improvement is noted, that strongly suggests that there is a significant degree of resistance to the dewormer and further diagnostics are needed. The DrenchRite® assay offers another alternative to serial FECRTs. This test hatches out larvae from a fresh comingled fecal sample and exposes the larvae to multiple concentrations of the major classes of dewormers (benzimidazoles, levamisole, avermectins and indirectly moxidectin). While the initial cost of this test is high, it may be quicker and less expensive in the long run. The test should be run when FECs are reasonably high and pre-coordination with the lab (Dr. Ray Kaplan, University of Georgia, (706) 542-0742) is necessary to ensure the correct sample is submitted.

Anthelmintic resistance is a major problem in small ruminant herds. Current deworming protocols should utilized a targeted selective treatment approach (deworm only clinically affected animals identified by FAMACHA, 5 Point Check, or FEC), or a strategic deworming approach based on the lifecycle and epidemiology of the parasite of concern (either presumptive or identified via larviculture or PCR). These approaches are designed to minimize production losses while preserving a refugia of non-resistant parasites, decreasing anthelmintic resistance selection pressures. It is especially important to minimize progression of resistance in the breeding and replacement herd, in market animals that are being reared with the permanent herd, and in any animals that will be utilizing pasture used for the permanent herd. In heavily parasitized herds, or those with multi-drug resistant parasites, the herd may need to be subdivided and a tailored approach developed for each class of animal (e.g. market stock vs. replacement stock vs. mature breeding stock). Producers that keep good records will ultimately have a better basis for developing an educate parasite control protocol base on knowing the historical patterns of clinical parasitemia, evidence for (or against) specific drug efficacy, and identifying chronic offenders for culling. Since parasite resilience is a heritable trait, culling chronic offenders can improve overall herd resilience. Remind producers that when they purchase replacements, they purchase both the parasites, and their anthelmintic resistance patterns. As a general guidelines for using anthelmintic drugs in small ruminants: DO NOT routinely rotate dewormer classes, DO use oral routes of administration, and DO dose appropriate for weight – increasing the dose for goats by 1.5-2x the label sheep dose depending on the product used. Anthelmintic dose charts that includes extended withdrawal information are available online for goats, sheep, and camels; the links are included below in the references.

Several non-chemical adjunct control methods may be considered for nematode parasite control. First, evaluate the herd’s nutritional program. We commonly assume that parasites caused the unthrifty animals, but stock reared on a poor plane of nutrition will also be less resilient to strongyle infestation, especially when protein is inadequate. Unfortunately, pasture rotation alone is ineffective for parasite control, but inter-species rotation with non-susceptible species (e.g. cattle or horses) or haying stocked forage can decrease existing parasite loads on pasture. FECs can be reduced by feeding condensed tannin forages (e.g. sericea lespedeza) either via grazing or as supplemented hay or pellet, but these feedstuffs must be incorporated as a significant percentage of the diet. Copper oxide wire particles have also received attention for parasite control in goats (not sheep due to high risk for copper toxicity); although the COWP boluses are safer than copper sulfate drenches, the herds liver copper status should be determined before initiating treatment and then regularly monitored. COWP boluses are increasingly being frequently administered at high levels by dairy goat owners, independent of their veterinarian, increasing the risk for copper toxicosis.

Tapeworms

*Moniezia* spp. are the most common tapeworm of sheep and goats in the U.S. Since the segments are easy to see when passed in the feces, they are often a concern to owners. However, they are rarely pathologic and treatment is not typically indicated. One exception may be high levels of infestation in youngstock, where they can be associated with altered gut motility and decreased intakes. Tapeworms are not treated by the common macrocyclic lactone anthelmintics; praziquantel can be used extra-label in sheep and goats.

Further reading
Ten to remember: Small ruminant haemonchus control

1. **Resilience.** Instead of eliminating all gastro-intestinal parasites, our main deworming goal is to keep animals healthy and productive despite a moderate parasite burden. While high numbers of parasites can make any animal ill, certain production groups (youngstock and lactating animals) and highly stressed sick individuals are more susceptible to clinical parasitemia. Good overall nutrition and health is an important part of the parasite control program.

2. **70-30.** 70% of your farm’s parasite problem comes from roughly 30% of the animals. In addition to the high-risk groups, this 30% also includes individuals who just can’t handle a moderate parasite load as well as the rest of the flock/herd. These individuals are the biggest contributors to parasite eggs on pasture; culling them can improve overall herd resilience over time, decreasing the need for deworming.

3. **Resistance.** Small ruminants - especially goats - are REALLY good at creating dewormer-resistant parasites. Find a dewormer that works and stick with it until it doesn’t; do not rotate between drugs or you will hasten development of resistance to all the drugs used. If you think your dewormer may no longer work, or if you are unsure what dewormer will be effective in your animals, we can assess the dewormer efficacy in your herd through a Fecal Egg Count Reduction Test or a DrenchRite® Test.

4. **Refugia.** Ideally, the number of eggs on pasture from non-resistant parasites greatly outnumbers those from resistant parasites – this is the refugia. Since our biggest concern in the mid-west is *Haemonchus contortus*, the barberpole worm, we can use a targeted selective treatment protocol (FAMACHA) to identify only clinically ill animals for deworming, allowing the remainder of the flock to pass low levels of eggs that have not been selected for resistance.

5. **Oral Drenching.** Give all dewormers orally with a drench gun; do not use injectable or topical dewormers. Ideally, hold the animal off feed for 24 hours prior to deworming.

6. **Adequate Dose.** Use the correct dose and do not underestimate the animal’s weight. Sheep should be dewormed according the label instructions; goats are usually dewormed at twice the sheep or cattle dose. One key exception is with Levamisole, which can be toxic at higher levels.

7. **Emergency Reserve.** It seems like we always find out that our dewormers no longer work at the worst time when animals are critically ill. Plan ahead to have an “emergency only” drug that will work in a pinch – we recommend that you AVOID routinely using Meloxicam (Cydectin) so that this drug will still be effective for use in an emergency, buying time to find a long-term solution.

8. **Package Deal.** When you buy in a new animal, you are also purchasing that animal’s parasites, and any dewormer resistance in those parasites. Eggs from those resistant parasites will infest your pasture and the rest of your herd or flock, accelerating your drug resistance problem.

9. **Dry Lot.** *Haemonchus* needs grass to complete its life cycle. A true dry lot, without any grass or weeds, can be used to help break the parasite cycle or as a quarantine area for new animals.

**Pasture rotation**

While pasture rotation is great for maximizing forages, a typical two- to four-week cycle is actually perfect for growing gastrointestinal parasites. Pastures would need to rest for many months in order to significantly break the parasite cycle. Better options include alternate species grazing such as with cattle or horses (note: sheep, goats, and camelids do share parasites), or haying pasture between grazing cycles.
Managing Meningeal Worms
Kelly Still Brooks, DVM, MPH, DACVPM
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Although aberrant migration of the deer meningeal worm, *Parelaphostrongylus tenuis* is a commonly recognized cause of neurologic disease in South American camelids, *P. tenuis* is less frequently considered as a differential for spinal neuropathies in goats and sheep. In enzootic regions of the U.S. (Atlantic coast west to the Dakotas, Nebraska, Oklahoma, and Texas), meningeal worm should considered as a cause of asymmetric multi-focal neurologic disease in small ruminants as well as camelids, as well as for cases of scoliosis, “downers”, and suspected brain lesions in camels. In addition to classic camelid cases, we have observed an uptick in cases of meningeal worm among Midwestern goat herds. It appears that 2015’s unseasonably cool and wet summer has contributed to both greater and earlier exposure to the parasite, especially for goats grazing woodland habitat. These cases involve a range of ages from older pre-weaned kids (4-6 months) to mature animals. Both sporadic cases and herd outbreaks involving as much as 15% of the herd have been observed.

**Epidemiology**

*Parelaphostrongylus tenuis* normally follows an indirect neurotropic lifecycle that passes between the white-tailed deer definitive host and a variety of intermediate host terrestrial gastropods. L1 larvae from deer feces penetrate the foot pad of a snail or slug, where they mature to the infective L3 stage. These L3 emerge from the footpad into the mucous trail environment, where they can survived desiccation and cold stress. After either the gastropod or emerged L3 larvae are ingested by a white-tailed deer, the L3 will migrate through the intestines and peritoneum to the dorsal horn of the spinal cord, and then mature during migration through the subdural space to the cranium. As adults, they enter the dural venous sinuses and deposit eggs on the meninges. Both eggs and embryonated L1 larvae travel via the circulatory system from the venous sinuses to the lungs, are coughed up and swallowed, and expelled in the feces. This life-cycle typically takes four months to complete and little to no clinical disease is observed in infected deer. Camelids, goats and other accidental hosts become infected by ingesting infective L3 larvae present either in the snail or slug, or in the slime trail. Once ingested, normal larval maturation is disrupted, resulting in aberrant migration through the spinal cord and CNS. No eggs are produced, but the neural inflammation and parenchymal damage from aberrant migration causes clinical central nervous system disease with variable signs depending on the anatomic location of the lesions. Clinical disease is most common in the late fall and early winter, roughly forty-five to sixty days after the peak exposure period in the early fall. However, if moderate summer temperature reduce the gastropod’s normal aestivation period, clinical cases may start appearing much earlier in the fall. Exposure to white-tailed deer or common pasture/browse is a common finding in the case history.

**Clinical signs**

Common clinical findings include upper- and lower-motor neuron, and peripheral limb sensory deficits. Cases often present with progressive neurologic deficits that start as hind-end weakness and ataxia which may lead to complete paresis and death. Clients may perceive that the animal is lame, stiff, weak, base-wide, ataxic, slow or reluctant to move, or unable to rise. In the absence of primary brain lesions, most affected animals are initially bright and alert with a good appetite and no fever. Depending on the anatomic locations of the lesions, the physical exam may find evidence of ataxia, hemi- or tetra-paresis, impaired proprioception, decreased nociception, altered reflexes, or scoliosis; advanced cases may be tetraplegic. Acute-onset cranial nerve, vestibular, and brain signs may be identified in atypical camelid cases. In goats, the most consistent findings include asymmetric multi-focal neurologic disease with emphasis on pelvic limb deficits. The clinical signs often progress over the course of several weeks, terminating in death or euthanasia although presumptive sporadic recoveries have been reported. In addition to the neurologic signs, peritonitis and colic symptoms have been reported in goats after high-dose experimental infections and linear pruritic dermatitis lesions have been observed in natural infections.

**Differentials**

Major differentials for typical clinical presentations of *P. tenuis* include all other causes of spinal meningomyelitis. Antemortem differentiation of meningeal worm from other inflammatory and degenerative etiologies may be possible through CSF analysis. An increased CSF white cell to protein ratio is commonly observed with meningeal worm. When present, an eosinophilic pleocytosis is highly specific for cerebrospinal nematodiasis and can be used to rule-out other etiologies. Copper deficiency degenerative myelopathy (enzootic ataxia, swayback) is the most common cause of caprine spinal cord lesions and should be a top differential for hind-end weakness and ataxia occurring in multiple neonatal to weaning-age kids, especially those raised on a sheep concentrate/mineral or high molybdenum-diet. Low liver copper levels are diagnostic for enzootic ataxia. Additional sporadic causes of degenerative myelopathy include compressive lesions from vertebral malformations, trauma, abscess, or neoplasia. Those cases typically present with a more focal anatomic localization than usually observed with *P. tenuis*. Common differentials for inflammatory conditions to consider are those causing a systemic inflammatory response syndrome (SIRS) such as peritonitis, colic, and septicemia. Although meningeal worm can have a highly specific CSF eosinophilia, the presence of other inflammatory conditions may lead to a mixed leukogram. In these cases, the presence of increased white blood cells and protein in the CSF, specifically if greater than 100 white blood cells/µL, is diagnostic for inflammatory disease. Antemortem differentiation of meningeal worm from other inflammatory etiologies may be possible through CSF analysis. An increased CSF white cell to protein ratio is commonly observed with meningeal worm. When present, an eosinophilic pleocytosis is highly specific for cerebrospinal nematodiasis and can be used to rule-out other etiologies. Copper deficiency degenerative myelopathy (enzootic ataxia, swayback) is the most common cause of caprine spinal cord lesions and should be a top differential for hind-end weakness and ataxia occurring in multiple neonatal to weaning-age kids, especially those raised on a sheep concentrate/mineral or high molybdenum-diet. Low liver copper levels are diagnostic for enzootic ataxia. Additional sporadic causes of degenerative myelopathy include compressive lesions from vertebral malformations, trauma, abscess, or neoplasia. Those cases typically present with a more focal anatomic localization than usually observed with *P. tenuis*. Common differentials for inflammatory conditions to consider are those causing a systemic inflammatory response syndrome (SIRS) such as peritonitis, colic, and septicemia. Although meningeal worm can have a highly specific CSF eosinophilia, the presence of other inflammatory conditions may lead to a mixed leukogram. In these cases, the presence of increased white blood cells and protein in the CSF, specifically if greater than 100 white blood cells/µL, is diagnostic for inflammatory disease.
spinal neuropathy include lentiviral infection (caprine arthritis-encephalitis virus) as well as any other less-likely viral cause of nonsuppurative meningoencephalomyelitis, such as rabies, pseudorabies, border disease, and West Nile virus. Unfortunately, a non-essential phospholipid CSF monocyteosis has been reported in some caprine and camelid meningeal worm cases, which complicates exclusion of a viral meningeal infection and listeriosis. Other reported causes of spinal cord lesions include congenital anomalies and neoplasia.

Additional differentials may be considered with extremely mild or severe presentations; in all cases, eosinophilia in the CSF strongly supports a meningeal worm diagnosis. Advanced tetraparetic and terminal cases of meningeal worm may resemble common cases of “downer” animals, including polioencephalomalacia, listeriosis, pregnancy toxemia, and hypocalcemia. However, the hallmark clinical signs of listeriosis and polioencephalomalacia, (unilateral cranial nerve deficits and bilateral menace deficits concurrent with intact pupillary light reflexes, respectively) are not expected findings with cerebrospinal nematodiasis. Pregnancy toxemia and hypocalcemia can be readily diagnosed through serum β-Hydroxybutyric acid and ionized calcium levels. Less common differentials for downer presentations include brainstem abscess, bacterial or viral meningoencephalitis, tick paralysis, organophosphate toxicity, and lidocaine toxicity. In contrast, mild presentations of meningeal worm may mimic the transient flaccidity or stiff, spastic movement and hyperreflexia observed in the early stages of tetanus, but would not include the third-eyed eyelid spasm and tonic seizures classically associated with tetanus infection. Laminitis, polyradiculoneuritis, myotonia congenita, and nutritional muscular dystrophy (vitamin E and selenium deficiency) may also cause similar abnormalities in gait and posture. Finally, rhabdomyolysis should always be considered with any open neurologic case and small ruminants should be submitted for scrapie testing. In particular, the pruritic skin lesions reported in association with meningeal worm in goats are also characteristic of scrapie. Both of these diseases require postmortem testing for definitive diagnosis.

Diagnostic plan
Definitive diagnosis of Parelaphostrongylus tenuis is complicated both by the lack of antemortem tests as well as difficulty in locating the parasite on necropsy. Field diagnosis is based on exposure history, clinical presentation, exclusion of other differentials, cerebrospinal fluid analysis, and response to treatment. At necropsy, gross lesions are uncommon and the nematodes are rarely found on routine examination. Somewhat more frequently, secondary parenchymal changes typical of neural larval migrans may be observed. These relatively characteristic necrotic and eosinophilic inflammatory lesions may include axonal degradation, eosinophilic encephalitis, and hemosiderin-laden macrophages, and gitter cells; occasionally nematode cross-sections are observed in the sections. A nested PCR technique has been applied to formalin-fixed, paraffin-embedded tissues with some success even when the parasite is not observed in the tissue section. Although research efforts are focused on improving antigenic targets for immunodiagnostics and prophylaxis in cameldids, there are no commercially available serologic tests or vaccines.

Treatment protocols
Meningeal worm treatment protocols include extra-label high-dose, extended course fenbendazole, anti-inflammatorics, and supportive care along with single dose ivermectin for control of immature larvae. Although avermectin anthelmintic are used in prophylactic control programs, they are actively effluxed from the CNS and are considered ineffective for clinical cases. Typical treatment protocols call for five days of high-dose oral fenbendazole (50 mg/kg) along with a single dose of ivermectin to kill any intestinal and peritoneal L3.

In addition to anthelmintic therapy, anti-inflammatory drugs are indicated to address the existing CNS inflammation from the larval migration and modulate the secondary immune reaction to the dying parasites. Both glucocorticoid steroids and non-steroidal anti-inflammatorics have both been used at species-appropriate doses. Glucocorticoids, such as dexamethasone (0.1 mg/kg IM q24 hours for 5 days), are widely used against CNS inflammation. They decrease tissue inflammation and edema, improve vascular permeability, and facilitate neuronal excitability and impulse conduction, and reduce inflammatory chemokine driven neuropathology. Nonsteroidal anti-inflammatory drugs (NSAIDS), most commonly flunixin meglumine (1.1-2.2 mg/kg IV q24 hours for 3 days), non-selectively inhibit cyclooxygenase-mediated inflammatory pathways in the CNS as well as in peripheral tissues. Dimethyl sulfoxide (DMSO, 1 g/kg IV) may be administered to protect against continued oxidative injury to the cellular architecture by free radicals produced by phagocytic leukocytes (including eosinophils) during the inflammatory process.

Adjunct therapies include supplementation of key vitamins, fluid and electrolyte support, and attention to key husbandry practices. Vitamin E (α-tocopherol), like DMSO, is a non-specific antioxidant that is also critical to neurocerebral health. Vitamin E deficiency is a primary cause of neurologic and skeletal muscle degeneration; supplementation would counter the effect of reduced intake from anorexic animals as well as provide additional neuroprotective anti-oxidant effects. B-complex vitamins, including thiamine (B1) and cobalamin (B12), are vital to metabolic pathways necessary for normal brain and nervous system function. They are normally adequately synthesized by the rumen flora in healthy animals; however, parenteral B-complex supplementation is advisable in the face of decreased appetite or rumen function. Fluid therapy protocols depend on the animal’s requirement, desired route, and fluid types available. Dehydrated, anorexic, and/or severely depressed patients require intravenous fluid therapy for resuscitation and base maintenance requirements (50 mg/kg/day, non-lactating animals). Continuous administration of isotonic non-alkalinizing solutions are
frequently utilized in the hospital setting for this purpose; the fluid base may include 0.9% sodium chloride, acetated Ringer’s solution, or another balanced isotonic electrolyte solution supplemented with potassium chloride (20-40 mEq/L) and/or calcium gluconate (25-50 mL/L) as clinically indicated. Although intravenous fluid therapy can be managed on-farm in the right settings, if protracted, these cases will eventually require supplementation with partial parenteral nutrition, which absolutely requires a higher level of case management in a hospital environment. For more functional patients, intravenous hypertonic saline and/or oral fluid and electrolyte solutions are more practical and less expensive methods of correcting fluid deficits and minor electrolyte imbalances in the field. Hypertonic saline (4-5 ml/kg) is particularly effective at rapidly expanding plasma volume at the expense of the extravascular compartment, but must be accompanied by additional oral or isotonic intravenous fluids. Non-alkalinizing electrolyte solutions are typically indicated for adult ruminants. An appropriate oral electrolyte solution based on sodium chloride, potassium chloride, and calcium chloride salts will provide relatively high levels of chlorine and potassium. Alkalinizes such as bicarbonate, acetate, and propionate, which are commonly included in calf electrolyte solutions, are contra-indicated. Attention to primary husbandry considerations include shelter from sun and inclement weather, and functional access to feed and water. Deep bedding, intentional position changes, and physiotherapy that includes passive range of motion exercises are indicated to limit secondary pressure myopathy, compartment syndrome, formation of decubital lesions, and tendon contraction.

**Prognosis**

Treatment prognosis is dependent on severity of clinical signs at presentation. Ambulatory animals generally respond to treatment but may retain residual neurologic deficits; prognosis for downer animals is poor. Ambulatory cases typically have a fair prognosis for survival but may not recover all neurologic deficits; when a modified neurologic scoring system is applied (0-5, grade zero indicating no gait deficits, grade five are recumbent), clinical experience indicates that a one-score improvement after treatment is a reasonable expectation for responding animals. Continued improvement more than six months after treatment is not likely. Sporadic recovery has been reported in multiple species.

**Prevention and control**

Meningeal worm prevention strategies can be group into chemoprophylaxis and management-centric options. Routine administration of avermectin anthelmintic compounds is effective against early larval stages before they migrate into the CNS; unfortunately, this approach facilitates anthelmintic resistance in common gastro-intestinal nematodes such as *Haemonchus contortus*. Chemoprophylaxis is often administered year-round, but it can be targeted seasonally in some climates; for instance, in locations that experience significant hard freezes and hot, dry summers, 85% of meningeal worm exposure occurs from September to December. Management strategies include efforts to separate livestock from the source. Most producers elect to limit livestock access to wet and marshy areas or other high-risk habitat. In highly endemic areas, deer fencing, vegetative barriers, and molluscicides have been employed, but it is not clear if these methods are effective or should be recommended.

**Additional reading**


Questions on Q Fever
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Chances are, when Q-fever comes up as part of your professional practice, it is because of an abortion work-up, a trace-back from human illness, or questions from a client on obtaining negative testing for an animal or herd. It has gained new attention due to recent outbreaks of disease in people where it has been implicated in both acute flu-like illness and more rarely in serious chronic disease. As a “new” old disease, advancements in the areas of Coxiella epidemiology and diagnosis unfortunately have not drastically improved our ability to manage the disease due to inherent limitations in the diagnostic testing modalities, limited control options, and zoonotic potential. Small ruminant veterinarians need to be prepared to answer client questions about exposure risks, clinical disease, control and treatment options, and personal safety.

Organism, epidemiology, and zoonotic risk
Q-fever, which is known in animals as coxiellosis, is caused by the gram-negative intracellular bacteria Coxiella burnetii. This agent is ubiquitous in the environment, where it can in a spore-like form for an extended period of time and become airborne, moving a mile or more on wind currents. It is primarily spread through aerosol transmission or direct contact with infected animals or exposure to infective materials, which include abortion or birth products, vaginal discharge, milk, feces, urine, and semen from infected animals.

Clinical disease (abortion, stillbirths, and weak neonates) most commonly occurs in domestic ruminants (sheep and goats, to a lesser extent cattle), but other vectors, including cats, birds, and ticks, have been implicated in human exposure; prevalence as high as 40% has been reported in feral cat populations. Most livestock infections are asymptomatic; in fact, over 75% of domestic cattle dairies are positive for C. burnetii based on bulk tank sampling, though reports of clinical disease are much lower. Unfortunately, shedding patterns are highly variable, both between individual animals and across species, though all species may shed in the absence of clinical signs and for several weeks following parturition or abortion. The highest concentrations of C. burnetii are typically found in ovine abortion products and sheep will continue to shed in the vaginal mucous and feces; however, shedding in milk is more intermittent in sheep compared to the other species; placenta or vaginal fluid rather than milk are the best samples for screening tests. In goats, C. burnetii has also been identified in vaginal secretions from nulliparous and clinically normal goats, though higher numbers of organism are shed after coxiellosis abortion. Goats will persistently shed in the milk for several weeks following parturition or abortion; bulk tank milk PCR is an excellent sample for herd screening, especially in early lactation. Infected cows are least likely of the species to abort and shedding in vaginal fluids is relatively short-term. However, even clinically healthy cows may persistently shed the bacteria in their milk through early to mid-lactation; bulk tank milk is an excellent sample for herd screening. Coxiellosis is a reportable disease in most states.

Historically, Q-fever has been treated as an occupational disease associated with livestock exposure; high-risk occupations include livestock farming, food animal veterinary medicine, slaughterhouse employees, and animal research laboratory employees. The infective dose for Q-fever is very low. Q-fever is a nationally notifiable disease, diagnosis typically triggers an investigation into potential animal contacts, and then testing of those contacts. However in recent years (2000-2010), more than half of the reported cases of Q-fever in the U.S. did not have any reported contact with livestock. Roughly half of human Q-fever cases result in asymptomatic seroconversion two to three weeks following exposure; approximately 3% of the U.S. population is seropositive. Of those with clinical disease, most experience an acute “flu-like” illness for one to two weeks, characterized by a high (104-105°F) fever, malaise, gastrointestinal signs, and coughing, which may progress to pneumonia; roughly one in four people may experience a post Q-fever fatigue syndrome following recovery from the acute disease. Rarely, chronic Q-fever or other serious complications such as hepatitis, endocarditis, or meningoencephalitis can occur, especially in individuals with heart valve replacement or other underlying risk factors. Pregnant women are generally considered to be at risk for miscarriage or pre-term delivery; although data from the recent Netherlands outbreak did not find evidence for increased obstetrical morbidity, it may depend on the bacterial strain involved.

Diagnostic testing
In all species, diagnostic testing is complicated by inconsistency between clinical infection, serologic status, and organism shedding. It is possible for infected animals to be both seronegative and/or not shedding at the time of sample collection. Titers may persist years past infection; conversely, a significant proportion (10-20%) of animas in infected herds may remain seronegative despite actively shedding the bacteria. For these reasons, serology is best utilized as a herd-level test only; neither negative serology nor negative PCR rule out infection in the individual animal but repeated negative herd tests reduce the likelihood that that the farm is infected. Herd screening tests should be biased towards the highest-risk animals – those that recently kidded, lambed, or aborted; do not include youngstock.

Serologic testing options include complement fixation, ELISA, and indirect IFAs. While complement fixation is used for regulatory export testing, the other tests are more sensitive and should be used for general herd- or flock-level diagnostic testing. Serology can be performed on bulk tank milk samples as a herd-level screening tool; a positive test indicates that herd members have
been exposed and seroconverted. As with most serologic testing, there is a time lag between infection and seroconversion; if used in
the face of acute disease, paired samples should be taken two to four weeks apart. When used for herd screening at any herd size,
testing thirty animals is sufficient to identify a positive herd at 10% or higher prevalence (0.05% risk of error).

Antigen detection testing options include PCR, immunohistochemistry, and histology; culture requires a Level 3 biosafety
laboratory and is not generally performed for routine diagnostics. Histology and/or immunohistochemistry performed on the aborted
placenta is the most reliable method of determining if *C. burnetii* is responsible for a specific abortion event. PCR assays can be run
on abortion/birth products, vaginal secretions, milk, and feces to detect shedding; a positive PCR result indicates that the animal is
both infected and infectious, but due to high levels of subclinical disease, infection with *C. burnetii* may be incidental to the abortion
or exposure event. PCR can be performed on bulk tank milk samples as a herd-level screening tool; a positive test indicates that at
least one herd member is infected and actively shedding *C. burnetii*.

**Treatment**

There are no known effective treatments to prevent infection, reduce shedding, or “clear” a positive serologic status. Parenteral long-
acting tetracyclines (two injections at 20 mg/kg, given 20 days apart) may reduce the number of future abortions in a herd
experiencing an acute abortion storm. Oral administration of tetracyclines in feed is ineffective as a herd-level control mechanism.

**Control**

Due to the persistent and ubiquitous nature of *C. burnetii*, our inability to differentiate uninfected animals, and our inability to
definitively clear either animals or the contaminated environment, test-and-cull and depopulation schemes are not advised. Basic
hygiene and biosecurity measures, including proper disposition of abortion products, is necessary to limit transmission within the herd.

Although official quarantine measures are not usually implemented, common sense dictates that producers should not sell or move
animals between herds during abortion storms. Vaccines that are used overseas are not available in the U.S.

**Client recommendations**

1. If coxiellosis is diagnosed in the herd or flock, advise owners to report any cases of flu-like illness in the household to their
   medical practitioner; clinical signs typically appear two to three weeks after exposure.
2. Limit human access to high-risk areas such as kidding and lambing pens or at high-risk times for aerosolization (e.g.
   cleaning out bedded packs). Limit access of high-risk individuals (immunocompromised, pregnant, or those with heart-valve
   replacement or disease).
3. Use personal protective equipment for high-risk activities such as kidding/lambing, handling abortion products, and working
   in contaminated dusty environments. This includes disposable gloves, fitted face mask respirator (N95 or higher, under
   consultation with a physician), eye protection, and dedicated outer clothing and boots. Follow basic hygiene protocols
   including washing hands with soap and water and refrain from eating, drinking, or smoking in animal areas.
4. Milk from infected animals should be pasteurized before consumption, further processing, or sale. Current pasteurization
   standards (145°F for 30 minutes or 165°F for 15 second) will kill *C. burnetii*.
5. Do not move, sell, or buy animals, especially pregnant animals, during an abortion storm or from producers experiencing an
   abortion storm. Negative tests on individual animals are not sufficient to guarantee that new purchases are uninfected but
   repeated herd testing can identify low-risk herds or flocks.
6. Appropriately dispose of manure, abortion materials, and deceased animals. Follow local regulations, but typical options
   include composting (at least 90 days), incineration, deep burial, and disposing bagged material through biohazard or landfill
   channels. During transport or in arid, windy climates it may help to cover the manure pile to decrease wind-driven
   aerosolization and spread. Implement an effective pest control program. Do not use high pressure hoses to clean high-risk
   areas.
7. Once established, low-risk operations should be tested at least annually, all abortions should be presented for diagnostic
   evaluation, and in the absence of abortion, a subset of normal placentas should be submitted for screening via PCR.

**Additional reading**


and the Q Fever Working Group.” MMWR; 62(RR03); 1-23. Access at www.cdc.gov/mmwr/preview/mmwrhtml/rr6203a1.htm

at www.cdc.gov/mmwr/preview/mmwrhtml/mm6040a5.htm


Bracing for Surgery?  
What to Consider BEFORE You Cut (or Refer) a Cruciate Case
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Mississippi State, MS
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Braces for cranial cruciate disease
- Many companies to choose from online – most will take 1-2 weeks to receive
- Custom braces from 3D printed cast molding are the only potentially valid option
- Best evidence include Stem Cell and/or PRP injections as well
- Case selection is critical
  - No meniscal tear
  - No skin issues
  - Temperament
  - Body type and Body Condition
  - Less than 25-50% tear of cranial medial band
    - Most braces still have 2-4 of drawer
  - Cost 900-1800 to make depending on brand and size of dog
    - Does not include shipping, cast molding or rechecks
- The brace is a temporary fix if 50% or more tear
  - Still need surgery to fix primary problem
  - Brace will wear out and need adjustments or replacement
  - Good option for “buying” time or chemo patients
- Physical Rehabilitation for 15 pounds or less
  - GOALS
    - Increase muscle mass
    - Increase ROM and comfort
    - Increase stifle extension
  - Rule of 3s for success => surgery
    - No when to say when
    - 3 days of non-weight bearing lameness
    - 3 episodes of non-weight bearing lameness in 3 weeks
    - 3 months without improvement in intermittent but persistent lameness
- Pain relief is critical for leg usage
  - NSAIDs
  - Tylenol 3 or 4
  - Tramadol or Gabapentin?
  - Nutraceuticals
  - DIET FOR WEIGHT LOSS

Regional anesthesia for the canine and feline pelvic limb
- Local and regional anesthesia are common practices in large animal veterinary medicine. In the past, locoregional techniques have been underutilized in small; however, recently there has been a surge in their use with small animal practice.
- Quincke needles are designed specifically for locoregional techniques. Quincke needle bevels are blunter which allows for a better feel as the needle dissects through tissue planes.
- Local anesthetics are fast-sodium channel blocking agents. In their bottles local anesthetics are acidic and inactive. When injected into the body (comparatively alkaline), the local anesthetic molecules dissolve into HCl salts and active bases. The active bases diffuses across the nerve epineurium and cell membrane into the cytoplasm and block sodium channels.
Toxic effects of local anesthetic depend on the drug. Lidocaine causes dose-dependent neuro- and cardio-toxic effects. Bupivacaine has potent cardio-toxic effects. Inadvertent intravenous injection of local anesthetics must be avoided; therefore, always aspirate before injecting.

Rear limb locoregionals include lumbosacral epidurals, sciatic-femoral blocks, distal peroneal-tibial blocks.

A good understanding of basic anatomy, pharmacology of local anesthetic drugs, and patient physiology is essential in order to safely, and effectively, utilize local and regional anesthetic techniques.

The most common location for an epidural approach in the dog and cat is the lumbosacral window. In the dog the spinal ends near L5-L6. In the cat the spinal cord ends near S2.

Combining local and regional anesthetic techniques with parenteral analgesics can provide small animal practitioners more flexibility and better options for pre-, intra- and post-operative pain management.

Several locoregional techniques for canine and feline rear limb procedures will be discussed.

Topical fentanyl for dogs

- Expensive
- Staff education is critical
- Lasts for 4-7 days
- Can give well before surgery or after induction
  - Hold off on other mu agonists to avoid dysphoria
  - Consider NSAID and gabapentin for at home
- I use t-shirts for extra layer of protection

3 day buprenex for cats

- Consider mu agonist for surgery
- Good option for adjunct pain control postoperatively with blocks and NSAIDs
Small intestines and stomach
- Blood supply and anatomy
- Common procedures
- Perioperative drug selection
- Biopsy techniques
  - Incisional
  - Punch
  - Resection
  - Mesenteric lymph node and pancreatic sampling

Large intestines
- Blood supply and anatomy
- Common procedures
- Risks with location and ischemia or perforation
- Biopsy techniques
  - Higher risk of mortality
  - Endoscopic option
  - Rectal pull-through option or eversion
  - Resection

After you cut the guts
- Assessing suture line and blood supply/tissue health
- Proper instrument and table handling
- Lavage protocols
- Consider drain pro/cons
- Consider feeding tube pro/coms
- Changing gloves, suture and drapes

Essentials for veterinary anesthesia patient monitoring
- 2011 AAHA Anesthesia Guidelines for Dogs and Cats, Richard Bednarski, MS, DVM, DACVA (Chair), Kurt Grimm, DVM, MS, PhD, DACVA, DACVCP, Ralph Harvey, DVM, MS, DACVA, Victoria M. Lukasik, DVM, DACVA, W. Sean Penn, DVM, DABVP (Canine/Feline), Brett Sargent, DVM, DABVP (Canine/Feline), Kim Spelts, CVT, VTS, CCRP (Anesthesia)
- Most (all) anesthetic drugs at high enough doses can have serious/toxic effects on anesthesia patients. Good patient monitoring is a combination of objective (monitoring devices) plus subjective (hands-on) information. No monitoring device alone can provide all necessary patient information.
- Patient monitoring during anesthesia should be based on individual patient needs; however, minimal components include cardiovascular status, oxygenation, ventilation, blood pressure, body temperature and depth of anesthesia.
- Following trends during anesthesia monitoring is more important than point-in-time methods.
- Ideal monitoring begins with a trained individual dedicated to the anesthetized patient. A complete anesthesia record should be completed for every anesthesia patient and used during ALL anesthesia procedures (including heavy sedation).
- Individual monitoring parameters, advantages and disadvantages, will be discussed.
This lecture will focus on how to better utilize your technical staff for surgical patients and your practice. Everything from calculating drugs, monitoring and pain assessment will be highlights in this hour. The technicians’ role for pre and postop communication with the owner, how to use them for discharges, sales, weight loss and estimates to better serve the practice. Some equipment to help your technical staff care for surgery patients will also be discussed. This whole team approach translates to success for everyone!

- Certified and/or licensed veterinary technicians make excellent nurse anesthetists.
- The roles of a licensed veterinary technician include patient nursing care, patient assessment and surgical preparation, patient anesthesia monitoring, patient post-operative care, client education, supervisor and practice management.
- Utilizing the veterinary technician can be a major contribution to clinical practice by freeing time for the general practitioner and providing an invaluable service to the clients.
- Depending on the size of the practice it is ideal to have at least one or more technician with a major responsibility dedicated to anesthesia and patient welfare. Duties would include patient preparation for anesthesia, equipment upkeep, drug calculations (including CRIs) patient monitoring, and post-operative care.
- Duties for patient preparation and intra-operative patient covered by an anesthesia veterinary technician include IV placement, patient monitoring, IV fluids and CRIs, trouble shooting, post patient care, anesthesia and analgesia-related CE.
- The Academy of Veterinary Technicians in Anesthesia and Analgesia (ACVAA) is a board-certified specialty for licensed veterinary technicians dedicated to the practice of anesthesia and analgesia.
- Surgical nursing is a critical portion for patient care and recovery
  - TLC for wounds, incisions and patient well being
  - Balls, hoists and slings to aid technicians and better compliance for care of large or stubborn patients
- Surgical nurses for estimate explanation and deposits
  - Appears less confrontational
  - Owners more likely to ask questions
  - Saves you time in the exam room, unless you are needed for clarification
- Technicians for weight loss postoperatively
  - Obesity is critical topic for longevity, cancer and Arthritis
  - Check ins/weigh ins for progress
  - Follow-up phone calls
  - Suggestions for diets and treat changes
- General health care advocates at check-in, check-out
  - Builds clinic loyalty
  - Front staff crucial role too
  - Nutraceuticals – education and adjustments
  - Probiotics – indications and options
  - Proper diet selection and knowledge
Post anesthetic complications with the small animal patient

- Anesthetic recovery is the interval from the cessation of anesthetic drug delivery to the point at which the patient is extubated and has voluntary motor control.
- Factors that affect the length of recovery include patient health, length of the anesthetic procedure, anesthetic protocol, and patient, post-anesthetic body temperature.
- According to a Brodbelt, et al., study in 2007, greater than 50% of the canine and feline anesthetic related adversities occur during recovery.
- All anesthetic patients have the potential for poor recoveries. Difficult anesthetic recoveries can be due to multiple factors, including emergence delirium, dysphoria, inadequate analgesia, and general patient discomfort. In these cases, it is often advisable to delay the recovery to avoid further stress or injury to the recovering patient. The authors speculated that inadequate patient monitoring may have been the primary factor behind anesthetic recovery periods being over represented by increased mortality rates in small animal anesthetic procedures.
- Anesthetic monitoring should NOT end at recovery; instead, it should continue until the patient is extubated and has returned voluntary muscle control. The degree of monitoring, and parameters evaluated, depend on the procedure performed and the patient’s health. Patient monitoring should include at least cardiovascular and respiratory status, body temperature, analgesia, and patient (dis)comfort. Post anesthetic, patient monitoring parameters should be included within the patient’s anesthetic records.
- Brachycephalic breeds have an increased risk of post-extubation, upper airway obstruction. During sedation and anesthesia excessive peri-laryngeal tissues and hypoplastic tracheas predispose these patients to pharyngeal obstruction. Ventilatory function should be monitored closely with brachycephalic breeds during pre-operative sedation and post-operative recovery, and it is prudent to have induction agent, a laryngoscope, and an endotracheal tube immediately available in case of upper airway obstruction.
- Several anesthetic recovery parameters and scenarios, with methods to mitigate problems, will be discussed.

Thermals packs

- Ice for 15-20 minutes
  - Wait for body temperature to be above 98 degrees
  - Applied during inflammatory phase (first 2-3 days)
  - Can be used before therapy laser
  - Disinfect between patients
  - Do NOT use on skin reconstructive surgeries

- Therapy Laser
  - My postoperative protocol for wounds 8J/cm2
  - Immediate postop (NOT ON CANCER)
  - In the morning after bandage removal as well
  - Can be daily for 7 days or EOD for two weeks until suture/staple removal
    - Reassess incision
    - Confirm ecollar
    - Regular postoperative confinement
    - Patient temperament may negate benefit

- Game Ready System
- Bandage for 12-24 hours
- Warm Thermal Packs
  - Start 2-3 days out
  - 10-15 minutes each time
- No electric blankets or adhesive pads
- Patient compliance is easier than with cold
Young patients are the most common routine cases that can have issues, with hypothermia being the biggest issue. We will discuss
the complications that can happen with hypothermia and how to avoid them. We will also discuss ways to decrease your infection rate
for routine spay/neuter procedures and better surgical handling of patients in the general practice setting.

- A common mistake made by practitioners is assuming young patients are lower anesthetic risks compared to older
  patients.
- The first consideration when planning an anesthetic event is preparation. Anesthetic preparation includes equipment
  upkeep such as vaporizer calibration, and breathing system and machine leak checks. Monitoring equipment should be
  kept clean and in good working order.
- The anesthetic record is considered a legal document, an integral component of a patient’s complete medical record, and
  is signed by a licensed veterinarian. The anesthetic record chronologically should record all events that occur during the
  anesthetic procedure, including complications.
- Having one person dedicated to patient monitoring during the anesthetic procedure is an important contribution toward
  avoiding intra-operative complications.
- Common issues seen during anesthesia involving veterinary patients include adverse drug reactions, problems related to
  patient intubation, patient hypoxia and hypercapnia, improper levels of anesthesia and/or analgesia, cardiovascular
  related problems and inappropriate body temperatures.
- Hypothermia is a serious issues with all patients but juvenile patients are more at risk
  - Less fat stores
  - Blood pressure and immune system decrease with body temperature
  - Opening a body cavity more critical when is more than 50% of their total body mass
  - Even fast spay/neuter procedures have drops in temperature that extend past surgery time
- Preventative measures
  - Warming blankets
    - Electric
    - Water
  - Warm air circulators
  - Thermal shield blankets
  - Fluid line warmers
    - Not helpful unless volume and rate are effectively high
    - Warm fluid bag
  - Hot water bottles, warm blankets
  - Recovery cage
- With a decrease in body temperature and there immune system can also come an increase in infection
  - Ways to lower soft tissue juvenile infection rates
  - Should be less than 4% for clean or clean/contaminated surgeries
  - Patient’s endogenous flora account for most surgery infections
  - Proper shaving while supporting patient temperature
  - Proper patient prep will ideally lower flora without lowering patient temperature
  - Proper surgical room prep
  - Proper surgical room attire for all in OR
  - There is a risk with perioperative prophylactic antibiotics and they do NOT replace good aseptic technique
Remember fighting to stay awake when toxic plants were being discussed in toxicology class? You thought only large animal veterinarians treating herbivores needed to know about plant toxins. In this session, you will have a chance to review some common and, perhaps, not so common phytotoxins which may be of concern to small animal practitioners.

What impacts plant toxicity?
- Plant toxicity depends on many factors:
  - Plant ID/Toxins/Toxin types in plant
  - Animal species which is exposed
  - Stage of plant growth/Plant parts eaten
  - Animal health status
  - Availability of nutrients/Water for plants
  - Effects of weather/Season/time of year
  - Environmental conditions
  - Geographical location

What impacts plant consumption?
- Many factors influence toxic plant consumption:
  - Palatability
  - Abundance relative to “nontoxic” plants
  - Boredom
  - Hunger
  - Because they are “there”
  - Curiosity
  - Stupidity
  - Possible Darwinian Phenomena

“Dose makes the poison!!!”
Expressions of toxic plant/fungi dose
- Less commonly in terms of toxic principle:
  - Specific dosage of toxic principle (mg/kg of body wei)
- Concentration in diet (ppm)-COMMON WITH MYCOTOXINS
- More commonly in terms of amount of plant eaten:
  - Percent of body weight
  - Percent of diet
  - Incorporation of duration of exposure
  - Consumed once or twice; days, months or years of exposure
- Descriptive terms
  - “Pinch”
  - “Tad”;
  - “Boatload”;
  - “Enough to choke a horse”

The basic clinical management of suspected plant intoxications
Basic work up of a suspected plant intoxication/look familiar???
- Usually an EMERGENCY!!!
  - Simultaneously incorporates aspects of treatment and diagnosis
  - FIRST THINGS FIRST!!!
  - Possible rationale for having yourself or technicians cloned!!!
- Signalment + Clinical Signs/Clinical Circumstances
Who?
What Plant?
When?
Where?

Problem List
What’s wrong?
Physical examination and STAT laboratory testing, IF proximate to patient

“Big Picture” Problems
SUMMARY OF CRITICAL ISSUES AND TARGET SYSTEMS/ORGANS
TREAT THE PATIENT NOT PLANT, UNLESS EXPOSURE TO TOXIC PLANT OBSERVED!!
STABILIZATION OF THE PATIENT IS NUMBER ONE PRIORITY!!!

D.A.M.N.I.T.
D = Degenerative
A = Anomaly
M = Metabolic
N = Nutritional/Neoplastic
I = Infectious/Inflammatory
T = Traumatic/Toxic

PLANT POISONING SHOULD BE SUSPECTED WHEN
OBSERVATION OF PLANT INGESTION/DESTRUCTION/PLANT PARTS IN VOMITUS
Sudden death/Similar clinical signs in MULTIPLE animals
Rapid onset of afebrile syndrome or sudden death of a previously healthy animal.”

IF “OBVIOUSLY” AN INTOXICATION, GO IMMEDIATELY TO INTOXICATION TREATMENT!!!
List plausible toxic plants/Some not so toxic plants and other differentials
Most likely FINAL “Toxic Plant” diagnosis and WHY?
Not always possible
Helpful to know toxic mechanism of action of toxic plants
Correlation with observed problems
Incorporated into Rx/Dx

Plant toxicosis treated/Diagnosis confirmed
Sometimes not possible to do both!!!

Emergency treatment of suspected plant intoxications (SHOULD LOOK FAMILIAR!!!)
REMOVE THE ANIMAL(S) FROM THE SOURCE!!!
MIGHT ACTUALLY BE REMOVAL OF THE SUSPECTED SOURCE FROM THE ANIMAL!!!
House/Yard management

Diagnosis of suspected plant intoxications
PLANT TOXICITY + TOXIC PLANT CONSUMPTION = PLANT INTOXICATION
OBSERVATION OF PLANT PARTS IN VOMITUS OR GASTROINTESTINAL TRACT!!!
TOXIC PLANT ID!!!
IF THE ABOVE DOESN’T HAPPEN: Collect detailed and accurate history
Usually 1st stage of assessing signalment + clinical signs/clinical circumstances
NEXT STEP: Physical examination of the alive and/or dead animal
  o Usually 2nd stage of signalment + clinical signs/clinical circumstances assessment
• Updated Problem List/D.A.M.N.I.T./List of differentials
• Tentative diagnosis/Most likely differentials
• Correlation mechanism(s) of action to Problem List
• Evaluate the efficacy of any treatment in progress.
• Clinical pathology IF ALIVE
• Necropsy exam/Histopathology IF DEAD
  o Collect appropriate samples for toxicologic analyses
  o GARBAGE IN/GARBAGE OUT

Examples of “COMMON” plant intoxications
• Plants Affecting the GI tract
  o Just about every ingested “toxic” plant
  o Plants containing steroidal glycoalkaloids
  o Plants containing insoluble oxalates
  o Plants containing saponins
  o Plants containing toxalbumins
  o *Euphorbia* species and others
• Plants affecting the Heart
  o Plants containing cardiac glycosides
  o Plants containing polyditerpene alkaloids
  o Plants containing grayanotoxins
  o Plants containing taxine alkaloids
• Plants affecting the Nervous System
  o Plants causing neuroexcitation
  o Plants causing receptor-mediated CNS depression
  o Plants affecting the parasympathetic nervous system
  o Miscellaneous plants affecting the CNS

Examples of “COMMON” plant intoxications (CONTINUED)
• Plants affecting the Liver
  o Hepatotoxic blue-green algae
  o Hepatotoxic mushrooms
  o Plants containing pyrrolizidine alkaloids
  o Miscellaneous plants affecting the CNS
• Plants affecting the Kidney
  o Plants containing soluble oxalates
  o True lilies and cats
  o Grapes and raisins and dogs

Useful toxic plant electronic resources
• http://www.aspca.org/pet-care/animal-poison-control/toxic-and-non-toxic-plants
  o List of toxic and “nontoxic” plants
• http://www.ansci.cornell.edu/plants
• http://www.petpoisonhelpline.com/
• http://www.petpoisonhelpline.com/
• http://research.vet.upenn.edu/poisonousplants/Plants/tabid/5259/Default.aspx
• http://www.library.uiuc.edu/vex/toxic/comlist.htm
• http://www.vth.colostate.edu/poisonous_plants/report/search.cfm
There are a variety of online/electronic resources which small animal practitioners can use to access critical information about potential toxicants. In this lecture, these resources will be discussed and demonstrated for specific uses.

**General veterinary toxicology electronic resources**
- Useful lists of potential toxicants/Helpful bulletins/Information regarding
  - [http://www.petpoisonhelpline.com/](http://www.petpoisonhelpline.com/)
  - [https://www.aspca.org/pet-care/animal-poison-control](https://www.aspca.org/pet-care/animal-poison-control)
- Phone apps available
- Phone consultations available on a fee for service basis

**U.S. government websites providing helpful information for suspected intoxications**
  - Information from MSDS for household products
  - Information on product ingredients
- [http://www.fda.gov/AnimalVeterinary/default.htm](http://www.fda.gov/AnimalVeterinary/default.htm)
  - Website for Food and Drug Administration
  - Go to Animal & Veterinary tab for veterinary drug-specific information
  - Useful regulatory information on pet food contaminants/recalls
  - Mechanism for reporting adverse drug reactions/pet food-related incidents
  - Other helpful information on veterinary medications
  - Also useful information on human medications
  - PubMed literature search
  - Website for National Institute of Environmental Sciences
  - Information pertaining to environmental health
- [https://www3.epa.gov/](https://www3.epa.gov/)
  - Website for U.S. Environmental Protection
  - Environmental contamination
  - Superfund sites
  - Pesticides
  - Heavy metals
  - Organic pollutants
  - Website for Agency for toxic Substances and Disease Registry
  - Operated by Center for Disease Control
  - Information on many toxicants
  - Toxicological Profiles
  - Tox FAQs™

**Useful toxic plant electronic resources**
  - List of toxic and “nontoxic” plants
- [http://www.plantweb.ansci.cornell.edu/plants](http://www.plantweb.ansci.cornell.edu/plants)
- [http://research.vet.upenn.edu/poisonousplants/Plants/tabid/5259/Default.aspx](http://research.vet.upenn.edu/poisonousplants/Plants/tabid/5259/Default.aspx)
- [http://www.library.uiuc.edu/vex/toxic/comlist.htm](http://www.library.uiuc.edu/vex/toxic/comlist.htm)
- [http://www.vth.colostate.edu/poisonous_plants/report/search.cfm](http://www.vth.colostate.edu/poisonous_plants/report/search.cfm)
Miscellaneous helpful websites

http://www.msds.com/
Website for free access to material safety data sheets (MSDS)

http://www.snopes.com/
Useful for urban legends/e-mails forwarded by your mother

http://www.merckvetmanual.com/mvm/index.jsp
Generic animal disease information

http://www.ahc.umn.edu/rar/umnuser/formulary.html
Free veterinary drug formulary

https://www.avma.org/News/Issues/recalls-alerts/Pages/default.aspx
American Veterinary Medical Association website on pet food recalls
We live in a “medicated world.” If you doubt that statement, simply sit down and watch the cavalcade of pharmaceutical advertisements which takes place 24/7 on network television. Taking into account all of the prescription, over-the-counter (OTC) traditional AND herbal, and recreational drugs surrounding our pets, it should be no surprise that there is an increasing rate of dog and cat intoxications involving “drugs.” In fact, according to National Animal Poison Control Center, prescription medications and veterinary medications of top toxicants in 2014 [http://www.aspca.org/resource/shelter-health-poison-control/top-10-pet-toxins-2014]. ALWAYS KEEP MEDICATIONS OUT OF REACH OF CHILDREN AND PETS. FOR VETERINARY MEDICATIONS, ALWAYS READ LABELS AND FOLLOW LABEL INSTRUCTIONS.

Acknowledging that, to ALMOST ALL of our clients, pet survival takes precedence over successful diagnosis, it is important to remember the mantra “TREAT THE PATIENT, NOT THE POISON” (UNLESS THERE ARE WITNESSES). THEREFORE, before going on to specific “DRUGS”, the “BASICS” of managing any suspected intoxication, which apply directly to “DRUGS”, are reviewed here and will be quickly summarized during oral presentation.

Overview of basic clinical management of suspected “drug” intoxications

REVIEW of Basic Work Up of a Suspected Drug Intoxication/Look Familiar???:

**Usually an EMERGENCY!!!**

- Simultaneously incorporates aspects of treatment and diagnosis
- FIRST THINGS FIRST!!! PATIENT SURVIVAL OFTEN HIGHER PRIORITY THAN Dx
  - Determine what is actually in suspected “DRUGS”/Jump right to treatment???
- Possible rationale for having yourself or technicians cloned!!!

Signalment + clinical signs/clinical circumstances

- Which Pet or Pets (be sure all present and accounted for)?
- What “Drug” or “Drugs” (how certain/labeled or unlabeled/multiple drugs/legality)?
- When (approximate time or day/might be clueless)?
- Where (IF known/how certain of location/other possibilities)?

Problem list

- WHAT’S WRONG? SOME CLINICAL SIGNS “TOXICANT X” SPECIFIC/SOME GENERIC
- Physical examination and STAT laboratory testing, IF proximate to patient

“Big picture” problems

- SUMMARY OF CRITICAL ISSUES AND TARGET SYSTEMS/ORGANS
- TREAT THE PATIENT NOT DRUG, UNLESS EXPOSURE TO TOXIC “DRUG” OBSERVED!!!
- STABILIZATION OF THE PATIENT IS NUMBER ONE PRIORITY!!!

D.A.M.N.I.T.

- **D** = Degenerative
- **A** = Anomaly
- **M** = Metabolic
- **N** = Nutritional/Neoplastic
- **I** = Infectious/Inflammatory
- **T** = Traumatic/Toxic

Overview of basic clinical management of suspected drug intoxications (continued)

REVIEW of Basic Work Up of a Suspected Drug Intoxication/Look Familiar???: (continued)

- **DRUG POISONING SHOULD BE SUSPECTED WHEN**
  - OBSERVED “DRUG” INGESTION/“DRUG”/PACKAGING IN VOMITUS OR GI TRACT
  - Sudden death/Similar clinical signs in MULTIPLE animals
  - Rapid onset of afebrile syndrome or sudden death of a previously healthy animal.”
  - IF “OBVIOUS” INTOXICATION BY “DRUGS”, GO IMMEDIATELY TO EMERGENCY Rx!!!
- **ONCE ANIMAL STABLE/DIAGNOSIS STILL UNCERTAIN: CONTINUE STEPWISE WORK UP**
- List Drugs/Other toxic and nontoxic differentials
- Most likely FINAL “Drug” diagnosis and WHY?
Detailed clinical management of suspected “drug” intoxications, including Rx/Dx

EMERGENCY Rx/TREATMENT of Suspected “Drug” Intoxications (SHOULD LOOK FAMILIAR!!)

Remove the animal(s) from the source
- MIGHT ACTUALLY BE REMOVAL OF THE SUSPECTED SOURCE FROM THE ANIMAL!!!
- House/Yard management

Immediate veterinary care
TREAT THE PATIENT NOT THE DRUG, UNLESS DRUG EXPOSURE IS OBSERVED!!!
- STABILIZATION ABCs
  - Airway/Breathing/Circulation/Depression/Excitation/Fever/Hypothermia
- Supportive care

Decontamination/antidotal therapy AND/OR lipid infusion for specific “drugs”
- Decontamination is ANOTHER way to separate the source from the animal!!! Depends on the route of exposure and stage of the intoxication
  - Bathing if plant material on paws (think cats)
  - Emesis/GI lavage/Activated charcoal (repeated?) ± Cathartics if ingested “Drugs”
  - MAKE SURE INDUCTION OF EMESIS+ NOT CONTRAINDICATED
- Some specific antidotes for “Drugs”?/Lipid infusion limited to a few “Drugs”

Confirming diagnosis of suspected “drug” intoxications (not always possible)
- OBSERVED “DRUG” EXPOSURE/“DRUGS”/PACKAGING IN VOMITUS OR GI TRACT
- MIGHT NOT NEED TO GO MUCH FURTHER FOR IDENTIFIABLE “DRUG”!!!
- POSSIBLE EXCEPTION FOR “LEGAL” CASES WHERE “DRUG” CONFIRMATION NEEDED
- IF THE ABOVE DOESN’T HAPPEN OR “LEGAL”: Make sure detailed and accurate history!!!
  - Usually 1st stage of assessing signalment + clinical signs/clinical circumstances
  - NEXT STEP: Physical examination of the stabilized alive and/or very dead animal
  - Usually 2nd stage of signalment + clinical signs/clinical circumstances assessment
  - Tentative “Drug” diagnosis/Most likely differential is “Drug”
  - Correlation of “Drug” MOA to Problem List helps support FINAL “Drug” Dx.
  - Evaluate the efficacy of treatment in progress, especially antidotes for specific “Drugs”

Detailed clinical management of suspected “drug” intoxications (continued)

CONFIRMING Diagnosis of Suspected “Drug” Intoxications (NOT ALWAYS POSSIBLE/continued):
- Clinical pathology IF ALIVE/Possible laboratory analyses for “Drugs”
- Necropsy Exam/Histopathology ± Toxicology Testing IF DEAD
  - Collect appropriate samples for histopathology IN FORMALIN
  - Carefully labeled and separated samples/COC?/Refrigerated or frozen (best)
  - “Drugs”/Vomitus/Gastric Contents/Liver/Kidney/Brain (if CNS)/Urine???/Fat??
  - Knowledge of “Drug” MOA and pharmacokinetics/toxicokinetics can be helpful.
    - Often determines analyses for what and when.
  - IDEALLY, histopath/analytical results are consistent with one another and “Drug” Dx.
  - HOWEVER, GARBAGE IN = GARBAGE OUT!!!
    - “Rotten” tissues tell no tales!!!/Pathognomonic lesions might be MIA!!!
    - BUT, can’t analyze for “Drugs”, IF ideal tissue samples/source not collected.

- Challenges to diagnosing “Drug” intoxications.
  - For “Drug” known to have been given therapeutically, what does detection mean?
  - Analyses not available for all “Drugs”.
Ingredients in proprietary “Drugs” or illegal “Drugs” might not be known
Differences in analytical results between diagnostic laboratories?
Differences in interpretation?
Certainty of identification?
“Legal” issues
  • Illegal drugs detected?

Useful “drug” electronic resources
MIGHT NEED TO JUST KNOW INGREDIENTS OF DRUGS/POTENTIAL ADVERSE EFFECTS

- [http://www.ahc.umn.edu/rar/umnuser/formulary.html](http://www.ahc.umn.edu/rar/umnuser/formulary.html)
  - Free veterinary drug formulary
  - Website for free access to material safety data sheets (MSDS)
  - Information from MSDS for OTC household products/Lists of ingredients
- [https://www.crowd.org/](https://www.crowd.org/)
- [Psychoactive plants/drugs](http://www.fda.gov/AnimalVeterinary/default.htm)
  - Website for Food and Drug Administration
  - Go to Animal & Veterinary tab for veterinary drug-specific information
  - Mechanism for reporting adverse drug reactions
  - Other helpful information on veterinary and human medications
  - PubMed literature search
  - Veterinary Poison Control Centers
- [http://aspcapro.org/human-animal-medication](http://aspcapro.org/human-animal-medication) for pet professionals

Examples of “common” intoxications associated with various classes of “drugs”

**Prescription human medications**

- NSAIDs
  - Zorvolex (Diclofenac)→Gastric ulcers at very low dosages in cats and dogs.
- Antidepressants
  - Effexor (Venlafaxine) and Prozac (Fluoxetine)+→SSRIs→↑Heart rate + ↑blood pressure + hyperthermia + sedation, ataxia, tremors, and/or seizures
- Benzodiazepines and Sleep Aids
  - Xanax (Alprazolam) and Ambien (Zolpidem)+→GABA agonism→CNS depression
- ADD/ADHD Medications
  - Adderall (Dextroamphetamine/Amphetamine) and Ritalin (Methylphenidate)+→Stimulants→↑Heart rate + ↑blood pressure + tremors, seizures, hyperthermia
- β-Blockers→Much more severe toxic effects than seen with ACE-inhibitors
  - Tenormin (Atenolol) + Coreg (Carvedilol)→Bradycardia + hypotension
- Cholesterol Lowering Agents
  - Statins→Lipitor (Atorvastatin) and Crestor (Rosuvastatin)→Vomiting + diarrhea

**OTC human medications**

- NSAIDs
  - Advil (Ibuprofen), Aspirin, and Aleve (Naproxen)→GI + kidney + CNS (Ibuprofen)
- Pain Medications
  - Tylenol (Acetaminophen/Paracetamol)→Methemoglobinemia + hepatic necrosis
  - Pseudoephedrine→Sympathomimetic thermogenic stimulant
  - Caffeine→Neuroexcitation + PVCs + other cardiac arrhythmias
  - NSAIDs→Primarily GI + kidney + occasional liver involvement
  - Acetaminophen→Methemoglobinemia + hepatocellular necrosis
- Dextromethorphan (for coughing → Serotonin agonism → Tremors + seizures
- Imidazoline Decongestant → α₂ Adrenergic agonism → Bradycardia + hypotension
- Antihistamines → Agitation + sedation + abnormal heart rate and blood pressure
- Benzocaine (local anesthetic) → Possible methemoglobinemia
- Xylitol → Hypoglycemia + hepatocellular necrosis in dogs

Veterinary medications
- NSAIDS
  - Rimadyl (Carprofen) and Phenylbutazone → GI + kidney + liver (Carprofen in dogs)
- “Dewormers”
  - Ivermectins → GABA agonism → CNS depression (especially with mutated MDR1)

Herbal preparations
- Guarana (Methylxanthines) and Ephedra/Ma Huang (Ephedrine/Pseudoephedrine)
  - Ephedrine and pseudoephedrine are sympathomimetic thermogenic stimulants.

Recreational drugs and drugs of abuse
- Marijuana → Cannabinoids → THC → “Depression” + ataxia + incontinence
- K₂ → Synthetic cannabinoids → Variety of effects + concerns about adulteration
- Miscellaneous others, such as prescription painkillers various mild-altering “Drugs”
So Many Pet Food Recalls...is Manufactured Pet Food Actually Safe?

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Based on reports in the popular media and information on the Internet, one would be led to believe that pet food manufacturers are actually out to eradicate our pets. Frankly, that doesn’t sound like a particularly financially rewarding business plan. Admittedly, there is a high correlation between animals which eat commercially manufactured pet food and those who get sick. Similarly, most dogs which get hit by cars are also eating many of these same pet foods. Furthermore, ALL animals eating commercially prepared pet foods will die.

The simple truth is that MOST pet food manufacturers in the U.S. are extremely conscientious. However, human and machine errors do occur; pet food ingredients do come from all over the world, with food quality and consumer protection regulations not being globally uniform; pet food manufacturers cannot control what happens to their products during storage by a distributor or, even, by a pet owner; pets may have chronic, life-threatening diseases without ever exhibiting any overt clinical signs, and then the pet all of sudden drops over dead; and YES, there are those who would try to profit at the expense of the health and welfare of our pets.

Causes of pet food recalls
- Bacterial contamination
  - Contamination by *Salmonella* species appears to be the most common
- Mycotoxin contamination
  - Aflatoxins and DON appear to be the most common clinically relevant contaminants.
- Formulation errors involving vitamins and minerals
  - Can be intoxications or deficiencies
- Various medications
  - Previous concerns about medications used for animal euthanasia
  - Rendering of euthanized animals thought to be most likely source.
- Miscellaneous
  - Melamine/Cyanuric acid and other potential “zebras”

How does a veterinary professional recognize that contaminated pet food is causing disease?
- Animals from different households, with similar signs after consuming same pet food
  - Magnified by the same pet food being marketed under several different names
  - More widespread if contaminated ingredient is used in multiple products and brands
  - More likely when single manufacturing plant is sole source for a specific diet type
  - Drought-stressed corn-based diets are more likely to be contaminated by aflatoxins.
  - “Suspicious” when animals get sick after eating “cheaper” unheard of brand
  - Also “suspicious” when animals get “sick” immediately after eating new pet food?
- More questionable circumstances for suspecting contamination of pet food
  - Rumors of diets contaminated with known toxicants without appropriate signs???
  - Conspiracy theories originating from unreliable sources???
  - Single animal eating manufactured pet food, which subsequently gets sick/dies???
  - Any animal experiencing GI, cardiac, hepatic, or renal disease???
  - Any animal eating commercial pet food???
  - Enemy of owner works for pet food manufacturer????????

What should veterinary professionals do when pet food contamination is suspected?
- Make sure there is a detailed/accurate history, along with a thorough physical exam
- Consult previous health records to rule out pre-existing disease
- Consult with FDA and AVMA pet food recall websites to see if pet food already recalled.
- Perform appropriate diagnostic tests to eliminate other causes of observed disease
  - If case involves pet death, postmortem exam with appropriate sampling is needed.
- Carefully record all information/Maintain detailed medical records
- Get a sufficient amount of suspect pet food and store/label sample appropriately
  - Retain original container and labeling
  - Need enough sample so some can retained and multiple analyses performed

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For legal purposes, often best if sample from unopened bag of the same brand/lot #
Ideal for veterinarian to collect pet food sample and retain custody/ship sample
Determine the most appropriate method for long-term storage
  - Room temperature/Refrigerated/Frozen
  - Might need to consult with label/manufacturer/regulatory personnel

It is critical to record the following product information
- Exact name of the product and product description, as stated on the product label
- Type of container
- Product intended to be refrigerated, frozen, or stored at room temperature
- Lot #
  - Can sometimes be challenging to locate
- Best by, best before or expiration date
- UPC code
  - Also known as the bar code
- Net weight
- Purchase date and exact location where purchased.
- How the food was stored, prepared, and handled

Appropriate testing of pet food generally involves mycotoxin analyses/bacterial culture
- Not all of the sample should be sent off for analyses or sent back to manufacturer.
- A portion of the sample should be retained until the situation is completely resolved

Pet food manufacturer can””should???” be informed of potential problem with product
- Might be most appropriate after testing pet food, so analytical results can be shared.
- However, if there has been pet death, it might be best to contact manufacturer first.
- If pet food contamination is likely, manufacturer contact should involve veterinarian.
- Not all of the sample should be sent off for analyses or sent back to manufacturer.
- A portion of the sample should be retained until the situation is resolved!!!

There is mechanism in place for reporting pet food complaints to the FDA
- [http://www.fda.gov/AnimalVeterinary/SafetyHealth/ReportaProblem/ucm182403.htm](http://www.fda.gov/AnimalVeterinary/SafetyHealth/ReportaProblem/ucm182403.htm)
- If pet food contamination likely, veterinarian should be involved in reporting to FDA.
- Not all of the sample should ever be sent off for analyses or sent to manufacturer.
- A portion of the sample should be retained until the situation is resolved.

Potentially useful sources of information on mycotoxin contamination in pet food
- [http://www.fda.gov/AnimalVeterinary/default.htm](http://www.fda.gov/AnimalVeterinary/default.htm)
- [https://www.avma.org/News/Issues/recalls-alerts/Pages/default.aspx](https://www.avma.org/News/Issues/recalls-alerts/Pages/default.aspx)
The Neighbor Did it!
Common Malicious Poisonings and How to Prove it
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The goal of this lecture is to outline a practical approach to small animal intoxications, with real examples of malicious poisonings, attempted “peticides” which were actually canine suicides, and various tasty and yucky poisons pets get into!!!

In this presentation we will review the following
- Some fundamental principles of toxicology
- The basics of the clinical management of suspected intoxications
- A few examples of malicious intoxication attempts/successes
- Some tasty/yucky things pets will eat

Fundamental principles of veterinary toxicology
All veterinarians should know by their second day of practice that EVERYONE has someone who dislikes them enough to harm their pets!!! Furthermore, according to Paracelsus, “Solely the dose determines that a thing is not a poison.” This statement essentially means that ANYTHING is potentially toxic, depending on the level of exposure!!! It also means that, with regards to potential toxicants/intoxications:
- There is a dose-response relationship for potential toxicants.
- The greater the dose/dosage of a given toxicant, the more severe the clinical signs.
  - Exposure to toxicants and/or their metabolites must be sufficient to cause intoxication.

However, there can be what appear to be possible exceptions to these “principles” (e.g., endocrine-disrupting chemicals), so it is critical to carefully define the toxicant being discussed, the different toxic endpoints being evaluated for that toxicant, and to take into consideration possible species differences in the effects of different toxicants.

Overview of basic clinical management of suspected intoxications
REVIEW of Basic Work Up of a Suspected Intoxication (“Malicious” OR “Accidental”):
- Often an EMERGENCY!!!
  - Simultaneously incorporates aspects of treatment and diagnosis
  - FIRST THINGS FIRST/MOST CLIENTS PREFER PET SURVIVAL OVER CONFIRMED Dx!!!
    - Determine what is in “Toxicant X” (NEED LABELS)/Jump right to treatment???
  - Possible rationale for having yourself or technicians cloned!!!
- Signalment + Clinical Signs/Clinical Circumstances
  - WHICH PETS (names, breeds, ages+)?/WHAT ARE THE CLINICAL SIGNS???(video?)
  - Exposure to “Toxicant X”→≈HOW MUCH?/WHEN?/WHERE? (How reliable is info?)
- Problem List
  - WHAT’S WRONG? SOME CLINICAL SIGNS “TOXICANT X” SPECIFIC/SOME GENERIC
  - Physical examination and STAT laboratory testing, IF proximate to patient
- “BIG PICTURE” PROBLEMS
  - SUMMARY OF CRITICAL LIFE-THREATENING ISSUES AND TARGET SYSTEMS/ORGANS
  - TREAT THE PATIENT NOT THE POISON, UNLESS TOXIC EXPOSURE OBSERVED!!!
  - STABILIZATION OF THE PATIENT IS NUMBER ONE PRIORITY!!!

Overview of basic clinical management of suspected intoxications (continued)
REVIEW of Basic Work Up of a Suspected Intoxication (“Malicious” OR “Accidental”/continued):
- D.A.M.N.I.T.
  - D = Degenerative
  - A = Anomaly
  - M = Metabolic
  - N = Nutritional/Neoplastic
  - I = Infectious/Inflammatory
  - T = Traumatic/Toxic
- POISONING SHOULD BE SUSPECTED WHEN:
DIRECTLY OBSERVED TOXIC EXPOSURE/"TOXICANT X" IN VOMITUS OR GI TRACT
- Sudden death/Similar clinical signs in MULTIPLE animals
- Rapid onset of afebrile syndrome or sudden death of a previously healthy animal.
- Signs of unknown etiology/Other causes ruled out
- Recent change in diet or environment
- Neighborhood feuds/Love gone bad/Pet owner often certain of “CULPRIT”
- Very small, young, mean, noisy, annoying, and/or stupid animals!!!
- Might be a “Darwinian phenomenon” OR “aliens”, “bikers”, “local meth labs”
- IF “OBVIOUS” INTOXICATION, GO IMMEDIATELY TO EMERGENCY Rx!!!

ONCE ANIMAL STABLE/DIAGNOSIS STILL UNCERTAIN: CONTINUE STEPWISE WORK UP

1. List plausible toxic/Some not so toxic differentials
2. Most likely FINAL “Toxic” diagnosis and WHY IS IT “TOXICOSIS X”
   - Not always possible to CONFIRM Dx/Looking and acting like “Dx” might be sufficient.
3. Helpful to know toxic mechanism(s) of action (MOA) of “Toxicant X”
   - Good correlation of “Toxicant X” MOA with Problem List supports “Toxicosis X” Dx
   - Can incorporate knowledge of toxic MOA into successful treatment plan
4. “Toxicosis X” successfully treated/Diagnosis of “Toxicosis X” confirmed
   - Sometimes not possible to do both “Toxicosis X” Dx confirmed by laboratory testing

Detailed clinical management of a suspected intoxication, including Rx/Dx

**EMERGENCY Rx/TREATMENT of Suspected Intoxications:**

1. REMOVE THE ANIMAL(S) FROM THE SOURCE OF “TOXICANT X”!!!
   - MIGHT BE REMOVAL OF THE SUSPECTED “TOXICANT X” SOURCE FROM ANIMAL!!!
     - Baths for cutaneous exposures to “Toxicant X” (especially the paws of cats)
     - IF “X” eaten, Emesis/GI lavage or containment/Activated charcoal ± Cathartics
   - House/Garage/Kennel/Yard/Junk management
2. IMMEDIATE VETERINARY CARE!!!
   - TREAT PATIENT NOT THE POISON, UNLESS “TOXICANT X” EXPOSURE OBSERVED!!!
   - STABILIZATION ABCs
     - Airway/Breathing/Circulation/Depression/Excitation/Fever/Hypothermia
   - Supportive care
   - Decontamination/Antidotal therapy AND/OR Lipid Infusion for specific Intoxications
     - Decontamination is ANOTHER way to separate the source from the animal!!!
     - Depends on route of exposure/Stage of intoxication/Specific antidote (IF available)
   - Bath for cutaneous exposures to “X”/Emesis+ IF NO contraindications (“X” eaten)

**Detailed clinical management of a suspected intoxication, including Rx/Dx (continued)**

Some “guidelines” for stabilization ABCs

1. Ensure that the Airway is patent
   - Awareness of obstructions/bronchoconstriction
2. Establish normal Breathing
   - Awareness of breathing problems/impaired gas exchange
3. Correct Circulation deficits
   - Fluid/Electrolyte/Acid-base imbalances + various anemias with different etiologies
4. Control Depression of CNS
   - Correct metabolic disturbances/neurotransmitter imbalances
5. Control Excitation of CNS
   - Do nothing if very mild
   - Correction of electrolyte imbalances and possible glucose deficits
   - Anticonvulsant medications
6. Bring down Fever
   - Avoid use of NSAIDs for toxicant-induced hyperthermia
7. Treat Hypothermia
   - No ice water baths

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Supportive care
- Maintenance of vital functions/fluid therapy
  - Might also be used in stabilization
- Antibiotics/Analgesics/Diet modifications/Client communication/Client education

Patient stabilized/Dx uncertain → fine-tuned clinical signs/clinical circumstances reassessment
- Might be onsite/animal side in clinic/remote by telephone, text, or ???
  - Access to both premises and animals is ideal!!!
- A thorough and accurate history is often the key!!!
  - Prevents the chasing of many wild geese AND “innocent” parties!!!
- Information might be relayed by phone or be secondhand.
  - The accuracy of such information might be questionable OR “slightly” exaggerated!!!
- Asking the right questions is extremely important!!!
  - Might need to ask the same question several different ways!!!
  - Might need to seek out the individual really in the know OR the “CULPRIT”!!!
- A good physical examination is of critical importance and may need to be repeated!!!
- It is perfectly ok to use a stethoscope and a thermometer!!!
- So is careful observation of clinical signs/circumstances!!!
  - Direct observation of patient might yield very different information!!!!
  - Impacted by scheduling/lighting/finances/crowds/decomposition/”karma”!!!

Confirming diagnosis of suspected intoxications (not always possible)
- DIRECT OBSERVATION OF “TOXIC” EXPOSURE/”TOXICANT X” IN VOMITUS OR GI TRACT
- PRETTY MUCH TRUMPS EVERYTHING ELSE IF “TOXICANT X” IS IDENTIFIABLE
- BRING IN CONTAINERS/LABELING/MSDS/ANY AVAILABLE DOCUMENTATION
- MIGHT BE ALL DONE WITH Dx/EXCEPT "LEGAL" CASES REQUIRING Dx CONFIRMATION

Detailed clinical management of a suspected intoxication, including Rx/Dx (continued)
CONFIRMING Diagnosis of Suspected Intoxications (NOT ALWAYS POSSIBLE/continued):
- IF PRECEDING DOESN’T HAPPEN OR “LEGAL”: MUST have detailed and accurate history
  - Usually 1st stage of assessing/reassessing clinical signs/clinical circumstances
- NEXT STEP: Physical examination of the stabilized alive and/or very dead animal
  - Usually 2nd stage of clinical signs/clinical circumstances assessment/reassessment
- Updated Problem List/D.A.M.N.I.T./List of differentials
- Tentative diagnosis of “Toxicosis X”/”Toxicosis X” is most likely differential/WHY???
- Correlation of “Toxicant X”MOA to Problem List helps support Dx of “Toxicosis X”.
- Evaluate efficacy of treatment in progress, especially specific antidotes for “Toxicant X”
- Clinical pathology IF ALIVE/Might be able to do some chemical analyses for “Toxicant X”
- Necropsy Exam/Histopathology ±Toxicology Testing IF DEAD
  - Collect appropriate samples for histopathology IN FORMALIN
  - Collect appropriate samples for toxicologic analyses NOT IN FORMALIN!!!
  - CAREFULLY labeled and separated samples/COC/?/Refrigerated or frozen (best)
  - Bait/”Drug”/Plant/UFO/Vomitus/Gastric Contents/Liver/Kidney/Brain?/Urine?/Fat?
  - A variety of useful screening/confirmatory analyses for metals/organic toxicants
  - IDEALLY, histopath/analytical results agree with one another AND “Toxicosis X” Dx.
  - HOWEVER, GARBAGE IN = GARBAGE OUT
    - “Rotten” tissues tell no tales!!!/Pathognomonic lesions might be MIA!!!
    - Can’t analyze for “Toxicant X” IF appropriate tissue samples/source not collected

Examples of cases of malicious intoxication attempts/successes + work up
- “The Yellow Pills in the Hotdog”
- “The Stalker and the Suspicious Pan of Water”
- “Beware of Strangers Bearing Meatballs”
- “SHOW ME the Meth Lab”
- “Stay Out of My Stash”
- “ANY CIDF”
- Criminals have a tendency to break the law
Emerging “Peticides”
“Oldies but Goodies”

Some tasty/yucky things our pets will eat voluntarily + example of work up
Most to also be discussed in other lectures
- “The Grass is Always Greener”
- “Attempted “Pesticide at the Sale Barn”
- “Makeover Gone Bad”
- “Hit and Run or Otto accident”
- “Death Without Cavities”
- Play-Doh and Paint Balls
- Bread Dough and Home Brewing Supplies
- Anything Chocolate and Other Methylxanthines
- NSAIDs/Acetaminophen/Other Medications
- Compost Piles AND ANYTHING THAT SMELLS LIKE “POOP”
By altering pre-glomerular resistance, healthy kidneys can maintain relatively stable glomerular capillary pressures despite variations in systemic blood pressure. This pressure regulatory process is termed “renal autoregulation”. Autoregulation can be reduced when renal disease results in loss of nephrons. Compromised autoregulation allows high systemic blood pressure to be transmitted to glomerular capillaries. This glomerular hypertension has been documented by micropuncture studies in dogs and cats with surgically reduced renal mass. In these models, glomerular hypertension was associated with glomerular hypertrophy and sclerosis and proteinuria. Systemic hypertension is relatively common in dogs and cats with renal disease. In a recent study of dogs with spontaneous chronic kidney disease (CKD), 29/45 (64%) had systolic blood pressure ≥ 144 mm Hg and 14/45 (31%) had systolic blood pressure ≥ 161 mm Hg. In cats with naturally-occurring CKD, systemic hypertension has been observed in 19-65% of cases depending on the definition of hypertension.

Renal proteinuria can result from glomerular and/or tubular abnormalities in dogs and cats with CKD. Glomerular proteinuria may arise from immune complex disease or structural abnormalities involving the glomerular capillary wall (more common in dogs e.g., amyloidosis and x-linked hereditary nephropathy). Protein-losing nephropathy caused by glomerular capillary wall lesions is often accompanied by systemic hypertension and glomerular proteinuria can be exacerbated by intraglomerular glomerular hypertension that can result from systemic hypertension. Tubular proteinuria occurs when tubular reabsorption of protein from the glomerular filtrate is compromised. Whether caused by capillary wall lesions, tubular lesions, or intraglomerular hypertension, excessive quantities of protein in the glomerular filtrate may contribute to additional glomerular and tubulointerstitial lesions leading to loss of more nephrons. Indirect systolic blood pressure greater than 160 mmHg is often listed as the threshold for systemic hypertension in dogs and cats.

**Hypertension**

Systemic hypertension in animals has largely been thought to be secondary to another disease (e.g., renal disease and endocrinopathies), as opposed to idiopathic (primary or essential). This has recently been called into question. For example, in a report of 69 hypertensive cats, seen at North Carolina State University for ocular disease, revealed that at least 17%, and possibly as many as 50%, of cats had no identifiable cause for their systemic hypertension. Elliott and associates at the Royal Veterinary College in London have documented that approximately 20% of hypertensive cats, diagnosed in primary-care practices, were idiopathic. Another retrospective study, which used very strict criteria for the diagnosis of primary (essential, idiopathic) hypertension, revealed a prevalence of 11% in cats.

Described and potential etiologies of secondary hypertension include acute and chronic renal disease, hyperthyroidism, hypothyroidism, hyperadrenocorticism, hyperaldosteronism, pheochromocytoma, diabetes mellitus, and obesity. Chronic kidney disease has the greatest association with hypertension and may often be causal. A recent report suggested approximately 29% of elderly cats with CKD were hypertensive, with the range reported in 4 studies being 19-65%. In dogs with CKD, approximately one-third will be normotensive, one-third will have borderline hypertension, and one-third will be hypertensive.

Systemic hypertension may contribute to progressive nephron loss by causing irreversible glomerular damage via increased intraglomerular pressures and glomerulosclerosis. By altering pre-glomerular resistance, healthy kidneys can maintain relatively static glomerular capillary pressures despite variations in systemic blood pressure via autoregulation. Inappropriate dilation of the afferent glomerular arteriole occurs in dogs and cats with CKD and diminishes the ability of the afferent arteriole to protect the glomerulus from variations in systemic blood pressure. Although the exact mechanism of the CKD-associated hypertension is not known, a combination of glomerular capillary and arteriolar scarring, decreased production of renal vasodilatory prostaglandins, increased responsiveness to normal pressor mechanisms, and activation of the renin-angiotensin-aldosterone system (RAAS) may be involved. The increased renin secretion leads to increased production of angiotensin II and aldosterone. In addition to its direct pressor effects, angiotensin II also has a stimulatory effect on the sympathetic nervous system, increasing vascular tone, and, in CKD vasoconstriction of the efferent arteriole which further contributes to the intraglomerular hypertension. Finally, angiotensin and aldosterone may also stimulate renal tissue remodeling via increased matrix production and fibrosis.

The consequences of systemic hypertension are usually dependent on the magnitude and duration of the blood pressure elevations. Acute ocular and central nervous system abnormalities can occur associated with hemorrhage or edema formation. Renal damage associated with hypertension tends to be more chronic and characterized by glomerular lesions (e.g., glomerulosclerosis) and proteinuria. Finally, functional/adaptive changes like ventricular hypertrophy can occur due to increased after-load in patients with
hypertension. Diagnosis and treatment of hypertension in dogs and cats with CKD may prevent development of retinal and CNS lesions or may limit or slow progression of renal and cardiac lesions.

**Proteinuria**
Renal proteinuria is a diagnostic marker of the severity of renal disease and potentially a mediator of glomerular and tubular injury. Recent findings have demonstrated that proteinuria is associated with increased risk of developing azotemia in aged cats and progression of renal disease in both dogs and cats with azotemic CKD. In addition, studies have shown that therapies that reduce the magnitude of proteinuria may be renoprotective. Proteinuric renal disease is often associated with systemic hypertension, which can conversely exacerbate renal proteinuria and therefore, it is difficult at times to separate the effects of high systemic and intraglomerular pressures and proteinuria.

**Management**
Indications for the use of ACEI in dogs and cats with CKD include hypertension and/or proteinuria. The initial recommended dose for ACEI is: 0.5 mg/kg PO once daily. Treatment goals are an indirect blood pressure < 160 mm Hg and/or ≥ 50% reduction in baseline UP/C. The initial ACEI dose can be doubled if the desired outcome is not achieved. Doses greater than 0.5 mg/kg twice daily may result in further reductions in proteinuria; however there are no controlled studies proving these higher doses are beneficial. Questions often arise regarding the use of Benazepril vs. Enalapril. Most of the canine studies have been accomplished with enalaprilat whereas most of the feline studies have used benazepril. There are differences in excretion; enalaprilat (the active metabolite of enalapril) is excreted via the kidney whereas there is hepatic metabolism of benazeprilat. Use of ACEI is likely associated with reduced efficacy and increased risk of adverse drug events as azotemia increases. Increased monitoring should be employed in dogs and cats with serum creatinine concentrations greater than 3.0 mg/dl. Use of ACEI in dogs and cats with serum creatinine concentrations greater than 5.0 mg/dl is usually not recommended. In addition, use of renal diets with reduced quantity/high quality protein may have an additive effect on proteinuria when used with ACEI (Burkholder WJ, et al, J Vet Intern Med 2004; 18:165 and Cortadellas O, et al, ACVIM Proceedings 2012).
Gradual reduction of dietary salt is often recommended as the first line of treatment for hypertension; however there are no studies that document the efficacy of this treatment. The calcium channel antagonist (CCA), amlodipine is often recommended as the first choice anti-hypertensive treatment for cats. Recent studies have shown that treatment with a CCA alone reduces proteinuria in cats with CKD suggesting that intraglomerular pressures were also reduced by this treatment (see #8 above). In those cases where systemic hypertension persists after initiation of ACEI treatment, or is initially greater than 180 mm Hg, a CCA can be added. The overall risk of target organ damage to the eyes, brain, heart, and kidneys is thought to be minimal if systolic blood pressure is maintained at ≤ 150 mm Hg.

**What evidence exists that ACEI are beneficial in dogs and cats with CKD (the good)?**


**ACEI are not always effective in modulating proteinuria and hypertension (the bad)**
Unfortunately, ACEI treatment is not always effective in reducing hypertension and/or proteinuria. It’s well known that CCA (vs. ACEI) are usually more effective in reducing systemic hypertension in cats compared with dogs. In some cases renal proteinuria may initially respond to treatment with an ACEI and then “escape”. This escape may be associated with up-regulation of alternative pathways for production of angiotensin II. For example, elastase and cathepsin G may directly convert angiotensinogen to angiotensin II. In other cases, renal proteinuria may fail to respond to even the initial ACEI treatment. Potential reasons for this lack of efficacy may involve dosing (i.e., suboptimal doses) or advanced renal disease. Very few of our patients with proteinuric CKD undergo a thorough histologic evaluation. Dogs and cats with advanced histologic disease may be less likely to be responsive to any
pharmacologic influence. Similarly, there may be certain structural pathologies that are more likely to be responsive than others. Histologic correlation with response to ACEI treatment has not been evaluated.

In those cases where there is a poor response to treatment (i.e., less than 50% reduction in baseline UP/C), the use of angiotensin receptor blockers (ARB) (either alone or in combination with ACEI) has been recommended. A recent meta-analysis on the effects of mono-therapy and combination therapy with inhibitors of the renin–angiotensin system compared the efficacy of ARB and ACEI in people with proteinuric renal disease. Forty-nine studies involving 6181 participants reviewed randomized trials of ARB versus placebo, ACEI, or the combination of ARB and ACEI in patients with microalbuminuria or proteinuria for whom data were available on urinary protein excretion at baseline and at 1 to 12 months. The conclusions were the reduction in proteinuria from ARB and ACEI is similar, but their combination is more effective than either drug alone. However, the authors stated that the uncertainty about adverse effects and outcomes that are important to patients limits applicability of these findings to clinical practice (Kunz R, et al, Ann Intern Med. 2008; 148:30). There are no studies in veterinary medicine evaluating the safety and efficacy of ARB (alone or in combination with ACEI) for the treatment of proteinuric renal disease.

ACEI treatment may be associated with worsened azotemia in CKD (the ugly)

In some cases treatment of proteinuric renal disease with ACEI is associated with worsening azotemia. Fortunately, this adverse event is relatively rare. Most adverse effects occur in dogs with congestive heart failure (CHF) and the relative contributions of the ACEI vs. decreased cardiac output and Lasix therapy on renal function are unknown. It should be noted however that ACEI treatment of CHF have relatively few adverse effects on kidney function (The COVE study, J Vet Intern Med 1995; 9:243 and Atkins CE, et al, J Am Vet Med Assoc 2002; 221:1149). Adverse effects on renal excretory function would be expected to be more commonplace as azotemia increases. Increased monitoring for worsening of azotemia is recommended in patients with baseline serum creatinine concentrations greater than 3.0 mg/dl; use of ACEI in dogs and cats with serum creatinine concentrations greater than 5.0 mg/dl is not recommended.
Almost all (99%) of the body’s calcium (Ca) is stored in crystalline form in bone along with phosphate (hydroxyapatite). Less than 1% of the body’s calcium is typically present in the extracellular fluid and this circulating Ca exists in three forms: 1) that which is bound to serum proteins; principally albumin (~ 40%), that which is complexed with anions like citrate, bicarbonate, phosphate, lactate, and sulfate (~ 10%), and that which is ionized or active (iCa) (~ 50%). The ionized and complexed forms of Ca are diffusible, whereas the protein bound fraction is not. The Ca values reported on standard laboratory serum biochemistry profiles reflect the total Ca concentration (tCa). Most of the day to day Ca homeostasis is balanced by dietary intake and loss through the gastrointestinal tract with the kidneys having a relatively minor role. For example, 90% of ingested Ca is usually excreted via the GI tract but if necessary, greater than 100% of dietary intake can be excreted via GI secretory fluids. Within the kidneys, normally Ca in the glomerular filtrate is highly conserved via tubular reabsorption by the proximal convoluted tubule cells. The ionized form of Ca accounts for most of the biologic activity of calcium (e.g., muscle contraction, neuromuscular transmission, vascular tone, and cellular membrane and transport functions) and this active form is highly regulated by parathyroid hormone, calcitriol, and calcitonin. When this homeostatic control is impaired, serum Ca concentrations may increase above the normal range resulting in hypercalcemia. Although the term “hypercalcemia” is often used in reference to increases in tCa, its use is best reserved for those instances when the iCa is increased.

Parathyroid hormone (PTH)
Minute to minute control of serum iCa is influenced primarily by PTH and its essential role is to increase plasma Ca concentrations. The major stimulus for production and release of PTH is a decrease in plasma Ca concentrations. PTH exerts direct effects on bone and kidney and indirect effect on the gastrointestinal tract through calcitriol. PTH stimulates osteoclast activated release of Ca from bone (phosphorus is released as well). At the kidney, PTH increases renal reabsorption of Ca in the loop of Henle and distal convoluted tubule, increases activation of 25-hydroxycholecalciferol to the calcitriol (1-25dihydroxycholecalciferol), and increases excretion of phosphorus by decreasing its tubular reabsorption.

Calcitriol (1,25-hydroxycholecalciferol)
Calcitriol is the active form of vitamin D and its production is stimulated by PTH as well as decreased concentrations of phosphorus, Ca, and calcitriol itself. Calcitriol promotes absorption of Ca and phosphorus from the gut and resorption of Ca and phosphorus from bone. Cats ingest cholecalciferol from food (but it has limited biologic activity – it must be hydroxylated). Hepatic hydroxylation produces 25-hydroxycholecalciferol and then renal hydroxylation, which requires α 1-hydroxylase, produces calcitriol.

Calcitonin
Calcitonin’s primary role is limiting the degree of postprandial hypercalcemia. Together with PTH, calcitonin maintains iCa within its normal narrow physiologic range. At physiologic concentrations, calcitonin has limited biologic potency and even maximal secretion will not correct a sustained hypercalcemic state. At high doses, calcitonin may promote urinary Ca excretion.

Fibroblast growth factor-23
Fibroblast growth factor-23 (FGF-23) is a recently characterized “phosphatonin” hormone whose primary action appears to be regulation of phosphate. FGF-23 is secreted primarily from bone in response to increased plasma phosphate and calcitriol concentrations. Within the kidney FGF-23 causes down-regulation of phosphate transporters and vitamin D synthesis. These actions result in increased renal phosphate excretion and reduced renal calcitriol formation. In the parathyroid gland, FGF-23 decreases PTH production and secretion. FGF-23 concentrations are increased in cats with early stage chronic kidney disease and via the actions described above, help maintain normophosphatemia.

Effects of hypercalcemia in cats
Gastrointestinal signs are common and may include anorexia, vomiting, constipation (perhaps associated with decreased gut motility). Pancreatitis has also been observed in cats with experimentally induced hypercalcemia. Hypercalcemia will result in a calciuresis which can predispose towards the formation of calcium oxalate (CaOx) urolithiasis (iCa concentrations should always be assessed in cats with CaOx uroliths). Renal damage may occur if the Ca X phosphorus product is > 60-70 mg/dl. Increases in iCa concentration may also have adverse effects on renal tubules independent of soft tissue mineralization. Severe increases iCa concentration may result in neuromuscular/CNS dysfunction and cardiac arrhythmias.
Differential diagnosis: “gosh darn it” eponym
G: Granulomatous disease (granulomatous disease: 1 case in literature – SQ Nocardia pyogranulomatous mass in 6 year old CM DSH; O: Osteolytic; S: Spurious, supplements, schistosomiasis; H: Hyperparathyroidism, humoral hypercalcemia of malignancy, houseplants (calcitriol glycosides e.g., Cestrum, Solanum, Triestum), hyperthyroidism; D: Vit D toxicosis, drugs, dehydration, diet; A: Addison’s, aluminum toxicity, Vit A, milk-alkali (excessive calcium carbonate ingestion); R: Renal disease, raisin/grapes (dogs); N: Neoplasia, nutritional; I: Idiopathic, infectious, inflammatory; T: Temperature, hyper- and hypothermia

Feline differential diagnosis for hypercalcemia: “shirt” eponym
- S: Spurious laboratory results
- H: Hyperparathyroidism
- I: Idiopathic
- R: Renal
- T: Tumors – usually related to the releases of a fetal protein (PTH-related protein) which mimics the effects of PTH

Diagnostic approach
Mild hypercalcemia in an asymptomatic patient should be confirmed with a subsequent biochemistry profile after a 12 hour fast. If the hypercalcemia is persistent or in cases with moderate/severe hypercalcemia, an iCa concentration should be assessed to confirm true hypercalcemia. Any other abnormalities on the minimum data base (e.g., azotemia, lymphocytosis) should be appropriately pursued. A “PTH panel” that includes tCa, iCa, PTH, and PTH-rp should be assessed. Finally careful cervical palpation may reveal a thyroid/parathyroid nodule and radiographs, ultrasound, bone marrow or mass aspirate cytology, and bone marrow or mass biopsy may also be indicated.

Primary hyperparathyroidism
Benign adenoma of one of the 4 parathyroid glands (Siamese cats may be over-represented) is the usual cause of primary hyperparathyroidism. A cervical mass may be palpable and PTH concentrations should be above reference range ([PTH] in normal range is still inappropriate in face of ↑ iCa). Surgical removal of adenoma is the treatment of choice.

Idiopathic hypercalcemia in cats
Most common cause of hypercalcemia in cats in US over the last 10 years is idiopathic hypercalcemia. Diagnosis is made by exclusion; hypercalcemia is usually mild to moderate ([tCa] < 15 mg/dl) and [PTH] is low, [Vitamin D] is normal, and [PTH-rp] is not detected. Serum phosphorus concentrations are usually normal unless there is impaired renal function. Many cats with idiopathic hypercalcemia appear clinically normal; some have non-specific signs (e.g., wt loss, vomiting, and constipation) but all affected cats are predisposed to CaOx urolithiasis.

Cats with tCa > 13 mg/dl, [iCa++] > 1.0 mg/dl above normal, Ca X phosphorus > 60 mg/dl, urolithiasis, or evidence of renal disease should probably be treated; however there is no consensus on treatment.

Diet
Initially acidifying diets were implicated as a potential cause of the hypercalcemia. Alkalinizing diets may help; generally they contain less Ca and phosphorus than do maintenance diets and they decrease calciuresis and therefore CaOx formation. High fiber diets may decrease gut transit to decrease Ca absorption but these diets generally contain more Ca. Vitamin D content of diets is difficult to determine and the same wet vs. dry diet may have different Ca/Vitamin D content.

Glucocorticoids may reduce iCa in some cases, but high doses may be necessary (> 10 mg/cat/day). The mechanism is probably decreased gut absorption of Ca. In one short term study glucocorticoids had no effect on calciuresis.

Bisphosphonates: block enzyme pathways in osteoclasts to decrease bone resorption but may be associated with adverse reactions (e.g., esophageal damage, nephrotoxicity).

Primary chronic kidney disease (CKD) and hypercalcemia
In many cats with CKD and concurrent hypercalcemia the increase in Ca is caused by high complexed Ca rather than increases in iCa (i.e., not true hypercalcemia). Some cats with Stage 3/4 CKD (10-30%) have increased iCa with increased PTH = (tertiary hyperparathyroidism). The cause of increased PTH is unclear; an altered set point for release has been hypothesized. Calcitriol treatment had no effect on [PTH] in one short-term study.

Control of hyperphosphatemia is the most important aspect of management. Up to 20% of cats with Stage 2/3 CKD develop mild increases in [tCa] when eating a renal diet (perhaps caused by decreased dietary phosphorus increasing gut absorption of Ca due to decreased chelation in the gut). This hypercalcemia often resolves when the cat is taken off the phosphorus restricted diet; first try a 50/50 mix of the renal diet with a senior diet and then if the hypercalcemia persists try a straight senior diet.
Tumors
Lymphoma and squamous cell carcinoma in the cat are the tumors most commonly associated with hypercalcemia. Typically the serum phosphorus is decreased and the PTH-rp is increased (although undetectable [PTH-rp] does not rule out neoplasia).
Hyperthyroidism is one of the most commonly diagnosed diseases of the older cat. Geriatric cats with hyperthyroidism may also have concurrent chronic kidney disease (CKD). Systemic hypertension, proteinuria, and urinary tract infection (UTI) can be consequences of either hyperthyroidism or CKD. Hyperthyroidism can increase glomerular filtration rate (GFR) in cats with CKD which can attenuate or resolve mild to moderate azotemia. In addition, serum creatinine may be decreased in cats with weight loss and decreased muscle mass. In both cases, reductions in BUN and serum creatinine concentrations make it more difficult to detect concurrent CKD. Conversely, the CKD may depress thyroid hormone concentrations (euthyroid sick syndrome) making it more difficult to diagnose hyperthyroidism. Initial treatment of hyperthyroid cats with azotemic CKD should ideally be accomplished with a reversible anti-thyroid medication in order to assess any adverse effects on renal function. Systolic blood pressure and urine protein creatinine ratio (UP/C) should be evaluated prior to and after treatment. Urine cultures should be obtained as part of the workup of both hyperthyroidism and CKD. In either case, a concurrent UTI should be managed as a complicated UTI with long-term antibiotic treatment based on culture and sensitivity results.

Clinical signs/physical examination
Classic clinical signs of hyperthyroidism include weight loss, polyuria/polydipsia (PU/PD), and polyphagia in an older cat. Fewer than 5% of cats with hyperthyroidism are less than 8 years of age; the average age at diagnosis is 12-13 years. A thyroid enlargement (thyroid slip) can often be palpated in hyperthyroid cats, although some euthyroid cats will also have enlargement of one or both glands. In cats with concurrent CKD, kidneys may be small and/or irregular. Approximately 50% of cats with hyperthyroidism will exhibit PU/PD. A primary polyuria may occur as a result of thyrotoxicosis increasing cardiac output and GFR as well as increased renal medullary blood flow which has the potential to decrease renal medullary hypertonicity and urine concentrating ability. Some cats with hyperthyroidism may also have a primary polydipsia secondary to the effects of high thyroid hormone concentrations on the thirst center. Regardless of the mechanism, decreased urine specific gravity makes interpretation of azotemia problematic (is it pre-renal azotemia superimposed on decreased concentrating ability or renal azotemia?). Systemic hypertension is another common finding in hyperthyroid cats. High blood pressure may be caused by increased cardiac output, sympathetic tone, and arteriolar resistance and if sustained, can lead to intraglomerular hypertension, glomerulosclerosis, and proteinuria. No matter what the underlying cause, hypertension can damage the eyes, brain, heart, and kidney of affected cats. For example, tachycardia murmurs, and gallop rhythms may be associated with hypertrophic cardiomyopathy. Similarly, whether proteinuria arises from hypertension or CKD, progressive renal disease is a potential consequence.

Increased practitioner awareness of hyperthyroidism, an increasing population of geriatric cats, and increased diagnostic testing of older cats (wellness exams) has resulted in earlier diagnosis of hyperthyroidism in many cases. Clinical signs in these cases may be more subtle compared with an advanced case of hyperthyroidism. With earlier diagnosis, weight loss may be present but emaciation will be less likely and body condition scores will be higher. Similarly, PU/PD is less likely to be observed by owners and appetite and activity levels may be only slightly increased in cats with early hyperthyroidism.

Clinicopathologic findings associated with hyperthyroidism may include a slight erythrocytosis; perhaps secondary to increased tissue oxygen consumption. Serum ALT is increased in approximately 75-90% of cats with hyperthyroidism and is thought to be associated with malnutrition, hepatic hypoxia, and/or toxic effects for thyroid hormone on hepatocytes. Azotemia is observed in approximately 25% of hyperthyroid cats and may be due to dehydration, increased protein turnover (BUN), and/or CKD. Concurrently, urine specific gravity is often decreased as discussed previously.

Diagnosis
The best screening test for the diagnosis of hyperthyroidism is the total T4 (TT4) concentration. An increased TT4 is specific for hyperthyroidism however false negative results may occur with non-thyroidal illness (e.g., CKD). In cats with compatible clinical signs of hyperthyroidism that have a TT4 in the normal range, repeating the test in two weeks is usually the first recommendation. If results are still in the normal range on the second test, a free T4 (fT4) (measured by equilibrium dialysis) may be assessed. In comparison to the TT4, the fT4 is more sensitive but may result in more false positive results. A low normal TT4 with a high fT4 is more suggestive of non-thyroidal illness, whereas and high normal TT4 with an increased fT4 suggest hyperthyroidism (especially with compatible clinical signs). Rarely a T3 suppression test may be employed to help confirm a diagnosis. Nuclear scintigraphy compares the uptake of technetium-99m by the thyroid to the salivary glands (a normal thyroid to salivary gland ratio is approximately 1:1). Nuclear scintigraphy is both sensitive and specific and it is considered the gold standard diagnostic test.


**Pre-treatment evaluation**

Several studies have demonstrated that GFR declines with treatment for hyperthyroidism; this decline is independent of the treatment modality (medical, surgical, radioactive iodine). This decrease in GFR should be considered a consequence of the resolution of the hyperthyroid state and not a side effect of the treatment itself. The potential for a decrease in GFR to adversely affect the patient’s quality of life suggests that a thorough pre-treatment evaluation of the heart (potential pre-renal effects) and kidneys is important. The goal is to identify patients that may be harmed by permanent treatment of their hyperthyroidism.

Thoracic radiographs to assess the cardiac silhouette should be performed to rule out cardiomyopathy (especially in cases with a murmur or gallop rhythm). Any changes in the cardiac silhouette should be further evaluated with echocardiography. A baseline blood pressure should be determined and a complete minimum data base (CBC, serum biochemistry profile, UA) obtained. Although it is thought to be a better positive prognostic indicator than minimally concentrated urine, hypersthenuric urine specific gravity (>1.035-1.040) prior to treatment does not guarantee adequate renal function post treatment. Combining urine specific gravity and TT4 concentration was helpful in predicting post-treatment azotemia in one study (USG < 1.035 and TT4 > 7.8 µg/dL together were poor prognostic indicators). The urine dipstick and sulfosalicylic acid assays are unreliable screening tests for proteinuria in cats and therefore a feline specific albuminuria assay and/or a urine protein/creatinine ratio (UP/C) should be utilized to quantitate proteinuria/albuminuria. Persistent proteinuria of renal origin is a poor prognostic indicator for progression of CKD in cats; however pre-treatment proteinuria does not appear to be predictive of post-treatment azotemia in hyperthyroid cats. Urinary excretion of NAG was also not predictive of post-treatment azotemia in hyperthyroid cats. Urine cultures are recommended in all cases but are required in cats with pyuria and/or hematuria; UTI occurs in 12-22% of hyperthyroid cats and 13-30% of CKD cats and with both underlying diseases, the infection is often clinically silent. Pre-treatment GFR measurement may be a useful predictor of post-treatment renal function. Although exceptions exist, pre-treatment GFR values > 2.25-2.5 ml/min/kg body weight are thought to predict adequate post-treatment renal function. Finally in cats with suspected CKD, renal imaging with ultrasound and/or radiographs is recommended to further assess kidney tissue architecture and help rule out ascending infections, uroliths, and renal infiltrative disease. In cats that have obvious CKD and/or severe hypertension (> 180 mmHg), these issues should be addressed prior to treatment of the hyperthyroidism.

**Effects of treatment of hyperthyroidism on renal function**

Regardless of the modality utilized (thyroidectomy, methimazole, I131), successful treatment of hyperthyroidism decreases renal excretory function, resulting in an increase in the serum creatinine concentration and a decrease in the GFR. The major changes in serum creatinine concentration and GFR occur within the first month post-treatment and then renal function tends to stabilize. Although renal function tends to stabilize after 30 days, it is advisable for clinicians to monitor serum creatinine concentration for at least 6 months after the cat has become euthyroid. Importantly, it has been shown that cats that develop post-treatment azotemia do not have decreased survival times compared with hyperthyroid-treated cats that remain nonazotemic. However, cats with azotemia prior to initiation of treatment for hyperthyroidism appear to have decreased survival compared with cats that become azotemic following treatment.

**Effects of hypothyroidism on renal function**

Just as hyperthyroidism tends to increase renal function, hypothyroidism tends to decrease renal function. Diminished GFR could have significant consequences for hyperthyroid cats with preexisting renal disease that become hypothyroid as a result of treatment. Cats with iatrogenic hypothyroidism are not only more likely to develop azotemia, but hypothyroid cats with azotemia also have decreased survival. Supplementing cats with iatrogenic hypothyroidism secondary to I131 with thyroid hormone, or reducing the dosage of anti-thyroid medication, to achieve a euthyroid state, may improve renal function. Identifying and treating cats with iatrogenic hypothyroidism therefore is important. Inasmuch as hypothyroidism may not occur for as long as 3-6 months after radioiodine treatment, monitoring total T4 for at least 6 months post-treatment should be accomplished. A low total T4 concentration alone is not sufficient for diagnosis of iatrogenic hypothyroidism because euthyroid sick syndrome may be present. The combination of reduced total T4 concentration and elevated TSH concentration is consistent with iatrogenic hypothyroidism and thyroxine supplementation or adjustment of antithyroid medication should be considered.

**Summary**

Successful treatment of hyperthyroidism has the potential to unmask pre-existing CKD, but the associated changes in renal function are usually mild and renal function largely stabilizes within 1-2 months of the hyperthyroid treatment. Overall survival of those cats that do become azotemic does not differ from non-azotemic cats. Therefore, treatment of hyperthyroidism is recommended with the target total T4 in the lower half of the reference interval, without creating hypothyroidism. Increases in serum creatinine concentrations may occur over several months post-treatment (due to decreases in GFR and increases in muscle mass), so monitoring renal function for 6 months following restoration of euthyroidism is recommended. When treating cats with evidence of CKD prior to treatment, the decreased survival times associated with pre-therapy CKD should be discussed with owners, and continued monitoring
of renal function for months following return euthyroidism is necessary. Due to the increased risk of azotemia and poor prognosis in cats with iatrogenic hypothyroidism, total T4 (and TSH when appropriate) concentrations should be monitored for at least 6 months after euthyroidism is achieved, and iatrogenic hypothyroidism should be corrected via adjustment of anti-thyroid medication or thyroid supplementation if necessary.
Chronic kidney disease (CKD) is a common problem that adversely affects both quality of life and survival time. Although the prevalence of CKD in the general small animal population is ill-defined, CKD may affect up to 10% of dogs and 35% of cats in referral hospital populations (Polzin and Osborne 1986; Krawiec and Gelberg, 1989). Neophron damage associated with CKD is usually irreversible and can be progressive. Renal failure results when three-quarters or more of the nephrons of both kidneys are not functioning. Whether the underlying CKD primarily affects glomeruli, tubules, interstitial tissue, or renal vasculature, irreversible damage to any portion of the nephron renders the entire nephron nonfunctional. Healing of irreversibly damaged nephrons occurs by replacement fibrosis and therefore a specific etiology is often not determined. Chronic kidney disease occurs over a period of weeks, months, or years and since it is often not possible to improve renal function in CKD, treatment is aimed at stabilizing renal function. In addition to dietary therapy, there is increasing evidence that treatment with ACE inhibitors can decrease the progressive nature of CKD by attenuating systemic hypertension, intraglomerular hypertension, and proteinuria.

By altering pre-glomerular resistance, healthy kidneys can maintain relatively stable glomerular capillary pressures despite variations in systemic blood pressure. This pressure regulatory process is termed “renal autoregulation”. Autoregulation can be reduced when renal disease results in loss of nephrons. Compromised autoregulation allows high systemic blood pressure to be transmitted to glomerular capillaries. This glomerular hypertension has been documented by micropuncture studies in dogs and cats with surgically reduced renal mass. In these models, glomerular hypertension was associated with glomerular hypertrophy, sclerosis, and proteinuria. Systemic hypertension is relatively common in dogs with renal disease. In a recent study of dogs with spontaneous chronic kidney disease (CKD), 29/45 (64%) had systolic blood pressure ≥ 144 mm Hg and 14/45 (31%) had systolic blood pressure ≥ 161 mm Hg. In cats with naturally-occurring CKD, systemic hypertension has been observed in 19-65% of cases depending on the definition of hypertension.

Renal proteinuria can result from glomerular and/or tubular abnormalities in dogs and cats with CKD. Glomerular proteinuria may arise from immune complex disease or structural abnormalities involving the glomerular capillary wall (e.g., amyloidosis and x-linked hereditary nephropathy). Protein-losing nephropathy caused by glomerular capillary wall lesions is often accompanied by systemic hypertension and glomerular proteinuria can be exacerbated by intraglomerular glomerular hypertension that can result from systemic hypertension. Tubular proteinuria occurs when tubular reabsorption of protein from the glomerular filtrate is compromised. Whether caused by capillary wall lesions, tubular lesions, or intraglomerular hypertension, excessive quantities of protein in the glomerular filtrate may contribute to additional glomerular and tubulointerstitial lesions leading to loss of more nephrons.

**Diagnosis of hypertension and proteinuria**

Current recommendations are that blood pressure be measured in a quiet area prior to examining the patient, typically in the presence of the owner and after a 5-10 minute period of acclimation. The ACVIM Panel on Hypertension suggests discarding the first measurement, then obtaining a minimum of 3, preferably 5-7, consecutive measurements with less than 10-20% variability in systolic blood pressure. The animal’s disposition, body position, and heart rate, the cuff size and measurement site as well as all measured values should be recorded in the medical record. Many clinicians suggest that hypertension be documented on more than one occasion before accepting the diagnosis (unless ocular lesions compatible with systemic hypertension already exist).

Diagnosis and management of proteinuria in cats and dogs with CKD should be accomplished in a step-wise fashion. The specificity of the dipstick screening test for proteinuria is poor and therefore confirmation of traditional dipstick positive proteinuria should be accomplished with a more specific follow-up test such as the sulfosalicylic acid (SSA) turbidimetric test, UP/C, or species specific albuminuria assay. The second step is assessment of proteinuria is to determine its origin. Renal proteinuria can adversely affect the prognosis of dogs and cats with CKD and therefore, physiologic or benign proteinuria and pre- and post-renal proteinuria should be ruled out. Subsequently, via serial monitoring, it should be determined if the proteinuria is persistent or transient and if persistent – is the magnitude stable or increasing or decreasing over time? Persistent proteinuria is defined as at least two positive tests at two week intervals. Relatively mild proteinuria in dogs and cats with spontaneous/naturally-occurring CKD appears to be a negative predictor of survival. In dogs and cats with the remnant kidney model of CKD, proteinuria is associated with nephron hypertrophy and increased intraglomerular pressures. Persistent proteinuria of renal origin of a magnitude ≥ UP/C of 0.4 in cats and ≥ 0.5 in dogs with azotemic CKD should be treated with an ACEI and/or dietary protein reduction. Borderline proteinuria is defined as a UP/C between 0.2 and 0.5 in dogs and 0.2 and 0.4 in cats.
What evidence exists that systemic hypertension and/or proteinuria are detrimental to canine and feline kidneys?


8. In dogs with naturally occurring CKD, the relative risk of uremic crises and mortality was approximately three time greater in dogs with UP/C’s > 1.0 (n=25) compared with dogs with UP/C’s < 1.0 (n=20). In this study the risk of an adverse outcome was approximately 1.5 times greater for every 1 unit increase in UP/C and the decline in renal function was greater in dogs with higher UP/C’s. Jacob F, et al. Am J Vet Res 2005;226:393.

9. In cats with naturally occurring CKD, relatively mild proteinuria (UP/C’s > 0.4) appear to be negative predictors of survival. Increasing proteinuria was associated with increasing serum creatinine concentrations and increasing systolic blood pressure (presumably related to glomerular hyperfiltration). UP/C, age, and serum creatinine concentration (but not blood pressure) were independently associated with mortality. Syme HM, et al. J Vet Intern Med 2006;20:528.

10. Proteinuria has been associated with increased risk of mortality due to all causes in cats that have normal renal function when their proteinuria is first detected. Walker D, et al. J Vet Intern Med 2004;18:417.

11. In 141 client-owned cats with naturally-occurring systemic hypertension, amlodipine treatment decreased both blood pressure and proteinuria. Proteinuria (UP/C) (before and after treatment as well as the change in UP/C was the only variable related to survival in these cats. Jepson RE, et al. J Vet intern med 2007;21:402.


14. In a prospective, longitudinal cohort study of non-azotemic cats ≥ 9 years, 95 cats (median age = 13) were followed for 12 months or until death or azotemia developed. 29/95 (30.5%) developed azotemia (Sr Cr > 2.0 mg/dl). Proteinuria at presentation (median UP/C of 0.19 vs. 0.14) was significantly associated with development of azotemia. Jepson RE, et al. JVIM 2009; 23:806-813.

15. In cats with the remnant kidney model of CKD, hypertension was associated with more severe glomerular histologic lesions. Mathur, et al, AJVR 2004;65:1006-1013.

16. In 59 cats with CKD (serum creatinine concentrations > 2.0 mg/dl, USG < 1.035, and history and clinical signs compatible with CKD vs. AKI) with postmortem data and biochemistry and urine protein data (obtained during the last 2 months of life) the UP/C was positively correlated with both renal interstitial fibrosis score and maximal glomerular volume. 34 of the 59 cats (58%) were classified as proteinuric. Chakrabarti, et al. Diagnostic Pathol 2013;50:147-155.

17. When cats with CKD surviving for more than one month (surviving group, n = 34) were compared with cats with CKD surviving less than one month (non-surviving group, n = 16), UP/C was significantly higher in the non-surviving group. In the surviving group, UP/C was the only clinicopathologic variable that exhibited a consistent alteration (increase) in relation to first visit data and was most likely to be associated with mortality. Kuwahara, et al. J Small Anim Pract 2006;47:446-450.

18. When client-owned cats with stable CKD (n=112) were compared with client-owned cats with progressive CKD (n=101), median UP/Cs in the progressive group were higher than the stable group (0.27 vs. 0.14). A 0.1 increase in UP/C was associated with a 24% increase in risk of progression of CKD. Chakrabarti, et al. J Vet Intern Med 2012; 26:275-281.
19. In 69 cats with CKD (serum creatinine concentrations > 2.0 mg/dl, USG < 1.035, and history and clinical signs compatible with CKD vs. AKI) with postmortem data and time-averaged systolic blood pressure (SBPOT) (obtained over a mean of 284 days) the SBPOT was positively correlated with maximal glomerular volume, hyperplastic arteriolosclerosis, and glomerulosclerosis. 34 of the 69 cats (49%) were classified as hypertensive (SBPOT of 159 mm Hg vs 136 mm Hg in normotensive cats). Chakrabarti, et al. *Diagnostic Pathol* 2013;50:147-155.

NSAIDs in Dogs with Liver and Kidney Disease
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Chronic kidney disease (CKD) is a common problem that affects an estimated 0.5 to 7% of dogs. Radiographic signs of osteoarthritis (OA) occur in 20% of dogs. The majority of OA and CKD are acquired and both conditions are more prevalent in older dogs. Use of non-steroidal anti-inflammatory drugs (NSAIDs) has dramatically improved the quality of life for many dogs with OA. The potential nephrotoxicity of NSAIDs however make their use problematic in dogs with CKD. Thorough evaluation of renal function prior to the use of NSAIDs and follow-up monitoring for any adverse effects on renal function is extremely important in the older dog. Newer evidence suggests that the cyclooxygenase (COX) II enzyme is important in maintaining renal blood flow (RBF) in dogs and therefore, COX II selective/specific NSAIDs at least have the potential to adversely affect renal function in dogs. In contrast, the hepatotoxicity associated with NSAIDs in dogs appears to idiosyncratic and unrelated the COX selectivity of the drug.

Potential nephrotoxicity of NSAIDs
Renal damage and disease can be caused by acute or chronic insults to the kidney. The terms renal disease and renal damage are used to denote the presence of renal lesions; these terms however imply nothing about renal function or the cause, distribution, or severity of the renal lesions. Acute kidney injury (AKI) often results from ischemic or toxic insults and usually affects the tubular portion of the nephron. Early detection of AKI facilitates appropriate intervention that can arrest or at least attenuate tubular cell damage and the development of established acute renal failure (ARF). In contrast, nephron damage associated with CKD is usually irreversible and can be progressive. Pre-existing CKD increases the risk of AKI associated with the use of potentially nephrotoxic drugs.

Renal prostaglandins help regulate RBF and glomerular filtration rate (GFR), renin release, and sodium excretion. Potential adverse effects of renal prostaglandin inhibition with NSAIDs can include decreased RBF and GFR, hypertension, salt retention and edema. Since both COX-1 and COX-2 enzymes are present/expressed in the canine kidney, any NSAID, regardless of its COX specificity or sparing properties, has the potential to produce adverse renal effects. In particular, dogs express higher basal levels of COX-2 in the kidney than some other species and may be uniquely sensitive to the nephrotoxic effects of COX-2 selective drugs. Although a number of studies have shown no adverse effects of the commonly used NSAIDs in dogs with normal kidneys, increased BUN and creatinine are common adverse events listed for NSAIDs at the FDA Adverse Drug Event website. Dogs in field trials of deracoxib and firocoxib had increased BUN at the end of the trials, while dogs treated with etodolac did not. In cases where RBF is decreased (e.g., dehydration and decreased cardiac output), the vasodilatory effects of renal prostaglandins are critical in the maintenance of renal perfusion and the potential for adverse effects associated with NSAID use is increased. There is also concern that patients treated with drugs that can decrease GFR (such as angiotension-converting enzyme (ACE) inhibitors) may have increased renal toxicity when treated with NSAIDs. Studies of elderly human patients have confirmed this effect, but in a study of normal dogs treated with enalapril and tepoxalin no alteration of GFR was noted.

Risk factors for acute kidney injury
Dehydration and volume depletion are perhaps the most common and most important risk factors for development of AKI/ARF. Hypovolemia not only decreases renal perfusion which can enhance ischemic damage, but also decreases the volume of distribution of potentially nephrotoxic drugs. In addition to hypovolemia, renal hypoperfusion may be caused by decreased cardiac output, decreased plasma oncotic pressure, increased blood viscosity, systemic hypotension, and decreased renal prostaglandin synthesis. Any of these conditions can increase the risk of AKI associated with the use of NSAIDs.

Pre-existing renal disease can increase the potential for nephrotoxicity and ischemic damage by several mechanisms. The pharmacokinetics of potentially nephrotoxic drugs can be altered in the face of decreased renal function. Animals with renal insufficiency also have reduced urine concentrating ability and, therefore, decreased ability to compensate for prerenal influences. Renal disease may also compromise the local production of prostaglandins that help maintain renal vasodilatation and blood flow. Age has been identified as a risk factor because many geriatric dogs have pre-existing renal lesions and sub-clinical loss of renal function.

Use of NSAIDs in dogs with chronic kidney disease
In dogs with pre-existing renal disease, the use of NSAIDs has the potential to exacerbate the renal disease and further decrease renal function and therefore NSAIDs should be avoided whenever possible in such animals. Hypertension and proteinuria associated with CKD are negative prognostic indicators and the potential for NSAID adverse effects may be increased in dogs with these complications. Certainly, the more advanced the stage of CKD, the greater the relative contraindication for the use of NSAIDs. It is important to remember that as early as stage II, > than 75% of the patient’s nephrons are no longer functional and the patient’s ability to auto-regulate RBF is compromised.

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Recommendations surrounding the use of NSAIDs in CKD patients are largely speculative, but practical suggestions include:

1. Maintain good hydration in these patients at all times
2. Increase the monitoring of these patients for early signs of AKI
3. Increase the monitoring of these patients for hypertension.
4. Use the lowest efficacious dose of a NSAID
5. Use analgesic drugs with less renal toxicity in place of NSAIDs
6. Monitor quality of life indices on a regular basis. In people, small stable increases in BUN and creatinine are often tolerated in rheumatoid and osteoarthritis patients on NSAIDs, because no other drugs maintain adequate quality of life.

Early recognition of acute kidney injury
Numerous urine parameters can herald the development of AKI in patients with initially normal renal function. The value of monitoring these parameters in CKP patients receiving NSAIDs has not been assessed. Increased urine turbidity or changes in urine sediment (increasing numbers of renal epithelial cells or cellular or granular casts) may be indications of AKI. The acute onset of tubular glucosuria (normoglycemic glucosuria) or the acute onset or increases in proteinuria may also be indicative of AKI. The interpretation of these urine parameters is enhanced by knowledge of baseline values.

Detection of enzymes in the urine such as gamma-glutamyl transpeptidase (GGT) and N-acetyl-beta-D-glucosaminidase (NAG) has proven to be a sensitive indicator of early AKI. These enzymes are too large to be normally filtered by the glomerulus, and, therefore, enzymuria indicates cell leakage, usually associated with tubular epithelial damage or necrosis. Urinary GGT originates from the proximal tubule brush border and NAG is present in proximal tubule lysosomes. In studies of gentamicin-treated dogs, increased urinary GGT and NAG activity was one of the earliest markers of renal damage/dysfunction. Interpretation of enzymuria is aided by baseline values obtained prior to a potential renal insult; 2 to 3-fold increases over baseline suggest significant tubular damage. Urine enzyme/creatinine ratios have been shown to be accurate in dogs prior to the onset of azotemia obviating the need for time urine collections.

Potential hepatotoxicity of NSAIDs
Hepatocellular toxicosis associated with administration of carprofen has been reported in a retrospective study of 21 dogs. The most common clinical signs were anorexia, vomiting, lethargy, and diarrhea. All of the dogs had elevations in serum ALT, 20 had elevations in serum ALP, and 18 were hyperbilirubinemic. The elevations in serum ALT were greater than the elevations in serum ALP in 16 dogs. Eighteen of the dogs had histologic evidence of hepatic necrosis characterized by multifocal to extensive vacuolar change, lytic necrosis, and apoptosis. It’s interesting to note that 7 of 9 dogs evaluated by urinalysis had changes ranging from mild to severe proteinuria, normoglycemic glucosuria, and/or renal epithelial cells and casts in the urine sediment. Four of the affected dogs died or were euthanized within 3-5 days of presentation. The authors speculated that an idiosyncratic hepatopathy that may have been caused by the interaction of glucuronide metabolites of the acidic NSAID with plasma and hepatocellular proteins; resulting in the formation of antigenic NSAID-altered proteins causing immune-mediated damage to the liver.

Early recognition of hepatic damage
NSAID-associated hepatotoxicity appears to be a sub-acute toxicity (within 2-3 weeks of initiation of treatment) and unrelated to the type (COX selectivity) of the NSAID. Baseline liver enzyme values should be established prior to treatment. Post-treatment, any anorexia, vomiting, lethargy, or diarrhea should prompt NSAID discontinuation and re-assessment of liver enzymes compared to baseline values. In dogs that remain clinically normal, reassessment of liver enzymes should be performed between 2-3 weeks after the NSAID treatment is started. If pre-treatment assessment reveals mild to moderate elevations in liver enzymes, pre- and post-prandial bile acid concentrations should be measured. Mild to moderate pre-treatment elevation in liver enzyme in the absence of liver dysfunction is probably not a risk factor for NSAID-associated hepatotoxicity. In dogs with reduced liver function (e.g., abnormal bile acids or hyperbilirubinemia), NSAIDs should be avoided, if at all possible.

Recommendations surrounding the use of NSAIDs in patients with decreased liver function are largely speculative, but practical suggestions include:

1. Maintain good hydration in these patients at all times
2. Increase the monitoring of these patients for early signs of hepatotoxicity
3. Use the lowest efficacious dose of a NSAID/increasing the interval between doses
4. Use alternative analgesic drugs in place of NSAIDs
Most bacterial infections of the lower urinary tract respond quickly to antimicrobial treatment; however, urinary tract infections (UTI) associated with defects in the host immune system (complicated UTI) often fail to respond or recur after antibiotic withdrawal and can be a therapeutic challenge.

**Etiology**

The most common bacterial pathogens associated with UTI in the dog include *Escherichia coli*, *Klebsiella*, *Staphylococcus*, *Enterococcus*, *Proteus*, *Pseudomonas*, *Enterobacter*, and *Streptococcus*. These are dermal or intestinal floras that ascend the urethra and then adhere to the mucosa of the bladder and multiply. Although many enteric organisms are anaerobes, the oxygen tension in urine probably inhibits growth of strict anaerobic bacteria and therefore, anaerobic UTI is rare. A recent study of recurrent and persistent UTI in dogs showed that 25% of culture positive urine specimens had two or more bacterial species isolated. Mycoplasmal infections are relatively rare but have been associated with recurrent or persistent UTI in dogs. Mycoplasma should be considered in dogs with persistent pyuria and negative urine culture, dogs with persistently alkaline urine and negative urine cultures, and dogs with persistent or recurrent UTI that don’t respond to appropriate conventional antibiotic treatment.

**Normal host defense mechanisms**

The status of host defense mechanisms appears to be the most important factor influencing the pathogenesis of UTI. Normal voiding is an efficient natural defense mechanism against UTI. Mechanical washout as a result of complete voiding is responsible for removing greater than 95% of non-adherent bacteria that gain entrance into the urinary bladder. Increased urine production and frequency of voiding enhance washout of bacteria. Disorders that decrease the frequency and/or volume of voided urine, or that result in an increased urine residual volume may predispose animals to UTI. Normal urine residual volume for dogs is less than 0.2 to 0.4 ml/kg body weight.

Bacteria are normally present in increasing numbers from the mid to distal urethra, but seldom do these organisms cause UTI in normal dogs. The high-pressure zone in the mid urethra and spontaneous urethral contractions help prevent ascension of bacteria. Differences in epithelial morphology (decreased epithelial receptor sites) also help decrease bacterial colonization in the proximal and mid urethra. The length of the urethra and bactericidal prostatic secretions in male dogs are thought to decrease the incidence of UTI compared with female dogs, however, nearly equal gender distribution in recurrent/persistent UTI has recently been reported. In both sexes, the valve-like nature of the vesicoureteral junction helps protect against bacterial ascension to the upper urinary tract.

Colonization of vulval and preputial luminal mucous membranes by nonpathogenic flora serves to decrease colonization by uropathogens. Normal flora occupy most of the epithelial receptor sites, produce bacteriocins that interfere with uropathogen metabolism, and have a high affinity but low requirement for essential nutrients required by uropathogens. Mucosal secretions also help prevent adherence of uropathogens to epithelium; immunoglobulins coat pathogenic bacteria and glycosaminoglycans form a protective barrier over the mucosal surface.

The antibacterial property of urine is an additional important host defense mechanism against UTI. Urine is frequently bacteriostatic and sometimes can be bactericidal depending on its composition. Low pH and high concentrations of urea and weak organic acids in concentrated urine inhibit bacterial growth. Although polyuric disorders may increase washout of non-adhered bacteria from the bladder, UTI may occur due to decreased antibacterial properties of urine.

**Complicated vs. uncomplicated UTI**

Uncomplicated UTI are infections without detectable underlying structural or functional abnormalities in the host's defense mechanisms. This form of infection is easiest to treat and is usually cleared soon after appropriate antibiotic treatment is initiated. Complicated UTI are associated with a defect in the host's defense mechanisms; i.e., interference with normal micturition, anatomic defects, damage to mucosal barriers, alterations in urine volume or composition, or systemic immunocompromise. In almost all cases, the underlying defect must be corrected in order to eliminate the UTI.

Abnormal micturition often results in incomplete voiding and retention of urine, which allows for multiplication of bacteria within the urinary tract. Damage to mucosal barriers may result in UTI, depending on the extent of the lesion and concurrent introduction of uropathogens. It is interesting to note that pathogenic bacterial inoculation of the urinary bladder in experimental animals usually fails to establish a UTI unless the uroepithelium is first damaged by a chemical or mechanical insult. Any time the urinary bladder is catheterized; bacteria are carried up the urethra to the bladder. If the catheter is inserted too far and damages the bladder mucosa, the chance of infection increases. Anatomic defects may allow ascending migration of bacteria (e.g., indwelling urinary catheters or an
ectopic ureter) or may damage mucosal barriers (e.g., urolithiasis, neoplasia, urachal remnant, thickened bladder wall due to chronic inflammation). Altered urine composition (glucosuria or excretion of irritating drugs like cyclophosphamide) can enhance the environment for bacterial growth. In addition to the above local factors, systemic disorders such as renal failure, hyperadrenocorticism, prolonged steroid administration, neoplasia, and diabetes mellitus can result in complicated UTI. In a recent retrospective study, acrobic urine cultures from 159 dogs with diabetes mellitus yielded bacterial growth in 34 cases (21%).

Elimination of clinical and laboratory signs of complicated UTI with antibiotic treatment is usually not possible; signs either persist during antibiotic treatment or recur shortly after antibiotic withdrawal. Although antibiotic treatment is the cornerstone of management, the status of host defense mechanisms is thought to be the single most important determinant of the outcome of UTI treatment. In complicated UTI, antibiotic treatment should control the pathogenic bacterial growth for a period sufficient to allow host defense mechanisms to prevent colonization of the urinary tract without further antibiotic administration.

**Recurrent UTI**

Recurrence of clinical and laboratory signs of UTI can be classified into two groups: relapses and reinfections. Relapses are infections caused by the same species of bacteria usually within several days of cessation of treatment. In this case the previous antimicrobial treatment failed to eliminate the infection. Relapses may be due to use of improper antibiotic or dose, emergence of drug-resistant pathogens, or failure to eliminate predisposing causes that alter normal host defense mechanisms and allow the persistence of the bacteria. Urinary tract infections that relapse are frequently associated with a higher degree of antimicrobial resistance compared to the original infection. Relapses in male dogs may be caused by chronic prostatic infections.

On the other hand, recurrent UTI may be reinfections. In this case, the previous antibacterial treatment cleared the first infection and the urinary tract has subsequently become infected with another bacteria. In most cases the time between reinfections is greater than the time between relapses. Reinfections often indicate failure to eliminate predisposing causes that alter normal host defense mechanisms. Alternatively, reinfections may be iatrogenic (follow-up catheterization) or spontaneous. Reinfections with less invasive bacteria (e.g., *Pseudomonas aeruginosa*) generally suggest the host's immune system is compromised.

**Treatment**

It is important to try to identify those patients with immune system defects; therefore a complete physical examination should be performed on all animals that present with signs of UTI. If a simple UTI is suspected, bacterial sensitivity results are not available, antibiotic treatment choice should be based on bacterial identification or the gram-staining characteristics of the bacteria. Clinical experience at several different veterinary teaching hospitals indicates that intelligent choices may be made about bacterial susceptibility to antibiotics. Without benefit of bacterial sensitivity testing, the following are the drugs of choice for the bacteria listed: E. coli - enrofloxacin; *Proteus* - amoxicillin-clavulanic acid; *Staph* - amoxicillin-clavulanic acid; *Strep* - amoxicillin-clavulanic acid; *Enterobacter* - tetracyclines; *Klebsiella* - enrofloxacin; *Pseudomonas* - tetracycline. If bacterial identification is unknown, treatment is best based on the gram-staining characteristics, i.e., ampicillin/amoxicillin or amoxicillin-clavulanic acid for gram-positive bacteria and trimethoprim-sulfa or enrofloxacin for gram-negative bacteria. In cases of suspected or known complicated UTI or in cases of recurrent UTI, bacterial culture and sensitivity of the urine is necessary.

Cystocentesis is the preferred method of collection for urine culture and sensitivity. The urine sample should be submitted in a sealed container for culture as quickly as possible. Refrigeration is recommended if a delay in culturing is anticipated. Many practices inoculate a blood agar plate with urine and then submit the plate for identification and sensitivity if there is bacterial growth after incubation. Minimum inhibitory concentrations (MIC) and Kirby-Bauer agar diffusion tests can be used to determine bacterial sensitivity. The Kirby-Bauer method is acceptable for most UTI; however the MIC technique is often advantageous with apparently resistant UTI.

Steps to follow for management of a UTI are given in Table 1. The duration of therapy of lower UTI must be individualized and should be based on the cessation of clinical signs and elimination of abnormal urine sediment as well as a negative urine culture. In general uncomplicated lower UTI should be treated for 2 to 3 weeks, while complicated UTI should be treated for a minimum of 4 weeks. Verification of proper selection of antibiotic therapy can be made after three to five days of therapy, by assuring that the urine is sterile. The urine sediment, however, may be still abnormal at this time.

Recurrent UTI should always be evaluated by urine culture and sensitivity. Additionally, attempts should be intensified to identify defects in the host immune system. Double contrast cystography and ultrasonography may be used to rule out anatomic abnormalities and mucosal lesions of the bladder. In male dogs, semen and prostatic wash cytology and culture as well as ultrasonographic examination should be employed to rule out bacterial prostatitis. Excretory urography, ultrasonography, and renal biopsy may confirm the presence of pyelonephritis; however these parameters may be normal in chronic pyelonephritis. Finally, consideration should be given to the possibility of otherwise asymptomatic hyperadrenocorticism causing recurrent UTI, especially infections associated with low numbers of WBCs and RBCs in the urine sediment.
The prognosis for complicated UTI is always guarded in comparison to uncomplicated UTI. The single most important treatment for a complicated UTI is correction of the underlying defect in the host defense mechanisms. If predisposing factors cannot be identified or corrected, relapses and reinfections are common. For animals with frequent infections, which cannot be cured, low dose (1/3 to 1/2 of the conventional daily dose) antimicrobial administration at bedtime may be recommended after the urinary tract has been sterilized with standard dose antibiotic treatment. This allows the drug to be present in the bladder overnight supplementing the animal’s defense mechanisms. Low (sub therapeutic) dosages of antibiotic may reduce infections by interfering with bacterial fimbria production and therefore uroepithelial attachment. For recurrences due to gram-positive bacteria, penicillins are recommended; while for recurrences caused by gram-negative bacteria, trimethoprim-sulfa or enrofloxacin is recommended. It should be noted however, that long-term, sub therapeutic antibiotic treatment could predispose the animal to a resistant UTI. Any “break-through” UTI should be treated with therapeutic antibiotic dosages on the basis of bacterial culture and sensitivity.

Table 1. Steps to follow for management of urinary tract infections
- Diagnosis based on history, urine sediment, and ideally urine culture and sensitivity.
- Selection of an antimicrobial agent.
- Reculturing of urine in 3-5 days to ascertain effectiveness of selected antimicrobial agent.
- Examine urine sediment 3-4 days before discontinuing antibiotic treatment.
- Recheck urine 10 or more days following cessation of therapy.
- Recurrent urinary tract infections should be evaluated for underlying predisposing factors (e.g., contrast radiography, CBC, serum biochemistry profile, ACTH stimulation test).
- Frequent reinfections may need to be treated with prophylactic doses of antibiotics after initial inflammation has been cleared up with standard dose antibiotic treatment.
How to Help Owners with the Hardest Decisions: Assessing Quality of Life in Pets and Knowing when to Say Goodbye (Parts 1 and 2)

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Knowing when is “time” is not an easy thing to do unless a pet is in the active stage of suffering – at that point, it is usually clear to all involved. But most pet owners do not want to have their pet get to the point of extreme suffering. But when do you make that decision? This presentation will give attendees tool to help guide owners through the decision process to provide them with guidelines to do what is best for the pet and the family.

Melinda’s phone call to me started off as most of our calls do, with lots of heartfelt tears. It was clear Melinda needed support and additional education through this tough time. Chance, her 4 year old male Staffordshire Terrier, greeted me at the door for our in-home hospice consultation, clearly unconcerned that he has both severe mitral and tricuspid valve insufficiency, along with atrial fibrillation. Melinda understood the gravity of his condition and was well-coached by the cardiologist. Her most pressing issue however, as with most of our clients, is knowing When to make that final decision. It’s the most important question we are asked as doctors and although our clients want a specific timeline, more personalized patient and client information is needed to most comprehensively evaluate quality of life (QOL) and reach an educated, informed, and supported choice that fits not only their pet’s medical condition but also the family’s wishes. “Quality of Life” applies not only to the pet; it applies just as much to the family!

The most commonly used objective measurements for quality of life by veterinarians are mobility, appetite, pain, and proper voiding. I certainly do not disagree with any of these but the presence of quality of life based on these items should not be answered with a “yes or no,” but rather “if… then”.

There are numerous objective QOL scales available that do a wonderful job addressing these, and other, clinical signs of the pet but, in my opinion, leave out the other 50% of the equation; the family’s time, emotional, physical and financial budgets. This is why I always start hospice consultations with open-ended questions. I need to get an idea of what the family values most in their pet’s daily life, where their “stop point” is in relation to the pet’s disease condition, and what their idea of a “good death” is for their pet.

The goal is not to evaluate the QOL for the family (although I feel owners want and deserve my opinion) but rather to help them uncover their own thoughts, feelings, and boundaries for their pet surrounding end of life decisions. These questions help me gauge the family’s time, emotional, physical and (when appropriate, financial) budgets:

1. Have you ever been through the loss of a pet before? If so, what was your experience (good or bad, and why)? (Side bar: “Have you ever been through this before?” is usually the first thing I ask. I find that families experiencing quality of life evaluation for the first time generally need more hand-holding and more direct language about the process ahead. They tend to wait for that hand-written letter from their pet saying “I’m ready now, Mom.” This is not just my observation, it is what I hear from these pet owners time and again after the loss of their pet; “I can’t believe I waited that long.”

2. What do you hope the life expectancy of your pet will be? What do you think it will be?

3. What is the ideal situation you wish for your pet’s end of life experience? (at home, pass away in her sleep, etc.)

4. Do you hold any stress or anxiety about any of these issues? (This section is meant to help identify the main concerns the family has.)
   - Pet suffering
   - Desire to perform nursing care for pet
   - Ability to perform nursing care for pet
   - Pet dying alone
   - Not knowing the right time to euthanize
   - Coping with loss
   - Concern for other household animals
   - Concern for other members of the family (i.e., children)

After some discussion, it was clear Melinda most valued the physical companionship Chance brought her. He followed her everywhere, even when it was clear his breathing was labored. She was aware that his condition could deteriorate rapidly at any time, leading to death in minutes to hours at best (a condition I categorize as “imminent”). Knowing the significant anxiety that accompanies dyspnea and the happiness her presence brings him, Melinda placed great value on the quality of death for Chance. Her worst fear was coming home after work to find that he passed away on his own, not knowing if he was in pain or stress during that death phase. Melinda’s stop-point came a couple weeks later when Chance no longer followed her to the next room; she knew it was
time. She wanted to be with him and to lean on the support of family at that crucial moment, which is why we met at Chance’s favorite spot on the beach at sunset the next day to peacefully say good-bye.

Ideally, every family’s budgets and boundaries align with the disease process at hand. For Melinda it did, but this is not always the case. The family that places greatest weight on both the happiness of the pet in addition to avoiding an emergency situation at all costs needs to understand the significant risk they run by waiting too long with imminent conditions…. This determines what clinical signs should be weighted most heavily to evaluate quality of life. We have to start moving away from the standard “call me when he stops eating”! Appetite truly does not concern me for the 85 lb Labrador that has severe osteoarthritis. This dog may never stop eating and the family must not rely on this clinical sign to ever manifest itself. The little Yorkie with congestive heart failure that suddenly refuses food, however, definitely concerns me. Each disease process has it’s own set of clinical signs that should be weighted most heavily.

If the pet is declining in health and there are no additional diagnostics or treatments the family is either willing or able to explore, then quality of life is either an imminent concern or will be some point soon. If the family’s emotional, time, physical or financial budgets are being drained there is a subjective time period in which euthanasia is an appropriate decision to make. This period could be hours, days, weeks, or even months. Before this specific period, I will refuse to euthanize since there is clearly a good quality of life. After this period, however, I will insist on euthanizing due to suffering of the pet. During this larger subjective time however, it is truly dependent on the family to make whatever decision is best for them under the guidance of a supportive medical team. Some owners need time to come to terms with the decline of their pet while others want to prevent any unnecessary suffering at all. Everyone is different. After all, owners know their pet’s personality better than anyone, even the vet!

Chance was clearly a happy boy that loved his mom dearly, watching her every move and following her to the kitchen, just 15 feet from where I was sitting. Melinda, a 25 year old professional, found Chance in the Florida Everglades as a puppy during a college field trip. He grew up with Melinda during her first years as an adult and now helps her feel secure while living alone. She has given Chance the very best quality of life thus far but with such a life-limiting and condition, is facing the difficult and inevitable loss of her boy. Although tired and breathing more rapidly than normal, Chance is happy. He has no perception of what “heart failure” means and no emotional reaction to his physical condition. He is living in the moment (isn’t that what we love about our pets anyway!?). The drawback is that once in pain, animals cannot sense an ending to their hurt. As humans, we can take a pill knowing that the headache will eventually subside but animals have no perceptron of their suffering ending. This key point is at the heart of quality of life evaluation; how do we measure happiness and prolong it as long as possible.

**Pain and anxiety**

Pain in animals is another important topic that all pet owners should be well versed on. It’s the main topic I discuss during my in-home hospice consultations. Myself, and many other professionals, believe that carnivorous animals, such as cats and dogs, do not “hide” their pain, rather pain simply doesn’t bother them the same way it bothers humans. Animals do not have an emotional attachment to their pain like we do. Humans react to the diagnosis of cancer much differently than Fluffy does! Fluffy doesn’t know she has a terminal illness, it bothers us more than it bothers her. This is vastly different than prey animals like rabbits or guinea pigs, who must hide their pain to prevent carnivorous attacks. If you’re interested in learning more about pain and suffering in pets, grab Temple Grandin’s book “Animals in Translation” and read chapter 5.

When discussing the decision to euthanize, we should be just as concerned about anxiety in our pet as we are about pain. Personally, I feel that anxiety is worse than pain in animals. Think about the last time your dog went to the vet. How was his behavior? Was he nervous in the exam room? Did he give you that look that said “this is terrible!”? Now think back to when he last hurt himself. Perhaps scraping his paw or straining a muscle after running too hard. My dog rarely looks as distraught when she’s in pain as she does when she’s anxious. It’s the same for animals that are dying. End stage arthritis patients begin panting, pacing, whining, and crying, especially at night time. Due to hormonal fluctuations and other factors, symptoms can usually appear worse at night. The body is telling the carnivorous dog that he is no longer at the top of the food chain; he has been demoted and if he lies down, he will become someone else’s dinner. Anti-anxiety medications can sometimes work for a time but for pets that are at this stage, the end is certainly near.
Waiting too long
An interesting trend that I did not expect when starting my hospice practice is that the more times families experience the loss of a pet, the sooner they make the decision to euthanize. Owners experiencing the decline or terminal illness of a pet for the first time will generally wait until the very end to make that difficult decision. They are fearful of doing it too soon and giving up without a good fight. Afterwards, however, most of these owners regret waiting too long. They reflect back on the past days, weeks, or months, and feel guilty for putting their pet through those numerous trips to the vet or uncomfortable medical procedures that did not improve their pet’s quality of life. The next time they witness the decline of a pet, they are much more likely to make the decision at the beginning of the decline instead of the end.

What about a natural death?
Yes, there are those pets that peacefully fall asleep and pass naturally on their own, but just as in humans, this is rare. Many owners fear their pet “passing alone” while others do not. Occasionally I am asked to help families through the natural dying process with their pet. For different reasons, these families are against euthanasia. I explain everything I possibly can, from how a natural death may look, how long it may take, what their pet may experience, etc. Inevitably, almost all of these families regret doing this. Most of them comment afterwards “I wish I would not have done that, I wish she didn’t have to suffer.” A natural death can be difficult to watch, especially for non-medically oriented people. Most people can watch a human family member in pain much more easily than they can their pet. To an extent, we can talk other humans through physical pain or discomfort. Humans can perceive an ending to their pain (via medication or even death) but there is little emotional comfort we can offer a pet that is suffering, they simply cannot perceive an ending to that pain. Families take this guilt difficultly and I do my very best to not only readily suggest euthanasia when appropriate, but prepare families for a “worst-case” scenario should they chose to wait.

Weigh your options carefully
If the most important thing to you is waiting until the last possible minute to say goodbye to your baby, you will most likely be facing an emergency, stress-filled, sufferable condition for your pet. It may not be peaceful and you may regret waiting too long. If a peaceful, calm, loving, family-oriented, in-home end of life experience is what you wish for your pet, then you will probably have to make the decision a little sooner than you want. Making that decision should not be about ceasing any suffering that has already occurred, but about preventing suffering from occurring in the first place. Above all, our pet do not deserve to hurt.

I’ve heard from countless pet owners that the death of their pet was worse than the death of their own parents. This might sound blasphemous to some, but to others it’s the cold truth. Making the decision to euthanize a pet can feel gut-wrenching, murderous, and immoral. Yes, those are strong words, but that is what our pet families experience. They feel they are letting their pet down or that they are the cause of their friend’s death. They forget that euthanasia is a gift, something that, when used appropriately and timely, prevents further physical suffering for the pet and emotional suffering of the family. Making the actual decision is the hardest part of the experience and I’m asked on a daily basis, “Doc, how will I know when it’s time?” Let me shed some light on this difficult discussion.

Quality of life scale
When evaluating quality of life, personalized patient and client information is needed to reach an educated, informed, and supported choice that fits not only their pet’s medical condition but also the family’s wishes. In short, quality of life applies not only to the pet; it also applies to the family!

Pet’s quality of life
Score each subsection on a scale of 0-2:
- 0 = agree with statement (describes my pet)
- 1 = some changes seen
- 2 = disagree with statement (does not describe my pet)

1. Social Functions
   a. Desire to be with the family has not changed.
   b. Interacts normally with family or other pets (i.e., no increased aggression or other changes).

2. Natural Functions
   a. Appetite has stayed the same.
   b. Drinking has stayed the same.
   c. Normal urination habits.
   d. Normal bowel movement habits.
   e. Ability to ambulate (walk around) has stayed the same.
3. Mental Health
   a. Enjoys normal play activities.
   b. Still dislikes the same things. (i.e., still hates the mailman = 0, or doesn’t bark at the mailman anymore = 2)
   c. No outward signs of stress or anxiety.
   d. Does not seem confused or apathetic.
   e. Nighttime activity is normal, no changes seen.

4. Physical Health
   a. No changes in breathing or panting patterns.
   b. No outward signs of pain. (See Resources Below)
   c. No pacing around the house.
   d. My pet’s overall condition has not changed recently.

Results
1. 0 - 8 = Quality of life is most likely adequate. No medical intervention required yet, but guidance from your veterinarian may help you identify signs to look for in the future.
2. 9 – 16 = Quality of life is questionable and medical intervention is suggested. Your pet would certainly benefit from veterinary oversight and guidance to evaluate the disease process he/she is experiencing.
3. 17 - 36 = Quality of life is a definite concern. Changes will likely become more progressive and more severe in the near future. Veterinary guidance will help you better understand the end stages of your pet’s disease process in order to make a more informed decision of whether to continue hospice care or elect peaceful euthanasia.

Resources

Family’s concerns
Score each section on a scale of 0-2:
- 0 = I am not concerned at this time.
- 1 = There is some concern.
- 2 = I am concerned about this.

I am concerned about the following things:
1. Pet suffering
2. Desire to perform nursing care for your pet
3. Ability to perform nursing care for your pet
4. Pet dying alone
5. Not knowing the right time to euthanize
6. Coping with loss
7. Concern for other household animals
8. Concern for other members of the family (i.e., children)

Results
1. 0 - 4 = Your concerns are minimal at this time. You have either accepted the inevitable loss of your pet and understand what lies ahead, or have not yet given it much thought. If you have not considered these things, now is the time to begin evaluating your own concerns and limitations.
2. 5 - 9 = Your concerns are mounting. Begin your search for information by educating yourself on your pet’s condition; it’s the best way to ensure you are prepared for the emotional changes ahead.
3. 10 - 16 = Although you may not place much value on your own quality of life, your concerns about the changes in your pet are valid. Now is the time to prepare yourself and to build a support system around you. Veterinary guidance will help you prepare for the medical changes in your pet while counselors and other health professionals can begin helping you with anticipatory grief.

Basic quality of life assessments
Let’s face it – some people just need an easy way to evaluate a pet’s quality of life. I’m not saying I agree with this method, but for some, this is all they can mentally handle during these delicate days.

The most traditional method is when you ask a family to record the top 5 favorite things of the pet and when they stop doing 3 or more of them, it is ‘time’. My apprehension to this method is that it does not take into consideration the pet’s ailment.

One twist I like to add to this is adding something that the pet hates to that list. There are certain things that just ‘bug’ our pets – and when they stop caring for those things, it can be a sign that they are simply tired and do not have the energy to ‘care’. My own dog hated the Goodyear blimp that flew over our house. The week he passed – he didn’t make a peep at it coming into his air space.
Another uncomplicated way to track quality of life is to get two jars – one labeled ‘good day’ and the other ‘bad day’. Have the owner put a penny in the appropriate day jar based on the pet’s behavior, habits, daily functions, etc. Then after a few weeks – you can see if the pet is having more bad days than good and it is probably appropriate to recommend euthanasia.

A much better quality of life scale was created by Alice Villalobos, DVM and is called The HHHHHMM Scale. This takes into consideration hurt, hunger, hydration, hygiene, happiness, mobility, and more good days than bad. It can be downloaded by following this link: http://www.pawspice.com/downloads/QualityofLifeScale.pdf

Advanced quality of life assessments
After helping thousands of families with determining when is ‘time’ – I have realized that much of that assessment is ruled by the pet’s ailment. As mentioned above – the pet in heart failure is very different than a pet with arthritis. The questions that you evaluate are very different. Appetite in arthritis is not as important as it is in heart failure. Respiratory effort is vital in heart failure while not so much (except for painting due to pain) in arthritis.

Due to this – the questions I have owners ask everyday is based on the ailment. Lap of Love has created an online interactive tool that owners can use to evaluate their pet’s quality of life. They create their pet’s profile and choose from a variety of ailments. Based on the ailment selection, the questions and parameters they evaluate are different.

This tool is free for vets and the public at large and can be found at www.pethospicejournal.com

Using this scale in conjunction with the family’s quality of life has helped many owners feel empowered over their decisions – whether to continue or euthanize their pets.

Suggestions on using any quality of life scale:
1. Complete the scale at different times of the day, note circadian fluctuations in well-being. (We find most pets tend to do worse at night and better during the day.)
2. Request multiple members of the family complete the scale; compare observations.
3. Take periodic photos of your pet to help you remember their physical appearance.

Summary
How I wish the answer to the question ‘when is time’ was simple and clear cut – however, it is not. It is our duty to assist owners with end of life decisions and to help end and prevent suffering of animals. There are many ways to help families explore quality of life questions but the one way that is an injustice to our profession is if you simply say, ‘Call me when it’s time’. Owners need more than this and animals deserve more.
Although medicine may not be able to cure a pet’s terminal disease or old age, we can certainly help the owner keep their pet comfortable, clean, and happy, which is important not only for the welfare of the pet, but for the human-animal bond. With increasing number of positive experiences families have with human hospice coupled with the ever-increasing status of our pets in society, clients are requesting this care for their aging or terminally ill companion animals. Knowing what hospice care is, how to provide it in the clinic or home, and how to assist families in the mitigation of suffering will ensure you provide top-quality care at this delicate time.

Hospice terminology
The American Veterinary Medical Association views veterinary hospice as care that will allow a terminally ill animal to live comfortably at home or in a facility, and does not believe that such care precludes euthanasia. We define veterinary hospice is defined as: A family-centered, medically supervised, and team-oriented service dedicated to maintaining comfort and quality of life for the terminally ill pet until a natural death occurs or the family elects euthanasia. It is important to note that a natural death is not the goal for veterinary hospice, it is simply a reality for many terminally ill pets whether they are in hospice care or not. The main purpose is comfort of the pet before death, whether from natural death or euthanasia. This care can take place in the clinic or home: the home is often preferred because it is where pets are most comfortable. However, education and medical direction begins at the clinic. Using the word ‘hospice’ to describe this care will help families realize that their pets are at the end of their lives and unable to be cured. Many times just the use of this word is a relief to pet owners! This terminology also illustrates that comfort and quality of life are the most important goals for these pets.

When describing hospice care to clients, it’s helpful to use the MMM approach: medicate, meditate, and mitigate.
- **Medicate** includes assisting the client in identifying, predicting, and treating pain and anxiety in his or her pet during hospice care. Transdermal and transmucosal administration is used frequently with pets that cannot tolerate oral medications.
- **Meditate** encompasses the discussions and education the owner will receive during the difficult time leading up to the passing of their pet. Much of the hospice discussions we have with owners revolve around helping them identify what they wish for their pet (do they wish to wait until the last moment knowing they are risking an emergency situation, or would they rather guarantee a peaceful passing via euthanasia).
- **Mitigate** refers to death or “end of suffering.” Presenting it to clients in the latter way helps them realize that end of life also means an end to severe pain and/or poor quality of life. Remind clients that euthanasia is not just about stopping suffering that is occurring at the moment, but preventing it from occurring in the first place.

Most common conditions for hospice- overview
   **Mobility (see sample Hospice Consultation Summary and end of this document)**
Lack of mobility is one of the most common reasons clients seek hospice care from veterinarians in our organization, mainly for canines. Making the pet comfortable, both physically and mentally, is the priority with these cases and the veterinarian can provide many therapeutic and environmental options, including:
- **Medical therapy** (NSAIDs and other pain medications)
- **Physical therapy** (heat therapy, massage, frequent short walks around the house)
- **Complementary medicine** (acupuncture, laser)
- **Household and handling improvements** (nonstick flooring and slings)
- **In-depth conversation** about quality of life and how to make the decision to euthanize (more on this later)

   **Cancer**
Cancer (of all types) is another common reason hospice patients require our care. The terms oncology and chemotherapy can cause pet owners to feel anxiety and uncertainty. The veterinary team needs to encourage owners to consult an oncologist to determine whether their pets may benefit from chemotherapy.

The most common elements of hospice care for cancer patients include:
- **Pet owner education and discussion about:**
  - Type of cancer, associated signs and conditions, and disease progression
  - Therapeutic options and prognosis
  - Pain assessment and provision of appropriate pain management
Proper nutrition
• Assistance with therapy once a treatment plan has been developed.
• Administration of gastrointestinal protectants (for patients receiving chemotherapy), appetite stimulants, and any other medications needed to treat secondary clinical signs.

Renal failure
Renal failure is the most common organ failure disease seen in pets requiring hospice care. Its chronic progression leads many families to question the longevity and the appropriateness of the treatments they are providing. A very direct conversation about the slow decline their pet will experience, regardless of our ability to treat the symptoms, has proven very useful for families managing this disease in their pet.

Although not typically considered sufferable, renal failure is uncomfortable and can lead to severe nausea, vomiting, diarrhea, dehydration, anorexia, and anemia. Pain medications, especially those given transmucosally or transdermally, such as buprenorphine, can be of great benefit in some patients. Although the absorption is still questionable given transdermally, owners report improvement.

Hospice therapy should also include:
• Diet changes (keep the pet eating)
• Fluid therapy
• Appetite stimulants
• Anti-emetics
• Antacids
• Client education about disease progression

Disease classification and preparation
To help veterinarians and families categorize diseases and how they should be handled, we have placed the most common conditions seen in our practice into 3 different categories: Imminent, non-imminent, and intermediate.

Imminent conditions
During the late stages of these conditions, severe symptoms will arise suddenly and progress quickly. Generally speaking, we encourage these families to make the decision to euthanize sooner rather than later to prevent any imminent suffering of their pet. When the disease enters the end-stage, it will progress so rapidly that we will most likely not be able to make it to their home for a peaceful in-home euthanasia. They may either wait until their pet declines, knowing they will have to rush him/her to the emergency clinic if he/she does not pass on his/her own, or they should make the decision to euthanize a bit sooner than they want in order to prevent a stressful situation. Below is a list of imminent condition with the general method of death (these may vary greatly, listed here are my experiences).

a. Congestive Heart Failure – fluid may back up into the lungs, “suffocating” or “drowning” the patient.
b. Hemangiosarcoma – depending on location of primary tumor, pet my bleed out into the abdomen, chest cavity, or lungs. Pet will usually become unconscious before passing (may appear like cardiac arrest to the family), or may experience a seizure.
c. Osteosarcoma – too many times we get a frantic call from families whose pet has experienced a pathological fracture and is screaming in pain. With this diagnosis, no time is too soon to euthanize.
d. Seizures/Brain tumor – at some point, an intra-cranial condition will most likely progress to such a point that will render the pet incapacitated or non-responsive. Usually, we get this call when the pet has had a particularly bad seizure and the owner “does not want to go through that again.” These conditions are usually expensive to treat and maintain and can be quite difficult for the family to manage and watch on a daily basis.

Intermediate
Lap of Love Veterinary Hospice commonly sees conditions that do not fit in either the imminent or non-imminent categories. They should be treated with caution and education on a case-by-case basis. Most of these will start impeding a major organ or musculoskeletal function as they progress. Families need to make this decision based on the comfort of the pet, not his/her mental capacity.

There are 2 types of intermediate conditions:

1. Steady & Progressive – These conditions will progress slowly and start to affect quality of life based on the location of the primary tumor.
   a. Most mechanical neoplasias (those that push on or in an organ or functional part of the body – major examples below)
      1. Lymphoma
      2. Mast Cell
      3. TCC
      4. Nasal Tumors
      5. Pulmonary neoplasia
2. Chronic – These chronic conditions progress slowly but have the ability to decline quickly when compensatory capacity is exhausted and the organ finally shuts down completely. Although families have time to plan for this, they are encouraged to not wait until the complete cessation of organ function.
   a. Liver failure – can be painful in humans but not always, commonly treated with heavy pain medication just in case
   b. Renal failure – not considered to be extremely painful in humans although most report a general feeling of “malaise.” This is treated symptomatically; appetite stimulation, some pain medication if needed, and fluids if tolerated by pet.

**Non-imminent**

These conditions generally progress slowly. Families have time to consider their options, what they want for their pet, and how exactly they envision those last moments. These families have a difficult decision nonetheless. They have the “luxury” of time, but this can sometimes make the choice even harder. They feel so guilty for setting a day to put their friend to sleep. These families are reminded that this slow, progressive decline is not always what Mother Nature intended. In the “wild,” pets that are declining in health are removed from the heard via predation. Regardless of their mental capability, Mother Nature does not allow her animals to continue on and on in a state of non-mobility. We put our pets in a perfect environment with shelter, food, and water at their every whim. The duty we have for bringing them into our world and out-live their more natural counterparts in the “wild” is to not allow them to continue in a state of helplessness or anxiety. It is our job to prevent them from developing urine scald, bed sores, and systemic infection that would otherwise set in (or treat appropriately if needed). At some point, regardless of mental capacity (this is an important point you MUST explain to owners), they will need to make that difficult decision. Conditions considered non-imminent:
   a. Osteoarthritis
   b. Cognitive Dysfunction Syndrome
   c. Degenerative Myelopathy

Pain and anxiety are the most important aspects of managing these pets. Tramadol and/or Paxil or another SSRI if needed are used frequently. The author’s success with a benzodiazepine such as Xanax (alprazolam) has been less than stellar (more than half have an adverse reaction to it, becoming more anxious than before).

1. Tramadol is generally started at 5 mg/kg, and can go up to 10 mg/kg q 6-8 hours as needed for pain/restlessness. This helps the pets sleep… and owners get to rest too!
   • This is very bitter tasting, animals may stop eating altogether with this medication. Consider compounding to transdermal if needed.

2. Gabapentin – 5-20 mg/kg PO BID – TID, sedation is the dose-limiting effect (ie, when sedation occurs, back down a bit on the dose). Pet need to be on this medication for a few weeks to reach full effect. Frequently combined with tramadol (tramadol on as needed-basis for “back up”).

3. Paxil – 0.5 mg/kg to start and if well tolerated can increase to 1 mg/kg. Many say not to use with Tramadol (Serotonin Syndrome) but this is rare and usually only in high doses. Author uses Paxil if tramadol isn’t doing the trick (lowering the tramadol dose of course).

4. Melatonin – good for early evening restlessness, can’t do much harm here. We generally use 5 mg per 50 lb PO. These are available at any drug story over the counter.

For these families, these statements help them understand how and when to make the decision to euthanize (when appropriate):
   • It’s not too soon.
   • There is no veterinarian that would refuse to euthanize him/her right now.
   • You are absolutely within the appropriate time period, now you as a family need to decide what’s best for him/her.
   • You know her better than anyone; think about what she is feeling and the changes that she’s experiencing.
   • The guilt you may feel by waiting too long and allowing her to possibly suffer may be worse than feeling that you did it too soon. (This is a very interesting trend I see – families much more often feel they have waited too long instead of cutting the suffering stages short and doing it sooner.)

**Hospice services**

Hospice services and treatments can be provided on an outpatient basis or in the client’s home.

**Medical therapy**

Specific treatment for disease process in order to manage symptoms

1. Supportive medical therapy, including pain and anti-anxiety medications and subcutaneous or intravenous fluids
2. Nutritional support, including appetite stimulants and alternative feeding strategies (eg, tube feeding)
3. Physical therapy, including moist heat therapy, mas sage, and laser therapy
4. Complementary medicine (eg, acupuncture)
5. Client education, including how to administer medications and fluid therapy and foods to feed when pet’s appetite is waning

**Household handling**
• In-home evaluation: Provide suggestions for reorganizing the household for senior pet mobility/safety, such as:
  - Barricading stairs
  - Moving food bowls
  - Using nonslip surfaces
  - Improving traction by shaving hair between pads or using traction booties

• Sanitation:
  - Diapers or Chux pads (“puppy pads”)
  - Waterproof bedding (baby mattresses are an alternative to expensive dog beds as they are waterproof)
  - Baby powder, waterless shampoo, and shaving hair around the perianal area help keep pets clean and comfortable.

• Life enrichment: Keeping the pet’s mind active and alert can make a huge difference in quality of life. Owners can simply change typical pet games:
  - Instead of tossing the ball in the back yard, roll the ball to the dog while he is in bed.
  - Long walks can be replaced with an inside activity, such as “hide and seek,” a game many dogs enjoy, or simply short frequent walks around the house to maintain core muscle.
  - Pets with a high food-drive may love a Kong toy (kongcompany.com) filled with their favorite treats or unique bowls (aikiou.com) that encourage them to seek out food in compartments.

Senior/geriatric boarding
Many pet owners do not take vacations or leave town because they worry their pets won’t receive appropriate care or may pass while they are gone and also do not want to burden the interim caretakers. Offering senior/geriatric boarding can alleviate these fears and can include:
  - Putting nonslip mats in cages
  - Locating pet in the main treatment room so the veterinary team can monitor the pet carefully
  - Use slings for walks and making sure pet gets walked often (if pet is mobile enough)
  - Providing daily updates (phone/email/text) to owner and sending pictures. The AVMA recommends that if a veterinary clinic is not able to provide hospice care, that the clinic should refer the pet owner to a veterinarian or veterinary service that offers hospice options.

Targeting geriatric patients
Don’t be afraid to discuss hospice and end-of-life options with clients. Owners are usually anxious and distressed when confronted with an aging pet and impending loss. The veterinary team can help these clients by asking about their expectations and fears as well as explaining the hospice process. It is important for owners to feel at peace when remembering the last few months of their pets’ lives.

While veterinary clinics usually have a standard of care developed for patients 6 years and older (blood analysis, preventive procedures, etc), geriatric patients have different needs. In order to implement the geriatric/hospice care described in this article, the veterinary team needs to target these patients. This can be as easy as determining which patients are over the age of 12; and if these patients have not been to the clinic in the past year, calling the owner to discuss what care may be needed.

Keeping it simple
Many pet owners with geriatric pets want to avoid stressing their pets as much as possible and may also have financial concerns. This includes avoiding unnecessary clinic visits and procedures. Therefore, an open discussion about the pet’s disease, appropriate medical therapy, and ancillary services, with the emphasis on quality of life, is one of the first steps in beginning hospice care.

Avoid making pet owners feel guilty if they choose to cease treatment or decide against it. For example, if an owner decides against having his or her pet’s bloodwork checked every 6 months (to evaluate long-term NSAID administration), don’t threaten to cease medical treatment. Instead:
  • Educate the owner on potential side effects, highlighting the importance of presenting the pet for treatment if any adverse effects are noted.
  • Have the owner sign a liability waiver refusing bloodwork to protect you and your practice.
  • Help the owner plan a compassionate approach to end-of-life care for his or her pet.

Hospice handouts
In the same manner that veterinary clinics provide pet owners with a puppy/kitten package, detailed end-of-life information for patients should also be available. Some things to include are:
1. Disease sheets with detailed information about the illness affecting the pet, including end-stage clinical signs
2. Daily diaries that describe appetite, thirst, urination, defecation, mobility, and clinical signs of disease, which are important things to monitor while a pet is in hospice care as they help determine overall quality of life.
3. “Quality of life” scales help give a measurable value to owners; the pet can be evaluated daily or weekly and ideally by more than one person in the family, which provides a more accurate evaluation of the pet. Make sure to teach the owner(s) how to accurately use the scale. (See related lecture on Quality of Life.)

4. Adjunctive services you support and trust (preferably mobile) in the area, such as acupuncture, massage, mobile grooming, in-home pet sitting.

5. Local pet loss groups or grief counselors, contact local human hospice for a good referral source.

6. In-home hospice and euthanasia services (if clinic does not provide these services), such as in-home evaluation, rechecks, diagnostics, fluid therapy, bandage changes, and prescribing/administering medication. Try using a pet sitter who is also a certified veterinary technician.

7. Emergency clinics in the local area, if your clinic does not offer 24-hour emergency care.

8. Specific euthanasia information, including:
   - When and how to schedule euthanasia at your clinic, and if your clinic offers euthanasia in the home.
   - How to handle an emergency situation, such as nights or weekends, when a veterinarian may not be available. For example, “rescue” pain medication (high dose tramadol is author’s favorite) to get the pet through the night if emergency is care is not available or possible.
   - Aftercare information (owners need to plan ahead), including services your clinic provides and prices.
   - Local pet crematories or cemeteries, services that will pick up the pet at the home after it has passed, etc.

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**Sample hospice consultation summary**

**Pet name**

“Emma” Smith, 90 lb, 14 year old spayed female Labrador

**Appointment date**

April 30, 2013

**Regular veterinarian (rDVM)**

Any Clinic Veterinary Hospital, Tampa

**History**

Emma is a sweet Labrador with advanced mobility issues. She has been maintained on her current medications for a few months but owners are starting to see her have anxious moments and concerned for her quality of life. She had a spinal fusion performed when she was about 4 years old after an accident which playing. Emma also has had no conscious proprioception in her hind limbs for almost 12 months. Last weekend Emma was doing very badly (panting, pacing, whining, unable to stand) and her owners were quite concerned. They contacted Lap of Love Veterinary Hospice to evaluate quality of life and an in-home consultation was scheduled.

**Owner’s main concerns**

Quality of life, addressing pain

**Current medications / treatment**

Prednisone 20 mg once a day, Rimadyl 75 mg twice daily, Gabapentin 400 mg twice daily (dose split 300, 300, 200 mg through the day), Tramadol 400 mg split through the day (150, 150, and 100 mg doses)

**Exam findings**

Emma was bright, alert and responsive… she’s a happy girl! She has a history of cataracts, has prominent corneal edema, and decreased mobility.

- Cardiovascular – HR 60 bpm
- Respiration – RR ~50, lung auscult normally
- Other - MM pale pink, crt <2 seconds, BCS 3/9, temperature WNL, significant muscle wasting consistent with old age
- Signs of Pain – no overt signs of pain such as panting, crying, moaning; just discomfort associated with normal advanced arthritis.
- Mobility – Emma is usually able to get up on her own but occasionally requires assistance. During my stay she was mostly lying down sleeping but I was able to see her get in the pool on her own (requires help to get out), and walk around the yard to urinate and defecate (postured well!). Her front limbs are also clearly sore and she walks very gingerly. Previously diagnosed spondylitis is clear in the rigidity of her back and gait in general.
- Cognition - She is a VERY happy girl with a wonderful appetite still. Emma is excited to see her owner and responds normally to ques.

**Doctor’s observations**

Emma is progressing normally for her amazing age! Emma does not appear to be in a great deal of pain but humans with advanced arthritis report a general feeling of “malaise” that can border on severe discomfort. Inability to stand, vomiting, diarrhea, constipation, ataxia, anorexia (not eating), changes in drinking, and “spaciness” (dysphoria) are all signs we may see in the future. At some point, one or more of these signs will not be controlled by medications and quality of life may become a concern. You have the “luxury” of time to decide what is best for your family and Emma. She has far outlasted any standard we have in medicine, or any standard
Mother Nature has set! That’s a testament to your amazing care for her! We are here to help walk that path with you and assist in interpretation of Emma’s condition.

**Recommendations**
1. Continue all previously prescribed medications. Recommend weaning off Prednisone though (not good to have both Prednisone and Rimadyl) – give half dose for 5 days, then ¼ dose for 5 days, then every other day ¼ dose for 3 days, then stop altogether.
2. Kidney values and other blood parameters may continue to be monitored at your regular veterinarian’s office if you wish.
3. Continue feeding her whatever she will eat – it will help keep her energy up.
4. Continue pool swimming! This may be your best indication whether or not she’s happy and is a great for her mobility.
5. Hypersalivating may be a sign of nausea so keep a look out for this.
6. Encourage water consumption with fresh water in multiple locations. Adding ice cubes sometimes entices them to drink more.
7. Over the counter medications that may help:
   a. Fish Oil Capsules, 1000 mg – for Shelby, give 7 capsules per day.
   b. Sam-E (liver protectant) – give about 250 mg per day
   c. If upset stomach is suspected: Famotidine (OTC Pepcid AC) 10mg, give 2.5 mg by mouth every 12 hours. Most Pepcid AC comes in 20mg, so give 1/4 tablet twice a day
   d. Lomotil (diphenoxylate) for diarrhea - 0.05-0.1 mg/kg (so up to 4 mg for Emma, check concentration of drug) by mouth every 6-8 hrs

**Medications & adjustments**
1. Tramadol – pain medication, use as needed. 150 mg every 6 hours during the day and 250 mg every 6 hours at night. Emma’s top range would be about 8 tablets – you may use this dose if she is in emergent need of comfort before euthanasia can be performed. Give as directed.
2. Gabapentin – give as directed, recommend increasing to 1000 mg, twice daily or spread out over 3 doses if needed. This is a pain medication as well and should be given every day.
3. Prednisone – wean down as described above.

**Sample DNR verbiage for pets (sample only, legal review is recommended before use)**

**Do not resuscitate order**

**Owner’s statement**

Based upon informed consent, I, the owner or authorized agent of the owner for the above pet, hereby direct the withholding or withdrawing of cardiopulmonary resuscitation (artificial ventilation, cardiac compression, endotracheal intubation and defibrillation) from the patient in the event of the patient’s cardiac or respiratory arrest.
The Art of Euthanasia and the Science of Death
Mary Gardner, DVM
Lap of Love
Brandon, OR

Dani McVety, DVM
Lap of Love
Lutz, FL

The euthanasia appointment is unparalleled in emotion and sentiment. There are few things in veterinary medicine, or life moreover, that require as many outward displays of empathy, compassion, and commiseration from a doctor. The tone of voice, delivery of words, bedside manner with both patient and client, and the ability to honor moments special to the family become a delicate dance around death that the doctor and staff should carefully choreograph and continually improve. The client expects their pet to be saved in an emergency situation but helping the family and pet feel comfortable, understood, and secure in their most vulnerable moment, the death of their friend, truly transforms the professionals into heroes.

The perfect euthanasia
Dusty was a 13 year old Doberman Pincher with the perfect life; a family that loved her, two adult children that grew up with her, and 5 acres to patrol when she wasn’t lounging on the couch. At this delicate age, her hips were failing, unable to support her weight though her attitude remained mostly happy. Dusty’s owners knew it was time. On a cool, crisp day, the family gathered around, feeding her treats as they sat outside on a blanket telling stories of her younger years. The veterinarian pulled into their driveway and their hearts sank. It was time. After gently describing the process and answering questions, a sedation injection was given to relax Dusty. She slowly became more and more relaxed and her family was able to see one last glimpse of their girl in a painless, calm state, unlike the past few restless months of pacing and panting. A few minutes later the doctor asked “are you ready?”, and with much tear-shed, the family hugged their beautiful girl as she peacefully passed away in their arms.

Yes, this account may seem idealistic but it happens every single day, and will continue to happen more frequently as our pets rise in hierarchy within the family circle; it is, in fact, what I wish would have occurred with my own dog, Dusty. I wish we would have been at home, or at least been given the option. And although kind and considerate, I wish I had not felt rushed or ignored by clinic staff that was clearly incapable and uncomfortable handling a 25 year old girl grieving the loss of her first dog. I wish they did not use the cephalic vein so that I would have been able to hold her more tightly as she left this world (I had to insist they place the catheter in the room and not remove her from me). I also wish they would not have tried to console me so much, expecting me to stop crying with their words. And mostly, I wish I did not have to get back in my car and drive home without Dusty.

Now as a veterinarian, I cannot imagine saying goodbye to one of my own pets anywhere else other than at home (unless, of course, in an emergency situation). Although this is not always possible, available, or affordable to all of our clients, we can at least modify, improve upon, and perfect certain aspects of the in-home euthanasia appointment to make it as good as it can possibly be regardless of location. Euthanasia is, and always will be, an art form that should be carefully rehearsed to perfection.

Your curbside appeal
You are the doctor. You have years of education and experience behind you. You’ve done this hundreds, maybe thousands of times. Your client has experienced this once, maybe twice, if ever. You are the director of this performance; the performance of a lifetime in the eyes of the client. The way you and your staff look, act, and smell will be embedded into the minds of your audience forever; they will never forget this moment so make it count. When dining at a fine restaurant, the server never asks you if you would like more water, they simply fill up your glass. Bring this level of service to your clinic; do not ask what can be done, predict their needs and fulfill them effortlessly and without being asked to do so. The Platinum Rule states “treat others the way they want to be treated.” Remember this as you read on.

Location
Remember that the theater and lighting sets the mood of the performance. Most clients will choose a grassy patch outside or even the backseat of their car over an exam room. Suggest this change of setting whenever possible to avoid negative associations with your clinic. But no matter how cold, impersonal, or dirty the exam table is a warm compassionate touch is the one thing that can make a less-than-desirable physical setting a place of comfort and love.

Preparation
Your staff should be aware of any pre-arranged euthanasia appointments and families should be instructed to call the clinic upon their arrival. Escort them from their car into the clinic through a back door and assist with immobile animals when needed. A stretcher or basket with a plush blanket will envelope your patient from the beginning and ensure the client that their pet’s comfort is your top
priority. Don’t ask if they need help, just do it. All paperwork should be filled out except for the client’s signature (even the date and pet information). If the family requests a private cremation, do not go over urn selection at such an emotional moment. Have a nice, standard urn your clinic uses unless the client requests to pick one out specifically. Gently suggest, “it will be easiest to handle all the paperwork and payment now so you don’t have to do this after.”

Prepare a few items to place in the exam room for euthanasias:

1. Lighting sets the mood. Two lamps are much preferable to florescent lights (use a flashlight if you need to better visualize a vein later instead of turning on the lights).
2. A wireless doorbell, which can be purchased at any home improvement store, will aid in more discreet and appropriate client-staff communication. Give the client the wireless doorbell button and the chimer to a designated staff member. The client can then push the button after each step if they would like time alone with their pet.
3. A small mirror can be hung on the wall or placed in a basket. Women want a quick look before walking back into public after such a traumatic moment.
4. Small travel mascaras can also be placed in a basket for the above reason.
5. Tissues, of course.
6. Water bottles are nice touch. Crying can make you dehydrated quite quickly.
7. Small decorative bag for the pet’s collar, hair clippings, or anything else personal to the pet that the family will take home.
8. Air-dry clay flattened and cut with a round or heart-shaped cookie cutter. Crayola Model Magic is a well-liked brand and costs less than $0.50 for each print.

First impressions
Always smile. Not a jolly “someone just told me a joke” smile, but a true and compassionate smile with solid eye contact with both the owner and the pet. Make sure the client sees that immediate connection you have with their animal; it’s why you became a veterinarian in the first place. Be happy to see both of them and greet them with a warm “it’s nice to see you Debbie, and as always, it’s so nice to see Max, he always makes me smile.” This may sound strange but when said with warmth it conveys a caring and loving touch. A hug or (at minimum) two-handed handshake will go even further to express your empathy. (More on the value of physical touch later.) You may even venture a heart-felt “How are you, hanging in there?” followed by “stupid question, I know. I’m here for you.” Clients usually give a half-smile with this statement. If possible, sit on the floor with large dogs. At minimum, a short doctor’s chair that allows you to lean forward to listen is best. Touch the pet often. Remember to use both the client’s name and the pet’s name frequently (at least every 10 minutes) during your discussion; this engenders a familiarity and level of comfort even with strangers. If the client is able, try to have at least 2-5 minutes of small talk before you jump into the euthanasia explanation. This is the perfect time to set the mood and calm your client’s nerves just a bit.

When performing in-home euthanasia, most veterinarians attend to families alone. There are many reasons for this but most importantly it allows for an unobtrusive physical presence in a client’s home. In a clinic, however, many clients may feel comforted by the presence of a familiar technician especially if they are alone with their pet. This technician or support staff should try to sit closer to the client than to the doctor and therefore appear more as a supportive presence, not as a doctoral assistant. This person should be comfortable honoring silence and allow the doctor to fully lead the conversation.

Communication during euthanasia
Board complaints about veterinarians are frequently the result of euthanasias gone wrong which most commonly stem from improper or incomplete communication by the doctor. Proper communication is at the heart of any positive interaction in life, no matter how big or small. We’ve all heard the saying “they don’t care how much you know until they know how much you care.” Clients may not remember exactly what you say, but they will remember how what you said made them feel (good or bad!). Remember that human physicians that have never been sued spend an average of 3 minutes more in the exam room than their counterparts that have been sued 2 or more times (Levinson 1997).

Your greatest gift
Euthanasia is commonly referred to the “greatest gift” we can give our patients when needed. This may be true, but there is an equally important gift to provide the families; assurance and support that they are making the right decision or that you agree with how they are making the decision (these are not always the same thing). Many families contact our service because their regular veterinarian has made them feel judged, misunderstood, or simply guilty. Even if euthanasia is not the same choice you would make for this pet, if you are going to perform the procedure it is your duty to remove that guilt from the client’s back. It is your gift to them; lasting and profound confidence that they made the right choice at the right time. This is not a moment for “what-ifs,” it is a time to remind them they have done a good job caring for their pet, no matter how big or small those good deeds were.
Setting expectations early
Describing the process of sedation and euthanasia is the first rehearsed performance the doctor makes during this most important appointment and it should appear as fluid and natural as possible. Choose each word carefully and deliberately. A doctor’s gentle confidence is the first step to putting the client’s mind at ease. And any professional thespian knows that even when you forget your lines or something on the set goes wrong, you must improvise and never let the audience see your apprehension!

After your introductory small talk and concurrence with the client’s choice of euthanasia, the procedural explanation begins. It is best to start with an opened ended question like “Have you ever been through this before?” Many times you will identify specific concerns through previous bad experiences that can be addressed directly during your following explanation. A common description for sedation and euthanasia may sound something like this, “I am going to give Max two injections. The first one is back here under the skin (point to lumbar area), just like a vaccine. It is a heavy sedative mixed with a heavy pain reliever and will take about 3-5 minutes to set in. He’s going to feel very calm and comfortable. Remember he might react more to this injection than he normally does with a vaccine, but that’s because he’s not feeling like himself today. When Max is nice and calm, I will shave a little area on his back leg to find a vein (we use butterfly catheters routinely, adjust this for indwelling catheters). I will then ask you if you are ready. Once you’re ready, I will give him the second injection in the vein. This second one will take about 30-90 seconds to take effect and is an overdose of anesthesia. Similar to going “under” for surgery, it will effect the brain first, then the respiration, then the heart. It’s very peaceful. The two things I will prepare you for is that his eyes will not close all the way and his bladder might relax. It is very rare, but if anything else happens I will explain it at that time. Do you have any questions at all?”

Sedation (See related lecture on Sedation Protocols for Euthanasia for more detailed information.)
In our in-home euthanasia practice, all pets are given an intramuscular (IM) or subcutaneous (SQ) sedation injection (unless medically unnecessary due to an emergency). This slow sedation is integral to setting a calm and relaxed mood for the procedure. It is humorously, yet appropriately, considered “secondary sedation of the owner.” IM or SQ is highly preferred over intravenous (IV) sedation due to the time required to take effect and the gradual onset of “sleepiness.” Clients much prefer to see their pet become sedated slowly; watching a beloved pet go immediately unconscious has been described by a client as “worse than watching my wife sedation due to the time required to take effect and the gradual onset of “sleepiness.” Clients much prefer to see their pet become sedated slowly; watching a beloved pet go immediately unconscious has been described by a client as “worse than watching my wife faint after childbirth.” A 3-10 minute sedation time is ideal, depending on the pet, (and especially when children are involved) as the pet appears to simply fall asleep naturally.

Here are some tips for explaining the IM or SQ sedation process (these do not always apply to IV sedation):
1. Use words like “calm,” “relaxed,” and “comfortable.” These imply a warm feeling that their pet will have instead of a physical state of unconsciousness that does not always occur with IM or SQ sedation.
2. Never use the word “asleep” when describing sedation. Clients will expect their pet to be nonresponsive with closed eyes, which is generally not the case.
3. To avoid client complaints about their pet’s reaction to the sedation injection, explain that their pet may react more to this injection than they regularly do with a vaccine due to his/her current state of discomfort. If more explanation is warranted, you may elaborate with “when you have a migraine and someone touches you, it can be painful; something that is generally not painful becomes uncomfortable when you are already in a state of pain.” This reminds them that their pet has received many injections in the past and that they should not expect a sick pet to react the same as a healthy one. (This is also a trait seen in human hospice. Patients report that the sensation of a simple touch can become painful as their bodies begin to shut down.)
4. Remind the family that “sedation will take about 3-5 minutes but every pet is different.” A second sedation injection is warranted occasionally, but never give a third unless absolutely necessary; you will have lost complete authority with the family if you must give a third injection, so make the first (and definitely the second) count! Remember that animals with a high body condition score, dyspneic, or very painful will not always sedate as quickly or as deeply; dose your drugs appropriately and prepare your clients before administering.
5. If the pet is not sedating appropriately, timely, or if the disease process is prohibiting complete sedation (as the case with many dyspneic or overly painful patients), remind the family that sedation is not physiologically necessary since the euthanasia injection, by itself, is an overdose of sedation. Explain that you would rather their pet pass from the euthanasia drugs than from suffocation or extreme pain. In other words, “we don’t want to wait for him to be completely sedated from this first injection as this might cause unnecessary suffering.”
6. Remind them you will ask “are you ready” before proceeding with the second injection.

After administering the sedation injection, ask the family if they would like to spend this time alone (only if they have an immediate way of alerting the staff for help, like a wireless doorbell, in case the pet has a side effect). “Would you like me to stay with you or would you rather be alone with Max?” You can also offer for your staff member to remain with them as well. If they are hesitant, always stay. This is a few minutes of your time that will go a long way at conveying your compassion for them. This is also a wonderful time to ask questions about their pet and to smile about happier times. Favorites conversation topics include stories about
how they chose the pet’s name, what kind of youngster he was, how his brother/sister is handling the decline (and whether they are concerned about the pet’s housemates after this loss). Always compliment the pet no matter how poor the pet’s physical presence is, “what a distinguished face he has,” or “she has such a kind aura about her.”

**Euthanasia**

Explaining the euthanasia injection concisely and gently ensures that the family has the information they need without worrying about dramatic and atypical occurrences such as vocalization or seizures that rarely occur. Even mentioning these unwanted (yet possible) side effects of euthanasia can anchor the owner’s perception of the appointment (and of you) to one of pain and misery, even if they do not occur. This is why we prefer to only warn about the pet’s eyes not closing all the way and the bladder relaxing. Variations on this explanation are tailored around the family and the pet. Adequate sedation should decrease unwanted effects to about 1 out of 30 patients:

1. Remind clients that death is a phase, not a moment. The body will shut down different systems at different times and in different ways depending on the individual and the disease process. No death is the same as the next; coming into this world is not always simple, and leaving this world is no different. Euthanasia is the best medical tool we have to make the process as easy as possible, and even then, medicine is not always perfect.
2. If the owner requires more explanation on the process of dying, say “have you ever been under anesthesia for surgery?” Most people have. Remind them how peaceful and painless slipping into that deep sleep was, and that the doctor made an otherwise painful procedure (like getting your wisdom teeth removed), painless with the use of anesthesia. By inducing unconsciousness via pentobarbital, we are providing the pet the same painlessness. They quite literally go to sleep and not wake back up.
3. Eyelids are muscles; they will not close all the way but rather relax like the rest of the body.

**The final act of kindness**

When the pet is calm and comfortable, your physical movements to the back leg will announce that the time is near. (Use of the saphenous is highly preferable for administration of euthanasia in order to stay out of the family’s way.) Do not remove the euthanasia syringe from your pocket until you are ready, especially with children in the room who tend to be needle-phobic. After shaving (save the hair in a small bag or glass bottle for the family!) explain that you are going to place a catheter. After, or if using a straight needle or butterfly catheter, look the family in the eye and ask “are you ready.” Many families will comment “no” or “I will never be,” after which your response should be “he’s ready.” Upon confirmation from the family, begin administration. The author’s preference is about 1 mL per 5-10 seconds depending on the size of the pet (longer for smaller pets). Always keep one hand on the pet; this will not only show your affection for the animal but also allow you to predict any changes that may indicate stretching or muscle tremors as the pet passes away which can be immediately and gently explained to the family (again, this is rare though).

Occasionally the heart will continue to beat for a few minutes after administration of euthanasia. This is not a concern and will vary with the patient’s circulation and cardiac output. The best way to prevent a client’s concern about their pet’s heart not stopping immediately is to wait at least 60 seconds after administration before listening. Do this by slowly pushing the last 1-2 mL of the drug, leave the syringe in the catheter for a few seconds while you look at the pet and gently pet him, slowly remove the syringe, re-cap, flush the catheter, and gently place your stethoscope in your ears. If there are still cardiac sounds, use your judgment as to whether you will need to simply wait a bit longer or (rarely), or give another euthanasia injection (tell the family “and now I’m going to give the rest,” always bring the bottle into the room with you!). If you need to wait a bit longer, tell the client, “there were a few escape rhythms, I will check again in a minute,” or “he’s gently passing, let’s give him another minute, keep talking to him.”

**Plan B**

Common peripheral veins may not be immediately accessible. The medial accessory branch of the down leg on a laterally recumbent pet (good blood pressure), sublingual vein, ear vein, and even tail veins have been used by the author. Cutaneous vessels supplying tumors are also appropriate when visible and perfused. Above all, remain calm and confident in your technical ability. Alternative routes are always available. Some appropriate explanations may be:

1. “His blood pressure is a bit low, I’m going to try another vein.”
2. “Instead of using my normal areas here, he’s telling me I need to give this a different route here.”
3. “His body is declining quite rapidly, I’m going to give this in the most efficient way possible.”

The AVMA Euthanasia Guidelines allow for other routes of pentobarbital administration (with unconscious sedation only):

1. Intra-cardiac – If needed, gently place your hand over the thoracic cavity and say “I’m going to give this in a central vein that will bring it directly where it needs to be.” Shield needle and syringe from the family with your other hand. Aim more cranial and ventral than you think and leave room in the syringe for air and/or blood. 1 mL per 10 lb is recommended. (Cooney 2012)

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2. Intra-renal – this is a standard protocol for cats by many in-home euthanasia veterinarians. Say “I’m going to give the second injection through the abdominal cavity into a large vessel, it generally takes anywhere from a few seconds to a couple minutes.” 80% will pass before you have finished giving the full injection. Give 3 mL per 10 lb in the cortex, even in the smallest of kidneys (most skilled practitioners will recommend 6 mL). (Cooney 2012)

3. Intra-hepatic – if needed, this is a good alternative to intra-peritoneal as it causes death in 2-5 minutes. Explain “I’m going to give the injection near a highly perfused organ, he’s going to pass away in just a few minutes.” Use 2 mL per 10 lb and aim cranially just under the xyphoid process. (Cooney 2012)

4. Intra-peritoneal (pre-sedation not required at this time) – there is some evidence that abdominal irritation from barbiturate injection (Wadham 1997), but IP is still a good alternative, especially for fractious cats. 3 mL per 10 lb is recommended (Cooney 2012)

A gentle ending
As doctors, we know there is a difference between physiologic death, cerebral death, and legal death. We define this moment to the client with the cessation of cardiac function via confirmation with a stethoscope. There are many gentle ways to verbally confirm a pet’s passing with a client, usually combined with a gentle nod of your head:

1. She has her wings.
2. She has passed.
3. She is free.
4. She is free of pain.
5. She is watching over you now.
6. She’s gone. (This is not ideal though many clients will say it themselves.)

Other post mortem changes
There are natural post-mortem changes in the body that may occur. Address these concerns specifically if they arise.

1. If leaving the family alone shortly after the euthanasia, remind them about muscle twitching. “It is completely normal and usually happens in the first 5-10 minutes, generally on the muzzle or shoulder.” If seen, give a gentle reminder that death is a phase, not a moment.
2. In the author’s experience, agonal breaths are seen more commonly in animals that are close to the natural dying process (within minutes to hours of a natural death), and twice as commonly in cats. “Although rare, this is normal and tells me she was close to a natural death. It is simply a spasm of the diaphragm, just like a hiccupper, and while it looks strange, I can assure you she has already passed and feels no pain at all.”
3. Clients will occasionally comment that their pet’s body is still warm after death. Body temperature decreases at only about 1.5 degrees F. per hour; exothermic reactions can continue for quite some time, as will peristalsis of the intestines, which may occasionally be seen along the abdominal wall. (Guharaj 2003)
4. Give clients permission to remain with the body for an extended amount of time; give them the doorbell and allow them space. If they require more time, suggest they take the body home for a few hours (or even a day) before home burial or returning to your office for cremation. No drastic decomposition will occur and many families find this an essential part of the healing process, especially for children.

Closing of the appointment
When the family is ready, prepare the clay paw print (this takes practice) and give to the family along with the hair clippings and any other pet belongings such as a collar, blanket, toys, etc. You do not want them to leave empty-handed. Remember to honor the silence. Do not try to console, that is not our job. Your consolation comes in the form of your presence and a gentle touch. When the family is ready to leave, have a staff member walk them to their car, preferably through a back door. If bringing the pet home, ensure the most tidy and dignified method of transportation of the body. A stretcher for larger pets and basket for smaller pets is essential; never a body bag unless specifically requested by the family and clearly necessary.

Improving non verbal communication during euthanasia & the power of touch
We have already mentioned numerous ways to use non-verbal communication; solid eye contact, two-handed handshake, leaning forward to listen, touching the pet (and/or sitting on the ground), and the use of the client and pet’s name (ok, this is verbal communication but elicits a non-verbal effect). A short doctor’s chair ensures you are not taller (and therefore subconsciously more powerful) than the client; this is another reason why sitting on the floor with large pets is so endearing to clients. Physical touch is also a very important part of this non-verbal communication and is interestingly the most proven method for servers increase their tip (and theoretically the level of service the customer believe they had). The most benign placed for physical is either on the elbow or on the shoulder. (Driver 2010)
Review of important points

1. Be comfortable with your sedation protocol (see related lecture on Sedation Protocols for Euthanasia). Use it frequently and be ready to manipulate it when circumstances require.

2. Rehearse your sedation and euthanasia explanation over and over. Do it in front of a mirror, record yourself saying it, ask your staff for feedback. This is your first performance of the last appointment and your best opportunity to convey your care for both the people and their pets.

3. Always stay calm, you are the “rock” in the room, the one that has all the answers and guides the appointment. Do not react to a client’s emotions; be empathetic and compassionately confident.

4. Honor the silence, do not try to fill it.

5. Never underestimate the power of physical touch. Physical touch says more about your genuine care and concern than your words ever will.

6. Remind the family they are making the right decision. Always.

7. Compliment the pet; be affectionate and caring towards him/her.

8. Know your answer to “how do you do this.” Make it optimistic; “This is an honor, I’m privileged to be part of this memory with you.”

Euthanasia is not only a necessary, essential, and permanent part of our job, but also an art form that requires immense personal focus, unparalleled empathy, and a unique form of compassion for families in varying situations. As the only health care profession with the authority to end life, the veterinary staff has the duty to ensure that our clients fully understand each step we are taking and why. Euthanasia is truly an art form in which the human aspect plays just as much, if not more, of a role as the medical and technical skills. We will be the ones to change the face of human medicine; we are the experts, we are the leaders, and we should be the best.

References

The competence to mitigate pain and suffering by treating diseases and/or symptoms in animals without the ability to communicate both the intentional and potential outcomes of such treatments such that trust and rapport is gained with the client is akin to expecting to know how to ride a bike by simply reading an article about the physics of balance and rotational force. Without the belief and trust that the client and the doctor have the same desired outcome, even if that desired outcome is simply the comfort of the pet, trust and rapport will not be established and the client may not accept the treatment plan that you, as a doctor, went to medical school to learn.

As veterinarians we have two parties to serve in almost all areas of medical interaction; the owner/client and the patient. (Shelter medicine is the only exception to this rule, as treatment of animals in a shelter setting rarely include an owner.) In human medicine, the client and the patient are generally the same entity. Even in pediatric medicine, the parent is the guardian of the child, not the owner of that child. The parent generally has the levity to make decisions, but if that decision is not in the best interests of the child (as reasonably determined in a court of law), then the parent will in fact lose the ability to make decisions for that child. In fact, it took a groundbreaking case in 1984 (In re Guardianship of Barry, 445 So.2d 365 (Fla. 2d DCA 1984)) to determine that a parent can serve as proxy for their dying infant child’s wishes, allowing the removal of life support in this case. In veterinary medicine, however, our clients served as proxy for their pet’s wishes in almost every interaction they have with a veterinarian; from the decision to amputate a limb, chose surgical versus medical treatment, and even the removal of “life support” is a common path that the veterinarian must walk the client through on behalf of the pet. Legally, the clients are in fact owners of the patient and our communication and established rapport with that owner is imperative if we are to gain the trust such that our medical knowledge will be put to use for the betterment of the pet and/or the treatment of a disease. Learning how to gain that rapport is where the rubber meets the road!

In this interactive talk, we will use real-world examples and demonstrations to illustrate various aspects of nonverbal communication. We will discuss the 4 major types of clients, how to adjust your body language to gain and maintain rapport and trust with them, and how to compensate for problems that may arise. Attendees will be able to immediately identify these clients and implement different ways of nonverbally communicating by both reading their clients’ body language more accurately and changing their own to reach the desired outcome.

Important topics we will discuss include:

- Identify the 4 major types of clients.
- How to start the conversation: tone of voice to use, where to stand or sit.
- How to address client concerns and outbursts.
- Specific words to use (and not use) for different types of clients.
- How to react and what to say when things do not go perfectly.
Ethics and Euthanasia:  
What “Convenience Euthanasia” Truly is and How it Can Ruin Our Profession  
Dani McVety, DVM  
Lap of Love  
Lutz, FL

When it comes to ethical-border-line euthanasia requests, we have a very important decision to make as veterinarians, but we need to ask the right questions from the start. Instead of deciding whether or not you are comfortable euthanizing that pet, the question should be “what are the alternatives for this pet.” By requesting euthanasia in the first place, the family is communicating to you that the human animal bond is broken. We can either help change the situation for them (remove the pet from their care via adoption or euthanasia), or do nothing by sending them home because “I just can’t do it.” And in my opinion, doing nothing is professional suicide; you’ve now ruined any rapport you had with that family, a small loss that does not create societal trust and respect for our profession. Helping a family, in whatever way, is far preferable than sending them home with a broken human-animal bond.  
Remember, medicine is not our product in the veterinary world, the human-animal bond is. Without that bond, they are not coming into our clinics. When euthanasia is requested, the family is telling us that there’s something wrong with that bond and they care enough to tell you about it instead of letting the dog or cat go on the side of the road.  

So what should be done in these extreme cases of uncomfortable euthanasia requests? Allow me to push the boundaries a bit; in my opinion, we must take responsibility for the pet in some way. As a housecall hospice veterinarian, if I am at a home of a pet that I do not feel comfortable euthanizing, and with an owner that simply cannot go on, the pet will come home with me. Yes, it’s happened. And have I euthanized animals that I may not have euthanized if they were mine? Absolutely. Have I euthanized animals that other veterinarians have refused to euthanize? Absolutely. Have I euthanized animals whose owners were completely at a lost, unable to go on for many reasons, and with tears in everyone’s eyes (including mine), we knew it was a difficult but good decision? Absolutely. And when those families hug me, knowing that I did not judge them for that tough choice we made together, that I did not force an altruistic or idealistic view on them, and that I partnered with them in opting for the best alternative option for their pet, a new level of respect is earned.

Euthanasia definitions

- **Convenience euthanasia** is a very subjective term. We use this phrase when euthanasia is requested for a pet that would otherwise be deemed adoptable under most circumstances and the family is unwilling to explore these options. For example, “my pet doesn’t match the decor in my home any more” (yes, I’ve heard this). Personally, I do not offer convenience euthanasia in my practice, we offer support and resources to re-home these pets.

- **Non-medical euthanasia** is a term I use when describing a request that is not related to the medical stability of the pet. This is a broad term that includes behavior issues (such as aggression or improper elimination in the home), in addition to emotional or lifestyle changes of the family that precludes the pet from experiencing a quality of life.

- **Non-imminent medical euthanasia** is a term that describes situations like the 12 year old cat. These conditions may be manageable or even curable under the right circumstances, but for whatever reason, those circumstances do not exist. This includes the parvo puppy that may survive with intensive care, the 5 year old intact female with a pyometra, or the young cat with a broken leg. Without the right resources and conditions (which may be too expensive), this pet would potentially suffer greatly. Rarely will I turn down this type of euthanasia request.

- **Medical euthanasia** describes most of the euthanasiass that occur in our clinics; a choice that is made when the quality of life of the pet is deemed unsustainable by both the family and the veterinarian.

Non-medical & convenience euthanasia rules

- Do not euthanize a pet that you do not feel comfortable euthanizing. Period. (But say “no” carefully, keeping these other rules in mind.)

- Always help the family explore alternative options and think about how those options will effect the family and the pet down the road. Remember that a shelter is the deadliest place for a pet to be. Write them down, discuss them, think about what effect those alternatives have on OTHER animals in society.

- If you are comfortable euthanizing, even if you don’t completely agree, you must help the family understand that although this is difficult for you (and them), you care greatly for the pet and the greater good.

- Do not get involved in cases if you don’t plan to help, you will do more harm to our profession by judging and berating clients that if you simply hand them a number to a different veterinarian (preferable), or at least the local shelter or rescue organization.
Better Data Saves Pets:
How Veterinary Informatics Can Help Pets and Practices;
Use Your Practice Software to Improve
Patient Care and Anticipate Disease
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Integration of clinical medicine with information systems in veterinary medicine has been a challenge. Many practice information management systems (PIMS) provide tools that are simple, sometimes intuitive to use for reporting, but they cannot often provide summary or access to complex medical information. We show how, using basic tools, you can find and get powerful information from a clinic’s PIMS that can help clinicians respond to the changing environment and enable the hospital to provide better care to their patients and clients. We use parvovirus and lyme disease as our models to show how information can be extracted and utilized to hit the goals stated above.
Your Master Problem List: Working for or Against You?

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In this highly connected world where our cell phones serve to organize our lives, carrying all of our important information, clients are beginning to expect their pets’ data to be as electronically portable as their own. Unfortunately the reality is that veterinary electronic medical records are not yet capable of truly portable data. Online pet health record systems such as Patterson’s ePet Health have the ability to mine data from specified medical record systems, but the data is not “transferrable” to other clinics. It’s only available for viewing online. Agreed upon data standards such as a shared data model as well as terminology and messaging standards are required to make true electronic transfer of medical information a reality. Until they are fully realized, “data” transfers must be done manually or as “pseudo-data” pdf files, and we will continue to have to read through pages of records in order to “extract” the list of previous problems when receiving a new patient.

Through the creation and maintenance of the Problem and Diagnostic Terms subset of SNOMED CT and the Veterinary Extension of SNOMED CT (VetSCT), The American Animal Hospital Association recognized this need and chose to promote standardization of the Master Problem list as a good place to start. The Master Problem list serves as an important part of nearly all medical records giving a quick historical view of the patient’s medical history.

Standardization in the purest sense means that everyone in a given group (practice, practice group, veterinary profession, etc) uses the same “standard” list, in this case for the Master Problem list. Practically, we’d also like it to be a controlled terminology or list which is managed according to good principles of terminology, avoiding the common pitfalls that will be reviewed in the next section. Standardization of problem list terms helps to avoid the inevitable spelling errors that plague medical records, as well as helping to enforce purity of the data stored as a “master problem”, i.e. only problems and diagnoses should be stored, not owner notes, demographic info, procedures performed, etc. It also helps to ensure consistent and reliable recording and retrieval. When free text is used for a Master Problem list, great variability can be introduced in the terms used to describe any given problem. For example, a veterinarian with an interest in cardiology (or one recently trained by veterinary cardiologists) is more likely to record “Arrhythmogenic right ventricular cardiomyopathy”, while older veterinarians may be more likely to record “Boxer cardiomyopathy”. In order to retrieve all cases, one would have to first compile the list of all possible ways to refer to this one cardiac disorder, and many other examples can be easily found (cardiac failure vs heart failure, kidney failure vs renal insufficiencies, etc).

Standardization using an externally agreed upon standard, such as SNOMED CT and VetSCT, gives additional benefits. Using external standards gives the ability to share or transfer the Master Problem list electronically. As long as both the sending and receiving systems are able to store and process codes from the standard, then the Master Problem list can be sent in a data format that can be truly imported into any EMR and be immediately available for use. This would be an important step towards true electronic transfer of medical information. Furthermore, since SNOMED CT has been adopted by United States public health and is encouraged as part of the US government’s Meaningful Use guidelines, use of SNOMED CT and VetSCT means that our medical records would be compatible with public health and human medicine. While realization of any benefit from this is clearly years down the road, it is important to retain that capability for the future. And finally, the logical structure of SNOMED CT and VetSCT provides relationships between medical concepts that make advanced query construction possible.

Most EMRs today come with a built-in list of diagnostic codes or master problem list codes. Unfortunately experience has shown these local lists to have many issues as they are not managed according to good principles of terminology. The most common problem that users immediately notice is that the lists are incomplete, missing many of the problems or diagnoses needed. Most systems allow the user to overcome this by adding their own problems to the list. Unfortunately this capability is one of the main causes of the other two most common problems with local lists: duplication and ambiguity. Users rarely have the time or training to do a thorough review of the lists to be sure that the problem they need is in fact not there. If we use our earlier example, if “Boxer cardiomyopathy” is not quickly and easily found, the average user would add it without thinking to look for “Arrhythmogenic right ventricular cardiomyopathy”….leading to two versions of the same disorder: duplication. Once you have duplication in the list, then searches for records with one of these diagnosis terms will not return the cases stored with the other. Retrievals will be incomplete; records will be missed. The other problem seen with list items added by users with no terminology training is that the terms are often ambiguous, lacking the clarity to ensure that the term means the same thing to every user that reads it. For example, a busy practitioner who’s just seen a “congested kitten” might search for “congestion”. With more appointments waiting, if he doesn’t find it immediately, it’s likely that this overworked practitioner might decide to just quickly add what he needs and add “Congestion”. Unfortunately this term is not specific enough to inform the next veterinarian. Someone else might see a dog with pulmonary congestion on radiographs and choose this same term “Congestion”. Both are congested, but they are very different clinically. Similar issues could arise with findings in locations like “fundus” (stomach or eye?) or “knee” (horse’s carpus or dog’s stifle?). When
ambiguous terms are allowed in the list, you lose data accuracy. If the same code is being used for cases that are actually different diagnoses, you cannot be sure that you are returning cases diagnosed with the disorder in which you’re interested. We also find that a wide variety of “types” of terms get added to the Master Problem list when users are allowed to add at will. Everything from procedures performed (spay, pedicure, anal glands) to notes about the animal’s disposition (caution with pedicure, will bite). These are not “Master Problems”, and including them here reduces the purity of the data.

The solution to the issues with the local lists included in EMRs is to replace them with well-managed, standardized lists. The question then becomes….Do we internally manage our own standard for our practice(s)? Or do we adopt an externally managed standard?

The main benefit of locally managed lists is that you have total control over wording. Assuming that your doctors can agree on terms, you can use what is preferred by your doctors. Plus you don’t have to rely on an outside entity and their update schedules and resources. Unfortunately most practices and/or practice groups don’t have anyone on staff with terminology experience (there are only a handful of veterinary professionals with this training across the United States). This means either investing in a salary for a trained terminologist, which most practices cannot afford, or training someone currently employed by the practice/group to be the terminologist-on-staff. The terminologist will need to maintain centralized controlled of the lists. In other words, the software must be locked down to restrict modification of the lists to only the trained terminologist to avoid users propagating the issues discussed. If these conditions can be met, then locally managed lists can be well controlled and locally “standardized”. But remember, local lists are only understood or usable by the local system(s). These codes cannot be transferred and understood by external systems.

In order to preserve the ability to exchange data with external systems, you must use an external standard. Externally managed standards such as the AAHA Problem and Diagnostic Terms are managed by trained terminologists, so there’s no need to have staff trained in terminology management. This does mean that you will have to rely on that external entity for additions and changes to the standard, though there should be a process for making requests and you should take advantage of it. You will also have to be sure that your system is prepared to handle the updates as the external standard changes and matures. This is very vendor dependent, as the software has to be set up to handle the updates.

For small animal general practice, the only external standard available for master problem list or diagnoses is AAHA’s Problem and Diagnostic Terms (AAHA PDT). The AAHA PDT is a subset of concepts and descriptions from SNOMED CT and VetSCT. It’s designed to be used as an interface list in systems for recording problems or diagnoses in small animal general practice. Its scope does NOT cover specialty medicine or species other than cats and dogs. Feedback so far has been positive regarding the coverage of cases seen in general practice as well as emergency medicine, though we expect that the subset will expand as more practices implement it.

At this point implementation of the AAHA PDT is in a very basic form in order to fit into current interfaces. Current systems generally store a text phrase (term) and an identifier, and do not provide a way to connect more than one term to a single identifier. With this limitation, it is not possible to take advantage of the fact that the AAHA PDT provides multiple synonyms for most of the concepts in the subset. The Editorial Board that reviews terminology issues affecting the PDT has chosen a single term for each concept as the “preferred” term, and this is the term that is generally implemented in existing systems. Unfortunately because veterinarians have a wide variety of preferences, this preferred term may not match the preferences of every hospital. This is the reason that the AAHA PDT has multiple synonyms available for each concept. Vendors should be encouraged to restructure their interfaces to allow for the ability to connect multiple terms to each concept. This will dramatically increase user acceptance since everyone’s preferences can then be available when they are looking for codes or doing code based case retrieval. Another issue that many vendors are struggling with is the fact that the SNOMED identifiers are 64-bit integers, much larger evidently than most systems are using for identifiers. For example, in order for IDEXX Cornerstone to be capable of loading the AAHA PDT, they have to alter the identifiers in order to make them fit into their identifier field. This works ok for now, but will cause problems in the future when we start to send data between practices. Vendors need to be made aware of this issue and should be encouraged to plan for this need.

If you’re not lucky enough to be using a system that can load the AAHA PDT for you (such as IDEXX Cornerstone, ImproMed or some of the new cloud based systems), then you will have to tackle the task of importing the codes and migrating your system on your own. Unfortunately, I have yet to find a system that offers a batch import functionality. Manual entry is the most common, typing each code and term in one at a time. Some vendors may offer to load the codes for you via their support mechanism (Cornerstone is an example of this). But before you even begin the process of getting the codes into your system, you must consider legacy data. Legacy data is data that you may have already stored in your system using your system’s current list. If you haven’t coded any problems or diagnoses, then you have no legacy data to be worried about. You can just enter the codes and terms and get started using them. However, if you have already been coding problems or diagnoses then you have to consider how you are going to handle those previously coded cases. Currently, the process with Cornerstone is that as part of the steps that Cornerstone support takes to load the AAHA PDT, they inactivate all old codes. So your old cases will be coded using the inactive list, and any new cases will be coded using the AAHA PDT. While this is expedient, it creates a data wall between your old cases and your new cases. We suggest that a more data friendly approach would be to look at the list that you’ve been using, compare it to the concepts in the AAHA PDT, and create a “map” between the two lists. A map is just a file that records equivalency between the old codes and the new ones so that you
have a way to reconcile your old cases with the new terminology. The map creation is best done by a terminologist or someone who has been trained to be sensitive to the details of terminology and recognizing when 2 ideas or concepts are truly equivalent and what to do when they are not. The mapping process may help to identify any terms that you’ve been using that might need to be included in the AAHA PDT. The map can also serve as a guide if you decide that you want to review your old cases and code each of those diagnoses/problems with the new codes in order to have them retrieved via the new coding system. Vendors should be encouraged to develop a mapping functionality that tracks the relationship between inactive codes and the new AAHA PDT codes. If this were built into the system, then searches could be automated to access the map and retrieve cases coded with inactive codes that have been deemed as equivalent to current AAHA PDT codes. This would remove the need to recode legacy cases.

And finally, once you’ve accomplished your migration, AAHA PDT implementers must be ready to handle updates. In April and October of each year, a new release is made available that includes new concepts that have been requested, new synonyms for existing concepts, and notices of concepts that have been retired or removed from the subset. With each update, any new concepts should be added to each system’s list. New synonyms should be reviewed to consider if this new synonym might better meet the preferences of the practice (once systems all multiple synonyms for each concept, then addition of these synonyms will be all that is necessary…no review required). And finally any retired concepts should be inactivated in the system’s list. Vendors should be prepared to update the terminology lists in each system, or at least provide you with the tools you need to do it internally. At present it is unclear how this will be handled in systems, such as Cornerstone, that have the AAHA PDT available.

While we all would like to be able to choose a system out-of-box that will allow us to store our medical data in a format that will easily transfer to other practices, this is just not available in current systems. And unfortunately, creating a fully standardized record with all of these goals in mind is beyond the resources that veterinary medicine has available for standardization. We can only move forward using small, targeted steps, such as standardizing the master problem lists. But if we keep our goals in mind and move forward one step at a time, we can improve our data and as a result improve our medicine. This is the ultimate goal.
Client engagement is essential for the success of veterinarians and their teams. If the client doesn’t connect with the veterinarian, the veterinary staff, or the clinic, then compliance with recommendations and therapeutics will go down. Compliance is a constant struggle that has many complicating factors, but we know a trusting relationship with clients that are engaged in their pets welfare, improves compliance. More compliant owners take better care of their pets, offer a more satisfaction to their veterinarian care teams, and improve finances.

Getting clients to engage in their pets’ healthcare can be difficult. How can veterinarians connect with owners? In the age of social media and digital services this should be easier then ever with a little knowledge and commitment. This discussion focuses on those methods. Traditionally we engage in communication with our clients in the exam room, through phone calls, and through the mail with postcards and newsletters. In the last 15 to 20 years, veterinarians have had modern technological ways of engaging their clients via e-mail, texting, blogging, websites, and Facebook. Now, there are even newer ways to leverage digital media to engage our clients such as smartphone apps, integrated wearable pet technology, patient portals, video blogging, and newer social media services such as Instagram, Pinterest, and Twitter.

As we move more toward electronic health records, another way to more fully engage clients with their pets’ health is through complete transparency and open medical records. There is a growing movement within human medicine to more fully engage patients in their own medical care by offering patients the opportunity to view their entire or partial medical records online. Such a concept causes discomfort amongst most clinicians, yet research has shown that open records are greatly valued by the patients and can lead to more complete care. Allowing patients open access can provide important updates to the notes, automated access to lab testing results, and a more accurate drug list reconciliation. All of these lead to a more complete medical record. This final discussion on engaging our clients will focus on offering open records to clients and how this service may benefit veterinary medicine along with some of the potential challenges.

**Traditional methods**
A large amount of veterinarian staff, technician, and doctor time is spent in the examination room with the client and the pet. This is where, traditionally, we have focused on providing the true “perception of value” to our clients. The exam room is where we make money and bond the client by establishing trust and rapport. Veterinarians commonly hope they can establish compliance, improved outcome, and economic success in the exam room. Usually, either by showing off superior knowledge about a subject (client education) or a dictatorial relationship (telling clients what they will do).

After the examination is over and the client has left, clinics use telephone calls for follow up questions, lab results, more client education, reminders, and scheduling purposes. All of this direct and synchronous contact with the client reminds the client of the clinic’s brand and purpose. Finally, veterinarians in the past and the present still rely heavily on the postal service to engage clients. The time honored tradition of the vaccine reminder postcard is still effective and valuable. Companies used to clamor for the opportunity to help us create newsletters that could be mailed to clients with the goal of brand recognition, client education, and further bonding to the clinic.

**Modern technology**
Nothing can replace good interpersonal communication within the exam room and the value of synchronous conversation over the phone, but in the last 15 to 20 years, technology has allowed veterinary clinics to expand the ways in which they interact with clients and engage them with their pets’ healthcare. Though many clinics still drag their feet on the value of e-mail communication with clients, it has finally started to gain traction. Electronic records systems and patient portals allow for easy asynchronous e-mail interaction. Clinics can share their newsletter, reminders for vaccines, reminders for bloodworm, lab results, surgical images, educational material, drug information, etc. all through an email.

Just as veterinarians and their software vendors are making it easy to e-mail clients, research is showing that fewer people actually check or read their email so we must find other ways to inform clients of their brand and engage them with healthcare. Using a business’s website to provide information and blogs has long been a recommended practice. Through wit, humor, and knowledge a clinic can inform their clients about important local subjects. Unfortunately, somehow, the writer of the blog must inform the client of a new posting and persuade the client to navigate to the website and read the article. A boring blog that isn’t updated routinely will not get return readership.

Thankfully, a clean and modern website design will tell clients about the quality of the clinic almost as much as the front room they enter. A clinic’s website can convey a massive amount of information if it is organized well and easy to navigate. Besides the
blog, a client library, recommended vaccines, hours, services, videos, etc. can be made accessible from the website. However, similar to the blog, how does a clinic drive traffic to website?

In the last 10 years a new technology known as social media has become widely accepted. The most commonplace, comfortable, and accessible for most veterinarians is Facebook. Through Facebook, a clinic can post their blogs, share interesting animal stories, offer healthcare tips, and inform clients of updates to the website with direct links. Clients that follow the clinic will see Facebook posts in their feed and can easily be directed to the website with little effort on their part. Unfortunately, just as veterinarians are becoming comfortable with this technology and similar to e-mail, this author has been informed by a few young persons that Facebook is for “old people.”

Onto the next piece of technology that can be used to interact with veterinary clients, text messaging. This is technology that has been around consistently, is solid, stable, cheap, and ubiquitous. EVERYONE has texting capabilities regardless of age or generation. Most people will look at a text immediately when they receive one! Veterinarians can use text messaging in place of or adjunct to e-mail with a far greater chance of being noticed and read. Texting services allow for appointment reminders, vaccine reminders, reminders to give heart worm prevention, or updates on hospitalized patients.

Newest and developing technology
There are still newer methods that veterinarians can take advantage of to interact with their clients. In recent years, it has become easier to create a personalized business app that clients can download. While functionality is often similar to the full fledged website, these apps can make the telephone number of the clinic more accessible on a client’s actual phone. Clinics can send out push notifications reminding all of the clients that the first of the month is great time to give a heart worm pill. Clients can access the clinics blogs, find the nearest emergency clinic, and, perhaps, even have some basic access to their pets vaccine records.

For the extra ambitious veterinary clinic, video blogging and creating a clinic YouTube channel can be an incredibly effective method to interact with clients. Entertaining visual media can be a more effective channel to engage clients than written blogs. Or the two styles can be combined. A written blog on brushing teeth can have an imbedded video demonstrating the method. Common surgical procedures, information on vaccines, ear cleaning, or any other seasonal information can be shared in a video format. When a new video is ready and uploaded, leveraging a clinic’s social media network such as the Facebook or newer services such as Twitter or Instagram can inform clients of new content. Entertaining and informative content routinely updated will drive clients to the clinic’s brand and engage them with their pets’ healthcare.

Another fascinating new technology that clients are using more is wearable tech. As we humans use our Fitbits and Apple Watches for day to day activity, our clients can also use similar technology for their pet that records patient data. A clinic that partners with the client and this technology can use that data to identify trends in patient healthcare giving the client a feeling that they are part of their pets’ healthcare team.

With more electronic records systems being adopted, patient portals are becoming more common in human medicine. Veterinarians are and should follow suit. These portals give clients access to some basic information regarding their pets records, usually medications and vaccine information. They can request information from veterinarians, request drug refills, and request appointments. While some vendors already offer this, it will become more commonplace in human and veterinary medicine for clients to simply schedule their appointments via the portal as well.

Open records
Vaccine records and drug refills are great information for clients to have access too, but we should also consider offering our clients more. If our medical data and lab data and imaging studies are electronic, then the medical record can be shared with all stakeholders; from specialists to clients. More importantly, this unfettered access to records will further allow the client to act as a healthcare team member that has become more fully engaged with their pets’ health.

While the concept of “open” records may be unnerving to practitioners, research has shown that such a level of transparency is eagerly embraced by human patients. Clinician’s initial concerns with open access are often less problematic than anticipated. Such transparency should build a more trusting relationship and further engage clients in their pets’ medical decisions and treatments. Clients and clinicians can work together to ensure accurate lab result interpretation (a surprising number of abnormal lab results are skipped by interpreting clinicians!), drug lists, and follow up information. Medicine is shifting away from a paternalistic approach and towards a partnership. Open records will help this process.

There are barriers to opening up records to clients. The first is technological. The author is unaware of any electronic health records vendor or practice management software vendor that offers this or is working on it. Gathering the information from such disparate areas as DICOM images, JPEG ultrasounds, free text writing, PDF scans, communication entries, and lab results into a singular readable source has many hurdles to overcome.

The second major barrier is human. Veterinarians and staff members will have reconcile their real or imagined concerns with their written medical records being fully visible to their clients. A thorough understanding of what clients can and cannot see will be
necessary. All staff members would need to be trained on how to handle clients that offer new information to be added to the record or who believe some of the record is erroneous in addition to a standardize process for recording medical data.

**Conclusion**

For a veterinary clinic to survive and fulfill its goal of providing high quality medicine to patients, client engagement is required. Thanks to the advances of technology there are now more ways then ever before that a clinic can reach and interact with their current or potential clients. This will lead to more educated clients with more visits to their trusted veterinary office with better compliance and better outcomes for the business and pets. As we explore ways technology currently allows us to interact, we should always be mindful of what is next. Veterinarians, in general, often wait for technologies to become so mature before adopting them, they risk society moving past that technology and onto something new. Full open medical records offer a unique way that a veterinary clinic could potentially build a trusting and engaged relationship with their clients while leveraging the latest technology has to offer.
Practical IT Solutions for Practicing Veterinarians
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Veterinarians are highly educated individuals that, at some point in their career could follow the electrons through a Kreb’s cycle while identifying the vastus lateralis muscle during a lunch period. However, during all courses required to earn a DVM, there are several missing pieces to their education. We all know, from personal experience, and the veterinary literature that veterinarians are woefully unprepared to take on the role of manager, human resources expert, or business guru. In the modern veterinary clinic there is yet another area that veterinarians are poorly prepared or educated: Information Technology (IT).

A modern veterinary clinic is filled with computers, printers, routers, switches, digital x-ray, electronic ECG machines, SD cards, cameras, tablets, and any number of other gadgets or gizmos. Our clients demand access to medical records through a patient portal, access to veterinary advice via e-mail, text reminders, and copies of their digital lab work or radiographs. Yet, regardless of age or generation, veterinarians often lack basic IT needs requiring them to hire or consult an expensive IT expert (you get what you pay for!), rely on the sage wisdom of a salesperson, or just wing it using a Best Buy credit card hoping that someone on the staff can make it all work together.

This discussion will focus on some extremely basic concepts of electronic devices, convincing those devices to communicate, and some basic security principles. With a common understanding IT principles, veterinarians will be able to more objectively assess their current assets and expansion needs using the basic acquisition documents of a Statement of Work and a Request for Proposal.

The basics: computing devices

Let’s first review some common styles of computers that clinics may currently own or consider purchasing.

**Desktop**
This is the style of computer we are most commonly familiar with running a version of Windows or Mac OS X (more on that later). It commonly have a DVD/CD drive, audio in/out, a several ports on the front and back, and connect to a monitor. These run on their own and do not require additional hardware to perform.

**Server**
Many clinics that use Practice Management Software (PMS) own a server. This looks like a desktop, but might be larger. Inside it has a lot more hardware and capabilities. The server processes and stored data at a central hub.

**Laptop or portable**
Like a desktop, this is a fully functioning stand alone computer with many of the same ports and capabilities but without cords to keep it in place. By definition, it is smaller and portable so it can roam across the clinic.

**Thin client**
Also known as WYSE terminal, terminal computer, or “dummy” computer. This computer is NOT self contained. It is usually a small box connected to a a monitor, keyboard, and mice. However, all of the processing is done at a central server. The thin client allows you to access programs remotely from the server and display those in front of you. This is a great, cheap way to get a bunch of identical “computers” around the entire clinic, but every single device is dependent on the server to function. If the server dies, crashes, is accidentally shutdown, etc., ALL of the terminals stop functioning at the same time! Additionally, since all of the THIN CLIENTS must send data over the network to a single computer, a robustly engineered network is required.

**Tablet**
These are self contained, portable, and usually held in your hands. They range in design and capabilities but usually focus on using a touchscreen to input data. Tablets are similar to portable computers, but often work just different enough to cause some headaches. If tablets are integrated into workflow, a full understanding of their function and design is necessary prior to purchase.

**ChromeBox/ChromeBook**
These are a newer, and often inexpensive desktop or portable computers using Google’s Chrome OS. Much of their functionality is the same as a traditional computer, but Chrome is different enough to offer unique integration challenges. This is especially true with regards to server based PMS and printers.

**MicroPC**
Not be confused with a thin client, this is simply a small desktop computer. They are still fully functional, but in a small form factor often eliminating the CD/DVD drive and various ports.

The basics: cords

Everybody has a “box o’ cords” and it can be confusing what each cord is supposed to accomplish. Additionally, when purchasing a new computer, there is an alphabet soup of ports for cords to plug into, but many people aren’t sure what they need or what they do.
**USB**
Universal Serial Bus cords are a standardized cord developed in the 90’s and the most commonly used cord to connect anything to a computer. There are several kinds of USB cords. As rule, if you are using a USB 2.0 cord or USB 3.0 it will generally be compatible with most modern peripherals. This cord is limited to a length of about 3 meters.

**PS/2**
Once commonly used to connect keyboards and mice to the computers. Some old keyboard or mice may still require these cords and many desktops may still have a place to plug these in.

**VGA**
A common port found on computer monitors, video cards, HD TVs, and LCD projectors to carry video out from the computer into the display. This cord has been around a very long time.

**DVI**
A newer, but still common video display cord that may be next to the VGA port or replace the VGA port.

**HDMI**
This cord is commonly used with HD-TVs, Blu-Ray Players, and Video Game Consoles. It is now found on many computers as a universal way to carry audio and video to a digital display monitor. Typically this cord is limited to about 15 meters without using an extender.

**Ethernet**
Also known as Cat 5e or Cat 6 cables, these are generally how your desktop computer connects to the network. This is also the cable that creates the network transferring data across all computing devices in the building. Cat 5e is an ethernet cable capable of up to Gigabit Ethernet (1000Base-T). Cat 6 is capable of up to 10-Gigabit Ethernet. Both versions can run up to 100 meters. Cat 6 is the newer standard.

**The basics: wireless**

**Bluetooth**
A wireless signal used for short range communication between devices such as a phone and headphones or a keyboard to a computer. Depending on versions (1.0 to 4.2) there are various speeds and reliability. For practical purposes, the range is typically around 3 meters and sensitive to interference.

**802.11**
This is the standard wireless signal used by computers, laptops, tablets, and smartphones to access the network and the internet. There are several versions with the most current and fastest version being 802.11ac. Most wireless access points are backwards compatible (802.11a/b/g/n/ac) and should be able to operate within the 2.4 GHz and 5 GHz bandwidth range.

**The basics: operating system**
This is the software that allows you to operate the computer. The two most common styles are Windows and Mac OS X though a newer Operating System from Google called Chrome is also available. These almost universally are navigated with a mouse and point and click system that most individuals are familiar with. It is important to note that Chrome OS is a newer system that may not be compatible with important peripherals used in a clinic; most notably printers and label printers. Ensure that any vital hardware and software is compatible with Chrome OS before purchasing a Chrome OS device.

Tablets often run iOS (Apple based tablets), Android (Google/Samsung Based Tablets), or Windows OS (Surface Pro).

**The basics: internet connectivity**

**DSL**
Connects the clinic to the internet over telephone lines. Speeds range from 256 kbit/s to over 100 Mbit/s depending on technology and services paid for.

**Cable**
Connects the clinic to the internet via coaxial cable (cable companies). Speeds can vary up to 250 Mbit/s or 400 Mbit/s.

**Fiber**
Connects the clinic to the internet via a direct fiberoptic cable having a potential speed of 10Gbit/s. This is the most expensive, yet fastest and most reliable method of accessing the internet.

**T1/2/3**
Dedicated internet lines with very high reliability ranging in speeds from 1.5 to 44 Mbit/s. They often have a significant expense associated with them and are frequently installed if/when a clinic upgrade their phones to Voice over IP.

**The basics: network hardware**

**Router**
The router forwards and managed data as it travels across the network. In most small businesses, it controls the data between the computers and the internet.
Switch
A device that connects everything together on a computer network to process and forward data. They manage the flow of data across the network. Unmanaged switches simply forward data on. More advanced routers offer more efficient control of this data.

Firewall
A technological barrier that monitors and controls the incoming and outgoing data from the network to the internet. Hardware based firewalls allow for more effective programming and security rules then software only firewalls. The firewall is one vital component of network security to prevent hackers accessing the clinic’s networks and its data.

WiFi access point or router
This is the device that connects to the network and allows devices to access the network/internet wirelessly. There are many different standards and specifications that allow different speeds, ranges, and number of devices. See above regarding 802.11a/b/g/n/ac.

Assessing your needs
With some basic definitions and understanding, it is important that a clinic run an in-house assessment of what they currently have. There should be an organized network map pointing out all computers and network access points throughout the clinic. The clinic should evaluate workflow and interview front line users as to where problems exist. The network should be evaluate taking into account the internet speeds (upload and download) purchased for the clinic and the ethernet speeds available via the computers, cords, and network hardware.

Once current hardware and software is understood, a strategic plan can be established for acquisitions of new “stuff.” Almost all new “stuff” in a clinic needs to take its IT infrastructure into account. New PMS (server or cloud based) requires a good network infrastructure and appropriate computers, laptops, and printers. A new digital X-ray machine requires network and computing access +/- cloud access for teleradiology and remote back up. Even new lab machines often have the ability to be networked for electronic lab importation to the electronic records. All of this technology must be connected to be functionally useful to patients and staff.

Statement of work
Once a need has been identified and an acquisition will be made, a Statement of Work (SOW) can be helpful to focus the scope of the project and truly understand the needs of the clinic. The SOW defines project specific activities, deliverable actions, and timelines while also creating a plan for change management. It will often include detailed requirements for the project along with budgetary constraints. This document, when well crafted, will help guide the entire project ensuring that a good decision has been made and the new acquisition adequately meets the needs for which it was intended.

The SOW can be as vague or detailed as the project manager wants, though more details ensure a greater understanding of what is needed. Common subjects that are addressed by the SOW include Background, Purpose, Budget, Scope of Work, Deliverables, Deliverables Schedule, Project Reporting and Communication, and Change Management.

Soliciting vendors
With a thorough understanding of needs, goals, budget, and staff associated with an acquisition project, collecting bids from various vendors is next. Vendors often flood overworked veterinarians with sale pitches that may or may not fit within the stated goals of the project. How can we identify vendors that are offering solutions to the specific goals of the project. This is where the Request for Proposal (RFP) helps.

The RFP is a written, organized solicitation given to vendors that may be able to offer appropriate solutions. It provides an understanding of the needs of the clinic and allows for the clinic to control how information comes back, hopefully in an organized fashion. The ultimate goal is to identify solutions that meet the needs and budget of the clinic.

The RFP informs vendors that the clinic is looking to acquire a product or service giving them a thorough understanding of what the clinic is looking to accomplish. The RFP builds off the SOW, requiring the clinic to specify exactly what it proposes to purchase. Since this a standardized document distributed to all potential vendors, it will encourage vendors to understand that this is a competitive process and ensures that vendors respond factually to identified requirements.

An RFP should include Summary, Background on the business, Proposal Guidelines, Purpose of the Project, Project Scope, Project Timeline, Budgetary Constraints, Vendor Qualification Requirements, Proposal Evaluation Criteria, and Contact information for proposal submission or questions.

Conclusion
IT hardware and software can be daunting. In fact, IT has become so large and important to business functionality that advanced degrees are available just for network security, network engineering, and IT support. Yet, veterinarians and their staff are often required to make expensive purchasing decisions based on their personal knowledge and experience with little to no formal training. Even a basic understanding of the components that make up “IT” will help veterinarians better assess their current situation, their current needs, and plan for the future.
Once it has been decided that an acquisition is necessary, using commonplace documents can help the clinic ensure a competitive bidding process that finds the best solution for their needs. The SOW forces a clinic to fully understand its goals and identify specific needs that must be addressed in any acquisition. The RFP explains those goals to potential vendors and ensures that only vendors with appropriate solutions offer proposals.
Most veterinary clinics rely on a singular piece of software to accomplish their day to day tasks. This Practice Management Software (PMS) is a combination of financial, invoicing, scheduling, and electronic health records. The PMS is one of the most important aspects of running the business and when that software goes down, clinics are left scrambling. This software is advanced and massive with a thousand different capabilities that few clinics are able to fully understand or leverage. PMS are also expensive to maintain, access, support, and upgrade. A modern, well organized, and efficient PMS should be installed that is able to handle the clinic’s current and growing needs without encumbering the staff or the budget.

Understanding the broad based categories of PMS available to veterinarians, server based and cloud based, will better allow clinics to make short and long term choices for their PMS. No veterinary clinic should feel that they are trapped in a proprietary software solution and should consistently review whether their needs are being met with their current choice. With some basic knowledge about the benefits and challenges of both styles of software, clinic owners, IT managers, and practice managers will be better equipped to decide what is ideal for their unique needs.

Server based PMS

Typically, all PMS data is stored within a relational database design. This database must be accessible to many computers simultaneously to gather new data and to view previously stored data. Since the 80s and 90s, this database has largely been centralized on a server style computer connected to the other computers of the clinic via the Local Area Network (LAN). In multi-clinic set ups, the server can be housed at a central clinic and remotely accessed from computers in other physical locations. The server is accessed simultaneously by all computers and must be designed to accommodate this level of processing requiring faster processors, more RAM, larger hard drives, and faster network connections. Additionally, since the database is one of the most valuable aspects of a business, the server should automatically backup the database and have best security practices in place. Due to these extra needs, servers tend to be significantly more expensive then the computers that access them.

As a general rule, if your clinic’s database is kept on a physical computer and you know exactly where that physical computer is located, you are running server based PMS. There are many benefits to keeping a database on a server within the clinic. The clinic knows exactly where their database is located and can physically touch or alter the computer containing that database. They can customize the server and the network connection to the server based on their needs. The clinic has direct control over the database file and can back it up daily, weekly, monthly, and yearly on local hard drives (external drives, RAID drive, jump drives, SSD), or cloud drives (Dropbox, Google Drive, etc.). They have control over security, who can see the data, and how that data is used. If a clinic has an IT or database specialist they can mine the database using computer coding (SQL). Often times, the server is purchased so it is a physical (though depreciating) asset.

This more traditional software model of a one time purchase with incremental upgrade costs to newer versions is comfortable for many people used to buying software packages for their own computers. Rather than an ongoing monthly expense, the clinic can buy the software outright and not worry about paying for it again. To increase comfort levels, the software packages for a server based PMS will typically be offered by a well established company with a long track record of reliability. Many of the most popular softwares have been around for 20 or 30 years! Through years of experience, server based PMS often have a understanding of how veterinary clinics work and what the workflow looks like. Additionally, through consolidation, these server based PMS vendors may have larger parent companies that have deep pockets to support ongoing development and technical support. Large server based PMS vendors aren’t going to go out of business anytime soon, have large support teams, and work hard to integrate with other large healthcare IT vendors such as ANTECH, IDEXX, etc. Having a large number of human and financial resources allows the server based PMS to work on many levels of integration simultaneously.

Yet, there are also detriments to server based software beyond their significant expense. Since coding a stand alone relational database PMS takes a lot resources, these large companies can be cumbersome and lose their agility. The software may go several years between major upgrades and those upgrades often must be purchased at additional cost. Server PMS vendors also have the ability to create proprietary ways in which they store the clinic’s data making it difficult for the clinic to leave a software platform once they are established.

While software limitations are important to consider, the single largest risk with server based PMS is that the clinic is dependent on a singular piece of hardware. While some larger clinics may have redundant servers in place, many clinics will only be able to afford one server. If the hard drive fails, electrical cords gets chewed by a kitten, a virus destroys the data, or any other number of “bad
things” happen to this one computer, the entire business’s data and ability to function comes to a halt. The server is the single most important piece of a computerized clinics infrastructure and its most vulnerable.

Often times the database is stored within a single location on the hard drive of the server and that server is connected to the world via the internet making it vulnerable to hackers. Good network and internet security is vital to keeping the clinic’s data secure. Either the clinic pays a local IT company, the PMS vendor, or handles this security itself. Keeping the server hardware up to date and the operating system and the antivirus software and the printer drivers and the firmware updated all fall to the clinic.

Server based software provides reliability and traditional feelings of control because the clinic knows exactly where their data is located. They can also plan on one time purchases infrequently. However, server based PMS also provides vulnerability since the clinic is relying on a singular piece of hardware to run its business. The clinic also must understand and implement good network security, backup protocols, and appropriate redundancy all of which takes time, money, and expertise.

**Cloud based PMS**

“The Cloud” is a commonly used phrase used to describe data that is no longer stored on the local hard drive, but somewhere else in the world. Think about “The Cloud” as an external hard drive that you cannot see, but access through the internet. Common consumer level cloud based services include DropBox, Google Drive, iCloud, or Amazon storage. The cloud offers a plethora of benefits, the most often cited is increased security, cost savings, and synchronization across computers.

Similar to a server, the database for a cloud based PMS is stored at a location, but is site. Therefore, the data is always in sync with the computers that access it locally and remotely. However, the database in the cloud accesses and sends data using the processing power of hundreds of very expensive high powered servers somewhere else in the world. The clinic no longer has to use its own resources to purchase, setup, and maintain the workhorse computer for database storage and access. These costs in time and money are shifted from the clinic to professionals that manage server for thousands of companies. Additionally, the cloud vendor is able to offer higher level security setting to provide redundancy, high levels of uptime, and prevent hacking with high level encryption.

The big difference between server and cloud is the knowing that your data is securely available anywhere in the world with encryption and backup. Most importantly, the clinic is no longer responsible for its own IT maintenance using clinic staff that not trained to handle it anyway. The business is no longer reliant an a single hard drive or a single computer (server) to function. Even if the cloud server farm is hit with electricity losses or flooding or a hurricane, there is (or should be) adequate redundancy that the clinic’s data is safe and accessible at all time.

Cloud based PMS will also alter IT costs. Rather then a “high cost of entry” with large one time purchases, the cost is typically a Software-As-A-Service (SaaS) model or subscription based. The vendor provides the PMS for a monthly fee. This allows the clinic to offset large purchases and reliably budget IT allocation on a monthly basis. New upgrades and tweaks to the software happen incrementally throughout the lifetime of the subscription so the clinic can rest assured they are using the most current version of the software with the most current functionality readily available.

Cost savings can be found with the IT budget in regards to computers as well. The clinic no longer needs high powered computers but simply a device to that can access the network and the internet. Because all of the data flows over the network and out through the internet, though, a robust network with proper termination, high upload/download speeds, quality switches, and firewalls are necessary. This is a significant potential cost as many clinic networks are not designed for robustness, efficiency, and security that cloud based PMS requires.

Another detriment that many clinicians feel with cloud based PMS is that they don’t know physically where their database is stored. It may be in San Diego, New Mexico, or New Jersey. This data is also outside the security protocols of the local clinic so it is often hard to predict who has access to the database. While cloud based storage offers improved network security, human security can still be a concern and must be examined with any potential cloud based PMS vendor.

Since the software is SaaS, the costs will never go down or disappear. In fact, as with most things, it is virtually guaranteed that at some point, the monthly fees will go up. A long term evaluation for 1 year, 3 year, and 5 year costs is valuable to truly assess total costs associated between server or cloud based PMS.

Another significant challenge to cloud based PMS is the “newness” of the concept in veterinary medicine. Many of these vendors are small without the backing of large parent companies. This is changing as large vendors acquire them, but size may matter. A small company with limited clients may be in peril of going under. If a clinic’s PMS vendor goes out of business, what happens to their data? Smaller companies may be more agile in rolling out new functionality, but they may also lack the human resources to make quick shifts. Small PMS vendors may also lack the network and resources to fully integrate with important peripherals and ancillary companies such as labs, imaging equipment, or marketing/portal services. All of this must be taken into account to ensure a cloud based PMS can truly offer a long term solution to a clinic.
Conclusion
Server based PMS offer the benefits of traditional understanding and comfort levels because veterinarians will know exactly where their clinic’s data is stored. The server is customizable to the needs and wants of clinic. However, this comfort comes at high costs in terms of financial, human, and knowledge resources while also keeping a business tied to a potentially vulnerable piece of hardware. Cloud based PMS may offer a way of removing this vulnerability and shift many of the human, knowledge, and financial costs to another company. Cloud based PMS also will provide the most current and up to date software available from that vendor at all times. However, a lot of trust is needed in the way the cloud based vendors set up security, maintain the data, and support their software. Many of these companies are smaller with shorter track records creating potential upheavals within the clinic should the vendor fail or be purchased by another company.

Every clinic should feel comfortable exploring the functionality of their current PMS and comparing it against what is available. Any clinic looking at upgrading their PMS, either to a new version or a new system, should create a Statement of Work and thoroughly explore the costs and benefits of server based or cloud based PMS vendors. Both styles have a lot to offer clinics and thorough review will allow a clinic to find the solution that best fits their current and future needs.
What is evidence-based medicine?
“Evidence-based medicine (EBM) is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients”. This means integrating individual clinical expertise and the best available external clinical evidence from systematic research (Sackett et. al. 2000).

The heart of EBM is that scientific methodology is most likely to determine what is likely to be true and what is false. It should increase our confidence in what we are doing is effective. It is not about dogmatism or evangelizing zealots but another tool to help provide quality medical care.

**EBM involves three general steps**
1. Phrasing a clinical question that is pertinent and answerable.
2. Identifying evidence that addresses the question.
3. Critically appraising the evidence to determine whether it applies to the patient.

**Quality of evidence**

**The hierarchial ranking of evidence in human medicine**
In veterinary medicine, we lack many of the higher quality sources of information to answer clinical questions. There is a need to collect and analyze data from private practices to generate some of the case control studies and case series to create the knowledge base to make scientifically valid decisions. The objective of this discussion is to present the work VMDB has done among North American Veterinary Colleges to gather and analyze data for publications and advancement of knowledge and propose a process for private clinical veterinary practices to participate in developing this knowledge base.

**History and work of VMDB**
Veterinary Medical Databases (VMDB) has collected and analyzed data since 1964. VMDB was originally funded by NCI (National Cancer Institute) to collect data on cancer cases using punch cards and mainframe computers and coded with SNVDO (Standard Nomenclature of Veterinary Diseases and Operations). When NCI discontinued funding, VMDB was transferred to a consortium of veterinary colleges and continued. VMDB has evolved over the years using changing technologies from magnetic tape, CD’s, usb and now via the Internet to transmit data. VMDB has remained current by migrating from SNVDO when it became apparent it was not being maintained and switched to SNOMED CT (Systematized Nomenclature of Medicine Clinical Terms), the premiere international medical terminology.

The data from VMDB has been used for over 100 articles relating to diseases and diagnoses from the various specialty hospital associated with the veterinary colleges. The disadvantage of this data is that it reflects a small, skewed subset of data seen by referral clinics. VMDB is at a stage where it is now possible and desirable to collect data from small animal, large animal and exotic veterinary practices to collect, analyze and generate knowledge more generally applicable. Disease surveillance, epidemiological trends, demographics, effectiveness of therapies and various other trends can be identified for clinical decision making.
Role of private practices in disease surveillance, epidemiology and advancing veterinary knowledge

Various surveys have shown that the vast majority of veterinary practices are computerized using some type of practice management software. This fact makes possible the collection of large data sets from a variety of clinics across North America. Using natural language processing, encouraging incorporation of SNOMED CT and using data analytical tools, it will be possible to report on demographics and statistical analyses of risk factors, effectiveness of treatment and other important trends and outcomes.

Collection of data will be secure and anonymous. No identifying data for owners, animals, clinics or clinicians should be stored. Unique identifiers will be used but there will be no ability to connect those back to any specific person or animal. Geographic location will be determined by city, state and 5 digit zip code to ensure privacy for clients and veterinarians.

The benefit for clinics will be an ability to review reports and trend generated from data collection. Breed specific issues and risk factors can be identified. Epidemiological and disease surveillance reports can be distributed to clinics located within certain geographic area to support decisions on vaccinations and disease awareness.

Benefit to animal welfare

Using data from private clinical practices will be beneficial to improving the health and welfare of animals. Identification of breed risk factors for disease and disease surveillance will improve the overall health of animals. Evaluation of therapies for effectiveness or lack of effectiveness will ensure appropriate therapies are used. Identification of adverse reactions to drugs and treatments can prevent risks for animals. For example, the discussion about vaccines and fibrosarcomas can be confirmed or denied. If fibrosarcomas are vaccine related, do certain vaccines have a greater risk or is fibrosarcoma just an injection risk occurring with various injections.

One health

Dogs and cats live 24/7 with people in the same environment as their owners. Do certain diseases that are increasing in occurrence in dogs and cats reflect the same trend in people, thus using pets as sentinels for environmental exposures? Correlating results for disease trends in animals may help identify disease trends or environmental risks in people.

Technical plans

An automated system of data submission will be developed in collaboration with the practice management software companies. This will allow identification of the demographic fields and data model in the EHR (Electronic Health Record) used at a participating clinic. Initially, Natural Language processing will be used to extract terms and map to SNOMED CT concepts. The data from those clinics using the AAHA Problem and Diagnostic list will be easier to store and analyze. Over time, VMDB will help participating clinics and software vendors to incorporate one or more of the veterinary terminologies (AAHA Problem and Diagnostic list, the Virginia Tech Veterinary Extension, and the VMDB Diagnostic and procedure list) into their EHR.

Getting involved

Any clinic or veterinarian wanting to submit data can email VMDB at vetnet@vmdb.org with the clinic name, contact person name, email address, phone number, and name of the practice management software used at the clinic. VMDB will then contact the clinic to analyze their current software and plan for automatic extraction and submission of data to the VMDB servers. Participating clinics will have free access to search the VMDB private practice database once they have submitted data for a six month period. Over time, the data collected can be analyzed for demographic, morbidity and mortality, treatment outcomes, disease surveillance and other trends of interest.
Gone are the days when all you had to do was trust that word-of-mouth, a Yellow Pages ad and a few brochures or fliers would be enough marketing to bring the clients in your door.

Today’s savvy business owners and managers are depending on technology specifically social media to keep their brands top-of-mind with their target audiences. If your practice has been trying to avoid jumping on the social media bandwagon, 2013 is the time to set your fears aside and go for it.

By now, you’ve no doubt learned the power of social media. It’s something that companies of every size, in every part of the world, are embracing in order to remain competitive. For many of us, however, the thought of social media is quite intimidating.

How often should I post on Facebook? On Twitter? Do I need a blog? What if someone posts a negative comment on one of my social media platforms? How will I make the time for me and my staff to actively participate in social media? Does social media replace traditional forms of marketing?

The questions go on and on. Here are five social media tips to get you started on the right foot, with input from social media expert Eric Garcia.

1. Update your website
Through social media, you’ll often drive current and potential clients back to your website. Make sure your website is updated, easy to navigate and looking its best at all times. A bad website could turn off potential clients.

The expert (Eric Garcia) says:
“Prominently display all of your social media links on every page of your website so clients and prospects can easily link to the various social media channels you are part of.”

2. Interact with followers
It sounds crazy, but social media is about being social. You or someone from your staff should be engaging with your followers at least a few times per week. Comment on other Facebook pages regularly and respond to the comments your followers leave on your page.

Do not delete comments, even if they are negative. Unless comments are inflammatory in some way, simply respond to them and set the record straight for other followers who might have seen the initial comment.

Post things that initiate a conversation between your followers—link to interesting articles or photos and ask how your followers feel about them.

The expert (Eric Garcia) says:
“Engaging with your followers will constantly put your practice name in front of people. Clients and prospects don’t follow you on social media to hurt you, but rather to support your practice. The likelihood of negative comments is very small. One way to engage followers is to post a photo of clients and their pets and ask your followers to caption the photo. Facebook, for any practice, should contain a fair mix of education, fun and personalized socialization.

3. Post daily
Ideally, your business should be posting on at least one of the social media platforms every weekday. Content can, and should, vary.

Some posts could include new items related to pet health care in your area, interesting images from your practice (unique X-rays are always a big hit), fun pet-related videos, blog posts, etc. Sharing popular posts from other industry pages is a quick and easy way to come up with a post for the day.

The expert (Eric Garcia) says:
“Post daily by sharing the same content across all of your social media channels and remain consistent. If you post a blog on Facebook regarding dental awareness, make sure to post the same blog on Twitter and share a related video on YouTube to keep the message consistent.”

4. Consider a blog
Although a bit more time-consuming, a blog is a great way to connect with your clientele. It gives you the chance to show them who you are on a more personal level. Your blog can go in any direction you want it to, as long as it is a reflection of who you are, letting your clients feel like they are getting to know you, your staff and your practice.
Write posts in the first-person point of view, using many photos, videos and other forms of multimedia. Also, link to relevant sources and related articles as often as possible. Blog posts can feed your other social media efforts—after a blog is posted, link to it in a Facebook post and a tweet. Ideally, you should post to a blog weekly.

**The expert (Eric Garcia) says:**
“Blogs are a great way to enhance your online presence through organic search engine optimization. When posting blogs, make sure to tag and categorize each blog post under a related category. And don’t stop after posting your blog – share it on various social media channels; be sure to tell your clients about the blog in person as well.”

5. **Supplement your marketing**
Social media should complement your traditional forms of marketing, not completely replace them. Make sure all of your marketing efforts reflect a consistent brand—colors, fonts and imagery should be similar throughout.

**The expert (Eric Garcia) says:**
“Don’t try to become part of every social media channel at one time. Take the time to focus on one social media channel and do it really well, then start exploring additional channels to be part of.” There’s never been a better, or more important, time to make social media a part of your overall marketing strategy. Don’t let it scare you. Start slowly and work your way up to meet your posting goals. Before you know it, it’ll be second nature.

**Search engine optimization**
Search Engine Optimization: The process of affecting the visibility of a website or a web page in a search engine's "natural" or un-paid ("organic") search results. In general, the earlier (or higher ranked on the search results page) and more frequently a site appears in the search results list, the more visitors it will receive from the search engine's users. SEO may target different kinds of search, including image search, local search, video search, academic search, news search and industry-specific vertical search engines.
What’s New in 2016 for Social Media
Eric Garcia
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In an era where most restaurants have stronger Wi-Fi than coffee, it’s hard to truly comprehend, let alone stay on top of, the sheer ubiquity of modern technology. Not only is technology everywhere, but it’s also speeding up each day. If we want our businesses to keep up, we’ve got to be on the move as well. The digital realm continues to integrate into most major aspects of our daily lives, ballooning into a practically palpable world where we can book flights and check sports scores, literally without lifting a finger. Meanwhile, major Social Media outlets like Twitter and Facebook keep on expanding their user bases consistently each quarter. The transition toward a world that’s entirely online has already begun, and folks, we’re a long way from dial-up.

Need more proof? In the year 2013, Apple sold more iPhones than most countries have people. Yes, feel free to read that twice for impact. And if you caught a glimpse of The 2014 Apple Worldwide Developers Conference (WWDC) this June, you know that they have no intention of slowing down their rollouts or sharing their market dominance in the near future.

Now, close your eyes and imagine even more people, with even faster technology. Open your eyes and voilà! It’s already happening. This expeditious trend isn’t just an illusion. As long as the rate of technological paradigm shift continues to increase exponentially, things will only get faster. This is theorized as the Law of Accelerating Returns and continues to make technological gains quicker and smarter than ever before.

What does this mean for businesses like yours? Well, whether you’re a household brand name like Coca-Cola or a newly opened veterinary business, your product or service, of course, comes first. But your engagement with your current and prospective clients, general accessibility and online presence now all come in at a close-ranking second. This pushes business owners to adapt or lose relevance in the all but prolonged digital age.

Still, not everyone is sold on the rise of Social Media. Some chalk it up as a temporary fad; others simply won’t budge when it comes to their tried and true marketing methods. While there are traditional methods of advertising and marketing that maintain their pertinence over time, (word-of-mouth marketing, for example, will never die) a growing amount of these dated-mediums are simply falling by the wayside.

On the other side of the equation, we can assure you that Social Media isn’t something that’s going to fade away anytime soon. While the Social Media-Medium itself that’s used to communicate might wax and wane over time, it’s the interconnectivity itself that remains not only relevant, but increasingly imperative to utilize, and ultimately master.

With most adults now online, the time to build your company’s digital infrastructure is now. Social Media allows you to quickly engage with your audience in an absolutely seamless manner. If it’s a new skillset you’re boasting, a sale you’re offering, or even a simple news announcement, your Facebook Page will allow you to capture your audience’s attention in a heartbeat. Deliver your content directly into their newsfeed, while measuring clicks, traction and response rates in real-time.

For those that have already mastered Facebook, Twitter is another fantastically savvy way to engage with your clients, friends and fans. Take it from this legendary Twitter savant, who despite a plunge in his recent approval ratings maintains one of the largest and most successful Twitter accounts in history. It’s about leveraging the platform that suits you best, and presenting your audience with what matters most.

At the end of the day, you want to live and work happily, and ultimately grow your business into the most rewarding and viable endeavor possible. With such a teeming array of Social Media websites in existence, it may just be a matter of figuring out which ones are right for you and your business, and then getting down to it.

Start with Facebook, Twitter, and Google+, and you’ll see how simple these platforms really are to use. And even if after this article, you still want more proof that your demographic actually uses Social Media, Dog Lovers International has you covered. Well, what are you waiting for? You’re only a few clicks away.
Yelling at Yelp!
How to Manage Your Online Reputation
Eric Garcia
Simply Done Tech Solutions
Lutz, FL

We all remember that fateful day when we passed through the doors of our High School for the very first time. Brow sweaty and backpack brimming, we knew that our reputation from years past would follow our footsteps down the hallway, but that we’d also have an entirely different arena in which to create anew.

The scenario itself may have changed, but many of the principles remain the same. Our reputation still precedes us when we enter a new environment, though now the World Wide Web is usually the messenger, for better or worse. Whether it’s a new job, a meeting, a fundraiser, a date, or something in-between, there are more ways than ever for those around us to do their homework, on us, before meeting face to face.

That’s right, a simple Google keyword search can bring up anything from your business website, to your Facebook page, and hopefully not those Instagram pictures from your best friend’s wedding. While it may seem like you have no control over what people find out about you online, the truth is that you have more of a say in the matter than you think.

Google has a remarkably comprehensive system in place for coordinating user search results and delivering them in a timely, deliberate and meaningful fashion. What might seem like random results at first glance is actually a highly methodical process unfolding in real-time. “Crawlers” (know singularly as Googlebot) continuously sort through billions of web pages from each corner of the globe, algorithmically analyzing, assessing, ranking, and indexing web pages and sorting through each applicable result.

So what does this mean for you? It means that when you’re looking for new clothes online and nonchalantly typing in “Summer Outfits Sale”; your results aren’t random. In fact, they’re far from it. Google, which accounts for approximately 70% of search engine market share, is calculating your keywords and weighing-out millions of results before delivering them to you, all in about 0.5 seconds flat. The key takeaway here is this: business owners can, and frankly need to, monitor and enhance their business’s search results in order to maintain and leverage their Online Reputation.

For example, when searching online for a local veterinary practice, customer reviews will gravitate toward the top of the screen, and instantly display the name, website, address, phone number, and here’s the kicker, one to five star average rating, of nearby practices. In a Google Search (by far the most common kind of online perusal) this one to five score is the combined average rating of the “Google Reviews” that have been left for your practice. These scores can dramatically influence the amount of new clients that find, and ultimately choose your business. This is why business owners must safeguard and even encourage the establishment of a positive Online Reputation in order to fully optimize their practice.

Ok, so Keyword Searches and Google Reviews are all important factors to consider when managing my Online Reputation. But what can I do about it and how can I use these tools to enhance my practice? Well, we’re happy you asked.

Quick tips: Improve your online reputation
1. First, you’ve got to search for your business online. If you’ve never looked, you might be a bit intimidated at what you might find. But you can do it, dive right in. We recommend that you search on Google, Bing, Yahoo and Yelp, and then “Claim Your Listing” once you’ve found the right result. Verify that this truly is your business, and presto, you’ve claimed your online listing.
2. Next, setup your online alerts. By using both google.com/alerts and mention.net you’ll have two powerhouse services monitoring the Internet for results about your business. Now you’ll be notified by email whenever a new result or review is available. Convenient? We think so.
3. Now, bookmark your top online listings in a folder within your browser. This will save you time when you wish to check back on your results, and lets you easily review your listings at least several times a month.
4. Here’s the fun part; engage with positive reviews! The average veterinary practice receives one review per month. You can reply to a positive review online, call and thank the client, or get creative and send a personalized Thank You card. Engage how you see fit, but definitely reach back to those who’ve been impressed with your practice. This little bit of effort can most definitely go a long way when it comes to encouraging positive reviews and ensuring client retention.
5. Finally, go the extra mile. You can engage directly with happy clients at checkout by encouraging them to leave a positive review for your practice. If they’re onboard, send them a friendly reminder email at the end of the week, with a direct link to help them leave a Google Review.

It’s really that simple to enhance your online presence and manage your online reputation. Doing so could mean the difference between countless new customers, and a haphazard web-presence. By pro-actively managing your Online Reputation, you’ll stay
ahead of the curb, reach more clients and ensure your veterinary practice maximizes its resources. In the digital age, you may not be able to control everything that’s published online, but you can certainly nudge it in the right direction.
Creating the “Easy” Experience for Clients
Brian Conrad, CVPM
Meadow Hills Veterinary Center
Kennewick, WA

Year after year we sit and preach to our staff about offering WOW Service and instill into them that they must exceed client expectations every week, every day, at every appointment. … How are we doing? I will guarantee we are not doing as well as we think we are. Have you ever taken a moment to consider what it is you are asking them? You tell them: “Go Exceed Expectations”. What in the hell does that mean? First off, do we know what the client expectations are? For a few that can answer that honestly as Yes, then tell me how does the client perceive if the client expectation has been exceeded? If we as the management and leadership team struggle to define this, imagine our poor receptionist who hears time and time again to go exceed expectations. After all, his/her next pay raise could be counting on it.

While I am playing devil’s advocate slightly and yes, I truly do believe in incredible client service and exceeding expectations but I have found over the years it is very subjective. We have focused at Meadow Hills Veterinary Center where I manager on “Making it EASY” for the client. If you tell your staff to make it EASY to do business with your clients than they have a much better understanding of their role. After all, we can list out all kinds of ways to make life easy for our clients and their pets. See the difference?

In 2010, The Harvard Business Review published results from a study they conducted using over 75,000 consumers to learn more about exceeding expectations vs. making business easy on customers. The first intriguing fact was 84% of those surveyed indicated their expectations had not been met in one of their most recent service interactions. 2 Critical factors came from the study that suggests Delighting Customers does not build loyalty but reducing their effort does. Interesting. So the first finding indicates that if we do more of the work for the client there is a higher probability of them sticking around for next year’s services. Finding number 2 stated Acting deliberately on this insight can help improve service reduce customer service costs, and decrease customer agitation and defection. So in essence, if we have our team members in our veterinary clinics make it easy on our clients such as reminding them 7 days before they are about to run out of a long term medication for their pet than they are going to be much more loyal and the probability of them staying with our clinic is high.

Let’s put this in terms we can relate to. Weight loss is a fairly easy concept to understand. If input of calories is less than output of energy in calories (i.e. burn off of calories) then you will lose weight. Obviously the opposite is true with weight gain. If we know this easy equation of eating and exercising a healthy lifestyle, then why is it Americans will spend some 35 billion dollars on weight loss products, pills, powders, additives etc. Why? Because it seems so much easier to take a magical pill than be good about what you eat and hit the gym. Kodak makes incredible cameras yet they are in bankruptcy. We don’t use these incredible camera but instead take the majority of our pictures with our smart phones because it is so convenient and easy. With a touch of a screen we are posting them on social media and texting them to family and friends. We are willing to give up some quality for ease.

Relate this back to your veterinary hospital. Are there systems and processes that place too much work on our clients? Are there ways to cut down on the amount of effort which is placed on the client? During a recent lecture on phone call systems in the veterinary office, participants identified clients calling to get updates as one of the top 5 reasons the phone rings. With a focus to lower the effort and anticipate the client’s needs for continual updates, imagine if we texted them as the patient headed into surgery, another text as they exited, and another as the patient woke up safely from anesthesia. This just removed 3 times the client has to attempt to call in to see how their baby is doing from a simple spay or neuter procedure.

Don’t assume you are doing all of the work. Ask the client in a survey using a “Customer Effort Score (CES). This is generally expressed on a scale of 1-5. One indicating very little effort was put forth by the client and a five indicating the client felt they had to do more than expected. The question would read: “On a scale of 1-5 how much effort did you have to put forth to have you and your pet’s needs and requests taken care of?” You of course would define what the scale of 1-5 indicating for the answering client. Very interesting information. In fact, a client who gives Meadow Hills Veterinary Centers where I manage a score of 3-5, I am immediately on the phone with them to find out where we went wrong. Take the time to learn more about your clients’ needs and wants. This activity can be a real eye opener. Make sure you are constantly sharing the results with the staff and making changes as necessary.

Still not convinced? The study continued on and found of the consumers that indicated effort was low on their part, 94% indicated they would be back for re-purchase of services or products. 88% indicated they would increase their spending. Only 1% noted they would speak negatively about the company. Of the clients that noted a high effort score, 81% said they would speak negatively about the company.

Let’s look at a couple of examples of companies in the United States that have put this into action. Chrysler sent a development team to the parking lots of grocery stores across the country. What they found is mom or dad coming out in the middle of the day with kids and arms full of groceries fumbling for the resemblance of the van keys and having a difficult time getting into the car. In came the invention of the auto-slider door on the min-van in efforts of eliminating effort. A big success for the families.
Lowes Home Improvement store launched “My Lowes” a web-based platform which organizes all of your personal preferences for your home and garden projects. It will keep track of the paint colors for each room of the house, specific annual plants and quantities you use for your garden each year and even remind you to purchase and replace your air filter. In other words, they are doing the work for you to keep your home maintenance organized. It’s working for them too in the way of re-purchase revenues.

In 2012, a report was release on grocery store trends. One of the indicators which was measured was the reason a grocery store is selected. 3 out of the top 5 reasons were for reducing effort on the consumer with convenient location, simple to shop, and easy in and out access. It is another indicator this paradigm shift for our veterinary hospitals is real and much needed for us to continue to find success with our clientele.

I encourage you to sit down with your staff and make a list of the reasons clients come into your doors. Exams, prescription refills, surgery, dentals, diagnostics, retail purchases, boarding, grooming etc. Make a road map of each reason and steps that have to be taken and list out ways your team can take on more of the effort for the client. Start to survey the Customer Effort Score and make sure the changes are being noticed and recognized by your clients. If they are not, then you need to go back to the drawing board and evaluate where the team is missing the effort connection. Taking a proactive approach will end result in a happier, more loyal, and more profitable clientele.
Interactive Case Studies:
Management Scenarios Gone Wrong

Brian Conrad, CVPM
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Welcome to a very unique CE session where the outcome of the next hour will be decided by you the audience. The participants will be given a list of real-life management scenarios that have gone wrong over the years. We will summarize various categories and then vote to determine which scenarios we will discuss. I will give the pertinent details of the case studies and then have a group discussion about how you the audience would handle a similar situation. We will then give details about what really happened, lessons learned, and action taken to prevent this all from occurring again. You all may select scenarios which involve human resources, marketing, communication, client relations, financials, law and ethics, and/or technology. Obviously human resources comes up often and plays a large role in the amount of time we spend in our day. While I don’t know where the session will take us, I have included some scenarios below in regards to hiring and retaining staff as that seems to be a recurring popular theme. I look forward a dynamic and productive session together.

The concerns, questions, and complaints I most often hear at Continuing Education conferences are I can’t keep good staff or train them. It of course never seems to be the fault of the owners, managers, or practices. The youth of today just aren’t like they were in my day said one practice owner recently in Kansas City at CVC. So where should the fault lie? I am not as concerned as who to blame as opposed to the mind set the practices must take in order to gain some ground on this issue. I hate to burst any practice owners/managers bubble but for every time I hear “I can’t find any good employees” I hear “I wish the practice I worked for was managed better”.

Several years ago I too along with our practice owners at Meadow Hills Veterinary Center in Kennewick, WA felt the strain of staff turnover. Each year we set a theme for our management team during our annual strategic planning session. In 2004 the theme was set as the “Year of the Staff”. Sure we always wanted to treat the staff well, but were we? It was time to take a mental inventory and evaluate areas for improvement and build on them.

It was at that moment I defined “My Promise” A promise which states “No team member will leave the practice feeling unchallenged, concede to a lack of direction, or have professional growth hindered”. A bold statement yet so simple. I realize there are companies out in the world able pay higher hourly wages, afford well rounded benefit packages, and perhaps even work schedules with ideal hours. But can they compete with “My Promise”?

It was a turning point for the company. At the same time a challenge to our management group not allow this promise to go unfulfilled. The idea of an empty promise leads us to the next factor of ultimate responsibility. The responsibility to say each employee’s success or failure is dependent on the systems and communication channels the management team puts in place. Failure by any team member is a direct reflection of the management and leadership style itself. For us to realize our new obligation and find success, the promise had to be interwoven with management undertaking the act of ultimate responsibility.

Let’s take a look at an example of ultimate responsibility: A new employee is confused with creating a vaccination schedules for a puppy on their second wellness visit. Our normal reaction tends to be: “We have told her 5 times how to determine the vaccination schedule, I think she is just not bright enough for the job.” Ultimate responsibility would have us examining our training methods for the new employee. Asking ourselves, did we spend enough time practicing and role playing? Do we have prepared materials for them to use as references as the new staff learns and becomes confident? Perhaps the training methods are not where the problem is but in our hiring techniques. Maybe we hired somebody trying to fill an immediate void that never belonged in the clinic to begin with. This is where the change has to occur with the management team in taking the responsibility to say we failed the team member, they didn’t fail us. The management team must be humble, learn from their mistakes and move on.

The idea of ultimate responsibility must funnel down to your senior staff. It is often time the senior staff witnessing job performances of the younger staff. We recently surveyed all of the staff that was trained in the last 18 months. One question posed was “What was the hardest part of fitting in at the clinic”? One of the popular responses was they felt the staff was quick to offer blame for any mistakes made. Finger pointing simply put. Since completing the survey results we have worked with our staff to create a culture in which now they realize the mistakes made by others are mistakes they need to help offer further training and guidance to. Instead of pointing a finger and gossiping to others, they are taking the challenge on their shoulders to find a solution. Not an easy transition style of thinking.

The staff you have in your hospital is already capable and ready for further challenges and growth. We as owners and managers just simply turn a blind eye consciously and unconsciously. It is not until the clinic enters a crisis situation such as losing key senior staff member that we are reminded. It is scenarios such as this where the management team is forced to promote or ask more of a team member to compensate for the loss. Not surprisingly, the individuals steps up to the plate. The sad part is they had it in them the entire time. We just didn’t realize it and take advantage of the talent sooner. I was recently reminded of this lesson a couple of weeks.
ago. I was in need of some gift certificates that needed to be created for a promotion which was soon concluding. I kept putting off the project because of time constraints. I finally had a team member offer to create the gift certificates on the computer for me. I reluctantly agreed. The next day I arrived in my office to find the certificates were completed in color with our logo and a creative theme for each month of the year (The certificate which was valid for July had a 4th of July theme on it, October had Halloween on it and so on). I was dumbfounded at the thought and creativity that went into the certificates.

We often hear the clichés maximize the staff’s strengths and minimize their weaknesses or don’t try to fit a square peg in a round hole. As non-original as the quotes are they do hold truth. Part of the promise is to identify each team member’s tools and talents and having them working positively and in unison for the clinic.

Betsy just celebrated her sixth year anniversary with Meadow Hills. She started in our boarding department and has since cross-trained in every other department of the hospital. While she does a fantastic job no matter where we have her scheduled, it wasn’t until recently where we truly found her ultimate strength. She now oversees all of our hospital assistants. From the hiring and training to evaluations and establishing communication lines with other departments, it is a perfect fit for her. One she excels in. We now focus our attention on making sure she is successful in her new endeavor. The management team is there to offer guidance and support and a little bit of cheerleading when needed. At the same time we relinquished some of our control in order for her to continue to grow. Not always an easy feat for practice owners and managers but necessary for success when done in a controlled manner. Betsy and others continue to take on more responsibility and with proper road maps and open doors will continue their professional growth for many years to come.

Ah, now the difficult part of the promise. Identifying these talents and strengths and opening the doors for growth and opportunity before it is too late. We start the process by encouraging and soliciting feedback from each member of the staff. It is not uncommon for me to invite an employee into my office and plainly ask “how are you doing” or “How do you feel about your job”? . When time permits I will take an employee on an errand, community event, or just simply for coffee. One on one time which aids me in determining if the staff is staying challenged and enjoying working for the company. A time to talk about personal and professional goals. During these essential meetings I find staff members are unaware about the possibilities the future may hold.

Take Kari a seven year veteran with us. For six years she worked in Reception and as a part time bookkeeper for us as she began her family. With the kids preparing to enter pre-school she was ready to go back to work full-time and wanted to be further challenged and possible seek work in the field of her bachelor’s degree. I took an hour of my day to take her to lunch. During this time we sat and explored options drawing a map with 3-4 areas she could advance with Meadow Hills. Over the last year she has trained in our treatment area becoming a coordinator for our surgeries and dentistry’s. I will never forget her satisfied face as she boasted to me she completed the placement of her first IV catheter. She will be attending her first major CE conference at CVC East and we look for her to take on even more responsibility when she returns. A lunch and an hour of time is a small price to pay in order to retain a valued employee.

With the promise goes sincere appreciation and gratitude. Rather than celebrate birthdays we focus on staff anniversaries. A poster is created each month to reflect each employee celebrating an anniversary and notating how long they have been with the company. After 3 years of service the ladies receive a gold star necklace. The gentleman receives a gold star tie tack. At five years a diamond is placed in the center of the star. At ten years the ladies receive diamond earrings. A special way to appreciate our gold star employees and simply say Thank You.

Going back to 2004, I am reminded of a night of celebration and thanks we created for the staff. What was unusual about this evening is our primary focus was to show the staff’s families how important each team member was to our hospital and to the clients and pets they serve. The night started off with a tropical themed dinner complete with real Hawaiian leis for each employee. The program presented by the two practice owners reviewed each position with the clinic and the immense responsibility, dedication, and training demanded by each role. The program continued with a video compiled of clients speaking to the staff on what their compassion, professionalism, and expertise mean to them and their pets. The night concluded with a slide show of each employee at work played to the song “Simply the Best” by Tina Turner. A group photo was taken that evening. The picture remains framed in my office. While some of the faces have changed, the defining message remains clear of ultimate responsibility and of a simple promise to work hard each day to fulfill. Nothing is more rewarding than to turn out my office light at night, walking out to my car and thinking “I saw someone grow today.”

A simple promise. It is time for you as a practice owner or manager to think about creating your promise. A journey to create a culture and environment with challenges growth, and opportunity for your staff. A renewed dedication to ultimate responsibility. At times you will have to be innovative and creative. The satisfaction you will endure while establishing long term professional relationships with your staff will benefit your practice immeasurably.
Make Your Next Team Meeting Work  
Brian Conrad, CVPM  
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At one point or another each of us discussed the possibility of holding a staff meeting to get everyone together to solve the day to day problems the clinic is currently facing. It seems like a good idea but when I do talk with hospital owners and managers I generally hear the following about their staff meetings:

We don’t have the time, we can’t get the staff to attend, they just turn into complaint sessions, or they are so expensive. I agree, with these thoughts I wouldn’t want to have meetings either. In fact, five or six years ago we had similar sentiments about our staff meetings at Meadow Hills Animal Hospitals in Kennewick, WA where I manage. I can remember a meeting that was so dull, boring and unproductive I was ready to give up in the idea of having meetings all together. I am so happy we pushed through those difficult times and now have excellent staff meetings each month. If some of the descriptions above sound like your team meetings it might be time to reevaluate and make some changes.

Before we are able to jazz up our meetings we needed to offer some additional structure I like to refer to the who, what, where, and when. Without the structure we have no basis for determining success or failure.

Our first goal was to focus on purpose for the meeting. What is it we wanted to walk out at the end of the meeting and be able to say we accomplished? Spending some time a week before hand creating pre-determined objectives set the tone for the meeting. Based on the objectives we were able to create a timeline to run the meeting off of. The timeline and objectives communicates a plan and places importance on the meeting itself. Our objectives are revised each meeting but include housekeeping items, pharmaceutical updates, client comments and concerns, staff education, team building and more.

The second goal our management team put in place was consistency for conducting the meetings on a regular basis. Whether you determine your practice needs weekly, monthly, or quarterly meetings it is important to show the staff you are committed to the meetings on a regular basis. We so often speak of creating value for the client but in this case we need to create a perception of value in the team’s eyes. On August 2nd 2016 Meadow Hills Animal Hospital, will conduct their August general staff meeting. While I cannot tell you the exact agenda at this time, it is important the staff understands the importance of these meetings. Meeting dates are schedule out a year in advance and are included on each employee’s schedule to demonstrate that importance.

The third roadblock we wanted to overcome at Meadow Hills was a proper environment to hold the meetings at. For many years we used to have our staff meetings in the treatment area of our hospital. As the staff grew so did the problems associated with this incorrect environment. Between the IV pumps beeping, the 6 month old puppy barking and wanting to come out and play, it was hard to get the staff to focus with so many distractions. We even had staff sitting around corners not able to make eye contact with each other. With some thought and investigating we were able to locate several reasonable alternatives. Economically priced hotels such as the Holiday Inn Express or Comfort INNS usually have smaller meeting rooms that can be rented for a reasonable fee. Often times these hotels are willing to negotiate a special rate if you are able to commit to holding your meetings throughout the year. Another idea we found to work for smaller groups is many of these hotel properties have continental breakfast rooms. The rooms have no purpose the rest of the day making them prime picking for holding your staff meeting in them. We also found a pizza parlor down the road that has a meeting room. We are offered the room for free with the purchase of lunch. With some creativity and negotiating we are now able to have our meetings in an effective environment for under $75. A small fee to pay to obtain everyone’s attention during such an important time.

Well the next question raised is who is going to take care of our clients as we are off-site for our meetings. The solution we have been using for over five years was hiring a part-time receptionist with her sole job being to answer phones, make appointments, and sell products while we are away. The added benefit is when our full-time receptionist request time-off we are able to use our trained staff meeting receptionist to step in.

With the structure in place we are now able to concentrate on the nuts and bolts of the meeting. As I said earlier our first goal is to create objectives you want to accomplish for your own meeting. Below are some examples of activities that you may want to include on your next agenda.

Continuing Education for the entire staff plays a vital role to our success here at Meadow Hills. Because of the significance to the training we schedule 30 minutes of the meeting to be dedicated to staff CE. The education sessions may be on medical topics, pharmaceutical supplies, client service, or other business related topics. We select a veterinarian or senior staff member to prepare and present the presentation. We make the presentations client useful. By client useful I mean we concentrate the information to be relevant to questions and concerns clients will have. Our staff doesn’t need to know if a specific antibiotic crosses the blood-brain barrier but they do need to know if the medication needs to be refrigerated and given on a full stomach. I have listed two our recent topics to give you an idea of the information covered.
Ears, ears, ears
One of the number one reasons clients get frustrated and leave a practice is the mismanagement of ear disease. Pus, bacteria, yeast, blood, fungus, mites, foreign bodies, and the list goes on. How do we diagnose but more importantly how do we treat and prevent reoccurrence. Home management and patience are the keys. Are we supporting our clients well during these frustrating times?

Dealing with difficult clients
The holiday months bring out the best and worst in all of us. What do we do when a client comes in with an agenda to attack? We know it is not our fault but they are standing in our lobby. What to do….what to do? Sometimes running out of the building is not an option.

Pharmaceutical Representatives are excellent resources for additional education for the staff. Take advantage of the opportunity and let the hospital and your clients benefit. When using one of your reps take the time to meet with them and discuss the agenda on what it is you would like covered. There is no sense in discussing a product to the entire staff if this is a product that will never be carried in your hospital. Make sure the pertinent information to be discussed will challenge the entire audience and be useful.

We have taught the staff to live and breathe customer/client service. We have spend 10-15 minutes talking about positive and negative personal experiences by staff members with other businesses. When client service is brought on a personal level the desire to improve and exceed expectations is heightened. Staff is encouraged to share a story about an experience they have had in the last month. From there we learn from the experience and relate the experience into the veterinary clinic talking about how it could help or hinder us in a similar situation.

Changing pace for an hour can be beneficial to the team in many ways. Once a quarter we try to find a team building activity for the staff to participate in during the staff meeting. It is a time for the staff to switch gears, get to know each other and learn to work with each other better. Below are a couple of examples of successful events.

During our February staff meeting a few years ago we had an employee come up with a brilliant idea in keeping with the Valentine themed month. Each team member was given a package of valentine day cartoon cards. The ones you see in stores for the elementary school children to pass out in class. Over the next week staff members took the time to fill one out for each of their co-workers. On the cards they listed out at least one positive trait they admired of their co-workers. At the staff meeting time was taken to hand out everyone’s cards in their own decorated valentine bag. The staff then opened each one and received over 30 separate compliments. In addition the staff went around the room and read one of their cards. What was extra special was each of the staff took the cards home and their families were able to see how appreciated and what vital roles they play for the clinic on a daily basis.

Last summer we created a photo scavenger hunt for our 3rd quarter team building exercise. We allowed 1 hour for each team of 5 employees to venture out into the community and look for items listed on a sheet of paper. Each item had a different point value. The team with the most points at the end was deemed the winner. We accomplished our main objective which was to have a fun outing together after a busy summer. Two added benefits was the staff bonding that took place and the strategy and thought process that was used by the teams. I will never forget one of our new receptionists commenting on how fun one of the doctors was as he got to know her better.

As our staff meetings positive momentum grew, we wanted to be able to measure how effective our communication was during the meeting. We developed and implemented a consistency quiz to administer to the staff at the conclusion of each meeting. The purpose is to establish consistency and show value for the time and money spent on the meeting. The quizzes are taken on an individual basis. They are reviewed and corrected over the next few days. Any misunderstandings are discussed assuring everyone is on the same page. This tool is a keystone to our success. Questions are pertinent to information discussed at the meeting and usually are 10 questions in length.

Celebrating Success is a great way to end your staff meetings. We spend the last 5-10 minutes focusing on what is going right. This may be client retention or compliance rates, it may be someone’s work anniversary of 5 years; it may be a heart felt thank you card from a client. Whatever the case may be we all have success stories in our clinics that need to be celebrated and the staff congratulated on being a part of it. Focusing on this behavior and making a big deal of the clinics accomplishments will only encourage similar behavior.

With some creativity and prep work your staff meetings can be revamped into an efficient fun, and productive time. Your goal should be to leave the meeting with you and your staff on the same page, feeling energized know decisions have been made and positive changes are coming. So take the challenge today and see how you may jazz up your next staff meeting.
Preparing Your Hospital for Change

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We all have had some type of success in our practices. Some more than others. But what differentiates ongoing success is for the ability to realize we must continue to be innovative and change to meet the demands, wants, and needs of our clients and future clients. Where we tend to slip up is we see and feel success in our practices and we have a human nature element in all of us to sit back and say “finally, we made it”. I made this mistake at the two practices I manage in Kennewick, Washington. It relates to our client service. We held weekly meetings, we created staff incentives, we solicited client feedback, reviewed other companies out of our industry who we could learn from. The list goes on and on in all of the effort, training and attention we placed on client service.

One day the phone rings and we promptly answer it with a welcoming message. It turns out to be a national company that would like to use Meadow Hills Animal hospital and 4 other companies in the United States to create a new training film on customer service. Wow, you can only imagine my excitement along with practice owners. We had done it. We have created the most amazing client service experience for our clients and now others want to duplicate it. A sense of excitement but also relief had swept over me. I could now concentrate on other areas of the hospital because it was obvious we had mastered the experience process and we were done in that avenue. Fast forward 24 months after filming. Our client retention is slipping, new client counts are down, and overall client satisfaction is at its worst levels since the company’s inception.

What had happened? We were making the BIG mistake so many companies do. Once you find that magical formula you just sit on it like a sacred cow. A naïve perspective of thinking that once you get there you can sit back and enjoy the fruits of your labor.

We now knew that whether it was our client service, our pharmacy sales, or our marketing programs for dentistry that we had to continue to change and evolve to continue that success we wanted for the company. It was a hard lesson to learn but one we try to reflect on often so that we never forget to change, grow, and mature.

Perform a self-evaluation

- How would you describe the culture of your veterinary clinic as it relates to change and innovation?
- What do you believe is the most unique or distinct feature about your veterinary clinic?
- What concerns do you have about your competition or possible competition that would dilute out your uniqueness?
- If you had a magic wand and there were no obstacles in your way, what is one thing you would immediately change about your organization? What would you never change?
- If your clinic closed its doors for good tomorrow, what would your community and the industry lose?

The idea of change is relatively simple. The idea if implementation and accountability is at the heart of innovation. Over the course of the next hour we will look at real life examples of innovation and change implementation.

We will look at techniques and systems to get staff excited and on board. The goal of the session is to have concrete objectives to take back your practice and start implementing immediately. But this of course is “subject to change”!
Attracting new clients

The World Wide Web can be a mysterious and confusing place. Is Google just a website or is it an all-seeing Internet-empire? Does my veterinary practice need active Social Media accounts to help me reach a wider audience and succeed online? And how important is it for me to optimize my visibility online anyway?

Luckily, I’m here here to answer all of your questions and help you navigate the myriad of digital elements that come with running a successful veterinary practice in the 21st century. To answer briefly; Google is both a website and an empire, Social Media is an imperative medium for most if not all businesses, and Search Engine Optimization is crucial to your success. I’ll be focusing on the later in this month’s blog and will provide you with 3 simple and proven steps to improve your Search Engine Optimization (SEO) strategy.

Simply put, SEO means page visibility, and your natural page rank across online search engines. SEO can result in an immediate boon to your practice, as a surprisingly large percentage of business starts online with a simple Google search. If you’ve heard me speak before then you already know Google is the #1 search engine (by far). In fact, in 2014 alone we collectively searched Google trillions of times. We searched so much that it prompted Google to release a “Year In Search” compilation, which as a side note, we recommend watching since it just might make your day.

Why focus on Search Engine Optimization and make sure that this element is as fine-tuned as the rest of your business? Well, if you remember our “Safeguarding Your Online Reputation” article, you remember that Google has “Crawlers” which comb through billions of web pages from all across the Internet, algorithmically analyzing, assessing, ranking, and indexing web pages. They rank pages based on a multitude of factors (known as an algorithm), but the key to a successful page is to leverage the factors that you can control. A better page rank means a stronger reputation and more visits to your website, which ultimately will convert to more pet owners walking in the door.

That last line is the crux, so hang on to the takeaway: a successful SEO strategy can convert for your business and thus provide more money in your pocket. Great! I understand. Now, how do I actually implement this and apply it in real-time?

Top 3 tips to improve your SEO

1. Secure as many positive Google Reviews as possible for your business. Again, we encourage you to read our previously published “Safeguarding Your Online Reputation” for a full analysis of Google Reviews, but simply put, this is a crucial element to enhanced SEO. Reviews instantly provide credibility to your business, while bumping you up in Page Rank.

2. Head over to the ‘Moz Local’ website and register your business. This simple tool will help local search engines and new pet owners find your practice online, all while enhancing your SEO efforts. While you’re at it, go ahead and register your business on Yelp, Google+ and Bing. All of these tools help to enhance your Page Rank, and will allow your business to be recognized easily across each search engine.

3. Make sure your content has a naturally included list of keywords that people will use when they search for you. These keywords shouldn’t be forced or crammed excessively into your website. They should however, be clear, confident and relevant. A few solid keywords might be “animal hospital”, “veterinarian” “animal clinic”, and the city or region you’re doing business in. All of these words help Google pinpoint who you are, what you do, where you’re located, and effectively deliver results to users with more accuracy.

Measure client satisfaction

Google has a remarkably comprehensive system in place for coordinating user search results and delivering them in a timely, deliberate and meaningful fashion. What might seem like random results at first glance is actually a highly methodical process unfolding in real-time. “Crawlers” (know singularly as Googlebot) continuously sort through billions of web pages from each corner of the globe, algorithmically analyzing, assessing, ranking, and indexing web pages and sorting through each applicable result.

So what does this mean for you? It means that when a pet owner is looking for the best care online and nonchalantly typing in “pet hospital in your city”’; your results aren’t random. In fact, they’re far from it. Google, which accounts for approximately 70% of search engine market share, is calculating your keywords and weighing-out millions of results before delivering them to you, all in about 0.5 seconds flat. The key takeaway here is this: practice owners can, and frankly need to, monitor and enhance their practice’s search results in order to maintain and leverage their Online Reputation.

For example, when searching online for a local veterinary practice, customer reviews will gravitate toward the top of the screen, and instantly display the name, website, address, phone number, and here’s the kicker, one to five star average rating, of nearby
practices. In a Google Search (by far the most common kind of online perusal) this one to five score is the combined average rating of the “Google Reviews” that have been left for your practice. These scores can dramatically influence the amount of new clients that find, and ultimately choose your practice. This is why practice owners must safeguard and even encourage the establishment of a positive Online Reputation in order to fully optimize their practice.

Ok, so Keyword Searches and Google Reviews are all important factors to consider when managing my Online Reputation. But what can I do about it and how can I use these tools to enhance my practice? Well, I’m happy you asked!

Quick tips: Improve your online reputation

1. First, you’ve got to search for your veterinary practice online. If you’ve never looked, you might be a bit intimidated at what you might find. But you can do it, dive right in. We recommend that you search on Google, Bing, Yahoo and Yelp, and then “Claim Your Listing” once you’ve found the right result. Verify that this truly is your practice, and presto, you’ve claimed your online listing.

2. Next, setup your online alerts. By using both google.com/alerts and mention.net you’ll have two powerhouse services monitoring the Internet for results about your practice. Now you’ll be notified by email whenever a new result or review is available. Convenient? I think so.

3. Now, bookmark your top online listings in a folder within your browser. This will save you time when you wish to check back on your results, and lets you easily review your listings at least several times a month.

4. Here’s the fun part; engage with positive reviews! The average veterinary practice receives one review per month. You can reply to a positive review online, call and thank the client, or get creative and send a personalized Thank You card. Engage how you see fit, but definitely reach back to those who’ve been impressed with your practice. This little bit of effort can most definitely go a long way when it comes to encouraging positive reviews and ensuring client retention.

5. Finally, go the extra mile. You can engage directly with happy clients at checkout by encouraging them to leave a positive review for your practice. If they’re onboard, send them a friendly reminder email at the end of the week, with a direct link to help them leave a Google Review.

Say thank you!

In a world that’s moving this quickly, who really has the time to say “please” and “thank you”? We might think that our world is so constantly filled with stimuli, that maybe nobody would hear it if we said, “Thank you”, just a little more often.

It may feel like these tiny, syllable-sized gestures are antiquated or meaningless in our modern day environment. However, this couldn’t be further from the truth. In fact, those precious two little words might be more important now than ever before. Scarcity solicits demand, right? We might be busier than ever before, but perhaps our need to express gratitude is also more prominent than ever. At Simply Done Tech Solutions, we believe this to be true.

Our need to express a heartfelt, “Thank you”, has never been more relevant or imperative, than it is right now.

Despite what you might have been told, this simple phrase is emblematic of a whole lot more. It can make or break a friendship, or even a relationship with your own clients. In the wide world of veterinary medicine specifically, we are so dead-set on acquiring new clients, we hardly designate enough attention to tell our existing clients how much we appreciate them, and thank them for coming in. Now why would we work so hard to build our practice, market effectively, provide stellar service, and stop short of giving thanks?

A few simple phrases can help you to retain your clients, but even more importantly, create an ongoing, genuine bond of solidarity and trust. I recommend that you make saying the following few sentences a habit. You’ll thank us later:

“Thank you for bringing your pet in to see us. Thank you for being a wonderful pet parent, and most of all, thank you for choosing to trust us with your pets health care needs.”

This type of response to a new or established client may only take seconds to say, but can literally make a world of difference. Of course, it’s got to be genuine, and of course you’re busy and of course the phone is ringing again. Still, you can’t overlook the importance of gratitude as a cornerstone of building any healthy relationship.

The central point here is this; the effort really matters, because we really matter. Simply taking the time out each day to thank your clients and let them know explicitly of your appreciation and their importance can be surprisingly rare. In this fantastic 3-minute video, Dr. Laura Trice explains the importance of simply saying, “Thank you”, and how this relates to some of our innermost needs.

I used to work closely with a widely respected veterinarian, Dr. Eddie Garcia (no relation, I promise) who would call each and every one of his clients within 72 hours of their initial visit. He would do this with no ulterior motive or hidden reasoning. He would simply call to say “Thank you for visiting our practice. If there is anything we can do for you we are only a phone call away.” He strongly encouraged both positive and negative feedback, in whatever form it came. He would use this feedback to learn about the wants, needs and fears of his clients, and thank them if their visit was sub-par as well. I can hear you asking, “Wouldn’t this level of openness leave him vulnerable to hours of time-consuming critique?” While that’s a fair question, the kicker is this:

- A majority of phone calls were left on an answering machine (well, voicemail box now days).
• People were so excited about the calls they called him back just to thank him for him calling them.
• Dr. Garcia boasted a 90% success rate of retaining upset or displeased clients.

I watched Dr. Eddie Garcia make this type of phone call everyday for over 10 years (watch him in action). Yes, he really made these calls each and every day, and yes, they really did make a difference.

Calling both new and existing clients is equally important, and can’t be emphasized enough.

Whereas most of the time clients might simply express their grievance or general feedback to a spouse, if anyone at all, Dr. Garcia used their direct feedback to forge a bond, improve his practice and retain his clients in a single call. You can do the same.

When I used to ask Dr. Garcia what motivated him to go above and beyond to make these phone calls, he had quite a simple explanation: to stay true to his mission. In his mission he outlined that his veterinary practice “will meet and exceed expectation”. These phone calls were his little way of making sure that he exceeded his client’s expectations of what an attentive and caring veterinarian looked liked. And it did.

From phone calls to Vetstreet’s “Thank You” emails, there are plenty of ways to effectively implement gratitude into your veterinary practice. Here is a 3-step-solution to implementing ‘Thank You’ into your practice today:

3 steps to saying thank you

1. Implement a protocol to have your team print two reports at some point, consistently, each day. These should consist of two parts: (a.) New client report from the day before. (b.) Appointment schedule report from the day before.
2. Decide in your practice who the appropriate person is to make the call. I usually recommend that associates call their own clients in order to create a genuine bond. If associates do not have the time to do so, the practice owner or medical director may make the call. I’ve recently heard the idea of practices delegating this responsibility to a receptionist or technician. The reason they do this is because they’ve express that pet owners are more likely to share a negative experience with the receptionist vs. the owner or associate. Choose the person who you feel would be great at taking this task on (there is no better person than another in my opinion).
3. Begin by calling all new clients and only choosing 3-5 existing clients from the appointment schedule report from the previous day. You don’t need to call back every existing client to say thanks, but spot-check and call a few.
Communicating to a New Generation of Clients

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Texting pet owners

Whether it’s at home or in the office, we know that communication can make or break important relationships. Solid communication leads to both trust and transparency, allowing you to enhance your connections and grow a network of strong, confident relationships.

With over 90% of adults in America utilizing cell phones (a statistic that is also rising), I think it’s a perfect time to address the benefits of text message communication when it comes to contacting pet owners. Leveraging this technology now puts you a step ahead of the curve, allowing you to send friendly updates and messages to a pet owner that may be curious about how their pet is doing, or may have simply forgotten about an appointment. The beauty of the text-message feature is that you can customize and control your messages at all times and communicate with any pet owner in mere seconds.

I recommend utilizing one-way text messages with pet owners, because this approach has some distinct advantages over two-way texting that is more commonly used when engaging with friends and casual colleagues. There are a variety of reasons that go into this recommendation, but the predominant reason is because two-way text messages open you up to non-stop communication, a whole host of follow-up expectations and perhaps most importantly, potential legal repercussions. Instead, one-way text messages allow you to dictate the content and tone of communication, insulating you from additional liability while still giving you the benefits of instantaneous communication.

If you’re interested in getting started with a text message service for your veterinary practice, I recommend using free tools such as textport.com, or purchasing a cell phone line (which typically will run you less than $12 per month), effectively skipping out on the extra phone but using the extra number. Once using the extra line, you can download the texting app for that specific carrier that work specifically with your mobile device (whether it’s iOS, Android or other) and program effective auto-reply statements such as the following:

This is an unmonitored number; Please call our practice directly at (xxx) xxx-xxxx
This allows you to send a particular pet owner in the right direction, without assuming the liability of the two-way text message.

You can also easily set auto-reply messages and keep track of any messages that you’ve sent out.

I recommend sending pet owners simple and concisely constructed text messages whenever a pet is dropped off for surgery, a procedure or appointment, as well as boarding or grooming services. Even just a simple text message can help you to alleviate any potential worry from a pet owner, and demonstrates your accountability and care in one fell swoop. Something simple, yet compassionate will do:

Hi Mr. Garcia! Elvis is doing great and is recovering from his procedure. We will call you shortly!

Thanks to rapidly accelerating forms of technology, text messages can be sent to any type of mobile device, and even the new Apple Watch (or any wearable technology)!

Even a brief text message can go a surprisingly long way when it comes to building a steadfast bond with a pet owner. Not only are you adapting to new technology, but you are going above and beyond to stay in touch and alleviate any possible concerns that could crop up along the way. This is a significant way to differentiate yourself from competitors, cut down on extra calls from pet owners wanting updates, and show that you’re truly committed to every step of the client experience at your practice.

Yes, new technology does open up new avenues of liability, most of which are covered by opting into the one-way text message feature instead of the two-way. Still, you will need a consent form from your clients before sending them text messages. You can easily capture this consent by adding a brief, additional section to any new client registration form, or a drop off procedure/authorization form. While the adjustments are simple, they’re important steps of creating a technologically equipped infrastructure for your veterinary practice.

The resources at your disposal don’t stop there. There are also third-party companies such as Vetstreet, Petly or ePetHealth that allow you to send pet owners text messages regarding due and upcoming appointments. These services seamlessly integrate with your existing practice management software, and will send text messages to clients automatically. Automation is absolutely imperative for this level of communication, and some services like Vetstreet and Petly will offer two-way texting for confirmations. A simple, “Y” or “N”, will allow clients to confirm or dissolve appointments, and a list will automatically generate to display who has confirmed their appointments and who has yet to do so. You can then make follow-ups as necessary, leveraging the convenience and quick communication that these services have to offer.

The bottom line for your veterinary practice? These features are an outstanding way to increase compliance and reduce missed appointments that cost your veterinary practice a significant amount of money over time. It’s proven that pet owners are more likely to respond to a text than a phone call, and by adapting to text-message technology off the bat, you’ll be quick to notice increased compliance and efficiency within your veterinary practice. Still, it’s important to remember that this form of technology isn’t meant to
replace more traditional methods of communication such as routine phone calls, but is instead meant to supplement traditional communication. Calling your clients regularly remains important, but is in fact complimented by the extra effort of text-message communication.

It’s never too late to get started. In fact, the sooner you adapt to these recommended features, the sooner you’ll start to notice results.

Mobile website
When you work in Information Technology, keeping up with Google is a lot like checking the weather. You can avoid keeping up with what’s coming if you want, but by doing so you risk being caught off-guard in a downpour. Simply by thumbing through the most prominent tech blogs on a routine basis (in conjunction with setting up a few Google Alerts) it’s easy for me to keep track of what major changes are coming about in IT, and on the web as a whole. This month, I want to share with you a major change that’s recently occurred to the Internet-giant Google, and their approach to web-ranking. We’ll explore what you can do to leverage their most recent changes, and explore how to utilize these changes to enhance your veterinary practice and garner more business than ever.

The announcement that Google would be factoring mobile compatibility into their comprehensive web-rankings beginning April 2015, caused immediate shockwaves across the web. No modern day business is truly exempt from these changes, that is unless of course they don’t have a website, or use the Internet at all (which is an entirely different issue). Modern, competitive veterinary practices rely more than ever on the web, and consequently, they rely on Google too. That’s because Google sets the tone for what exists on the Internet as a whole, controlling, monitoring and distributing an unthinkably large amount of data each day. In fact, Google possesses over 65% of all search market share (yes, feel free to confirm this statistic with a Google search), effectively making or breaking what exists on the web in any capacity. While it may be easier than ever to start a blog or launch a website, it’s much harder to truly thrive on the web. This is why my goal is to help you adapt and leverage the absolute latest technological developments to enhance your veterinary practice.

As of late last month, Google has overhauled their web-ranking system, and is now incorporating the functionality of websites on mobile devices as a major component of Google Rankings. I know that pet owners and veterinarians alike use their smartphones more than ever, and Google has inevitably caught onto this trend. If a website is not optimized for mobile, it will now fall dramatically in search rankings conducted on mobile devices to reflect this shortcoming. In other words, if your veterinary practice website is not comprehensively optimized for both mobile and desktop, your business is at risk of losing traction and falling behind the competition. On the other hand, practices that can rapidly adjust may be able to outpace competition that has faltered behind such adjustments. If practices choose not to adapt to the new formula, they risk losing out on a significant amount of business.

The trend of accessible data on the web is now skewed toward mobile, and Google has fully adjusted to this reality. Comprehensive Simply Done Tech Solutions analytics finds that on average, 33% of all organic (non-paid) traffic generated to a veterinarian’s website is from mobile. It’s important to note that this percentage is increasing over time, and in some cases, veterinary practices actually secure more mobile traffic than they do via desktop. So what makes a good mobile website, and how can you ensure that you’re well equipped moving forward? Look below for some examples of a successful mobile build, and read on for a step-by-step guide on how to optimize your veterinary practice’s website.

A good mobile website strips down the best of a desktop site, and makes it easily available for the user to access on their mobile device. Mobile devices are smaller and more compact, and thus the aesthetics and build of a proper mobile website reflects this. A good mobile site will emphasize big buttons that are accessible on the go. This might mean a tap-to-call button that lets the user call your primary business line with a single finger-tap. The same functionality can be lent to locators and GPS features that can ostensibly bring a new client right to your door. A proper mobile build will show off the best of your brand with a custom color pallet and seamless links to Social Media. Well-conceived and implemented mobile builds will also boast simple bits of information that any prospective or current customer might need to know (think location, phone number, slogan). Now that we’ve got the basics down, let’s look at a simple step by step process to adapt your website to mobile, today.

Online patient portal
Let’s start off by understanding what an online patient portal is. An online patient portal communicates information from your practice management software into an online secure patient portal. Information that is transmitted usually consists of the patient’s name, birthdate, reminder dates, prescriptions recently filled and great educational material. Depending on whom you choose as your online patient portal provider will depend on the information transmitted online. Clients register for their online patient portals by registering online via the clinic website or by inputting an email address into the client’s electronic file. Once your client registers and accesses the portal they will see their pet’s up to date information.

The benefits of an online patient portal greatly exceed the total cost. Clients who have access to an online patient portal help increase the compliance percentages in your practice. They are able to see when their pet is due for services and read about the importance of the service due all in one place 24 hours a day 7 days a week. Reminders are automatically sent to the client’s email
address on file. Prescriptions are listed allowing clients to easily request a refill reminder by email and even request the refill online. An online patient portal is full of other fun features for clients. They can upload photos, blog with other pet owners, read about pet care and more. By having an abundance of information on the patient portal for clients to view you are increasing the likelihood of the number of visit. Your practice name and information is made prominent throughout the portal so clients can familiarize themselves with you and your practice.

**Email reminders**

It is difficult to think that 100% of all mail makes it to its desired destination. An incorrect address can be the key reason for mail being delivered to the wrong destination. A majority of practices still rely on the postal services to deliver reminder cards to their clients. There is a good reason why. Statistics show the return rates on post cards are still very high. It is not recommended to remove post card mailings in your practice. However, with our new generation of clients relying on e-mail as their primary form of communication, it is important to implement an e-mail reminder protocol for your practice. If the reminder post card never makes it to the client the probability of that client coming in to satisfy their reminders start to decrease. Even though pets are a priority in house holds across America they still rely on you to remind them of what medical attention their pet(s) needs.

To implement e-mail reminders in your practice start off by contacting your practice management software technical support team to see if there is a feature already offered to you at no additional cost. If not, consider moving toward a patient portal where clients will greatly benefit from not only e-mail reminders but a variety of other online services. Finally, if all else fails consider getting started with your own method of e-mail reminders. Start off by contacting an e-mail messaging provider. Talk to your practice management software technical support team about extracting reminders and client e-mail addresses in one file excel file. This file can easily be uploaded into the e-mail messaging provider’s database and sent out as an e-mail reminder.
By the ripe age of twenty, I was strapped in as the VP of a competitive veterinary marketing firm. At twenty-three, I had launched my own firm entirely: Simply Done Tech Solutions. By clueing in early to the necessity of digital adaptation, I was able to arm myself with the best technology, and instruct industry peers, firms, veterinary practices and friends how to do the same. Keeping a leg up in our world means having a finger on the pulse and an eye on the next step at the very same time. However, it’s this same constant connectivity that can take a hazardous toll on attention spans, peace of mind, and ultimately cause some nasty burnout along the way. So how do we leverage technology, but do so with intention? How can we maintain our status as technological savants at Simply Done Tech Solutions and maximize our efficiency, yet still remain cool, calm and collected? This month, I’m proud to present you with #UNPLUGGED:

I’ll give proven tips on how to instantly maximize your effectiveness, while making sure you stay master of your digital tools, rather than the other way around.

In 2014, we can get more done in twenty-four hours than we once would have ever thought possible. If your iPhone alarm clock rings at 5:30AM, you can pop on your automated coffee maker by 5:45AM, take the dogs out for a quick walk, and be finished replying to emails by the time you crack into work 8:00. At this rate, you might even be able to finish the never-ending stack of callbacks you’ve been working on by 2:30PM, and still have time to order GrubHub off your iPad for a late yet impossibly convenient lunch.

There’s only one problem here. You might just be moving so fast, that you can hardly taste your lunch or even slow down by the time you get to dinner! The frenetic lifestyle brought on by instantaneous Internet access may put us ahead by leaps and bounds when it comes to doing our work, but it can also wind us up and throw us into a myriad maze of never-ending tasks. This leaves us at odds, somehow increasing our efficiency but somewhat dissatisfied and always striving to do even more. Many of us wind up multi-tasking to increase our efficiency still, responding to texts in-between emails and trying to catch up on an interesting Huffington Post article with the leftover minutes before our next appointment.

A recent study published by Stanford has compiled some intriguing data on the effects of multi-tasking. I encourage you to read the report in full, but the summary is this: when you commit to too many things at once, you suffer, and the work suffers. Although we’ve gotten accustomed to doing more, we can’t do it as well or give it our complete full attention. Imagine trying to watch ten television sets at the same time. Now how much dialogue and plot can you really remember at the end of thirty minutes?

Ok, so what do we do about all of this? How do we increase efficiency while still avoiding burnout and Internet-fatigue? Simple. Just unplug. As hinted by the title of this article, you can have the best of both worlds and I’ll teach you how. Designate some time each year to regroup and get away from it all. Yes, that includes Facebook and Twitter. You don’t have to deactivate entirely, but log-out and take a long and intentional break. Maybe even change your passwords to something that can’t be input with muscle memory. Prepare in advance, pay bills early and set up auto-responders for your important email accounts. When done correctly, you can unplug for as little as a week, but as much as a month, without losing any headway on your projects or work. In fact, you might even find that when you come back from your digital sabbatical, you’re feeling better than ever. For me personally, a bit of time to unplug from the world allows me to prioritize friends, family and myself. I find myself gaining a heightened sense of creativity and an eagerness to put my ideas into action when I get back to work.

Want some help getting started? Here are some tips that can help you kick things off when you’re ready to try your first official #UNPLUGGED session.

**How to #unplug in 5 simple steps**

1. **SCHEDULE Time Away**
   You don’t need to go far. Or you can go as far as you like. The point is, get out of the routine you’re in and find somewhere peaceful where you can achieve stillness and quiet. This could be a beach; it also could be a family or friends house. Find somewhere you’ve been meaning to spend time, and go!

2. **ALERT Important Contacts of your plan**
   Before you go, you’ll need to prepare. Inform your staff, your colleagues and friends of your plan to unplug. Make sure to prepare by wrapping up your current projects and sending a definitive message to those who should know.

3. **SET Up Your AuTo-Responder via email**
   Be thorough yet clear. Let people know you’ll be away and for how long. My away message looks a little something like this (It was inspired by Fast Company’s):
PLEASE NOTE:

I will be taking a digital sabbatical from June 25 - July 9. I will be completely UNPLUGGED from the world. During this time I will not be able to generate any activity through phone, email, or any social network, including, but not limited to, glancing, checking, syncing, wi-fi connecting, pinning, sharing, googling, commenting, liking, loving, tagging, favoriting, plus-oneing, tweeting, vining, messaging, texting, face timing, or uploading.

4. Establish Emergency Contacts
I recommend letting family and a few select friends know how to contact you in case of an emergency. You may have a landline connected where you’re staying, or a cell-phone that is disconnected from Social Media accounts and kept on silent. Even if you’re only gone for a little while, it’s important that you can be contacted if something important comes up.

5. Unplug and smile!
It has begun, you’re officially UNPLUGGED! Pay close attention to the sights, the sounds and the sensations around you. When you come back, you’ll be refreshed, recharged and ready to give 110% to exactly what matters most. I promise.
1. Manage, maintain and monitor
   - Why we don't fix people
   - Challenges ahead
   - New laws
   - Make a plan now
   - Understand your options

2. Changes in the labor market
   Generational influence
   Millennials will represent the largest part of the labor force beginning in 2016. Many more mature members of the workforce are moving swiftly toward retirement and in some cases resulting in a loss of business intelligence in the process, commonly referred to as skills gap. The needs, experience and expectations of the various generations in the workplace influence the management of your practice. Is this being considered and do you have a plan in place to survive this new ground in employee management?

   WHAT TO DO NOW-Update training to be diversified in how it's delivered, varying the methods to incorporate what will be useful for all team members. Consider special training for managers/supervisors on how to be connected to different generations. Reinforce that diversity of age is an asset to the practice and all team members are valued for their contribution to the success of your business. Succession planning is crucial to the success planning of a practice.

   Hiring practices
   The challenge for obtaining qualified candidates for open positions will continue to stretch many practices to the edge of their capability through 2016 and beyond. Job hopping will continue as the usage of smart phones have made it very easy to have instantaneous access to job opportunities. The latest assessment of the labor market identifies that nearly 86% of employees are looking for work at present and are evaluating positions outside of their current job type.

   WHAT TO DO NOW- Practice must have mobile accessibility to their website/social media along with career information to attract job seekers. Candidates want to apply through their mobile access so having an application online is not just a good idea, it's critical to capturing potential candidates. Consider upgrading to being able to apply online for positions, employee referral programs and aggressively looking for candidates all the time. Assess how successful the interview process has been in finding, and choosing the right candidates. Implement a strong retention program and reduce down the need for hiring.

   Immigration
   Executive Order that enables undocumented workers a path to citizenship or permanent status in the US through enhanced work permits and protection from deportation for up to 3 years if they meet an outlined set of requirements. Employers should prepare for individuals who identify that their status to work may be questionable and they now wish to meet the criteria as identified in the executive order. At the point that they are identified, they may be protected under certain state laws, while the employer may be in violation of the federal law for knowingly employing an undocumented worker.

   WHAT TO DO -Assure that the I-9 form you are using is the most recent (dated ___) and implement a policy that indicates penalties to employees found to have falsified their interview information or qualification for employment. Assure this is not a discriminatory claim, as it applies to anyone who would falsify their qualifications, interview information or anything specifically during the hiring and introductory periods of employment with the practice. Social media sites, blog posts and Face book need to have more information about what the culture at the practice is like and why employees work for the practice. Social media is being used advantageously to attract strong talent to a business.

3. Wage and hour law changes
   States are taking a more active role in the determination of minimum wage, in some cases even cities are declaring a minimum wage for employees within their geographical limits. The Department of Labor has committed to more aggressively pursue employers who in anyway do not comply with rules governing employee wages, this includes penalties/fines, and allows for civil liability as well. In particular, cases where employers were not negligent but were deliberate in their actions, criminal prosecution will be applied. Changes to what is a salaried employee must be paid at minimum is also changing in accordance with the FLSA Act.

   WHAT TO DO NOW-At least yearly, all positions in the practice should undergo review for compliance with state, municipality and federal requirements. Assure all employees are classified correctly and that all mandatory notices on new minimum wage rates are posted.
4. Medical and recreational marijuana- coming to a state near you

As Colorado, Alaska, Washington, Washington DC and Oregon are all illustrating to the rest of the country what recreational, not just medicinal marijuana can do for the financial well-being of a state, many more states are strongly considering getting on the "go green" bandwagon. While the states are enacting their own legislation, it's important to note that the federal laws have not changed and are not expected to in the near future. Off work usage and the impact on the workplace are still being studied while employers must decide what is safe for the business, and not invasive on employees off work activities.

WHAT TO DO NOW: Drug free workplace policies are becoming paramount in creating safety for employees, clients and patients. Any drug testing policy will need to reflect the updated laws of that state and how to manage any issues where an employee is demonstrating impairment that suggests adverse marijuana usage. All team members should have yearly training to assure they understand the intent and application of the policy. Managers/supervisors need special training in identifying situations where impairment due to marijuana usage may be present. Review your policy regularly as the laws are changing all the time.

5. Employee benefits

Paid sick leave -by city, by state, by number of employees

Many states have determined that employees should be granted paid sick leave to attend to medical concerns in themselves or immediate family members. The laws often include how the time can be used, how much time is available and who must comply. Compliance is often by the employer's number of employees and the number of employee hours worked.

WHAT TO DO NOW-As these laws can be very detailed and shifting, do make sure that your benefits plans, and other policies that might be covering this same topic agree with one another. If sick leave is part of a state law, it is likely that tracking that time separate from PTO is a good practice as well. Employees should be able to identify what time they have available easily either through online access or recording on their paystub.

Affordable Care Act- changes for 2016

In 2015, the Affordable Care Act (ACA) outlined a mutual responsibility requirement between the employer and employee on coverage. If the employer has 50 or more employees as determined by the formula provided, that employer must provide at least minimum coverage to full time employees. For an assortment of reasons, this was not actually applied to companies of 50-99 employees, but rather in a modified version to employers of over 100 employees. Now, in 2016, businesses with over 50 employees will have to offer coverage to over 95% of their employees not to incur a penalty.

WHAT TO DO NOW- Be sure to use the formula provided for the ACA to accurately count employees in application of this requirement and perform the count regularly if your employee numbers move in either direction due to seasonality, work load or operational elements. Employers must certify that they have not reduced their workforce to avoid having to comply with the ACA, look carefully and document how decisions are made regarding employee count.

Employee vs. practice privacy

Many employers are allowing employees to bring their own devices (BYOD) to work and use them for work purposes. This includes access to the business network, internet and applications/software. Questions can arise over privacy, data security and social media access.

WHAT TO DO NOW- Update Social Media Policy at least yearly. Track any issues you have during the year to determine what policy changes should be adopted. Have a BYOD specific policy. Maintain the appropriate level of network and internet security, enforce policy consistently among all employees.

Employee rights - volunteers and interns- categorize them carefully

Growing laws and legislation to protect individuals who should be paid for the contribution in the workplace- California Nevada, Washington DC, Illinois, Oregon and New York have laws in place for 2016.

WHAT TO DO NOW- Update your policy to comply with changes.

Accommodations for Pregnancy and Childbirth

Upgrades to current pregnancy accommodation regulations are being further defined on the state and on the federal level. Many states have specifically identified accommodations for pregnancy and childbirth along with similar medical conditions to entitle reasonable accommodations that have not been addressed in the past have been clearly outlined. These changes include modifying workplace schedules, redistributing job duties, job function and special seating.

WHAT TO DO NOW-do not just have a policy, have a written step by step procedure that all supervisors/managers are trained to use and assure that it is consistently applied. Remember that many of the pregnancy leave regulations include maternity as well as paternity. Follow up regularly with all employees utilizing this policy to assure that they are also informed and can provide feedback on updating on a regular basis. Evaluate current legislation - Family Medical Leave Act, Pregnancy Discrimination ACT, to assure the practice knows what laws apply and how many employees you have to have to require compliance, each law can vary on what commits you as an employer to have to be in alignment with the regulation.

Same sex marriage and rights of individuals regarding LGBT
While 2014 marked a landmark change in the allowance of same sex marriage, 2016 will likely show an increasing number of state laws that affect employers than in any prior years- these requirements include availability of benefits, rights of same sex spouses, and protection under the law against workplace discrimination.

WHAT TO DO NOW-All supervisors/managers should receive yearly training on assuring a workplace that supports diversity and respects employees as well as clients without discrimination or harassment. Training should include how to investigate claims and how to minimize employer liability. Assure that your benefits policies identify how to apply and assure equality in access to these benefits.

Workplace bullying
With nearly 75% of all businesses reporting that they are aware of workplace bullying, how to respond to complaints is essential. Bullying can evolve into a wide variety of issues that not only can effect productivity, absenteeism, morale, healthcare costs, and workman's compensation claims, but also enter into harassment, discrimination and civil liability.

WHAT TO DO NOW-Don't wait to become part of a statistic, set into place a zero tolerance policy accompanied by yearly team training. Employers should be quick to respond to any report and disciplinary action up to termination. Employees should be encouraged to report any areas of concern without fear of reprisal. Proactivity is key in reducing down occurrence.

Domestic violence
While domestic violence takes place in the home, it has an impact in the workplace, affecting productivity, attendance and longevity of employment. It is for these reasons that legislation and requirements are being adopted by federal and state agencies. The EEOC particularly has specifically outlined what is considered discriminatory in regard to victims of domestic abuse.

WHAT TO DO NOW: Employees are encouraged to report that they have placed restraining orders against spouses or significant others, or if they have concerns about safety that surround their place of employment. A safe environment with a protocol that all team members apply- this includes access of non-employees to private areas of the hospital, how to handle aggressive individuals, and safety in or around the practice. In some states, leave may be part of the state requirement for victims of violence for counseling or legal issues.

Ban the box
Many states are becoming more concerned that employers are using a rapid screening for criminal convictions as a measure to disqualify potential employees without further consideration. The law has been established in several states to not allow the question of prior criminal convictions to be part of the initial job application. Inquiry into past convictions (not just arrests) can be obtained later in the interview process or when the offer of employment is conditionally extended.

WHAT TO DO NOW: If not currently performing pre-employment background checks, it's time to re-evaluate. Remove any questions on employment applications that ask about criminal convictions. Train all team members about what is appropriate and not appropriate to ask during each stage of the interview process. If considering employment for an applicant assess how long ago the offense occurred, the serious nature of the conviction and if it specifically applies to your practice.
Video coaching can be a useful tool for identifying strengths and weaknesses in the consulting room. Sheila Grosdidier explains the benefits of this style of training, what to consider when implementing it, and what results to expect.

Remember the last time you went to veterinary conference and speaker shared with you some great insights about how to improve your practice, enhance your client compliance to recommendations or perhaps just how to assure you were being consistent with your wellness exams? You walked out of that conference room motivated, perhaps even inspired to change your process, encouraged to truly engage your clients to gain their acceptance of your recommendations. But somehow, between these great ideas and the opportunity to apply them in the consulting room, there is a distinct disconnect. This is the reality for many within a veterinary practice - doctors, nurses, assistants and receptionists alike. Most are already very competent and experienced at what they do, so in order to make a dramatic change in how a practice conducts a routine activity, they must be compelled to evaluate it from a totally different perspective. Each client's outpatient visit is composed of several different stages: a step by step consulting room protocol, a comprehensive patient physical examination, client discussion regarding health concerns, the veterinarian's recommendations, review of follow-up and the visit's conclusion. Videotaping makes it possible for all team members to review visits, examining all their component parts and identifying how clients respond to recommendations, body language and specific procedures, as well as how they act when alone in the consulting room. This is particularly useful as for most vets it is simply not practical (or possible) to make the same sort of systematic assessment either during the actual consultation of afterwards from memory. This article will cover a number of essential components that should be considered and implement in order to realize the full potential of video coaching in practice.

Is video coaching for you?
Take a moment and ask yourself these questions with regard to your veterinary practice:

- Are you certain that clients are being offered a consistent standard of care?
- Do team members have the tools and resources to meet the clients' needs?
- What future training do you see your team needing to best grow the business?
- How does your team best learn?
- What training have you specifically done in the past that has targeted client interaction on an individualized basis?

While there is little argument that the costs of training practice team members are rising, significant debate does exist over what methods of training will ensure consistency, enable the adoption of new skills, and create an environment that allows team members to coach themselves to achieve greater performance. When done correctly, recording employee/client interactions on video for one-on-one review can be one of the most valuable training tools available to change ineffective behavior into consistently productive client communication.

Communication
In 2005, a survey in the US by the American Management Association identified that more than half of employers use video surveillance as a way to reduce theft and violence (Grosdidier 2009), while only 16 per cent of those businesses used that information for evaluating employee performance (Guerin 2013). While some practices might already be using video technology as a method of monitoring for theft prevention or safety, the ultimate aim of video coaching is obviously very different. Those differences include how the information will be captured, what will be done with the video and how its information can change your practice.

**Box 1: Benefits of using video for coaching in veterinary practice**

- Team members can review specific aspects of a real interaction with a client, not simply go on what they remember about it;
- They can view and compare videos back-to-back to see improvement over time;
- They can review an interaction multiple times, focusing on things such as their body language, word choice, speech volume and cadence, and find something different during each viewing;
- They can see a complete visit from the client's perspective, not just their own; They can find out what clients do in your consulting room when they are not present, then think of what they could do to make this a productive time for them and the client.
One of the most important factors, and one on which the success of this process will hinge, is clear communication with your team members, assuring them that this will not be a covert operation but rather an openly acknowledged training tool for reviewing staff and client interactions and subsequent coaching for future performance. This is not about looking for mistakes; it's about looking for ways to improve. Each of us has had those moments when we replay a conversation with a client and wonder, 'Could I have done something differently?'. Seeing the entire interaction can provide clear answers. So remember that the team will need to be reassured that the main goal of filming is for those images to help them choose just the right words in order to communicate more effectively with clients in the future. Make it absolutely clear that it is not about policing the practice.

Rules for everyone
Because there could be some initial resistance from the team about being recorded on video, it's important to establish clear, steadfast rules that will be outlined and followed so as to reassure the team that this is a training tool to be used in a positive manner. Recording an examination should only proceed after the client has been advised and agreed. A consultation could begin, for example, by saying, 'Ms Jones, we are videotaping today for training purposes. Is that okay with you?'. Usually clients are glad to give consent, but if the client does not approve, then the camera must be turned off. Signs must be posted to alert clients and remind team members that the practice is using video cameras and that it is not a secret activity. While there may be a sense of natural apprehension at the outset of using video, over time, team members will probably have to be reminded that the video is running - it will become part of the landscape. Establish that the video will be used as a tool for training only, not for surveillance.

Implement a clear policy that outlines how the video will be recorded and evaluated to ensure maximum benefit to the employees as well as to the practice. To get the most out of the process, this should not be a one-off activity. Plan to evaluate it monthly and set goals with a time line for each team member, which can then be evaluated at a later date.

In some respects, this style of coaching is going to be as important for the growth of the entire consulting room team, not just for the individual that you are coaching on a one-to-one basis. Ultimately, this individual will model a change in behavior, creating a ripple-effect for others to follow. A policy example can be found in Box 2.

Box 2: Example policies for team video coaching
- The practice uses video as an individual staff coaching tool to evaluate interactions with clients exclusively. In the consulting room;
- Recording will take place in identified rooms on specified dates and times. All consulting room assistants or technicians, receptionists, kennel assistants and vets will be expected to participate, as the entire visit in the room will be recorded. The video camera is to be set up correctly so the entire room will be visible;
- All staff who greet clients and guide them into the consulting room are to state that the visit is being recorded and request the client's permission (eg, 'Ms Jones, for training purposes we are recording this on video today. Is that okay?'). If the client declines, the video camera must be turned off;
- Team members will have an opportunity to review their video and pick which of the clips they would like to review with their supervisor. Each team member will use an evaluation checklist and submit it to the supervisor before the review;
- The supervisor will review the clip one-on-one with the team member and complete the checklist for comparison with the team member's checklist. An agreed goal will be established for the next videotape session;
- An incentive could be included, for example, all team members who complete four video reviews in a 12-month period will receive a bonus and be entered into an annual draw for a day off.

Rules for everyone
Be sure to have your team members assess themselves before their review with their supervisor. It is likely that the team members will readily pick up on how to improve and know what they need to do before the review. This will help them feel more confident and supportive of your coaching over time. Reinforce this action by pointing out what they are doing on their own to improve and how that not only directly impacts the well-being of pets, client satisfaction and the health of the practice, but also enhances their skills. This is where an incentive program can accelerate results.

It is fair to expect that the team will be apprehensive when the initial videotaping begins, as it is a new experience and there is a tendency to feel self-conscious. Remember, the videotaping is not going to be taking place in all of the consulting rooms, all of the time. It's at training tool, not a surveillance activity. The goal is to have four or five appointments per team member being coached, which equates to about a few hours in an afternoon or a morning once a week. These should be reviewed about twice a month initially, and then, when the coach and the team member reach the desired results, they can be scheduled on a monthly basis.

Want to get started the easy way? Offer a reward to the first team member to try it. One clinic offered an afternoon off for the vet who videotaped the morning of appointments. The practice had no problem getting someone to volunteer, and when the vet told the rest of the team how well the process went, and that they learned good information from the coaching, there wasn't any issue with getting the rest of the team on board.

Star performers
Each year, many veterinary practices spend considerable amounts of money on professionally produced videos to train their team. However, practices that start filming and reviewing consulting room footage find that they start coming across examples of excellent work. Did you just watch a nurse give a perfect explanation of why a pet needs to have a senior profile? Did you see how well a vet responded to an angry client? When you witness these moments in the video, consider creating your own training video. This gives you a tool that highlights the stars you have on your team and which is specific to the standards in your practice. These in-house videos are amazing tools for training new staff. Ask your team members if they would be comfortable sharing their expertise by showing how to perform at that star level in such special videos. Creating a collection of these interactions is incredibly instructive to new employees.

Think positive
Your team members will probably be concerned that this will be a negative experience that consists of looking for what they are doing wrong. Demonstrate that it's about building on their current skills and creating positive reinforcement (Joseph 2013). Start the discussion by asking them to identify what they did well and then what they would do differently. Focus on the interaction, not the person.

Summary
Veterinary clinics should constantly strive to adopt new and better techniques and videotaping can be a valuable tool that can offer consistency when adopting new methods. How we remember our interactions with clients can vary and videotaping can provided an accurate and effective method of reviewing and evaluating these interactions in order to improve performance. Remember to create a clear framework and team policies for recording videos, coach vets and team members one on one to achieve the best results, and always set goals to make sure that objectives are actually achieved.

When clients are asked what was most memorable about visits, what do they say? Is it a vet's diagnostic ability or technical knowledge, or is it the connection that was made with the client? Video can dramatically identify what the strengths and weaknesses in the consulting room are and provides an opportunity to closely evaluate them in a way that no other instruction can offer.

References
Before you begin, a disclaimer. Frequently I find conference audiences interested in quick fixes that can somehow bypass the arduous, complicated work of leadership. There is no such quick fix. Still today’s growth tools, probably more so than ever before, are easy to use and extremely effective. Innovation, experience and technology have all converged to give practices a great shot at growth. Just keep in mind that none of these fly on its own. Every good growth and leadership tool requires a great leader to operate it.

Leadership training
Despite a lot of evolution over the past 20 years, the ranks of our industry leaders are still largely comprised of self-made men and women, eager to succeed, but not equipped with the experience, communication, and skills to accomplish every company’s ultimate goal: to get employees to think and act like owners.

Before you think about business growth, you should think about leadership. If you had a lemonade stand in Hell, it’s likely that initial business would be great irrespective of your leadership skills, but soon, the supply line of water and lemons to make your product and the number of employees falling ill from pitchfork pricks would erode at profits. Ultimately every business requires some good leader to get things to run smoothly, to get people to get along, and to get people to work passionately.

Fortunately, there are a lot of excellent sources of leadership education. Organizations like the CVC and AAHA have embraced the notion that complex concepts are best described using a variety of teaching styles and settings. Attend a conference by one of these organizations and managers may be asked to work in small groups, listen to panel discussions, attend Ted-Talk-like lectures, write essays, or sit in on roundtable discussions. These organizations are drilling down into catchall terms like leadership, communication, coaching and team building and asking both instructors and students, ‘what does this mean?’

The Veterinary Hospital Managers Association has had exponential growth in recent years, not just in membership, but also in the quality of their leadership education and their commitment to it. Both the VHMA and CVC-Advantahave a wealth of free leadership resources available in digital, print and video format. VHMA also encourages learning through mentorship and has a number of ways for managers to connect with other hospital leaders. Their online member forum is like the VIN of Veterinary Practice Management. It provides great information and support in a daily, ongoing feed.

Vendors like Zoetis and Merck know that there is a direct connection between strong leadership and strong sales. These companies have a wealth of free resources available to practices, world-class teachers that can be delivered right to the practice door. They can even outline a plan for how to continue both the professional and business growth after the class has ended.

If you and your practice are going to grow, you’re going to grow because of your leader’s efforts and his or her communication and leadership skills. Leadership education is better and more available than ever before. Go to the VHMA and look at the qualifications to become a certified veterinary practice manager. Even if you never sit for the test, the list of qualifications can serve as your own personal syllabus on a journey that you must take if you are going to grow your practice in the long term.

Employing technicians specializing in a field
There are more and more options for technicians to expand their knowledge base and grow their skills. This is excellent news for practices interested in retaining quality employees and getting workers to help grow the business as though it were their own.

In my work, I see more and more practice’s employing technicians who specialize in dentistry. Since their knowledge and experience can exceed that of the practice’s veterinarians, they can be an enormous asset. They can take over client education at the practice and, because of their credentials, bring a lot of credibility to what they say and teach. They can build out safety protocols for anesthesia and bring an added dimension of oversight to the entire surgery/dental department that ensures that the practice doesn’t just grow, but thrives. Their enthusiasm for what they do can infect the whole team. At the practices with which we work, the ones that employ techs with a specialty in dentistry bring in tens (if not hundreds) of thousands of dollars in dental revenue annually. Some of these techs lead departments that rival the production of the practice’s associate veterinarians. They are a source of inspiration for the rest of the team members. They teach the team that veterinary nursing is a career that comes with a good salary, respect, job enrichment, and lots of opportunities to self-actualize.

Along the same lines, companies like Oncura, a business that produces a ‘self aware’ ultrasound machine, can provide associates and technicians certified education on how to accurately perform both abdominal and chest ultrasounds, then watch their work (and if necessary, take over the controls) in real time. It’s been an enormous growth opportunity for some practices. By training technicians to do the work, valuable veterinarian time can be channeled in other directions. Live, remote oversight of the ultrasound process by boarded veterinarians ensures that the ultrasound images are diagnostic and interpreted accurately. In some cases, practices are using the Oncura device as part of a senior screening program that catches medical conditions that would otherwise go undiagnosed in the
traditional annual appointment. The service opens up a whole new level of care for owners eager to keep their pets living long, healthy, active lives and gives small practices an excellent way to compete with larger veterinary groups and specialty practices.

‘Want to be trained as an ultrasound technician?’ How do you think that sounds to an employee that’s been with you for years, proven his or her worth, and who is eager to continue to grow? Do you think such a growth opportunity would help you retain that employee? Could it be a way to have the employee’s earning power keep pace with his or her annual salary increase? The fastest growing hospitals are growing their business by growing their team members.

**Expense management tools**

Our practice managers should be on the floor where they can observe the team in action and trouble shoot efficiency and customer service opportunities. If they’re holed up in their office pouring over the schedule or breaking down invoices into specific expense categories, they’re not making effective use of their time.

During the lecture, we’ll talk about online scheduling software that dramatically lowers the amount of time spent working on the employee schedule. This software streamlines an otherwise tedious job, calculates total staff wage, reduces overtime, and helps practice managers to better budget the practice’s payroll.

Companies like Henry Schein allow practices to have their inventory invoices separated according to the accounting-expense-category to which the product is assigned. This really streamlines the data entry process. It means that total expenses, by account, can be taken directly from the individual invoices and loaded into the accounting software. The process makes comparing revenue in the POS software to expense in the accounting software straightforward. With just two reports, practice managers can make an immediate, accurate assessment about their inventory management and markup.

**Advanced communication services**

I recently was assigned an article for Firstline Magazine in which I was asked to explore managers’ thoughts on third party communication systems. I was shocked by how many (100%) felt that their communication company was ‘indispensable’. It was not because they were relying on them to handle their reminder cards, but looking to them for help with the small universe of other communication jobs we find ourselves having to do in today’s digital age. Here are few of those jobs and the benefits that they bring to practices.

Targeted emails: Communication companies now have a facile way of looking in your software and distinguishing patients by breed, sex, age, time that they’ve been at the practice, services that they’ve received, and so forth. Singling out specific subsets of your patients and targeting them for a service that directly meets their needs means less advertising money spent and a wider conversion rate.

Reputation management: Third party communication companies can help you keep track of what’s being said about you online and on which sites it’s being said. They can also help you stay proactive about your reputation by sending out surveys to clients before they turn to sites like Yelp to express their dissatisfaction. Surveys provide you with a more objective look at your practice’s client satisfaction. What’s more, your communication partner can assist you with driving these favorable reviews to more visible sites, like Google, where they are more likely to make a difference in your search engine results and how your prospective clients view your business.

Additional services include newsletters; help with social media sites, inactive client conversion campaigns, texting services, and more. With so much available to you, for so little money, relationships with third party communication companies are a sure bet. Be sure to understand fully what they are offering and to stay on top of monitoring their success at delivering it.

**Focus on compliance to preventative services**

More clients think of their pets as family than ever before. Services like dentistry, annual wellness bloods, and more involved services like preventative-health ultrasound screening or radiographs don’t seem like extravagances, but valuable diagnostic tools to keep loved ones remain healthy, active, and feeling good. Companies like Zoetas and Antech have been hard at work building tools to help practices communicate the value of prevention and increase team members’ ability to communicate that value.

**Portable and self-aware diagnostic tools**

A growing number of companies are making significant advances in ultrasound machines. Today, practices can purchase machines that are portable (SONIMAGE P3 by Konica Minolta) and that can provide remote, real-time feedback by boarded diagnosticians working off site (the previously mentioned device by Oncura). Portable laser machines can be rented to clients who can be trained to use them on their pets at home thereby decreasing the amount of time spent working on such patients inside the practice, increasing client compliance to treatment, and driving practice revenue.

**Payment solutions**

The past few years have brought in a new wave of payment solutions that put more veterinary services within reach of more clients. Companies like Vetbilling.com manage payment plans for clients off-site. Plans are written by the practice and on the practice’s
terms and the hospital collects 100% of the funds. Companies like Care Credit have made it easier for clients to get approved for credit and insurance companies like Trupanion, that offer a certificate for a free 30 days of coverage, help practices sell the value of pet insurance and to drive higher patient coverage rates.

We're living in a fast paced and quickly changing world. Our learning curve is steeper than ever before, but the wave of technology and change has also brought us a host of business-growing solutions. Never before have we had such an opportunity to make an impact on our clients, on the quality of our work, and on our savings and growth. Explore the vendor hall at this year’s CVC and be bold enough to ask for help. It’s essential to these companies’ success that you succeed. Go ahead and farm out some of the responsibility for growing your practice, just don’t forget that you will never be absolved of the responsibility for managing it.
Some time ago, receptionists were renamed client care representatives. Likely the name change was someone’s way of reminding all of us that the first people to whom our clients are introduced are tremendously valuable. Trained well, client care representatives have the ability to capture phone shoppers; to catch hundreds of minor, but important client and patient medical chart details; to increase client compliance, and to market services of the practice.

In this lecture we’ll talk about the job we want accomplished by these people that we seat at ‘the desk’; talk about how to engage them in what they do; and review the leadership elements that build a great team.

Client care representative job description
Your choice to go into business was a kind of commitment. In essence your decision to go out on your own and start your own practice was a statement: ‘I have something unique to offer. I have chosen to provide oversight to the care your pet receives because I want to ensure you quality. The outcome of your pet’s health is so important to me, I want to take full responsibility for every part of the service you receive at this hospital.’

If that’s true then ‘the people at the desk’ have to be more than phone answerers and note takers. They have to be a voice of the practice and really, a voice of you. When they pick up that phone, they should do it the way you would do it. They should pick it up as though they had the desire and power to make a difference.

Start to build your veterinary reception team with an understanding of what you want accomplished beyond ‘picking up the phone by the 3rd ring’. Appreciate the value of the job itself. Understanding how important (and hard) it is to manage a room full of clients and pets like a practice owner, to manage a desk load of other responsibilities like a practice owner, and to manage a phone that rings with every kind of question and request like a practice owner is the first step in building a great team. It’s the first step because it teaches you that the person who is willing and capable of doing all of that deserves a good deal of respect.

Start with respect
Why is it that this most difficult job is given to those for whom we have the least regard and for whom we provide the least training? Many practice owners will participate in hiring the hospital’s technician, but the client care reps, well, ‘the senior front desk person handles them’. At some practices, doctors won’t even bother learning the front desk team’s names. Their passive dismissal of the group as a whole is what typically fuels our industry-wide front-versus-back war. We silently bless prejudice against a group that has done nothing to win our disfavor except show up to work, receive little to no training for what they do, and quake beneath an intense day of multitasking and demanding clients.

When you reach out into the world and request someone to take a client care representative job at your practice, let them know by the ad that you write that this is a job that’s extremely important and valuable. Let them know that you appreciate their interest in applying for the position. When they send in their resume, thank them and show them how glad you are to meet someone who may soon be an extremely valuable and respected member of your team.

During the lecture, we’ll review some hiring practices for you to consider when looking for and interviewing a client care representative, but keep in mind this most important point: a great client care representative is an extension of every one of your client service ambitions. Show your gratitude and respect for anyone willing to take on that very big job.

There’s no getting around your responsibility to train
If your practice is like many others, training isn’t training; it’s sitting next to someone who’s been there longer and watching. It’s pick it up as you go. It’s follow along as best you can.

The training issue is a frequent source of dismay for me. I regularly go to practices that are quick to point out the flaws of their front desk team, yet can’t produce a single page of a training manual. They can’t even produce an outline of a training manual. If they do have a training manual, they have no record of it ever being used. As I said before, training is typically relegated to someone whose only qualification for the training job is that they were hired first.

Don’t train what to do, train how to do it. You don’t just want the phone picked up; you want it picked up in a particular way. You don’t just want people checked into your practice; you want them checked in in a particular way. Use your mission statement as a way of qualifying your training so that your client care representatives understand the spirit of the job, not just the job.
Stay realistic
Remember that you want client care representatives to answer phones; check in clients; look for charts; manage charts; balance a cash drawer; deal with any number of computer glitches and issues; communicate to clients through email, portals, text messages, social media and so forth; eat their lunch in their laps; and hold their bladder till the ‘other one’ comes back from break. It’s a huge job and it often comes in punishing waves of busyness. What’s more, the only way to do it, is typically to do it all at once. While we always list multitasking as a requirement of the job, we fail to acknowledge that asking someone to multitask is leaving them wide open to make mistakes and consequently to fail.

Conclusion
Building a great receptionist team starts with appreciating the value of the job you are trying to get done, with training to do the job that you would like done, and with an exploration of how to adjust the workflow of the job so that it’s doable not just endurable.

It’s likely that the rigors of being a client care representative will never go away. The general practice model, with its ‘help all clients at all times’ mission, just won’t allow for it. The person assigned to manage all of the communication and foot traffic coming into the practice will always be challenged.

But it’s very likely that we can make an enormous difference in how our client care team performs by showing them respect for what they do, training them how we would like them to do it, helping them to do their work when they need it, and rewarding them and praising them appropriately when they succeed.

When someone at the desk calls out sick, team members run away from, not run towards the responsibility of filling in. Why is that? Answer that question correctly and you will have taken your first step in building a client service team that sells your practice.
The Front vs. The Back War increases employee turnover, lowers employee productivity, increases the number of patient and client care mistakes that your practice makes, and is emotionally draining on everyone. So why do we allow it to persist?

Let’s consider some of the instigating factors in the battle.

**Communication blackout**

What we don’t know can hurt us, or at the very least can increase our level of frustration and anxiety. Let’s say we need a bag of food from ‘the back’. With a full lobby and a line of people waiting for our help, we pick up the phone, hit the intercom button and call out, “Can I get a 5 pound bag of CD, please?”

Nothing. You ask again while an impatient client stares you down. “Hello? Can anyone hear me? Can I get a 5lb bag of CD?”

The silence gets you boiled. “How many times have we had a meeting about this?” you rhetorically ask your coworker. “What on earth are those people doing back there?”

Those people are actually nowhere near the intercom and can’t hear your call. One is doing the inventory in the basement and the other is outside walking a patient. It’s a perfectly innocent workflow issue, but because you can’t see what’s going on you immediately jump to the worst conclusion. “Lazy good for nothing…I’ll be right back,” you tell the client. “I’ll get it for you myself.”

Once in ‘the back’ you really let loose, though there is no one around to hear. “Where in the hell is everybody? Is everyone on break, because we are slammed up front!”

Judy emerges from the basement with a boxful of supplies. “What’s your problem?” she asks on the defensive. “What are you yelling about?”

“What does it look like? I’m yelling about you and Joanne! I’m friggin slammed up front and you people are no where to be found!”

And so begins The Front versus The Back war of 2016. If only your practice had had a camera system in place, you could have seen Judy in the basement doing her job and Joanne in the dog walk doing hers, but left in the dark, you imagined the worst and the war began.

Communication blackouts can frequently be the source of ‘The War’s’ first shot, but another source is more insidious. It’s the underbelly of us all, the need to feel superior to others, to believe that our work is more important, that we’re smarter, that we’re right.

A group of technicians huddles around a computer monitor in the back and watches the latest appointment pop into place. The doctor on duty is the first one to comment. “What’s he doing!? He’s putting a vomiting 16 old cat next to a lethargic rabbit? How the hell am I supposed to see that? Someone tell him to fix it.”

Marge is only too happy to tell the new guy, Bob, that he messed up. She approaches him from behind and taps him on the shoulder. “Did you just book the vomiting cat?”

“Yes.”

“Dr. Karen says you need to move it now. She’s extremely pissed. So can you move it, please? Thank you.”

Another ‘War’ shot fired.

I have a backyard brood of poultry. One day, I’m going to write a book called, Everything I Need to Know About People I Learned from Chickens, because I’m convinced that chickens’ natural inclination to brutally peck each other into a social order, to ostracize, and to form exclusive cliques, is a broader representation of what we humans do more subtly every day.

But while some of us work in barnyards, we don’t have to act like we live in them. People who work with others must rise above their basest instincts.

Our job’s atmosphere and the standards by which all employees interact is referred to as workplace culture, a term that can be treacle-sweet for many conservative business owners who see goals like revenue, efficiency, care and service as much more important.

But when I talk about workplace culture, I’m not advocating for hand holding and Kumbaya; I’m talking about a calculated look at what our employees feel, how they interact, the tone we set in the workplace, and our respect for health and happiness as a way to increase productivity and most importantly to stop fighting, work together well, and feel good about our jobs.

A few of you may be saying ‘But we tried to improve our practice’s culture. It didn’t work. People weren’t responsive. We advocated for behavioral guidelines. No one listened’.

Failure of workplace culture programs has nothing to do with the merit of trying to improve your employee’s physical and emotional well-being. Don’t let failure to solve the problem convince you that the problem isn’t worth fixing.
Culture is made possible both by the attention we pay to it as leaders and as a by-product of the business model that embraces the fundamental building blocks of any team: communication, respect, and cooperation to name a few. Leaders should stop thinking about culture as a job perk, and start thinking about it as a necessary component to smooth, productive, long-lived operations.

So my recommendation for ending The Front versus The Back War is not to go gunning for the snarkiest individuals at the practice, but to drop an atom bomb of culture on the entire building. The fallout of your actions will have long lasting, glowing effects.

**Mission**

Begin with a articulating your aspirations for workplace culture (that is your aspirations for how it feels to work at your practice and how you expect people to behave) in your mission statement. Choose the words carefully. Mission statements are the real anchor points for all you do and say in your business. Like the foundation of a house, your mission statement must be resolute about what you are trying to accomplish.

Here’s a test that I like to try whenever someone wants to share his or her mission with me. Firstly, I don’t allow them to read it. I make them face me and pretend that they’re trying to entice me to take a job at their practice by extolling the same virtues that are expressed in their mission statement. “Mr. Halow, all of our employees are valued. At our practice, everyone is expected to get along and work together well.”

“Oh!” I reply because this job is sounding tempting, “if I take a job here, how will I notice that I am valued?”

This kind of role play tests the owner and practice’s commitment to the lofty language in the mission and can take a document that feels disconnected from day-to-day operations and make it relevant.

**Discuss culture in writing in your employee and training manuals**

Secondly, articulate your expectations for both behavior and communication in your practice manual. The latest HR trend is to create Workplace Civility Guides in which you outline your expectations with respect to salutations, day- to-day office chitchat, ostracism, name-calling, nicknames, and so forth. It may seem silly. Company policies that insist we say good morning to our coworkers? Laugh if you like, but enough feelings have been hurt by our failure to observe the simplest of social etiquettes that a proactive approach isn’t a bad idea. Outlining how you want people to behave and how you would like them to react is an excellent jumping off point for an interview discussion on the matter, an excellent way to market your practice (who doesn’t want to work for a business that cares about employee feelings?) and an excellent way to begin any coaching session on the subject.

**Overtly Advocate for Culture**

Thirdly, be the practice culture’s advocate. If you observe employees behaving in a manner that is counter productive to the practice’s feel-good culture, address it. Not addressing cultural issues as soon as they arise is more than inaction; it’s passive approval. It’s a passive gesture on the leader’s part that the person on the receiving end of the bad behavior is not important. It’s inaction that could be deadly to your relationship to that individual moving forward.

Do not pussyfoot around negative interactions in your building. People that decide to rain on your parade should blow their storm clouds over to someone else’s practice. When I coach team members on this subject, I never shame them. My negative behavior record is probably no better than anyone else’s. But I have recognized the deleterious effects of negative behavior in the workplace and learned to sit on my knee-jerk reactions. I expect others to do the same. As one colleague of mine says, “We can sit here all day long and listen to why you believe it’s okay to behave negatively in the workplace. At the end of the discussion, I’m still not going to allow you to do it.”

**Conclusion**

For any leader concerned that they don’t have the ability or gumption to end The Front versus The Back War, know these things. Showing employees that you respect them, that you believe in them, and that you are eager to see them succeed will not leave you feeling emptier, but fuller. Addressing what everyone understands to be an unpleasant situation will leave you feeling lighter, not heavier. Standing up for kindness and consideration will not leave you short staffed, but will provide you with a remaining group of employees that will invest extra time and energy to help you out. Many of you have tried many things to improve your business. Try working on what you know is most important to you and everyone in your employ: happiness.
Don’t give up on selling flea tick and heartworm products (FTHW) products in house. Your products, with the assistance of manufacturer incentives, are nearly always competitively priced. As online stores grow in popularity, explore an online store of your own as a way to remain competitive.

Despite the ubiquity of online stores and big-box-store loss-leader promotions, there is a future for FTHW sales in your practice. As I previously mentioned, your prices are typically competitive against those of online or discount stores. At your practice you also have the value of convenience and one-stop shopping. At the end of a long workday, your client doesn’t want to have to drive from your practice to the mile-long aisles of Costco to purchase their Frontline just to save a dollar or two. (Besides, won’t they end up spending an additional 40 dollars on junk that they never expected to purchase once they walk through the doors?) People are tired and eager to finish their daylong to-do list. Don’t add to the stops that they have to make on the way home. Sell them the medication that their pets need while they are in the building.

Think about the language you use
Learn to inquire about their need for FTHW products in a productive way. “Do you need any more flea product?” is almost always answered negatively. Instead ask, ‘How many doses of Revolution do you have left at home?’ as a way of starting a discussion on the need for year-round protection. Other practices will ask clients to bring in all their remaining doses (and any other medications) with them in a plastic bag. The request forces everyone to take an exact look at what kind of medication the pet is receiving and how often.

Believe in what you sell
Is year-round flea medication really necessary? We say it all the time, but do we believe it? How compliant is your own team (or you for that matter) with year-round protection? If you’re not compliant, ask why. Uncovering your team’s reluctance to follow the same direction that they’ve been giving to clients for years may clue you into more effective messaging for everyone involved.

Switch to orals
Many studies have shown that orals are not only more efficacious in stopping all parts of the flea life cycle, but because they are dosed out every three months, their use boosts client compliance.

Explore an online store of Your own.
Online stores have come a long way since the old Vetcentric days. Today’s stores are easy to use and do an excellent job at following up with clients and driving compliance. In studies provided by Vets First Choice, clients were three times more likely to be compliant with recommendations to dose their pets year-round with a flea preventative if they purchased their flea product through the online store as opposed to stopping by the clinic to pick it up. Clients were also three times more likely to be more compliant with prescription diet recommendations if they purchased it through the online store’s auto-refill service.

The value of prevention
I’ve been working in the veterinary business for nearly 20 years. Each year, at every conference I attend, there is some lecture about the value of year-round FTHW compliance and every year, I’m shocked that the lecture room is packed and sometimes overflowing.

But sit in and listen and you’ll find out why. Real data about real disease, once disclosed, is jaw dropping. Dr. Mike Dryden, a frequent speaker at such sessions, is packed with information on the value of prevention based on his first hand experience conducting research on the topic. In fact, fleas, tick and heartworm parasites are everywhere and the primary and secondary impact of an infection by any of them is cause for much of the disease we treat in our practices annually.

We eat with our pets, ride in the car with our pets, sleep with our pets, and hug and kiss our pets. Keeping them parasite-free is just smart. It’s better for our pet’s health, better for our health, and better for our wallet. As a team, rediscover the value of the products that you sell and understand the value of buying them through your business as opposed to another source. Talk about year-round protection and regularly evaluate your client’s compliance with your recommendations. These products are still a valuable source of pet health and veterinary practice revenue.
It’s rarely pleasant to talk to clients about financial matters. Perhaps it’s because we presume that if the client is unhappy about the price, it means that they are unhappy with who we are as individuals. Then without benefit of training, we find ourselves stumbling through a conversation about money, exacerbating an already uncomfortable situation by talking with our foot in our mouth. To successfully take on a discussion about money with a client, we need to have a set of systems, conditions, and skills in place:

- An understanding of our real relevance
- Our clients’ trust
- A team approach to care
- Belief in our pricing
- An understanding of how to communicate value
- Use of language that underlines both our investment in the outcome and our expertise

An understanding of our real relevance
Before you can successfully defend your prices, you have to believe in the value of what it is you’re pricing. Start with the question, ‘Who are we and what do we do that is worthwhile?’ The answer to this question is typically articulated in the mission statement, a document that cites your relevance to clients, patients, employees, veterinary medicine and the community. Too often mission statements are given cursory attention in our industry, a mistake that leaves us most vulnerable to competition. If people have a choice between an Internet answer to their issue or a more time-consuming, expensive stop at your veterinary practice, you better have a very good reason for them to choose the latter. First writing about your value, and then regularly discussing it, not only helps you realize your full potential as a healthcare provider, but it gives you a reservoir of understanding to draw from when faced with the need to talk to clients about your practice’s value.

Our clients’ trust
Simply stated, if people don’t trust you, they’re not only circumspect about your pricing, but about your actions and words. The best way to earn trust from your clients is straightforward. If you want to be trusted, be trustworthy. If you tell clients that their concerns about value and price are meaningful to you, then have systems, tools, and training in place, visible to all, to let everyone know that you are walking your talk. Premade estimates, an accurate reminder system, follow through, meaningful callbacks, pet insurance, clear and open accounts-receivable policies, and regular team meetings about service and product worth are all actions that fortify your verbal commitment to care and value.

It’s also important that the entire team demonstrates its commitment to client service and patient care. When client issues arise, all team members should be empowered to do whatever needs to be done to satisfy the client. Most practice owners and managers would agree that solving clients’ concerns about value is much more complicated than voiding something from the invoice, which is the knee jerk reaction of those inexperienced with handling client complaints. For this reason, they may be reluctant to allow all team members to take action on behalf of a client without a supervisor’s permission. This is a mistake. Few, if any of your employees have owned a business, and probably don’t fully understand the fine line between satisfying clients’ needs and fiscal responsibility. Without giving them a chance to take full charge of client satisfaction however, they’ll never completely understand how challenging it is to be conciliatory to clients’ concerns and profitable at the same time. Allowing employees to shoulder the full weight of a client complaint, with coaching from you, more deeply involves team members in service, demonstrates your confidence in the employee, gives them some way of ‘making up for’ the service error, and best of all, underlines how important it is to avoid such mistakes in the future.

A team approach to care
More impactful than an individual caring about service is an entire team collaborating on service. Focusing on a team approach to service does two things. Firstly, it delivers a more consistent and reliable client experience. Our client service missteps don’t happen so much because we fail to care as individuals, they happen because we fail in communicating client need to one another along the cycle of service. Simply put, it’s not that we don’t run with the ball; it’s that we drop it in the handoff. Secondly, each stride forward you take as a group rallies a sense of team pride and gives employees the sense that their individual effort is part of a larger win. Successful morning huddles and other systems to ensure that all members of the team know who is coming in for the day, what their needs are, and the gender of all pets, improve the value of what you do and your practice’s espirit de corps.
Meaningful pricing
In order to stand up for what you charge, you should believe in the merit of your pricing. Too many veterinary professionals don’t understand the fees behind their services and products. Indeed the system may have little rhyme or reason to it. Sometimes prices are put in place because ‘they seem fair’ or because ‘that’s what the other practices charge.’ Employees may observe owners making up a price for a service without using any kind of formula. They may see managers remove service items from an invoice or giving away money to clients who refer the practice to others. At the end of the day, many of us fail to stand up for our service or product pricing because we believe that it has been created arbitrarily.

The best pricing schemes are ones that are logical and straightforward; that take into consideration non-fixed costs (typically the inventory costs), the fixed expenses (unchanging costs like rent, utilities and labor) and the desired mark up. Veterinary service pricing however, gets much more complicated because we price services and products as loss leaders, as part of packages, as part of wellness plans, and with consideration to what our competitors are charging. These additional variables complicate the pricing formula considerably because in addition to considering the three above-mentioned costs, we have to consider how much profit we are losing on our other pricing schemes. Taking so many variables into consideration is more headache than most managers care to take on. They end up alighting on pricing that ‘feels right’ or what they think clients will accept, rather than taking the time to actually determine if their pricing is profitable.

It’s too bad. There are a number of pricing tools available to help managers make sense of prices. Many veterinary practice consultants have developed pricing tools that not only take a lot of the guesswork out of pricing, but also often provide tens of thousands of dollars worth of additional revenue in the first year that they are used.

In my experience, the best prices are ones that the entire team have discussed, understand, and market. I regularly visit a diversity of practices in many locales, both affluent and poor. I’ve come to discover a surprising fact. Lower prices do not improve sales, nor do they improve compliance. Wherever I go, compliance is not driven by price, but by team buy-in to the value of the service for the money charged.

Lastly, let me say this about pricing. Spending months educating team members and clients about pricing and value, then turning around and giving someone 25 dollars off their bill for referring a client is contradictory to the importance of your practice’s pricing structure. While pricing promotions can have a place in your practice, don’t readily default to them. A bouquet of flowers, a present selected specifically for a client based on their interests, even a handwritten card are more meaningful than money and serve to reiterate to clients and employees alike that you are serious about service and care.

An understanding of how to communicate value
The best client interactions are structured to demonstrate the value of your practice’s services and products. There are three steps to successful client discussions about value, each of which is discussed below.

Identify client need and demonstrate you care
The first step in a successful client transaction is determining what the needs of the client are and letting the client know that they matter to you. Show that you comprehend and care by making eye contact, displaying appropriate facial expressions that indicate you are attentive and empathetic, by keeping an open body posture, and by saying things like, ‘I understand how you feel’ or ‘I understand what you are going through’. Even repeating the client’s needs back to them in your own words is very effective at establishing that you both understand and care.

Demonstrate how your service satisfies the client’s needs
Next talk about the products and services that you offer and how they will serve to meet the needs of the client and mitigate their pet care issues. It’s important that this isn’t a retrospective of everything you offer, but rather a specific recommendation for a product or service that matches the client’s needs. This is an extremely important distinction. For example, if a client expresses an interest in protecting their pet against fleas, our job is not to list the various products that we sell, rather to assess the situation at hand, and recommend a single solution. Most competitors are marketing a wide selection of products sold at low price. As small businesses, we’ll almost always be outcompeted on both fronts. To be successful, we must play to our biggest strengths, our care and our expertise. That’s why it’s important to limit your advice on veterinary care to one solution that, in your expert opinion, will be the most helpful.

Catalyze a plan of action
The last step in this interaction with clients is to invite them to participate in an action plan moving forward. To spend all the time you have getting to know their needs and providing education, then saying something like, ‘it’s up to you’ is not only a waste of your time, it distances you from the client. Someone who is committed to the well being of an individual is not wishy-washy about advice. They have an opinion and they state it! Veterinary professionals who offer a list of options believe that by doing so, they’re avoiding the appearance of being pushy. In fact, their strategy may be more deleterious than they know. Faced with many options, clients often get
confused or worse, frustrated. Many don’t perceive a bunch of options as helpful, rather as a sign of the professional’s general
disinterest. At the end of the day, each of us wants to know what others think. We call our best friends and family members with
questions and the ones who love us most give us their unequivocal thoughts on whatever issue is at hand. Tell the client what to do
and let them know it comes from a place of caring and expertise. It’s the best way to forge lasting bonds and loyalty.

Think about the words you choose
So you believe in the services and products you offer. Your team collaborates on service and care. You enter into each client
interaction ready to listen to their needs, match those needs with your services, and then catalyze an action plan. Still compliance and
sales slowly ebb to your larger competitors. What can you do? My advice is to carefully think about each time a client chooses to
leave your practice or purchase a product elsewhere and work as a group to think of better ways to communicate who you are and
what your practice offers. Here are some ideas to get your started:

Client wants a script for a preventative so that they can purchase it elsewhere
“Are you sure you don’t want to purchase it from us? With the manufacturer’s promotion, the doses at our hospital are less expensive,
yet still come with guarantees that are superior to any other online store or pharmacy outlet. We have time today. If you come in, we
can help you select the correct product to match your pet’s lifestyle and show you how to apply it correctly at no additional charge.
Mrs. _____, we work hard at getting this kind of support from our vendors because we want to be the sole care provider for your pet.”

Client wants to purchase their pet’s chronic medication from an online store.
“Are you aware that we have an online store? It’s just as affordable and there is no shipping charge. Additionally, as owner of the
store, I have full oversight over it. I can promise you that none of our products are short dated or diverted, a claim I’m not comfortable
making with any other online resource for medications. Our client care representatives are free now. I’ll have one pick up and take
your order over the phone. She’ll also send you a link to a video demonstrating how to log into and navigate the site. I think you’ll
find it helpful for future use.”

I regularly need my dog’s Rimadyl filled, but you are too far away.
“If I understand you correctly, our practice is out of the way for you and you would like a closer place to purchase your pet’s chronic
medication. Why don’t I set you up with an automatic refill and free shipment from our online store? I fully guarantee all the
products sold in my store and using our store allows me to remain the sole health care provider for Bingo…a role that I have worked
all of my life to achieve.”

My bill is too high.
“Let’s go into one of the exam rooms and discuss it. From the medical history I see that both the technician and the doctor did a
thorough history and it looks like there is a clear reason why each of these services is recommended. May I offer you one of the
payment solutions we have for our clients? Each is affordable, will give you the ability to pay your bill, and still give Bingo the care
that you and the doctor agree he needs.”

I don’t want a payment option. I have too much debt already.
“I understand that you don’t want to take on more debt and would like to decline the flea product that you and the doctor agree Bingo
should have. With your permission, I can set up an automatic shipment of one dose of the product every month from our online store.
It reduces your pet’s bill today by 62 dollars, but doesn’t compromise Bingo’s care a bit! I’d also like to give you this handout on a
pet insurance that we recommend. Flea products are just one of the many expenses all pet owners face. This is a trusted insurance
company. It is affordable and will mitigate those future costs.”

You people are the most expensive practice around!
“I understand your concern about price. We regularly discuss our pricing as a group and try to find ways to make things as affordable
as we can for our clients without compromising care or quality. We don’t want to lose your business and want to remain Kitty’s
healthcare provider. Can I suggest this insurance plan? Of all the ones on the market, we believe this one is the best. Insurance means
that you don’t have to lower the quality of care Kitty receives by going elsewhere and it will spread the cost of care out over time.
Here’s a brochure and a promotional coupon that you can activate today. Is there anything else I can do to be helpful? I hear what you
are saying and want to make a difference.”

Why do I have to pay for a physical exam?
“A full physical exam, along with any diagnostic work that is done, is the best way to fully understand Bingo’s health issue. We do a
full physical exam on every patient before it is treated because the risk of making an error is too great otherwise. Physical
examinations review every major body system and give the veterinarian more information on how the illness is affecting the body as a
whole. It would be unethical to diagnose a pet without a physical exam and a breach in care and safety on our part that the doctor
would never risk. He is too dedicated to his role of veterinary care provider for our community to do otherwise.”

Conclusion
Your business hinges on your clients’ belief in the value of what it is you do. In my experience, veterinarians are extremely dedicated,
ethical individuals. When our concern for clients and pets goes unrecognized, it’s not because it doesn’t exist, it’s because we’ve
failed to demonstrate it. Taking time to review how you talk about money is a gateway to a clearer practice identity, increased teamwork, stronger relationships with clients, improved compliance, and a general increase in the health and longevity of your patients.
Increasing Compliance with Wellness Profiles
Bash Halow, LVT, CVPM
Halow Tassava Consulting
New York, NY

Grow your business on services in which you believe. Don’t choose to grow wellness profile compliance simply because the industry
tells you to. The future success of your business doesn’t ride just on revenue, but on your willingness to realize the kind of care and
service you know internally is right for the people and pets that you serve.

Sell wellness profiles if you believe in the value of the information they provide and if you know how to act on that information in
a meaningful way. I recently encountered a veterinarian who expressed concern about the value of 4dx results. “VIN hosts a lively
debate on what to do with the results. As a veterinarian, I’m torn. Tests cost my clients nearly 50 dollars. If they show that the
patient has been exposed to Lyme, I’m not sure what I do with that information and I think a lot of other veterinarians would agree.”

Concerns like these are real and important. As a business owner, your best asset is the trust that exists between you and your
clients. If you’re going to take money from them, you should be able to do so knowing that you’re being of genuine service. Practice
what you believe in; provide what you know to be of value; care and believe in what you do. That’s where the real revenue from
‘wellness profiles’ or any other service that you provide comes from.

If you’re convinced (not if AAHA or the AVMA, or Idexx is convinced) that wellness profiles are beneficial, then I would like you
to take that information to your team. Start out with a meeting of your fellow associates and enjoy a discussion on the matter. Emerge
from the meeting with an agreement on the value of wellness profiles, to whom they should be recommended, and an understanding of
what you’re going to do with the information when you get it.

Take the results of the meeting to the whole practice team. Engage your crew in a discussion about what wellness profiles are and
why they work. Give real examples of how they’ve helped patients in your practice. Don’t just have one doctor, manager, or owner
preach this message, but get the entire veterinarian team on board with helping the team members to understand. Doctors are
considered leaders and coming at the group as a united front lends credibility to what you are telling them, underlines the importance
of what you are saying, and gets the group enthused.

Ask the team for their thoughts on how to promote the profiles to clients. In our work with clients we invite teams to look at the
‘Cycle of Service’, a timeline of every client education juncture (when they find the practice on the web, when they call the practice,
when we confirm appointments, when they arrive at the practice, when they are taken into the exam room, etc.). Allow team members
to formulate their own ideas of how they would like to communicate the value of wellness profiles to clients. Because they are
progenitors of these ‘how to’ protocols, you’ll find that they are invested in the outcome and eager to see them succeed.

Often someone in the group will get hung up on the question of value. Do the benefits exceed the costs? It’s an excellent question
and one that should never be shamed or dismissed as mere grumbling. Employees that express concern about money are
demonstrating their empathy for clients; it’s exactly the quality that you’re looking for in a member of your team. Employees that
express concern about money are also picturing themselves in the rooms ‘trying to sell’ clients on wellness profiles. They’re
expressing a real concern that they don’t know how to do what you’re asking them to do or that you’re setting them up for failure. Be
a good leader. Thank them for taking the wellness-profile-project seriously. Thank them for looking for a way to help you achieve
this practice goal. Help them find the answers that they seek.

One solution is to work with companies like Antech to put together ‘wellness packages’. In many cases, you’re most likely already
doing a stool sample and/or a heartworm test for a patient, ask Antech if they would be willing to create a lab package that includes a
wellness profile as a way to save clients money and drive sales. Wellness packages make compliance easier for everyone and
streamline the practice’s workflow. Practices that organize their wellness profiles into specific packages based on species and age
always grow their laboratory sales, catch more disease in its earliest stages, and decrease the amount of mistakes made internally.

After a few weeks of exploring the best ways to communicate your thoughts to clients, you may be ready to develop some
marketing materials. Use the following list to stimulate your own thoughts on how best to educate clients.

Build Customized Wall Art: These days, word processing software is easy to use and sophisticated. Entreat your doctors and room
techs to sit down and sketch out an idea for a wall chart or handout that would be helpful in conveying the value of a wellness profile.
Pass the drawing off to someone who is agile with software like Keynote or Pages and build a professional looking poster customized
with your practice logo and colors. Print it professionally and invest fifteen dollars in a nice frame. Done. The entire process can
take as little as a month. When you’re through, you’ll have a teaching tool that looks great and that everybody eagerly uses.

Turn Handouts Into Webpages: Hopefully your website is built in an easy-to-use platform like WordPress. Open up a blank page
on the site and transfer content, that you would otherwise have built into a brochure, onto the web. If the document is optimized
properly it will increase your practice’s online visibility and serve as a link that you can send to clients following any visit and any
discussion that you have had about wellness profiles. It’s a way to keep the dialogue going after the visit and a way to convey that
your team is professional and sophisticated.
Turn success stories into blogs
Everyone enjoys a touching story about a dog, cat or other companion animal. The next time your wellness profiles succeed at changing the life of a client or a patient, ask the client for permission and write about it! Don't just talk about the medicine involved; talk about the ‘heart’ of the story and what it means to the relationship of the pet and pet parent. Optimize the blog for search engines, post it to your site, and cross post it to your social media sites. Attend the lecture for more information on this topic or reach out to the author directly for more specific guidance in this area.

Write scripts
The key word in this title is ‘write’ not ‘scripts’, since it's the process of writing that helps us to learn what we want to say and how to say it. When I conduct ‘script writing parties’, I gather team members in groups and have them write collectively. After the writing session, they’re asked to read aloud what they’ve written and invited to talk to the rest of the team about why they chose the words that they did. Almost no one leaves the meeting reciting their script at the next client encounter, but they do leave with their mental wheels turning. Having thought about what to write, they are more likely to reflective of what they say and how they say it the next time they are in a position to educate a client.

Practices educate clients about the wellness profiles during puppy and kitten visits; as they promote the benefits of pet insurance; as they prepare the patient for their first preventative dentistry (“Since we’ll be doing Fluffy’s first dental cleaning next year, we’ll run a wellness profile this year to get a base line for next year’s blood results”). We’ll discuss these and other ideas in the lecture itself.

Remember to regularly talk about the value of wellness profiles and your team’s success with promoting them at your employee meetings. Celebrate success stories and share objective compliance data with the team to stimulate thought and discussion on what everyone needs to do to improve. Make sure that your team understands that their efforts are making a difference in the lives of patients and lengthening/improving important client/pet relationships.

There is a direct relationship between preventative medicine and your mission statement. Finding a way for your team members to successfully promote wellness profiles empowers them to teach and gives them a chance to see the enormous positive benefits of their actions.
For years we, as veterinary professionals, have been consumed by increasing compliance. Assuming that most pet owners truly want to care for their pets properly, the issue becomes more about effective education than about desire, technique, or cost. When I can create a good connection with pet owners and am able to deliver my message in a way that they are willing and able to receive, pet owners are more informed and eager to follow my instructions. While it might appear that the surge in communication technology has made this process easier, we have found that there are so many technological options available to the modern practice that communicating with pet owners in the veterinary market can be a very frustrating experience.

During this lecture, we will focus on three aspects of modern communication technology: surveys, reviews, and apps, and we will explore how these very efficient channels can help you increase your practice compliance.

**Surveys**

For years, we have been asked our opinions on topics that range from our country’s political system, the taste of a particular soft drink, or the service we received during a phone experience with a major brand. We have grown accustomed to sharing our opinions in hopes that they will incite change, compliment those who are doing a great job, or call out those who are not. Pet owners are no different and are an extremely valuable source for information about your practice and their experiences there.

We have adopted a post-appointment survey process at our practice that automatically reaches out to every client via text and/or email after each transaction. Clients are asked questions about their experience and are allowed to submit responses quite easily from their smart phones, tablets, or desktop computers. Our survey content has been custom-created to dive deeper than simply “was this a positive experience?” or “did we exceed your expectations during your last appointment?” by including verbiage that tests the client’s understanding of how they would like our practice to care for their pets.

Questions that have proven to be effective at assessing the client’s experience include:

- Did the doctor demonstrate that he/she understand your needs by providing solutions that directly relate to the reason for the visit?
- Did the staff explain things using easy to understand terms and phrases?
- Do you understand the care that was provided in the practice?
- Do you understand how to properly care for your pet?
- Are the recommendations provided during the visit reasonable and easy to implement?
- Do you feel prepared to care for your pet?
- Do you feel comfortable contacting the practice with additional questions?

Asking the “right” questions is just the beginning of the survey game. You should also consider implementing a consistent process which reaches a large portion of your client base. Resist the temptation to make wide sweeping changes based upon a few comments. Track results until trends become obvious, and then implement change slowly. After processes and procedures are changed, be certain to re-survey those groups that initially made the comments suggesting that change was necessary to gauge effectiveness of the change.

Although we will dive deeper into the world of surveys during the lecture, here are a few tips that I have found very important:

- Keep the number of questions asked during a survey low. We like 5-7 and find a sharp drop-off in participation when more questions are asked.
- Be consistent. Survey repeatedly. Pet owners will develop an expectation of being asked their opinions.
- Be willing to change.
- When possible, thank pet owners for their participation and suggestions.
- Do not survey too much. Many programs allow you to send post-appointment and custom-made surveys without a client appointment. Asking for feedback too frequently can cause a person to stop responding.
- Track your results, and use a program that allows you to categorize the responses back to specific aspects of your practice.

**Reviews**

Mobile technologies, websites that have been created to gather and publish people’s opinions on products, services, or brands, and the public’s acceptance of these trends has created an opportunity for veterinary practices to gather data about how they are performing. While many veterinary professionals consider reviews and the entire online review process to be the bane of their existences, I will demonstrate how the review process can be used for your benefit.
This is a lecture about compliance and using the review concept to gather information that will allow your practice to make change, monitor how well your message is being received, and be a part of the review process. Handling negative reviews is a subject that requires much more attention we can cover during this session.

As we discussed earlier in the lecture, we have become accustomed to sharing our opinions. The trend that is online reviewing has changed how today’s savvy consumers select products and brands. For that reason, your practice needs to have an active role in gathering and publishing the most accurate reviews about your practice. One technique is to attach a request for a review to each of the post-appointment surveys you send to clients. While surveys generally gauge a client’s feelings on a scale of better or worse, reviews actually ask for text-based comments, allowing clients to be most specific in their opinions. If your practice has the ability to do so with an internal system, I strongly suggest that you request a survey after each appointment. Should you not have an internal system for gathering survey results, you can direct your clients to public review sites like Yelp, Google Reviews, City Search, or Facebook. Often times, simply demonstrating that reviews are important to the growth of your business is enough to excite clients to participate.

One of the very valuable (and most often overlooked) aspects of online reviews is the relationship between the words used in review comments and the words used by those searching online for practices like yours. Think about how you search the Internet. If you were looking for a veterinarian in your area, would you search Google for “veterinary,” or are you more likely to search for phrases like “best veterinarian Any town, Iowa?” More likely it’s the latter. Assuming that is true, let’s consider some of the comments that can be made using reviews. Clients often submit reviews that contain verbiage like “Dr. Smith is the best veterinarian in Any town,” “I live in Any town and love Dr. Smith,” or “Any town loves Dr. Smith and his veterinary team.” Do you see the overlap in content? Please know that the search engines like Google, Yahoo, and Bing can only read text. Properly gathering and positioning valuable content from online reviews on your hospital website is critical for getting your message shared with others searching the web.

Although we will dive deeper into the world of reviews during the lecture, here are a few reasons your practice would want to participate in the review process (all of which, by the way, can increase compliance). You can expect to:

- Develop a solid base of accurate, very positive reviews and comments that can “cushion” the blow of the less than accurate or blatantly false negative reviews.
- Develop a much more engaged audience who not only will feel more loyal to your practice, but also will provide indicators about how well your message is reaching them and ultimately providing them with the information necessary to properly care for their pets in the manner you suggest.
- Increase search engine rankings for your practice, if (and only if) the review results are available and properly positioned on your website.
- Have your clients understand and be comfortable with the predictable space where they can leave comments. They will feel that they have a voice. The Internet of full of review outlets. If you do not provide a canvas on which they can “paint”, they will paint elsewhere - perhaps in a location with which you are unfamiliar and are not able to monitor.

Practice APPs

I am certain that you will agree that smart phones and mobile devices have changed the way we communicate. If you are like most of the population, you utilize your smart phone and the apps installed within it many times throughout the day. These apps are designed to solve problems, provide instant access to information, offer entertainment, build loyalty, increase brand awareness, and generally make the lives of consumers much more convenient. Do you have an app for your practice? I believe that you should, as this is one of the most valuable opportunities today to reach clients.

One of the best ways to apply app technology to your practice is to utilize a service that will build and brand an app for your practice. Your practice purchases the app from the developer and provides the app as a free service/benefit to your clients through the Google Play or iTunes APP Store. This opportunity allows you to increase brand awareness by properly positioning your practice logo on what has become the most valuable piece of digital real estate available - your client’s smart phone. If done correctly, your client will consciously or sub-consciously view your logo each time he/she accesses the apps on his/her smart phone. This access opens up an opportunity for communications that has never been available before.

A word of caution. There is a trend in the digital world for companies to create “mobile websites” that may “look” like apps when downloaded and saved as “shortcuts” on a client’s smartphone. These mobile sites are NOT apps and offer very limited functionality when compared to a true app that is most commonly delivered via the Google Play or iTunes APP Store. While I will demonstrate the difference during the lecture, the rule of thumb should be how the APP is delivered to your clients. If it is available via Google Play or iTunes, it is an APP. If it is a shortcut on the client’s phone to a mobile website, it is not an APP but rather a dated approach to web design that unfortunately looks like an APP but lacks most (if not all) the features that make APPs such a valuable piece of your digital strategy.
Although we will dive deeper into the world of APPs during the lecture, here are a few reasons and features of today’s APPs that clearly demonstrate why your practice should immediately implement an APP as part of a digital marketing and compliance strategy:

- APPs are inexpensive to build and maintain (usually under $500 to build and $100/month to maintain).
- Having your APP and logo on a client’s cell phone promotes brand awareness and allows him/her instant access to your information.
- Clients can request appointments, Rx refills, and food refills with the touch of a button.
- Practices can “push” messages to everyone that has the APP installed very easily. These messages can be related to specials, reminders to dose pets for heartworm, or any time-sensitive topics.
- Clients can call the practice with the touch of a button.
- Practices can easily implement technology which allows those clients who have the APP installed to receive messages automatically when they enter the practice. These messages can center around specials, provide welcoming messages, or suggest conversations that the doctor would like to have during the visit.
- Loyalty or “compliance-promoting” programs can be developed which gamify the process of compliance and reward clients for caring for their pets in the manner that you recommend.
- Photo sharing allows clients to take pictures of their pets and share them (with your logo as branding) across the social channels like Facebook.
- Clients can track pet exercise and log activities, symptoms, or behavior for your review during the next visit. Pet exercise can be tracked very accurately using GPS and provide a very accurate idea of how long and how far a pet is walked/exercised.
- Clients can access emergency phone numbers and information with the touch of a button.

**In summary**

Today’s world of communication can be very complicated. The many opportunities that are available for you and your practice to communicate with your clients can be overwhelming and missed without a good plan for implementation. This lecture will highlight some of the most valuable digital opportunities and provide examples of how we utilize these resources to excite pet owners to properly care for their pets by complying with the recommendations we make.
Treating birds and exotic pets
Is it worth it? Are there enough clients to make it worthwhile? Does it generate enough revenue? Done the right way, exotic pet practice can be productive, progressive, and profitable. So, how do you make it this way? You need to take several steps.

**Start with continuing education on exotics**
Before you can charge clients for your exotic pet services, you must educate yourself and your staff about exotic pets. Decide which species you will treat. Birds? Small mammals (rabbits, rodents, ferrets)? Reptiles? Amphibians? Less commonly known species like sugar gliders, pot-bellied pigs, opossums, skunks, wallabies, kinkajous, and primates?

Start by focusing on wellness care. Learn what’s “normal.” For each species, educate yourself about diet, husbandry, light and temperature requirements, humidity needs, appropriate cage substrate, normal behavior and social structure. Then, learn what’s “abnormal.” Learn to recognize common clinical signs and diseases. Learn basic supportive care and disease-specific treatments, and don’t forget - exotics information is constantly evolving, so you must keep current!

**How do you get educated about exotic pets?**
Veterinary schools in the past have taught little to no bird and exotic animal medicine. Many veterinary schools do not have exotic pet departments, so most veterinarians have little to no clinical experience with birds or other exotic pets when they graduate from school. One way to gain experience is through externships that are ideal for “hands-on” experience. Many private practices that treat exotics, zoos, wildlife centers and sanctuaries, and laboratories are great places to gain exotic animal experience.

Every veterinary hospital that treats birds and exotic pets should also have a library to cover the care of different species. There are several excellent textbooks, journals, and magazines that review basic bird and exotic pet medicine and surgery. In addition, there are many useful on-line courses through websites such as the Veterinary Information Network that provide great information on exotic pets, as well as numerous list-serves, chat groups, and forums (such as ExoticDVM@yahoogroups.com) in which practitioners can share information on exotic animal care. Remember, however, as you participate in this on-line networking, that exotic pet care information quickly becomes obsolete as new methods of treatment evolve; there is quite a bit of anecdotal information out there on exotic pets – the “IME, or “In my experience…” - that is not necessarily supported by scientific study and not necessarily all correct.

Another place to gain education about bird and exotic pet care is through membership in veterinary associations. The Association of Avian Veterinarians, the Association of Exotic Mammal Veterinarians, the Association of Reptile and Amphibian Veterinarians, and the Association of Zoo Veterinarians, all are great places to learn about exotics and worth the expense of membership if you’re serious about learning. These organizations provide up-to-date information through conference lectures and publications and offer ongoing networking opportunities. Best of all, these groups offer membership not only for you, but also for your colleagues and staff (associates, residents, interns, veterinary technicians, and support staff).

**Getting set up to treat exotic pets**
How do you get started? Before you can see any exotic pet, you must acquire some basic equipment tailored to examine these animals. A few essential items include a pediatric/infant stethoscope, small surgical instruments, a few cages with small bar spacing or Plexiglas surrounding them to prevent escapes, and small gauge catheters, needles, and blood tubes. Other basic equipment you will need includes un-cuffed endotracheal tubes for birds (as the standard cuffed tubes used in dogs and cats can cause necrosis of birds’ tracheal walls that are made of cartilage that doesn’t stretch), metal gavage feeding tubes (to force feed birds and reptiles), small bandaging material, and a Gram’s stain kit (to stain stool samples), and an electronic scale that weighs in 1-gram increments to weigh small species. If you are going to be working more seriously with exotic pets, you might consider getting more advanced equipment including a nebulizer to deliver aerosolized medication, a lighted nasal speculum to look in the oral cavities of rabbits and rodents, specialized dental instruments (rasps, burs, etc.) for rabbit and rodent dentistry, Doppler cuffs in small sizes (1mm, 1.5 mm) to measure blood pressure, an intravenous fluid pump to deliver small volumes of fluid slowly, microvascular surgical instruments, a centrifuge to spin small blood tubes, rigid endoscopes (2.7 mm, 1.9 mm, 1 mm) with a light source/camera/video equipment for working inside small pets, an in-house chemistry machine (such as the Abaxis machine) that can calculate hematlogy results from small blood volumes, a digital x-ray machine capable taking high detail films of very small pets, and an ultrasound machine with a high frequency (12-16 mg Hz) probe for high resolution images of shallow structures. In addition, you need an account with a send-out laboratory capable of processing small exotic pet blood samples, as well as a relationship with a pharmacy that can reliably compound drugs into liquids in volumes appropriate for small exotic pets. Finally, you should also have at least some client education handouts on exotic pet nutrition, housing, and husbandry to cover the most commonly seen exotic pet species.

**What exotic pet services will you offer?**
If you’re thinking about incorporating exotics into your practice, you’ll want to consider what services you’ll offer. Will you focus only on preventative medicine and wellness care, or will you see all species for illness, as well? Will you promote annual to bi-annual...
Unfortunately, since many exotic pets (i.e. hamsters, budgies) are considered “disposable” or “replaceable” pets and are not valued as much as the beloved family dog or cat, many exotic pet owners shop around to get the lowest prices when it comes to treating their animals. Many small exotics (rodents, rabbits, small birds) have short life spans, and some of these pets may be part of a large

Another factor to consider is whether you will treat exotic pets on an outpatient basis only, or whether you will hospitalize sick exotic animals. If you hospitalize these animals, will you have overnight care? What will you do if you have a critically ill exotic patient? Will you see exotic pets on an emergency basis after-hours? If you are treating exotics during business hours, you must have some provision for after-hours emergency care. Seeing exotic pet emergencies can be a real practice builder, as many exotic animals are not brought to the veterinarian until they are sick. In general, most exotic pets are not brought in for regular check-ups, and many exotic pets are not regularly handled by owners, so many owners don’t realize that their animals are ill until they are really ill. Many exotic animals are prey species that hide their illness, so that they are not eaten by predators, until they are no longer able to. If you are going to treat exotic pet emergencies, you should decide whether you will treat only client-owned pets or whether you will also see non-client owned exotic animals. You may also want to consider having an answering service screen your after-hours emergency calls, as once the word gets out that you are treating exotic pet emergencies, you may be flooded with emergency calls. Another thing to consider if you are going to accept exotic pet emergency calls is to form a relationship with a local 24/7 emergency veterinary center in which you can train the emergency veterinarians in basic, life-supportive exotic pet care, so that you can refer emergency cases to them after your business hours. You can provide them with exotic textbooks, basic drugs, and equipment, and be available to them by phone for exotic pet advice once they are treating the emergency. They can refer emergency calls to you for assessment. Then you can assess these cases on in phone conversations with the clients, and refer them back to the emergency center for temporary “babysitting” care until your hospital reopens. You just need to explain to clients that the emergency center care is supportive only, not necessarily diagnostic. The emergency hospital gets business, and you get support plus new clients, so everyone wins.

Another factor to consider, once you have gotten more comfortable treating exotic pets, is whether you will provide phone consultations for other veterinarians seeing exotic species. Will you accept referrals? Once your colleagues realize that you are willing to see these animals, you may receive a many referrals from other local veterinarians not willing to see these cases.

Once you decide to treat exotic pet species, you should consider whether you will do exotic pet house calls. House calls are convenient for easily-stressed, infrequently handled exotic pets. However, with house calls, you are limited in the diagnostic tests you can do in someone’s home, and these visits may not always be cost effective. If you’re going to do them, you must account for your time, your technician’s time, and travel costs, and you must be sure to keep good records for legal purposes. House calls for exotic pets may be better left for multiple pets that the owner is unable to bring all together to the hospital.

A final factor to think about if you decide to treat exotic pets is whether you will work with pet stores and breeders that sell these species. Pet stores throughout the country commonly sell exotic species, and in these stores, the emphasis often on “herd” health, not on the individual pet. This kind of thinking often limits you in what kinds of diagnostic work-ups you are allowed to do, and you are often faced with competing interests of animal vs. the corporation, as well as pre-conceived ideas about what is appropriate exotic pet care. Working with corporations also has certain legal ramifications that are not present when you work on client-owned animals. Working with pet stores, however, can be a great PR move for your practice.

What about exotic pet product sales?
Just as many veterinary hospitals that treat dogs and cats sell food and other products for these animals, clinics that treat exotic pets often sell products for these patients. If you are going to treat exotic pets, you will want to consider the sale of products for these animals. Often it is difficult for exotic pet owners to find quality items for their animals, so the sale of exotic pet products can be very profitable. Possible items to sell include food, treats, nutritional supplements, cage bedding, toys, grooming tools, pet carriers, and instructional DVDs and books on particular species. Having these items available in your hospital, particularly if you recommend these items to your clients when you examine their pets, provides them with “one-stop-shopping” convenience. Factors to consider when deciding whether to sell certain products include whether you actively recommend these items, whether you have space in your hospital to stock these items, whether you treat enough of a particular species to justify sale of species-specific items, whether you will sell these items before their expiration date, whether they are easily available in stores locally or on-line, and if they are, whether you will be able to sell them at a competitive price. If you sell other items on your hospital website, you might consider selling exotic pet products there, too, to avoid having to stock them in the hospital and to make it convenient for exotic pet owners to get the products they need.

Setting fees to treat exotic pets – how much will you charge?
Unfortunately, since many exotic pets (i.e. hamsters, budgies) are considered “disposable” or “replaceable” pets and are not valued as much as the beloved family dog or cat, many exotic pet owners shop around to get the lowest prices when it comes to treating their animals. Many small exotics (rodents, rabbits, small birds) have short life spans, and some of these pets may be part of a large
collection of similar species and are therefore considered less important individually. Ironically, some exotic pets cost less to replace than to treat. The fact that many of these animals are small, very cute, and have a low purchase price makes some exotic pet owners mistakenly think their pet’s care should be inexpensive.

Do not undervalue exotic pet care in your practice. Exotic animal care should cost as much as, if not more than, dog and cat care for several reasons. Exotic species are often more time consuming and labor intensive to treat, and they frequently require specialized equipment and training to work with. In addition, they commonly need more frequent monitoring, medicating, and feeding because of their higher metabolic rates.

Here is some advice and a few guidelines when it comes to setting fees for exotics. Do not compromise your standards by not offering the best medical care you can provide because of price. Recognize the inability or unwillingness of some exotic pet owners to spend large amounts on their often inexpensive pets, and be willing to work with clients in providing the best care possible within their budgets. Explain your treatment plans thoroughly and in detail, and be sure to review your recommendations in layperson terms. Only doctors (no other staff) should review estimates with clients to ensure they receive an adequate explanation and greater perceived value for services.

Beware of “shopped prices” - fees owners call around about to comparison shop. Commonly shopped fees include examination fees, the cost for neuter/spay, and the charge for fecal analysis. Compare your fees to those of other colleagues in your area that treat exotic pets, and be sure to keep your initial visit price competitive. Do not set the initial fee too low as to undervalue your services or to reinforce the notion that exotic pets are not as valuable as dogs and cats, but do not set the fee too high as to dissuade people from coming. Get clients in your door, and then justify your fees for other services once they come in. Convincing exotic pet owners to come in the door is sometimes the hardest part. Greater client volume generally outweighs higher fees for fewer clients.

**How will you advertise your services for exotic pets?**

“If you build it, they will come” does not always apply. In general, veterinarians are not taught in school to market their services, and many veterinarians do little to no marketing other than a sign or a website. Offering the best medical care is not enough. If you’ve got it, you must flaunt it. But how?

There are many ways to advertise the fact that you treat exotic pets. First, you can incorporate exotic animals into your logo on stationary, business cards, etc. You can do this either with a picture or with words (“exotics,” “birds,” “rabbits,” etc.), indicating that you treat these species. You can also include exotics in your motto. For example, my exotic animal hospital’s motto is, “Exceptional care for extraordinary pets,” next to our logo which has a bird, a rabbit, a ferret, and a lizard. Branding your practice is essential to any animal hospital’s success, and a picture is worth a thousand words. So, having exotic pets in your hospital’s logo and sign will bring you referrals from people just driving by. Perhaps more than any other place, the Internet is where your advertising dollars should be focused. Start with a prominent Internet presence: a user-friendly website that includes essential key words (bird, rabbit, ferret, reptile, exotics, etc.) so that your site comes up high in an Internet search for species-specific pet care. Exotic pet practice is a niche industry, so key words are important. Be sure to search engine optimize your website using these words!

While your ultimate goal should be to come up high in the organic search results for exotic pet terms on search engines, you may want to invest, as well, in some Internet marketing by developing ad campaigns on Google, Yahoo, Bing, Facebook, etc., that focus on a series of key words that are commonly searched when owners are looking for exotic pet care. These ads should link back to your website. This cost-per-click” advertising can become quite expensive if it’s not monitored carefully to ensure it is driving business into your practice. You should start with a limited maximum daily budget for each ad and a moderate cost per click. Monitor your campaign’s progress weekly (via Google Analytics) and make adjustments to key words and cost-per-click accordingly. You should track the number of appointments that result from your ads to determine the return on your investment. As this can be very complicated and time consuming, you may want to seek professional advice. Remember, to make money, you have to spend some money!

You should ensure you website, which indicates that you treat exotic pets, is listed in Google’s local business directory. To do this, visit Google’s “Local Business Center,” and register your business on-line with Google. Google will send you a code via snail mail that you enter on line in their Local Business Center site to confirm your listing. Within 6 weeks, your local listing is posted and is also a place where clients can review your services. While good reviews can be a great source of referrals, remember clients can provide both positive and negative reviews on-line, so be careful to monitor this site closely!

Another way to market your exotic pet services is email marketing, which is faster, cheaper, and easier than direct mail. With email marketing, you can easily target thousands of clients, referring vets, pet stores, and pet clubs with just a single click, and you can target specific recipients (species, age groups, geographic locations, etc.). You can base emails on “calls to action,” or things you’d like exotic pet owners to do, such as microchipping and boarding (for all species), and dental prophies and vaccinations (for ferrets). Email marketing can be labor intensive if you have to do the work manually, since a limited number of emails may be sent out at any one time, but many companies will interface with your veterinary software programs to send emails out directly for you.
Other ways to market your exotic pet services include referrals from other exotic pet owners, pet stores and pet clubs, plus testimonials on species-specific pet care websites. Satisfied clients are a great source of new business, but remember, bad news travels as fast as good news. Therefore, never burn bridges with clients!

Pet clubs, such as bird clubs, rabbit societies, reptile groups, etc., often are national with local chapters and can be sources of referrals of both sick and healthy pets. You can stock clubs with business cards and hospital flyers, offer to speak at their meetings, advertise in their newsletters, and provide members-only discounts in return for client referrals.

Pet stores can also be a terrific way to gain new business. Many of these stores sell exotic pets and need help with their care. Animals in the store, as well as pets sold to clients, may become new patients, plus many stores will agree to hand out your practice’s business cards and flyers in return for your services. Meet with the store owner or manager, and remind him that the healthier you are able to keep the exotic pet after it is sold, the more business (in supplies, food, etc.) the store will likely get back. Also, keeping in-store pets healthy means these pets are more likely to be purchased, and healthier pets mean happier consumers who are more likely to return to the store for future pet purchases. With stores, however, some animal care decisions may be based on dollars and cents, not on individual pet welfare, so discounting your in-store services in return for client referrals may be a way to gain their business.

One thing to remember when you first start treating exotic pets is to not take on cases you’re not comfortable with. Referring cases to a specialist (avian, companion mammal, or any other specialty) is not equivalent to failure. Such a referral may help the patient fastest and may lead to happier, more satisfied clients who ultimately return to your hospital.

In sum, you can make treating exotics both fulfilling and profitable. But before you can offer your services for a fee, you must teach yourself the basics. Once you are ready to see exotic patients, get the word out, and market your practice! Exotic pets deserve great care. There are many exotic pets out there but few truly great exotic pet vets. Don’t be afraid of them – be one of them!
Most any time a group of veterinarians gather to discuss their practices, the subject of marketing arises. Over the years, the "hot" marketing ideas or trends that are debated have shifted, but the theme has remained constant; most practices want to want to spend as little as possible to produce as much revenue as possible. Simply put, they want to spend their marketing budget as efficiently as possible. Today, we will look at some marketing techniques that deserve the industry's attention.

**Evaluate everything first**

Before you begin designing a marketing plan for your practice, you should fully understand your current efforts. To do so, have your practice manager gather financial numbers which will reveal your current annual marketing spend or commitment. Make a list of marketing expenses. Be consistent on how you are looking at the numbers. For example, choose to look at things from a monthly or an annual view. This allows for these expenses to be viewed in the same light. After you THINK you have compiled an accurate list of marketing expenses, pull your monthly credit card statements. Search line by line to make certain that you have identified all possible expenses that could be considered part of marketing. Include Internet hosting fees, Yellow Pages listings, and any marketing consultant fees.

Next, contact each provider, and have them provide you with a list of the services that they provide. Ask if there are measurements available to include analytics, traffic totals, and/or any information that can be used to substantiate the expense and perhaps draw parallels between the effort (expense) and the result felt by the practice (most often, an increase in revenue). Then, compare your services, and look for overlapping or duplicate services. Conduct this assessment every six months, so that those small monthly fees and charges are accounted for and that providers are held accountable for the services that they claim to provide. Very often, practices find that they have purchased services that overlap and/or that they are not utilizing to their full potential.

The practice of reviewing your current marketing plan may uncover funds that can be banked or reassigned to more beneficial services. This practice also begins the conversation of exploring new marketing opportunities. Here we go.

**The yellow pages**

Years ago, we all did it. People needed to find your phone number and, outside of calling 411 and being charged, they needed the book. It was so common to use the phone book that the people used it as a place to gather information and to "shop." Times have changed. No one uses the phone book anymore. While some may argue that the older population may still utilize this medium, the number of people still doing this is trending down rapidly. There are many stories out there of practices that tried to back track leads and were not able to draw a single new client back to the phone book. Drop your space down to the most basic ad - perhaps only a listing of your address and phone number.

**Yellow pages websites**

While I don't want to spend too much time on the Yellow Pages because of its obvious low return on investment, let’s address the Yellow Pages related website trend. The Yellow Pages had to reinvent itself in the digital space. The Yellow Pages created very basic template sites that offer slightly more than an online phone listing. Many practices are promised rankings in the search engines from The Yellow Pages, and often those sites do pretty well for a very limited number of keywords. The real problem is that these sites are expensive, are very limited in their performance, and actually become competitor sites to your "real" practice website. Why pay for competition? A good search engine optimization (SEO) firm should be able to accomplish what the Yellow Page sites do for a fraction of the cost.

**Phone number tracking**

We are constantly looking to track the effectiveness of marketing efforts. There is a trend out there that involves a phone number that is dedicated to the practice so that the provider can "show you" how many people dial that number. Neat, right? The question to ask those providers is, "What happens to the phone number when I stop using your service?" All the programs I have seen will not transfer the number to your control, as it is their property. So, basically the number becomes assigned to another business or is disconnected. Why would you want to market and promote a phone number that your practice does not own? You should not participate in such programs but should use other techniques to determine the effectiveness of a marketing program.

**Tracking marketing efforts**

When marketing your practice, you must attempt to determine the effectiveness of your efforts. Simply put, is the marketing paying for itself? Social media and website efforts can be tracked using analytics (tiny bits of code placed on websites that track a user’s
experience). A good SEO firm should walk you through the traffic reports for your website each month, speaking in terms that you understand, so that you have a clear picture of how your efforts are affecting the traffic on your website. While determining the amount of revenue from an effort can be difficult, you should track your numbers on a monthly basis and be focused on your marketing efforts. For example, trends should show an increase in the amount of revenue for the services or products you promote while (and for a short time after) any campaign is in effect. Remember, if you don't track it, you can't manage it.

In order to get a clear view of what is happening, sometimes you have to get back to the basics. Every campaign should involve a call to action and a mention code. Your receptionists are the keys to learning why clients are coming into your practice. Each caller or visitor in your practice should be greeted with the same phrase: "It's great to see you/hear from you. What exactly made you call to schedule this appointment/call to inquire about us/visit the practice today?" If asked, and you listen carefully, clients will tell you which marketing efforts are working. Track these results on a daily, weekly, and monthly basis. A graph of these results will be an amazing asset.

What marketing strategies are hot today?
The most important marketing tools are digital. We have moved into a fast paced world, whereby cell phones are not used to make calls. Desktop computers, tablets, websites, and social media accounts are communication portals that allow immediate access to a world of information. To be successful today, your practice must have a strong presence in this space. If you do not, you will be left behind. It is that simple.

Where do I start?
Remember earlier we discussed having a digital marketing assessment conducted on your practice? You need to start there. Contract with an outside firm to perform a review of your current digital footprint, the traffic it is creating, and how it compares with that of the competition. An analysis of this sort should be fairly time consuming on the reviewer's part and may cost between $500 and $1,000. Very often, these fees will ultimately be deducted from any products purchased from the firm; thus, essentially the analysis is free provided you make a purchase.

Later we will take a look at some analysis results and discuss the information that is returned.

After receiving your analysis and recommendations for your practice's digital campaign, you will have a better idea of the specific techniques or strategies that will work best for your practice. It is important to understand that a practice needs to be prepared for many techniques. For instance, practices without websites should not start social media campaigns. Start with the website, and grow into the social media.

The following topics will be discussed in greater detail during the lecture, but here is an outline to follow:

Practice website
If you do not have a website for your practice, this should be a priority.

Key factors to consider when developing a website:
- Is the website custom made or a template design?
- How is the content of the website developed? Is it "duplicate content"?
- Will the website be built SEO friendly?
- How much SEO does your practice need?
- Will you own the content, design, and URL?
- How much will your website cost over 3 years?

Search engine optimization
The next step in developing an effective digital presence will be exploring your SEO needs.

Key factors to consider when developing an SEO plan:
- SEO is an ongoing expense and is necessary in order to remain relevant.
- Without SEO, your message may be unheard.
- Effective copy is a cornerstone to good SEO.
- SEO is not "one size fits all" and should only be purchased after analysis.
- Your SEO program must change regularly.
- You must track and understand all efforts.
- SEO and social media are related.

Social media marketing
After your website and SEO programs have been implemented, it is time to consider social media.

Key factors practices should consider when developing a social media plan include:
Who will manage the social sites?
Which sites are most important (Start with Facebook, YouTube, and a blog).
Where will you get content?
Practices must own the rights to use content and pictures from the Internet.
All social efforts should be directed back to your website.
Post relevancy and consistency are extremely important.

Mobile
We live in a mobile world. Operating without a mobile website will result in many missed opportunities to share your message. While we will focus on the mobile space in another lecture, the following factors should be considered when developing a mobile plan:
- How does your website currently perform on smartphones and tablets?
- Does your presence meet pet owners’ expectations?
- Understand "thumb navigation."
- Mobile searches will soon surpass those conducted on a desktop.
Who’s Treating this Patient?
GPs and Specialists Can Practice Better Together
Laurie Hess, DVM, DABVP (Avian)
Veterinary Center for Birds and Exotics
Bedford Hills, NY

What is a referral?
- process of referring
  - the act of referring somebody/something to somebody else, i.e. sending a patient to consult a medical specialist
- somebody or something referred
  - somebody or something that has been referred, especially a patient who has been sent to a medical specialist

Who refers? General practitioner → specialist
- Most common; complicated cases beyond GP’s comfort; cases requiring specialized equipment or techniques GP doesn’t have
  - Specialist → specialist
- From one specialty to another, i.e. Internist → Cardiologist; Oncologist → Surgeon

Referrals have occurred forever, but: are they different today?
More referrals to specialists?
- More specialists are available: 2008-2009 Pfizer study showed @ 9,600 specialists practicing in U.S.
- GPs refer to specialists because pets need 24hr care, specialized treatment, advanced diagnostics
- There are a greater number of practice specialty categories
- Greater acceptance by pet owners of specialized fields in veterinary medicine, as in human medicine

Fewer referrals to specialists?
- More general practitioners are in practice now and more are holding on to clients because they need to earn $
- Fewer clients are willing to spend $ on pets at specialists because of discretionary spending
- GPs may feel that with hard economic times, clients may not want to spend $ at specialist so don’t refer

How do vets explain referrals to the client?
AAHA referral brochure: Simple, concise, takes burden off GP in explaining referral process to client

Different types of referrals: direct vs. indirect
Direct referral
- GP provides medical care to a pet (has true doctor-client-patient relationship) up to a certain level
- GP is familiar with specialty services of specialist
- GP believes specialist can provide a service to that pet that GP cannot
- GP refers pet’s owner directly to specialist

Indirect referrals - 2 types
Doctor-client relationship present
- GP provides care to client’s pet (has true client-patient relationship)
- Client has another pet that GP doesn’t feel comfortable treating
- GP refers client’s second pet to specialist

Doctor-client relationship not present
- GP receives call from a non-client asking to treat pet that GP is not comfortable treating
- GP refers non-client’s pet to specialist

Different types of referrals: active vs. passive
Active referral
- GP actively explains pet’s case to specialist via phone call, email, fax, letter
- Effective communication between GP and specialist is essential to make referral work & results in:
  - Better feedback to GP from specialist about case, better medical care for pet
- It’s all about communication! Tried and true tips for better communication:
  - Direct vet-to-vet dialogue (eliminate the middle man!)
Phone calls 1st thing in the morning, before crazy day of appointments start
Direct emails from vet-to-vet, not through general clinic email that vet may never see
Personalized letters, addressed directly from vet to vet
Biannual specialist letters to GPs, reminding GPs that specialist is happy to receive referrals
Support staff (practice manager, receptionist, tech) follow-up on communications sent by GP to specialist
Annual token thank you gifts from specialist to GP, thanking for referrals

Passive referral
- GP takes no direct action to explain details of pet’s case to specialist
  - GP makes notation in pet’s medical record to “Go see Dr. So-and-So” specialist, or
  - GP mentions specialist’s name to pet owner, or
  - GP gives specialist’s contact information/business card to pet owner to make an appointment

Specialist feedback = relay of medical information from specialist back to GP
- Is essential for referral process to benefit pet
- Is critical if GP wants to continue to receive referrals
- May be active or passive
- Is often the step where the referral process fails!!

Specialist feedback can be active vs. passive

Active feedback
- Specialist takes action to relay medical findings and treatment back to GP via call, email, fax, letter, text
- GP is continually updated by specialist on pet’s care & status

Passive feedback
- Specialist doesn’t contact GP directly but filters information about pet back to GP through owner or hospital support staff, or
- Specialist gives owner a copy of pet’s medical records to give back to GP
- GP misses out on critical information regarding pet’s treatment when specialist fails to actively speak with him/her

When referrals go wrong: the problem - COMMUNICATION BREAKDOWN!!! At several levels:
- Failure of GP to communicate pet’s initial medical problem to specialist
- Failure of GP to send appropriate medical records to specialist before referral
- Failure of specialist to keep GP updated on pet’s medical care & status

What happens when a referral doesn’t work
- Once vet-to-vet communication breaks down, medical care may be suboptimal & pet’s treatment suffers
- GP’s frustration may make it less likely he/she will refer in the future to specialist
- Specialist’s frustration may make him/her less likely to provide feedback to GP in the future
- The pet is ultimately the one that suffers

Effective referrals are based on successful communication!
- In a 2008-2009 Pfizer study of 20,000 GPs who referred to specialists, most important factors, in addition to quality of medicine, that determined whether GP referred to a specialist were communication-related:
  - Client service, specialist’s responsiveness, communication between specialist and GP

How referrals should go
- For direct referrals (doctor-client-patient relationship present):
  - GP treats pet; GP decides to refer pet to specialist
  - GP tells client @ specialist and provides client with specialist’s contact information
  - GP actively contacts specialist to discuss referral and to tell specialist that he/she wants feedback
  - Specialist actively refers pet back to GP for further care, if appropriate
  - GP treats pet & actively/repeatedly updates specialist re: pet’s status throughout treatment
  - In future, GP is more likely to refer again to specialist, and both vets and pet benefit from collaboration

- For indirect referrals (no doctor-client-patient relationship present):
  - GP receives call from non-client asking to treat pet GP isn’t comfortable treating
  - GP is familiar with services of specialist
  - GP refers non-client to specialist by providing pet owner with specialist’s contact information
GP tells specialist that client is not truly GP’s client
Specialist thanks GP for referral
Specialist doesn’t need to provide follow-up back to GP, as pet owner isn’t truly GP’s client

**How to streamline the referral process**
Making referrals faster, smoother, better - implementation of a standardized hospital referral protocol:

- **For original referral by GP to specialist**
  - GP’s manager/receptionist/technician gives client specialist’s contact information +/- makes appointment for client
  - GP’s manager/receptionist emails/faxes pet’s medical records to specialist
  - GP’s manager/receptionist/technician follows up with specialist to ensure records received before pet’s appointment
  - GP contacts specialist directly to alert @ referral
  - GP informs specialist of desire for feedback
  - COMMUNICATION BETWEEN GP & SPECIALIST IS RECORDED IN PET’S MEDICAL RECORD

- **For specialist’s follow-up with GP**
  - Specialist contacts GP after seeing pet re: findings and treatment, after each visit or every few visits, if >1
  - Specialist provides written findings & treatment to GP each time pet is seen, so GP is up-to-date
  - Essential if pet needs emergency medical treatment by GP
  - GP’s manager/receptionist ensures that that all documentation re: pet’s care is sent to specialist
  - Specialist’s manager/ follows up with GP’s hospital to confirm receipt of all documents
  - COMMUNICATION BETWEEN GP & SPECIALIST IS RECORDED IN PET’S MEDICAL RECORD
  - Use of pre-made GP referral forms and pre-made specialist follow-up forms:
    - Minimizes miscommunication/missing information conveyed improperly/illegibly in medical records
    - Minimizes frustration & wasted time by both GP and specialist in relaying critical medical information
    - Forms should be part of hospital’s medical software program
      - So all communication appropriately recorded in patient record
      - If forms not part of software, forms must be scanned into medical record
  - Use of pre-made forms with fill-in the blanks:
    - Referral form for GP to send to specialist
    - Follow-up form for specialist to send back to GP
    - Ideally is part of hospital’s software program, so all communication appropriately recorded in patient’s record
    - If not part of software, must be sent to other vet & scanned into patient’s medical record as forms are used

**Forms are the key to streamlined referral success**

**Referral form: GP → specialist**
Referral form to small animal specialty facility: simple, short, easy for GP to fill out, print & fax, or scan in & email

**Referral form: GP → specialist**
Referral form to specialists at a large university: more detailed, longer, downloadable as pdf for GP

**Referral follow-up form: specialist → GP**
- Follow-up letter from specialist:
  - Part of veterinary software program, saved directly into pet’s medical record
  - Emailed to GP through medical software program
  - Short, easy to read, digital
  - Fill out boxes (cut and paste from SOAP)
  - Print and fax, or print/scan/email, to GP

**Key points for successful referrals**
- GP’s responsibilities:
  - Communicate referral to specialist BEFORE client’s appointment
  - Ensure appropriate medical records are sent to specialist BEFORE client’s appointment
  - Indicate to specialist whether client is truly GP’s client and convey to specialist re: desire for feedback
Specialist’s responsibilities:
- Thank GP for referral
- Provide written feedback to GP after seeing patient
  - 2008-2009 Pfizer study: GP most concerned about feedback after specialist treats pet, not during treatment
- Ensure documentation of patient’s treatment is sent back to GP, each time specialist sees patient
- Refer patient back to GP for follow-up care, if appropriate

What happens when referrals are done right
- Pet benefits from advanced medical care
- GP benefits: owner appreciates that GP did right thing in referring, GP learns from specialist with feedback
- Specialist benefits: employs specialized training to help pet, earns GP’s respect & will likely receive future referrals, gains financial compensation for pet’s treatment
- Healthy pet, happy owner, satisfied vets – all win!
Designing and building a low-stress, pet-friendly hospital is wholly dependent on considering how animals are affected by their environments. Different situations, different stimuli - including new sights and sounds and smells - and different environments are all stress-inducing scenarios for animals.

But by viewing the veterinary hospital environs from the animals’ perspectives, we can provide approaches designed to create positive, stress-free relationships between pets and veterinary practices.

We can break down the elements of a well-designed hospital building into six categories:
- Floor plans.
- Design for dogs.
- Design for cats.
- Animal housing.
- Lighting and sound engineering.
- Mechanical engineering.

Floor plans
Stress reduction should start before the pet even enters the building. Does your practice require a waiting room, or can your patients be directed from the parking area directly to an exam room either via a small greeter’s desk or into an exam room with an outside entrance?

If a waiting area is essential to your practice style, one alternative, in the appropriate climate, would be to build a porch large enough to accommodate waiting clients so pets can benefit from being out-of-doors. For indoor waiting, consider creating different seating areas within the space so cats and dogs can be separated.

In fact, separation of species is a concept that can and should be carried throughout the hospital. Separate areas are defined as spaces that are physically separated by noise, ventilation, and visual barriers so dogs cannot hear, smell, or see cats and vice versa. One way to begin to incorporate this concept is to designate at least one exam room for cats only.

Within treatment areas, it is also ideal to develop strategies for blocking a patient’s view of other dogs or cats that are receiving treatments. There may be some limitations to this idea in critical care hospitals where monitoring of patients is the number one priority, but within the confines of the type of medicine you practice, consider reducing the stress of one animal watching treatments on another.

It is acceptable to provide glass above dog and cat eye-level between the divided treatment areas to allow the hospital to remain visually connected for doctors and staff.

Just like separate treatment areas, separate ward areas are defined as spaces that are physically separated with noise, ventilation, and visual barriers so that dogs cannot hear, smell, and see cats and vice versa. Because dogs routinely make noise in wards, dog wards and cat wards should not share a common wall, and there should be at least two doors between housed dogs and cats to prevent sound leakage when one door is opened.

Generally the focus is on the separation of dogs and cats, but hospitals seeing a large number of birds and/or exotics should also develop separate areas for housing and treating these pets.

Design for dogs
Dogs will generally reveal their distress through barking, shaking, whimpering, hiding and other visible behaviors. In other instances, dogs will shut down behaviorally which can be mistaken for non-compliance.

The goal for creating stress-free environments is to minimize all types of fear reactions, those both outwardly displayed and internalized. The goal is to act, not react. For example, instead of focusing on the control of barking noise, the aim is to prevent the stressors that cause dogs to bark.

Exam rooms
An outdoor exam and treatment option is a great tool for some dogs that are terrified in hospital settings. Dogs can avoid the indoor areas of the hospital by coming directly to the outdoor exam room or treatment area from the parking lot. In order for this option to be viable, outdoor treatment areas must be at least partially covered for comfort and all-weather use, cleanable, and fully enclosed with non-climbable fencing for safety.

Indoor exam rooms should be designed to be large enough for dogs to be examined or treated on the floor with enough space for owners and medical staff to easily move around the dog.
Non-slip flooring

Reducing the possibility of slips, trips, and falls (STFs) has become very important within the human healthcare industry. This pressure to make human hospitals safer has created new medical flooring options that can also be used in reducing stress for dogs in veterinary hospitals. The fear, and the real possibility, of slipping and losing footing is a common problem for canine patients but it is one that we can avoid.

Non-slip flooring should be considered for each of the following spaces in a hospital:
- Client areas - lobbies and exam rooms.
- Treatment areas.
- Wards.

Non-slip flooring is defined as flooring that provides a coefficient of friction (COF) of at least 0.60 when wet, according to ASTM D2047. Designers can readily obtain COF data from product manufacturers. In addition to achieving this standard, the floor must look solid to dogs, and should not have a high gloss or polished surface, as this can create the psychological impression that the floor is slippery. The following flooring types qualify:

- Safety sheet vinyl floors. Many of these have COFs of 0.80 or greater when wet. New safety floors are easier to clean than their older counterparts, and can be manufactured without a sparkly appearance. With proper installation, some safety sheet vinyl products may even be used in wet areas of buildings, and therefore may be used throughout a hospital.
- Safety vinyl tile. This inexpensive product is not acceptable for wet areas or areas requiring high sanitation.
- Rubber tile and sheet flooring. Rubber products don’t typically seal well around drains so they are often limited to the dry areas of hospitals, but otherwise rubber flooring can be an excellent choice. Rubber flooring also helps to reduce acoustical reverberation within a space.
- Some porcelain tiles. Tile has to be selected very carefully to balance cleanability with slip resistance, particularly when the tile is wet. Epoxy or urethane grouts should be used to create a sanitary finished surface.
- Some resinous flooring. The problem with resinous flooring (epoxy, etc.) is that in order to achieve the proper wet COF, the floor can be difficult to clean. It is helpful to work directly with the flooring manufacturer to find the best balance between cleaning protocols and safety.

Design for cats

Cats are highly susceptible to fear and stress in uncertain surroundings, particularly veterinary hospitals. Some known feline stressors that occur in veterinary settings include: confinement, transport, changes in environmental temperature and/or ventilation, light patterns, unfamiliar smells, noises, dogs, other cats, irregular schedules, unpredictable handling, the presence of unfamiliar human contact, the inability to engage in natural feline behaviors, and the lack of control over environment.

Cats brought into the hospital in carriers should have a towel draped over the carrier and should be placed off the floor on a raised, stable surface. Feline waiting and exam rooms should be equipped with tables or countertops to rest carriers.

Feline exam rooms should include a variety of enrichments to help ease fear through play and exploration. Include a climbing structure in the exam room to allow cats a choice of vantage points. The structure should be low enough so that cats can be retrieved, with enough distance from the ceiling to ensure cats don’t escape into the ceiling space or onto the tops of cabinets. Windows that provide cats with a view to the outside can also be stress-reducing.

Within a cat’s enclosure it is important to have both an option for privacy and an option for an unobstructed view out. Traditional vertical cage bars restrict the view for the animal. Stress-reducing cages employ horizontal barred gates with unobstructed views out of their environments. Privacy screens can be attached to these bar gates, allowing the cat a hiding space when it is medically appropriate.

Enriching an enclosure for a cat in a medical setting is a difficult task. That said, some degree of enrichment is important to provide housed animals with a sense of control and comfort.

Animal housing

While pets do not typically stay in hospitals for extended periods of time, a well-designed enclosure is still important to reducing fear and promoting health.

One tool for reducing stress in housing areas is to separate dogs by their level of reactivity and other behavioral needs. This can be achieved by including more than one dog ward within your hospital. Another option would be to create some fully-enclosed rooms for individual dogs. These are effective for reducing the level of fear and reactivity in dogs for the following reasons:
- Dogs will have greater sound isolation from one another.
- Dogs will have greater air flow isolation from one another.
- Dogs will have a greater sense of defensible space.
Options for providing stress-reducing wards doesn’t end with the rooms themselves. The design of the housing and caging also plays an important role for a variety of reasons.

The slamming sound that cage doors make when they are closed and the creaking sound they make when they are opened can be avoided with improved products. Most manufacturers now produce quiet latches that click shut rather than slamming. Choose both quiet latches and hinges, if offered by the manufacturer.

Fear and agitation can be caused by reflective surfaces, especially among cats. Low-stress housing must be manufactured with low-gloss interiors. Based on cats’ and dogs’ abilities to see into the UV spectrum, some bright white plastics should also be avoided. Matte-finish stainless steel, high-pressure laminate, and light-colored resin products are all more appropriate choices.

For dogs, the balance between visual isolation and visual connection is tied to the needs of the particular dog, whether the dog is being housed for long or short periods, and whether a visual barrier is temporary or permanent. Permanent barriers on the fronts of kennels are not always appropriate because they can encourage some dogs to jump up to see out.

Given that dogs in medical settings are housed for short periods, most will benefit from the ability to see out of their enclosures. Based on a balance of the information available, provide the following:

- Dogs do poorly when facing each other at a close distance in dog wards. A design that prevents dogs from looking at other dogs across an aisle is best.
- The possibility of a clear view out of the enclosure at the dog’s eye level.
- The possibility to put up a temporary barrier for the extremely fearful or anxious dog. (Note that dogs with critical medical conditions should remain fully visible.) A simple sheet or towel hung on a run door will work. A manufactured product that serves the same purpose can also be purchased. Barriers should not be part of the permanent design of the kennel door.
- Greater privacy on other portions of the run to give the dog a sense of enclosure. For example, it has been established by a majority of experts that isolation panels between runs are necessary.
- If glass is used, the room must be ventilated through the enclosures for proper air flow.

Because dogs vary so much in size, the rules of thumb require discretion and interpretation from the hospital management team and the design professional. Healthy adult dogs must be able to move freely and express normal behaviors and assume normal postures within their enclosures to meet basic animal welfare standards. The only exception that applies is a necessary restriction of movement for medical reasons. Thus, if a healthy dog is too large to physically turn around in a cage without touching the cage walls, he should be housed in a run.

Runs in medical settings should be wide and shallow rather than deep and narrow. Deep and narrow runs promote poor behaviors and make the relationship between the dog and the caretaker more difficult. Wide and shallow runs encourage natural interactions between dogs and caregivers, allowing more space for staff to position themselves next to the dogs. These enclosures are also designed better for the size and shape of a dog.

Stress-free environments for healthy cats must also allow for normal behaviors and postures. A 36-inch-long cat cage will allow for this type of movement more effectively than the typical 24-inch cages.

**Lighting and sound engineering**

**Daylighting**

The benefits of daylighting have been well-established. They include:

- Reducing patient recovery time.
- Improved staff productivity in office environments.
- Improved retail sales.
- Improved performance in education settings.

Due to the fact that dogs and cats are psychologically similar to humans, daylighting provides the same healing benefits. Below are some criterion for effectively included daylighting in your building:

- In moderate climates, develop open plans, oriented on an east/west axis, that have a greater connection to the out-of-doors.
- In the Northern Hemisphere, window openings on the south side are most effective. These openings can be designed with overhangs that let in the low winter sun but block the high summer sun.
- Reduce glare. Avoid conditions where direct sunlight can come through a window or skylight without being softened by a translucent glazing system, louver, or sunshade.
- Locate openings in ways that benefit your animal patients the most.

There are a variety of ways to incorporate daylighting including windows to the exterior, high light shelves and clerestory lighting, and in the interior with overhead skylights and light tubes. Use caution when incorporating skylights to ensure a balance between daylighting and the introduction of too much heat.
Dimmable lighting
The veterinary industry has traditionally recommended a lighting level of 50 foot candles (fc) for areas where patients are examined, which may also include wards. This level of bright lighting can be stressful for patients, so lighting should be designed to dim or step down to 30 fc or less when full lighting levels are not required.

Animals’ hearing
The range of hearing for humans is 25 Hz – 20 kHz. Dogs hear ranges from 20 Hz up to 50 kHZ and cats hear frequencies measuring from 25 Hz – 64 kHz. In other words, dogs and cats hear high-frequency sounds that cannot be heard by people. In a building, high-frequency sounds may be emitted from electronic equipment, motors, and lighting.

In addition to these high-frequency noises, buildings typically emit low-frequency rumblings from mechanical systems. These low-frequency sounds have been shown to be stressful for humans, and may also be stressful for animals.

Considering that animals hear noises we don’t hear, and hear them better, preventing mechanical noise and vibration is a critical goal for designing stress-free, healing spaces for animals.

Here are some tips to prevent unnecessary noise from reaching animals in your hospital:

- Locate rooftop mechanical equipment over spaces other than animal wards.
- Provide mechanical equipment with internal vibration isolation.
- If you have an existing building, replace any old fluorescent lighting with new fixtures utilizing electronic ballasts. This will eliminate the buzzing noise that old fixtures emit.
- Locate other motors and mechanical equipment such as housekeeping vacuums, medical suction pumps, etc. in remote closets far away from animal and medical spaces.

Mechanical engineering
Indoor air quality affects the wellbeing of people and animals. While it is unlikely that a few hours in a poorly ventilated animal hospital can result in any lasting impact, veterinary hospitals are especially prone to poor indoor air quality because of odors, humidity, chemicals, heavy cleaning, and hair from pets. Research about the effects of building ventilation systems on both people and animals reinforces the idea that stress-free, pet-friendly hospitals should be ventilated with care and expertise.

Separation of species
Once again, separating dogs from cats has its advantages. In this case the requirement is to separate air exchange between dog and cat exam rooms, treatment and ward spaces. This separation may be achieved by 100 percent exhaust systems, or by providing separate mechanical units to serve the dog and cat areas of the building. The latter is a more practical approach for exam and treatment spaces that do not otherwise require 100 percent exhaust.

Negatively pressurize ward spaces
Differential pressurization is a well-known tool to control the spread of odors and airborne contaminates in hospitals. Ward spaces must be negatively pressurized, meaning that more air is exhausted than supplied from these spaces.

Air exchange minimums
More air needs to be exchanged in animal wards than in offices to comply with accepted animal health standards. At minimum, 12 - 15 air changes per hour are required in animal wards. The number of recommended air changes varies on the configuration and use of the ward. For rules of thumb, use these guidelines:

- Isolation wards should have 20 - 30 air changes per hour depending on the disease isolated and the size of the room. Small rooms need more air changes than large rooms.
- Run wards need 12 - 15 air changes per hour.
- Dog cage wards need 12 - 20 air changes per hour to control odors, unless the air is vented through cages.
- Cat cage wards need 15 - 20 air changes per hour to control odors, unless the air is vented through cages.

Ventilated caging
One of the biggest problems with housing animals in cages is that the cage itself is a barrier to adequate room air exchange. The solution to this problem is to provide individually ventilated cages. Individually ventilated cages are relatively simple to use. Air is supplied in front of the cage, is pulled through the cage, and is then exhausted from the top into the building exhaust ductwork. The simplest installation for ventilation of cages is to build the cages in permanently.

While ventilated caging may seem to be an extravagant expense for a hospital, ventilating through the cage generally allows for the overall room air exchange rate to be cut down. Therefore, if you are housing a number of animals, vented caging can pay for itself.

Pheromone dispensers
The use of pheromone dispensers can be very useful in reducing stress in spaces where species are separated. Locate enough power outlets in cat and dog wards, exam rooms and treatment areas to plug in pheromone dispensers to allow for the buildup of pheromones in these spaces.
Summary
The design of a stress-free, pet-friendly practice originates from a very simple idea. By considering all of the spaces within a hospital from our animals’ perspectives, we can create environments that are more responsive to their needs and health. This new point of view is beginning to shape the forward momentum of the veterinary industry and can help you differentiate your practice in today’s market.
Research shows that no more than 25% of one’s overall success is attributable to general intelligence or IQ. A good portion of that remaining 75% is related to emotional intelligence competencies: self-awareness, self-management, social awareness, and relationship management. After this session, you will understand the components of emotional intelligence (EI), understand the importance of EI as a competency in need of measurement when selecting for leaders, assess your own EI in order to have a frame of reference for selecting for EI in others, possess tools to identify ways to improve your own emotional intelligence, and be able to identify EI characteristics of effective leaders to measure in a selection process. You will leave the session with resources or tools for measuring EI, such as interview questions, written exercises, behavioral tools, and EI inventories.

The difference between IQ and EI
General intelligence (measured as IQ) is typically focused on analytic reasoning, verbal skills, and spatial ability. It’s a murky concept with many definitions and not the best predictor of work success. Emotional intelligence (EI) is the capacity for recognizing our own feelings and those of others, for motivating ourselves, and for managing emotions well in ourselves and in our relationships.

It’s critical that our team members and leaders have emotional intelligence in order to maximize our own personal success and the success of the practice. For every 1% improvement in the service climate, there is a 2% increase in “profit.”

EI and the brain
Emotions begin in the limbic area. There is an open loop relying on external cues. The main purpose of the innermost part of the brain is survival. Open loops mean that we affect and are affected by someone else’s emotion (e.g., ICU patients get better if someone is present with them). A leader transmits signals that can alter hormone levels and immune and cardiovascular functioning of others. In fact, after a 15-minute conversation, body rhythms and physiology match—a concept called mirroring. Leaders “manage meaning” for us. About 60% of perceptions about organizational climate can be traced directly to the leader. Workers are more likely to be overtaken by negative emotions (hijacked) after interactions with the boss rather than with peers or clients. Distress from hijacking lowers performance.

The components of EI

Social awareness includes empathy, organizational awareness, and service. Relationship management includes inspirational leadership, influence, developing others, change catalyst, conflict management, building bonds, and teamwork and collaboration.

When it comes to emotional self-awareness, the inability to notice our true feelings leaves us at their mercy. People with greater self-awareness are better pilots of their lives and are more confident about personal decisions. As a person matures, emotions begin to shape and improve thinking by directing a person’s attention to important changes. For example, a teacher becomes concerned about a lesson that needs to be completed for the next day. The teacher moves on to complete the task before concern takes over enjoyment.

You can develop social awareness through empathy. Empathy is the ability to recognize another’s emotional state. In research on married couples, empathy appears to include matching the physiological changes of the other person. Developing empathy leads to greater emotional stability, greater interpersonal sensitivity, and better performance at school, work, volunteer jobs, as well as with family.

The art of social relationships involves managing emotions in others. To excel at people skills means having and using the competencies to be an effective friend, negotiator, and leader. One should be able to guide an interaction, inspire others, make others comfortable in social situations, and influence and persuade others. The subtle and complex abilities that underlie people skills are being attuned to others’ emotions, promoting comfort in others through the proper use of display rules, and using your own emotional display to establish a sense of rapport.

Using EI to hire leaders
So, now that you understand EI, how do you hire people with an abundance of it so that they and your organization maximize success? First, let’s talk about traditional interviews. Have you ever hired someone who seemed fabulous in the interview and bombed on the job? Why does this happen so often? Interviews fail because we over-rely on our intuition and our “gut feelings.” People just aren’t good at predicting future behavior. If we were, would the divorce rate be 50%? We more often select for general intelligence or technical competencies rather than emotional intelligence.
When hiring for EI, the successful selection methods include the structured interview, both situational and behavioral; written exercises, written exercises, including reactions to case studies and EI assessment; and behavioral assessments, including presentations and role plays.

In structured interviews, types of interviewing questions include:

- **Credentials/Technical:** “Why did you pursue the undergraduate degree that you did?” (Self-Awareness)
- **Experience:** “Describe a time when you were presented with a problem about which you did not have all of the information to solve the problem. How did you proceed?” (Social Awareness, Self-Management)
- **Opinion:** “Coming in as a new employee, what would you do to get oriented to the organization?” (Social Awareness, Relationship Management) and “Choose one or the other. I have little trouble taking prudent risks or I am particularly cautious about change. Explain your answer.” (Self-Awareness)
- **Situational/Hypothetical:** “Assume you are confronted by a donor who is unhappy with something the organization has done. The donor is calling to let you know that they are going to discontinue paying on their pledge. What do you do?” (Relationship Management)
- **Behavior Description:** “Tell me about a time when you felt least effective or were most frustrated in your efforts to deal with a conflict between yourself and a co-worker.” (Self-Management, Relationship Management)

In behavior-based interviews, the best predictor of future behavior is past behavior in a similar circumstance. Here are some guidelines:

- **DON’T** ask questions that can clearly be answered by reading the application or the resume.
- **DO** balance the types of questions you ask.
- **DO** make the majority of your questions behavioral description questions.
- **DO** determine the “best” answer BEFORE the interviewing process begins.

In written exercises, you want the candidate to identify critical incidents. Use job descriptions, challenges, successes, and essay questions. Develop case studies, or have the candidate write media plans, case statements, or grant proposal outlines.

Three types of behavioral exercises are interpersonal role plays, tests of people skills, and knowledge application. Another is having the candidate give a presentation, which assesses communication, public speaking, networking, and training. Yet another behavioral exercise is mock meetings, which assess facilitation skills, leadership ability, teambuilding skills, and group process skills.

In order to assess the results of the EI-based exercises, you have to know what the correct “answer” is before you pose the question or elicit behavior. Narrative description is not enough. Match the most desirable answer to the dimensions of EI, including self-awareness, self-management, social awareness, and relationship management. Develop rating or scoring rubrics ahead of time so that you can easily evaluate the responses and compare the candidates.

**Examples of EI-based interview questions**

Here are some examples of EI interview questions that test self-awareness.

- **Impact on others**
  - Tell me about a time when you were surprised about the positive impact your behavior or words had on a coworker, a customer, or an employee. How did you learn this information? What did you do when you learned this information?
  - Tell me about a time when you did or said something and it had a negative impact on a coworker, a customer, or an employee.

- **Emotional and inner awareness**
  - Tell me about a time when you were distracted or preoccupied about something. How did you know? What impact did that have on your performance? What impact did it have on others at work?
  - Tell me about a time when you were in a good mood at work. How did that affect your performance? What impact did your mood have on others at work?

- **Accurate self-assessment**
  - Describe a time when you received feedback about your performance and were in agreement. What did you agree with?
  - Describe a time when you received feedback about your performance and you disagreed with that feedback. What did you disagree with?
  - Was there ever a time when you initially disagreed with feedback you received and later came to accept it? Tell me about that.

Here are some examples of EI interview questions that test self-management.

- **Emotional expression**
  - Tell me about a time when you were angry with someone at work. What did you do?
Has there ever been a situation at work where you said something and later regretted saying it? Tell me about that.

Describe a situation at work when you were very enthusiastic about something. How did your enthusiasm affect others?

Courage or assertiveness
- Tell me about a time when you spoke up about something in the workplace. What was the issue? Why did you speak up about it? What did you say? What did others think?
- Has there ever been a situation at work where you wish you had said something in a meeting or encounter but didn’t? Tell me about that.
- Describe what you did the last time someone blamed you for something at work that wasn’t your fault. What did you do?

Resilience
- Tell me about a time when you felt that you were defeated at work. What did you do?
- Tell me about a time when you were distracted by or preoccupied about something. What did you do?
- Tell me about a time when you felt like giving up on something. What did you do?

Awareness in the moment
- Tell me about a time when you realized that a conversation wasn’t going very well. What did you do?
- Tell me about a time when you realized that you weren’t speaking up during a meeting. What did you do?
- Tell me about a time when you realized that something was best left unsaid. What did you do?

Planning tone
- Tell me about a time when you deliberately planned the tone of a particular conversation. How did you do that? What result did it have?
- Tell me about a time when you missed an opportunity to set the tone in a discussion. What happened?

Respectful listening
- Think about a time when you didn’t understand something in the workplace. What did you do?
- Describe a situation when you didn’t understand why someone was acting a certain way or taking a certain position on some issue? What did you do?
- Describe a time when you jumped to conclusions.

Feeling the impact on others
- Tell me about a situation when you sensed something was bothering a peer or coworker. How did you know? What did you do?
- Describe a situation when you knew that something was wrong with a relationship you had with a peer, customer, or supervisor. What did you do?
- Relate a situation in which you determined that something that you did or said didn’t go over very well. How did you know?

Service orientation
- Tell me about a time when you offered assistance to someone without being asked. What did you do?
- Describe a situation when you offered assistance to someone even though it was outside of your job description. What did you do?
- Was there ever a time when you resented helping someone at work? Tell me about that.

Building relationships
- Who are some key people in your organization who you currently must work with on a regular basis to get your work done? Describe your relationship with these people.
- Describe your present responsibility for building and maintaining relationships at work. Whom do you build relationships with? How? Why?

Collaboration
- Describe a time when you had to solve a problem that involved or affected other people within the company. How did you solve it?
- Have you ever implemented an idea or solved a problem and had your solution met with resistance? What do you think you could have done to avoid the resistance?
- Describe a time when you sought someone’s ideas or opinions about a project or idea you were working on.

Conflict resolution
- Tell me about a dispute with a peer. What was it about? What did you do? How did it end up?
Tell me about a time when someone suggested something that you disagreed with. What did you say?

How have you resolved differences with peers or others?

Tell me about the process you use to resolve your difference.

- Organizational savvy
  - Did you ever have an opportunity to advance a new idea at your last job? How did you go about doing that?
  - Tell me about a time when you gained support for an idea that you had. How did you do that? Why was this idea important to you?
  - Describe a time when you couldn’t get support for an idea that you had. What happened? Why was this idea important to you?
In days past, managers had a goal of stability and predictability. The only thing predictable in today’s environment is that change is coming. Simple or complex, from a new phone protocol to a new facility, you can embrace the change.

The learned response to change is caution but caution can lead to paralysis. Today’s acceptable level of performance is tomorrow’s failure. If your practice has a culture with quick reflexes, you can pick up the speed that will protect you in the marketplace.

Fast growth necessitates change. To deal with growth, we are really talking about how we handle change in the workplace. Change compels us to get out of complacency.

Reaction to change: Panic or stay cool
Because change is scary, panic ensues and kills concentration. Action is important, but impulsive action makes things worse. If you stay cool, you can scramble without getting spastic. You can maintain a sense of urgency but still be steady under fire.

Is your practice complacent?
- The practice is not losing money; no big threats.
- The practice compares itself to the industry average, not the industry leaders.
- The typical manager or employee can work for months without encountering resistance or questioning.
- There is no highly visible crisis.
- The practice measures itself against low standards.
- Planning and control systems are rigged (or non-existent) to make it easy for everyone to reach goals.

When faced with change, you can wait for instructions or take the initiative. You must figure out what your business needs and be part of the solution.

To get change-ready:
- Push decision making down to the lowest possible level.
- Begin sharing information.
- Talk, but listen too. It is a two-way street.
- Encourage participation.
- Get into the trenches with front-line employees.
- Help people see the “why” of change, and work with them to discover “what.”

We confuse getting ready with progress. Roll up your sleeves, improvise, and LEARN. Inertia is more crippling than mistakes.

Frequent questions asked by those affected by change:
- “What is in it for me?”
- “Why is this important?”
- “How do these people even know what the problems are? They don’t bother to ask us.”
- “Do they really think they can change the entire practice at once?”
- “How much of our time and their money will they sink into this dry hole?”

The first step is to mobilize energy and commitment by sharing values and a clear vision. People do not argue with what they help create. You will get buy-in through participation. The group who will implement change must agree on what the problems are and why they need to change.

If change is dictated from the top, people will resist having solutions imposed upon them by individuals who lack familiarity with day-to-day operations. This resistance is expressed through lack of motivation and commitment to the change. You must have top level support—not control.

Try harder.
- People respond to change by putting out more effort. The greater the change, the more effort to stick with the same habits.
- You can’t handle change if you don’t change.
- Today’s problems are caused by yesterday’s solutions.

Try easier.
- Simplify, and search for different solutions.
- Get rid of things that are in the way.
- Innovate. Bust out of old routines.
The second step is to develop a shared vision
Do you have a mission, vision, and values? Vision must be put into action; owners and key managers must inspire others in the organization toward it. Vision is “what can be seen,” so focus on specifics about how to improve the business and how those changes will benefit the employees, patients, and clients.

**Components of shared vision**
- Describe a desirable future.
- Be compelling. It should be much BETTER than the current state of things.
- Be realistic. Make it within the grasp of the hard-working folks who will make it happen.
- Be focused on a manageable set of goals.
- Be flexible.
- Make it easy to communicate with all levels of employees.

You waste time and energy when change creates a culture of complaint and “poor me.” We want the good old days. We fret about the future, and it does not improve a thing.

Spend energy on solutions by buckling down and channeling your energy on productive things. Action is better therapy than tears. You can redirect emotions to produce results.

The third step is to hire qualified employees. Get the right people on the bus and the rest OFF! Answer the question: What isn’t getting done well? What is holding the practice back?

The surest security is a willingness to take risks. You need NERVE. Without risks, there are no breakthroughs. If we don’t move forward without road maps, we will trail the competition. Use your imagination and extend yourself. Put adventure into your work and see how far you can reach.

Plan for employee turnover and be cognizant of these issues:
- Would the departure of a key employee upset the applecart?
- As volume of business increases, some people will not keep up.
- Without clearly defined roles, new employees will leave.

**Systems are the answer to this problem.**
Identify the leaders in your practice. Good leaders have three characteristics:
- They believe that revitalization and change is the key to competitiveness.
- They articulate their conviction in the form of a credible and compelling vision.
- They have the people and organizational skills to implement their vision.

Rely more heavily on your strengths. You must thrive on pressure and not always rely on strengths. A culture of inflexibility develops if people rely too much on their strengths. Don’t let your strengths become your weaknesses. Keep learning, develop new directions, adapt. Show respect for what works.

The fourth step is to focus on results, not activities. If you cannot measure it, you cannot manage it. Operations experience and expertise is critical. You must identify and meet short-term goals, and regularly discuss them. Welcome destruction, change is destructive. It bothers people. Protecting “what is” sabotages “what could be.” Destruction is a condition for survival. It’s messy, makes noise, and people get upset. Reward disturbing the peace.

Step five is to implement systems. Establish your infrastructure for growth with HR, project management, and quality control. Create checks and balances for your practice. Reduce stress and make workloads predictable and manageable. This also helps with managing turnover.

Change makes people scared. They get jumpy about doing anything that makes them look bad. If we can’t tolerate failure, we don’t develop new competencies. Failure is a master educator.

Don’t be careless, but fail quickly and fail often. Success is usually a byproduct of screw-ups.

Step six is to start change at the periphery. The likelihood of success is greatest when change is implemented in small, autonomous units. Success breeds success.

Change brings out the “so-so” in people. Pressed to keep up, we are tempted to shave standards. We want to get even with the organization for requiring change. Raise your standards and pursue total quality. Instead of accepting less than your best, improve on it. Don’t tolerate so-so performance. Tough times define the culture.

**Change and growth: What is in it for me?**
- Clear advantage over the status quo
- Compatibility with people’s desires and values
- Requirements that are understandable
- Option for people to experience the change model at a small scale first
- The possibility of people observing the suggested change in another setting
Change triggers survival instincts. Looking out for number-one takes priority over looking out for the client. Focus on the team; there is strength in numbers. Rather than maneuver to protect yourself, focus on protecting the client… the client determines the fate of the organization.

Step seven is to train employees, and train again. Invest in training and supervision of the employee. They can’t read your mind. There is a direct correlation between training and productivity. Our goal is continuous improvement and change. This is a process, not an event.

Loyalty creates traitors. The organization tries to change, but the culture won’t let it. There is danger in being chained to outdated traditions and ways.

Show insensitivity to history to show respect for the future. Break with the past and celebrate achievements that make the organization more competitive. Defending the past cannot protect you from the future.

Step eight is to implement technology to support operations. Give people the tools they need to do their jobs. You cannot run a business on a wing and a prayer. Strategically plan and budget for technological improvements.

Change is hard on hope because problems cloud our view. We focus on what is wrong instead of possibilities. Bad news drives good news away.

Opportunity comes disguised as problems. The way you think influences the way you frame the situation, thus influencing the ability to deal. Believe in opportunities, and you help them appear.

Step nine is to improve quality. Deliver a consistent client experience. Develop checklists and scripts to recover from service failures.

Increase client satisfaction (value). Listen more than ever to what employees and clients say about their experience with your business. If clients are negatively impacted by a system (or lack of one), fix it quick! Complaints and surveys are a great thing.

Increase competitiveness by staying current with:
- What is important to your clients
- What is happening in the marketplace
- What your competition is doing
- What your practice is doing well
- What you need to do to improve

Change creates problems, and we look to blame others. Badmouthing the boss becomes a pastime. None of this slows down change or fixes the problem. Blaming is a devious way of avoiding responsibility.

Identify problems with a solution. Get busy doing what you can do instead of second-guessing somebody else’s efforts. Push for a culture of personal responsibility.

Step ten is to expand services, not just service. Add new procedures and profit centers. 80/20: Do you know the rule?

Adults get bogged down in routine and habit. Grown-ups resent the difficulties of change.

Kids embrace change. They thrive on it and treat it like a plaything. Kids insist on variety. Change is what keeps them from boredom. Life is a constant stream of break-throughs. Be curious instead of worried.

Maintain positive cash flow and control costs. Don’t “burn” cash. Running out of cash is the most common reason a business fails. Spend cautiously and get paid promptly.

The single biggest impediment to growth is the inability of the culture to change with the growth. We are often failures at implementation.

6 implementation problems
- It takes more time than we thought (76%).
- Major problems surface that we did not predict (74%).
- Coordination of activities was not effective enough (66%).
- Competing activities and crises distracted attention from implementing the strategic decisions (64%).
- Capabilities (skills and abilities) of employees involved with change were not sufficient (63%).
- Training and instructions were not clear to lower-level employees (63%).

Reaction to change: A sense of loss and anxiety
A common reaction to change is a sense of loss and anxiety. The typical employee spends eight hours a day doing, in general, fairly routine tasks, and the completion of those tasks becomes the culture. People will adapt, but there is a predictable series of responses.

Stages in reaction to change
- Shock
- Defensive retreat
- Acknowledgement
- Acceptance and adaptation
People get stuck for a few reasons:
- Change is not a single event.
- Everyone comes to the table with a different level of experience relative to change, and they have different coping skills.
- People who are emotionally fragile are particularly vulnerable during times of change.

Minimizing the negatives of change and growth
To minimize the negatives of change and growth remember to keep your cool and handle pressure smoothly. Keep your responses non-defensive. Develop creative and innovative solutions to problems. Be willing to take risks and try out new ideas. Be willing to adjust priorities. Demonstrate enthusiasm for long-term goals. Be open and candid. Participate. When a decision needs to be made, make it.

Management Development is the process of building the current and potential performance capabilities of an organization’s managers. The focus is on skill development and understanding how to think and behave as managers.

Functions of management development
- Enhance management skills.
- Shape the corporate culture.
- Promote leadership style.
- Reward and recognize managers.

Critical dimensions
- The person’s concept of their role
- The skills demanded by the role
- Attitudes and psychological factors

Change in role
The fundamental dimension of successful transition involves changing the concept of one’s organizational role from a performing doer to a managerial role of supervising the work of others.

Predicting the future is easy. It’s trying to figure out what is going on now that is difficult.
Client focus audit: Rate your key-customer service focus

- Why would key potential customers want to do business with you?
- What are you doing to ensure that your customers will be with you tomorrow?
- Are you fulfilling the promises you make to your customers every day?
- Are you respecting your customers’ time in every aspect of the service process?

Client focus audit

**Satisfaction**
- You treat the pet in a timely manner and within the estimate provided.
- You follow through with commitments.
- You exceed the client’s expectations within the confines of the treatment plan.

**Service**
- You understand the client’s expectations and exceed them.
- You pay attention to the client’s changing needs and perceptions. You are proactive!
- You focus the BUSINESS on the needs, desires, and wishes of the client.

**What is customer satisfaction?**
Customer satisfaction is focusing on the contractual and cultural expectations between the hospital and the client. Service is guided by the understanding of that agreement.

Customer service is making an effort to consistently and proactively serve the client; to be of service, not just follow a policy or a rule or an agreement.

**Implementing customer service**
- Change must come from owners and veterinarians. They must lead by example.
- Employees must be empowered and trained to make good business decisions.
- Balance the notion that the customer is always right with what is right for the business.

**Implementing customer service**
You can’t afford to fail at this!
- Listen carefully to find creative ways to exceed clients’ needs. Do not rely on assumptions.
- Focus on the customers’ evolving needs. Is service the same to you at every stage of life?
- Eliminate non-value-added mentality: “We’ve always done it this way.”
- Provide choices. Instead of “no,” how about, “Let’s explore alternatives.”
- Establishing a customer service culture is everyone’s responsibility.
- We all must take on the attitude of, “We are in business to take care of our customer!”

**Back to basics**
- Pick up the telephone!
- Is everyone trained properly?
- Do you have performance reviews that enforce client service?
- When is the last time you surveyed your clients?

**Client focus audit**
- How effectively do you communicate with your customers?
- Would your customers agree that you are not just MEETING their expectations but EXCEEDING them?
- Do policies in your practice make it easy for customers to do business with you?
- What is your USP? (Unique Selling Position)
- What do you consider your competitive advantages?

**Service or satisfaction?**

**Customer service**
- Where did you receive excellent customer service?
- Why was that service exceptional?
- What do customers like least about doing business with your organization?
Communication style

- How important is communication to good customer service?
- Does everyone communicate in the same style?
- Circle ALL the words that describe you when you are at WORK.
- Service

<table>
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<th>1</th>
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<td>3</td>
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<td>Supportive</td>
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<tr>
<td>4</td>
<td>Manipulative</td>
<td>Manipulative</td>
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Communication style

- Count the circles in each quadrant.
- Which quadrant has the most circles?
  - 1, 2, 3, or 4?

My communication style

Are you:
1. Analytical
2. Driver
3. Amiable
4. Expressive

Read about your style. Does it sound like you?

Communication with different styles

- High Analytical
- High Driver
- High Amiable
- High Expressive

Communication and service

- Understanding communication styles can be useful in customer service.
- How does communication impact customer service?

Exceptional customer service

Benefits of exceptional customer service

- Goodwill in the community
- Community support when real problems occur
- Pride in your agency and your career
- Better working environment; internal and external customer service
- Increased success with clients

What customers value

- Tangibles: appearance of equipment and personnel
- Reliability: ability to perform the expected service
- Responsiveness: willingness to help customers
- Assurance: knowledgeable, courteous employees, and their ability to convey trust and confidence
- Empathy: caring, individualized attention to customers

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What customers want
- Dignity and respect
- Services meet expectations
- Success
- Help with problems
- Treated as individuals
- Respect their self-image
- Respect their time
- Someone on their side
- Correct information

Put a smile in your voice
- Turn to the person next to you.
- Decide who will be the caller and who will answer.
- Turn your back to each other.
- With a frown on your face, introduce yourself to your partner. Tell him/her your name and ask if you can do anything to help him/her.
- Now with a smile on your face, ask the same.
- Can you tell a difference?

Tools of the telephone business
- Be direct.
- Be helpful.
- Be courteous when screening calls.
- Be careful with transfers.
- Be knowledgeable.
- Offer an explanation for delay.
- Thank the caller.
- Take good messages.

Customer service barriers
- Company policy
- Job specialization
- Lack of coordination between departments
- Remote decision-making power
- Arbitrary policies; inflexibility
- Top priority is cost-cutting
- Indifferent employees
- Not enough creative problem-solving
- Failure to listen to customers

Service recovery
- You will make mistakes. What angers customers is when the mistake is handled poorly.
- 70% of disgruntled customers will return if the business apologizes for an error.
- 95% will return if the frontline employee recovers impressively.

What is “service recovery?”
- Make it easy and convenient for customers to complain.
- Identify unhappy customers before they complain or go away.
- Turn mistakes into positive service stories.
- Keep track of reasons customers complain so problems can be eliminated.

Service recovery steps
1. Apologize.
2. Listen and empathize.
3. Fix the problem.
4. Offer atonement.
5. Keep your promises.
6. Follow up.
My customer service plan

- Plan for improving INTERNAL customer service with my co-workers
- Plan for improving EXTERNAL customer service with my customers

3 points to transition

- Mastery - Loving what we do and being competent in what we do. It makes our service EASIER.
- Chemistry - Building and nurturing strong relationships with others. It makes our service FRIENDLIER.
- Delivery - Willingness to meet the needs and exceed the expectations our customers every time. It makes service FASTER.

Where are we headed?

- Easier - Combines competency and care
- Friendlier - Creates a customer-focused culture
- Faster - Speeds up processes

Mastery

- Loving what we do and being competent in what we do. It makes our service EASIER.
- Each of us has a right to participate in educational trainings, work-related skill-building workshops, seminars, and instructional opportunities that reinforce our competency and professionalism.

Chemistry

- Building and nurturing strong relationships with each others. It makes our service FRIENDLIER.
- Each of us has the right to enjoy what we do and to have opportunities to build strong, positive interpersonal relationships with coworkers, staff, and customers.

Delivery

- Willingness to meet the needs and exceed the customers’ expectations every time. It makes service FASTER.
- Each of us has a right to provide excellent, swift customer service that has that WOW quality.

Are you an influencer?

- Has integrity with people
- Nurtures other people
- Has faith in people
- Listens to people
- Understands people
- Enlarges people
- Navigates for other people
- Connects with people
- Empowers people
- Creates and mentors other influencers
Veterinary medicine has gone through a tremendous change and so must our paradigms for delivery of service. It has gone from owner- and medicine-driven to client- and experience-driven. The new consumer is looking for an experience instead of a purchase.

The magic of client enchantment
Service magic is a set of learned skills developed through desire and determination. The service magician seeks to delight and stand apart from the crowd. It is not a bag of tricks or an illusion. It is rapport, communication, trust, and credibility. It is the art of crafting awe and wonder and creating pleasant surprises.

Service Magic is:
- Unexpected
- Unpredictable
- Valuable
- Memorable
- Reproducible

There are three well-researched approaches. The first is the RATER approach, and acronym for:
- Reliability is the ability to provide what was promised dependably and accurately.
- Assurance is the knowledge and courtesy of employees, and their ability to convey trust and confidence.
- Tangibles are the physical facilities and the equipment, and the appearance of personnel.
- Empathy is the degree of caring and individual attention provided to clients.
- Responsiveness is the willingness of the staff to help clients and provide prompt service.

John Goodman of TARP, a D.C. based customer-service research firm, performed a critical incident study of 1,000 hospitals to find out what behaviors delighted clients and how that affected loyalty. Three top delightful behaviors cited were:
- Consistently good service
- Proactively provides information
- Tells me of new advancements

The client is consistently delighted when:
- We do what we say we will do.
- We regularly communicate.
- We provide them with something new.

An emotional connection is created between the service deliverers and the delighted client who voluntarily tells love stories about those who serve. This extreme level of satisfaction is called CLIENT TRUST.

Service Magic Is Not…
- …happenstance or a fluke
- …dependent on an extravagant gesture
- …a complex set of practices
- …boastful or arrogant; rather, it’s confident
- …a value-added client service; it is unpredictable and unique
- …service with imagination more than service with generosity

The Service Magic Method is a two-step process—two very large steps: Opportunity quest and matching technique to opportunity.

Opportunity Quest
Points of impact and moments of truth where the client:
- Observes
- Assesses
- Evaluates

We need a cycle-of-service map or a moment-of-truth assessment.

Opportunities meet 4 criteria
The point of contact is necessary but routine
- Checking in
• Establishing credentials
• Placing an order
• Listening to instructions

The point of contact is annoying or unappealing
• Waiting in line
• Being kept o hold
• Waiting-room experiences
• Having to make choices

The point of contact is a creaky operation or a perilous process
• Getting lost
• Misreading instructions
• Missing a step
• Changing the routine

The point of contact is undistinguished or value-add-neutral
• Competitors are all doing it the same way.

Don’t add magic when
• Custom and propriety would be dislocated or disturbed
• Clients would view the change as peculiar or puzzling rather than pleasant
• Safety or welfare could be compromised
• The delivery system is broken

Match technique to opportunity
• Misdirection or direction: Distract the client’s attention from an unpleasant encounter or contact.
• Pattern interruption: Humans always search for patterns to make sense of what they experience. Do something different than what is expected in the moment.
• Force: It seems like a fair and free choice, but the outcome has been predetermined. Reframe the choices to make them seem more desirable. Shape the experience.

5 service-management tools
• Inclusion
• Personalization
• Patter
• Using confederates
• Outs or escapes

Where it happens
The three Ps of service magic
• Place
• Process
• Performance

Place magic
• Environments that delight, support, and enliven
• Magical places: venues that attract and please, subtly enhanced by human endeavor

Components of place magic are
• Nature or natural
• Signage, direction, and observation
• Convenience

Know your clients
• Understand who will be using your services.
• Wear their shoes.
• Organize the flow of people and ideas.
• Create a focus.
• Communicate visually.
• Avoid overload. Create turn-on.
• For every ounce of treatment, provide a ton of treat.
• Keep it up.
Process magic
- Policies, procedures, and routines that make transacting business with an organization, easy, positive, and memorable
- Meet client need with experiences of awe and memories of amazement

Two types of process
- Algorithmic
- Heuristics

Algorithms
Algorithms are step-by-step processes, kind of like rules of law, that focus on precision, replication, exactness, and dependability
- Red Rule algorithm: most effective; tested through trial and error; designed to maximize effectiveness, safety, and predictable outcomes (get it done right)
- Blue Rule algorithm: designed to maximize efficiency and sameness (get it done in a predictable manner; rote task)

Heuristics
- Rules of thumb for doing work, such as, “Greet each client warmly,” or “Treat patients with respect”
- Implicit in heuristics is that there is some sort of judgment call that has to be made about good service.
- When complex judgment calls are part of the service situation, creating complex guidance seldom results in improved service.

Adding magic to heuristics
- Never mess with the core expectation of consistency.
- Magic is provided by the person implementing the process, not the process itself.
- It cannot be too extreme, because the client will know that it is not likely to happen again.

Magic and algorithms
Red rule magic (Get it done correctly.)
- Make sure the alteration fits the process.
- Ask, “Is it reasonable that this brand of magic will not disrupt existing relationships?”
- Make sure it is done by the whole team.
- Done in matched tones (change the experience, not the process)
- Must be subtle
- Magic can happen alongside the process.
- Must be of the same nature as the core offering

Blue rule magic (Get it done in a particular manner.)
- Remember you are tampering with trust.
- Select a process that clients “must endure.”
- Don’t alter the process without looking at the whole experience.
- Include props (checklists, job aids, cueing devices) for the service provider in order to enforce consistency.

Restoring magic
Service-recovery scripts
- Coaching employees to say the right things and recover from service problems
- Should match the practice’s mission
- Discover what the client values and develop scripts around those issues.
- Get input from people who deliver the lines.
- Coaching employees to say the right things and recover from service problems
- Keep it simple.
- It IS a performance.
- It is a safety net.

Six steps to recovery
- It begins off stage.
- Clients know best.
- Fix the client, then fix the problem.
- Offer a fair resolution, then atone for inconvenience.
- Give employees responsible freedom.
- Follow up.
Off stage
- Create a clear policy for problem solving.
- Create a system for measuring and monitoring satisfaction.
- Recycle that information back to the staff.

The client’s eyes
- An expectation of fairness
- Personalized treatment
- Fix it fast. Don’t let it happen again.

Client first, then the problem
- We usually jump right to fixing the problem. DON’T! Instead, put the client first.
- Psychological repair comes first.
- Grovel and apologize.

They need...
- Information
- Recourse
- Expertise or know-how

Fair resolution/atonement
- Apologize and acknowledge.
- Listen and empathize: ask open-ended questions.
- Fix the problem quickly and fairly.
- Offer atonement.
- Follow up.
- Keep your promises.

Involve the client
- Clients who participate in the recovery process feel more magic.
- Contrary to popular belief, they bring a sense of fairness to the table.

Responsible freedom
- Give your employees some autonomy.
- Create policies that allow them to share in the process.

Follow-up
- Nothing creates magic more than follow-up.
- Some clients fear retaliation and will not voice their concerns when they happen, (especially in health care).
Service 301:
Managing the Client Service Puzzle
Shawn McVey, MA, MSW
McVey Management Solutions
Austin, TX

(From Harvard Business Review’s “The Four Things A Service Business Must Get Right”)

Best practices aren’t enough
- Most managers/owners are waiting for that one great idea or the magic bullet to fix customer service.
- Other managers/owners try to implement best practices from other companies or conferences.
- But, there is no such thing as a good idea in isolation! There are only good ideas in the CONTEXT of an overall service model/plan.
- In other words, what worked for them may not be what works best for you!

Service context
To consistently and sustainably delivery service excellence, the entire service context has to work together.
- What your service offering consists of
- How you fund the excellence you want to provide
- How you manage employees to deliver quality service
- What you do to help customers enhance – not erode - service

Service offering
- Determine how customers define “excellence” when it comes to your offering.
- Your management team must be absolutely clear about which attributes of service the business will compete on.

Most business failure is because of trying to design a service model to cover a huge range of customers and remain competitive across them. Successful businesses know what they do well that matters to their customers and choose to focus on that and not over-invest in what they do poorly.

Organizations that are superior at all service attributes usually demand a price premium of 50% over their competitors and most industries don’t support that type of premium. You can choose between excellence paired with inferior performance on one hand or mediocrity across all dimensions on the other. Accepting that inferior performance in one dimension fuels superior performance in another is the first step to designing excellent service.

ACTION: Define your attributes
Managers need to determine which attributes to target for excellence and which to target for inferior performance – based on the needs of your customers. Discover the relative importance customers place on attributes and then match the investment in excellence with those priorities.

“Wal-Mart’s value proposition”
- Ambience and sales help are least valued by Wal-Mart’s customers, low prices and wide selection are most valued.
- The trade-offs Wal-Mart makes are deliberately informed by these preferences.
- They optimize the service offering to cater to their customers’ priorities and REFUSE to over-invest in underappreciated attributes.
- Shoppers whose preferences match Wal-Mart’s strengths self-select into its customer base.

Commerce Bank’s strategy
- Commerce Bank has been able to grow its retail customer base dramatically even though its rates are among the worst in its markets and it has made limited acquisitions.
- They focus on the set of customers who care most about the experience of a physical branch.
- Customers are varied in demographics (young, first-time bankers, urban professionals, elderly retired folks) but united in priorities (ambience, convenience & friendliness).
- They added to ambience with interior elements like high ceilings and natural light and added a fun contraption for redeeming loose change.
- For attributes less important to their customers (price, product range), they are willing to cede the battle to competitors.

So, ask your customers
What do your customers generally agree constitutes excellent service? Is it convenience? Friendliness? Extended hours? Closer proximity? Ambience? Lower prices? Provide your current customers a list of possible service attributes that you believe your hospital provides well and ask them to prioritize the list in terms of matters most to them. Focus your service offering on the few attributes that matter most.
List of possible attributes
- Ambiance – smell, lighting, comfort of lobby, ease of movement through hospital
- Price/cost
- Convenience – proximity, hours, availability of appointments, duration of visit
- Friendliness – courteous staff, remembers my pet’s name, smiles, etc.

What attributes would you use to describe your practice?

Service design audit: The offering
- Which service attributes do you target for excellence?
- Which ones do you compromise on in order to achieve excellence in other areas?
- How do your service attributes match up with your targeted customers’ priorities?

Funding mechanism
Consider how you will pay for the increased cost of the excellence you’re seeking to provide through your service offering.
- Charge the customer
- Create savings & value-added services (win-win)
- Spending now to save later
- Having customers do the work

Charge the customer
- Make sure the customer is charged in the most palatable way.
- Management should creatively consider what feels fair to its customers.
- Often, the least creative solution is to charge more for the particular service feature you are funding.

What are some ways you could creatively & palatably charge customers to fund excellent service?

Starbucks’ appeal
Starbucks’s appeal is that customers canlinger almost indefinitely in a coffeehouse setting. Rather than putting meters next to their overstuffed chairs to charge customers for their length of stay, they charge a premium price for the coffee. The higher priced coffee pays for all that goes into creating and maintaining the coffeehouse setting…furniture, staffing, lighting, etc.

Commerce Bank convenience
They stay open late and on weekends – because their customers care about convenience and ambience of the physical branch. It pays for that service by giving a half percentage point less in interest for deposits. It could fund extra labor hours by charging for evening or weekend visits, but a slightly lower interest rate is more palatable to its customers.

Create a win-win
Create synergies between operational savings and value-added services. Very clever management teams discover ways to enhance the customer experience even while spending less. Some of these innovations provide only a temporary competitive advantage, as they are quickly recognized and copied – others are surprisingly durable.

Progressive’s win-win
The Company immediately sends out a van to assist a person involved in an auto accident – often arriving on the scene before the police or tow trucks. Customers love this responsiveness and ranks service highly, even though they wouldn’t be willing to pay more for it. Progressive funds this “excellence” because it allows them to save money on insurance fraud since a representative is sent to the scene – lower fraud loss & lower legal fees.

How can you find win-wins?
Most managers’ impulse is to imagine what new value could be created for customers and then to ponder how that could be funded through cost-savings. Rather, consider what are your biggest cost buckets? With these in mind, managers can then simultaneously determine how to reduce costs and create a value-added service.

Spend now to save later
It is possible, if somewhat painful, to make operational investments that will pay off by eventually reducing customers’ needs for auxiliary service in the future.

Intuit’s call center
They provide free customers support (very uncommon in the software industry), even though call centers are expensive to staff. They have their development people fielding calls so that subsequent versions of its offerings will be informed by direct knowledge of what users are trying to accomplish and how they are being frustrated. This investment drives their feedback-driven improvements for better software with the next offering.
Have the customer do the work
If the goal is service excellence, you must create a situation in which the customer will prefer the do-it-yourself capability over a readily available full-service alternative. If the self-service option is truly preferable, customers should be willing to take on the work for nothing or even pay for the privilege.
What work could you transfer to the customer?

Airline ticket kiosks
Frequent fliers now prefer kiosks because they provide readier access to useful tools like seat maps and often shorter lines.

Service design audit
The funding mechanism
- Are customers paying as palatably as possible?
- Can operational benefits be reaped from service features?
- Are there longer-term benefits to current service features?
- Are customers happily choosing to perform work (without the lure of a discount) or just trying to avoid more miserable alternatives?

Employee management system
Ensure that your workforce management activities (recruiting, selection, training, job design) empower employees to deliver the excellence embodied in your service offerings. If your business requires heroism from your employees to keep customers happy, then you have a bad design. Employee self-sacrifice us rarely a sustainable resource. Instead, design a system that allows the average employee to thrive.

Employee management system
- Ask yourself, what makes our employees reasonably able to achieve excellence?
- And, what makes our employees reasonably motivated to achieve excellence?
- Thoughtfully considered, the answers will translate into company-specific policies and programs.

Commerce Bank’s formula
This bank chooses to compete on extended hours and friendly interactions and not on low price or product breadth. Since strategy informs employee management, Commerce decided they don’t need straight-A students to master their limited product set, so they hire for attitude and train for service. Many people are reluctant to acknowledge a trade-off between aptitude and attitude, but failure to accommodate this economic reality in the design of the employee management system is a common culprit in flawed service.

Commerce Bank’s team
- Managers use simple weed-out criteria like “Does this person smile in a resting state?” rather than trying to maximize across a wider range of positive characteristics.
- Because “friendly” people prefer working with other “friendly” folks, the employees are empowered to recruit co-workers.
- When they experience great service in another setting, they can hand out a card printed with a compliment and a suggestion to consider working for Commerce.
- Because they hire for attitude, they must engineer things so that even lower-aptitude employees will reliably deliver great service.

Service design audit: The employee management system
- What makes employees reasonably able to produce excellence?
- What makes them reasonably motivated to produce excellence?
- Have jobs been designed realistically, given employees selection, training and motivation challenges?

Customer management system
In a service business, employees AND customers are both part of the value-creation process. Articulate which behaviors customers must demonstrate to get the most value from your service. Then design your service specifically to foster those behaviors.

Keep it simple
To get customers using the new self-check-in kiosks, airlines ensured that travelers could complete the transactions with far fewer keystrokes than the check-in personnel used to need. By contrast, retail stores that offer self-service checkout machines haven’t made using those machines easy for shoppers. Stores expect shoppers to shoulder responsibility for fraud prevents by weighing bags during check-out, which makes customers anxious to they avoid the machines.

Your customer management system
Ask yourself, which customers are you focusing on? Which behaviors do you want? And which techniques will most effectively influence behavior? Manage customers in a way that is consistent with the service attributes you’ve chosen to emphasize overall.

Influencing customers
- Instrumental – incentives and dis-incentives like discounts and late fees
• Normative – the use of shame, blame and pride that gets us to return shopping carts and pick up trash when no one is looking
• Which methods have you found most effective for influencing your customers’ behavior?

Service design audit: The customer management system
• Which customers are you incorporating into your operations?
• What is their job design?
• What have you done to ensure they have the skills to do the job?
• What have you done to ensure they want to do the job?
• How will you manage any gaps in their performance?

Integrating the elements
Successful service companies have a working plan that incorporates all four elements into the service design. Within each of those areas, however, it is hard to spot any best practice. The whole business depends more on the interconnection of the four than any one element.

Cleveland clinic
• Consistently ranked among America’s most eminent hospitals and a leader in pioneering cardiac care for decades.
• No financial rewards tied to doctor productivity, allowing doctors to spend time on innovation.
• Organizing centers by disease, rather than by specialization which encourages cross-collaboration.
• No clear exact source of their advantage, but the choices made complement one another and come together in a smoothly operating system.

Service design audit: The whole service model
• Are the decisions you make in one dimension supported by those you’ve made in the others?
• Does the service model create long-term value for customers, employees, and shareholders?
• How well do extensions to your core business fit with your existing service model?
• Are you trying to be all things to all people or be specific things to specific people?
10 “Must” Things I Would Do in Practice
(Parts 1 and 2)
Mark Opperman, CVPM
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1. Clearly state to my entire team the mission and vision of my practice. Everyone needs to know who we are, what we are and what I want us to be “when we grow up”. I will live that vision in my words and actions.

2. Determine my practices Niche and make sure my practice stays true to that Niche

3. Hire "10" Employees and Develop a training program to insure standards of medicine and customer service. Use video evaluation to "inspect the expect".

4. Incorporate an employee incentive program so that the team has a stake in the success of our practice.

5. Hire "10" associate veterinarians, develop an excellent mentorship program, pay them Pro Sal and after 5 years offer them the option of a percentage buy in to the practice.

6. Use the best in technology; laboratory equipment, digital radiology, VOIP phone system, veterinary software program.

7. Develop an amazing website, be active on social media, be a very high touch, high tech practice.

8. Determine a fair fee schedule, make sure all team members understand our fee schedule and are in support of it and teach the team to show the value of our services to our clients.

9. Hire a "10" practice manager and work with him or her to understand the objectives of our practice and make sure they are being met and hopefully exceeded.

10. Enjoy every day, share that joy with my team as well as the success of the practice. Give back to the community and to those we serve, both clients and patients.

The ten "must" things I would do in my practice
1. Clearly state to my entire team the mission and vision of my practice. Everyone needs to know who we are, what we are and what I want us to be “when we grow up”. I will live that vision in my words and actions.

How to create a "ten" practice

I. What is a "Ten" Practice?

A. Factors that go into Creating a "Ten" Practice and Others that We Must Overcome When veterinarians first open their practices, the excitement of doing so and also the "fear" due to the economic involvement or indebtedness, are factors that help to make the Initial practice successful and grow.

B. When a veterinarian reaches a certain point where he/she is economically more secure, the excitement diminishes and the practice stabilizes.

C. A type of "clinical depression" sets in.

D. There is a "fear" to incorporate change and, due to this, the practice stabilizes and sometimes levels off or deteriorates. At this point, many veterinarians become disheartened with the practice and the profession.

E. Therefore, one must recognize that change is important and one must be willing to take the steps necessary to achieve the attainment of a "ten" practice.

F. If you are not on the economic edge and not making it financially, you do not have anything to lose and might as well go for it.

G. One must be committed to go through one's fear. If there is anything in your work environment that you do not enjoy, then you must be willing to change it.

H. Your practice is a Rorschach of your life. How you run your practice is how you run your life!

I. One must commit to change.

J. "Life Sickness" can happen.
    a. Caused by the unwillingness to change when all that which is all around you is changing.
    b. This is the opportunity we have to really make a difference in our lives.
    c. This is the only opportunity

K. What things in your life add meaning to your life?
    a. We have the opportunity to have our practices any way we want if we just have the courage to do it.

2. Determine my practice's Niche and make sure my practice stays true to that Niche.
How to enhance your practice's "perception of value"

Make the office visit a pleasurable experience

- Warm and Professional Atmosphere
  - Current reading material
  - Client education
  - leash hooks attached to reception desk
  - Coffee
  - Kiddie Corner
    - Treasure chest of toys, coloring books
  - Pet carriers
  - Personalized leashes
  - Background music
  - ID collars and tags
  - Obtaining patient weight during each visit
    - Microchip
  - Uniforms and name badges
  - Being attentive to the client's needs
- Internet Pharmacies
  - What percentage of your income comes from pharmacy sales?
  - How to effectively deal with internet pharmacies

Exam room report card

3. Hire "I 0" Employees and Develop a training program to insure standards of medicine and customer service. Use video evaluation to “inspect the expect”.

4. Incorporate an employee incentive program so that the team has a stake in the success of our practice.

VMC team incentive program

The problem with most incentive programs

We have all read about various employee incentive programs and you may have even tried to implement some of these programs in your own practice. Often, incentive programs reward employees based on a specific goal or metric, such as the number of new patient visits or increased sales of specific services such as dental services, fecal tests or microchip implants, or sales of specific products, such as heartworm prevention or dental products. Unfortunately, I have found these types of programs are not highly effective in motivating team members. The problem with most of these incentive programs is that not all team members are involved in a particular program, so results are short lived. Also, the amount of additional bookkeeping required to manage such programs tends to become burdensome. In many cases, an ineffective team incentive program can even create back-biting and tension among team members. Naturally, the intent of any incentive program is to reward your team for their dedicated efforts and to let them share in the fruits of their labor. There is a type of team incentive program that accomplishes this goal, and it is one that we have successfully implemented in many of our consulting practices. In the years since we first began using this incentive program, I have found that, without question, the results are extremely positive and long-lived. This is not the type of incentive program that produces short-term results, nor does it only provide benefits to only a few select employees. Instead, all team members are rewarded based on the quality and quantity of effort they have put into the practice.

How this incentive plan works

The VMC Team Incentive Program has two components that are critical to its success. The first is the incorporation of a team approach within the practice; the second is effective communication between employer and employee.

Simplified, the VMC Team Incentive Program works as follows. First, we calculate the increase in the practice's gross income for one quarter as compared to the same quarter of the previous year. A predetermined percentage of this amount (usually 10%) is set aside in an employee incentive bonus fund. After the end of each quarter, employees are evaluated by the practice manager or the owner of the practice using individual employee evaluation forms. Based on their individual evaluation score and the number of hours worked, each employee receives a proportional amount of the employee incentive fund.

The team approach

For this incentive program to be successful, employees must work together as a team and each must know what their place is within the team. Every team member must know what exactly he or she can do in order to help increase productivity, efficiency, and profitability within the practice. Therefore, a list of specific tasks and procedures should be presented to each team member. For example, receptionists must be instructed to take a "full service approach" with clients and to verify that patient information
regarding past vaccinations and other routine procedures is obtained on all patients entering the practice. Exam room technicians must be instructed how to effectively market the practice's services and to educate clients about topics that pertain to their pet's health, such as preventative procedures, dietary needs, and flea and tick control programs. In this manner, every team member will know exactly what they can do personally to increase the efficiency and profitability of the practice.

Open lines of communication

The second important component of this incentive system is the performance evaluation. As stated, each team member will be evaluated by the practice manager or the practice owner after the conclusion of each quarter. For part-time employees, the evaluation score obtained will be adjusted based on the number of hours the team member worked during that quarter. This helps to ensure that team members are rewarded based on their time spent in the practice as well as for the quality of their performance. All performance evaluations need to function as an open line of communication between the employer and employee. In addition to having the employer evaluate the team member, the team member also should be asked to evaluate him or her own performance using the same evaluation form prior to their performance review. Evaluation forms will then be compared and reviewed with the team member during their review and this exchange will provide a forum for discussion. It is important to keep an open mind during this process. If a team member feels they were judged unfairly in a particular area and can substantiate that claim, then a change may need to be made in his or her evaluation score. By reviewing these evaluations with team members, they will become aware of how their performance is perceived by the practice owner and what they personally need to do in order to improve their evaluation scores. It has been my experience that this incentive system develops "10" employees in a short period of time.

As stated, performance reviews should be done quarterly to give team members the opportunity to improve upon their performance and therefore receive a greater proportion of the incentive fund. It should be the goal of the practice that all team members eventually score 100 on their evaluation and are able to share equally in the fund.

A program for all-full-time and part-time

Once quarterly evaluations have been completed and reviewed with your team, the next step is to adjust the scores based on the average number of hours worked during the evaluation period. As an example; if a team member worked an average of 20 hours a week, their score would need to be cut in half, because 20 hours is half the amount of hours of a full-time employee (40 hours). The score is adjusted based on the average number of hours the team member worked during that quarter as compared to a full-time 40 hour a week employee.

The beauty of this program is that there is no discrimination. A kennel assistant may get the same or greater bonus than a receptionist or technician—it all depends upon how well they do their job and the number of hours they work. This is a very important aspect of the incentive program. I truly feel that one person is not more important than another. If someone does well in their job and therefore gets a good evaluation, they have just as much right to receive a bonus as anyone else in the practice.

Modifying the program to suit your practice

There have been many variations to this program implemented within various practices. In younger practices or in a practice that might have hired a new veterinarian or built a new building, we have reduced the 10% of the increase in gross. The 10% of the increase in gross is the maximum figure I would suggest you use to fund the incentive program, but in many cases it can and should be less.

I would also suggest you set a minimum score a team member must receive before the adjustment for hours in order for the team member to receive any money from the incentive program. So, as an example, we might say that an employee must get a score of 70 or 80 (out of 100) on their evaluation if they are to get anything out of the incentive fund. Again, this score would be before the adjustment for hours. Why would we want to give a bonus to a team member whose performance rating was only a 50 or 60? Another caveat I normally include in the incentive program is that an employee must have been employed during the entire incentive period. If a team member comes in during or leaves during the quarter, they will not receive anything from the incentive program; it is an all or nothing situation.

Give your team an incentive!

The VMC Team Incentive Program has had phenomenal success in the practices where it has been introduced. It rewards team members based on their individual performance and contribution to the practice. It opens up lines of communication and informs the individual team member what they need to do in order to improve their performance. This program avoids the major pitfall of other employee incentive programs which is that employees who are not truly dedicated and making a conscientious effort are rewarded along with everyone else on the team. There is nothing more frustrating to a hard-working employee than to have another team member who is marginal and does not put forth that concerted effort still be equally rewarded.

That is why we stipulate that team members must receive a 70% or greater on their evaluation, before the adjustment for hours, if they are to receive any funds from the incentive program. I have found that this incentive program creates a "team approach" and has a long-term beneficial effect, not only on the practice's profitability, but also on the employees themselves.
We are all well aware that the success of a practice depends in a large part on the quality of team within it. Bringing our employees into an incentive program such as this, creating a team environment, and rewarding team members based on the accomplishment of the practice's goals, is truly a win-win situation for all involved.

In implementing this incentive program, almost any performance evaluation form can be used, just as long as the grading scale can be converted to a numeric scale of 0 (low) to 100 (high). I would recommend that you utilize evaluation forms that are not only specific to the veterinary industry, but also customized to each team member's job description within the practice. (VMC, Inc. has many templates for job descriptions that are customized for various positions within a veterinary practice, such as receptionist, technician, veterinary assistant, etc. Contact VMC or visit www.vmc-inc.com for more information.) If you opt to create your own evaluation forms, I would suggest that evaluation forms consist of 20 criteria that will be scored from 0 to 5 points with a maximum of 100 points.

Once you have scored performance evaluations, you will then determine the percentage of hours each team member works as compared to a full-time employee. So let's say you determine an employee works an average of 30 hours a week, 30 divided by 40 equals 75%. So you would calculate take 75% of the employee's evaluation score. Once you have adjusted scores based on the hours team members work, the next step is to add up all the adjusted scores and divide each score by the total to figure out what percentage of the incentive fund each team member receives. You will find the calculation sheets and the Excel team incentive worksheet provided with this package will assist you in this process.

**Keep your team motivated**

In some cases, practices have found that, after a year or two, the incentive bonus program loses its edge and no longer motivates employees. Team members may even start to see the incentive bonus as an entitlement. If this happens with your program, you may want to consider applying the Dolphin Theory of Management. The Dolphin Theory of Management comes from the dolphin shows we have all seen. How do trainers get a dolphin to jump out of the water and through a hoop? Well, it is a process that is first begun by placing a hoop under the water. The trainer will coach the dolphin to swim through the hoop underwater and, when that happens, the dolphin is rewarded with a fish. After repetition and reward has ingrained this behavior in the dolphin, the trainer will raise the hoop a little and repeat the training. Each time the dolphin learns to go through the hoop, the hoop is raised a little more until eventually the hoop is out of the water and the dolphin is jumping through it.

Okay, now let's use that theory and make the analogy that our team members are dolphins and the hoop is the evaluation form. As your team members achieve a score of 90 or 100 on their evaluation form, you must "raise the hoop," or update the evaluation form. Remove some of the criteria they are already performing well on and substitute in new criteria that challenges the team member. Yes, this will be a lot of work, but it is so very necessary. Team members can never feel that their job is a dead-end. They should know that there are always new challenges ahead and that management is going to help them to achieve them. This is the Dolphin Theory of Management and you will find it will help to keep your team motivated and "jumping through the hoop."

**Quarterly is a must**

A cornerstone of this program is quarterly performance evaluations. I have known some practices to try to do six month, or even yearly, evaluations and the result was that the program really didn't work. For some reason, quarterly performance evaluations are a must. Yes they are time consuming, but consider this: when you do quarterly evaluations, they don't have to be as in-depth as if you just did yearly evaluations. So you should only have to spend about 30 minutes per employee on the quarterly evaluations. In addition, I would argue that one of the real values of the incentive program is that employees receive feedback on a quarterly basis, which is something they truly desire. In fact, this may be one of the most motivating factors of the incentive program- not be the actual cash reward, but instead, the quarterly feedback on their performance. It is absolutely necessary to the success of this program that you conduct effective quarterly reviews incorporating the Dolphin Theory of Management.

**Scoreboarding**

The final key to success with this program is keeping your team informed as to how well the practice is doing. This is a management concept known as scoreboarding. Consider this: how well would a football, baseball or any team do if they did not know the score until the game was over? Well, that is how many of your team members feel. They have no idea of how well the practice is doing and what they might do to help improve the situation. It is not fair to team members to not tell them until after the quarter is ended if there was an increase in gross or not. So, as part of the incentive program, it is imperative that team members be informed of the practice's success. This can be done by simply posting a graph on the wall in your team lounge that shows income from last year, and then having someone updated the current income on a weekly or monthly basis. You may find that, as the end of the month draws near, if the practice has not exceeded its income from the previous year, team members may band together and step up their work in order to increase the income. It is crucial that you let team members in on the "secret" of how well the practice is doing and make sure they know what they personally can do to help.
Commonly asked questions
- "What should I do if there has been no increase in income between one year and the next?"

If there is no increase between one quarter as compared to the same quarter of the previous year, the practice should still do evaluations on all team members. In fact, you can turn this negative situation into a positive one. During the review, team members can be informed as to what they can and should do in order to ensure that there is an increase of gross in the next quarter. It may be helpful to analyze exactly which profit centers did not perform well over the past quarter and set some specific goals for the quarter to come. This otherwise negative situation can therefore turn into a very positive one and give added motivation to your team.
- "Should I continue to do annual performance evaluations in addition to the quarterly ones I am doing for the incentive program?"

One of the quarterly evaluations should also serve as the annual evaluation. I recommend that a practice only conduct four evaluations per year. Naturally, each of these evaluations will be used to determine compensation out of the incentive fund, but the evaluation that falls closest to the anniversary date of employment should be used to determine any adjustments to the employee's base salary or hourly rate.
- "How can I use my practice's existing evaluation forms with this program?"

Regardless of what evaluation form is used, there must be the possibility of scoring zero to 100 points. A tool to convert individual evaluation scores to this scale is provided in the Excel2014 Team Incentive Workbook, which is included with this packet. You could also perform the calculations manually in order to convert your evaluation form to a 0-100 scale. This method is described below.

The simplified formula is as follows:

- 100 points / No. of Areas (criteria) = Points Per Area
- Points Per Area / Highest Possible Score = "N"
- Total Score For All Areas * "N" = Evaluation Score

So, let's assume that your evaluation form has a total of 20 different criteria upon which an employee is graded. The grading system is such that scores range from 1 for unsatisfactory to 4 indicating excellence. Then, by dividing 20 into 100 points, you determine that each criterion is worth 5 points. The highest score available for each criterion is 4, so you then divide 5 points by 4. The resulting figure is 1.25. Therefore, once an employee has been evaluated, you should total the scores for all twenty areas and multiply this total by 1.25. The resulting figure will be the score you will use in determining the employee incentive bonus. Again, a perfect score must be 100 points for the purposes of this program.
- "Should team members be told how their bonuses were calculated?"

The answer is a resounding yes! Team members need to be told what their score will be and, in fact, during the discussion with the employee, your evaluation as well as their own evaluation of themselves should be reviewed and the final score should be determined. It is not recommended that you share other employees' scores with a given employee, however, that is most likely to occur.
- "How can I keep my team informed of how well the practice is doing and whether or not there will be a bonus at the end of the incentive quarter?"

This may be the most important question of all. Team members need to be continually updated and aware of how well the practice is doing. The concept is known as scoreboarding, as previously described. I would recommend that you post a graph comparing each week's income to that of the same week of the previous year. If weekly income is not available, then at least post the monthly income as a target figure and, week by week, indicate how well the practice is doing to reach that target. It is extremely important to the success of this incentive program that team members be informed on a continuous basis, not only how well they are doing in regards to their job, but also, the financial success of the practice.

Team members need to "know the score," or how well the practice is doing, but equally important is that they know what they can do personally in order to improve the practice. As described previously within this article (see "The Team Approach" on pages 1-2), employees should be given a list of the specific ways they can improve the practice's efficiency and productivity, which will then result in increased gross income.

At your team meetings, I suggest you discuss this topic with all team members. Receptionists must know that he or she must provide excellent customer service both over the phone and in person. They should be taught to ask the client if they need any additional products when they are receiving out the client, and to offer to send phone shoppers your hospital brochure. Exam room assistants should make sure they review the pre-exam check list and inform clients of additional preventative procedures the pet might need for optimum wellness. The kennel assistant should be instructed that they must be friendly when admitting a patient, not make the client wait and even help bring the patient to the client's car upon discharge. Everyone plays a critical role in the practice's success, no one person is more important than another and each must know what their role is to help the practice become even more successful.

5. Hire "10" associate veterinarians, develop an excellent mentorship program, pay them ProSal and after 5 years offer them the option of a percentage buy in to the practice.

805
6. Use the best in technology; laboratory equipment, digital radiology, VOIP phone system, veterinary software program.

7. Develop an amazing website, be active on social media, be a very high touch, high tech practice.

10 reasons to update your website
1. Significant changes have taken place recently with Google Search engines and you may not be getting the traffic you have previously without adapting to these changes.
2. Are you mobile friendly? Your website needs to be easy to access on smartphones, iPads, tablets, etc.
3. Your website should be fresh and inviting. Tired, old websites, say we don't keep up with the needs and interests of clients.
4. Your clients want someone who has good information and will become of their regular community of online resources.
5. Your website should be in alignment with your marketing efforts, pull those efforts through your site.
6. Does your site identify all new services, products and activities you offer? Give viewers a reason to visit you now.
7. Know your bounce rate. Are potential clients coming to your site and then immediately leaving? You need a fresh face to attract and encourage viewers to stay and return.
8. Does it load easy on a mobile device? The site needs to be updated regularly to assure ease of access.
9. Navigation of a website has improved dramatically in the last couple of years, is your site simple to move around?
10. Social media integration is mission critical; your website is the landing page to encourage viewers to see your other social media sites.

8. Determine a fair fee schedule, make sure all team members understand our fee schedule and are in support of it and teach the team to show the value of our services to our clients.

How to determine your client fees fairly & accurately
1. “Shopped” and “Exposed” Services
2. Calculate Your Fees Based On:
   A. Overhead costs per minute
   B. Direct costs at percent (%) mark-up
   C. Return on time to the doctor
3. In Hospital Service Fees - Overhead costs per minute
   A. Calculate overhead costs/minute/DVM
   B. Formula: ALL expenses- compensation to doctors (both owners & associates) including related costs - inventory costs + number of hours the doctors are scheduled (both office hours & surgery) +60
4. In Hospital Fees - Direct costs
   A. Direct costs definition: Inventory costs, costs of materials used in the procedure
   B. Formula: 2x cost due to costs of ordering
5. In Hospital Fees - Return on time to the Doctor
   A. Return on time to the Doctor definition: Per minute cost of the doctor’s time spent on a procedure
   B. Formula: Cost of DVM’s time/minute x # of minutes spent on procedure
   C. In hospital procedures
   D. Surgery
      1. General/Soft Tissue
      2. Orthopedic

9. Hire a" I 0" pr-actice manager and work< with him or her to understand the objectives of our practice and mal<e sure they are being meet and hopefully exceeded.

Veterinary practice manager

Introduction
The purpose of this position is to provide a variety of analytical, evaluative, advisory, coordinating, supervisory and technical functions in support of the (Practice Name]. Areas of responsibility encompass veterinary practice administration, operational plans, training of staff, ordering and stocking supplies and equipment, funds and resource management, data automation, staffing, safety and security, and review/evaluation/analysis of the past performance of the practice.
Primary job responsibilities

- The fundamental responsibility of the veterinary practice manager is to effectively use all available resources to provide the best possible patient care to animals treated/handled by the hospital, as well as to ensure the profitability of the practice.
- Serves as financial manager. Responsible for developing budget estimates and revisions utilizing knowledge of the functions and requirements of hospital. Make recommendations to practitioners for distribution of funds allocated for medical and non medical equipment and supplies. Advise practitioners on the formulation and development of corrective actions necessary to improve the effectiveness and profitability of the practice.
- Consult with the hospital's accountant. Obtain assistance and advice in preparation and review of various analytical reports, unaudited financial statements (profit and loss), departmental profit and loss statements and tax returns. Draw on expertise in determinations on business projection analysis and review, as well as investment assistance. Review statements for trends, bring to the attention of veterinarians any apparent financial problems, and recommend adjustments and corrections. As required, draw on the accountant for assistance in the development of an appropriate accounting system or for advice on the need for updating the existing system.
- Establish inventory control system. Assure adequate stocks of supplies are available. Negotiate best prices for all products purchased.
- Manage supply activities. Supervise maintenance and proper storage of supplies and equipment. Analyze and monitor the depletion rate of supplies and equipment and provide necessary guidance in the requisition to replenish needed requirements. Responsible for the quality control of supplies and equipment and ensure items are serviceable and readily available for use.
- Rectify financial issues that arise between {Practice Name] and its clients. Use a variety of methods in collection of delinquent accounts. Determine when special financial agreements are appropriate; ensure that agreements made are properly followed.
- Plan and organize all veterinary administration and personnel activities. Directly assist the practitioners in the management of assigned personnel. Select/promote/separate administrative and paraprofessional team members. Formulate job descriptions, set salaries, prepare payroll. Schedule team members in appropriate areas at all times.

10. Enjoy every day, share that joy with my team as well as the success of the practice. Give back to the community and to those we serve, both clients and patients.

Charity account

Protocol

In order to allow the doctors to provide charity services while, at the same time, controlling this expense, it has been decided that a charity account will be establish for each doctor. This account will allow each doctor a pre-determined dollar value to be used in the rendering of medical and surgical services, per their discretion, through the charity account. If a doctor exceeds the amount in his or her charity account, then that doctor would need to "borrow" the funds from another doctor if they wish to render additional charity services.

Establishing a charity account is an effective means of allowing a practice to offer charitable services while still maintaining this expense within acceptable norms.

The Charity Account is fairly easy to set up. Set up a new client account for each doctor in the practice management software. The new client account should be named "Dr. Name – Charity Account". The rest of the information in the account set up is not important. Then enter a credit of $X,000 into each of these doctor accounts. This can be done using account adjustment and the reason would be charity account. A suggested annual amount would be $1000 - $2000 per year per doctor.

Begin printing the fee exception reports daily. Any time a doctor charges less than the stated fee, it will show on the fee exception report. An account adjustment entry is then made in the “Dr. NameCharity Account” for the amount that was "discounted", with the client's name and invoice number noted. You can also transfer a credit from the charity account to a specific client if the doctor requests it. Once the account is depleted, the doctor should discontinue offering charity services unless they wish to pay for those charges out of their own pocket.

NOTE: You cannot "dock" one's pay, but should monitor this behavior so that it does not reoccur.

Important guidelines

1. Charity accounts are set up annually. Each doctor is assigned a $X,000.OO balance.
2. Charity accounts are to be used for professional services ONLY.
3. Do not use more than the amount credited to each doctor on an annual basis.
4. Doctors can approve use of their account by another doctor.
5. The charity account cannot be used for family members.
6. The charity account has to be used for established [Practice Name] clients.
7. The charity account cannot be used for any service for which the clinic will be invoiced.
8. This includes send out lab-work, cremation/burial services, etc.
Save Money by Controlling Staffing and Inventory Costs
Mark Opperman, CVPM
Veterinary Management Consultants
Everygreen, CO

I. Inventory Costs
   A. Your inventory costs to gross should be:
      1. Drugs and Medical Supplies 10.1% (includes radiology, hospital supplies, but excludes food)
      2. Heartworm and Flea/Tick Products 4.5%
      3. Diets 3.1% (therapeutic and retail)
      4. Over The Counter Retail .4% (toys, collars, shampoo, etc)
      Total Inventory as a percentage of Total Revenue = 15-18%
   B. Let’s look at some of the reasons why your inventory costs might be out of line
      1. Shelf life
      2. Duplication of product
      3. Factor of mark-up
      4. Factor of total income
      5. Embezzlement

II. Do You Have and Effective Inventory Control System in Place
   A. Can you control all your inventory with your computer software program?
   B. Items that go through the receiption process can be controlled by your computer system, items that don’t go through your
      receiption process must be controlled by an alternate system
   C. Let’s discuss both inventory control systems
      1. Manual - Red Flag Inventory Control System (see page 135)
      2. Computerized inventory control with your veterinary software program
         a. Establishing effective inventory control with your computer
            1. Review your type categories
            2. Set up a “manual” type category
            3. Review all inventory items in the computer for the following:
               a. Buy-to sell ratio
               b. Mark-up
               c. Auto calculation
               d. Description
               e. Review other information
            4. Perform a physical inventory
            5. Enter new numbers into the computer
            6. Print re-order reports
            7. Use your inventory control system
            8. Do a physical inventory at least once a year

III. Controlling Your Support Staff Costs
   A. Your total support staff costs to gross
      Total Expense for non-doctor staff positions
      (gross W-2 wages, excluding benefits and employee payroll taxes)
      1. Receptionists 4.4%
      2. Credentialed Technicians 4.9%
      3. Veterinary Assistants 5.6%
      4. Ward Assistants 1.2%
      5. Managers 4.0%
      (includes hospital administrator, practice manager, team leader, book keeper)
      6. Groomers 0.4%
      7. Other not identified 0.2%
      8. Payroll taxes 2.3%
      9. Retirement, profit sharing contributions 0.7%
      Total Support Staff Costs as a percentage of Total Revenue = 23-24%
   B. Breakdown of support staff costs to gross
   C. Associate doctors should run around 14% of gross income, but be paid on a percentage of their individual production.
      1. ProSal
   D. What are the causative reasons why support staff costs to gross might be too high in a veterinary practice?
      1. Scheduling
2. Overtime
3. Poor utilization and training
4. Factor of income
5. Embezzlement

E. What can you do to bring your inventory and support staff costs into line in your practice?
How to be Happy in Veterinary Medicine
(Parts 1 and 2)
Kimberly Pope-Robinson, DVM, CCFP
1 Life Connected Consulting
San Clemente, CA

1. Feelings are not to be judged, or ignored. I believe that feelings are our passion connecting with our physical self and becoming numb and/or cynical does not lead to a content or sustainable career in veterinary medicine.

2. Wellbeing overview – Learn to recognize the water line, sinkers, and balloons.

3. Water line is the “fear of failure” or our own personal shame. “Shame is the intensely painful feeling or experience of believe we are flawed and therefore unworthy of acceptance and belonging!” Brene Brown

4. Know the difference between guilt and shame - shame is who we are, guilt is our behaviors. I find we tend to jump to shame versus guilt as a profession. “Recognizing we made a mistake is far different than believing we are a mistake.” Brene Brown.

5. Understand and learn your “why”, this is the core to shame resilience. Find how to remember your “why” (filling your balloons) when things become challenging.

6. When you feel negative (sinking under the water line) – write down 3 positive things that speak to your “why”.

7. Define perceived threats (Red and Blue trains) – Book reference “Riding the Blue Train”. Learn to recognize which train you are on and accepting that it is okay to have brown gauze moments.

8. We come into the industry all on blue trains, however industry dynamics promote red train stations; long hours, physically and mentally demanding, deal with the public in emotional situations, having no options for pets (not just money concerns), and have no insurance buffer for the financial conversations.

9. It is not only Compassion Fatigue that is driving people out of vet med. Defined how Engagement, Perfectionism, and Compassion Fatigue show up in the profession.

10. Subway story, think of one justifiable reason for others actions will help to find peace in forgiveness so as to not take things personally.

11. Empathy is the antidote to shame, 4 attributes to empathy.
   a. To be able to see the world as others see it.
   b. To be non judgmental.
   c. To understand another person’s feelings.
   d. To communicate your understanding of that person’s feelings.

12. To have empathy for others, we must be able to be empathetic to ourselves.

13. “Self Forgiveness is the foundation to a sustainable career in the veterinary industry.”

14. 4 key summary points;
   a. You control your response
   b. You create your environment
   c. Work through emotions
   d. Find self forgiveness.
How to Manage Emotions in Tough Conversations with Clients

Kimberly Pope-Robinson, DVM, CCFP
1 Life Connected Consulting
San Clemente, CA

1. Feeling like you are trapped between a rock and a hard place with these discussions is not an isolated feeling.
2. Medically trained, technically trained, and communication training not generally the primary problem. I have found the highest anxiety in having these conversations is related to our emotions.
3. Remembering why we entered the profession, it can sink us, but it also floats us.
4. Money is not our driver, however profit is slim.
5. Provide a definition of high-level view of “compassion fatigue” in the profession.
6. Top personality traits I have observed to those entering the profession; analytical, compassionate, people pleasers, Type A, Introverts, workaholics, and perfectionism (high achievers). These are great traits to get into and through vet school, not so great for a sustainable career.
7. Remembering the value that you bring can be difficult and often we fall into traps early on in the career to avoid these conversations.
8. The traps;
   a. I’m new and learning
   b. I own that window
   c. Discounting prejudice
   d. Make a client for life
   e. Skipping recommendations.
10. Then we have those moments of clarity, cases we will never forget. No matter how much we share with a client, it will not remove all legality concerns.
11. 13) The answer is in partnership; set expectations. Preparing for sticker shock is not leading the conversation with money.
12. Reflective listening and providing structure/direction are critical as often dealing with emotional situation.
13. A reflective statement does not mean that you agree with the individual. Being heard can be extremely powerful in letting people move forward.
14. If the client can’t afford having a next step in mind, remember that there is a right and wrong way to the multiple estimate approach.
15. How to not take it personal – remember this is a partnership.
   a. What we know-
      i. Medical needs
      ii. Financial requirements
      iii. General prognosis
      iv. Emotional expectations
      v. Home care needs.
   b. What they know-
      i. Financial capabilities
      ii. Attachment to pet
      iii. Emotional capabilities
      iv. Home care follow through abilities.
16. The right path is not always because of financial or medical drivers, how to learn to accept all outcomes. Learning to let it go is key!
17. “Self forgiveness is the foundation to a sustainable career in veterinary medicine.”
18. 4 key summary points;
   a. You control your response
   b. You create your environment
   c. Work through emotions
   d. Find self forgiveness.
Better Alternatives to Across-the-Board Fee Changes

Denise Tumblin, CPA
Wutchiett Tumblin and Associates
Columbus, OH

With the rising popularity of dollar stores and thrift shops, it may seem consumers are only concerned about price. Is that also the case for pet owners coming through the doors in America’s veterinary practices? A new study tried to find out.

To develop The Nationwide and Purdue University Veterinary Price Index, economists at the Krannert School of Management set out to better understand the pricing of veterinary services from 2009 to 2013. They analyzed millions of veterinary transactions drawing from pet-health insurance claims provided by Nationwide. The analysis revealed that veterinary fees are rising at a slower rate than consumer prices overall and that fees have shown a slight decrease over the study period.

These results paint a gloomy picture for pricing power in the profession. Yet some practices — perhaps yours — have increased fees. A comparison from Benchmarks 2013 and 2015 reveals price increases of varying degrees.

<table>
<thead>
<tr>
<th>Service</th>
<th>2015 Fee</th>
<th>2013 Fee</th>
<th>% Increase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete physical exam</td>
<td>$52</td>
<td>$50</td>
<td>4.0%</td>
</tr>
<tr>
<td>Senior exam</td>
<td>$53</td>
<td>$52</td>
<td>1.8%</td>
</tr>
<tr>
<td>Medical progress exam</td>
<td>$42</td>
<td>$40</td>
<td>4.8%</td>
</tr>
<tr>
<td>CBC with chemistry panel</td>
<td>$136</td>
<td>$122</td>
<td>10.3%</td>
</tr>
<tr>
<td>Canine heartworm antigen test</td>
<td>$39</td>
<td>$37</td>
<td>5.4%</td>
</tr>
<tr>
<td>Urinalysis</td>
<td>$46</td>
<td>$44</td>
<td>4.5%</td>
</tr>
<tr>
<td>4 cm skin tumor removal</td>
<td>$155</td>
<td>$149</td>
<td>4.0%</td>
</tr>
<tr>
<td>Anterior cruciate repair</td>
<td>$726</td>
<td>$630</td>
<td>15.2%</td>
</tr>
<tr>
<td>Six radiographs, digital</td>
<td>$87</td>
<td>$85</td>
<td>2.4%</td>
</tr>
</tbody>
</table>

How are some high-flying practices increasing fees in a time of consumer price sensitivity? They deliver the value that justifies higher prices. If this sounds like old news, ask yourself, “Am I really doing everything I can to improve my client’s understanding of the level of service I offer?” It isn’t enough to communicate it in the exam room. Clear, consistent value messages need to be communicated by staff, by doctors, by your website, your practice’s appearance and your involvement in the community. The value you offer needs to be reflected in everything you do.

Understand the cost of providing a service and a product. Knowing the true cost of something helps with pricing decisions. Products are easy, right? It’s the cost of the inventory item. But wait - don’t forget about labor to prepare and place the order, to unpack and check the shipment and stock the hospital shelves, as well as the overall cost of housing the inventory. Some sources put the true cost of an inventory item as high as the invoice amount plus 40%.

Calculating the cost of services presents an even greater challenge. You must factor in labor, the cost of all necessary materials and supplies, facility and equipment costs, and administrative overhead. This exercise might seem like a lot of work for little benefit, yet it provides a huge “reality check” for you and your team on the expense side of the pricing equation. Do your fees truly cover your costs, or are you in the red? Traditional thinking related to setting fees looks like this:

Cost + Profit = Price

A fee increase is the easy solution to offset rising costs and maintain a reasonable profit. Yet this strategy might not be feasible if your community is economically-challenged or your fees are the highest for the area. Business in the new normal requires a different pricing approach. A new, healthier model for pricing might be a LEAN strategy. Start with the price the clients will accept and manage your costs to maintain the desired profit margin:

Price = Cost

Minnetonka Animal Hospital in Minnetonka, MN has used LEAN strategies for years to minimize waste. Owner, Dr. Kaaren Howe, is eco-conscious and passionate about protecting finite resources. “By making LEAN changes, we have eliminated waste and are less stressed, more efficient, and more productive. An added bonus - our inventory and staff costs are 4% lower!” Follow Dr. Howe’s lead and reduce waste to manage costs as part of your pricing strategy.

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Make strategic fee changes. Instead of annual, across-the-board increases, review quarterly and determine which fees to increase, which fees to leave alone, and in some rare cases, which fees you’ll reduce. Monitor the quality of your patient care and client service to ensure both continue to reflect your fee structure (and vice versa). Teach and mentor your staff to boost everyone’s comfort and confidence with talking about money and communicating your value.

- **Price shopped products and services.** A market-based approach is best for price-sensitive services such as vaccinations and elective surgeries. Conduct a Community Survey to gather market data. How do you stack up compared to others? The range of fees in the market place and your target client demographic will guide your pricing decisions. If your goal is to offer the most affordable price in your community, then set your fees at the low end. If you’re targeting a demographic that will pay more for demonstrably better service, then set your fees at the mid-to-high end. You can have the highest fee in the community as long as your quality and reputation supports your pricing. Consider the level of medicine and customer service you offer compared to others. Contrast your number of doctors, practice hours, and the quality of your facility. Do clients believe the value you deliver is greater than the price you charge?

- **Price inventory items based on cost.** The median markup on medication dispensed through the in-house pharmacy is 150%, and through the on-line pharmacy it’s 100%. The median markup on heartworm, flea and tick items is 95% in-house and 80% on-line. Markups on drugs for chronic conditions are 100% in-house and 85% on-line. Apply the markup to the cost, including shipping and sales tax, and add a handling/dispensing fee of $6 to $12. Example:

<table>
<thead>
<tr>
<th>Pharmacy Type</th>
<th>Markup</th>
<th>Handling Fee</th>
<th>Final Price</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-house pharmacy</td>
<td>150%</td>
<td>$10</td>
<td>$35</td>
</tr>
<tr>
<td>Online pharmacy</td>
<td>100%</td>
<td>$10</td>
<td>$20</td>
</tr>
</tbody>
</table>

If you make bulk purchases to get a better price, keep the savings versus passing it on to the client.

- **Employ variable pricing in special situations.** There may be times when you want to influence client behavior, reward a client or patient demographic, or increase business during certain times of the day or days of the week with modified pricing. Examples of variable pricing include a discount on dentistry during National Pet Dental Health Month, a discount for senior citizens, Preventive Wellness Plans at a lesser cost than the ala carte price, concessions for humane or rescue organizations, and price concessions during a block of time that is traditionally slow in the practice. Variable pricing can be an effective tool, but carefully consider all the factors and keep your discount under 15%.

- **Price non-shopped services based on value.** A client can see the tangible benefits of your services in a happy, healthy pet. Continue to look for opportunities to influence clients’ perception of value. What’s important to them and keeps them coming back? What’s a turn-off? If you don’t know, ask them. Does your practice facility and level of client service match your price? People think and hear with their eyes, so make sure your image and value match. Consumers want reliable, predictable, familiar service. Be consistent, so clients know what to expect every time.

- **Reduce a fee if there’s no other solution.** For any service or product you hesitate to offer (or don’t charge for) because you think it’s over-priced, consider lowering the fee. Take this approach only after you’ve exhausted all other efforts to change the practice’s internal perception of the value of this item. Talk with your staff to explore why the item is priced at this level, and calculate the true cost of providing it. If you still believe the item is over-priced, then lower it. Reducing the fee might help make your team more comfortable offering the service every time it’s appropriate. Plus, charging something is better than nothing.

Get your team on the value bus. If the doctors and staff don’t see the value of your services, it’s time for some refresher CE. The entire team must be knowledgeable about your medical protocols and what’s involved in providing patient care. Receptionists should observe outpatient appointments, dental procedures, and surgical procedures, so they truly understand the quality of care provided start to finish. Every technician and assistant should spend time at the front desk to gain a better appreciation of all that’s involved in delivering stellar customer service. Calculate and share the true cost of your most-common services and products. Ask your highly-skilled communicators to teach less-confident co-workers how to be a more effective patient advocates. For team members who still fail to see the value of your medical care in spite of your robust education and mentorship program, free up their future for something else and get them off your bus.

Offer a variety of payment options. Start with cash, check, or credit card. Then consider other options that might be a good fit in your practice. Promote pet insurance for future medical needs. Offer third-party options like CareCredit. Consider pre-arranged auto-deductions from clients’ bank account. Revisit Preventive Care Plans that offer budget-friendly payments. Seek staff input about the payment options clients ask for. Based on your staff’s feedback, ask your practice manager to research the information needed to make a decision about whether that payment option will work for your hospital.

Use these recommendations and the worksheet that follows to analyze your pricing and determine if you’re on track or have opportunities to improve.
Comparing your value-based fees

Compare your value-based fees with Well-Managed Practices® using this worksheet. Enter your exam fee in the spaces provided and apply the following factors to determine your new fee. Source: Benchmarks 2015 – A Study of Well-Managed Practices® by Wutchiett Tumblin and Associates and Veterinary Economics.

<table>
<thead>
<tr>
<th>Source</th>
<th>Professional Services</th>
<th>Diagnostic Services</th>
<th>Diagnostic Imaging</th>
<th>Lab Work</th>
<th>Nonelective Procedures</th>
<th>Anesthesia (incl. intubation)</th>
<th>Hospitalization – LEVEL II (with IV)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Percent of exam fee</td>
<td>Your exam fee</td>
<td>New fee</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Type of Service</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Complete physical exam ($50; 20 minutes)</td>
<td>79.8%</td>
<td>$</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medical progress exam</td>
<td>79.8%</td>
<td>$x 0.798</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Extended exam</td>
<td>125.7%</td>
<td>$x 1.257</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hospitalized patient exam</td>
<td>71.8%</td>
<td>$x 0.718</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Blood pressure evaluation</td>
<td>71.8%</td>
<td>$x 0.718</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>ECG; 4 lead in-house</td>
<td>138.0%</td>
<td>$x 1.380</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tonometry</td>
<td>72.0%</td>
<td>$x 0.720</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abdominal ultrasound</td>
<td>478.8%</td>
<td>$x 4.788</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Digital Radiograph (two views, 8x10)</td>
<td>288.3%</td>
<td>$x 2.883</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Digital Radiograph add’l (one view, 8x10)</td>
<td>115.7%</td>
<td>$x 1.157</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>BUN</td>
<td>59.8%</td>
<td>$x 0.598</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CBC with chemistry panel</td>
<td>239.4%</td>
<td>$x 2.394</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cytology (fine needle aspirate)</td>
<td>109.7%</td>
<td>$x 1.097</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Feline leukemia, FIV, heartworm antibody test</td>
<td>116.7%</td>
<td>$x 1.167</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Giardia ELISA</td>
<td>107.7%</td>
<td>$x 1.077</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Canine Heartworm 3DX test</td>
<td>73.8%</td>
<td>$x 0.738</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anesthetic monitoring (electronic)</td>
<td>55.9%</td>
<td>$x 0.559</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gas anesthetic, sevoflurane</td>
<td>209.5%</td>
<td>$x 2.095</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(30 min, 60-lb canine)</td>
<td>207.5%</td>
<td>$x 2.075</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gas anesthetic, isoflurane</td>
<td>(30 min, 60-lb canine)</td>
<td>142.6%</td>
<td>$x 1.426</td>
<td>$</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Overnight, excl. exam &amp; inpatient services</td>
<td>149.6%</td>
<td>$x 1.496</td>
<td>$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
How Associates Earn More by Producing More Without Selling their Souls
Denise Tumblin, CPA
Wutchiett Tumblin and Associates
Columbus, OH

Revenue production comes into play whether a doctor’s compensation is tied to individual production, or he or she receives a fixed salary. Yet maintaining a “patient first” focus is imperative for continued success.

Patients drive the medicine in your practice, and the medicine drives the business. Yet, paying attention to the business is crucial to your ability to continue to elevate patient care, invest in new technology, and invest in the doctors and staff. A practice-wide focus on enhancing patient-care, brainstorming ideas for growing services, and sharing information about the costs of running the practice helps associates better understand the patient-medicine-business connection.

Mine your data
On your journey to enhance patient care and grow revenue, make your practice management software your very first stop. A wealth of information lies within, just waiting to help you shine a spotlight on opportunities to jump-start growth. You’ve likely heard various versions of Peter Drucker’s credo, “What gets measured gets managed,” or Robin Sharma’s version, “What gets measured gets improved.” So, measuring is your first step. Your second step is a plan of action to accomplish your goals.

Determine what to measure
Perhaps your goal is to increase the number of patients who are in harmony with your medical protocols. The practice sets the following group goals:

- Goal: Increase the number of thyroid panels by 25 percent
- Goal: Increase the number of heartworm tests by 25 percent
- Goal: Increase the number of fecal tests by 35 percent
- Goal: Increase the number of feline wellness examinations by 35 percent

Run a report from your practice management software for the number of the above items completed last year.

Brainstorm ideas for how to hit your targets
Is everyone confident discussing the patient benefits of these services? Ask your most effective communicators to share communication tips with less-confident co-workers, and then practice. Do your client educational materials need an overhaul? Ask for volunteers to update your handouts to better communicate the message. Are you using visual aids to help communicate the importance of these services? We think and hear in pictures, so visual aids during appointments can help clients see the light.

Post results where the team can see and report your progress
Visuals work for team members too. Graphs are a quick and easy way to see results and patterns in the results. Learn from your successes and your failures. If you’re not hitting your targets, what do you need to do differently? Fine-tune your approach to ensure success. If you’re exceeding your targets, celebrate your success! Then, aim higher.

Sustain the gains
John E. Jones said, “What gets measured gets done. What gets measured and fed back gets done well. What gets rewarded gets repeated.” Reward your team for accomplishing their goals. Have a pizza and salad party. Bring an ice cream truck to the practice for a fun staff break. Go rollerblading, hiking, bowling or whatever your staff likes to do for fun. Once you’ve hit your targets in these areas, choose three or four new areas to spotlight.

Additional patient-care ideas
- Revitalize your team’s approach to dentistry. Make sure every staff member observes a dentistry procedure start to finish, so they can speak truthfully and passionately about the value of those procedures with clients. Be mindful of the words you use to describe periodontal services to clients. Boarded dentist Dr. Cindy Charlier, frequently reminds practices to “Consider not the cost to the client but the cost to the patient if the client doesn’t accept your recommendation.”
- Take a fresh look at preventive-care plans. Implementing wellness or preventive-care plans might be sitting on your “to-do” list. Or perhaps you decided the considerable time and effort required to implement a program wasn’t worth it. Regardless of the reason you’re not offering them, it’s time to revisit this option. New data from the American Animal
Hospital Association and IDEXX reported that 8 out of 10 pet owners indicated an interest in preventive-care payment plans. And their interest level wasn’t determined by income.

- Rejuvenate your nutrition mojo. The latest statistics from the Association for Pet Obesity Prevention are that 54 percent of dogs and cats in the US. Are overweight or obese and that 90 percent or more of pet owners don’t recognize that their pets are overweight. As the pet advocate, you have a special opportunity to provide nutritional education to clients that’s essential to rewrite those statistics.

- Spark growth with a new service. Adding new services to your repertoire creases fresh energy and enthusiasm for the entire team. New services are another opportunity to differentiate your practice (and for a doctor to differentiate him or herself), expand the offerings for existing patients and attract new clients.

**Let’s talk compensation and benefits**

The majority of practices now pay their doctors some form of incentive-based compensation. About 26% pay purely based on production. Another 50% pay doctors a guaranteed base plus a percentage of production over a required minimum. The remaining 24% pay their doctors a fixed salary.

Practices who use a blended rate – i.e., one percentage applies to all medical service and product production – typically pay their doctors between 16% and 21%. Where they fall in the range is dependent upon the practice’s staff-to-doctor ratio. The more staff the practice provides to assist the doctors, the lower the percentage paid to the doctors. The additional staff members allow the doctors to produce at a higher level, which increases doctor compensation. The practice also has an added layer of overhead because of the additional staff members, which the doctors must help support.

Practices who use a split-rate – that is, one percentage applies to medical service production, and a different percentage applies to medical product production – typically pay their doctors between 22% and 26% for services, and 4% and 8% for products. The service/product split – that is, how much of medical revenue comes from services and how much from product sales - and the staff-to-doctor ratio will both impact where you end up in the stated ranges.

To make any percentage-based compensation system work, every team member must understand what is and isn’t credited to the doctor’s individual production. Doctors receive credit for all medical service revenue provided during an outpatient appointment, in-hospital treatment, or dental and surgical procedures. Doctors also receive credit for medications and therapeutic foods dispensed during an outpatient appointment, during in-hospital treatment, or at the end of a patient’s hospital stay.

Prescription refills and additional food or product purchases that don’t involve a doctor are credited to a hospital provider. The doctor receives credit for the refill only if it requires his or her time to review the record, assess if the medication or dosage needs to change, and give direction to the staff member who will fill the prescription. Doctors never receive credit for boarding, grooming, or retail purchases.

When multiple doctors collaborate to treat a patient, the doctor who provides each point of care receives credit. For example, if Doctor A examines and admits a patient to the hospital on Day 1, and Doctor B provides or supervises the hospital treatment on Day 2, Doctor A gets credit for everything on Day 1, and Doctor B gets credit for Day 2.

Doctors who lead with what is in the best interest of the patient can earn more. Maintain a patient-first mentality that always, always remembers it’s about the medicine, not the money. With a patient-first approach, the money follows the medicine.

**Figure 1 – Starting salaries for employed veterinarians**

<table>
<thead>
<tr>
<th>Years of Experience</th>
<th>Median</th>
<th>75th Percentile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 1 year</td>
<td>$70,000</td>
<td>$70,000</td>
</tr>
<tr>
<td>1 to 2.9 years</td>
<td>$73,500</td>
<td>$75,000</td>
</tr>
<tr>
<td>3.0 to 5.9 years</td>
<td>$76,800</td>
<td>$80,000</td>
</tr>
<tr>
<td>6.0 to 10.9 years</td>
<td>$85,000</td>
<td>$85,000</td>
</tr>
<tr>
<td>11.0 to 15.9 years</td>
<td>$85,000</td>
<td>$87,500</td>
</tr>
<tr>
<td>16.0 to 19.9 years</td>
<td>$87,000</td>
<td>$89,800</td>
</tr>
<tr>
<td>20+ years</td>
<td>$87,500</td>
<td>$90,000</td>
</tr>
</tbody>
</table>

**Figure 2 – Benefits in well-managed practices®**

<table>
<thead>
<tr>
<th>Benefit</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retirement</td>
<td>Simple IRA - 3% match</td>
</tr>
<tr>
<td>Health Insurance</td>
<td>65% of premium OR $258/month</td>
</tr>
<tr>
<td>Continuing education</td>
<td>$2,000 to $2,600</td>
</tr>
<tr>
<td>Paid time off</td>
<td></td>
</tr>
<tr>
<td>0 – 3.9 years</td>
<td>10 days</td>
</tr>
<tr>
<td>4 – 9.9 years</td>
<td>14 days</td>
</tr>
<tr>
<td>10+ years</td>
<td>17 days</td>
</tr>
</tbody>
</table>

How to Get 16% ROI on Your Practice
Denise Tumblin, CPA
Wutchiett Tumblin and Associates
Columbus, OH

As a practice owner, you’re well-versed in the risks and rewards of owning a business. You may even have days when it feels like the risks and hassles outweigh the rewards by a large margin. Owners of Well-Managed Practices tell us their biggest challenges are maintaining efficiency, productivity and profitability. If you’re nodding your head in agreement, read on.

It’s a fact: Downward pressure on profit continues. Well-Managed Practices now have an average owner return (ROI) of 12 percent. That’s what’s left for the owner after all operating expenses are paid, including variable expenses, fixed expenses, staff compensation, facility costs, associate and owner doctor compensation and owner management compensation.

That’s low. Declines in profitability impact your ability to pay the bills, offer competitive wages and benefits, buy new equipment and protect the investment value of your veterinary practice. It’s time to improve profit. Aim for an ROI 2 to 4 percent higher than last year. Aim for a 16 to 20 percent ROI within the next three to four years. See Figure 1 – How to Get to 16% ROI.

Seem impossible? Suspend your disbelief just for a moment and stick with me. Instead of, “We can’t do . . .” or “That will never work because . . .”, open your mind and explore the possibility of, “What if we could do . . .?” or “How can we make a change in . . .?” A profitability turn-around takes planning, focused attention and changes to your processes to boost efficiency and productivity. Some of the required changes may be painful. You’ll possibly encounter resistance along the way. Persevere and encourage your staff to hang in there because the results will benefit everyone.

Watching what you spend may come naturally in your practice. You work with a practice budget, compare your numbers to the WellMP benchmarks, and adjust your spending when necessary. If so, kudos to you and your staff! But if you’re not quite where you’d like to be when it comes to taking charge of your expenses, now’s the time to put your expenses on a diet.

Rather than adopting the “starvation” approach to accumulate the extra cash, start with these Five Easy Slim Downs and these benchmarks to help you pinpoint where your spending is a little heavy. Then get started with your practice slim down to save that extra $10,000 to $20,000 and boost your ROI to 16% or above.

1. Pare down your drug inventory. If your shelves are looking a bit bloated, it’s time to eliminate the excess. Veterinarians have many wonderful drugs to choose from to treat patients. But carrying every wonderful medication that’s available ties up a lot of cash and creates confusion for the staff and for clients. Doctors – unite! Create a list of the medications that you believe in the most. Conduct a scientific comparison of the duplicate products you have on your shelf. Consider the pros and cons, safety, and efficacy of each. Make your case scientifically and medically and come to a consensus among the doctors about what’s your best and second choice. Then eliminate any other redundant items from your shelves.
   a. Tip: Stock $10,000 to $16,000 of drugs and medical supplies per full-time equivalent doctor, or about one month’s supply. This includes heartworm, flea and tick products and excludes diets.
   b. Tip: Spend 8% to 9% of revenue on drugs and medical supplies. Spend 4% to 6% of revenue on heartworm, flea and tick products.
   c. Tip: Move infrequently-used medications to your on-line store.

2. Evaluate your labor cost. What one or more things could you do differently to increase efficiency and productivity in your hospital? It’s not unusual for different practices to have the same level of staff support, but significantly different levels of doctor production. I’m currently working with two practices, each with a 4 to 1 staff-to-doctor ratio; one generates about $440,000 of medical revenue per FTE doctor and the other generates $670,000 per FTE doctor. What accounts for the $230,000 difference? Explore the following opportunities to rev up your practice’s productivity.
   a. Do more with less. Bump your pay scale to attract more skilled and efficient employees. We’ve all experienced the employee who seems to get twice as much done in half the time as two other employees combined. You might find that an employee who merits $18 an hour could easily complete the work of two, less productive $12 an hour employees. The result: an annual savings of $10,000 to $12,000 depending on the benefit package.
   b. Streamline your processes. It’s easy to get into the routine of “that’s the way we’ve always done it.” Take a fresh look at your protocols – are you doing things the easiest, most efficient way, or could you streamline the process? Are staff members duplicating efforts? Eliminate the redundancies. Are you taking extra time to track information that no one is using? Then stop.
   c. Tip: Hold a contest for your staff. Ask each staff member to submit one or two ideas to improve efficiency throughout the hospital (reception, exam rooms, treatment, surgery, boarding, etc.). Give awards for the top four ideas (first, second, and third place, and honorable mention). Be sure your awards are meaningful and
compelling. For example, first prize gets a paid day off; second prize gets a gift certificate for a local spa; third prize a gift certificate for a favorite local restaurant; and honorable mention gets tickets to the movie of their choice. Or, you could let the winners choose which award they would like out of your offerings.

d. Get organized. Clutter and untidy work stations add to the chaos of busy days. Spend a day eliminating the mess. Move frequently used items to more accessible parts of the hospital to eliminate wasted steps. Move rarely used items to storage. Get rid of items in storage that you haven’t used for a year or more. Adopt the creed: reduce, reuse, recycle. The hospital will look better, and the doctors and staff will feel better and be more productive!

e. Convert under-utilized space to a medical purpose. Some hospitals have idle or under-used space that’s begging for use as a medical area. For example, convert a food storage space to another exam room. Convert an under-utilized retail space to a patient discharge room. Convert an under-utilized storage space adjacent to treatment to a dental suite or a procedures room.

f. Tip: Hold a contest for your staff to solicit their ideas about under-utilized areas of the hospital that could be converted to medical use. Give awards for the top ideas (see suggested prizes above).

3. Bump up your use of technology. Update and/or replace hardware to reduce wasted time waiting for the computer to process or recovering from a crash because the system can’t handle the hospital’s current needs. Update your software to the latest version. Replace your software if the company hasn’t provided updates for years or their support is poor. Convert to electronic medical records to eliminate wasted time searching for lost or misplaced records. Technology saves time and reduces frustration when used well.

a. Tip: Hire a trainer from your practice management software company to spend a day with your staff teaching them more about your software’s capability. Staff members know the basics. But they may not be aware of all the shortcuts that help streamline their work, or the options that help enhance client service and patient care. The return you’ll receive will be much greater than the cost of the training. Example: One veterinary practice estimated that the knowledge they gained from the training saved three staff members an hour a day, which amounted to an annual labor savings of about $15,000.

4. Revisit your administrative costs. It’s easy for fixed overhead spending to creep up without realizing it. Don’t let the word “fixed” change your mind about giving these expenses another look.

a. Use e-mail for reminders, newsletters, educational materials, and other client correspondence instead of the U.S. postal service. Postage adds up and clients may actually prefer to receive information via e-mail.

b. Take stock of your office supplies. Organize your inventory in one central location so everyone knows what you have on hand before requesting and ordering more. Change reorder points to minimize the amount of inventory you have on the shelf before placing a new order.

c. Evaluate employee health insurance. Talk with your insurance agent about health insurance policies with higher deductibles and co-pays. Sometimes the premium savings is greater than the difference in the deductible, so you can offer to pay part or all of the difference in the deductible and still lower the practice’s cost. Ask your agent to research other policies with lower premiums and similar coverage options. Consider having employees cover part of their health care.

d. Assess your Workers’ Compensation Insurance rates. Coverage managed by a private insurance company, if an option in your area, might offer better rates than a fund managed by your state.

e. Conduct an energy audit in your practice. A professional energy audit gives you a clear picture of where your practice is losing energy and what you can do to save money. Possible resources to conduct the audit include your state or local government energy or weatherization office or your electric or gas utility company. Per www.energy.gov, you can save 5% to 30% on your energy bill by making the recommended upgrades. Visit www.greenyour.com for an energy audit checklist.

f. Investigate the possibility of refinancing your debt. If you’ve got any high-rate loans, act now to see what your options are for getting into a more favorable rate.

5. Think twice before investing in equipment. Do the math to determine if the equipment purchases you’re planning will pay for themselves in a reasonable timeframe. Investing in equipment helps you enhance patient care and client service, and grow your practice. But fabulous equipment rarely used, is a poor investment. Take the time to evaluate how often you’ll use the equipment and the revenue potential before taking the plunge.
Figure 1 – How to get to 16% ROI

Gross Revenue 100%
Variable Expenses 23%
Fixed Expenses 8%
Staff Compensation 22%
Facility Expenses 8%
Total operating expenses (61%)

Amount Available for Associates and Owner 39%

Doctor Compensation 20%
Owner Management Compensation 3%
Total Doctor Compensation (23%)

Owner Return on Investment 16%

Reinvestment – Equipment 3%

Remaining Amount Available to Owner 13%

Compare your expenses to these benchmarks
Variable Expenses (as a percentage of total revenue)
Drugs and medical supplies (includes radiology, surgery and hospital supplies but excludes food, shampoos, etc.) 9.9%
Heartworm, flea, and tick products 4.3%
Laboratory 4.1%
Diets (therapeutic and retail) 3.0%
Over-the-counter retail products (e.g. toys, collars, shampoo) 0.4%
Credit card fees 1.6%
Bad debt, collection fees 0.1%
Cremation, care of remains 0.6%
Sales and use tax 0.7%
Medical waste disposal/radiation badge monitoring 0.1%
Practice vehicle expense 0.1%
On-line pharmacy-drug cost 0.1%
On-line store-food cost 0.1%
Total 25.1%
### Fixed expenses (as a percentage of total revenue)

<table>
<thead>
<tr>
<th>Expense</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accounting services</td>
<td>0.3%</td>
</tr>
<tr>
<td>Advertising and promotion</td>
<td>0.8%</td>
</tr>
<tr>
<td>Bank charges (monthly maintenance fees)</td>
<td>0.1%</td>
</tr>
<tr>
<td>Business consulting services</td>
<td>0.4%</td>
</tr>
<tr>
<td>Business gifts and flowers</td>
<td>0.1%</td>
</tr>
<tr>
<td>Business meetings</td>
<td>0.1%</td>
</tr>
<tr>
<td>Charitable contributions</td>
<td>0.1%</td>
</tr>
<tr>
<td>Continuing education, meetings, and travel</td>
<td>0.6%</td>
</tr>
<tr>
<td>Entertainment</td>
<td>0.1%</td>
</tr>
<tr>
<td>Equipment repairs, maintenance, and support contracts</td>
<td>0.5%</td>
</tr>
<tr>
<td>Franchise tax and other taxes</td>
<td>0.1%</td>
</tr>
<tr>
<td>Health insurance</td>
<td>1.8%</td>
</tr>
<tr>
<td>Laundry and uniforms</td>
<td>0.1%</td>
</tr>
<tr>
<td>Legal</td>
<td>0.1%</td>
</tr>
<tr>
<td>Liability insurance</td>
<td>0.1%</td>
</tr>
<tr>
<td>Licenses and permits</td>
<td>0.2%</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>0.2%</td>
</tr>
<tr>
<td>Office and computer supplies</td>
<td>0.9%</td>
</tr>
<tr>
<td>Payroll service costs, retirement plan administration fees</td>
<td>0.2%</td>
</tr>
<tr>
<td>Personal property tax</td>
<td>0.2%</td>
</tr>
<tr>
<td>Postage, freight, and delivery</td>
<td>0.2%</td>
</tr>
<tr>
<td>Printing</td>
<td>0.1%</td>
</tr>
<tr>
<td>Professional dues and subscriptions</td>
<td>0.3%</td>
</tr>
<tr>
<td>Technical (IT) support contracts</td>
<td>0.4%</td>
</tr>
<tr>
<td>Telephone, answering service, internet connection</td>
<td>0.5%</td>
</tr>
<tr>
<td>Workers’ compensation insurance</td>
<td>0.6%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>9.1%</strong></td>
</tr>
</tbody>
</table>

### Non-doctor Staff Compensation (Gross W2 wages as a percentage of total revenue)

<table>
<thead>
<tr>
<th>Expense</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wages</td>
<td>21.6%</td>
</tr>
<tr>
<td>Payroll taxes</td>
<td>2.5%</td>
</tr>
<tr>
<td>Retirement contributions</td>
<td>0.6%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>24.7%</strong></td>
</tr>
</tbody>
</table>

### Facility Expenses (as a percentage of total revenue)

<table>
<thead>
<tr>
<th>Expense</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual rent or mortgage payments (excluding property taxes, insurance &amp; utilities)</td>
<td>5.1%</td>
</tr>
<tr>
<td>Utilities (gas, water, electric)</td>
<td>0.9%</td>
</tr>
<tr>
<td>Janitorial, housekeeping, and garbage</td>
<td>0.4%</td>
</tr>
<tr>
<td>Facility repairs, maintenance, lawn care, and security monitoring</td>
<td>0.8%</td>
</tr>
<tr>
<td>Property insurance</td>
<td>0.3%</td>
</tr>
<tr>
<td>Real estate taxes</td>
<td>0.5%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>8.0%</strong></td>
</tr>
</tbody>
</table>

### Reinvestment

<table>
<thead>
<tr>
<th>Expense</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical equipment</td>
<td>0.8%</td>
</tr>
<tr>
<td>Computer equipment</td>
<td>0.4%</td>
</tr>
<tr>
<td>Facility improvements</td>
<td>0.8%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>2.0%</strong></td>
</tr>
</tbody>
</table>
Productivity and efficiency often top the list of practice owners’ concerns, and all eyes are fixed on revenue growth. We asked the 2015 Benchmarks participants to name one change that would have the most significant positive impact on their practice. The top responses were:

1. Expand or renovate the facility
2. Strengthen the skills of the practice manager and healthcare team
3. Expand services
4. Hire a new associate – for some, an additional doctor; for others, a replacement

Patients drive the medicine in your practice, and the medicine drives the business. Yet, paying attention to the business is crucial to your ability to continue to elevate patient care, invest in new technology, and invest in the doctors and staff. A practice-wide focus on enhancing patient-care, brainstorming ideas for growing services, and sharing information about the costs of running the practice helps the doctors and staff better understand the patient-medicine-business connection.

Mine your data
On your journey to enhance patient care and grow revenue, make your practice management software your very first stop. A wealth of information lies within, just waiting to help you shine a spotlight on opportunities to jump-start growth. You’ve likely heard various versions of Peter Drucker’s credo, “What gets measured gets managed,” or Robin Sharma’s version, “What gets measured gets improved.” So, measuring is your first step. Your second step is a plan of action to accomplish your goals.

Determine what to measure
Perhaps your goal is to increase the number of patients who are in harmony with your medical protocols. The practice sets the following group goals:

- Goal: Increase the number of thyroid panels by 25 percent
- Goal: Increase the number of heartworm tests by 25 percent
- Goal: Increase the number of fecal tests by 35 percent
- Goal: Increase the number of feline wellness examinations by 35 percent

Run a report from your practice management software for the number of the above items completed last year.

Brainstorm ideas for how to hit your targets
Is everyone confident discussing the patient benefits of these services? Ask your most effective communicators to share communication tips with less-confident co-workers, and then practice. Do your client educational materials need an overhaul? Ask for volunteers to update your handouts to better communicate the message. Are you using visual aids to help communicate the importance of these services? We think and hear in pictures, so visual aids during appointments can help clients see the light.

Post results where the team can see and report your progress
Visuals work for team members too. Graphs are a quick and easy way to see results and patterns in the results. Learn from your successes and your failures. If you’re not hitting your targets, what do you need to do differently? Fine-tune your approach to ensure success. If you’re exceeding your targets, celebrate your success! Then, aim higher

Sustain the gains
John E. Jones said, “What gets measured gets done. What gets measured and fed back gets done well. What gets rewarded gets repeated.” Reward your team for accomplishing their goals. Have a pizza and salad party. Bring an ice cream truck to the practice for a fun staff break. Go roller blading, hiking, bowling or whatever your staff likes to do for fun. Once you’ve hit your targets in these areas, choose three or four new areas to spotlight.

Re-energize your practice’s compliance efforts
If your team has run out of steam on compliance, perhaps it’s time to change your vocabulary. Could “client compliance” become “patients receiving the healthcare necessary to ensure a long, healthy life in harmony with your medical protocols”? Yes, it’s long-winded compared to compliance, yet much more patient-centric and may resonate more with your team.

In the book If Disney Ran Your Hospital, author Fred Lee describes four levels of motivation. The first is compliance, and it’s defined as “doing what someone makes me do,” which isn’t very motivating. The author suggests we instead need to move through
the other levels of motivation – willpower (doing what I believe I should do), imagination (doing what I want to because I feel like it) and habit (doing what comes naturally) – to get to real nurturing, uplifting motivation. So let’s change what we call “compliance” to move clients (and the practice team) to the “habit” level of motivation.

**Additional patient-care ideas**

- Revitalize your team’s approach to dentistry. Make sure every staff member observes a dentistry procedure start to finish, so they can speak truthfully and passionately about the value of those procedures with clients. Be mindful of the words you use to describe periodontal services to clients. Boarded dentist Dr. Cindy Charlier, frequently reminds practices to “Consider not the cost to the client but the cost to the patient if the client doesn’t accept your recommendation.”
- Take a fresh look at preventive-care plans. Implementing wellness or preventive-care plans might be sitting on your “to-do” list. Or perhaps you decided the considerable time and effort required to implement a program wasn’t worth it. Regardless of the reason you’re not offering them, it’s time to revisit this option. New data from the American Animal Hospital Association and IDEXX reported that 8 out of 10 pet owners indicated an interest in preventive-care payment plans. And their interest level wasn’t determined by income.
- Rejuvenate your nutrition mojo. The latest statistics from the Association for Pet Obesity Prevention are that 54 percent of dogs and cats in the US. Are overweight or obese and that 90 percent or more of pet owners don’t recognize that their pets are overweight. As the pet advocate, you have a special opportunity to provide nutritional education to clients that’s essential to rewrite those statistics.
- Spark growth with a new service. Adding new services to your repertoire creases fresh energy and enthusiasm for the entire team. New services are another opportunity to differentiate your practice (and for a doctor to differentiate him or herself), expand the offerings for existing patients and attract new clients. The past two years, rehabilitative therapy topped the list for Well-Managed Practice participants, followed closely by laser therapy. Behavior counseling made the list of future additions for the next two years. So take a look at your own “services to add” list – and finally add one!

**Revenue metrics – How the puzzle pieces fit**

The key to revenue growth is to understand all the pieces and identify the opportunities for growth. Shown below are the 12 critical components of revenue and comparisons from Well-Managed Practices®. Measure your practice’s results against these benchmarks to identify your opportunities for growth.
Revenue

Your WellMP SM Practice
All revenue $648,200/Doctor $____
Medical revenue $582,000/Doctor $____
Other revenue $66,200/Doctor $____

Average doctor transactions (ADT) Transactions

WellMP SM Practice
ADT $184 $___
Exam $52 $___
Other $67 $___

All transactions 5,300/Dr. ____
Medical transactions 2,900/Dr. ____
Other transactions 2,400 Dr. ____

Fees Services
The overall fee structure and the service/product mix are the two main determinants of a hospital’s ADT.

Active Clients Visitation
Your WellMP SM Practice
Your WellMP SM Practice
931/Dr. ____
Medical 3.1/yr. ____
Other 2.6/yr. ____

Accounts Receivable New Clients Retention
Your WellMP SM Practice
Your WellMP SM Practice
1.5% ____
Stable 18/Mth/Dr. ____
4.4 Yrs. ____
Transitional 35/Mth/Dr. ____
2.4 Yrs. ____

Awareness Visits Scheduled Age of Active Patients
How well known is your practice in the community?
Your WellMP SM Practice
70% ____
< 1 yr 9% ____
1.1 - 3 yrs 20% ____
3.1 - 6 yrs 23% ____
6.1 – 9 yrs 22% ____
> 6 yrs. 26% ____
The “LEAN” System:  
Be More Efficient Without Cutting Corners  
Denise Tumblin, CPA  
Wutchiett Tumblin and Associates  
Columbus, OH

Products made in Japan 50 years ago evoked images of mass-produced, low-quality goods. Today Japanese brand names such as Toyota, Lexus, and Honda signify quality and durability. How were the Japanese automakers able to improve quality and reduce cost? Did they just get lucky? No, they got Lean!

While Lean started in manufacturing, Lean processes translate to service industries too. There are tremendous opportunities for the use of Lean in healthcare. A study of human healthcare facilities concluded that the facilities spent 75% of their time on non-patient tasks related to communicating, coordinating, and documenting care. In addition, a 2003 report by the New England Journal of Medicine reported a 45% defect rate in human healthcare. The overall goal of applying Lean strategies in healthcare is to initiate a process of continuous improvement to improve patient outcomes while lowering costs.

Several human healthcare facilities have used these statistics as a lever for implementing Lean. The results are impressive. ThedaCare, a Wisconsin-based health system, reduced inpatient total cost of care by 25% while improving patient satisfaction to nearly 100%. Seattle Children’s new surgery center reduced nonoperative time by 50% versus the main campus surgery center.

Lean defines waste as any activity clients view as not adding value to their experience and not meeting their needs. By focusing on activities which meet the needs of the client, you will realize benefits such as:

- Improved patient care
- Improved client satisfaction
- Improved staff satisfaction
- Reduced inventory
- Improved flow of patient care
- Reduced expenses

Waste goes beyond expired medications. Lean identifies seven areas of waste.

- Unnecessary services. Are you doing things that don’t need to be done, such as inadequate patient work-ups (running lab test A when you needed lab test B)?
- Mistakes. Does your staff regularly need to redo work and correct errors?
- Delays. Do equipment failures and wait times for charts or medications happen frequently?
- Unnecessary motion. Could you increase efficiency by moving equipment or supplies to reduce or eliminate wasted effort?
- Over-processing. Review your protocols and processes on a regular basis to determine if they are still relevant and provide value.
- Excess inventory. This goes beyond the products stocked for retail sale and doctor use. For example, too many files leads to the need for more cabinets and more floor space. Do you have files and equipment you have not used for months cluttering your work area?
- Excess transport. Do you juggle patients and clients among rooms?

Wasting the creative and technical skills of your employees is an additional area that practice owners may not consider. Let your staff shine! Look for opportunities to tap their skill sets. Ask your team to identify opportunities to eliminate waste and present plans to implement their ideas. The traditional top-down management style places stress on the owner to lead initiatives and ensure implementation. Lean processes empower employees to inspect their own work and redesign processes and protocols to maximize efficiency. The result: staff members have newfound enthusiasm for their work and more time to practice medicine.

One of the strengths of Lean is its focus on action. You can get started right away with a small project, see immediate results that excite you and your staff, and leverage this momentum to take on a larger project. The first step - designate a Change Agent. The leader of your Lean initiative must have an open mind about change and be able to make things happen. He or she can seize upon a frustrating experience and turn it into an opportunity to start a Lean project. For example, a staff member may struggle to find needed medical equipment in a storage area. The Change Agent can use this experience as an opportunity to rearrange the storage area in a way that makes items easy to find and reduces frustration and wasted time.

The Change Agent begins the Lean project by implementing the 5S System for the targeted area of improvement. While originally 5S was used as a tool for maintaining clean work areas, it has evolved into a systematic method for reducing costs, improving work flow, and empowering employees to assist in reducing waste. The staff members closest to the service now have the authority and tools needed to improve work processes or work areas.
Once the first Lean project has been identified, begin by taking photographs or video of the area of focus. Before and after pictures are a powerful tool for showing staff members the benefits derived from Lean initiatives. Next, utilize the 5S steps.

1. **Sort**
   a. Eliminate unneeded items within the target area. Dispose of items that aren’t needed. Fight the urge to hold onto items because you might need them in the future.

2. **Set-In-Order**
   a. **Current State.** During this step, document the location of each item and the current work flow. Create a map of the area to outline it, identify the large items, and map the flow of patients, clients, employees, and paperwork. Label all significant items, so they are easily identifiable to staff members. This process, called mapping a value stream, creates a one page picture or flow chart of the current process, and helps identify redundant steps and unnecessary motion.
   b. **Future State.** With the current state mapped, now create a future state value stream map. How can you eliminate waste identified in the current state value stream map? What is the ideal flow for completing a task? Brainstorm with your staff to create an area which has great flow, is well ordered, and reduces unnecessary movement. Items are now well-labeled and anything can be found within seconds. Everything has a place and there is a place for everything.

3. **Shine**
   a. Inspect, clean, organize, and de-clutter the area and items within the area. Repair or replace frayed cords, bad bulbs and batteries, and worn-out parts.

4. **Standardize**
   a. Create standards and visual controls such as signs and checklists to improve efficiency and reduce errors. Signboards and color code indicators provide important information at a glance.

5. **Sustain**
   a. Teach employees your Lean processes and protocols, so everyone understands the benefits. Continue to evaluate additional Lean opportunities.

As with any change, you may encounter staff resistance. People may not understand the need for change, may fear it will lead to more work, or may not understand that they will be playing a key role in determining the changes. Commit to the program, explain the need for the changes, and address the fears of the resisters. Unfortunately, about 10% of the workforce might remain resistant to the change and leave. But those who embrace Lean will enjoy improved safety, work flow, and reduced costs that lead to increased customer satisfaction, employee engagement, and practice profitability. Don’t wait to realize the benefits of Lean. Implement Lean in your practice now!
Practices must stay on top of current trends in staff compensation and benefits to remain competitive and attract efficient, effective and productive employees. Pay scales must also reflect the level of education, experience, skill and personal attributes employees bring to the table. Yet there’s more to keep in mind when it comes to keeping employees happy, satisfied in their position, and with the practice. Staff members tell us that competitive wages, appreciation for a job well done, and growth/career opportunities are the top three things that are most important to them as an employee. How does your practice rate in delivering the top three for your staff?

**Begin with compensation and benefits**

Results from Benchmarks 2015 show that Well-Managed Practices™ spend between 21% and 25% of revenue on staff compensation, payroll taxes, and retirement contributions. This includes all non-doctor staff positions; hospital administrators, practice managers, bookkeepers, receptionists, credentialed technicians, veterinary assistants, kennel/ward attendants, and groomers are in this number. Where you fall in this range will depend on the cost of living in your area, the skill set of your staff, and your staff-to-doctor ratio. See Figures 1 and 2 for the latest pay scale and benefits offerings.

If your staff costs are high, start by evaluating productivity. Often the issue isn’t over-spending, but rather lower-than-expected productivity. Low productivity has a variety of reasons. Sometimes it’s caused by giving away or significantly discounting care that the practice provides. Sometimes it’s related to a lower-than-warranted fee structure. Sometimes it’s due to a lack of skills or inefficient processes. And, sometimes a practice employs people who are a poor fit and who put a damper on the morale of the other team members. Before you start thinking of ways to cut staff expenses, first determine why your cost is high.

**Show appreciation**

Showing appreciation actually starts with communication. Staff members tell us that the number one thing that would most increase their job satisfaction is better communication with co-workers. Enhanced training and continuing education is a close second – and communication is integral to an effective training and education program. The staff also tells us the number one change that would have the most significant positive impact on the practice is improved internal communication.

Effective communication is crucial for business success. Getting everyone on the same page enhances efficiency and productivity, reduces stress and frustration, eliminates wasted efforts, and cultivates teamwork – all of which results in lower costs and higher profit. It’s also important to recognize your staff members respond to a variety of communication “styles”. Here’s how to interact with people with different personalities kindly, sensitively, and effectively (excerpts from Benchmarks 2015).

**The director**

Personality Characteristics: Competitive, ambitious, goal/results-oriented, task-oriented, confident and independent

To effectively communicate with:

- Be brief, direct and concise
- Avoid unnecessary details
- Focus on results and return on investment
- Provide options

**The mediator**

Personality Characteristics: Introverted, calm and supportive, loyal and team-oriented, great teacher, trainer, mentor

To effectively communicate with:

- Be patient and logical
- Friendly and neutral tone
- Steady, low-key approach
- Involve in planning process and idea generation

**The socializer**

Personality Characteristics: Charismatic and outgoing, talkative and very social, self-assured and sensitive, creative, visual learner

To effectively communicate with:

- Allow time for social interaction
- Avoid harsh or aggressive tone
- Put details in writing
- Use a whiteboard for discussions

**The analyzer**

Personality Characteristics: Introverted, meticulous and organized, detail- and task-oriented, fear of work being criticized

To effectively communicate with:

- Be organized and logical
- Support position with facts
- Ensure each point is understood
- Understand motivated by quality of output/data
Orientation, training, and mentorship

While the orientation and training provided to new employees is often considered mentoring, a true mentorship program goes beyond day-to-day training and focuses on accelerating the mentee’s professional development. A mentor shares knowledge and experience, points out other resources and networks that may be helpful to the mentee, provides critical feedback related to the mentee’s communication skills, interpersonal relationships, technical abilities and leadership skills, and challenges the mentee to move outside his or her comfort zone.

Employers who offer mentorship programs are more attractive to potential employees and enjoy better staff retention. Employees are more productive and satisfied in their position. Turnover, and its resultant cost, is lower. Informal mentoring occurs all the time and is very effective. Yet a formal mentoring program, done well, will give an added leg up.

In a formal mentoring program, both the practice and the mentee set specific goals and measure the results. Mentorship is available to all employees who meet the program criteria. Participants are paired up based on compatibility, and mentors receive training. Management promotes the mentorship program internally, so everyone sees the benefits of participating.

One key to an effective mentorship program is identifying the skills the mentee wants to build and the practice’s goals for the program. A mentee must visualize his or her future - where does he want his career to develop? Will she have a special interest niche in the practice, such as acupuncture, avian, rehab or surgery? Will he be the go-to person for internal staff CE? Will she begin on a path to practice ownership? A key success factor for a mentorship program is the mentee knowing what success looks like and what he or she wants to accomplish, and having a sense for the kind of guidance that would be most beneficial.

Likewise, the practice must set clear objectives for the program. How do you visualize the future of the mentee in the practice? What areas of professional development are most important to you? Do you want to expand a doctor’s medical training to add new services to the practice? Strengthen the veterinarian’s communication skills? Expand the associate’s understanding of the costs of running the practice?

Set staff members up for success

Managers need to put team members in situations where they’ll thrive. This process starts as early as the first interview. Ask candidates what they love to do, not just their past work experience. Working in your area of passion leads to happier and more efficient employees. Here are some ways to get team buy-in:

- Ask employees I reviews which of their skills are under-utilized. Find ways to use these talents to benefit the practice.
- Ask employees to submit a personal SWOT analysis prior to their performance review, and use it to supplement their evaluation.
- Pay close attention to the strengths and weaknesses the employee pinpointed. Tailor the employee’s goals for the coming year based on the SWOT to help foster buy-in and accountability for the goals.

Figure 1 – Pay ranges by position

<table>
<thead>
<tr>
<th>Position</th>
<th>&lt;3 yrs.</th>
<th>3 – 5.9 yrs.</th>
<th>6+ yrs.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital Administrator</td>
<td>No response</td>
<td>No response</td>
<td>$23.00</td>
</tr>
<tr>
<td>Practice Manager</td>
<td>$18.60</td>
<td>$22.00</td>
<td>$20.00</td>
</tr>
<tr>
<td>Office Manager</td>
<td>$19.50</td>
<td>$16.00</td>
<td>$18.75</td>
</tr>
<tr>
<td>Bookkeeper</td>
<td>No response</td>
<td>$14.75</td>
<td>$16.60</td>
</tr>
<tr>
<td>CSR/Receptionist</td>
<td>$11.50</td>
<td>$12.50</td>
<td>$14.25</td>
</tr>
<tr>
<td>CVT/RVT</td>
<td>$15.00</td>
<td>$16.00</td>
<td>$18.00</td>
</tr>
<tr>
<td>Veterinary Assistant</td>
<td>$11.50</td>
<td>$12.25</td>
<td>$13.25</td>
</tr>
<tr>
<td>Kennel</td>
<td>$9.00</td>
<td>$11.00</td>
<td>$11.50</td>
</tr>
<tr>
<td>Groomer</td>
<td>$9.25</td>
<td>$14.00</td>
<td>$24.25</td>
</tr>
</tbody>
</table>

Figure 2 – Benefits in well-managed practices®

Retirement                Simple IRA - 3% match
Health Insurance

Continuing education (per employee, per year)

<table>
<thead>
<tr>
<th>Position</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Managers</td>
<td>$1,000 - $1,500</td>
</tr>
<tr>
<td>Client Services Representatives</td>
<td>$350 - $500</td>
</tr>
<tr>
<td>Technicians</td>
<td>$500 - $950</td>
</tr>
<tr>
<td>Veterinary Assistants</td>
<td>$340 - $500</td>
</tr>
<tr>
<td>Kennel</td>
<td>$250 - $390</td>
</tr>
</tbody>
</table>

Paid time off

<table>
<thead>
<tr>
<th>Experience</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 3.9 years</td>
<td>10</td>
</tr>
<tr>
<td>4 – 9.9 years</td>
<td>14</td>
</tr>
<tr>
<td>10+ years</td>
<td>17</td>
</tr>
</tbody>
</table>


Resources to help evaluate your compensation and benefits

Benchmarks 2015 – A Study of Well-Managed Practices®

Compensation and Benefits by AAHA Press

On-line sources such as www.salary.com or www.payscale.com
5 Physical Exam Hacks for Better Accuracy and Client Talk
Ernie Ward, DVM, CVFT
Seaside Animal Care
Calabash, NC

Most veterinarians don’t allow a meager 1800 seconds to perform an examination. That’s 30 minutes to welcome, examine, diagnose, offer advice, obtain products, review usage and follow-up, invoice the client, and schedule da return visit. Good luck. Why are we so stingy with the time offered to our patients and clients? Time won’t solve all our professional problems, but it highlights where our priorities are. I can’t force you to spend a mere 30 minutes per appointment, but I can suggest some compelling tactics to make each second of your exams more meaningful.

Connect hearts, minds, and paws
First impressions count every time. This means whether you’re meeting a client on the first or fortieth occasion, those first few seconds set the tone for the rest of the visit. Regardless of how hurried, distracted, or overwhelmed you may be, your success with the current appointment is determined by how present you are in that moment. If the client perceives you’re distracted, disinterested, or disconnected in any way, things typically don’t go well. I’d also add that I’ve found if an animal senses you’re not thrilled to be there, they’re less agreeable and often downright nippy.

Before you begin each appointment, know client and pet names, gender, age, pertinent medical history and reason for visit. Being prepared allows you to focus on faces instead of charts. Enter the exam room smiling, relaxed, confident, and grateful for the opportunity to practice your art. Extend a warm handshake, pet the patient, and sit down. I recommend sitting whenever possible to eliminate any potential intimidation, foster intimate communication, and aid attention. Deliberately focus on listening. Too often, veterinarians have diagnosed and decided before asking and examining. I find when I approach exams unbiased, I’m a better diagnostician.

As the conversation continues, pay close attention to the client and patient’s body language. Do they understand what you’re saying and doing? Do they agree? Are they comfortable? If your compliance rates aren’t what you’d wish, chances are your clients aren’t connecting with your advice. Ask questions such as, “Does that make sense?” “What do you think about that?” “Do you think Rover will be okay trying that?” Staying connected means actively engaging and ensuring clients agree with our recommendations.

Make comfort a priority
Managing veterinary appointments is tricky because we’ve got to accommodate both human and animal needs. What pleases people may not comfort canines and what comforts canines probably won’t make Himalayans happy. When considering my consultations, I ask, “Is everyone as comfortable as we can make them?”

Train your staff to evaluate animal and human body language as soon as they enter your hospital. Are they stressed, nervous, or angry? If so, have protocols for easing apprehension. Many times a warm smile, gracious greeting, comfortable seating, and offering water, tea or treats can allay most worries. Teach your team to engage in conversation, introduce clients to one another while waiting, and pay attention to the energy and attitudes of clients. Make it your goal to lift spirits instead of avoiding anxiety.

When you interact with your pet patients, critically analyze your movements. Aim to use minimal restraint, be exceedingly generous with treats and distractions, and intensely focus on the pet’s responses. You’ve probably only got about 600 seconds of hands-on contact; make them as pleasant as humanly possible.

Compliance insurance
You’ve got less than 1800 seconds to connect, comfort, and practice medicine during a standard appointment. Some veterinarians fear if they emphasize communication they may minimize medicine. Not true, if you’re tactical. To help keep my team centered on my patient’s medical needs, we use a variety of single-page examination reports. These reports are divided into the medical history, physical examination, and a checklist of meds, supplies, and services we recommend.

The medical history includes the most common questions we have for wellness visits by species and age, queries for frequently seen medical conditions, and more extensive behavioral assessments. The exam portion highlights our standard physical examination findings by anatomy and organ system, and the checklist ensures clients leave with what they need.

Provide a copy of the report to your client during discharge. Review the pertinent details and highlight areas that need further attention. At the completion of the appointment, make sure to answer, “When will we see you again?” Don’t be afraid to schedule an appointment six months or more in advance. The client can always change it if necessary. Booking future appointments today is an excellent way to encourage follow-up and routine visits. I’m constantly amazed at how powerful this tactic is in practice. Advance scheduling works because most people honor their commitments.
1800 seconds spent wisely on each appointment can be incredibly valuable. Don’t waste them waiting in the lobby or consultation room. Don’t squander them performing a meandering meaningless exam. Don’t fritter them away creating stress and anxiety. Every second we have is priceless. Create daily tactics that guarantee you’re making the most of your most valuable resource – time.
Make your Peace between Profit and Charity in Practice
Ernie Ward, DVM, CVFT
Seaside Animal Care
Calabash, NC

- “You care more about my money than my pet!”
- “I wish you could be my doctor.”

In my 26 years of veterinary practice, I’ve heard those words on countless occasions, sometimes from the same client. Those sentiments highlight the struggle we have within our profession: Why do veterinarians have an agonizing association with money? Where do we draw the line between profit and compassion? How can we do good for animal welfare while doing well financially in our practices? The answers may be both simpler and more complicated than you think.

How did we get in this mess?
Veterinarians were revered in ancient times because we cared for our tribe’s food animals. Our roots intertwined with commerce as livestock attained economic value. During the previous two hundred years, the limits on our compensation became based on the “replacement cost” of the animal. In other words, we could only charge commiserate to the value of the patient we were treating. The limitation on our fees had the unintended consequence of hampering primary medical progress in veterinary science. For most of modern veterinary medicine’s history, we’ve been consigned to scrounging for human medical research scraps. It’s only been in the past fifty years that major pharmaceutical companies have recognized animal health as a profitable sector worthy of their resources. And that gets to the real reason of how we got in the mess.

Our limiting legacy
It wasn’t until the 1970’s that small animal veterinary medicine as we know it today came into existence. Prior to that, a farm-call veterinarian would administer the occasional vaccine or offer basic treatment for a dog or cat. Why? Because there wasn’t any inherent financial value in pets. The farmer could justify spending £50 on a cow he could sell or milk but not on a dog with a broken leg. While his family dearly loved the dog, he just couldn't rationalize paying much for treatment.

It was in this environment that early small animal veterinarians established the money-guilt we experience today. The first pricing schedules were grounded in the legacy of “replacement cost” and arbitrary mark-ups based on what veterinarians thought clients would pay. Feeling uncomfortable raising their fees for examinations and professional services (it was only a cat, after all), many veterinarians expanded their offerings into areas they weren’t entirely at-ease. The battle between “selling” and “serving” commenced. There were a multitude of paths our profession could’ve, and probably should’ve, chosen to elude our current quandary. Despite all of this, I believe part of our predicament can be traced back a few more decades to a beloved icon.

Blame it on Herriot
I also blame James Herriot for our profession’s money-guilt. Herriot set the “veterinary virtuosity bar” unreachable by most of us by accepting a few eggs in exchange for years of specialized education, investing in expensive equipment and medications, driving in dangerous conditions, leaving our families, and then staying up all night curing an ailing animal. I can’t match that. Many veterinarians swayed by his chivalry cringe when conversing about currency for our services. “That’s okay, a handful of eggs will be sufficient.” His memoir, “It Shouldn’t Happen to a Vet,” takes on an entirely new meaning now, doesn’t it?

We carry this ethos in our DNA today. Even as pets have risen in prominence and incomes allowed owners to demand high-tech tests and treatments, we still harbor hesitancy about money. Some of our colleagues consider anything more than a few eggs is too much to charge. Others are convinced that financially successful veterinarians are greedy, uncaring, or probably both. In my experience, there’s room for both eggs and elitism in the veterinary profession.

Making peace with profit and philanthropy
Businesses exist to produce profit. As long as an enterprise pursues profit in an ethical manner, making money can benefit society. For example, if my practice revenue grows, I am able to hire additional staff members and pay a fair wage, invest in helpful technology, and give back to my community. Or not, if I’m a greedy louse. I can’t do either if my practice isn’t profitable.

Whenever the debate of should veterinarians do more … (fill in the blank with the cause of your choice), I typically say, “Yes!” I say this because we should be doing more. More community outreach and involvement, more political engagement, and more personal development. In my experience, the best businesspeople aren’t greedy, they’re giving. Most truly successful people I know understand to sustain success they must invest in their staff, contribute to their neighborhoods, and retain high standards, ethics, and morals. I’m incredibly proud to call myself a veterinarian and just as proud of my entrepreneurial accomplishments.
**Roadmap to peace**

The first step toward reconciling the pull between wealth and charity is to define yourself and your practice objectives. What does it mean to be a veterinarian to you? What are your metrics for personal and practice success? How do you know if you’re achieving your goals? Too often we allow others to determine who we are and how we practice. This creates tension, frustration, and, ultimately, disillusion. Burnout is the result of dissatisfaction with an undefined journey.

Self-reflection is essential because I feel most money-guilt is anchored in poor professional self-confidence. Blame it on our history, the economy, or Herriot. The fact remains that many veterinarians would rather swig anal gland juice than talk about money with clients. Maybe it’s because I started my first practice without any full-time employees in a poor, rural setting, but I became comfortable looking clients in the eye, telling them how much money they owed me, and saying, “Thank you.” I became “money-calm” because I’m convinced I provide a valuable service. That’s not arrogance; that’s self-confidence and it can help you relax around remuneration.

The next step is giving back. I’ve always focused on one or two major fundraisers for my clinics each year. Most of our philanthropy involves animal and children’s charities. We also created a fund within our business to raise monies to aid financially distressed families with their veterinary bills. I’ve discovered that whenever your staff witnesses you giving back to charities and needy clients, they’re better handling the inevitable, “All you want is my money!” complaints.

Inserting yourself into animal welfare issues is another stride toward being proud of your profit. One of the chief responsibilities of success is applying your influence to causes you’re passionate about. Being financially fruitful often opens opportunities for change you’d never access form the cheap seats. Movements and charities need disciplined, ambitious, and bold leaders. These attributes are most often associated with successful businesspeople. Use your power to fight for better treatment for animals.

**Falling off the tightrope**

Treading this tightrope isn’t easy. I can’t adequately explain nor resolve this issue in a few hundred words. Clients will still say ugly things to you and about you and stress doesn’t evaporate overnight. You’re going to want to quit and take the eggs. Don’t. Not only is our profession counting on you, your career and happiness are at stake. It gets better the more confident you become. Absolving yourself from the guilt of receiving money for your expertise is crucial for professional peace of mind. Self-confidence helps shields you from the sting of criticisms and allows you to develop into the veterinarian you dreamed. For that, I thank Dr. Herriot. His writings shaped who I am today and instilled in me the power and sanctity of the human-animal bond. It’s up to each of us to turn yesterday’s basket of eggs into today’s credit cards and cash. Perhaps no other profession can do as much good while doing well. I’m proud to be Ernie Ward, Veterinarian.
One of the more awkward affairs to follow is an aging heavy metal band. Bare chests, bravado, and body-hugging buckskin have decayed into pruned pouts, blank stares, and mom jeans. I wonder if hip Millennial pet owners sometimes look at today’s veterinary practices and liken us to Iron Maiden rocking the local Travelodge? Millennial pet owners are here and poised to dominate the future of veterinary medicine. It’s time we transition from a Boomer-centric practice model or risk looking like out-of-shape and out-of-touch geezers to a new generation of clients.

Fortunately, we’ve got a little time to prepare for this new wave of pet owners. In approximately three years, Millennials (born 1981 to 2000), will begin outspending Baby Boomers (born 1946 to 1964) on everything from cars to cats and dresses to Dachshunds. They’ll also be buying dresses for their Dachshunds according to consumer research firms such as GfK and Wakefield. 63% of Millennials agree that pets should be pampered and treated as family members with 76% reporting they’re likely to splurge on luxury items such as clothing, custom beds and expensive treats. Over 75% prefer pet supplies made from natural or organic materials, are BPA-free and hypoallergenic. Right or wrong, 86% feel “natural” pet food is essential and favor retailers and brands with a socially- and environmentally-responsible platform. In other words, younger pet owners are choosing clinics and companies committed to protecting our planet and people. What other strategies can veterinarians use to attract and retain Millennial pet owners?

Taking requests
I recently saw one of my all-time favorite 1980’s New Wave bands play. They’d just released a critically-acclaimed album and embarked on a North American and European tour. Together thirty years and still making vibrant, contemporary music. One of the cool things they did that night was solicit song requests on social media. I hash tagged my treasured tunes, along with about 200 other concertgoers. After the show, the guitarist told me they ripped the idea from a (much) younger band earlier in the year and wanted to keep current. “If you stop growing, you’re dead,” he said.

Veterinary clinics need to actively include Millennials in decision making. While Boomers relied on doctors to decide what was best for their pet, the Internet Generation was raised on choosing for themselves. Baby Boomers were told what to do as children while Millennials were brought up being asked what they’d like to do. This is a significant shift and directly affects how veterinarians should engage young clients. Shared decision making strategies have been around for years; they’re no longer an option. Vets must allow Millennials to feel they’re in complete control of their pet’s care and provide extensive information, resources, and time to choose.

Instant second opinions
Control is key because Millennials will be fact-checking you constantly. As soon as you exit the exam room, young clients immediately consult Dr. Google to authenticate your advice. This is one of the reasons I started offering clients an iPad with a website to review whenever I needed to leave them alone. I realized they’d be searching online behind my back so I figured I’d at least lead them to a trusted track.

I believe we must do a better job of explaining our examinations (“actively articulate your actions” I like to say), providing written diagnostic rule-outs, and offering treatment options, especially to Millennial pet owners. If you neglect to mention a common alternative test or therapy, they’re going to find out. Don’t fear this; embrace it. This generation values education and information, our professional strengths. Shift your communication style from dictums to discussions and enjoy the strong bonds you’ll forge.

Mobile is the modern home
Over 87% of Millennials report their smartphone never leaves their side and they surf the web using two to three devices a day. Strategic online marketing is essential for future success. Your website must look fabulous on multiple platforms, tell your story in a succinct and compelling way, and offer ways to interact at the virtual push of a button. This includes online shopping, appointment booking, communications, and pet records. Regardless of what is or isn’t possible, what has or hasn’t been done, and what you feel you can or can’t do, young pet owners are demanding these services and more. How are we going to handle virtual vet visits, non-veterinary medical care and opinions, and the avalanche of bad online pet care advice? The veterinary profession must solve these economic, legal, and ethical challenges and create safe and effective methods for veterinarians to provide the services of the future. Quickly. If veterinarians don’t, outside providers will continue to win the, hearts, revenues, and smartphones of tomorrow’s pet owners.
Better be social

One of my dear college buddies is now head of Facebook Media Partnerships. He helps develop how Facebook delivers news. That's important, because about 88% of Millennials get their daily news from Facebook. 44% reveal they check Facebook “several times” a day and 36% and 33% prefer Pinterest and Twitter to learn about their world. Newspapers and television are so 2000.

You probably already dabble in social media. It’s time to deep dive. Collaborate with your team and experts to determine your social media strategic objectives, deployment, monitoring, and measurements. Resources dedicated to social media are necessary to remain competitive in today’s online world. If you’re unsure how to proceed, seek outside help. Time is too precious to wasting it posting content that doesn’t generate results. You also don’t want to go viral for something stupid. “Leave Britney alone!” anyone?

Passive aggressive anonymity

You’ve probably heard of a disgruntled client who went on social media to viciously attack a veterinarian. Cyberbullying of vets got so bad in the 2015 “America’s Favorite Veterinarian Contest” that it had to be cancelled. A single “F” or one-star rating is enough to scare many prospective clients from trying a veterinary clinic. Millennials take online reviews, rating websites, and crowd wisdom very seriously. Many report they trust online advice over friends and family recommendations. Studies show younger adults believe anonymity promotes greater honesty. I think they’re probably right until I get dinged with an unfounded and unsubstantiated negative review or comment.

Online reputation monitoring should be a part of your daily routine. It can be as simple as creating free Google alerts for your clinic and team or as comprehensive as using a professional service. If you’ve already received a few harmful reviews, you should probably hire a pro to help. If you haven’t you may want to go ahead and purchase some peace of mind. I hate to tell you to spend money on this sort of thing, but I’ve seen too many clinics suffer the damage a handful of haters can cause.

The great news is online reviews can boost your practice. Don’t be afraid to ask clients to rate you. I instruct my receptionists to subtly slip in, “It was great meeting you today! If you’re happy with how Fluffy did with Dr. Ward, we’d really appreciate if you’d tell your friends or rating us on our Facebook page.” Sometimes that gentle nudge is all it takes to get five stars.

Iron Maiden, Saxon, and Judas Priest are still touring. Ozzy is a TV star. I don’t view them anymore as doddering doofs donning denim and doling out drivel; I see them as teachers. You can continue doing things the same old way and probably get by with it for years to come. The greatest hits will always be hits. If you want to grow, evolve, and remain contemporary, you’ve got to change. The Millennials are coming. It’s up to each of us to write new songs or be content playing our greatest hits. There will always be an opening at Travelodge on Tuesday night.
Introverts Unite…Separately: 10 Tips to Survive and Thrive in an Extroverted Profession

Sarah Wooten, DVM
Sheep Draw Veterinary Hospital
Greeley, CO

With jungle gym careers, increased competition for jobs, more complicated customer interactions (social media, yelp, and google reviews, anyone?), veterinarians have to establish critical relationships and communicate important messages to their clients and colleagues. In a business world that largely relies upon extroverted strengths, finding space to be heard can be a challenge for the introverted veterinarian.

Fortunately, extrovert-centric self-promotion is so last decade. Today - influencers will stand out if they can build other people up and commit to listening over talking.

Before we can talk about the strengths of introversion, it’s important to understand what introversion is. An introvert is someone who seeks solitude to recharge energy. Introverts draw energy from within themselves. Introversion is not shy. Shy is behavior or behaviors caused by anxiety in social settings. Characteristics include needing and desiring time alone, tendency to think before speaking, prefer to dig deep vs. small talk, may be less likely to express emotion and can be difficult to read, enjoy writing over talking, and can be private, quiet, and reserved.

In the American workplace, there can be barriers that prevent introverts from becoming influential leaders. These can include an emphasis on team approaches that can be draining and stifling to the creativity of an introvert, a tendency to shy away from self-promotion, pressures within the American business culture to behave like an extrovert, feeling talked over by extroverts, pressures to make decisions quickly, and the stress that goes with these barriers.

It is important for introverts to know that they are not alone in feeling frustrated by these perceived barriers, and that being an introvert is not a weakness. In order for introverts to establish professional success, we must first stop trying to behave like extroverts, and instead, focus on the strengths that come with an introverted nature.

Introverts must schedule quiet time to recharge, prepare, find creativity and solutions, and not burn out. Preparation and ability to focus is a strength of introverts, but they must have time alone to exploit this strength. Quiet time allows preparation by visualization, and increases the likelihood of success of professional encounters with clients and staff. Even a few minutes of solo concentration can help an introvert increase focus and influence.

How do you find your quiet time? How can you prioritize this sacred time and use it to your best advantage?

Where can you go without distraction?

What activity (or activities) provide you with recharging quiet time?

Think about a challenge that you are currently facing. How can taking some quiet time help you with influencing change?

A second strength of introverts is preparation: Introverts are game-changing strategists. We hate to wing it. We like a game plan, and rehearsal, and all details ironed out well before its game time. This allows an introvert to present himself or herself as the expert on the topic at hand, and increases confidence of both the veterinarian and the client.

Another area of strength includes intentional and attentive listening to clients, an ability to read body language, and tuned-in empathy, which encourages clients to share feelings and questions more readily. It also gives an ability to help clients make hard decisions. Introverts tend to display tremendous listening skills, which is critical to patient management and successful practice. Introverts can use this ability to gain credibility and achieve better compliance, especially through the use of open-ended questions and focused conversation to really understand the problem before provided customized treatment and diagnostic plans.

Introverts also display a penchant for writing, and writing for influence, whether, through one-on-one interactions with clients, via veterinary or pet publications, or through thoughtful use of social media is yet another way introverts can influence and lead our profession.
When communicating with clients, there are subtle changes you can make in your delivery that can have big payoffs in increased compliance, better client retention, and increased new clients. If you use the right words, the word gets around. In this session, we are going to identify simple communication tweaks that will strengthen your confidence, reduce confusion in the client, and instill a sense of goodwill between client and veterinarian. In this session, we will go through two ways to communicate an idea, the pros and cons of each, and how changing what you say or how you say it might get more ‘yes’ from your clients.

1. ‘I recommend’ vs. ‘your pet needs’
2. ‘We need to run a CBC, chem, T4, urinalysis, etc.’ vs. ‘We need to get some blood and urine for some tests.’
3. Silent physical exam vs. talking your client through the physical exam.
4. ‘I noticed the following abnormalities on your pet’s physical exam – list them’ vs. ‘First let’s talk about what is good. Your pet’s heart sounds good, no murmurs’
5. ‘Do you have any questions?’ vs. ‘Does that make sense?’ Sometimes clients are too overwhelmed to respond to an open-ended question, and asking a simply yes/no question is more appropriate and easier for them to process.
6. ‘I’ve been looking on the internet and I found…’ eye roll, pursed lips, gusty sigh and fill-in-the-blank immediate negative reaction vs. ‘great job researching. I can tell you really care about your pet. Here is what I know…’
7. ‘I feed blue buffalo’ – what are your responses? What is successful? What is not? CROWDSOURCING! What I’ve found: in our clinic we have abnormal gut flora, increased urine pH with increased numbers of urinary stones. Here are some other diets that you can try…
8. ‘I don’t want to heartworm test, my pet doesn’t go outside.’ What do you try?
9. ‘your pet needs a dental, here have a giant estimate’ vs. ‘things look pretty good today, however I am concerned about your pet’s dental health. See this tooth? It’s infected, and that bacteria goes into the bloodstream every time your pet chews, which puts additional wear and tear on the internal organs.
10. ‘your pet is fat, cut back calories 20%’ vs. ‘I’ve noticed an upward trend in your pet’s weight. Have you? This additional weight puts your pet at risk for all the same issues that overweight humans have (make it relevant). Here is the exact amount of calories per day to feed of EVERYTHING THAT GOES IN YOUR PET’S MOUTH to prevent unhealthy weight gain. (give written instructions)
11. ‘I recommend a lepto vaccine’ vs. ‘leptosporosis is a growing human and pet health concern in our area. We’ve diagnosed some cases, and in order for you to know about the risk, I’ve been super intentional with counseling pet owners on the risk to both you and your pet. It is a simple two booster vaccine and requires a yearly booster. Do you want to do that today? It will save you some time and money vs. coming back later to do it.
12. ‘restrict your pet’s exercise after surgery to prevent surgical complications’ vs. ‘in a pet’s mind they are either fixed or broken. If they feel ‘fixed’ they will overdo it, and that could cause big problems for healing. Protect your investment in this surgery, and keep your pet in a kennel/on a leash at all times/or a specified amount of time. Then ask for clarification. Are the exercise restriction instructions clear?’
13. ‘coconut oil/apple cider vinegar/cesar milan’s alpha roll/fill in the blank questionable-internet recommendation will not fix the problem’ vs. ‘it sounds like you have been trying some things at home that aren’t working. Let’s work together to try and figure this problem out. I believe I can help you.’

In conclusion
1. Make sure the client understands what you are saying. Finish with ‘does this make sense?’
2. Say ‘your pet needs’ vs. ‘I recommend’
3. Draw parallels to human health issues to make the issue relevant and better understandable for the client.
4. Encourage the client when they make the effort to do research, even if it erroneous.
5. Give positive reinforcement when the client asks hard questions: ‘that is a good question’
6. Make sure you address the reason the client came in. Ask ‘did we address your concerns today?’ If you end up having to fix something else before addressing the client’s primary problem, address that as well. ‘I know you came in because your cat is urinating on your bed. I believe the pain that your cat is experiencing from dental disease is contributing to this problem, so we fix that, and hopefully the problem will stop. If it doesn’t, I need to know.’
7. Utilize fear free tactics when possible, humor works really well to break the ice and lower your client’s anxiety levels.
Tips from the Trenches:
Subtle Ways You Sabotage Client Relations
Sarah Wooten, DVM
Sheep Draw Veterinary Hospital
Greeley, CO

Often times we as veterinarians are unaware of how we are impacting our clients mentally and emotionally, and how that might open doors or create barriers to care. In this session, we will go over several subtle ways in which we can break down client relations, and what to do instead.

Possible barriers to productive veterinarian-client relationships:

1. Not acknowledging the difficulty of the situation right away – establishes empathy and instills loyalty.
   a. Emergency – I’m sure you wanted to be at the veterinary hospital today, right? This was part of the plan for the day.
   b. Poor prognosis: this is hard. I’m really sorry.

2. Refusing to compromise on a diagnostic or treatment plan, i.e. only offer the best, and if the client declines, don’t give them a second option that may be more palatable to them.

3. Using big words.

4. Not using discharge instructions and handouts.

5. Not being sympathetic to client concerns for high estimates. The struggle is real!

6. Not developing your best euthanasia best-side manner. ‘no one cries alone’ – a good end inspires incredible client loyalty, and for good reason. This is hard.

7. Not asking for clarification on the part of the client after you explain something. “Does this make sense?”

8. Assuming that every client wants an in depth explanation. Learn to read your client. Some clients want to know everything about their pet’s condition – they will geek out on everything you give them. They thrive on explanations like ‘your pet has gastroenteritis secondary to dietary indiscretion. We are going to give an injection of maripotant to reduce vomiting and gut inflammation, and then this is an antibiotic called metronidazole that is designed to…on and on.’ Other clients are what I call ‘going to the mechanic client’ – they don’t care about the disease process, they just want it fixed and not to happen again. They get overwhelmed easily with information. They are happy with ‘Your pet has diarrhea. Give this and if it doesn’t clear up in 24-28 hours, call me.” Start with basic explanations, and then offer more information if the client seems interested.

9. Assuming that every client is not doing his/her best. Not giving praise for a job well-done.

10. Using closed body language, not maintaining eye contact, and not giving your undivided attention to the client in the room.

11. Using adjectives like ‘kinda’ when describing your recommendations, response to therapy, etc. ‘after surgery, your pet will be kinda groggy’ or ‘I kinda recommend a urinalysis’ etc. Using ‘kinda’ can be like ‘um’ – a space filler when you aren’t feeling confident in your communication. Think before you speak, so you can be intentional, clear, and what you say can have the greatest impact.

12. Not understanding your client’s level of anxiety. Asking “how are you?” can go a long way. Smiling, using humor when appropriate, and meeting your client where they are will all break down barriers to care and establish trust and rapport.

13. Not understanding or addressing your patient’s anxiety and fear. Not utilizing fear-free tactics. Letting the client know that you understand their pet is nervous and that it is natural. I draw parallels to how I look when I visit the dentist.

14. Trying to convince a reticent client. They are either going to listen, or they are not. You lay out the facts to the best of your ability – that is your job. If you have a good relationship with the client, you can try this: “Ok. I definitely hear you. I think it would help me to help you and your pet the best if you explain to me your reasons behind your decision.” What the client does with it is their decision, not yours, and if you are too pushy, you can piss them off. When they don’t do what you say, document it. Sometimes, the answer is initially no, but if you show respect to the client, respect their decision, and maintain the relationship, sometimes the answer changes over time to ‘yes’.

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Putting the Treat into Treatment:
Teaching Pet Owners to Get Pets from the
Living Room to the Exam Room in a Calm State
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Walking tools that help owners gain control over their pet’s forward motion and direction without the use of force are front clip harnesses and head halters. It’s important to use fixed length leashes and to avoid retractable leashes, especially with fearful animals, as numerous problems are likely to occur with such tools and there’s a general lack of control.

Cat harness and leash can be used to teach kittens and relaxed adult cats to follow and have protected time outdoors. Such walking devices also make the relationship more interactive and provide another form of control useful for minimal restraint during exam if the animal is accustomed prior.

Teaching animals to relax in the car is another important way to reduce stress before the animal has even arrived. Decreasing the anxiety of being in a crate, then reducing aspects of the car that might be stressful (such as noises and motion sickness) along with Victory Visits to fun places can help reduce anxiety. For some animals simply training outside of the car or sitting in the car while getting their meal, treats or doing training may be a helpful place to start.

Victory Visits are one way to practice elements of the veterinary visit that are made to be more like the actual vet visit while being kept positive and at a rate the animal tolerates. Victory Visits are done in preparation for actual exam and treatment at the hospital and are preventive in building a positive association with the clinic. Puppy classes and kitten socialization guidance are other strategies for building a more social, less aggressive and more handling tolerant animal from the start.

To get animals to move on or off of things or to approach something targeting, luring and tossing treats are all helpful strategies. Treating is one helpful strategy for counter conditioning an animal to the veterinary experience. But, in many cases the treat rate is far too low and the reward value or treats given is minimal for the animal. Rather than one to three harder treats given during the visit have an arsenal of available treats, toys and other rewards to employ. And, employ the tastiest, most enjoyable treats possible at a fast rate to keep the animal occupied during exam and procedures. One tactic is using distractions and feeding or keeping the animal’s attention on something else throughout the procedure. Or, another useful strategy for decreasing anxiety longterm is conditioning the animal to tolerate handling.

AAHA guidelines are helpful for incorporating behavior guidelines and checks into regular veterinary visits to address problems early and often. The sooner a behavior issue is addressed the better chance it has for being resolved. Many issues get worse, not better, when left on their own without treatment.
Fear Free Handling—
Understanding the Art, Design, and Feel of this Fashion Tech-Style
Jonathan Bloom, DVM
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Many pet owners fail to identify, and are reluctant to address, conditions such as dental disease and arthritis in their pet because they don’t see the disease, and they don’t appreciate the negative impact that it has on the body. In contrast, most pet owners are EXPERT at identifying fear and anxiety in their pets, and owners are very much aware of how a negative experience can impact both their pet’s mental health and wellbeing. Experience and careful observation reveal that those sentiments are not restricted to just the outpatient visit, but also apply and contribute to the pet owner’s reluctance to hospitalize their pet. In addition, pet owners are reluctant to approve necessary routine procedures such as blood collection and x-ray for much the same reason. Implementing strategies to maximize patient comfort is the most prudent way to create successful experiences while visiting the hospital. Pet owners visiting my practice have been overwhelmingly accepting and appreciative of the efforts made to ease their pet’s fear and anxiety.

Technicians have been performing common procedures such as blood draws, nail trims, and x-rays the same way for decades. But just because it has become the norm, doesn’t mean that staff like it! No staff member likes being bitten by a dog, or scratched by a cat. No staff member loves donning protective leather gloves to hold a cat for a nail trim or to remove an IV. No staff member loves stretching pets out in an unnatural and uncomfortable position to take an x-ray. And no staff member loves working in an environment with barking and whining pets. We all work very hard and want to be appreciated for what we do. It is much more rewarding when pets can be gently controlled for nail trims and IV placements, when pets can be calmly positioned for x-rays, and when the background noise from the dog wards can be kept to a minimum.

Identifying fear and anxiety in the hospitalized pet
A problem well stated is a problem half solved! Proper care for out-patient procedures and for hospitalized pets start with the proper identification of fear and anxiety. Veterinary healthcare providers need to pay more attention to signs such as trembling, hiding under bedding, vocalizing, body position, and lack of comfort behaviours etc.

The pet’s surroundings and housing
Common belief has long been that dogs are colour blind. Dogs can however see many of the same colours that humans can see. Fear Free™ has developed a colour palette that was selected to be both positive and visually comfortable. Bright lighting can also be uncomfortable for pets. Dimmable lights are ideal. Cages are believed to best suited for housing when there is opportunity for looking outward with few obstructions, or have the option to retract to an area when less sensory stimulation is present is also ideal. This can be achieved by providing boxes, tents, or partial covers so pets can choose their level of stimulation.

Sample collection
Attention should be paid to commonly performed procedures such as blood collection, urine collection, IV catheter placement and removal, treatment of skin wounds etc. Procedures used to make these more comfortable experiences often include the use of compression wraps, topical anesthetics such as Emla cream, pharmaceuticals, and environmental control.

Radiology
For years, pets have been forced into dark X-ray rooms, stretched out onto hard table tops, placed on their backs with their legs being squeezed by the vice like grip of lead lined gloves, while their limbs are pulled in 4 different directions. There is nothing natural or enjoyable about this for pets. Retakes are numerous, and X-rays are often of compromised quality. Several options including compression wraps, pinch induced behavioural inhibition, or pharmaceuticals are often used to create faster, better quality, more comfortable x-ray experiences for pets.

Post-operative care
Pets often experience stress and anxiety due to the direct result of pain. Careful attention must be paid to regularly assessing and addressing pain relief in our hospitalized pets. Environmental control is also critical in ensuring a smooth, comfortable post-op recovery including consideration given to noise levels, music, pheromones, body positioning etc.

My staff have never been more eager to accept a fresh and innovative healthcare initiative in the past as they have been for FEAR FREE™. They realized that they are surrounded by calmer, happier, and more easily handled pets. As a result, staff are working in a more safe and in a more enjoyable work space. Subsequently, staff satisfaction and staff morale have never been higher. The creation of a Fear Free™ philosophy and culture benefits pets, pet owners, hospital staff, pet healthcare, and the business as a whole.

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As a frequent consultant and instructor, I have the incredible opportunity to meet many of you within your own practices. When I ask if you chart your dentistry, most of you enthusiastically report that you do. This is a positive change from the past. Now, I would like to take this opportunity to look at the dental chart in detail and review the importance of this document.

But before we start looking at the document and how to record your findings, I recommend we review some anatomical terms as they relate to the mouth and associated structures.

First, you should know the proper and expected dentition of puppies and kittens versus adult dogs and cats. In the puppy, there are no deciduous first premolars or molars. In the kitten, there are no deciduous molars. Also, it is important to know the eruption schedules. In the puppy, primary or deciduous teeth begin to erupt around 3-6 weeks of age. Usually, by 6 months of age, the adult teeth are replacing the primary teeth. Also, it should be mentioned here that many of the “micro-breeds” tend to experience a delayed eruption. This is important information when planning treatment options for extracting retained deciduous teeth.

Begin the assessment of the head by looking at your patient squarely in the face and note any swellings or asymmetry while the patient is still awake and conscious. Note any facial abnormalities such as unilateral facial swellings.

If the patient is cooperative, a conscious intraoral exam can be very beneficial in case planning as well. Things to note are; the bite, tooth occlusion and any tooth-to-tooth contact and any tooth-to-tissue contact. The tissues of the gingiva, the mucosa and the lips all should be examined and notes made of any abnormalities. Also, note any odor, discharge, swelling, tumors, etc.

Once you feel a good conscious intraoral exam was performed, the patient should be anesthetized for the comprehensive assessment. This is when you are able to gather the most information. One person should be performing the examination and another should be recording the information. It is a good practice to have the person who is performing the anesthesia make the notations on the dental chart.

**Tooth identification**

There are several different methods of referring to each tooth. A tooth can be identified with an abbreviation. For instance, the left upper fourth premolar would be noted as LUPM4. The benefit of that is that everyone can understand that.

The other method is called the modified Triadan numbering system. The first number refers to the quadrant that the tooth is found. The second and third refer to the tooth position starting rostral and moving caudally. The right upper arcade is the 100 series, 200 is the left upper arcade, 300 series is the left lower arcade and the 400 series is the right lower arcade. Tooth 401 would be the right lower first incisor and tooth 411 is the right lower third molar.

Deciduous teeth are the 500, 600, 700 and 800 series in the same pattern.

The modified Triadan numbering system applies to the cat dentition as well. The difference is that they have fewer teeth. The cat have no upper first premolars, they have no first and second premolars on the lower arcades and there is only one molar in each arcade.

So here are some easy landmarks for you:
- The first incisor is always 01
- The canines are always 04
- The first molars are always 09

**Anatomical direction:**
- Tooth surfaces that touch the front lips – labial
- Tooth surfaces that touch cheeks – buccal
- Tooth surfaces that touch palate – palatal
- Tooth surfaces that touch tongue – lingual
- Anterior portion of a tooth – rostral
- Posterior portion of a tooth – caudal

So, once you have performed the complete visual oral assessment, it is time to start making notes on the dental chart. The purpose of a dental chart is to make record of the state of the mouth on that day. A veterinarian that was not involved in the procedure at all should be able to understand exactly what condition each tooth was in just by looking at this chart.

Often I have gone to practices that state they chart their dentistry. In fact what they do is circle all missing teeth and “x” out all extracted teeth. Complete charting involves much more than that. There should be adequate room to make notes as to signs,
diagnosis, treatments, prescriptions and take home instructions. An anatomical graphic showing every expected tooth in that species should be present and large enough that you can make notations of periodontal probing depths on at least two surfaces.

**Periodontal probing**

Since the statistic is that 70-85% of all companion pets over the age of 3 have periodontal disease, we need to make notations as to the pocket depth on each tooth. Without these numbers, there is no way that we can follow the progress of the therapy.

There are a number of periodontal probes available. I find that it is easiest for measurement of pocket depth is you choose a Williams periodontal probe. This instrument has markings at each mm. There is a heavier band at 4-5mm, 9-10mm, 14-15mm. This instrument is positioned parallel to the crown and gently guided under the sulcus of the tooth until the tip reaches the ceiling or the floor of the pocket. The intention of the technician using this instrument is to detect and measure periodontal pockets and clinical attachment loss. At the very least measurements should be recorded at the deepest pocket depth on the mesial and buccal aspects of the teeth and the lingual and palatal aspects of the teeth. Any pocket depth greater than 1 mm in a cat or 3mm in a dog is considered a periodontal pocket.

Other critical notations are tooth fractures; enamel fractures, uncomplicated crown fractures, complicated crown fractures, uncomplicated crown root fractures, complicated crown root fractures and root fractures. The classification of these fractures can be found at www.avdc.org.

An explorer is the very pointed tipped instrument used to enhance tactile sensation. This instrument allows the technician to detect any abnormalities in enamel integrity. The sharp end will transfer a change in feel when in contact with tooth resorptions, enamel hypoplasia and carious lesions.

**Other gross clinical observations**

All other abnormalities should be noted:
- Discolored teeth
- Fractured teeth
- Mobility
- Furcation exposure classification
- Tooth resorption classification (www.AVDC.org)
- Fistucae
- Crowding
- Tooth rotation
- Abrasion versus attrition
- Enamel defects
- Foreign bodies
- Oral masses
- Supernumary teeth
- Stomatitis

A very comprehensive list of appropriate abbreviations can be downloaded from http://www.avdc.org/traineeinfo.html.

Intraoral radiographs are taken and those findings associated with each tooth should be noted on the record. Once the veterinarian has made a diagnosis and treatment plan, this is shown and noted on this chart as well.

As you progress in increasing your dentistry skills, there will be more and more things diagnosed and different treatment options will be offered and provided. This document will be your way of providing a means of clear communication for individuals within your practice and to those you are referring care.

The standard of care expected by the state boards in relation to dentistry is increasing every year. AAHA standards also make it clear that good record keeping and charting for dentistry services provided is expected. Since the down turn in the economy has hit dentistry services and surgery services hard across the country, we should look at this opportunity as a “speed bump”. Speed bumps are provided in order for us to slow down and evaluate the current conditions. This is an excellent opportunity for us to take this skill to the next level.
A current trend in veterinary medicine is a reduction in elective surgeries in small animal practice. Spays and neutering procedures are being done earlier and earlier at the rescue or at low cost clinics. That change has affected the small animal practice dramatically which has focused much attention to the mouths of our patients.

Small animal general practitioners are looking towards dentistry as a way of increasing wellness care while supporting the operating and treatment room activities. With an increased focus, there is an increase of the number of dental procedures being performed in practice and this necessitates us to look at both equipment maintenance and technician safety.

There is nothing more frustrating than equipment failure during a procedure for both the veterinarian and the technician. Therefore, it is important to schedule some time during the week that will be devoted to equipment maintenance.

**Dental unit compressor maintenance**

Some dental units come equipped with a small compressor. Some practices have a compressor outside of the dental department and the units are connected with quick-connections. In either case:

- Oil-cooled compressors have either a view port or a dipstick with which to monitor the oil level. This should be checked weekly. Consult your compressor’s owner manual to determine the type of oil required.
- Condensation also accumulates in the barrels of the compressor. There are drains (either wing nut type or screw type) at the bottom of the compressors. These air storage tanks need to be drained weekly for busy dental departments and monthly for smaller departments.

**High speed handpiece maintenance**

**After use**

1. Remove the handpiece from the dental unit tubing.
2. Wipe the outside of the handpiece with a clean gauze or paper towel moistened with water or alcohol. If you have a handpiece equipped with a fiber optic light source, make sure that is wiped clean as well. Do not use harsh cleaning solutions and do not vibrate in the ultrasonic cleaner.
3. With the bur in place, spray a short burst of special handpiece lubricant (refer to manufacturer’s owner’s manual) into the air drive hole. This is usually the smaller and often shorter of the holes.
4. Reattach the handpiece to the dental unit tubing and depress the foot pedal for 30 seconds allowing the lubricant to circulate through the handpiece and to expel any excess oils from the air line. Allow the lubricant from the handpiece to discharge onto a paper towel and inspect for color. This should all be clear. If not, repeat the lubrication process described above until it is clear.
5. Remove the old bur.
6. Dry the exterior of the handpiece thoroughly (any excess oil will soak through the autoclave pouch, disrupting instrument sterility and will risk paper char).
7. Follow manufacturer’s owner manual for autoclave time and pressure (Never exceed 135 degrees C).

**Before use**

1. Place a new or sterilized bur in handpiece. Secure in appropriate bur into the chuck of the handpiece and finger tighten the chuck closed around it by mounting the Chuck Wrench or by releasing the Push Button on the end cap of the Push Button Type handpiece.
2. Spray lubricant into drive air hole.
3. Allow handpiece to run for 20-30 seconds.

**Cartridge replacement**

1. If, after lubricating the high speed handpiece, there is excessive drag (the handpiece is not spinning with adequate RPM), it may be necessary to replace the turbine cartridge.
2. A bur should be in place.
3. Use the manufacturer’s end cap wrench to remove the end cap turning the wrench counter-clockwise.
4. Gently push the turbine out by pushing gently on the bur.
5. Remove debris from the turbine from the inside of the handpiece with a cotton tipped applicator.
6. Insert a new turbine into the head of the handpiece by aligning the locating pin to the guide dot on the head.
7. Make sure the back of the cartridge sits flush with the back of the handpiece.
8. Secure the end cap back in place with the end cap wrench.
Low speed handpiece maintenance
1. If your low speed handpiece has a motor section with a detachable sheath, the motor does not need to be sterilized.
2. Slide the attachment ring up to detach the sheath.
3. Dental motors and sheaths require a higher viscosity oil than high speed spay.
4. One to two drops of oil in the drive airline is all that is necessary.
5. Attach the motor to the drive airline and run to distribute the oil.
6. Wipe away the excess with a paper towel.
7. The straight sheath does not require lubrication.
8. Clean the outside with a moist gauze or paper towel and dry.
9. Place in a sterilization pouch and sterilize.

Disposable polishing angle
1. It saves maintenance times because you simply throw it out.
2. No cross contamination.
3. 90 degrees reciprocating head
   a. Does not wind into long hair of some animals.

Autoclaveable prophy angle
1. Dip the head of the prophy angle in a small amount of handpiece cleaning solvent.
2. Run for 1 minute changing directions of the gears from forward to backward.
3. Wipe off and insert prophy cup.
4. Periodically follow the manufacturer’s instruction in the owner’s manual and disassemble the prophy angle to oil the gears.

Hand instrument sharpening
1. Put a drop of sharpening oil on an Arkansas Sharpening Stone.
2. Hold the dental instrument either against a firm surface at a 90 degree angle to the floor with the toe facing you.
3. Place the oiled sharpening stone at a 115 degree angle and move up and down until a sharp angle is obtained.
4. Wipe filings and excess debris off with a conical stone.

Winged elevator sharpening
1. Match the angle of the back edge of the winged elevator against the Oiled Arkansas Stone.
2. Hold the instrument steady.
3. Move the stone down the back of the instrument on the right side, then the middle then the left.
4. Use the conical stone on the inside of the winged elevator to remove filings.
5. If there are notches in the instrument left from improper extraction techniques or if the instrument has been bent, send it off for professional instrument care OR replace the instrument.

Operator/technician safety
Ergonomics
1. Maintain proper posture
   a. Upper back
      i. Your elbows should be at a 90 degree angle
      ii. Use magnification and good lighting to reduce the need to bend the neck and shoulders
   b. Lower back
      i. Adjust the height of the seat so that your feet are flat on the floor with your knees slightly lower than the hips.
   c. Hands
      i. Hold the instruments in a modified pen grasp
         1. Neutral position
         2. Relaxed position
         3. Stabilized hands when possible

Personal safety
1. Contaminants
   a. Two foot spray, splash and spatter zone
   b. Wear eye protection at all times
      i. Goggles
      ii. Safety glasses
      iii. Chin length full face shields

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c. Wear mask
   i. Have a filtration level of at least 95%
   ii. Minimize goggle fogging

d. Protective clothing
   i. Really should protect your skin and work clothes (CDC)

e. Gloves
   i. Right size
   ii. Allow for good tactile sense

2. Radiation
   a. 6 foot 8 inches from beam when barriers are no available
   b. Primary Barriers (needed when within the beam)
      i. Lead gowns
      ii. Lead curtain
   c. Secondary barrier
      i. Dry wall is considered an appropriate secondary barrier
   d. Dosimeter
      i. Collar level
One of the biggest revelations for me in my training as a veterinary technician was that cats are not small dogs. That statement is especially true in relation to the subject of veterinary dentistry.

As with most other subjects in medicine, we must start with anatomy and the differences unique to our feline population as a species.

Unique anatomy

Tongue
The tongue is covered with papillae. These barbs point backwards and this enables the cat to rasp meat off of bones and to groom more effectively. Theses barbs catch debris and dirt while grooming. Unfortunately, because the barbs on a cat’s tongue points backwards, anything that gets attached to these barbs eventually gets swallowed. That includes things like hair and string. Hairballs are a common issue for cat as are string foreign bodies. Once tangled in the papillae, the material cannot be spit out.

Teeth
Cats have a unique dental formula. Theirs is I3, C1, P3, M1
I3, C1, P2, M1

Their canine counterparts are supposed to have 4 premolars and more than one molar. So, it is important to know that when charting the feline patient, their teeth on the maxilla start at the second premolar (there is no first) and on the mandible, the first tooth distal to the canine is the third premolar (there are no first and second mandibular premolars).

Gingival probing depths
In our feline patients, normal gingival probing depths are less than 1 mm.

Canine teeth
The canines have a vertical grooves that extends the length of the tooth. These sometimes become stained. It is important to note that the enamel is thinnest in this area and care must be taken not to spend too much time trying to remove the staining for fear of damaging the protective enamel.

Also, the pulp canal extends ALL THE WAY to the tip of the crown of the feline canine tooth. Any degree of chipping of these teeth require investigation. A “wait and see” approach is not appropriate. These injuries must be radiographed. Nearly all fractured canine teeth are painful and will become infected.

Common feline pathology

Juvenile onset gingivitis
This occurs before cats are 9 months old. There is a severe gingivitis and notable halitosis. Often these cats little to no tartar accumulation. The exact cause is unknown but treatment involves early detection and frequent (q 4-6 months) professional plaque removal with elegant home care. Usually, true juvenile onset periodontitis will resolve by the age of 2 years. It is often noted that juvenile onset gingivitis occurs frequently in specialty cats such as Abyssinians and Persians.

Canine tooth extrusion (supereruption)
Sometimes when a canine tooth is effected by chronic periodontal disease, there appears to be a greater crown height. Although the etiology is unknown, current studies reveal a statistical correlation between supereruption and tooth resorptive process.

It is important to note; when the veterinarian is making the recommendation for the extraction of a maxillary canine tooth when the lower canine tooth on the same side remains, it is critical that the client be prepared that there is a chance for maxillary lip impingement.

Alveolar bone expansion (osteitis)
Caused by chronic periodontal inflammation the alveolar bone around the upper canine teeth. When noted it is critical to evaluate the periodontal structures of these teeth radiographically to stage the periodontal disease in order to know the appropriate therapy. By Stage 4 periodontal disease and 50% bone loss, tooth extraction should be considered.

Tooth resorption
Tooth resorption is a common ailment in our feline population. Studies have reported anywhere from 20% to 75% of the feline populations will experience this disease process. These lesions are usually noted buccally but can occur on any surface. Statistically the most commonly affected teeth are the mandibular third premolar. On the cheek teeth, the lesions are commonly noted at the cementoenamel junction and with hyperplastic gingival tissue covering the affected portion of the tooth.

In the canine teeth, it is common to see tooth resorption more apically and may not be clinically apparent.

At one time these lesions were called neck lesions, FORLs (Feline Odontoclastic Resorptive Lesions) and cervical line erosions. The current terminology is tooth resorptions. They are classified:
• Stage 1: Mild hard tissue loss
• Stage 2: Moderate hard tissue loss but that does not extend endodontically
• Stage 3: Moderate hard tissue loss but that does extend endodontically but most of the tooth integrity is maintained.
• Stage 4: Extensive hard tissue loss that extends endodontically
• Stage 5: Crown has fractured off due to hard tissue weakness and the gingiva has extended over top of the remaining root tissue.

The etiology of this disease process is unknown and being researched extensively. Recent studies have grouped these lesions into two groups:

• Type 1: Resorption associated with periodontal disease where the tooth retains radiographic evidence of a periodontal ligament and a pulp canal. These teeth need to be treated with complete extraction.
• Type 2: Lesions associated with bone replacement. These teeth can be treated with crown amputation with intentional root retention.

Determination of treatment options can only be made radiographically.

**Stomatitis**

This is one of the most painful conditions for cats and definitely one of the most frustrating syndromes to deal with for the owner and the practitioner.

The etiology is unknown specifically but appears to be associated to an immune mediated reaction to either dental plaque or the tooth structure itself. Usually there are many factors involved; genetics, environmental stress, diet and viral infections. Although, for a while it was suggested that feline Bartonella virus was responsible for stomatitis cases. However, although many cats may be infected with Bartonella, it is more probably an opportunistic pathogen ad not the primary cause.

These cats present painful. They often drool, have a history of weight loss and poor appetite and their coats are unkempt due to their inability to groom themselves comfortably. Some cats only have inflammation around the caudal cheek teeth, while others exhibit a generalized inflammation.

This disease is very frustrating. Medical therapies such as corticosteroids, antimicrobials, Gold Salts, Interferon all have mixed long-term results.

When feline patients present with inflammation associated only with the teeth caudal to the canines, surgical extraction of the teeth and all root fragments caudal to the canines is the treatment of choice. It is recommended that if one is to treat a stomatitis case, they must have intraoral radiology capability. It is critical that all root tissue be removed as well. The accidental retention of root tissue will lead to continued gingival inflammation. Since in the best case scenario, extraction can provide a 50% chance of resolution, post-op radiographs are vital to insure that the case has been properly treated.

Patients also should be supported via feeding tube if anorexia is an issue until they are able to eat on their own.

Post-operatively most veterinarians will put these patients be on appropriate antibiotics for 2 weeks post-op with appropriate pain management.

In summary, feline patients have some unique issues that the trained veterinary technician should be aware of in order to support the veterinarian and the owners.
Are dental radiographs essential for professional veterinary dental care? Absolutely! In practices that use radiology to evaluate dentistry patients with obvious clinical findings, radiographs revealed additional pathology in 50% of dogs and 53.9% in cats. In cases that had no gross pathology present, radiology exposed clinically relevant findings in 27.8% of dogs and 41.7% in cats. So, when practices are providing dentistry without evaluating the health of the tooth below the gum tissue, they are missing a vast amount of disease.

To provide this service, a dental X-ray unit is not necessary. For some years, vet dentists used their regular medical X-ray unit successfully. The key is to use intraoral film. This task is awkward and time consuming. Often it means transporting the anesthetized patient to a totally different room in the practice. But, it can be done.

Many veterinary hospitals site cost to the clinic as the number one reason for not purchasing a dental radiographic unit. Unfortunately, they are mistaken. Of all pieces of equipment in a practice, this unit is relatively inexpensive. To purchase a regular dental X-ray unit, the cost would amount to about $4,000. If the practice goes digital, software and sensor can cost from $6,000 to ~$9,200.

A cost analysis is valuable when evaluating the profitability of equipment. Let’s say, for example, a practice is performing 3 dentistry per day. On the average they take (and this is very conservative) 10 radiographs per day. The average fee is $10-20 per view, so let’s split it in the middle…$15 per radiograph. That produces $150 per day. Do these 5 days per week; the practice generates $750 per week. Do these 50 weeks per year, the practices produces $37,000 per year. The equipment paid for itself in less than 6 months just on the revenue brought in by the images itself. I haven’t included the increased pathology found and the revenue generated by treating it. These numbers are very reasonable in a large, multi-vet practice.

Consider a small, 2 vet practice. Let’s imagine they perform 3 dentistry per week. They take 10 radiographs per week at $15 each. That is $150 per week. Do these 50 weeks a year and the practice has grossed $7,500 a year. The equipment in that scenario paid for itself in less than 2 years. After the equipment is paid off, except for incidental supplies, the rest is all profit.

To use a medical X-ray unit, it is preferred that the head of the unit can be lowered and the angle changed. A focal distance of 12 inches is best. To be able to use the bisecting angle technique is often necessary to reposition the patient. Different X-ray units have different technique charts but you can try using 100 mA, 65 kVp at 1/10th second and adjust the technique accordingly.

Dental radiographic units have heads that are more adjustable so that the patient does not have to be manipulated and repositioned as much. The radiographic detail is much better.

So, once a practice decides it is interested in providing this service, some training and education is required. Fortunately, there are many venues for this education. There are numerous training facilities across the country; there are convenient online courses, many wonderful books and journals. Recognizing normal versus abnormal requires some knowledge of each.

Safety is also an important factor. It certainly is a fact that digital radiographs require about 1/10th of the radiation required when exposing film. But, that doesn’t mean that one shouldn’t prudently provide radiation protection. Stay 6 feet away from the head of the X-ray unit, do not stand directly in the line of the beam, do not hold the sensor with your hand, and always wear your radiation badge.

There are hand-held X-ray units available. These are often sought because they do not take up a large footprint within the dental operatory. But, their approval is provided for use at an arm’s length. These units are heavy and that may be difficult.

Once you have obtained the equipment and you have training in getting diagnostic images, it is important to begin to understand the baseline for normal versus abnormal tooth development and pathology. I recommend the following book when you are first getting your feet wet in this service:

Atlas of Dental Radiography in Dogs and Cats, 1e by Gregg A. DuPont DVM FAVD DAVDC and Linda J. DeBowes DVM MS DACVIM DAVDC (Jul 25, 2008)

In a normal, young patient, it is important to know that the dentinal wall is very thin and the pulp chamber is wide. As the patient ages the dentinal wall thickens hence the pulp canal narrows. Also, in very young animals, the apex of the tooth is still open. As they age, the apex closes.

Indications for radiographs are vast:
- areas where there are missing teeth
  - Impacted teeth often cause dentigerous cysts. As the tooth is developing, there is a sac of epithelium that covers the crown of the tooth. During eruption through the gingiva, the sac is lost. If the tooth is embedded,
the sac remains and often begins to secrete fluid. As the fluid accumulated, a cyst forms. As the cyst enlarges, the surrounding bone is destroyed.

- to evaluate teeth experiencing periodontal disease
  - It is not enough to clean a periodontal pocket without radiographing the tooth to evaluate bone loss and whether or not the patient has secondary endodontic disease.

- to document destruction caused by tumors, including epulides
  - when evaluating tumor or epulide activity, radiographic findings should accompany the biopsy so that the pathologist can consider this in his/her diagnosis

- to evaluate discolored, worn or fractured teeth
  - Is there endodontic disease and what are the options for care?

- prior to extraction
  - What are the roots like?
    - Sometimes there three roots on a two rooted tooth
    - Sometimes the root is already fractured or resorbed
    - Sometimes mandibular molar roots curl at a 90 degree angle
    - Sometimes mandibular molar roots cross

- Are the roots ankylosed?
  - No point in trying to take out a root if it has been incorporated into bone (usually feline)

- What is the quality of the remaining bone
  - Is there sufficient bone to safely extract a tooth without fracturing the jaw?
  - Is there already a jaw fracture?

- post extraction
  - this documents complete root extraction
  - it documents the remaining integrity of the bone

- facial trauma

In summary, to provide complete and professional dentistry that will keep your patients happy and healthy, radiographic evaluation is very necessary. Extracting a tooth without radiographs is the same as repairing a femoral fracture without radiographs.

Some specialists feel that by the end of the decade, dental radiographs will be standard of care in veterinary medicine. Our clients are becoming ever so much more sophisticated in their expectations for their veterinary care. This is everyone’s opportunity to be ahead of the curve rather than trying to play catch up.
Periodontal Disease: The Most Prevalent Disease in Veterinary Medicine
Vickie Byard, CVT, VTS (Dentistry), CVJ
PetED Veterinary Education and Training Resources
Warminster, PA

Peri- : Prefix meaning around or about
-odont: having to do with tooth
-iium: indicates a biologic structure

If we use this information, it means that periodontal disease is a disorder of structure having to do with the tissues that surround and support the teeth, including the gums, cementum, periodontal ligament and alveolar and supporting bone.

The structures of a tooth
- Enamel: hard white substance covering the crown of a tooth
- Dentin: the main boney part of the tooth beneath the enamel and surrounding the pulp chamber and root canals
- Gingiva: the gums of the mouth. The gingiva is made up of epithelial tissue that is attached to the bones of the jaw and surrounds and supports the bases of the teeth.
- Gingival sulcus: the groove between the surface of the tooth and the epithelium lining the free gingiva.
- Free Marginal Gingiva: the portion of the gingiva that surrounds the tooth and is not directly attached to the tooth surface.
- Attached Gingiva: the portion that is firm, resilient, and bound to the underlying cementum and alveolar bone.
- Cementum: A bonelike substance covering the root of a tooth.
- Alveolar bone: The alveolar process is the thickened ridge of bone that contains the tooth sockets on bones that bear teeth.
- Periodontal Ligament: the fibrous connective tissue that surrounds the root of a tooth, separating it from and attaching it to the alveolar bone, and serving to hold the tooth in its socket.
- Furcation: the space between two roots

Periodontal disease is the most prevalent medical condition affecting our dogs and cats. As a matter of fact, it is suggested that most pets over the age of three years of age are experiencing some level of periodontal disease.

Bacteria in the mouth form a thin, slimy film on the teeth, otherwise known as biofilm. When that biofilm covers the teeth, it is called plaque. If the plaque is not removed, the minerals in the saliva join with the plaque and harden into a substance called tartar or calculus. The bacteria secret toxins and that sets off an inflammatory response. This is the primary cause of periodontal disease.

There are stages of periodontal disease
- Normal: Clinically normal. No inflammation evident
- Stage 1 PD (periodontal disease): Gingivitis without any attachment loss
- Stage 2 PD: Early periodontal disease. There is less than a 25% attachment loss and/or a stage 1 furcation involvement.
- Stage 3 PD: Moderate periodontitis: There is a 25-50% attachment loss and/or a stage 2 furcation involvement.
- Stage 4 PD: Advanced periodontitis. There is a greater than 50% attachment loss and/or a stage 3 furcation involvement.

Periodontal disease is much more than just an aesthetic issue for pets and their owners, although the odor may be the client complaint necessitating the visit. Periodontal disease can lead to oral discomfort, as well as tooth loss. It has also been strongly documented in human medicine a link between periodontal disease and numerous problems such as an increased risk of stroke, myocardial infarction, atherosclerosis and difficulty regulating diabetes due to the inflammation. It has also been suggested that people over 60 years of age may suffer from delayed memory as well. We now have studies in dogs showing a correlation between periodontal disease and microscopic changes in heart, liver and kidney tissue.

The periodontal patient needs first to have a thorough assessment, cleaning, and charting to determine the degree and severity of the disease process. Radiographs are also needed to determine if there are any teeth endodontically challenged secondary to the periodontal disease. A critical piece of the puzzle is determining the ability and willingness of the owner to provide care at home. Although there are treatments available, if the owner is not willing to provide meticulous home care, severely affected teeth should be extracted.

In the event you have an owner that is motivated to do home care, treatment options are:
Root planning and subgingival curettage

When periodontal pockets have been identified, it is imperative that the plaque and calculus be removed from the root surface. Ultrasonic and sonic hand pieces can be used hasten the work, since our veterinary patients are under general anesthesia. The scalers actually vibrate at a frequency that breaks down bacterial cell membranes. This does hold a therapeutic advantage. However, the tips do not provide the same horizontal flat surface as hand instruments do. Therefore, it is recommended to follow ultrasonic pocket treatment with engaging the curette with the root surface and pull with a downward motion in a cross hatch fashion.

The goal of root planing is to scale the root. Since the cementum is softer, it is more affected by tartar build up and inflammatory by-products. So root planing removes the roughened cementum, impregnated with toxins.

Care needs to be taken not to be overly aggressive in planing. Cementum itself does contain substances that augment attachment.

The pocket itself needs to be treated as well. A curette is used to debride the diseased tissue from the pocket, leaving a healthier tissue bed for healing and reattachment.

Perioceutics

These are products that are employed to provide a medicant to the disease periodontal pocket.

Doxirobe gel (zoetis)

This is a doxycycline polymer preparation that comes as a two syringe system. The polymer syringe mates with the antibiotic syringe. The plungers are depressed in a back and forth motion 100 times. A blunt cannula is attached and can be bent to whatever angle is most appropriate. The gel is introduced into the treated periodontal pocket (greater than 3mm deep). A few drops of water on the gel and the matrix harden. A plastic filling instrument or titanium covered beaver tail instrument is used to pack the material into the pocket. This will remain in place for 2-3 weeks. Another advantage of Doxycycline is that it has an anticollagenase effect. This aids in tissue reattachment.

Clindoral (TriLogic pharma)

This is a preloaded syringe system that comes ready to use. Attach the blunt cannula and with the head of the pet upright, instill the Clindoral filling the pocket. Hold the head in the same position for 1-3 minutes for complete gelation. An instrument can be used to pack the material. The material slowly resorbs over a 7-10 day period.

Consil and osteoallograph

Guided Tissue Regeneration. Both products are synthetic bone graft materials. This is a more advanced procedure and referral to a dental specialist may be indicated if your veterinarian is not familiar with this product. This is a material that is most effective for areas where there has been vertical bone loss. This procedure is a surgical procedure necessitating a surgical flap. Care must be taken to rule out any oral nasal fistulae or antral nasal fistulae. Otherwise, these materials will migrate into the sinuses and will be very irritating for the patient.

Systemic antibiotic use

When providing periodontal therapy, these sites are considered “open and draining”. Treatment by combining scaling and extraction is not an indication for systemic antibiotic treatment. There are, however, some specific indications for adding a systemic antibiotic:

- When local tissue is severely infected and periodontal therapy required surgery to expose bone or if teeth were extracted from severely infected bone.
- Osteomyelitis
- CUPS (chronic ulcerative paradental syndrome): mucosal immunopathy
- Prevention of bacteremia in specific cases
  - Patients with clinically evident cardiac disease
  - Patients with clinically evident renal or hepatic disease
  - Patients with prostheses; ocular, total hip replacements, patients with anterior cruciate repairs using nonabsorbable material
  - Patients with splenectomies
  - Patients with clean surgical procedures with severe periodontal disease
  - Chemotherapy patients
  - Patients with concurrent auto-immune disease

Home care

When periodontal therapy is provided, it is critical to provide explicit home care instructions:

- List all dispensed medications and when should the client begin the medications
- When may the patient eat next and what may they eat?
- When may the client resume or begin tooth brushing?
- When is their recheck appointment?
• When do you want to schedule the next dentistry? This is influenced by the client, the size and breed of patient, the budget of the client, etc.

Periodontal disease is the most common condition in our companion pet population. Prevention is the gold standard and that includes owners brushing their pet’s teeth…daily. Probably one of the most important activities the technician plays is in educating the client on how to maintain a healthy mouth.
As in many aspects, veterinary medicine lags woefully behind our human medicine counterparts. We, as a profession, are slow to adopt change; nowhere more importantly than in our ability or desire to address the profound personal needs of our colleagues. At an April 2012 meeting at the Betty Ford Center, the statement was made that statistics from the Centers for Disease Control and Prevention show that drug overdose has now surpassed automobile accidents as the leading cause of accidental or injury death in the country. It is estimated that 30,000 Americans will die from prescription drug overdose this year.

The reason that this became an important issue for me was that as of 2008, I began a 12-step program of my own. I live in a moderate sized town. Once I got entrenched in this program, I was alarmed at the number of professional and paraprofessional colleagues employed in veterinary medicine I met that were also dealing with an addiction of one sort or another. I began to think. Now, I am not much of a mathematician and I am certainly not a statistician but I began some simple math.

If I know 6 colleagues from one town in Pennsylvania, and there are 2,563 municipalities in PA then there may be over 15,000 colleagues in one state alone. Multiply that times 50 states…well, the numbers don’t add up considering there were only 90,000 some veterinarians employed in the US and about 71,000 veterinary technologists and technicians. But it made me aware of a serious problem within my profession. And, these are the numbers of colleagues that have found some sort of help. Statistics show that the number of people still struggling with addictive substances always outweighs those that find recovery.

So, is there a problem in veterinary medicine? Well, I think we need to look at the suicide rate of veterinarians. In March of 2010, Bartram published a paper in the Veterinary Record showing that our British colleagues are faced with a higher risk of suicide as compared to the general population and double the risk of other health care professions. What are some of the factors suggested in that paper?

- Personality characteristics of those entering the veterinary field
- Work related stressors
- Ready access to drugs
- Comfort with the concept of euthanasia
- Stigma associated with mental illness
- Professional and social isolation

It only makes sense that if suicide is an issue within our profession, we need to be looking at one of the precursors to that and that is substance abuse.

The Centers for Disease Control and Kaiser Permanente produced the ACE study. ACE stands for Adverse Child Experience. 17,000 Kaiser patients voluntarily participated in the study and it produced staggering data suggesting that trauma during childhood development dramatically increases the health, social and economic risks. See www.ACEstudy.org. We joke about the fact that it is the wrong reason to go into veterinary medicine because you don’t like people. As an instructor, I have repeated to countless students that there is a person at the end of every leash or holding every cat carrier. But I have to wonder how many of us gravitated towards this profession for that very reason. Having assisted with the administration of hundreds of MBTI (Myers-Briggs Type Indicator) personality tests in veterinary medicine, I have noted a strong tendency towards introversion in both the veterinarian and the technician populations.

So, first let’s consider personality types. What type do you identify with? In the 1950’s these personality types were identified as risk factors for heart disease:

Type A: Impatient, hurries, under pressure, prompt and often early for appointments, watches clock, walk/talk/eat rapidly, does multiple activities simultaneously, lives in the future/always planning, feel that ‘there's never enough time’.

Type B: relaxes readily focuses on the quality of their life paces themselves easygoing ‘one day at a time’ less ambitious lower incomes/grades less irritable.

The Type A personality group experiences a significantly greater chance of having heart disease because they experience stress more strongly. Many of us in the United States have not been taught to deal with stress appropriately.

What is stress?

- A “Physical, mental or emotional strain or tension.”
- A “condition or feeling experienced when a person perceives that demands exceed the personal and social resources the individual is able to mobilize.”
A “Physical, chemical or emotional factor that causes bodily or mental tension and may be a factor in disease causation.”

A “state resulting from stress; especially one of bodily or mental tension resulting from factors that tend to alter an existent equilibrium.”

There are individuals that can experience a stressful experience and move into an adaptive stage. The flip side are those that move into an exhaustive stage. If one is not able to move to an adaptive stage, the exhaustive stage of stress the individual is sensitized or primed for future stressors!

To be able to handle the frequent stressors appropriately, we have to find ways to adapt in healthy ways.

- Experiencing the feelings without guilt or shame
- Sharing the unpleasant feelings with a friend or confidant
- Exercise/yoga
- Community
- Mindfulness/meditation

Unfortunately, some that find stress unbearable, resort to unhealthy ways to de-stress…drugs and/or alcohol. If, healthy stress relievers are not cultivated, addiction can be right around the corner.

So, what is addiction or chemical dependency?

- Loss of control - abuse of substance
- Need-dependency (physical and psychological)
- Continued use despite consequences
- Tolerance (need more of substance to get the same effect)
- Withdrawal (physical, psychological)

Why can some people drink and never have a problem and other struggle so?

- Biology - genetic predisposition (10% of cases)
- Psychological - "addictive personality"
- Social - family dynamics predispose
  - one parent with substance abuse: the child is 4-5 times more likely to abuse
  - both parents abuse: the child is very likely to abuse
  - Adult survivor of child abuse
  - PTSD: self-medication

And how do you know you have a problem?

- Have you ever decided to stop drinking for a week or so, but only lasted for a couple of days?
- Do you wish people would mind their own business about your drinking-- stop telling you what to do?
- Have you ever switched from one kind of drink to another in the hope that this would keep you from getting drunk?
- Have you had to have an eye-opener upon awakening during the past year?
- Have you had problems connected with drinking during the past year?
- Has your drinking caused trouble at home?
- Do you ever try to get "extra" drinks at a party because you do not get enough?
- Do you tell yourself you can stop drinking any time you want to, even though you keep getting drunk when you don't mean to?
- Have you missed days of work or school because of drinking?
- Do you have "blackouts"?
- Have you ever felt that your life would be better if you did not drink?

Many of our veterinary colleagues are dying of this. If you think you have a problem, there is plenty of help available. It is not hopeless.

- Twelve-step programs (free and virtually everywhere and online)
- Rehab
  - FMLA
  - Entitlement
  - Under the Family and Medical Leave Act of 1993 (FMLA), most Federal employees are entitled to a total of up to 12 workweeks of unpaid leave during any 12-month period for the following purposes:
    - the birth of a son or daughter of the employee and the care of such son or daughter;
    - the placement of a son or daughter with the employee for adoption or foster care;
- the care of spouse, son, daughter, or parent of the employee who has a serious health condition; or
- a serious health condition of the employee that makes the employee unable to perform the essential functions of his or her positions.
- Under certain conditions, an employee may use the 12 weeks of FMLA leave intermittently. An employee may elect to substitute annual leave and/or sick leave, consistent with current laws and OPM's regulations for using annual and sick leave, for any unpaid leave under the FMLA. (The amount of sick leave that may be used to care for a family member is limited.

- Intensive Out-patient Programs

I have seen people that have struggled with addiction lose their license and inability to get to work due to loss of their driver’s license. Getting your veterinary license back is very hard and expensive. It is so much easier to address the wounds, learn new coping skills and get honest.

We also have to create a pathway to health for our coworkers. Create a strong policy and make it safe to say; "I think I may have a problem". If substance abuse is stigmatized in our work places, our dependent co-workers will struggle in silence. It is important to remember; substance abuse is a disease. The addicted colleague deserves the same compassion you would afford one that had cancer or some less stigmatized disease.
All-natural, organic, corn-free, grain-free, gluten-free, raw, BARF, kibble, canned, semi-moist, freeze-dried, frozen; there are so many types of pet foods available today. How do you know which to recommend to your clients or to feed your own pets? This can be quite a controversial topic! Advocates of many diet types often adamantly believe their choice of diet is the absolute best, and some will passionately defend their beliefs, whether based on facts, anecdotal information, or misinformation. Let’s take a look at some diet option types, pros/cons, facts and misconceptions.

**Home cooked diets**

Some owners enjoy cooking for their pets; others feel they are providing superior nutrition, some believe their pet will not eat or will not enjoy commercial foods, others prepare limited ingredient recipes. Preparing home cooked diets for pets is often more time consuming and expensive than feeding commercially available options. There are countless books, websites, articles, and blogs with recipes for home cooking dog and cat foods unfortunately very few are actually complete and balanced. Clients that wish to prepare their pets food at home should be strongly encouraged to consult with a veterinary nutritionist to ensure the diet is nutritionally complete. Clients should also be counseled to follow the recipe exactly as formulated and not to substitute or eliminate ingredients.

Limited ingredient home cooked recipes designed for elimination trials can be very useful to determine if a patient is sensitive to a particular ingredient. These diets are intended to only be used on a short term basis under the supervision of a veterinarian as they are often not nutritional balanced. Once the ingredient the pet is sensitive to is identified the veterinarian will often recommend a complete and balanced commercially available diet or consult with a veterinary nutritionist to develop a complete and balanced home cooked recipe. Owners preparing these elimination diets must not make any changes to ingredients unless directed to do so by a veterinarian. These patients should also not receive any treats or foods other than those in the elimination diet. It is extremely important to stress to clients they must follow the recipe exactly as formulated and not to substitute or eliminate ingredients.

**Organic and all natural diets**

The term “organic” refers to the handling and processing of ingredients and products; it does not describe the quality of a product. In order to use this term on the label pet foods and treats must comply with the USDA's National Organic Program (NOP) regulations. These regulations include ingredient sourcing, ingredient handling, manufacturing, labeling & certification of products using the term "organic" on the product label. When natural is listed on a food label the food should not contain any chemically synthesized ingredients. If “natural with added vitamins and minerals” appears on the label the vitamins and minerals may be chemically synthesized. “Holistic”, “organic”, “human grade”, “premium”, and “gourmet” are purely marketing terms and have no legal definition.

**Corn free diets**

Corn has also been labeled by some owners/breeders as a cheap filler that causes allergies and is not well digested by dogs and cats. Actually corn provides a highly available source of complex carbohydrates and is an excellent source of linoleic acid (an essential fatty acid which is important for healthy skin), essential amino acids and fiber. Despite what some owners believe documented allergies to corn are quite rare. The ingredients that are most commonly associated to adverse reactions in dogs are beef, dairy products, and wheat; to lesser extent lamb, chicken egg, chicken, and soy. In cats beef, dairy products, and fish are the most common ingredients reported to cause adverse food reactions. Actual food allergies are estimated to be responsible for only 1% of skin disease. Food sensitivities often improve from a diet change but this is not due to a food allergy but rather factors such as increase digestibility of the new diet, fat, or fiber content.

**Grain free diets**

There have been no studies indicating grain free diets are superior or healthier for dogs and cats. While some grain free pet foods provide excellent nutrition others may not. Potatoes and tapioca, which have a lower nutritive value than grains, are often used in place of grain in these diets. In fact potatoes and tapioca have less protein and more sugar than corn or oats. Some owners mistakenly believe grains are common causes of food allergies however as discussed above this is incorrect. Another common misconception is that dogs and cats eating foods containing grains are at increased risk of developing diabetes; there is no evidence to support this belief.
Gluten free diets
Corn gluten, which is found in some pet foods, is often blamed for GI issues by some owners and breeders as associated with what they perceive as celiac disease. Celiac disease is rare in dogs and has been reported primarily in Irish Setters. In reality, pets with celiac disease react to the protein in wheat, rye, and barley not corn gluten. Gluten is the concentrated protein from grain after all the starch is removed. Corn gluten is actually an excellent source of protein containing greater than 60% protein and is highly digestible.

Raw, barf, freeze dried and frozen diets
Commercial raw diets are available in many forms with frozen and freeze dried being the most common. These are often marketed as complete; others are intended to be fed with additional supplements. The internet is also full of recipes for Bones and Raw Food or Biologically Appropriate Raw Food (BARF) homemade diets. Many of these advocate a formula of 60% raw meaty bones with the remainder of the diet consisting of green vegetables, eggs, milk, brewer’s yeast, muscle meat and organ meats, yogurt, sometimes with some grain and legumes. Meals are not expected to be balanced but rather in theory to be balanced overall by varying the ingredients from one meal to the other. Articles explaining “how to feed raw” quite often advocate feeding muscle meats, organ meats, bones, and fish, with or without vegetables, fruits, grains, herbs, vitamins, and supplements. While some dedicated pet owners will follow these instructions in order to provide this “over time balanced approach” many others, however well intended, will not. Many owners will begin to make substitutions or not continue to dedicate the time and effort this method of feeding may require.

Studies on bacterial contamination of raw foods found that 20% to 35% of raw poultry and 80% of raw food diets for dogs tested positive for Salmonella spp, and 30% of stool samples from dogs fed these diets were positive for Salmonella spp. Raw pet food diets have also tested positive for Esherichia coli and Yersinis enterocolitica. Healthy pets may not show clinical signs of illness when ingesting these bacteria however immunocompromised, the very young and the very old are at significant risk. The pathogens found in raw meat also pose a significant health risk to young children, the elderly and immunocompromised people. It is therefore extremely important, anyone preparing these diets, to practice proper hand washing and that all utensils, bowls, and surfaces used for the preparation of raw pet food diets are properly cleaned and disinfected. Raw meat may also contain parasites such as Echinococcus spp, Neospora caninum, Sarcocystis spp, Toxocara canis, Toxoplasma gondii, and Taenia spp. It is also important to note, the feces of pets consuming raw diets can contaminate the environment which may also pose health risks to people and other pets.

Client communication and education
What if the diet a client is feeding isn’t complete and balanced? Or if you do not agree with the type of diet a client has chosen to feed? How can you explain your concerns and educate a client that their perceptions are incorrect?

- Listen to the client’s reasons for feeding the diet in question and their concerns with other diet options.
- Ask open-ended questions to ensure you understand their viewpoint. Be careful not to sound or appear judgmental.
- Avoid making disapproving comments, using a negative tone of voice, facial expressions, and body language. If you are perceived as judgmental, condescending, closed minded, or unwilling to listen and consider their point of view or beliefs clients are likely to become defensive or simply unwilling to listen to your advice.
- Gently point out any misconceptions or false information offering references to the correct information.
- Some owners turn to alternative diets out of concern that their pet won’t eat more conventional diet options. These owners may express concerns of their pet not eating as the cause for not wanting to change diets.
- Some owners will not be interested in hearing your advice. Others may listen; perhaps discuss or debate the issue with you but not be willing to make any change to their feeding practices. Be respectful and don’t take it personally; making a nutritional recommendation does not ensure the owner will follow your recommendation.
- As with any medical advice; document the patient’s nutritional history and your nutritional recommendation in the patient record.

Nutritional recommendation
Remember to make a nutritional recommendation for every patient every time they present to your hospital. Consider the patients age, weight, body condition and muscle condition scores, life stage, life style, and any health issues. Recommend the diet you believe is the best for that particular patient but remember you may also need to consider the owners perception of what is the best diet for their pet. When you have done your best to educate your client but they insist on feeding a diet you do not think is best, consider if there is a way to improve the patient’s diet. Perhaps the client would agree to have a veterinary nutritionist evaluate the diet and if not nutritionally balanced suggest improvements. If they are feeding a grain-free diet that does not contain highly digestible ingredients perhaps you can help them find one that does. If corn free is all the client will consider, research the available options and recommend the one you believe is best suited for your patient. Suggest or provide a referral to a veterinary nutritionist for the client that wants to prepare a home cooked diet or a raw diet.
Remember the goal of a nutritional recommendation is to provide the patient with the best possible diet. If the client is not willing to follow your recommendation, what recommendations can you make to improve the patient’s diet? Be a nutritional advocate for your patients!

References
L. Freeman, et. al, Grain-free diets: An alternative option, but don’t dismiss the grains, 2012
Nasogastric Feeding Tubes:  
The Technicians Role  
Ed Carlson, CVT, VTS (Nutrition)  
IVG Hospitals, Inc.  
Woburn, MA

Feeding tubes are generally tolerated well by most patients and most feeding tubes are relatively easy to place. There are two types of feeding tubes that may be placed by technicians – Nasogastric (NG) and Nasoesophageal (NE) tubes. Nasogastric (NG) tubes are inserted into the nostril, through the nasal cavity into the esophagus, terminating in the stomach. Nasoesophageal (NE) tubes are inserted into the nostril through the nasal cavity terminating in the distal esophagus.

Nasogastric (NG) and nasoesophageal (NE) tubes are useful for patients who are unwilling to or are unable to eat but have normal GI function. Placement does not require anesthesia and usually can be done without sedation by using a topical anesthetic. They are used short-term, usually less than 14 days, and are sometimes used until the patient is stable enough to be anesthetized for a longer term feeding tube placement if necessary. NE/NG tubes are contraindicated in patients that are actively vomiting, comatose, do not have a gag reflex, or have nasal tumor / nasal disease. Since these tubes are generally quite small (usually 8 French or smaller) patients may be fed only liquid food. Complications associated with NE/NG tubes include epistaxis caused by nasal mucosa irritation; aspiration pneumonia should the tube become dislodged due to vomiting or regurgitation; and esophageal stricture (a condition which is rare).

Nasogastric (NG) and nasoesophageal (NE) tube placement

Materials needed for placement include:
1. Tetracaine or proparacaine
2. Sterile lubricant jelly
3. Appropriate sized tube
4. 2.0 or 3-0 nylon suture material
5. Scissors
6. 22-gauge needle
7. Permanent marker
8. Empty 6cc syringe
9. 6cc syringe of sterile water
10. E-collar

To insert the NG or NE tube:
1. Elevate the patients muzzle and apply a few drops of a topical anesthetic, such as Proparacaine Hydrochloride Ophthalmic Solution USO, 0.5% into the nostril you have selected for tube placement. Allow 5 minutes before attempting to insert tube.
2. While you are waiting, place a stay suture as close to the nose as possible.
3. An easy way to place a stay suture is to quickly insert a 22-gauge needle through the skin at the point where the wing of the nostril meets the fur. With the needle in place insert a 2.0 or 3.0 nylon suture through the needle from the beveled tip and out the hub of the needle. While holding the suture remove the needle. Tie a square knot closely to the skin but loosely enough to allow passing another suture under it.
4. Measure and mark the tube.
5. The NE tube measures to the 7th or 8th intercostal space. The tip of the tube will fall in the distal esophagus.
6. The NG tube measures to the last rib. The tip of the tube will fall either slightly before or in the stomach.
7. Apply sterile lube to the tube.
8. Holding the patients muzzle with your non-dominant hand begin inserting the tube with your other hand. Press the patient’s nose upward using the thumb of the hand that is holding the muzzle.
9. Once you have inserted the tube about the length of the patient’s muzzle, lower the patients head pointing its nose downward slightly. These motions should help guide the tube into the esophagus rather than the trachea.
10. Continue to advance the tube until you have reached the mark you made when measuring the tube prior to placement. The patient may or may not swallow during placement. Gently stroking the throat may encourage swallowing which may assist the feeding tube to more easily pass down the esophagus.
11. If the patient begins to cough at any time during placement, STOP, remove tube and try again. Lack of coughing DOES NOT ensure the tube is not in the trachea!
12. Attach a 6 ml syringe to the tube and check for negative pressure.

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13. If negative pressure exists, flush 5 – 6 ml of sterile water into tube. If the patient coughs the tube is in the trachea. If the patient does not coughplacement MAY be correct; confirmation with a radiograph is recommended.

14. Cap the tube with an injection cap and temporarily suture the tube to the stay suture leaving long tails to secure the tube in place once proper placement is confirmed.

15. Confirm placement with a single left or right lateral post procedural radiograph.

16. Once proper placement is confirmed, secure tube with Chinese finger trap.

   The Chinese finger-trap suture is perfect for feeding tubes; it continues to tighten if the tube is tugged. Pass a long piece of suture through the previously placed stay suture and secure with a square knot. Then begin the Chinese Finger trap pattern. The suture is wrapped around the tube, crisscrossed and tied into a square knot. Repeat a minimum of 5 crosses. For red rubber tubes each throw should be pulled tight enough to make a small indentation in the tube. For silicone tubes pull tightly however indenting these tubes may cause them to become occluded.

17. Secure the other end of the tube in a second location either on the cheek (the facial nerves run across this area of the cheek so the suture should go through the skin but be kept as superficial as possible) or on the top of the head. Tie a loose square knot near the skin then secure the tube with a second square knot.

18. This may also be done with the 22-gauge needle method described above. Smaller diameter tubes may also be attached in this second location with surgical staples.

19. The patient may require an E-collar to prevent tube removal.

**Beginning assisted feedings**

Before beginning assisted feeding it is important to determine the patient's Resting Energy Requirement (RER). RER represents the energy requirement for a normal animal, which is not fasted, at rest under thermoneutral conditions. Two common methods for calculating the patient's RER are:

\[
\text{RER} = 70 \times (\text{body weight in kg})^{\frac{3}{4}} \\
\text{RER} = 30 \times (\text{body weight in kg}) + 70
\]

The first equation calculates a more accurate caloric requirement. The second formula tends to overestimate the actual caloric needs of very small patients and very large patients. The general recommendation is to begin feeding $\frac{1}{4}$ of the patients total RER for the first 12 hours and, if well tolerated, to increase by $\frac{1}{4}$ the total RER every 12 hours until full RER is reached. If at any time the patient vomits, discontinue feedings until vomiting has resolved, reduce volume by $\frac{1}{4}$ when feeding is resumed, and increase volume more slowly.

**Nasogastric (NG) and nasoesophageal (NE) tube feedings**

Only liquid veterinary diets should be used for feeding through NG and NE tubes. CliniCare® Canine/Feline Liquid Diet, which contains 1 Kcal/ml, is a good option. Emeraid® Intensive Care HDN™ Feline and Emeraid® Intensive Care™ HDN Canine are powdered formulas which are mixed with water and are also a good option. The volume of water added can be varied dependent on the diameter of the feeding tube, however the caloric density is reduced as the volume of water is increased. Trickle feeding via constant rate infusion (CRI) is most often used for hospitalized patients, although these tubes may also be used for bolus feedings and to administer oral liquid medications. Tablets should not be crushed and administered via these small tubes.

A number of liquid diets designed for people are also available. These diets are typically less expensive than veterinary liquid diets however are nutritionally inadequate and some may contain ingredients that are inappropriate for dogs and cats. These human diets are especially inappropriate for cats as they are too low in protein, taurine, and arginine.

**Troubleshooting feeding tubes**

Flushing the tube well with water after each bolus feeding, or every 4 hours for CRI’s, will usually prevent the tube from becoming clogged with food.

Some patients may be irritated by the feeding tube and require a buster collar to prevent them from removing the tube. Some patients with NE or NG tubes may experience excessive sneezing. If this happens, apply a topical anesthetic; gently lift the patients muzzle upward and apply 2 or 3 drops into the nostril the tube is placed in.

If the tube becomes dislodged, which usually causes coughing and/or discomfort and distress:

- If via CRI, STOP IMMEDIATELY!
- If flushing causes coughing – DO NOT FEED! Alert the doctor. The tube may need to be removed or replaced.

**Discontinuing assisted feedings**

When the patient is voluntarily eating 60% of its RER, assisted feedings should gradually be reduced and discontinued when voluntary consumption has reached the patients full RER. Long-term feeding tubes (E & PEG) should not be removed until the patient has been eating its full RER consistently and maintaining its body weight for a minimum of one week or more.
References
Textbook of Veterinary Internal Medicine, 7th Edition, Stephen J Ettinger and Edward C. Feldman
Clinical Textbook for Veterinary Technicians, 6th Edition, Dennis M. McCurnin and Joanna M. Bassert
Nutrition for the Noncritical Hospitalized Patient

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An important area of patient care that is not always given much thought is the nutritional needs of our hospitalized patients. How do we decide what, when, or how much a patient should eat? How should we be documenting what our patients are eating? We all know there are some procedures that we normally recommend fasting but when might this be contraindicated? Should patients that are vomiting or having diarrhea be fed? What are some methods for getting our patients to eat? How long should we allow a patient to go without eating before we consider assisted feeding? The answer to some of these questions ultimately is the doctors’ decision but remember as veterinary assistants and veterinary technicians we are advocates for our patients!

If there are no feeding instructions on a patient treatment sheet discuss the nutritional plan with the doctor responsible for the case. Are there any foods this patient should not be fed? Is there a particular diet the doctor would prefer the patient ate? If the patient refuses to eat that diet may we try other options? If a patient in the hospital is to receive a medication, the treatment sheet clearly indicates what medication, how much, route of administration, and when it is to be administered. Why should diet and feeding instructions not be indicated in the same way?

Understanding our patients’ nutritional requirements, documenting nutritional information in the nursing notes and discussing this during technician rounds is a vital part of providing excellent patient care! Did Fluffy like to be petted while eating her meal? Did she like her food warmed slightly or prefer it cool? Some patients won't eat if they can't smell the food; heating can bring out the aroma. Some nauseous animals dislike a smelly food, cooling can mask some of the aroma. Did Spot prefer canned food or dry? Often we only offer canned food to patients, epically those that are not eating. But many animals prefer day, especially if that's what they normally eat. Did Lucky like chicken or beef? Is Max food aggressive and lunges if you try to remove his food dish when he is in his cage? Passing along this information from one shift to the next is so important!

Even a patient that is well nourished at admission can rapidly become nutritionally depleted during hospitalization. Patients that are already malnourished at admission and that require prolonged hospitalization are at risk for increased morbidity and mortality due to poor nutritional status. Therefore, we must be proactive to ensure that nutritional support of hospitalized patients is not overlooked.

Nutritional status should be thoroughly assessed before aggressive nutritional support is initiated. Is your patient well nourished, undernourished, or at risk for becoming undernourished? A nutritional assessment can help identify patients that could develop a nutrient deficiency without nutritional intervention. A nutritional assessment should include a complete history (including a diet history), a complete physical examination, a body condition score, and laboratory testing (i.e., a complete blood count, a chemistry profile, and urinalysis).

Calculating calorie requirements

How do you know if a patient is eating enough? There are many other formulas to calculate patient energy needs, some for growth, weight loss, weight gain, extremely active individuals, and resting energy requirement (RER). A good starting point for most of our hospitalized patients is to calculate their resting energy requirement (RER). There are several formulas to calculating this, I recommend using one of the following and that you use the one you are most comfortable with.

RER = 70 x (body weight in kg) ¾ OR RER = 30 x (body weight in kg) + 70

For example: Sandy weighs 44 pounds, 20.0 kg.

30 x 20kg = 600 + 70 = 670

Sandy’s RER is 670 kcal per day

Next determine the caloric concentration of the food you have chosen to feed your patient. (Or that your patient has chosen to eat!)

The amount of calories in any food can be easily found on the company website or product guide. The table below contains the calorie content of some of the foods we commonly use in the hospital.

<table>
<thead>
<tr>
<th></th>
<th>size</th>
<th>kcal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hill's® Prescription Diet® a/d® Canine/Feline Critical Care</td>
<td>5.5 oz</td>
<td>179</td>
</tr>
<tr>
<td>Iams® Veterinary Formula Maximum-Calorie™ Canine and Feline</td>
<td>6 oz</td>
<td>333</td>
</tr>
<tr>
<td>Hill's® Prescription Diet® i/d® Canine</td>
<td>13 oz</td>
<td>369</td>
</tr>
<tr>
<td>Iams® Veterinary Formula™ Intestinal Plus Low-Residue™ Adult Canine</td>
<td>14 oz</td>
<td>417</td>
</tr>
<tr>
<td>Iams® Chicken for Dogs</td>
<td>13.2 oz</td>
<td>427.5</td>
</tr>
<tr>
<td>Iams® Beef for Dogs</td>
<td>13.2 oz</td>
<td>424</td>
</tr>
</tbody>
</table>
Iams® Lamb & Rice for Dogs 13.2 oz 427.5
Hill’s® Prescription Diet® c/d® canned 5.5 oz 163
Hill’s® Prescription Diet® i/d® Feline 5.5 oz 161
Iams® Veterinary Formula™ Intestinal Plus Low-Residue™ Adult Feline 6 oz 169
Iams® chicken for Cats 3 oz 97
Iams® Turkey for Cats 3 oz 95
Iams® Beef for Cats 3 oz 97
Iams® Veterinary Formula™ Intestinal Plus Low-Residue™ Adult Canine dry per cup 259
Iams® ProActive Mini Chunks™ dry per cup 379
Hill’s® Prescription Diet® c/d® dry per cup 383
Iams® Veterinary Formula™ Intestinal Plus Low-Residue™ Adult Feline dry per cup 348
Iams® ProActive™ dry cat per cup 372

Once you know the number of calories in a can or cup of the selected food simply divide that number into the patients RER. For example:

One 13 oz. can of Hill’s® Prescription Diet® i/d® Canine Gastrointestinal Health contains 369 kcal. We determined Sandy’s RER is 670 kcal. Therefore, Sandy needs to eat 1.8 cans per day to meet her resting energy requirements. (670 divided by 369 = 1.8)

This amount is usually divided into multiple feedings per day. Healthy pets, hospitalized for a spay or neuter, for example, can be offered half this amount twice per day. Other patients, such as a post-op abdominal mass removal may require smaller feedings every 4 to 6 hours. For anorexic patients or those that have not been offered food for a period of time should this amount can be introduced slowly, starting with perhaps 10 or 15% of the RER divided into four or 5 feedings per day. If the feedings are well-tolerated, then the amount can gradually be increased by 20 to 25% the following day and so on until the full caloric requirement is met. It is important to introduce food slowly in anorectic patients to prevent refeeding syndrome. This syndrome was first described in people with the liberation of Holocaust survivors during World War II. It was found that, after prolonged periods of anorexia, aggressive overfeeding leads to severe metabolic imbalances, which in some cases could be fatal. For example, as phosphorus and potassium shift back into the intracellular space, severely decreased serum phosphorus concentration can lead to hemolytic anemia. Typically, this is a concern once phosphorus concentration drops below 2 mg/dl and, at this point. Potassium concentration should be monitored closely and supplemented as well. Ideally, phosphorus and potassium concentrations should be monitored daily in these patients for the first few days of feeding. It is also important to remember that liquid diets are often 75% water, so reducing the patients IV fluid rate may be indicated as feedings increase.

In general, it is recommended if a patient has not eaten for 3 days or if they are not able to tolerate eating their total caloric requirements for 4 or more days, more aggressive nutritional support must be considered to ensure adequate nutritional intake. These patients may require some type of feeding tube or parental feeding which we will discuss in another lecture.

Nutritional history
While you're taking a patient's history, ask the client the questions below regarding the pet's current diet. You may notice some of the questions appear to be similar. The slight variation in the way the questions are asked is meant to prompt owners to think about all the food offered to their pets. For instance, one type of large dental chew is 600 calories and another medium-size one is listed as 188 calories.

1. Has there been any recent history of involuntary weight gain or loss? If yes, how many pounds and during what time period? (Remember, acute, involuntary weight loss is more significant than weight lost over a long period.)
2. Any history of nausea, vomiting, or diarrhea? If so, how often?
3. Any changes in the pet’s appetite, urination, or defecation? If so, describe the changes and duration.
4. Does the pet have any difficulty chewing or swallowing?
5. Does the pet have any allergies?
6. Any recent change in the pet’s diet?
7. How many people, including children, reside in the household? Who is primarily responsible for feeding the pet?
8. Are there other pets in the household? If so, do they have access to one another’s food?
9. What kind of food does the pet eat? (Get specific information, such as brand and product name.)
Getting your patient to eat

If a patient has not eaten for 3 days or longer, nutritional support should be considered if it is not contraindicated because of vomiting, regurgitation, or megasoesophagus or because nothing can be given by mouth while the patient awaits sedation for diagnostic procedures or surgery. Before initiating aggressive nutritional support, try warming the food and using coax-feeding techniques (e.g., hand feeding; petting and/or talking to the patient in a soothing manner during feeding time). Some hospitalized patients may eat only when alone and undisturbed. Because cats are more likely than dogs to develop fixed food preferences, cats are more likely to eat a familiar food. Patients that usually eat dry food often ignore canned food, and patients that usually eat canned food often ignore dry food. Therefore, it is always important to obtain a complete diet history from the client so that the patient’s food preferences are known before feeding is initiated.

I'm not a fan of syringe feeding. I find most animals hate it and find it difficult to force patients to ingest enough food using this method. However some doctors will request a patient be syringe feed and some patients may tolerate it. However if the patient protests I would discontinue the syringe feeding and suggest a different nutritional plan. If, at any time, the patient shows signs of nausea (e.g., turning away from the food, lip smacking, hyper-salivating, regurgitating, vomiting, trying to bury the food) or food aversion, coax feeding should be stopped and administration of antiemetic drugs should be considered.

When used appropriately, appetite stimulants can be useful; when used improperly, they can be very harmful. The misuse of appetite stimulants can create a learned food aversion if the patient is forced to eat before being ready. The patient should be thoroughly evaluated before the decision is made to use an appetite stimulant. An appetite stimulant may be considered if the patient is otherwise healthy and blood work results confirm that the patient has fully recovered. Diazepam, a benzodiazepine tranquilizer, is sometime used as a short-term appetite stimulant, especially in cats. Adverse effects of the drug include sedation and, sometimes, agitation and hyperactivity; rare cases of liver failure have been reported in cats after repeated use. Cypbroheptadine can have antihistamine and antiserotonin effects in cats. The drug is administered orally and may not be effective for up to 24 hours. Adverse effects reported by owners include hyperactivity, agitation, and aggression. Mirtazapine, a tetracyclic antidepressant, appears to be well tolerated in dogs and cats. Reported adverse effects include sedation, vocalization, hypotension, and tachycardia. Patients with renal disease or hepatic disease should be closely monitored. Antidepressants may affect the blood glucose concentration, so patients with diabetes mellitus should also be closely monitored.

Traditionally, dogs and cats with acute vomiting and diarrhea due to dietary indiscretion have been managed by withholding food for 24 to 48 hours for “bowel rest.” Fasting, even for this period of time, decreases the length of the intestinal villi, increases the risk of bacterial translocation, and reduces activity of intestinal disaccharide enzymes. In addition, the bowel does not necessarily “rest” when empty: during fasting, dogs experience migrating motility complexes or “housekeeping waves”; cats experience a similar motility pattern. During inflammation, normal motility is likely decreased and ileus and delayed gastric emptying are present.

“Feeding through diarrhea” (i.e., feeding while a patient still shows clinical signs) may help maintain the activity of small intestinal digestive enzymes and help preserve normal villi morphology. The presence of food in the intestine also decreases the risk of bacterial translocation. Further, feeding small amounts may improve intestinal motility and gastric emptying. In some cases of osmotic diarrhea, feeding worsens clinical signs; therefore, patients should be treated individually and feeding stopped if diarrhea appears to worsen.

When intractable vomiting is present, oral intake of food should generally be avoided, but for as short a time as possible. In dogs infected with parvovirus, early enteral feeding results in faster resolution of vomiting and diarrhea than does withholding food. Similar to feeding during diarrhea, feeding small amounts may improve gastric emptying and a return to normal motility. Highly digestible foods with a low to moderate fat content should be considered because high-fat diets may slow gastric emptying and promote vomiting in some patients. Parenteral fluids and electrolytes should be provided as needed, and antiemetics should be used if the presence of a GI foreign body has been ruled out.

If there is a history of dietary indiscretion and the patient is still eating and not clinically dehydrated, the diarrhea usually resolves with time. Switching to a highly digestible, low-residue food for 5 to 7 days may help the inflamed GI tract absorb nutrients while healing occurs. The animal can resume its regular diet after the diarrhea has resolved.
In many cases, however, conservative management is necessary, with restriction of food intake for 8 to 24 hours, followed by introduction of a bland, highly digestible diet fed frequently in small amounts. Failure to implement a conservative strategy is a common reason for poor response and relapse. Sometimes a secondary bacterial or fungal infection can develop, which may require medical therapy in addition to dietary manipulation. If a foreign body is involved, the introduction of a highly digestible diet is essential for tissue healing and for restoring a positive nitrogen balance after its removal. In some cases, dietary manipulation may be both diagnostic and therapeutic. For example, if the animal has food sensitivity or a true food allergy, switching to a number of different diets may be necessary. Dermatologic signs may be present, but GI signs can appear alone or with skin changes. Intestinal changes are reversible, and normal GI function can return in approximately 6 to 8 weeks. Dietary changes may involve switching to a novel protein or modified protein diet, such as hydrolyzed protein. Some cats are carbohydrate intolerant and develop secondary diarrhea from high-carbohydrate diets. Switching them to a low-carbohydrate, high-protein diet often resolves the diarrhea. Intestinal cancers are generally nonresponsive to dietary manipulation, but a high-quality diet can aid in the ability of the patient to heal, respond to chemotherapy, and maintain adequate nutrition. High levels of fish oils may help in the treatment of some forms of lymphoma.

**What can veterinary assistants and veterinary technicians do to provide excellent nutritional nursing care for our hospitalized patients?**

When a patient is admitted discuss the nutritional plan with the admitting doctor. Are there any foods they should not have? What should they be fed? How much? How often?

If the doctor has not taken a complete nutritional history from the client perhaps you can. Refer to the nutritional history taking suggestions above.

Don’t leave food continuously with a patient that is not eating. If you are nauseous do you like to have any smelly foods in your face all the time? And canned food left out for hours gets all dried out, discolored, and just plain gross!

Document in your nursing notes:

- What food does Fluffy like? Remember to include dry or canned, brand, flavor. Or maybe she only likes what the owner brought in? Do we have any left? If so, where is it?
- What food does Fluffy not like?
- Does Lucky like to eat out of a bowl? Plastic or metal? Prefers a flat plate?
- Does Spot eat better if hand fed? Or prefers to eat when no one is looking?
- Does Max prefer to eat outside of the cage? Or prefers to eat outside the hospital?
- Rex only seems to eat in an exam room when the owner comes to visit? Maybe he will eat for us in an exam room?
- How much food has Sandy eaten today?
- Zoe isn’t eating.
  - What have you tried tempting her with? Baby food? Canned tuna? Chicken? Cheese? For dogs maybe try canned cat food?
  - Have you tried putting a small amount in her mouth? How did she react?
  - Have you tried syringe feeding? How did she react?

Is your patient eating enough to meet their resting energy requirements (RER)?

- If the patient is only going to be in the hospital for the day or overnight as with a spay, neuter, dental, small mass removal, etc. not eating their entire RER should not be a problem. Provided, of course, they go home as scheduled and begin to eat once they are home. I recommend making a follow up phone call the day after discharge to ensure your patients appetite has returned to normal.
- If your patient is not eating or not eating enough to meet his RER please point this out to the doctor. Work together on a revised nutritional plan!
  - Ask the doctor if you can try tempting with foods not listed as acceptable on the treatment sheet. What other foods could you try tempting with? Baby food? Cheese? Tuna, Chicken? Canned cat food (for canine patients)?
  - If, despite your best efforts to encourage your patient to eat, any patient has not eaten for 3 days suggest a feeding tube! And remember that’s including days at home prior to being admitted not just days without eating in the hospital!

Be a nutritional advocate for your patients!

**References**

Firstline Magazine, How to conduct an effective interview with clients. Sep 1, 2010 by: Charlotte Higgins, AS, CVT

Compendium June 2013, Vol 35, No 6) Focus on Nutrition: Dietary Management of Gastrointestinal Disease by Marge Chandler, DVM, MS, MANZCVSc, DACVN, DACVIM, DECVIM-CA, MRCVS

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Nutrition for the Inappetent Cat, Elizabeth Thomosky, DVM, Diplomate ACVECC
Calculating energy (caloric) requirements

How do you know how much a patient should eat? Is Fluffy eating enough or too much? There are many formulas to calculate a patient’s energy needs — some for growth, weight loss, weight gain, extremely active individuals, and resting energy requirement (RER). The first step to determining how much to feed is to calculate the patient’s resting energy requirement. RER is a function of metabolic body size and represents the energy requirement of the patient while at rest at a controlled temperature. There are several formulas to calculating RER.

One simple formula is \( RER = 30 \times \) (body weight in kg) + 70.

**Example:** Sandy weighs 44 pounds, 20.0 kg.

\[
30 \times 20kg = 600 + 70 = 670
\]
Sandy’s RER is 670 kcal per day.

This formula is generally adequate for medium size healthy patients, however may not be accurate for small or large patients. The formula \( RER = 70 \times \) (body weight in kg) \( \frac{3}{4} \) provides a more correct estimate of the patient’s caloric requirements.

**Example:** Tiny weighs 3 pounds, 1.36 kg.

\[
70 \times (1.36kg) \frac{3}{4} = 88.1 \text{ kcal/day}
\]
Tiny’s RER is 88 kcal per day

or

\[
30 \times \text{ (body weight in kg) } + 70 = \text{ RER}
\]

\[
30 \times 1.36kg = 40.8 + 70 = 111 \text{ kcal/day}
\]
This calculation estimates Tiny’s requirement as 111 kcal per day; an over estimation of 41 kcal per day!

Once you have determined the patient RER, the next step is to calculate the patient’s Daily Energy Requirement (DER). DER is calculated by multiplying RER by a coefficient based on the patient’s life stage and body condition. A list of coefficients, for common life stages, to determine DER is listed below.

<table>
<thead>
<tr>
<th>Canine</th>
<th>Feline</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Growth DER</strong></td>
<td><strong>Growth DER</strong></td>
</tr>
<tr>
<td>Up to 4 months  = 3 x RER</td>
<td>Growing kittens = 2.5 x RER</td>
</tr>
<tr>
<td>Over 4 months   = 2 x RER</td>
<td>Normal neutered adult = 1.2 x RER</td>
</tr>
<tr>
<td><strong>Maintenance DER</strong></td>
<td><strong>Maintenance DER</strong></td>
</tr>
<tr>
<td>Average neutered adult = 1.6 x RER</td>
<td>Intact adult = 1.4 x RER</td>
</tr>
<tr>
<td>Intact adult = 1.8 x RER</td>
<td>Obese prone = 1.0 x RER</td>
</tr>
<tr>
<td>Obese prone = 1.4 x RER</td>
<td>Weight loss = 0.8 x RER</td>
</tr>
<tr>
<td>Weight loss = 1.0 x RER</td>
<td></td>
</tr>
<tr>
<td><strong>Work DER</strong></td>
<td></td>
</tr>
<tr>
<td>Light work = 2 x RER</td>
<td></td>
</tr>
<tr>
<td>Heavy work = 4-8 x RER</td>
<td></td>
</tr>
</tbody>
</table>

**Example:** If Tiny, in the example above, is a kitten and his RER is 88 kcal/day his estimated DER is 220 kcal/day (88 x 2.5).

It is important to remember all caloric calculations are estimates of the patient’s energy needs; actual caloric intake may vary from one individual to another. These calculations, however, are an excellent starting point for every nutritional recommendation.

Next, determine the caloric concentration of the food your patient is fed. The amount of calories in any food (and some treats) can be easily found on the company website or in their product guide. The caloric content of most “people” food that the patient is fed can be found on the products packaging or company websites.

Once you know the number of calories in a can or cup of the selected food, simply divide that number into the patient’s DER.

**Example:** In the above example we determined Tiny’s DER to be 220 kcal/day

Dry kitten food “X” contains 523 kcal/cup

\[
\frac{220}{523} = 0.4
\]
Tiny should be fed 0.4 cups per day

Example: Canned kitten food “X” contains 88 kcal/2.9 oz. can
220/88≈2.5

Tiny should be fed 2.5 cans per day

Example: Tiny’s owner wishes to feed both dry and canned versions of food “X”. A 2.9 oz. can contains 88 kcal. Subtract this amount from 220 (Tiny’s total DER) - 220-88=132
Then divide the remaining daily kcals by the number of kcals in 1 cup - 132/523=.25
Tiny could be feed 1 can and ¼ cup per day.

This amount may be divided by the number of feedings the patient will receive. Remember to be specific in your instructions to your client; include the name of the food you are recommending including the brand, the amount to be fed per day and per feeding, how many feedings per day, how many treats, and when the plan may change.

Example: Feed Tiny 1 can (2.9 oz.) and ¼ cup of Food “X” per day. I would recommend putting ¼ cup of the dry variety out for him in the morning and allowing him to graze on it throughout the day and feeding him ½ can of Food “X” in the morning and ½ can in the late afternoon.

Tiny is a growing kitten and these amounts will need to be adjusted as he grows until the time he is neutered. We will weigh Tiny and preform a body condition score at his next scheduled visit in 3 weeks. A revised nutritional recommendation will be made at that time.

You have indicated you will not be offering Tiny treats at this time. If you begin to offer treats (which most owners do) the amount of food Tiny is fed will need to be reduced by the number of calories the treats contain.

For many healthy pets being fed a good quality diet manufactured by a reputable company this maybe all the calculations that are required. But, being a nutrition tech, there are so many other calculations you may want to do!

**Dry matter basis vs. “as fed”**

Ingredients are listed on the label on an “as fed” basis; basically this means that the moisture content is included. In order to compare the nutrients in one food to another, especially when comparing a canned food to a dry food, you must convert each nutrient from “as fed” to “dry matter basis” (DMB). One method to calculate this is to subtract the percentage of moisture listed in the guaranteed analysis section on the label from 100% and then divide the percentage of the nutrient by this number.

- Step 1 – subtract the moisture content of the food from 100%.
- Step 2 – divide the nutrient content by the DMB.

**Example: a canned food label lists the moisture content as 75% and protein as 10% (as fed basis).**

- Step 1 - 100% - 75% (moisture) = 25% DMB
- Step 2 - 10% protein divided by 25% DMB = 40% protein
- This food is 40% protein on a Dry Matter Basis (DMB).

**Example: a dry food label lists the moisture content as 10% and protein as 18% (as fed basis).**

- Step 1 -100% - 10% (moisture) = 90% DMB
- Step 2 - 18% protein divided by 90% DMB = 20% DMB
- This food is 20% protein on a Dry Matter Basis (DMB).

Another simpler, yet perhaps not quite as accurate, method is to multiple the percentages of nutrients listed in the guaranteed analysis by 1.1 for dry foods and by 4 for canned foods.

**Example: a canned food label that lists the protein content as 10% x 4 = 40% protein (DMB).**

**Example: a dry food label that lists the protein content as 18% x 1.1 = 19.8% protein (DMB).**

Both of these calculations provide the estimated amount of the nutrients on a dry matter basis. If the actual amount is required it may be obtained from the pet food manufacturer.

**Calculating estimated carbohydrate content**

The amount of carbohydrates a pet food contains is not included in the guaranteed analysis, however it is easily estimated. Start by converting the protein, fat, fiber, and ash contents to the dry matter basis (as explained above). Add these percentages together and subtract the total from 100%. This is the estimated carbohydrate content of the food.

**Example: Food x contains**

- Protein – 32% DM
- Fat – 20% DM
- Fiber – 6% DM
- Ash – 12% DM
- Total – 70%
• 100% - 70% = 30%. This food contains approximately 30% carbohydrates.

Calculating calorie content
Calorie content is not required to be listed on the label of pet foods unless a claim of “light” or “less calorie” is listed on the label. Calorie content can be obtained from most manufacturers websites, product guides, or by calling the company support number. The calorie content can be calculated using the ‘as fed’ amounts listed in the guaranteed analysis of crude protein, crude fat, and the calculated carbohydrate content (as explained above) using the modified Atwater method. The modified Atwater value for protein and carbohydrates is 3.5 kcal/gram and for fat is 8.5 kcal/gram. The sum of these 3 results is added together and the result multiplied by 1.1 for dry foods or 1.2 for canned foods. This is the kcals/gram of the food. To convert to kcals/cup multiply the weight of the food in one 8-ounce measuring cup.

Example: Food x is a dry food weighing 90g/cup; on an ‘as fed’ basis it contains
  • Protein – 20.2%
  • Fat – 16%
  • Carbohydrates – 49.2%
  • 0.20 x 3.5 kcal/g = 0.70 kcal/g
    • 0.16 x 8.5 kcal/g = 1.36 kcal/g
    • 0.49 x 3.5 kcal/g = 1.72 kcal/g
      ▪ Total = 3.78 kcal/g
    • 3.78 kcal/g x 1.1 = 4.16 grams/cup
    • 4.16 g/cup x 90 g/cup = 374.4 kcal/cup

References
Acute pancreatitis (AP) is a common illness in dogs and cats. Although many cases are mild and easily treated, some cases develop severe complications that can be fatal. Mortality rates of dogs with severe AP have been reported to range from 27 to 42%. Survival rates in cats with pancreatitis are difficult to determine given the high incidence of concurrent disease, such as hepatic lipodosis.

Causes
The underlying cause of pancreatitis is not known. In dogs, dietary indiscretion such as table scraps, garbage, and high-fat foods are common causes of pancreatitis. Potassium bromide and phenobarbital are also known to cause pancreatitis in some dogs. Other risk factors in dogs include obesity and middle age or older (often over 5 years of age). The dog breeds of Miniature Schnauzer, Yorkshire Terrier, Silky Terrier, and the Miniature Poodle appear to be predisposed to pancreatitis. In cats, obesity and dietary factors are not thought to cause pancreatitis, but are attributed to a number of infectious diseases such as toxoplasmosis, FIP, and FIV. Both dogs and cats with diabetes mellitus appear to be prone to pancreatitis. Acute pancreatitis is more common in dogs and was once thought to be uncommon in cats. Although it is now thought that acute pancreatitis is actually more common and often missed in cats suffering from the disease. Cats frequently have more subtle and nonspecific clinical signs such as lethargy and anorexia. Chronic pancreatitis is seen more often in cats than in dogs.

Diagnosis
Diagnosis of pancreatitis is reliant on a combination of clinical signs, laboratory findings, and imaging. Clinical signs in dogs include vomiting, abdominal pain, depression, dehydration, anorexia, fever, and diarrhea. Severe cases may present with petechiation, icterus, ascites, and even shock. In some dogs an abdominal mass may be palpated. In cats the common signs include anorexia, lethargy, dehydration, and weight loss with vomiting occurring in less than 50% of cases.

Laboratory finds are nonspecific and quite varied. Some dogs present with a mild neutrophilia or severe leukocytosis with or without a left shift, anemia and neutropenia, or thrombocytopenia. Azotemia, increased liver values, lipemia, hyperglycemia, hypoproteinemia, hypercalcemia, and metabolic acidosis may also be seen. Elevations in amylase and lipase have been considered indicators of pancreatitis, however these enzymes may be normal in dogs with pancreatitis and elevated in patients not suffering from the disease. Canine and feline pancreatic lipase immunoreactivity (cPLI and fPLI, respectively) are enzyme-linked immunoassays that were developed to measure species specific pancreatic lipase activity. They are considered the most reliable indicators of acute pancreatitis; both can be affected by the severity of the disease.

Abdominal radiographs may be useful in the diagnosis of acute pancreatitis, however radiographs with non-specific findings are not unusual in patients with acute pancreatitis. Abdominal ultrasound is a more sensitive method of imaging and is often used in the diagnosis of pancreatitis in dogs and cats. An enlarged hypoechoic pancreas is suggestive of edema, hemorrhage or necrosis commonly seen in patients with acute pancreatitis. Other abnormalities such as peritoneal fluid, abscesses, and dilation of the pancreatic or biliary ducts may also be seen. Unfortunately, the sensitivity of ultrasonography when used alone to diagnose acute pancreatitis is generally low, with sensitivities ranging from 11 to 67% in cats and 68% in dogs.

Treatment
Abdominal pain, which may be severe, is common in patients with pancreatitis and should be addressed with analgesia. Fentanyl and other opioids are commonly used to improve patient comfort often in combination with low-dose ketamine and lidocaine in a constant rate infusion. Antiemetics such as maropitant, dolasetron, and ondansetron are recommended to reduce nausea in patients suffering from pancreatitis. Frequent aspiration of stomach contents is also recommended to reduce patient discomfort, nausea, gastric distention, regurgitation and decrease the risk for aspiration pneumonia.

Water intake is important in order to avoid or correct dehydration, and parental fluid therapy may be required. Electrolyte and acid base disturbances, if any, should be corrected before beginning nutritional support. Fasting these patients until clinical signs resolve to “rest the pancreases” and/or providing parenteral nutrition was long considered the appropriate nutritional management. However, premature intracellular activation of proteolytic enzymes is now thought to be the disease trigger rather than pancreatic stimulation. Studies in humans and rodents have shown that exocrine pancreatic secretion actually decreases during pancreatitis. Fasting to decrease pancreatic stimulation is therefore unnecessary and may be contraindicated. Withholding enteral nutrition decreases intestinal motility, compromises intestinal blood flow, and may cause intestinal villus atrophy. Prolonged fasting may also lead to abnormal internal bacterial flora and gastrointestinal barrier function. Patients with acute pancreatitis may have been anorexic for a number of days prior to presentation; fasting these patients, especially for multiple days, is not recommended. In dogs and cats enteral feeding is thought to prevent mucosal atrophy and therefore may reduce the risk of bacterial translocation and septic complications.
Studies in people have shown improved survival rates in patients fed via jejunostomy tubes, shorter hospital stays, and lower medical costs compared to those receiving parenteral nutrition. In veterinary patients, jejunostomy tubes are generally placed either endoscopically, fluoroscopically or surgically and are at risk of becoming dislodged after placement. Nasogastric tubes are more commonly used in veterinary medicine. These are less expensive and easier to place than j-tubes, and are often well tolerated in dogs and cats. Multiple human studies comparing nasogastric tube feeding to nasojejunal feeding have shown no significant difference in mortality rate, length of hospitalization, or infectious complication rates. The International Consensus Guidelines for Nutrition Therapy in Pancreatitis guidelines on nutrition therapy for acute pancreatitis, states that “postpyloric feeding is not required and support the use of NG feeding, although consensus agreement was not reached”. Many experts recommend NG tube feeding as the initial feeding method for patients that are unwilling to eat especially for patients not stable enough for general anesthesia or those not anticipated to require long-term assisted feeding. Enteral feeding, when started early, may help decrease vomiting and improve gastrointestinal motility. The goal of trickle feeding a liquid diet to these patients is often not to attempt to meet their resting energy requirements (RER), but rather to test to see if the patient can tolerate enteral feeding and to provide nutrition to the cells of the interstitial lining.

Some clinicians may prefer nasoepphageal tubes over nasogastric tubes because of concerns NG tubes may increase the risk of regurgitation and gasreosophageal reflex due to the irritation caused by the tube crossing the esophageal sphincter. A 2013 study compared the complication rates of these two tube types and found there to be no significant difference. NG tubes should be considered in patients with motility problems as they allow for liquid stomach contents to be aspirated. These patients often feel better after aspiration of liquid stomach contents; some may even begin to eat voluntary. NG and NE tubes require vigilant monitoring; if they should become dislodged they may cause aspiration pneumonia.

Only liquid veterinary diets should be used for feeding through NG and NE tubes. CliniCare® Canine/Feline Liquid Diet, which contains 1 Kcal/ml, is a good option. Emeraid® Intensive Care HDN™ Feline and Emeraid® Intensive Care™ HDN Canine are powdered formulas which are mixed with water and are also a good option. The volume of water added can be varied dependent on the diameter of the feeding tube, however the caloric density is reduced as the volume of water is increased. Trickle feeding via constant rate infusion (CRI) is most often used for hospitalized patients, although these tubes may also be used for bolus feedings and to administer oral liquid medications. Tablets should not be crushed and administered via these small tubes.

A number of liquid diets designed for people are also available. These diets are typically less expensive then veterinary liquid diets however are nutritionally inadequate and some may contain ingredients that are inappropriate for dogs and cats. These human diets are especially inappropriate for cats as they are too low in protein, taurine, and arginine.

Esophagostomy (E) tubes are a good option for patients that are healthy enough for a short anesthetic procedure. They are relatively easy to place and may be used in cats and dogs. They can be used by clients at home for longer term nutritional support if necessary and allow for a wider variety of canned diets to be used when blended with water. Medications may also be crushed and administer via E-tubes.

Gastrostomy tubes are also an option for long term nutritional support. Percutaneous Endoscopic Gastrostomy Tubes (PEG) are a good option when used for long term case. Clients are able to administer bolus feedings and medications at home using this method. General anesthesia is required and placement via an endoscope is recommended, although these are sometimes placed “blind”. Feeding via the PEG tube can begin as early as 12 hours post-placement, however waiting 24 hours is usually recommended. Once a PEG tube is placed it should not be removed for 10-14 days to prevent leakage of gastric contents into the abdominal cavity. This type of feeding tube can remain in place for months and maybe replaced with a low profile tube for longer-term use. The incision site may require superficial cleaning for a few days post-insertion. These tubes allow for a wide variety of diet options when blended with water. Medications may also be crushed and administered via PEG tubes.

**Parenteral nutrition**

Parenteral nutrition (PN) is generally reserved for patients with severe acute pancreatitis that cannot tolerate enteral feeding. Parenteral nutrition must be compounded and may not be readily available to many veterinary hospitals other than larger referral hospitals. Commercially available PN solutions for people are not designed to meet the needs of animals and may not provide adequate nutritional support. In some cases premade glucose and amino acid solution may be used short-term until the patient is stable enough to undergo general anesthesia for feeding tube placement or begins to eat on its own. The use of PN in veterinary patients has been associated with a risk of infection and intestinal atrophy, with subsequent risk of bacterial translocation, increased rate of sepsis; and metabolic complications such as hyperglycemia, blood electrolyte abnormalities, and hyperlipidemia. Aseptic technique is required and extreme care should be taken with the handling and administration of parenteral nutrition; if contaminated PN can become an excellent growth medium for bacteria. A study by Jensen and Chan (2014) showed patients receiving PN that were also trickle fed had a higher survival rate than those receiving PN only. Due to the risk of potential complications, the difficulty in obtaining and the high cost of providing parenteral nutrition, enteral feeding remains the preferred method for most acute pancreatitis patients. If PN is used, the recommendation is to begin trickle feeding as soon as the patient will tolerate it and gradually increase the enteral feeding.
**Dietary considerations**

A highly digestible diet, which is not high in fat, designed for patients with gastrointestinal disease is generally recommended for patients with pancreatitis. In some dogs breeds hypertriglyceridemia has been shown to be a predisposing factor. For obese and/or hypertriglyceridemic dogs, diets with a fat content of ≤10% on dry matter basis (DMB) are recommended to manage pancreatitis. Non-obese dogs and those that are not hypertriglyceridemic, diets with a fat content of ≤15% DMB are recommended. The recommended protein level for dogs with pancreatitis is 15 to 30% DMB.

Cats have a higher dietary fat requirement than dogs and can also digest and use dietary fat more effectively. This makes cats more susceptible to malnutrition and lean muscle loss during periods of anoxia and starvation. Insignificant levels of the amino acids arginine and methionine may contribute to the development of hepatic lipidosis in cats. For obese and/or hypertriglyceridemic cats, diets with a fat content of ≤10% on dry matter basis (DMB) are recommended to manage pancreatitis. For non-obese cats and those that are not hypertriglyceridemic, diets with a fat content of ≤25% DMB are recommended. The recommended protein level for dogs with pancreatitis is 30 to 40% DMB.

Cats with chronic pancreatitis may develop cobalamin deficiency. Subcutaneous or intramuscular injections of vitamin B12 are recommended, given weekly for the first month, and then continued every 2 to 4 weeks.

**Energy requirements**

Resting energy requirement (RER) represents the energy requirement for a normal animal, which is not fasted, and is at rest under thermo-neutral conditions. The equation 70 x (body weight in kg) ¾ is used to calculate RER. The general recommendation is to begin feeding pancreatitis patients 1/3 or less of the patient’s total RER for the first 12 hours and, if well tolerated, to gradually increase this amount every 12 hours until full RER is reached. If at any time the patient vomits, discontinuing feeding until vomiting has resolved, reducing the volume when feeding is resumed, and increasing the volume more slowly is recommended. In the past, an illness factor was often added to the RER when feeding critically ill, however this practice is no longer recommended. It has been shown that excessive nutrition during times of illness may increase the risk of hyperglycemia and other metabolic complications.

**References**


Taking the Mystery Out of Pet Food Labels
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Reading and understanding pet food labels is an important skill for all veterinary technicians to have. Clients often have questions about the food they are feeding their pet or which food would be best for their pet. Veterinary technicians are ideally suited to educate clients and make nutritional recommendations.

Pet food governing agencies and organizations
Having a basic knowledge of the agencies and organizations that regulate pet foods and pet food labels is the first step to understanding pet food labels.

The Association of American Feed Control Officials (AAFCO)
The Association of American Feed Control Officials (AAFCO) is a private organization but is not a regulatory body. All members of the AAFCO must be state or federal government officials. Pet food companies, pet food trade organizations (the Pet Food Institute for example) and professional organizations (such as the American College of Veterinary Nutrition) cannot be members, but may serve in an advisory capacity to AAFCO committees and investigations. The AAFCO develops model laws and regulations which are often adapted by states. Member government agencies often request assistance from AAFCO when revising existing or developing new regulations and laws. AAFCO also publishes ingredient definitions, official terms, pet food labeling information and standards for pet food testing. AAFCO established the current standards for dog and cat food nutrient profiles including the minimum standards for adult maintenance, growth, and reproduction profiles for both dogs and cats. The organization also set maximum levels for some nutrients in dog and cat foods. Testing protocols for evaluating metabolizable energy and feeding trial guidelines are also published by AAFCO. Many, but not all, states follow model bills and regulations for pet foods established by AAFCO.

The Center of Veterinary Medicine (CVM)
The Center of Veterinary Medicine (CVM), a division of the Food and Drug Administration (FDA), regulates pet food in cooperation with individual states. The FDA is responsible for regulating health claims, ensures food safety, approves food additives, as well as the specifics of some label requirements. The CVM regulates health claims made on pet food labels and product literature. Feed control officials in each state inspect manufacturing facilities and enforce these regulations. Pet food labels are legal documents and governed by the laws of the country the product will be sold in. All pet foods must meet the FDA’s requirements and the requirements of the state they are sold in.

United States Department of Agriculture (USDA)
The United States Department of Agriculture (USDA) inspects and regulates animal research facilities and may make unannounced inspections of these facilities. The USDA inspects ingredients used in pet foods to ensure proper handling and is responsible to ensure pet foods are labeled in a manner that makes it clear the food is intended for animals and not human consumption.

National Research Council (NRC)
The National Research Council (NRC) is a non-profit, non-government organization that evaluates research conducted by other groups and individuals. The NRC published “Nutrient Requirements of Domestic Animals” in 2006 that, in the United States, has been replaced with the AAFCO guidelines. However, these guidelines are still in use in some other countries.

Pet Food Institute (PFI)
The Pet Food Institute (PFI) is a national trade organization whose members include pet food manufacturers and pet food related suppliers. They represent the pet food industry, at the state and federal levels, before regulatory and legislative bodies.

Pet food label requirements
All pet food labels are required to have two main sections, each have legally required components: 1.) the principal display panel, and 2.) the information panel. The FDA definition of the principal display panel is “the part of the label that is most likely to be displayed, presented, shown, or examined under customary conditions of display for retail sale”. The information panel is defined by the FDA as “that part of the label immediately contiguous and to the right of the principal display panel”.

Principal display panel
The principal display panel must contain the product name, the species the food is designed for, and the net weight of the food contained in the package. There are specific requirements governing how products are named. If the name of a food is “Chicken for cats”, the food must contain 70% or more chicken as fed. “Chicken dinner”, “chicken platter”, “chicken entrée” must be 25% or more chicken total weight. “With chicken” must be at least 3% chicken. “Chicken flavor” is usually less than 3% chicken. Canned foods may not be greater than 78% moisture. However, foods with “chicken in gravy” or “in sauce” in their name are allowed to have moisture of 78% or more.
The principal display panel may also, and normally does, include the manufacturer's name and the brand name. A product vignette (picture/likeness) may also appear on the label. If a vignette is included, it must not misrepresent the product. Nutritional claims, such as “complete and balanced”, may appear on the principal display panel. All such claims are regulated and are discussed in the nutritional adequacy statement section below. Bursts and flags highlighting improvements, new products, or ingredient changes are allowed. However, the length of time this information is allowed to appear on the label is limited.

The information panel
The information panel must include the ingredient statement. Ingredients must be listed in descending order by weight and only standard AAFCO terms may be used.

A guaranteed analysis must be included listing the minimum percentages of crude protein and crude fat and the maximum percentages of crude fiber and moisture. Other nutrients are not required to be listed unless they are highlighted on the label. Nutrients not listed in the AAFCO Nutrient Profile must indicate “not recognized as an essential nutrient by the AAFCO Dog or Cat Food Nutrient Profiles.” Ash content is not required, but maximum ash guarantee is allowed. Cat food labels may state “low magnesium” if the maximum level is included and if the food contains less than 0.12% on a dry matter basis and less than 25 mg per 100 kcal metabolizable energy.

A nutritional adequacy statement must also be listed on all pet food labels except for treats and supplements. Claims such as “complete and balanced” must be substantiated by one of the three methods AAFCO allows to substantiate such claims: 1.) the formulation method, 2.) the feeding trial method, and 3.) the family method.

Dog and cat food producers that use the formula method to substantiate the claim a food is complete and balanced must formulate the food to meet AAFCO Food Nutrient Profiles using standard nutrient information about the ingredients the food contains or by having the product analyzed. The formula method does not ensure the availability of nutrients in the food or that the food is palatable. However, it is the easiest, least time consuming, and least expensive method. Foods using this method of validation must state it is “formulated to meet the nutritional levels established by the AAFCO Dog or Cat Food Nutrient Profile for (the life stage it was tested for)” on the product label.

The feeding trial method requires pet food companies to conduct feeding trials, using minimum standards established by AAFCO. All animals in the study are examined by a veterinarian at the start of the study and again when the feeding trial is completed. Participants are also required to have minimum baseline labwork performed, and body weights recorded weekly. This method may be used to validate that the food meets the requirements for growth, gestation and lactation, adult maintenance, and/or all life stages. The health of the dam at the end of the study and the size and health of the litter are taken into consideration with the gestation and lactation trials. Growth feeding trials must be conducted for a minimum of 10 weeks. The “all life stages” claim may be used when the offspring of a gestation and lactation feeding trial complete a growth feeding trial. Feeding trials for adult maintenance claims must be conducted for at least 26 weeks. The majority of nutritional deficiencies, if any, should be detected using this method of validation. However, nutrient excesses might not be evident during this timeframe. “Animal feeding tests using AAFCO procedures substantiate that (name of food) provides complete and balanced nutrition for (the life stage it was tested for)” is listed on the label of foods that have passed feeding trials. The feeding trial method is much more expensive and time consuming to conduct. It is generally considered the preferred method to substantiate nutritional adequacy claims.

The less common method of substantiating a food claim is the family method. Foods using this method must be in the same product line and be the same processing type (dry, canned, etc.) as a food tested using the feeding trial method. When analyzed, crude protein, calcium, phosphorus, zinc, lysine, and thiamin (as well as taurine and potassium for cat foods) must meet the levels of the food validated by feeding trials or the minimums established in the AAFCO Dog or Cat Food Nutrient Profiles for all nutrients. Products using this method must state “provides complete and balanced nutrition for (life stage), and is comparable in nutritional adequacy to a product which has been substantiated using AAFCO feeding test” on the label.

Treats and supplements are not required to include a nutritional adequacy statement on their label. Foods that do not meet AAFCO standards must be labeled “This product is intended for intermittent or supplemental feeding only.” Veterinary therapeutic diets should be labeled “use only as directed by your veterinarian.” These products must also list an AAFCO life stage nutritional adequacy statement or “this product is intended for intermittent or supplemental feeding only” on the label.

Feeding guidelines must be included on all dog and cat food labels, except veterinary therapeutic diets. These guidelines must be listed in common terms (for example a x pound cat, x cups of diet). These are only guidelines which are generally based on standard maintenance energy requirement (MER) equations with a safety margin. Actual amounts to feed should be calculated using one of several established methods and adjusted based on the pet’s body condition. However, it is important to note that pets consuming less than 80% of the recommended amount for their ideal weight may be consuming inadequate nutrients. These pets should be transitioned to a less caloric dense food.

The calorie content of dog and cat foods is not required to be listed on the label unless a claim of “light” or “less calorie” is listed on the label.
Manufacturer, distributor, or importer name and address must also be included. The company’s phone number and website are not required to be listed on the label, but may be included. A company that manufactures and distributes dog and cat food may state “manufactured by” on the label. Some companies have their foods manufactured by another company; these foods must state “manufactured for”, “imported by”, “distributed for” on the label. Foods not made in the United States must state “product of” on the label.

Universal Product Code (UPC), batch numbers, date of manufacture, and best used by date may also be included on the information panel.

References
Anesthesia carries with it a certain degree of risk and there is no single best way to anesthetize animals. Appropriate drug selection is important and largely dependent on the patient’s physical status, temperament, type of procedure, present and anticipated pain, familiarity of drugs available and cost.

One size does not fit all
It is generally not recommended to use pre-mixed drug cocktails like BAG or kitty magic. These cocktails do not allow for tailoring of the protocol to the specific temperament of the patient and often result in inappropriate dosing. There is also room for human error in mixing up large volumes of drugs, recording controlled substances and breaks in aseptic technique when pulling from a multi-dose vial. Best practice includes accurately drawing up each drug individually and then mixing them together for delivery. Designing an anesthetic protocol is easy if you remember PIMP, preanesthetics, induction, maintenance and pain management.

Pre-anesthetics and balanced anesthesia
Pre-anesthetics are an essential component to successful anesthesia. Pre-anesthetics help to calm and sedate anxious, frightened or fractious patients. It streamlines restraint and decreases patient and staff stress. Reduces the necessary dose of induction drug, helps to decrease the amount of inhalant needed for maintenance of anesthesia, promotes smoother inductions and calmer recoveries along with an easier to manage maintenance stage of anesthesia. Pre-emptive pain management, or giving an analgesic in anticipation of pain, should be part of the preanesthetic period. Balanced techniques along with pre-emptive analgesia can make post-operative pain easier to manage. Balanced anesthesia is the concurrent administration of smaller quantities of multiple drugs allowing them to work synergistically- combined drug effectiveness is greater than the sum of each part- which allows for the use of less of each drug minimizing side effects and maximizing therapeutic benefit.

Pharmacokinetics and pharmacodynamics
Understanding anesthetic drugs requires an appreciation of each drug’s pharmacokinetics and pharmacodynamics. Pharmacokinetics refers to the effect the body has on the drug and pharmacodynamics refers to the effect the drug has on the body. Drugs enter the body via the bloodstream and bind to specific receptors located in target organs and tissues to exert specific effects- for anesthetics these effects are usually central nervous system depression or stimulation. Agonists are drugs that bind to and stimulate a specific receptor found in target tissues. Antagonists bind to but do not stimulate receptors- most antagonists competitively bind to receptors and displace agonists effectively ‘reversing’ the action of the initial drug. A partial agonist is a drug that only partially stimulates a receptor and an agonist-antagonist binds to more than one receptor, agonizing one and antagonizing another.

Benzodiazepines like diazepam and midazolam are sedatives that work by increasing the activity of an inhibitory neurotransmitter- gamm-aminobutyric acid or GABA. This class of drugs is used for anti-anxiety and calming in ill or geriatric animals, skeletal muscle relaxation, appetite stimulation and as an anticonvulvulant. Benzodiazepines do not provide any analgesia and cause very minimal (if any) cardiovascular and respiratory depression. They have the tendency to decrease inhibition in young, healthy animals causing paradoxical excitement especially when given alone. Benzodiazepines are reversible with the drug flumazenil. Onset time is rapid and duration of action is 1-3 hours. Diazepam: propylene glycol based, for IV use only Midazolam: water based, can be given IM, SQ or IV

Phenothiazines like acepromazine or ‘ace’, is a tranquilizer often used as a preanesthetic in cats, dogs and horses. Acepromazine is an alpha-1 adrenergic and dopaminergic receptor antagonist that causes dose dependent sedation and a generalized disinterest in surroundings. It has no analgesic properties and it can mask the signs of pain without alleviating them. Ace can cause peripheral vasodilation that can negatively affect blood pressure and body temperature. It also has antiemetic and antihistamine effects. Onset time is around 15 minutes with peak effect at 30 minutes. Duration of action is variable and dose-dependent and can last 4-8 hours in small animals.

Alpha-2 adrenergic agonists provide sedation, muscle relaxation and analgesia. This class includes the drugs detomidine, dexmedetomidine and xylazine which are commonly used in both large and small animals. Dexmedetomidine is more specific for the alpha-2 receptor making the side effects associated with this drug class less prominent compared to xylazine and therefore safer for our small animal patients. Sedation and analgesia is dose dependent as is the duration of action. Peripheral vasoconstriction leads to pale mucous membranes and a reflex bradycardia. Vomiting and increase urine production are not uncommon. Some animals may be refractory to the drug. Co-administer with an opioid and allow patients to sedate in a quite place to decrease stimulation. Alpha-2 agonists are fully reversible with atipamezole (dexmedetomidine) or yohimbine (xylazine). The volume of reversal given should be...
the same as the volume of drug given previously. Dexmedetomidine: onset time is 5-15 minutes IV, 15-30 minutes IM. Duration of action is around 90 minutes.

**Opioids** provide analgesia by binding to specific receptors located in the brainstem and spinal cord. Some opioid receptors can also be found in peripheral tissues. Opioids don’t remove pain; they raise the threshold making pain more tolerable. There are two main types of opioid receptors, kappa and mu. Pain is mediated by mu receptors in mammals and kappa in birds. There is some evidence to suggest that kappa receptors exist in higher numbers in the gut compared to the peripheral tissues in mammals allowing kappa agonists to provide good visceral analgesia. Pure mu agonist opioids include morphine, hydromorphone, oxymorphone, fentanyl and methadone. This group of drugs fully bind to the mu receptor and are effective at providing analgesia. They can also cause sedation, especially in geriatric, pediatric or debilitated animals. Pure mu agonist opioids cause minimal cardiovascular depression and dose dependent respiratory depression by increasing the resting partial pressure of carbon dioxide or PaCO2. Bradycardia from vagal stimulation, vomiting from direct stimulation of the chemoreceptor trigger zone (CTZ), urine retention from decrease sensation and urge, antitussive properties and resetting of thermoregulation are not uncommon side effects of this drug class.

**Morphine**
Histamine release when given IV, poor oral bioavailability, vomiting is common, high lipid solubility make it a great for epidural use (preservative free formulation is recommended). Duration of action is 4-6 hours.

**Hydromorphone/oxymorphone**
Less vomiting compared to morphine, no histamine release IV, hydromorphone may cause hyperthermia in cats. Duration of action is 2-3 hours.

**IV fentanyl**
Very potent synthetic opioid, 3-8 minute onset time IV with a short duration of 20-30 minutes. Given as a constant rate infusion (CRI).

**Fentanyl patch**
Variable absorption that may not provide adequate analgesia if used as a sole means of analgesia. Takes 12-18 hours to reach therapeutic plasma levels and can potentially last for 3 days. Dose accordingly!

**Methadone**
Pure mu agonist and NMDA antagonist (helps prevent wind-up pain by blocking n-methyl d-aspartate). Good analgesia and less sedation compared to morphine. Duration of action is 3-4 hours.

Agonist/antagonist drugs stimulate one receptor while blocking another. Drugs like butorphanol and nalbuphine stimulate the kappa receptor while blocking the mu receptor making them only effective at treating mild pain in mammals. Because they block the mu receptor they can be used to reverse any unwanted effects of a pure mu agonist opioid while maintaining some analgesia.

**Butorphanol**
Mild analgesic with a ceiling effect (more drug does not equal more analgesia), short duration of action lasting 45 minutes- sedation may last longer, controlled as schedule IV

**Nalbuphine**
Mild analgesic, short duration of action lasting only 30 minutes, not a controlled substance

A partial agonist opioid is a drug that does not fully bind to the mu receptor making it less effective than a pure mu agonist like morphine. Partial agonists are drugs that only partially bind to the mu receptor exerting an effect that is not as great

**Buprenorphine**
Very high affinity for the mu receptor making the use of subsequent opioids ineffective until buprenorphine has worn off. Slow onset of action taking it nearly 45 minutes to provide pain control but long duration of action 6-8 hours, great transmucosal absorption in the cat but only about 30% bioavailable transmucosally in the dog, class III controlled substance

**Sustained release buprenorphine** (SR) is not an FDA approved product but is available through a compounding pharmacy. It is given SQ and has been shown to provide up to 72 hours of analgesia. Reversal is difficult and may require hospitalization.

**Simbadol**
High potency buprenorphine (1.8 mg/ml) dosed at 0.24 mg/kg and FDA approved for use in cats. Provides 24 hours of analgesia and can be q24 for up to 3 days. SubQ ONLY.

**Opioid antagonists** completely reverse the effects (analgesia, sedation and cardiopulmonary depression) of all circulating opioids including endogenous ones. These drugs should only be used in the face of an absolute opioid overdose because their use removes ALL analgesia along with any other effects. An acute awareness of pain can lead to catecholamine release from sympathetic stimulation which may result in cardiac arrhythmias, hypertension and possibly death. Redosing may be necessary due to the short duration of action of naloxone compared to most opioids.

- **Naloxone**: onset 1-2 minutes IV, 5 minutes IM, duration of action 30-60 minutes
- **Nalmefene**: onset 1-2 minutes IV, 5 minutes IM, duration of action 1-2 hours
Anticholinergics are sometimes used in preanesthetic combination to prevent bradycardia and decrease salivary secretions. Drug class effects include secretion reduction, bronchodilation, arrhythmias (glyco is less arrhythmogenic), thickening of respiratory secretions, and inhibition of intestinal peristalsis. Conservative use of anticholinergics is recommended and their use should never be a substitute for diligent monitoring.

Induction Drugs consist of injectable agents that allow the anesthetist to induce anesthesia quickly and to secure an airway. Injectable agents provide a safer, less stressful alternative to ‘gassing’ or ‘boxing’ down a patient. It is also less expensive, faster, with less waste/pollution and more control. All injectable anesthetics depress some vital organ function making the use of preanesthetics and subsequent lower doses essential.

Dissociative anesthetics are also referred to as cyclohexamines- these drugs produce a dissociated state along with sympathetic stimulation. Increases in cerebral blood flow and intracranial and intra-ocular pressure, CNS stimulation, primary cardiovascular depression with indirect sympathetic stimulation leading to increased heart rate, blood pressure and cardiac output. Sub-anesthetic doses of ketamine (and possibly Telazol) act as NMDA antagonists blocking central sensitization (wind-up pain) in the dorsal horn of the spinal cord. They are not analgesics in and of themselves and should not be used as a sole means of pain control however; ketamine is a great adjunctive medication contributing to a balanced approach to analgesia. Ketamine and Telazol are contraindicated in in patients that are sympathetically spent- severe trauma, stress, shock etc. This is due to the fact that only the direct cardiovascular effects will be appreciated. Not recommended for use in patients with cardiovascular disease, head trauma, intracranial masses, glaucoma, corneal ulcers, pheochromocytoma, hyperthyroidism.

Ketamine: dissociative anesthetic that causes muscle rigidity when used alone, commonly coupled with a benzodiazepine for induction. Not recommended for use with an anticholinergic as significant tachycardias can ensue, hepatic metabolism in the dog, renal excretion in the cat.

Telazol: Combination of a dissociative and a benzodiazepine. Tiletamine and zolazepam mixture, must be reconstituted with saline, use within 4 days if stored at room temp or two weeks if refrigerated.

Propofol and Propofol-28: Propofol is an ultra-short acting non-barbiturate induction drug with rapid metabolic clearance. Ninety seconds to peak effect and 5 minutes to redistribute, non-cumulative, administer slowly and steady to avoid over-dosing. Propofol has the potential to cause profound cardiovascular and respiratory depression and should be used cautiously. IV injection can be painful and muscle twitching and seizure-like activity has been reported. Repeated dosing is not recommended in cats as oxidative injury to red blood cells may result. Drug can be re-dosed intraoperatively at 1-2 mg/kg if patient becomes light. Pre-oxygenation is also recommended. Propofol-28 contains benzyl alcohol as a preservative giving it a 28-day shelf life. Regular propofol should be discarded 6-8 hours after opening.

Etidomide: Etidomide has little to no negative effect on the cardiovascular system- ideal induction drug for hemodynamically unstable patients. It may have brain protective properties after global ischemia, and inhibits steroid production by the adrenal glands for 3-6 hours after administration- not recommended in patients with hypoadrenalcortism. Vomiting and retching is common with underdosing and etomidate can be painful IV because it is based in propylene glycol. This drug is also expensive.

Alfaxalone: Alfaxalone is a neuroactive steroid substance that is used extensively in the UK, Australia, Europe and Canada. It produces hypnosis with reasonable muscle relaxation and a decrease in cerebral blood flow and cerebral oxygen demand. Dose dependent hypotension may be seen initially due to myocardial depression and peripheral vasodilation but the effects are often offset by the reflex tachycardia. Respiratory depression associated with the use of alfaxalone is dose dependent; the drug is non-cumulative and approved for IV use only. IM chemical restraint is off-label and works better for cats vs. dogs (very short duration of action). Alfaxalone is not an analgesic, it does not contain preservatives and should be discarded 6 hours after opening.

Maintenance of anesthesia is often achieved through the use of inhalant anesthetics. Inhalants are potent vasodilators and cause dose-dependent hypotension and respiratory depression. The two most common inhalants used in veterinary medicine are isoflurane and sevoflurane. The minimum alveolar concentration or MAC is the potency of an inhalant. MAC-50 is the amount of inhalant needed to keep 50% of patients non-responsive to surgical stimulation. MAC-95 or surgical MAC is the amount of inhalant needed to keep 95% of patients non-responsive to surgical stimulation and is calculated by multiplying MAC-50 for the species by 1.5. It is important to note that respiratory arrest can occur at 2 x MAC so proper dosing is essential to safety. MAC studies are done on patients who have not been given pre-anesthetics. The use of pre-anesthetics decreases the amount of inhalant necessary thereby reducing MAC.

Iofluorane: MAC is 1.3% in dogs and 1.6% in cats, low blood-gas solubility (rapid induction and recovery), 0.2% metabolized in the body. Sevoflurane: MAC is 2.3% in dogs and 2.6% in cats (sevo is less potent and requires higher vaporizer settings to maintain anesthesia), lower blood-gas solubility, 3% metabolized in the body.

Pain Management is an important part of the anesthetic protocol and balanced techniques should be employed whenever possible. In addition to opioids and alpha-2 agonists, the flowing drugs should augment the pain management protocol.
**Non-steroidal anti-inflammatory drugs (NSAIDS)**

NSAIDS (Non-steroidal anti-inflammatory drugs) block the production of specific prostaglandins by binding and inhibiting the cyclooxygenase (COX) enzyme. COX enzymes (COX-1 and COX-2) also have important homeostatic functions. COX-1 mediates prostaglandins responsible for renal and GI blood flow and platelet integrity and COX-2 mediates prostaglandins responsible for inflammation, pain, edema, fever as well as other homeostatic functions. NSAIDS should be avoided in animals with renal or hepatic dysfunction, coagulopathies, GI disorders, shock, hypotension/hypovolemia and they are not recommended for use in combination with corticosteroids.

**Lidocaine**

Lidocaine is a local-anesthetic and anti-arrhythmic agent with a rapid onset and short duration of action. Lidocaine is a prokinetic that enhances gut motility and help prevent ileus. It has MAC sparing effects when given as a constant rate infusion, a loading dose must be given prior to starting a CRI to achieve blood levels. Cats are sensitive to lidocaine and care should be taken when this drug is used.

**MLK (Morpine, Lidocaine, Ketamine)**

Drugs are combined into a bag of IV fluids and delivered at a surgical rate. Loading dose of each drug is required to achieve therapeutic levels.

**Tramadol**

An oral medication that has weak mu receptor effects along with norepinephrine and serotonin reuptake inhibition. Tramadol works well with NSAIDS as post-operative pain management. Gabapentin is effective at reducing hyperalgesia and allodynia associated with neuropathic pain and central sensitization as well as chronic and malignant pain. Gabapentin is not an analgesic but an adjunctive medication that allows true analgesics to work better by calming down the nervous system- gabapentin should be used in conjunction with NSAIDS and/or tramadol for best results. Amantadine is an NMDA antagonist and analgesic adjunctive medication. It is good at reducing central sensitization. Amantadine is excreted almost unchanged in the urine- reduce doses in the renal patient.
Lights Out!
The Anesthetic Induction Period
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Anesthesia is the reversible loss of sensation as a result of pharmacological agents that may affect all or part of the body. There are differing levels of anesthesia that range from conscious sedation for minor procedures to deep general anesthesia for more invasive techniques. Anesthesia induction precedes general anesthesia that produces unconsciousness and through balanced techniques also includes analgesia, muscle relaxation and amnesia.

Balanced anesthetica
Balanced anesthetic protocols incorporate multiple drugs and techniques from different classes to achieve a desired goal. Animals are prepared for anesthesia by performing a thorough patient evaluation, choosing adequate premedication, placing an IV catheter, initiating a controlled induction and securing the airway. The equipment and drugs used in the induction period vary depending on the patient’s health and temperament as well as the procedure being performed. The anesthetist’s role is to gather this information and in consultation with a veterinarian, develop a suitable induction plan for his/her patient.

Anesthesia induction
The careful administration of appropriate agents to bring about unconsciousness and to help facilitate endotracheal intubation or securing of the airway. The induction period begins with premedication administration and ends when all monitors are connected and the patient is on a maintenance level of anesthesia.

Getting ready
Once the patient has been premedicated, time is needed for the drug to take effect. The amount of time depends on the route and type of drug as well as the temperament of the patient and the environment. IV premeds require 1-2 minutes where as an IM injection may take up to 15 minutes to take effect. Anxious patients will often take longer to sedate and patients premedicated in a loud or otherwise stressful/active room will also be on edge and take longer to sedate. Best practice is to premed and then put the patient in a quite and dark room. Periodic monitoring of patients while sedating is essential. The period of time between premeds and induction is an ideal time to gather supplies and leak test equipment. For example one could:

- Set up and pressure test anesthesia machine
- Gather IV catheter supplies
- Choose three endotracheal tubes and check each cuff for leaks
- Corral monitoring equipment and check that it is working properly
- Plug in supplemental heating device and warm it up
- Fill out anesthesia monitoring sheet with patient information
- Calculate, check and draw up induction drugs
- Calculate fluid rate and set up fluids
- Formulate an emergency plan and calculate emergency drugs

IV catheters
All patients undergoing anesthesia should have an IV catheter placed. IV access is essential when performing successful anesthesia because having a catheter facilitates the delivery of IV anesthetics, avoids potentially harmful perivascular drug administration and provides access to rapid drug/fluid administration in emergency situations. IV fluid therapy in anesthetized dogs and cats is recommended to maintain venous access, to help correct hypovolemia caused by the vasodilatory effects of anesthetic drugs, to help replace insensible fluid losses common in the surgical patient.

IV catheter size is important because it influences the drug delivery rate and may cause unwanted patient side effects. For example, a small gauge catheter is easy to place and produces less inflammation; however, it may be difficult to deliver highly viscous drugs rapidly or with ease. The IV fluid administration rate is also limited by the internal diameter of the catheter. A larger gauge catheter may be more difficult to place but will deliver drugs and fluids with ease. It can cause phlebitis due to the constant contact it makes with the vessel wall. As a general guide, 22 gauge catheters work well in most cats and 20 gauge catheters work well in most dogs.

Gauge is a descending scale; large numbers correlate to small diameters (20 gauge is smaller than 10 gauge). French is ascending where large numbers correlate with large diameters (20 French is larger than 10 French).

Note: Intact animals, male and female, have tougher skin and often require a larger sized catheter or a ‘pilot’ hole to be made with a needle prior to catheter insertion. This hole prevents burring of the cannula from pushing it through tough tissue. To make a pilot
hole, choose a needle one size larger than the catheter you wish to place. Tent up the skin and poke a hole through the skin only. Then, use that hole to introduce the catheter and stylet through the skin and then proceed by placing the catheter into the vein.

**Induction drugs**

Induction is best achieved through IV administration of a drug because this route allows for rapid airway control and enables careful titration to provide the desired effect without over dosing.

The ideal induction agent would have a wide therapeutic index, rapid onset of action, quick metabolism or redistribution, no negative affect on other body systems (maintains respiratory drive), reversible, inexpensive, long shelf-life, and not be a controlled substance. This drug does not exist but a few come relatively close.

**Ketamine/midazolam**

This combination is a dissociative anesthetic (ketamine) coupled with a benzodiazepine (midazolam). Ketamine causes muscle rigidity whereas the benzodiazepine provides muscle relaxation.

**Pros**

This combination increases heart rate and blood pressure as a result of sympathetic nervous system stimulation. Ketamine antagonizes the NMDA receptor (an excitatory neurotransmitter in the brain) effectively blocking glutamate and stopping the amplification of pain signals. Induction is fast and drug is titrated to effect.

**Cons**

This combo can cause an increase in cardiac work and myocardial oxygen demand. In critical patients with depleted sympathetic reserve ketamine can drop cardiac output and blood pressure. The acidic pH of ketamine makes IM injections painful. This combination should be avoided in patients with a seizure history or history of head trauma, brain lesions, tumors and ocular disease and it can increase intracranial (ICP) and intraocular pressure (IOP).

**How to use**

A dose of ketamine is calculated based on the patient’s lean body weight. The volume of ketamine is matched by an equal volume of midazolam (or diazepam). Half of the calculated dose is given IV over 30-90 seconds then titrated to effect (until patient is able to be intubated).

**Notes**

Induction with ketamine/midazolam is slower than with propofol and patients will maintain more muscle and jaw tone. This is important to keep in mind to avoid over-dosing. Ketamine takes about 2 minutes to reach the brain and lasts about 20 minutes. Ketamine is a CIII controlled substance.

**Telazol**

This drug is a dissociative and sedative combination similar to ketamine/midazolam. The drugs in Telazol are tiletamine and zolazepam. This drug is reserved for induction of healthy dogs and cats as it cannot be tailored to meet the needs of the patient, it is not reversible and it has a relatively long duration of action. Telazol is a CIII controlled substance.

**Propofol**

A commonly used short-acting IV anesthetic with a relatively short shelf life.

**Pros**

Propofol is non-cumulative and it provides a rapid, smooth induction with a short duration of action. It can be used as a constant rate infusion (CRI) or total intravenous anesthesia (TIVA) in dogs or redosed via low-dose injections for procedures like a laceration repair, minor biopsies and endoscopy. It is good for head trauma as it doesn’t increase ICP and c-sections as it has minimal effects on fetus.

**Cons**

Propofol can cause significant respiratory depression, apnea and cyanosis which is more pronounced when the drug is administered rapidly or in large doses. Animals given propofol must be monitored closely and supported (assist respirations to avoid hypoxemia). Propofol is highly protein bound so patients with hypoproteinemia will require lower doses. It is not currently a controlled substance.

**How to use**

Propofol should be calculated as a range giving the anesthetist the information necessary to dose appropriately. Depending on how sedate the patient is, a dose within the range is given as a slow IV push over about 20 seconds (low end for very sedate and mid-range for less sedate patients). The patient is assessed and the drug is titrated to effect, e.g. until intubation is easily achieved. Propofol takes about 90 seconds to reach the brain and then about 5 minutes to redistribute making it important to be ready but also be a bit patient.

**Alfaxalone**

Alfaxalone is a neuroactive steroid molecule, with central effects. Despite being an analogue of progesterone, Alfaxalone does not bind to sex hormone, glucocorticoid, or mineralocorticoid receptors

**Pros**

This drug provides a rapid induction and hypnosis with reasonable muscle relaxation and a decrease in cerebral oxygen demand. It is not irritating perivascularly, it has a wide safety margin, smooth induction and rapid but smooth recovery. It is non-cumulative and can be dosed daily.
Cons
Dose-dependent respiratory depression and hypotension from myocardia depression and peripheral vasodilation that is offset by a reflex tachycardia.

How to use
A dose is calculated based on lean body weight and given IV over 60 seconds until the signs of anesthesia are appreciated, then it is titrated to effect. Rapid admin exacerbates side effects. Alfaxalone is a CIV controlled substance.

Etomidate
A short-acting injectable based in propylene glycol.

Pros
It is rapidly metabolized by the liver and plasma esterases (enzyme found in the plasma that cleave apart compounds and make them inactive) leading to a quick recovery. It produces minimal cardiovascular and respiratory depression and does not alter heart rate, cardiac contractility or blood pressure. This drug is a great choice for high-risk patients including those with pre-existing cardiac conditions and hepatic disease; it is not a controlled substance.

Cons
High cost, retching and myoclonus if patient is not adequately sedated or if it is underdosed, may cause pain on injection. Drug can inhibit steroid production for up to 4 hours.

How to use
Calculate dose based on lean body weight and administer the entire calculated dose as a slow IV push to well sedated patients.

Opioid
Opioids can be used to induce anesthesia is sick or debilitated patients.

Pros
Gentle and relatively safe induction as there is little negative effect on cardiovascular function.

Cons
Patients become very noise sensitive during this time so a quite induction area is essential. This method is really only useful in very ill or old patients that may not tolerate the cardiovascular effects of other induction agents.

How to use
A combination of an opioid and a benzodiazepine are given IV until patient is unconscious.

Inhalants
Inhalant anesthetics can also be used to induce anesthesia; however, mask or chamber inductions are stressful, airway control is slow and there is a great risk for contamination of the work environment. Over dosing of inhalant anesthetic during a mask or chamber induction is nearly always a given. Isoflurane and Sevoflurane are the two inhalants used with greatest frequency in veterinary medicine. Both have high vapor pressures requiring a precision vaporizer for delivery and low blood:gas solubility (the measure of the tendency of an inhalant to exist as a gas or dissolved in blood) making induction and recovery quick. The biggest differences between isoflurane and sevoflurane is their minimum alveolar concentration or MAC. MAC is the minimum concentration of drug in the lungs that is needed to keep 50% of patients immobile during surgical stimulation. It is used to discuss the relative ‘strength’ of the inhalant. Those with a lower MAC are more potent because less drug is needed to achieve the same effect. MAC is also used to help guide vaporizer settings.

Pros
Provides a relatively rapid induction and recovery with good muscle relaxation. The drug is eliminated primarily via the lung with little kidney and liver metabolism.

Cons
Dose-dependent central nervous system depression, dose dependent hypotension, myocardia depression and respiratory depression. Inhalants do not provide any analgesia and should never be used as the sole agent for painful procedures. There is also a risk of exposure to potentially harmful waste anesthetic gas. Inhalants are not safer than properly used injectable drugs.

How to use
MAC is used to guide the vaporizer setting for intubated animals and other drugs (adjuncts and premeds) are used to help keep the vaporizer setting as low as possible to avoid the negative dose-dependent side effects.

A patient’s response to IV anesthetic induction will depend on the rate of administration (most anesthetics agents are titrated to effect) and over dosing is more likely when drugs are given too quickly and their side effects are not appreciated as they happen. Physiologic factors such as blood volume, total protein levels and blood pH play a role in the distribution of drugs throughout the body. The overall health status and organ function will affect how quickly the drug is distributed, metabolized and excreted which ultimately affects when and how long those effects last. Animals that are ill or debilitated in some way, including advanced age will require less induction drugs than their healthy counterparts. Premedications and the patient’s response to them will also influence the type and amount of agent needed. Using premedications will reduce the amount of induction drug need and thus further reduces unwanted side effects. As an added bonus, premedications make IV catheter placement easier and less stressful for the patient and the staff.
Endotracheal tubes and intubation

There are three methods of selecting the proper sized endotracheal tube. One involves using the width of the nasal septum as a guide, another uses a chart with the animal’s lean body weight and recommended endotracheal tube (ETT) size and finally palpation of the outer diameter of the trachea in the region of the mid-neck. The most accurate way to choose a tube is lean body weight and breed.

Choose three tubes, the size you expect to place plus one half size smaller and one half size larger. If intubating a brachycephalic breed, choose 2 sizes smaller since these breeds often have stenotic or narrow tracheas. Measure the length of the tube alongside the patient so that the tip of the tube lies midway between the larynx and thoracic inlet. ETT tubes that are too short may not pass through the larynx and those that are too long may pass into a bronchus resulting in ventilation of only one lung. If the tube extends more than 1 inch past the incisors, it should be trimmed or a shorter tube should be used. Long tubes that stick out passed the incisors contribute to mechanical dead space that may lead to the rebreathing of carbon dioxide.

Intubation should be performed using the largest sized tube possible to minimize the need to overinflate the ETT cuff. A laryngoscope should be used to allow visualization of the larynx and to get the user used to handling the equipment for when it is needed for a challenging or emergent intubation. Sterile lubricant should be lightly smeared on the ETT cuff to help facilitate intubation and to help form a seal within the trachea. Lidocaine is helpful in desensitizing the larynx in cats. Cuff inflation after placement should be done with care.

Complications of endotracheal intubation

Esophageal intubation

Inadvertent placement of the ETT into the esophagus.

Clinical signs

Absence of EtCO₂ readings, patient will not maintain unconsciousness, inability to achieve a proper seal.

Treatment

Tube should be deflated, removed from esophagus and then placed into the trachea

Bronchial intubation

Intubation of one of the mainstem bronchi due to an excessively long ETT.

Clinical signs

Low pulse ox reading from one lung ventilation, difficulty keeping patient asleep

Treatment

Deflate cuff and gently pull ETT tube back so that the tip falls midway between the larynx and thoracic inlet. Use an ET tube of the same length to determine the length of the ET tube inside the patient.

Laryngospasm

Reflexive closure of the laryngeal cartilage usually from stimulation and inadequate anesthetic depth. Common in cats.

Clinical signs

Closed laryngeal cartilage and inability to place endotracheal tube.

Treatment

Desensitize laryngeal cartilage by placing a drop of lidocaine onto the area. Alternatively, deepen the plane of anesthesia to suppress the reflex. Do not continue to push the ET tube towards the laryngeal cartilage without first desensitizing it or deepening the level of anesthesia as it will only make the spasms worse.

Forceful intubation

Endotracheal intubation should be gentle and easy. Although the largest sized tube should be placed, do not forcefully attempt to place a tube that is larger than necessary. Damage to delicate tissues at the back of the throat can lead to edema and potential airway obstruction.

Clinical signs

Red or swollen laryngeal tissues, difficulty breathings upon extubation, complete airway obstruction.

Treatment

If patient is having difficulty breathings post extubation it may be necessary to re-anesthetize them and place another endotracheal tube (smaller). Provide the patient with supplemental oxygen and try to keep them calm, be ready to intubate with a much smaller tube.
Anesthesia literally translates into “without sensation”. Our goal as anesthetists is to provide unconsciousness, amnesia, analgesia and muscle relaxation for a variety of procedures both invasive and non-invasive. Our ability to carefully string our patients out along the line between consciousness and unconsciousness compromises homeostasis making close monitoring essential.

**Why monitor?**
Anesthetic emergencies are difficult to predict especially if the patient is not being monitored. Anesthetic emergencies can happen quickly and they can be devastating. It is better to be proactive rather than reactive when it comes to anesthetic complications. Our goal is to be able to walk that line with confidence by maximizing the safety of the anesthetic experience. There is no safe anesthesia, only safe anesthetists.

**Morbidity and mortality (M&M)**
Morbidity refers to the prevalence of complications occurring secondary to hypoxia. Mortality is the rate of anesthetic related deaths. Certain complications are more likely to increase morbidity and mortality:
- Excessive bradycardia
- Cardiac depression
- Vasodilation
- Hypotension
- Arrhythmias
- Hypoventilation
- Hypoxemia
- Hypothermia

These obstacles all make it difficult for nutrient rich oxygen to reach the tissues. Diligent monitoring allows us to recognize and treat these potentially life threatening problems.

**Monitoring basics**
According to the American College of Veterinary Anesthesia and Analgesia (ACVAA) guidelines on anesthesia, continuous awareness of the heart rate and rhythm along with the gross assessment of peripheral perfusion including pulse quality, mucous membrane color and capillary refill time are mandatory. Ventilation and oxygenation, anesthetic depth and body temperature are also important. If you didn’t have any monitors, you could still gather information using your eyes, ears and hands: Heart rate, pulse quality and vasomotor tone, respiratory rate and character, reflexes and tone, eye position and body temperature. Monitoring multiple parameters gives you a more complete picture of the physiologic status of the patient.

**Heart rate**
Be aware of the normal heart rate range for the species you are working with as well as what is normal for the breed and the individual. Give yourself a range that is specific to this patient and stay away from extremes. Bradycardia is an excessively slow heart rate that affects cardiac output and leads to hypotension and poor perfusion. For most medium to large-breed dogs, the low 50’s with a normal blood pressure is often tolerated. Smaller dogs have higher resting heart rates as do cats so for this population the tolerated low for small dogs might be 80 bpm and for cat 120 bpm. When possible, also monitor blood pressure and end-tidal carbon dioxide for the most complete cardiovascular picture.

Tachycardia is an excessively fast heart rate that affects cardiac output. The less time the heart spends in diastole, the less time it has to fill with blood so blood pressure is often affected by way of a decrease in stroke volume. Tachycardia also increases myocardial oxygen consumption and makes the heart have to work extra hard. Depending on the size of the patient and their normal resting heart rate, tachycardia in the dog is often in excess of 160-180 bpm and 240-280 bpm in the cat.

Some causes of extremes of heart rate (and potential ways to remedy them)

**Bradya**
- Anesthetic overdose (lighten up)
- Opioid administration (give an anticholenergic)
- Alpha-2 agonist administration (reverse or no treatment)
- Hypothermia (rearm)
- Hypoxia (oxygen therapy)
• 1st and 2nd degree A-V blockade (anticholenergics)
• High vagal tone (anticholenergics)

Tachycardia
• Too light (deepen)
• Painful (give analgesics)
• Ketamine administration (no treatment)
• Anticholenergics (decrease dose next time)
• Inotropes (decrease infusion)
• Hypovolemia (restore volume)
• Hyperthermia (cool)
• Hypoxemia (oxygen therapy)
• Hypercarbia (ventilate or eliminate rebreathing of CO2)
• Anesthesia recovery (comfort or no tx)

Pulse quality and vasomotor tone
Palpation of a pulse is a subjective way of approximating blood pressure. It is done by evaluating the height and width of the pulse pressure waveform compared to normal. Bounding pulse- vasodilation as seen in sepsis and hypovolemia, where the vessel is easily collapsible. Weak and thready pulse- vasoconstriction as seen with alpha-2 administration, poor cardiac function, tachycardia, small stroke volumes. It is essential that the anesthetist palpate normal pulses in a variety of patients to make it easier to determine when a pulse is abnormal.

Pulse quality is largely a reflection of stroke volume (the volume of blood pumped with each beat) and vessel size or vasomotor tone (the degree of vasodilation or vasoconstriction). Vasomotor tone regulates both peripheral and visceral perfusion. Vasodilation improves peripheral perfusion but can cause hypotension if too severe. Vasoconstriction can impair peripheral perfusion but also improves blood pressure. Assessing vasomotor tone can be done by evaluating mucous membrane color and capillary refill time. Normal mucous membrane color consists of a light to medium pink color and a capillary refill time of 1-2 seconds. Pale mucous membranes may indicate vasoconstriction (think about the pale gums of an animal given dexmedetomidine). Red mucous membranes may indicate vasodilation (think about a patient that is hyperthermic or too deeply anesthetized).

Many things can cause vasodilation or vasoconstriction and the way to correct it is to address the cause.

• Vasodilation
  o Systemic inflammation
  o Sepsis
  o Hypercapnia
  o Hyperthermia
  o Drugs (acepromazine, inhalants)

• Vasoconstriction
  o Hypovolemia
  o Heart failure
  o Hypothermia
  o Drugs (alpha-2 agonists, sympathomimetics)

Pulse sites
Pulses can be assessed from a variety of sites and each site offers a little different feel. It is essential for the anesthetist to become familiar each site. The femoral artery is located high up on the inner thigh where the leg meets the abdomen. This vessel is large and easy to palpate in most patients but can be challenging to find in obese or heavily muscleed animals. The dorsal metatarsal artery is located on the dorsal aspect of the hind limb distal to the hock and over the metatarsals. This vessel is very accessible and great for arterial catheter placement. It can be difficult to palpate in vasoconstricted, hypotensive or small patients. The coccygeal artery is located on the proximal ventral tail is also used for arterial catheter placement. The radial artery is just proximal to the metacarpal pad on the forelimb and a bit medial. Because it is located distally on the limb, it too can be difficult to palpate in vasoconstricted, hypotensive or small animals. The lingual artery is on the ventral portion of the tongue near the lingual frenulum. It is only useful in anesthetized animals and is a great place to easily grab a pulse intra-operatively. In exotics, cats and small dogs, placing your hand around the chest is another way to feel the heartbeat.
Respiratory rate and character
All anesthetic drugs provide some degree of respiratory depression making respiratory monitoring and support vital. A change in breathing is a good indication of a change in patient status. Respiration is comprised of tidal volume (TV), respiratory rate (RR), and minute volume (MV).

- TV (Vt) = the volume of air in a single breath (10-20 mL/kg)
- RR (f) = the number of breaths per minute (8-15 br/min)
- MV (V) = total volume of air breathed per minute (150-250L/kg/min)
- TV X RR = MV

Respiratory character describes the quality of the breaths being taken. Breaths can be shallow, deep, slow, fast, irregular or absent. Shallow breaths are often associated with a small tidal volume and they tend to be faster than normal breaths. The increase in rate makes up for the decrease in volume and normalizes the overall amount of air moved in one minute (minute ventilation). Deep breaths are characterized as having a large volume but slow rate. Apnea is the absence of spontaneous breathing and is common especially after induction of anesthesia. It is important that the anesthetist support the patient through this time by breathing for them until they can begin to breathe on their own. Bradypnea is a slow respiratory rate and without an end-tidal carbon dioxide monitor or blood gas analyzer, it is difficult to determine if these patients are adequately ventilating. Hypothermic patients and those that are a deep level of anesthesia will often exhibit this slow respiratory rate. Tachypnea is an increased respiratory rate with a number of possible causes: too light, too deep, hypoxia, hypercapnia, hyperthermia, hypotension, painful, septic, atelectasis etc. It may be helpful to give some larger breaths, check body temp, check BP, assess pain, assess anesthetic level etc to find the cause.

Ventilation is defined as the act of breathing and respiration is the actual gas exchange. The adequacy of ventilation and appropriate gas exchange can really only be determined through arterial blood gas analysis or end-tidal carbon dioxide measurement.

Reflexes and tone
The amount of muscle tone an animal has and whether or not their reflexes are present (and to what extent) gives the anesthetist an indication of the depth of anesthesia. The presence of reflexes indicates a lighter level of anesthesia but this is not always a bad thing. Brisk reflexes indicate that the patient is too light, but sluggish reflexes are ok as long as it doesn’t interfere with the procedure. The corneal reflex should always be present (unless paralyzed).

Tone can be assessed simply as none, some or lots. Jaw tone, anal tone and general muscle tone should be taken into account when assessing the depth of anesthesia. No tone may indicate that the patient is a little deep or adequately anesthetized; some tone is ok and even good as long as it doesn’t interfere or cause harm but lots of tone is not good unless you are trying to recover the patient.

Eye position
The eye is a little window into the central nervous system and its position can tell us a lot about how deeply our patients are anesthetized. If the eye is centrally facing the animal is either too deep or too light so it is important to also look at pupil size. A medium pupil indicates that the patient is light. A dilated pupil indicates a deep level of anesthesia and an immediate adjustment is necessary. A constricted pupil also indicates a deep level of anesthesia and an immediate adjustment is necessary. If they eye is in a ventral-medial position, the patient is at a good plane of anesthesia. Keep in mind that drugs can affect eye signs and pupil size making it important to assess the patient after pre-medication and before induction (opioids cause dilation; ketamine can affect the palpebral reflex etc).

Temperature
Anesthesia depresses muscle activity, metabolism and thermoregulation and leads to hypothermia. A good temperature range is 98-102 degrees Fahrenheit in dogs and cats perianesthesia. Hypothermia can contribute to an anesthetic overdose making necessary to decrease your doses in hypothermic patients! Animals less than 98 degrees Fahrenheit are considered hypothermic. Mild hypothermia is anywhere from 96-98 degrees Fahrenheit, moderate is 94-96, severe is 90-94 and anything less than 90 degrees is moribund due to CNS depression and imminent death if not corrected. Patients greater than 103.5 degrees Fahrenheit are considered hyperthermic (normal being 100.5-102.5). Cell damage occurs once the body temperature reaches 108 degrees.

YOU
Your eyes, ears and hands can make excellent monitors if you know how to put them to work!
Remember to be vigilant and look at the whole picture (co-existing disease, drugs given and currents meds, procedure, species, breed…). Seek knowledge, the more you know the more you know the more confident you will be. Remember to enjoy yourself, anesthesia is an art and a science and it can be fun.
The recovery phase begins once the procedure ends and anesthesia ceases to be delivered. This may include turning off the inhalant or discontinuing total intravenous anesthesia (TIVA). Recovery ends when the patient is sternal, alert, extubated and normothermic.

It is good practice to maintain the patient on 100% oxygen for 5 minutes after discontinuing inhalant. It is also common to disconnect the patient from the breathing system, occlude the patient end of the hoses and flush the system full of 100% oxygen then reconnect to the patient.

- Patients continue to be respiratory depressed and can benefit from an enriched oxygen mixture
- Waste anesthetic gas is corralled and disposed of decreasing exposure to personnel

Monitoring
Patients should continue to be monitored during the recovery period and the extent to which they are monitored depends on their overall condition.

- The ECG patches can be removed unless patient has a history of arrhythmias. Patients that have undergone major surgery like a splenectomy, GDV, pericardectomy, PDA ligation etc. should have the ECG maintained through recovery and likely post-operatively as well to check for arrhythmias. The most likely arrhythmias would be VPC’s but may progress to V-tach and negatively affect blood pressure.
- The blood pressure cuff can be removed if the patient has been stable and normotensive. Patients that have been hypotensive or cases where blood pressure needs to be monitored closely (renal transplant) should continue with readings. Most patients’ blood pressure will increase once the inhalant is discontinued and they start to recover making it acceptable to discontinue this monitoring parameter. Blood pressure lines and ECG monitors can make it difficult to transfer patients to gurneys and recovery cages so these monitors are some of the first to be removed.
- The pulse-oximeter should be maintained at least until the patient is extubated. This monitor is easy to place and maintain and is often hand-held making it simple to continue to use. Also, it will alert the anesthetist to hypoxemia-a condition that is likely after discontinuation of oxygen in the face of continued respiratory depression.
- Continue to monitor EtCO2 until disconnected from oxygen or extubated. Continued respiratory depression is common during recovery and hypoventilation will be reflected in the EtCO2 readings giving the anesthetist the opportunity to assist respirations and help ward off hypoxemia.
- Maintain supplemental heating if patient is normo- or hypothermic. Patients can lose heat during recovery because they are no longer under drapes and such. The temperature should be taken immediately post-op and then every 30 minutes until patient is able to maintain a normal temperature for two consecutive readings.
- Some patients may become hyperthermic during recovery from anesthesia and these patients need to be monitored more closely and steps may need to be taken to reduce their temperature. Ways to this may include just removing supplemental heat, removing blankets, wetting down paws (alcohol is not recommended) or placing a wet towel in a cage with a patient. Acepromazine promotes vasodilation and may help bring the body temperature down. NSAIDS can help if the hyperthermia is actually pyrexia secondary to inflammation. Post-operative infections will not manifest immediately, it takes a day or so for the bacteria to gain a foothold so this should not be a consideration unless infection was present pre-operatively. Opioid induced hyperthermia is self-limiting and doesn’t typically require intervention.

Nursing care
Good nursing care in recovery can have a dramatic positive effect on anesthesia recovery and post-operative comfort. Provide patients with a quiet and comfortable place to recover, keep them warm, express their bladder and offer food/water when awake. Hunger, hypoglycemia, hypothermia and a full bladder are all uncomfortable situations and can exacerbate pain and cause undue stress. Some patients will benefit from having cotton put into their ears to muffle outside noise and blanket over their cage to shield their eyes from the ambient light. Soft music (through a dog’s ear) and items from home (unwashed so they smell like home) are also comforting. When working with a post-op patient, be patient, go slow and narrate what you are doing to help calm the patient and to keep you calm as well.

Pain management may include a constant rate infusion (CRI) or intermittent boluses of opioids, placement of a cold pack over the incision, massage of tense muscles or those strained during surgical positioning, and anxiolysis. Other steps that can be taken to increase comfort include cleaning off any left over scrub solution or blood from the surgical site. Blood and scrub can be itchy and their presence may prompt patients to bother the surgical site. Gently remove this with saline or water during recovery.
Patients that are hypothermic can be placed on a soft, insulated surface with a forced warm air heating blanket and a blanket over top that gets tucked under the patient or the pad creating a bubble of warm air. This bubble shouldn’t be disturbed unless necessary to check body temperature. If IV fluids are being given they can be warmed near the patient so heat is not lost between the source and the patient. Once the patient is able to maintain their body temp above 99 F, the supplemental heat can be removed. Once removed, the body temp should be checked in 30-60 minutes to make sure it is maintaining.

Recovery area and extubation
Recovery should be smooth and stress-free and it should take place in a quiet area of the hospital where the lights can be dimmed as it is important to minimize unnecessary stimulation during recovery. Vital signs such as HR, RR, CRT and MM color should be monitored every 5-10 minutes during recovery and body temperature every 30 minutes. SpO₂, EtCO₂ and BP/ECG can also be monitored and should be if the patient is recovering from a major procedure or is compromised in some way.

A full recovery is defined as the point at which the patient is able to sit sternal with normal vital signs (HR, RR, body temp). Patients should be extubated when they are ready and showing signs which include alertness, swallowing and tongue tone. Patients do not have airway control until they are alert and able to swallow so these parameters are used to decide when it is safe to remove the endotracheal tube. Although massage and mild stimulation can be helpful is rousing patients, aggressive waking is contraindicated as it can lead to patient and staff injury. Recovering animals should be aloud to ‘sleep it off’ especially after a long or painful procedure. Some animals pop awake and start to chew on the tube, (these patients should be extubated quickly!) while others are content being able to breathe (brachycephalics). Do not deflate the endotracheal tube cuff until you are ready to extubate since vomitus or other fluid may be present in the pharynx and can be easily aspirated. Brachycephalic breeds will be alert, sternal and content with a tube in their mouths- these recoveries take a bit longer but these patients can be extubated when they are alert and sternal. Some patients will not be alert but will be swallowing slightly- these patients are not ready! Wait for alertness and tongue tone before pulling the tube unless instructed otherwise. Cats should be extubated as soon as possible as they are prone to laryngeal spasm upon extubation. After extubation patients should be watched closely every 15-20 minutes until a full recovery has been achieved- hypoventilation can quickly lead to hypoxemia when a respiratory depressed animal is breathing room air. The IV catheter should remain in place until full recovery and pain has been properly addressed. Catheters kept in over night should be wrapped and kept clean.

Post-operative pain management
Pain should be assessed as soon as the patient is conscious. Review the anesthesia record to gather information on which analgesics have been given and when. Additional analgesics should be given during recovery if the patient is due for them or if they are showing signs of pain. Pain can be assessed using any number of different pain scoring sheets including: University of Melbourne Pain Scale, Glasgow Composite Measure Pain Score or the Colorado State University Acute Pain Scale (author recommendation). Pain should be evaluated every hour for the first 4-6 hours following surgery and then regularly until discharge. High quality pain management should be instituted for at least the first 24 hours post-op and may continue if necessary. High quality means opioids plus NSAIDS (if tolerated) plus or minus sedation or adjunctive medications along with good nursing care. To go home medications for pain management
TGH meds: 3-7 days of analgesia depending on the type of procedure. PO drugs like tramadol, NSAIDS, gabapentin and amantadine are common. Certain opioids like fentanyl patches or buprenorphine can also be sent home because they are given either OTM or transdermal but the risk of abuse by humans is high.

Tramadol
Weak mu receptor agonist. Can be used in dogs and cats but has a very bitter taste and is often refused by many felines. Decent for acute and chronic pain management and it may have increased benefit if given chronically because it inhibits the reuptake of norepinephrine and serotonin. It should not be used in conjunction with other drugs of this nature or TCA’s or MAOI’s. Tramadol may increase the seizure threshold so use caution in animals with a seizure history.

Gabapentin
Antiepileptic at high doses and anti-hyperalgesic at moderate doses. Works well for the treatment of neuropathic pain. Drug should be stepped down when discontinued to avoid rebound pain. Liquid form may contain xylitol so it is not recommended for use in dogs and cats. It is not an analgesic in and of itself so it should be used with other drugs like NSAIDS or tramadol. Excreted unchanged in the urine so care should be taken when dosing renal patients.

Amantadine
NMDA antagonist and is a great analgesic adjunct for acute and chronic pain in dogs and cats. It blocks wind up or central sensitization in the dorsal horn of the spinal cord. It is used as an antiviral drug in humans and can be used in combination with NSAIDS or tramadol. Available in capsule and liquid. Excreted unchanged in urine so use caution in renal patients.
Fentanyl patch
Class II controlled substances. Transdermal patch will provide systemic absorption and provide 3-4 days of analgesia. If removed prior to 3 days it will continue to provide analgesia for 6-12 hours after patch removal.

Patch is very popular because it is easy to use but the absorption rate in dogs and cats can be highly variable. It may work really well for some patients and not at all in others. It takes about 6-12 hours in cats and 12-24 hours in dogs to reach blood levels so opioids should be supplemented until the patch kicks in. 12.5 mcg/hr; 25 mcg/hr; 50 mcg/hr; 100 mcg/hr. patch size is selected based on patient weight. The patch can be placed anywhere on the body where there is intact hairless skin.

Transdermal fentanyl or recucyra
Recently approved for use in the US. It is a transdermal fentanyl solution (50 mg/ml) that is applied to the skin between the shoulder blades 2-4 hours pre-operatively and will provide up to 4 days of analgesia. It is not meant to replace the use of other opioids during surgery but may prove to be enough analgesia when used in conjunction with other CRI’s like lidocaine, locals or NSAIDS to provide multimodal analgesia. Proper training is necessary prior to use and extreme care must be taken to prevent direct contact with the site of application for at least 3 days.

OTM buprenorphine
A common post-op drug used in cats. Buprenorphine is a partial mu agonist and a class II controlled substance. Transmucosal administration in the cat has almost 100% bioavailability. OTM means allowing the drug to be absorbed across the mucous membranes, not given orally. Oral administration is not efficacious due to the first pass effect. The bioavailability of OTM buprenorphine in dogs is around 30% so this route is not recommended in dogs.

SR buprenorphine
A compounded medication that is not FDA approved. The drug is set in a lipid carrier which allows it to absorb over a period of time. It is given SQ and may sting on administration but can provide up to 72 hours of analgesia. This drug is used more frequently in cats than dogs likely because they seem to tolerate it better. High doses can cause euphoria (purring, rolling, rubbing). This drug is very difficult to reverse and may require hospitalization for supportive care and a naloxone CRI in extreme cases.

Simbadol
A high potency buprenorphine for SQ use in cats. It provides 24 hours of analgesia and is well tolerated by most felines. Side effects are similar to SR buprenorphine but Simbadol is FDA approved and well regulated.

Rough recoveries
The majority of patients who have been given a balance of anesthetic drugs will have a smooth recovery. However, some will wake up dysphoric or painful despite our best efforts. The quality of the induction, the temperament of the patient, previous rough anesthetic recoveries and the length of the procedure can all affect the recovery. Evaluate analgesic history and effectiveness throughout the procedure and especially prior to recovery. If the duration of action of a sedative/analgesic has lapsed, consider re-dosing prior to recovery or at least be ready with analgesics.

Rough recoveries can be due to pain, dysphoria or emergence delirium and it is often difficult to determine which is which.

- **Dysphoria**: characterized by agitation, excitement, restlessness, excessive vocalization and disorientation. Patient is not responsive to human voice and will not look/focus on you. Cats tend to act as though they are hallucinating, they may open-mouth breathe, vocalize and pace. Dysphoria is often precipitated by opioids and patients that are dysphoric from opioids will not benefit from additional opioids. They may however, benefit from a low dose of butorphanol or naloxone to reverse the side effects of opioids. This may then lead to a painful patient because reversal removes or lessens analgesia in an attempt to calm the patient so adjunctive analgesics may be necessary to keep them comfortable.

- **Emergence delirium**: dissociated state of consciousness in which the patient is unaware of their surroundings. Patient may be excited, agitated, restless and they may vocalize. This most often occurs in patients who have no sedative drugs on board or when recovering from a dissociative anesthetic (ketamine, telazol). Other situations explained in the human literature that may preclude emergence delirium include hypoglycemia, hypoxia, severe hypercapnia, hypotension and distended bladder.

There are a few ways to handle this situation;
- **Resist**: Restrain the patient until they have exited this post-anesthesia excitement phase. This is not always possible or safe.
- **Rearrange**: Reanesthetize the patient using induction drugs, reintubate or supplement oxygen and allow the patient to wake back up (this is called a do-over)
- **Reanesthetize and also give a sedative like dexametomidine or acepromazine**
- **Just give a sedative and hope it works before someone gets hurt!**

- **Pain**: When dealing with rough recoveries it is important to address potential pain as well as anxiety since our patients cannot verbalize when they hurt. Painful patients are responsive to human voice and will relax and stop vocalizing once analgesics take effect. Dexametomidine is an excellent choice in patients that can handle the drug because it offers both analgesia and sedation and is fully reversible. It is possible to achieve the desired effect with very small doses given IV (1-2 mcg/kg).
- **Slow recoveries:** Some patients seem to take forever to wake up. Hypothermia can slow down drug metabolism and lead to a prolonged recovery. Keep patients warm as they recover and help circulation by gently massaging and moving the limbs. Post-operative opioids can also re-sedate a patient after surgery. These drugs are given to control pain so it is not recommended that they be reversed unless necessary. If necessary, reversal can be achieved with butorphanol or low dose and titrated naloxone to reverse the side effects but preserve most of the analgesia.

- **Rapid recoveries:** Rapid recoveries can be dangerous if the patient is dysphoric or delirious. Many rapid recoveries are due to patients waking up on inhalant alone - no real analgesia and very little premedications still on board. These recoveries can not always be predicted but by staying ahead of pain management and conscious of when premeds wear off, many rapid and rough recoveries can be avoided.
Our goal as anesthetists is to provide our patients with an expert balance between the effects of anesthetic drugs, both wanted and unwanted, the affects of underlying illness and the intensity of procedural stimulation. To achieve this, we must depress the central nervous system sufficiently to keep our patients unaware and relaxed, immobile and pain-free while at the same time maintaining near normal cardiopulmonary function. This compromise to homeostasis brings with it the potential for problems. However, the source for most anesthetic complications is human error (70%) and those errors are often associated with poor patient monitoring. Constant vigilance of the anesthetized patient allows us to be proactive rather than reactive through anesthetic depth adjustment and need-based patient support. As anesthetic drugs have become more refined and physiologic monitors more sophisticated, successful anesthesia is no longer defined as the lack of mortality- just surviving anesthesia isn’t enough- but by the lack of morbidity. Our ultimate goal is to bring patients through the anesthesia experience without any ill effects.

The American College of Veterinary Anesthesia and Analgesia or ACVAA published a position statement in 2009 regarding recommendations for monitoring anesthetized veterinary patients. The ACVAA recommends frequent and continuous monitoring of circulation, oxygenation, ventilation and body temperature by trained personnel. Appropriate physiologic support through the utilization of hands-on monitoring in conjunction with physiologic monitors can improve the practice of veterinary anesthesia. However, anesthetic monitors are only as good as the person using them- it takes a well-trained technician or nurse to be able to recognize abnormalities and know how to properly respond.

**Circulation**

Adequate blood pressure is necessary to deliver oxygen and nutrients to all tissues in the body. The components of blood pressure include systolic (peak pressure during contraction; stroke volume and arterial compliance), diastolic (minimum pressure during relaxation of the heart; systemic vascular resistance and heart rate) and mean arterial pressure (driving pressure for organ perfusion). Normal values: Systolic 100-140 mm Hg, Diastolic 50-100 mm Hg, Mean 70-120 mm Hg.

Arterial blood pressure is comprised of cardiac output (the volume of blood ejected over one minute) and systemic vascular resistance (the resistance offered by the peripheral vessels). Stroke volume (the volume of blood pumped with each beat) and heart rate make up cardiac output. Preload (the volume of blood returned to the heart), cardiac contractility (the intrinsic strength of the heart’s contraction) and afterload (the tension against which the ventricle must contract) all contribute to stroke volume. Afterload is also a factor in systemic vascular resistance as is vessel diameter.

Autoregulation is the body’s ability to maintain normal perfusion to vital organs despite changed in systemic arterial pressure. Autoregulation can maintain normal renal blood flow when systemic blood flow falls outside of 60-160 mm Hg range.

There are two ways to measure blood pressure, indirectly using non-invasive methods and directly using invasive methods. Non-invasive methods include the use of oscillometric blood pressure monitors and a Doppler; both utilize a pneumatic cuff placed over a peripheral artery. The width of the blood pressure cuff bladder should be 40% of the circumference of the limb to obtain an accurate reading. Cuffs that are too large may lead to underestimated pressures and cuffs that are too small may lead to overestimated pressures. It is better to choose a wider cuff over a smaller one if the ideal size is not available as the margin of error is less. The monitor should be set to read every 3-5 minutes for anesthetized patients. Blood pressures read more frequently do not given the limb a chance to recover and re-perfuse with blood; pressures read less frequently may lead to missed events.

The Doppler can be a more accurate way of monitoring blood pressure in small patients but is prone to user error. Cuff selection and placement, maximum inflation pressure, and deflation rate are determined by the operator and can vary greatly. In dogs, the pressure at which the sound of blood flow returns after cuff inflation reflects the systolic blood pressure. In cats the pressure more closely reflects the mean arterial pressure.

Invasive blood pressure (IBP) measurement is the gold standard of blood pressure monitoring. This method requires the aseptic insertion of a catheter into an artery (dorsal pedal, coccygeal, femoral, lingual, facial, auricular). Most monitors capable of measuring invasive pressures will display a continuous systolic, diastolic and mean arterial blood pressure reading as well as a waveform. By observing the velocity or upswing of the arterial pressure wave (anacrotic ascending limb) one can glean information about changes in cardiac contractility. This method of blood pressure assessment is essential in a number of clinical settings including major surgery, trauma and critical care.

Since oscillometric monitors estimate blood pressure and are great trend monitors, isolated abnormal readings may not be a cause for alarm. If the blood pressure is trending, evaluate the patient by assessing anesthetic depth and vital signs. Adjustments should be made based on findings (deepen anesthesia, assist respirations, provide more analgesia etc). If the blood pressure is trending down the
same algorithm is used but the corrective steps will differ. If sudden and significant changes in blood pressure occur, evaluate the patient and communicate with the clinician then recheck the pressure and make adjustments based on findings.

Hypotension is defined as a systolic blood pressure less than 80 mmHg and a MAP of less than 60 mmHg in small animals. The reduction may be significant enough to cause serious complications including shock and death. Consequences of hypotension may include hypoxemia, reduced drug metabolism, worsening of V/Q mismatch (see Reasons for Hypoxemia), delayed anesthetic recovery, renal failure, central nervous system (CNS) abnormalities (blindness, neurologic deficits), shock, cardiac arrest and death.

- Drug side effects/excessive anesthetic depth: Inhalants, acepromazine, anaphylaxis or histamine release (rapid IV morphine), radiographic contrast media, beta blockers, rapid IV antibiotics etc.
- Decreased venous return to the heart: Hemorrhage, fluid loss, compression of vena cava, increased intra-abdominal pressure, mechanical or manual ventilation
- Cardiac disease: Arrhythmias, valvular disease, cardiomyopathy, congestive heart disease, pericardial effusion
- Mechanical: Closed pop-off valve, overinflated re-breathing bag (decreases venous return)
- Shock/vasoactive substance release as seen with manipulation of damaged or diseased organs

Hypertension is less common in veterinary anesthesia but no less important. Hypertension can cause greater swings in blood pressure and end-organ damage and may be the result of:

- Mechanical error
- Sympathetic stimulation: Hypercarbia (indirect SNS stimulation), pain, light level of anesthesia
- Anesthetic drugs: Alpha-2 agonists, ketamine, inotropes, pressors
- Disease states: Pheochromocytoma, chronic renal disease, hyperthyroidism, increased intracranial pressure, heart disease, etc.

Treatment options for hypo- and hypertension depend on the underlying cause and may include an assessment and adjustment of anesthetic depth, assessment of analgesic efficacy, expansion of intravascular volume (hypotension) and possibly drug therapy.

The ECG peripherally detects the electrical signals generated by the heart. This monitor reveals information about the heart’s electrical activity but not its mechanical function. The ECG is essential in diagnosing arrhythmias and should be used during the pre-operative evaluation of high-risk or trauma patients, intra-operatively for all procedures, and post-operative for those cases requiring follow up information on the electrical stability of the heart.

**Conduction**

The P wave, the first small and usually positive wave signifies atrial depolarization initiated by the SA node. There is a brief pause between the P wave and the QRS complex which corresponds to the time it takes for the impulse to travel from the SA node through the AV node and bundle of His. This pause is called the P-R interval and it also represents the time between atrial depolarization and ventricular depolarization. The QRS complex represents ventricular depolarization. This complex starts out with a negative deflection (Q) and then takes a sharp and significant upswing (R) and then comes back down sometimes dipping just below baseline (S). Depolarization should happen quickly producing a narrow QRS complex. A short pause is seen after this QRS complex followed by the T wave, this is called the ST segment. The T wave represents ventricular repolarization. The time between the R wave of one complex and the R wave of the following complex is termed the R-R interval. This interval helps us determine heart rate and regularity. A normal sinus rhythm follows the conduction pattern from its origination in the SA node, through the AV node, Bundle of His, bundle branches and Purkinje fibers. Since the SA node has the fastest impulse rate it typically overrides the other pacemaker cells in the heart.

In the anesthetized patient, the ECG is used as a means of detecting dysrhythmias and not necessarily for diagnosing arrhythmias. ECG’s are a great way to monitor heart rate and rhythm but the ECG can appear normal when myocardial performance and blood pressure are poor.

**Common arrhythmias**

- 1st degree AV block: prolongation of the PR interval
- 2nd degree AV block: dropped QRS complexes
  - Type 1: variable PR interval (av node fatigue)
  - Type 2: constant PR interval before and after dropped beat (more serious- can progress)
- 3rd degree AV block: complete dissociation between SA and AV node
- VPC’s: Impulse originating distal to SA node which can effect hemodynamics
- V-tach: rapid ventricular rate, unstable rhythm
- V-fib: Irregular tachyarrhythmia, unstable, arrest

**Oxygenation**

Oxygenation is the process of taking oxygen molecules from inspired air and delivering them to tissues to sustain aerobic cellular metabolism. PaO2 is the measure of oxygen dissolved in plasma and it is determined by arterial blood gas measurement. This value
tells us the efficiency of the lungs to deliver oxygen to the blood and is measured in mmHg (millimeters of mercury). Normal PaO₂ values depend on inspired O₂ concentration. To determine the normal PaO₂ values, multiply inspired oxygen concentration by 4-5. For example, room air is ~ 21% O₂ so the normal PaO₂ value for an animal breathing room air is 80-110 mm Hg. A patient breathing 100% oxygen should then have a PaO₂ value of 400-500 mm Hg. SpO₂ is peripheral oxygen saturation of hemoglobin that is measured as a percent and estimates PaO₂. The numbers of concern for each value are not equal because they are based on the normal oxyhemoglobin dissociation curve. This curve illustrates the relationship between PaO₂ and SpO₂. The pulse ox utilizes wavelengths of light; deoxygenated blood absorbs red light whereas oxygenated blood absorbs infrared light. The ratio of red to infrared light provides us with an SpO₂ value. An SpO₂ of 95% and above represents adequate oxygenation and a precipitous drop in PaO₂ can be seen at an SpO₂ of 94% and below indicating early hypoxemia. When the SpO₂ falls to 90% the PaO₂ is as low as 60 mm Hg which is defined as severe hypoxemia and warrants treatment. There are five reasons for hypoxemia:

**Low inspired oxygen concentrations** is usually not an issue when using 100% O₂, but it is important to check the oxygen supply and make sure the flowmeter is on and in good working order. If using N₂O, check ratios or discontinue mixture. A sedated or anesthetized animal breathing room air is often at risk for hypoxemia making supplemental oxygen necessary.

**Hyperventilation** is common under anesthesia in spontaneously breathing patients because most anesthetics are respiratory depressants. An animal that appears to be breathing adequately may not be. The adequacy of ventilation can only be determined by an arterial blood gas or estimated using end-tidal CO₂ monitoring. Patients at risk for hyperventilation (obese, geriatric, dorsal recumbancy, deep anesthesia) should be manually or mechanically ventilated.

**V/Q mismatch** stands for ventilation (V) and perfusion (Q) mismatch. It is characterized by inflated alveoli that are not perfused (ventilation is good but blood flow is bad) or perfused alveoli that are not well inflated (blood flow is good but ventilation is bad). Some common causes of V/Q mismatch include anemia, inadequate ventilation, atelectasis, hypotension, positioning.

**Diffusion impairment** is an increase in the distance that oxygen has to travel to get from the alveoli to the capillary and vice-versa. Pulmonary edema, pulmonary fibrosis and pneumonia can all cause diffusion impairment.

**Shunting** is defined as venous blood by-passing oxygenation in the lungs and mixing with arterial blood decreasing PaO₂. Collapsed lung lobes, PDA and VSD can all lead to shunting and hypoxemia.

Many pulse-oximeters are prone to malfunction, bias and variation especially if they have been designed to be used on humans instead of animals. Some potential reasons for flawed readings include the fact that tissue, venous/capillary blood, and skin pigment all absorb infrared light and motion, location of the probe, wetness/dryness, tissue thickness, electrical/optical interference can effect readings. When inaccurate, the pulse-ox is usually inaccurately low. Check patient status first, then troubleshoot monitor because a poorly functioning monitor may indicate poor systemic perfusion.

All monitors have limitation and the pulse-ox is no exception. This monitor does not assess the adequacy of ventilation; it only estimates the amount of oxygen saturating the present hemoglobin. It also gives a pulse rate and in monitors with plethysmography (a graphic waveform of blood flow beneath the probe) you can glean some information concerning pulse pressure and volume status. Also, it can be misleading in anemic patients who may have an SpO₂ reading of 100% (available hemoglobin is saturated) when in fact their tissues are hypoxic. This monitor can give the anesthetist a false sense of security because animals on 100% oxygen won’t indicate impending hypoxemia until the PaO₂ falls to 80 mm Hg (all the way from 400!).

**Ventilation**

The capnometer gives us end-tidal carbon dioxide (EtCO₂) value which estimates PaCO₂. PaCO₂ is the amount of CO₂ dissolved in arterial blood and it gives us information about how well an animal is ventilating. Carbon dioxide is important in two ways, it defines the respiratory cycle and it is a by-product of cellular metabolism (cells produce it, lungs excrete it). As an anesthetic monitor, the capnometer non-invasively gauges the adequacy of ventilation and it helps guide assisted breaths and mechanical ventilation. This monitor increases our ability to detect potential problems by alerting us to leaks within the breathing system, apnea and fluctuations in respiration on a breath-by-breath basis, deviations in chest compliance and changes in cardiac output (including the effectiveness of chest compressions during CPR). It can also help us determine if a patient is properly intubated and alerts us when a patient is inadvertently extubated.

Normal PaO₂ is 35-45 mm Hg and the end-tidal CO₂ level is typically 3-5 mm Hg greater than the actual PaO₂. For this reason, the appropriate normal range for EtCO₂ is 30-45 mm Hg. Abnormal EtCO₂ readings may have a respiratory or a metabolic cause.

- Elevated EtCO₂ (> 45 mm Hg)
  - Hypoventilation, excessive depth, inappropriate ventilator settings, exhausted soda lime, machine malfunction, hyperthermia, airway obstruction, abdominal or thoracic restrictive disease, pleural space filling

- Low EtCO₂ (< 30 mm Hg)
  - Hyperventilation, light level of anesthesia, hypoxemia, pain, hypothermia, inappropriate ventilator settings, decreased cardiac output
Normal levels of inspired CO₂ range from 0-3 mm Hg and any value greater than 3 mm Hg may indicate a problem. Common causes of elevated inspired CO₂ levels include a leak in the breathing system or machine, excessive dead space, exhausted soda lime or inadequate fresh gas flows in a non-rebreathing system.

**Temperature**
Temperature can be monitored intermittently via rectal thermometer or continuously via rectal/esophageal probe connected to a mechanical monitor. Properly placing a reusable probe down the esophagus provides an easy and accurate core body temperature reading. Minimizing patient heat loss can be achieved by controlling conductive, convective, radiant and evaporative heat loss and by providing supplemental heat support. Insulating a patient from the cool environment with the use of blankets and towels and minimizing prep times can help maintain normal body temperature. Convective warming devices can also be employed and are an efficient way to maintain and improve body temperature under anesthesia. Rice socks, electric heating blankets and warm water bags/bottles/gloves should be avoided because they have the tendency to cause burns (Haskins, 1999). If they must be used, do not allow the heat source to contact the patient directly and remove once they cool to patient temperature as they will begin to absorb heat from the patient at that time.

**Consequences of hypothermia**
- The release of catecholamines in response to the stress of a decreased body temperature
  - Subsequent vasoconstriction, tachycardia and hypertension increase post-operative morbidity
- Coagulation deficiencies
  - Hypothermia impairs platelet function, decreases coagulation pathways and increases fibrolysis
- Decreased wound healing
  - Thermoregulatory vasoconstriction reduces wound oxygen tension, impairs oxidative killing by neutrophils and reduces collagen deposition
  - Hypothermia directly impairs immune function and increases post-operative wound infections (Sessler, 2006)
- Reduction in necessary amount of inhalant
  - Hypothermia increases solubility and decreases clearance leading to the significant potential for anesthetic overdose
- Hypothermia leads to post-operative shivering
  - Shivering greatly increases metabolic oxygen consumption and when coupled with residual respiratory depression and atelectasis, hypoxemia is likely.

The intelligent use of mechanical monitors will aid the clinician in supporting patients during times of compromised homeostasis as seen under anesthesia. Respecting the guidelines put forth by the ACVA to continuously monitor circulation, oxygenation, ventilation and body temperature will improve the practice of anesthesia and reduce patient morbidity by providing positive anesthetic outcomes.
The Ins and Outs of the Anesthesia Machine
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The anesthesia machine
The components of the anesthesia machine work together to deliver controlled amounts of oxygen and anesthetic gas to a breathing system. Anesthesia machines are designed to deliver volatile anesthetics in the presence of oxygen. One hundred percent oxygen is commonly used to deliver anesthetics for the delivery of adequate amounts of O2 to tissues. Oxygen delivery is reduced because anesthetics reduce tidal volume and cause some degree of respiratory depression.

Oxygen cylinders
Oxygen is often obtained as compressed gas in a pressurized cylinder. Pressurization is necessary to fit a large amount into a relatively small container. Various sized tanks are available and each size is designated with a letter A-H but the most commonly used sizes are E cylinders and H tanks.
When the cylinders are full they contain 2200 psi of gas with varying volumes based on size:

E-cylinders
- Small and easy to carry
- Holds ~770 L of oxygen
- Commonly found attached to an anesthetic machine

H-cylinders
- Huge and heavy
- Holds ~7000 L of oxygen
- Commonly found in a closet or chained to a wall

Other medical gases can be obtained in compressed cylinders as well. To keep all medical gases organized they are each assigned a cylinder color.
- Oxygen is green in the US, white in Canada and Europe
- Nitrous oxide is blue
- Air is yellow in the US, grey, white and black in Canada and Europe
- Nitrogen is black
All knobs, hoses and connectors associated with these gases should also be of the designated color.

Pressure gauge and regulator
Located on the cylinder yoke, this apparatus provides a safe, constant operating pressure within the machine regardless of the pressure in the tank. It also has a gauge connected to it that displays the tank pressure. The volume of gases that are not liquid at room temperature can be determined based on the tank pressure. The volume of gas is proportional to the pressure in the tank. For example, if a full tank is 2200 psi and contains 600 liters of gas then a half full tank will have a pressure of 1100 psi when it contains 300 liters of gas.

Flowmeter
The flowmeter is made up of a graduated glass tube and some sort of float (ball or plumb bob). It is typically expressed in L/min or mL/min. Oxygen enters the bottom of the flowmeter and exits the top. It allows the anesthetist to adjust the O2 flow rate. The flow of oxygen can be increased to speed the change of inhalant concentration in the machine.

Time constants (TC)
A time constant represents the volume of the machine in relation to the flow of gases. It takes approximately 3 time constants to see a 95% change in the concentration within the system when a change is made to the vaporizer. A typical small animal anesthesia machine volume (components of breathing system) will have around a 5 L volume and is dependent on the size of the absorbent canister, breathing bag size and diameter and length of the breathing hoses. Math

- $3 \times TC \times 5 \text{ L} / 1\text{L/min} = 15 \text{ min}$ With a flowrate of 1 L/min it takes 15 min to approach a steady state
- $3 \times TC \times 5\text{ L} / 2\text{L/min} = 7.5 \text{ min}$ With a flowrate of 2 L/min it takes 7.5 min to approach a steady state

Factors affecting changes in anesthetic concentration include hypoventilation and apnea. By assisting ventilation you facilitate gas exchange, this includes oxygen as well as anesthetic

CAUTION! Increases in flowrates should always be monitored closely because an increase in system flow also means an increase in flow to patient. Gases are dry and cool and can be very drying to respiratory tract and will cool patients very quickly. Never close
the pop-off valve when you have higher flows because pressure builds much quicker so you have less time to react! High flows waste anesthetic gas and oxygen and money!

**Vaporizers**  
Vaporizers are responsible for converting liquid anesthetic to a gas. It adds controlled amounts of anesthetic gas to O₂ and it can only release anesthetic in the presence of oxygen (or N₂O). Each vaporizer is calibrated for a specific agent based on that agent’s vapor pressure. It is essential to never fill a vaporizer with an anesthetic other than the one it is intended for.

**Oxygen flush valve**  
This button allows fresh oxygen to by-pass vaporizer and enter breathing circuit directly at 30-50 L/min. It is a handy but dangerous component because over pressurization is easy.

**Flush valve CAUTION**  
- Use with extreme care or not at all when the circuit is connected to a patient  
- NEVER use with a non-rebreathing system  
- NEVER use when pop-off is closed  
- DO NOT use in small patients  
- The use of this valve decreases anesthetic concentration in the system

**Fresh gas outlet and common gas inlet**  
The gas mixture (O₂ and anesthetic) exits the vaporizer and machine at the fresh gas outlet. The mixture follows a flexible tube and enters the breathing system at the fresh gas inlet. This port is necessary to hook up a non-rebreathing circuit.

**Flutter valves**  
Fresh gas enters the one-way inspiratory valve on its way to the patient and then is exhaled through the one-way expiratory valve. These valves are only utilized with a circle system to allow for re-breathing of gases.

**Pop-off valve/APL**  
The pop-off or adjustable pressure limiting valve prevents build-up of pressure or volume within the circuit. Most will vent pressure at 2 cm of water. Allowing pressure to build-up may impede venous return or cause barotrauma. It is good practice to always keep a hand on the pop-off valve (when you close it) to make sure that it gets re-opened. There are pop-off occlusion valves are available (SurgiVet.com).

**Carbon dioxide absorber**  
The carbon dioxide absorber contains absorbent granules that remove CO₂ from expired gas. Any gas that gets returned to the patient passes through the carbon dioxide absorber on its way back. Utilization of the carbon dioxide absorber lowers fresh gas flows, reduces waste of anesthetic and oxygen, lowers the overall cost of anesthesia by allowing for the re-breathing of gases.

Carbon dioxide absorbent is made up of granules of calcium hydroxide or barium hydroxide. When they come into contact with CO₂ an exothermic reaction takes place that produces both heat and water. The granules must be changed when exhausted. Many formulations have a pH color indicator to alert when to change it. Granules start off white and may change to lavender or blue when they have had their fill of carbon dioxide. This is a short-lived chemical reaction allowing spent granules to revert back to white once they are no longer being bombarded with CO₂. There are formulations available that elicit a permanent color change. If the status of the granules is in question, handle them; fresh granules can be easily chipped and crumbled whereas spent granules are very hard and brittle.

**Manometer**  
The manometer measures the pressure of gases within the anesthetic circuit and patient. This gauge is very useful when breathing for a patient or setting up a ventilator and should be referenced with each manual breath.

**Scavenging system**  
The scavenging system collects waste gas and disposes of it. Two types of scavenging systems exist, passive disposal made up of non-circulating ventilation systems or activated charcoal and active systems consisting of a central vacuum.
Components of breathing system

The breathing system is comprised of everything after the common gas outlet that breath passes through. This includes the breathing hoses, breathing bag, CO2 absorber, and scavenging system. The breathing system functions to deliver oxygen and anesthetic gas to the patient, to remove CO2 from exhalation and to facilitate manual ventilation.

Anesthetic systems/circuits

The two types of anesthetic circuits available include circle or rebreathing systems and the non-rebreathing circuit. The circle system is commonly reserved for patients greater than 7 kg and it utilizes the inspiratory and expiratory valves as well as the CO2 absorber. The non-rebreathing system is most often used for patients less than 7 kg and bypasses the bulk of the anesthesia machine utilizing only the flowmeter and vaporizer.

Circle systems

‘Y’ hoses: Two legs connected by a “Y” at the patient end
- Universal F: One coaxial tube that connects to inspiratory/expiratory valves using a small hose

Non-rebreathing systems

- No exhaled gas is returned to patient
  - Adequate oxygen flows required to prevent rebreathing of gases
  - 250-500 ml/kg/min
- Evacuated by scavenging based on O2 flows
- No CO2 absorber
- Many types, two common are both modified Mapleson D
  - Ayers T
  - Bain

Circle system vs. Non-rebreathing system

- Circle system
  - Low flows
  - Rebreathing of gases
  - More economical
  - Recycled air is warmer and humidified than fresh gas
  - May be cumbersome
  - More resistance to breathing for small patients
- Non-rebreathing system
  - Little resistance to breathing
  - Does not require a CO2 absorber
  - Allows inspired concentrations of anesthetic to be changed rapidly
  - Promotes hypothermia and drying of respiratory tract
  - Can be wasteful if used in larger patients (high flows)

Breathing bags

Breathing bags (also called rebreathing bags or reservoir bags) should hold a minimum of 60 ml/kg of the patient’s body weight. This can also be figured out by multiplying 5-6 X tidal volume (10-15 mL/kg). When choosing a bag size it is important to always round up. An appropriately sized bag allows patient to take a large breath, it allows the anesthetist to observe breathing and to breathe for the patient. The size of the bag does matter, if the bag is too big it impairs monitoring of breathing rate, adds volume to machine and slows changes in inspiratory anesthetic concentrations when settings are altered. If the bag is too small the animal is unable to take an adequate breath because the bag collapses on inspiration.

Endotracheal tubes

Many endotracheal tubes are intended for one-time use only. The biggest drawback of reusing them is that the cuffs tend to wear out over time. All ET tubes should be checked for leaks prior to each use. To do this, make sure the cuff inflates and stays inflated. You can also place an inflated tube in a bowl of water and look for leaks/bubbles. All leaky tubes should be discarded.

Mechanical dead space

Mechanical dead space is the area where bi-directional flow takes place. This includes the endotracheal tube and anything between the Y-piece and the ET tube such as the CO2 monitor adapter, swivel adapter, elbow etc. Excessive pieces should be kept to a minimum in small patients. It is important to note that hose length does not contribute to dead space but can increase the resistance to breathing.
As shelter intakes show a decreasing trend and live release rates tend to be increasing across much of the country, one population that is still at risk for euthanasia is pre-weaned kittens. It is common for shelters to take in multiple litters of kittens in a single day, often brought to the facility by Good Samaritans. Rarely do people realize that many of those kittens will be euthanized due to a lack of resources available to care for them and a lack of homes ready to adopt them when they reach an age at which they can transition to the shelter population. As shelters are successfully managing their feline populations and spay/neuter resources are helping to decrease intake, we are in a place now where saving this vulnerable population is a possibility.

It is not uncommon for technicians or assistants in shelters or private veterinary practices to occasionally bring home a litter of kittens to bottle feed. It is almost a rite of passage for any young technician. Inevitably, there are a few “foster failures” in every group of veterinary professionals. When an organization decides to make saving kittens a priority, a formal foster program will make the process run smoothly without putting the burden on the shelter employees.

How do shelters know if they can reasonably start a kitten foster program? First, you need the resources to care for kittens in foster. Online wish lists and “kitten showers” are excellent ways to solicit donations for fosters, but the organization needs to be able to cover all costs for the kittens in foster if donations were to cease. Shelter resource capacity also need to be considered. All animals that leave for foster will eventually need shelter services that may already be stretched. Consideration needs to be given to the capacity of the medical team to provide vaccinations, exams, treatments, counseling, and surgery. Foster kittens will also occasionally need emergency care, and if a staff member isn’t available a means to pay for emergency veterinary care will need to be in place.

Next, you need to ensure every cat you put into foster will be able to find a home when it returns to the shelter. Foster parents often get attached to their temporary family member, and if your shelter is still euthanizing healthy cats for “space” it is not a good time to start a foster program. Not only do you risk upsetting the foster parents and potentially damaging the shelter’s reputation, you also expend a lot of resources on cats that may not be able to find permanent homes. If the shelter live release rate for weaned kittens is not near 100%, the focus needs to be on improving that number before trying to save neonates. If your shelter has a very high live release rate for kittens and the resources and volunteer base to run a foster program, it’s time to move on to the next step.

Finding a foster coordinator early in the process is important. This can be a volunteer or a part or full time employee depending on the size of the organization. Many shelters have a very dedicated volunteer or unofficial foster parent that they can ask to coordinate the effort. This person serves as the link between foster parents and the shelter so it is important that they understand the organization and all aspects of raising foster kittens. The coordinator will also need to understand vaccination and deworming schedules so they can ensure kittens are kept up to date on preventatives.

The veterinary team (with the assistance of a consulting veterinarian if the shelter does not employ one) will need to write a thorough foster manual that details care for kittens and what to do with minor maladies. Examples of emergency conditions can also be noted in this manual. A detailed foster manual that clearly defines the responsibilities, protocols, and expectations will provide clarity and minimize problems as well as liability.¹ The more detailed the foster manual and the more educated the coordinator, the fewer phone calls the veterinary staff will need to field in the future.

Social media has become the primary means for many shelters to recruit foster families. Referrals from friends, newspaper ads, and youth groups are other effective means for finding volunteers. It is very helpful to have those with previous fostering or animal care experience be mentors to those without as much knowledge or experience. It is also helpful to have a mix of lifestyles and personalities in your foster group. For example, and older retired person is great to have for a kitten that needs socialization and a lap to sit in much of the day but they may not want to stay up all night to bottle feed. A younger student that stays up late to study may be more equipped to take care of a bottle baby.

Orientation classes are a great tool to educate foster families. Classes should be taught by a member of the veterinary staff who has knowledge of neonatal care. These presentations introduce new fosters to the organization and its mission and educate them on the importance of following the recommended guidelines. It is a good idea to teach volunteers what “normal” looks like, including going over milestone charts and describing how quickly kittens normally gain weight. This is also a good time to set realistic expectations. If the organization cannot or will not use resources for ICU care or specialized treatments, that needs to be explained up front. If kittens with significant medical issues will be euthanized rather than treated, that needs to be explained so foster families have realistic expectations before agreeing to bring home a kitten.

I also recommend fosters sign a contract. This will vary depending on the type of shelter, legal requirements in your area, and the requirements of fosters. For our municipal organization, the city attorney asked that all fosters sign a contract stating that they understood the risk of zoonotic disease, that they would follow all instructions for medical care, and that they would return the kittens.
to the shelter upon request. If foster parents will be required to have their pets vaccinated or have their cats tested for FIV and FeLV this should also be in the contract. Many organizations also ask foster to attest to not fostering for any other shelters.

Once the coordinator, supplies, and families are in place, you just need kittens. In order to move the kittens out of the shelter quickly, it is good to keep a list of which foster parents are immediately available. The foster parents should know that they are next in line so they can be available to pick up kittens should they arrive. The faster kittens can get out of the shelter, the more likely they are to not develop shelter-acquired illness. Either fosters can have supplies ready at their homes or starter kits of supplies can be ready so kittens can leave quickly. Before foster kittens leave the building, it is important that they have a record at the shelter and a record that can go with the foster that includes a specific schedule of recheck appointments for that animal(s). If the shelter has computerized records, it is easy to set reminders for their vaccinations and deworming schedule and to set a tentative surgery date.

Writing foster care protocols
As previously mentioned, a foster manual with detailed care instructions will aid the families in giving the kittens the best start in life while saving the veterinary staff time and resources. The foster manual needs to include housing, feeding, elimination, bathing, and socialization guidelines as well as a discussion of weight gain and milestones. It is also a good idea to provide answers to some common medical concerns and what to do in case of emergency.

The most important housing concern for neonates and infants is to provide warmth. Kittens do not regulate their own body temperature well in the first four weeks of life and can only maintain a body temperature about 12 degrees higher than their environment. This is due to their large body surface area with non-cornified skin, their lack of insulating fat, and the fact that they can’t vasoconstrict their peripheral vessels. Neonates are especially susceptible because they cannot shiver at birth. Overhead heat lamps, Snuggle Safe discs, and other commercially available sources of warmth can be used. Heating pads should be used with extreme caution to avoid thermal burns. Hypothermia has a negative impact on immunity, nursing, and digestion so keeping the kitten warm is vital to keeping them healthy. Foster homes should also be advised to house kittens away from unsupervised access by other pets or children and in an area that can be disinfected.

Nutritional support can be the greatest challenge for foster families. If a lactating foster queen is available, she is the best option for feeding orphans since she will provide nutrition as well as maternal care. Most lactating queens will accept the additional kittens, but it is important to place kittens with litters that are a similar size to avoid size discrepancy. Partial hand-raising is an option if a queen has recently weaned her own litter, if there is a discrepancy between the amount of kittens and the amount of lactation, or if the queen is not healthy enough to provide optimal nutrition. When orphans need to be bottle-fed, foster homes need to be supplied with sufficient formula and bottles. Orogastric tube feeding kittens is relatively simple, and some dedicated volunteers can be trained to take kittens that require tube feeding. Specific instructions for preparing and warming the formula, feeding frequency, daily weighing and monitoring of growth, positioning during feeding, and weaning need to be written in the foster manual. Signs of feeding problems and instructions for care should also be included in these manuals. Foster parents should also be warned of the risk of feeding cow’s milk and the risks associated with over-feeding. It is not uncommon for well-meaning individuals to unintentionally cause harm.

Kittens will generally start drinking formula from a dish around 3-4 weeks old. Solid food is first introduced by mixing canned food with milk replacer and offering it on a flat dish. The amount of milk replacer is slowly decreased until the kitten is primarily eating solid food. By 5-6 weeks, kittens can chew dry food and should be eating about 1/3 of their daily calories in the form of solid food. Dry food can be offered ad lib between meals of canned food. Weaning should be complete by 6-9 weeks old.

Elimination and defecation must be stimulated in kittens less than 3 weeks old. Normally this is done by the queen when she licks the kitten’s perineum, and the foster parent needs to mimic that in young orphans. Before and after each feeding, the foster parent needs to rub the kitten’s lower abdomen, genitals, and rectum with a cotton ball dipped in warm water. Kittens typically urinate with every stimulation and defecate at least one time per day. When the kitten starts eliminating on its own, it should be given a litter box with shredded paper or non-clumping litter. Stool should be placed in the litter box until the kitten learns to eliminate in the litter box.

Socialization is an important aspect of hand-rearing kittens that cannot be overlooked when developing care protocols. The sensory phase begins at approximately 3 weeks and lasts until approximately 2 months old. Opportunities to gain desirable conditioning during this time will improve the kitten’s ability to be a well-socialized companion animal in the future. Frequent, careful handling of the kitten is important to help it become accustomed to human handling. This becomes an important consideration when shelters attempt to socialize the kittens of feral queens admitted to the shelter.

As long as the kitten is healthy and gaining weight, it should be scheduled for surgery at about 6-8 weeks old. A plan should be in place for adoption following surgery. Many shelters use fosters as the primary source of off-site adoptions rather than having them enter the shelter population. It is also common for foster parents to be adoption ambassadors and find homes for their houseguests. It is important that the population management and flow through foster homes is managed in much the same way as the shelter population as a whole. It is also important for the kittens in foster to be included in plans for shelter population if they are to return to the shelter for adoption and placement.
Medical protocols
A medical exam should be performed at the shelter before kittens are sent into foster. Exams are important for many reasons, one of which is to identify special needs cases that may need to go to foster homes more equipped to handle their needs. High risk kittens should go to homes that can provide for their special needs and could handle the loss of a kitten without mental anguish. Weighing newborn kittens will assist with determining their likelihood of survival. Normal birth weight kittens are typically 100g +/- 10g, and birth weight is the best indicator of neonatal survival. One study of 477 kittens determined that the survival rate of low birth weight kittens was only about 40% while the survival rate of normal birth weight kittens was about 68%. For this reason, low birth weight kittens should be considered higher risk and placed in appropriate homes. Any kitten that does not have the benefit of colostrum or a replacement should also be considered high risk. Singletons can also be considered “special needs” because of the lack of a litter mate to provide warmth and socialization. If the history of the litter is unknown, singletons are also thought to carry some extra risk because infectious disease may be the underlying reason for arriving at the shelter alone.1

The initial medical exam includes a head to toe exam that checks for any congenital abnormalities. Examples of abnormalities seen in kittens include cleft palate, imperforate anus, gastroschisis, omphalocele, and abnormally large fontanels. Congenital defects will range from very mild to those that require euthanasia. Where the line is drawn will depend on the resources available to care for the kittens as well as the availability and experience of the consulting veterinarian. Age can be estimated by using weight and milestone charts. Kittens and queens also need to be checked for overall health and any signs of infectious disease. A Wood’s lamp exam should also be performed before placing the kittens in a home.7 Queens need to be tested for FeLV and FIV before being placed in a home, and kittens should be tested for FeLV before foster when possible.

A broad spectrum dewormer should be given to all kittens over 2 weeks old and repeated every 2 weeks until 2 months of age. If the kitten stays in foster beyond 2 months old, it needs to be dewormed monthly.1 Ponazuril is a useful coccidiocidal medication that can be given to young kittens before going to foster homes. Kittens and queens should be carefully checked for any ectoparasites. In very young kittens the safest way to manage flea infestations is to bathe and manually remove them. Fipronil spray (Frontline Spray, Merial) is labeled for use in kittens as young as 2 days old in some countries.6 If the kittens are over 4 weeks old, an MLV FVRCP vaccination should be administered. Kittens need to be revaccinated every 2 weeks while in the shelter’s care until 16-20 weeks old.10

You will need to work with the veterinarian to determine what medical issues constitute an emergency, something that needs to be examined at the foster’s next convenience, or something that can be watched at home. The clinical signs that fall into each group should be written clearly in the foster manual and explained at orientation so the families know what to do should something go wrong. It is a good idea to write basic flow charts or protocols for standard diagnostics and treatments for the most common conditions.

When writing flow charts it is important to remember that neonates are not just small cats. Kittens respond to disease in a limited number of ways so treatment is often symptomatic until the underlying cause can be identified. Clinically ill neonates tend to present with some combination of hypothermia, hypoglycemia, dehydration, and hypoxia. Treating these symptoms quickly can increase the chances of survival.11 Pain management is also very important in young animals because a permanent hyeralgesic response can develop without proper therapy.12 Kittens respond differently to some medications, and many drugs are not labeled for use in very young animals so it is important to check with a veterinarian before administering any medications. Also, working with neonates can be frustrating. It is not uncommon for a very young animal to die and for the veterinarians to not have answers as to why. In one study, when 107 kittens and puppies 0-7 days old were submitted to the Washington State Animal Disease Diagnostic Laboratory, a definitive diagnosis for cause of death was made only 72% of the time.13 We don’t always have all the answers, but this is especially true when working with our youngest and most vulnerable patients.

I suggest the following resources for further information and to find accurate weight and milestone charts as well as feeding recommendations:

References
15. ASPCA Professional (examples of foster contracts, applications, manuals): http://aspcapro.org/foster-care
Placement of an intravenous catheter and nasogastric intubation are two important technical skills in equine medicine. Intravenous catheters should be placed for relatively frequent, repeated boluses or constant rate infusions of intravenous medications and fluids. Nasogastric intubation should be performed as a diagnostic tool in horses with signs of colic and can be used for administration of large volumes of medications, nutrition, or fluids into the stomach.

Intravenous catheters can be placed easily placed in several vessels in the horse. They are most commonly placed in the jugular vein, which is a central vein, and are therefore “central lines”. This means that it is safer than peripheral vessels to use for large volumes and more irritating and concentrated medications. Other locations that are used for catheter placement in horses include the lateral thoracic vein, cephalic vein, saphenous vein, and digital veins. Typically, these vessels are not used for routine medication and fluid administration unless there is a problem with the jugular vein(s) (i.e. thrombosis) or another medical reason to avoid use (i.e. cellulitis or subcutaneous emphysema obscuring the vessel). The vessels in the limbs may also be used specifically for administration of antimicrobials or analgesics regionally (intravenous regional limb perfusion [IVRLP]). Placement, use, and monitoring of the catheter are fairly similar regardless of the location. There are several types of catheters that influence the method of placement. I will discuss over the needle, over the wire, through the needle/pull away catheter.

Intravenous catheters can be made of a variety of materials. These materials vary in their stiffness and reactivity, which play a role in their thrombogenicity. More thrombogenic catheters are only for short-term use. Available catheter materials, from most thrombogenic to least, include fluorinated ethylene propylene (FEP), polytetrafluoroethylene (PTFE), polyurethane, and silicone. Many of the catheters used in equine hospitals are made of FEP (short-term) or polyurethane (long-term).

Prior to placement, the site should be aseptically prepared. I generally recommend clipping if the catheter is going to be in place for more than a single injection. While catheters can be placed without local anesthetics, it is often easier, particularly in young animals and when sites other than the jugular vein are used, to place a bleb of local anesthetic. As noted, placement of an over-the-needle catheter requires the vessel well distended. If you are going to have to distend the vessel manually for yourself and you need to keep both hands free, you must either prepare an enlarged area or used a knuckle or the back of your hand for distension. As noted, placement technique will depend on the type of catheter being used:

Over-the-needle: The catheter comes over a needle that is the same length (a little longer) as the catheter and serves as a stilette. To place the catheter, the needle and catheter are put through the skin and into the vessel together. Getting into the vessel may require a quick, jabbing motion as it often moves away. Once you feel that you are in the vessel, you should check for a flash back of blood. The catheter and needle combined should then be advanced several centimeters to make sure the catheter is well seated in the vessel. Then, the needle/stilette should be held in place and the catheter fed off of the needle into the vessel.

Over-the-wire (modified Seldinger): A flexible catheter is placed into the vessel over a guidewire. To begin, a needle or short-over-the-needle catheter is placed into the vessel, well seated. A long guidewire is then placed through the needle or catheter. The needle or catheter is then removed being careful to NEVER let go of the guidewire. If necessary a vessel dilator is passed over the guidewire and into the vessel (bleeding will increase) being careful to NEVER let go of the guidewire. The catheter is then threaded over the wire and the wire is removed. Again, be careful to NEVER let go of the guidewire.

Through-the-needle/pull away catheter: A flexible catheter with a guidewire to make it more rigid is passed into the vessel through a needle or pull away catheter. To begin, a needle or short-over-the-needle pull away catheter is placed into the vessel, well seated. A flexible catheter with a guidewire is passed through the vessel and out of the vessel and a guard is placed over the needle to prevent cutting the catheter and the stilette is removed. If a pull away catheter is used, that catheter can be pulled apart leaving the catheter and stilette in place. The stilette is then removed.

Once placed, catheters should be flushed, usually with heparinized saline, to maintain patency every 6 hours. They should be monitored at those times for any signs of catheter site infection or vessel thrombosis/phlebitis. Catheter caps should be changed daily. It is important that catheters remain are clamped and capped when not in use and/or have a one-way valve in place to prevent air embolism. Particularly in the jugular vein, a large amount of air can be sucked into the vessel rapidly. A designated catheter or catheter port that is handled aseptically should be used if partial or total parenteral nutrition is being administered.

As noted above, indications for an intravenous catheter are the need for relatively frequent, repeated boluses or constant rate infusions of intravenous medications and fluids. Catheters can also be used in some cases for sampling blood if an appropriate discard device...
Shock occurs when the energy needs of cells are greater than the energy being delivered by the blood. Most commonly, there is a deficiency in oxygen delivery. Oxygen delivery is a product of oxygen content and cardiac output. Oxygen content is primarily dependent on [Hb] and SpO2 and cardiac output is the product of heart rate and stroke volume with stroke volume determined by preload, afterload, and contractility. Although conditions that affect either component can result in shock, in horses it is more common for shock to be caused by decreased cardiac output. Specifically horses are affected by conditions that result in decreased preload. The two main types of shock seen in horses that are due to decreased preload are hypovolemia and maldistribution of fluids. Examples of hypovolemic shock are hemorrhagic shock and fluid loss associated with large volume diarrhea or nasogastric reflux. Examples of maldistributive shock are endotoxic and septic shock that result in massive vasodilation and venous pooling of blood.

Clinical signs of hypovolemic and maldistributive shock are similar and associated with poor perfusion and decreased intravascular volume as well as the body’s attempts to improve oxygen delivery. Physical examination findings associated with poor perfusion and decreased intravascular volume are cool extremities, prolonged jugular refill, prolonged capillary refill time, poor pulse quality, and decreased/absent urination. Physical examination findings associated with the body’s attempts to improve oxygen delivery are increased heart rate, increased respiratory rate, decreased/absent urination, and production of concentrated urine. If available, hand held lactatometers and/or blood gas monitors can be used to assess plasma lactate concentrations to support inadequate oxygen delivery.

The goal of emergency fluid therapy is rapid restoration of preload, cardiac output, and oxygen delivery. This requires use of the intravenous route of fluid administration. A wide diameter (10-14g), short catheter with large bore fluid administration sets will maximize the speed of delivery. Depending on the type of fluid chosen and packaging, pressurized administration may be possible.

The two main alternatives for rapid volume resuscitation are: 1) using a relatively small volume of hypertonic saline (2-4 ml/kg or 1-2 L/adult horse [not generally recommended in foals]) and/or synthetic colloids (5-10 ml/kg or 2.5-5 L/adult horse or 250-500 ml/foal) to borrow/pull fluid from the interstitium into the vascular space followed by administration of larger volumes of isotonic crystalloids (at least 20 ml/kg) or 2) using isotonic crystalloid fluid alone. In either case, the “shock dose” of 80-90 ml/kg can be used as a guideline for the maximum amount of isotonic crystalloids to administer as a bolus. In general, it is rare to need the entire “shock dose”. I recommend starting with a 20 ml/kg bolus (10 L/adult horse or 1L/foal), reassessing clinical signs/physical examination parameters, and determining if additional boluses are required.

In horses with diseases that are associated with protein loss, the use of colloids in the resuscitation plan might have additional benefits. In the case of hemorrhagic shock, whole blood should be considered as part of the fluid resuscitation plan.

Nasogastric tubes also come in a variety of shapes and sizes. In most cases a fairly large diameter, firm tube will be used, but for more longterm use (i.e. feeding tubes), small diameter, flexible tubing with a stilette for placement is preferable. It is of vital importance to check for two things prior to administration of any medications or large volumes of fluids: you are in the right place (stomach, not lungs) and there is no significant (>2L net) reflux present. When passing a tube there are several ways to check that you are in the right place—you can often see the tube pass down the left side of the neck in the esophagus, you may be able to feel the tube in the stomach (if it is in the trachea and you shake it, it will rattle), there is negative pressure in the esophagus, and the stomach should have gas/contents. With a smaller, more flexible tube, these things may be more difficult to assess. In these cases, radiographs are the most accurate way to confirm placement.

Proper restraint is key to successful, safe nasogastric intubation. Physical restraint methods such as twitches or stocks may be adequate, but sedation should be used if necessary. Local anesthetic lubricant is available, but if it reaches the larynx it will make it difficult to get the horse to swallow. The most sensitive, and therefore most objectionable, part of passing a nasogastric tube for the horse is through the nasal passage. This should be done as quickly and smoothly as possible. Prior to passing the tube, get an idea of how far in you will have to go to get to the pharynx (you can measure from the nostril to the medial canthus of the eye or empirically in an adult horse its about 8-12 inches). The tube must be directed VENTRAL and CENTRAL (medial) through the nasal passage. Your hand should be placed over the horses nose with the thumb holding the false nostril out of the way and pushing the tube VENTRAL and CENTRAL. Be careful not to occlude the other nostril. If you run into a firm/boney structure it is the ethmoids—don’t keep pushing or you will get a pretty good nosebleed. In order to encourage the tube to go to the esophagus rather than trachea, keep the horse flexed at the poll. Horses will often swallow immediately as you get back to the larynx. If not gently “bumping” the larynx will generally encourage them to swallow. Be patient, but if it does not seem to be working, try going up the other nostril. When using small, flexible tubes, sometimes it is very difficult to get the horses to swallow. You can use an endoscope to confirm/assist placement in these cases.
As noted above, indications for nasogastric intubation include a diagnostic tool in horses with signs of colic and administration of large volumes of medications, nutrition, or fluids into the stomach. Oral fluids are at least as effective as intravenous fluids for correcting dehydration and maintaining hydration. They are less expensive and do not have the risk of vascular complications. Administration of fluids required for a 24-hour period can be dividing into multiple doses throughout the day or given as a constant rate infusion via nasogastric tube. No more than 6-8 L of fluids should be given at one time point. There are three main components to consider when calculating the volume of fluid for a 24-hour plan: maintenance, dehydration, and ongoing losses. In adult horses, maintenance requirements are 50 ml/kg/day and in foals, maintenance requirements are 80-100 ml/kg/day. In horses, estimating dehydration is very difficult. Traditional clinical signs of dehydration such as skin tent and dry/tacky mucus membranes have been shown to be completely inaccurate. Ongoing losses may be easy to measure, such as with horses that are producing nasogastric reflux, or difficult to determine, such as with horses with diarrhea or polyuria. Nasogastric tube administration is commonly used for treatment of horses with large intestinal impactions. There are several mechanisms of action that cause loosening and/or softening of gastrointestinal contents by laxatives. Laxatives used in horses include bulk laxatives that provide volume and help retain water (psyllium), surfactants that help incorporate water and fat (dioctyl sodium sulfosuccinate [DSS]), lubricants that are slippery and decrease water absorption (mineral oil), and hydrating agents that help attract and retain water (isotonic balanced electrolyte solution or magnesium sulfate). DSS can be irritating and may facilitate the absorption of mineral oil or magnesium sulfate and associated toxicities. Overdoses of magnesium sulfate alone can also result in magnesium toxicity. An isotonic balanced electrolyte solution has been shown to result in the largest increase in fecal water. Water has also been shown to effectively hydrate ingesta. Psyllium is frequently used for the prevention and treatment of sand impactions. Mineral oil can be used as a marker for intestinal transit in addition to it’s laxative activity.
Talking Toxic:
Things Technicians Need to Know about Toxicology and Common Intoxications
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The goal of this lecture is to provide veterinary technicians with important information pertaining to veterinary toxicology, in general, several small animal intoxications, specifically, and currently available toxicology-related electronic resources.

In this presentation we will review the following
- Some fundamental veterinary toxicologic definitions and principles
- The basics of the clinical management of suspected veterinary intoxications
- A discussion of several “common” small animal intoxications
- General veterinary toxicology electronic resources

Fundamental definitions for veterinary toxicology
- **Toxicology**- The study of poisons
- **Poison**- Any solid, liquid or gas, which can, in sufficient quantities, adversely affect a biological system
- **Toxin**- A poison of biological origin
- **Toxicosis**- The state of being intoxicated
- **Dose**- The terms for the total amount of a toxicant to which an animal is exposed
- **Dosage**- The amount of toxicant per unit body weight, often per unit of time

Basic principles of veterinary toxicology
- “The Dose Makes the Poison.” → Dose-Response Relationship- Generally, the greater the dose/dosage of a given toxicant, the more severe the clinical signs → Exposure to toxicants and/or their metabolites must be sufficient to cause intoxication.

OVERVIEW of basic clinical management of suspected intoxications

REVIEW of basic workup of a suspected intoxication (“malicious” OR “accidental”)
- Often an EMERGENCY!!!
  - Simultaneously incorporates aspects of treatment and diagnosis
  - FIRST THINGS FIRST/MOST CLIENTS PREFER PET SURVIVAL OVER CONFIRMED Dx!!!
    - Determine what is in “Toxicant X” (NEED LABELS)/Jump right to treatment???
  - Possible rationale for having yourself or technicians cloned!!!
- Signalment + Clinical Signs/Clinical Circumstances
  - WHICH PETS (names, breeds, ages+)/WHAT ARE THE CLINICAL SIGNS??? (video?)
  - Exposure to “Toxicant X” → HOW MUCH?/WHEN?/WHERE? (How reliable is info?)
- Problem List
  - WHAT’S WRONG? SOME CLINICAL SIGNS “TOXICANT X” SPECIFIC/SOME GENERIC
  - Physical examination and STAT laboratory testing, IF proximate to patient
- “BIG PICTURE” PROBLEMS
  - SUMMARY OF CRITICAL LIFE-THREATENING ISSUES AND TARGET SYSTEMS/ORGANS
    - TREAT THE PATIENT NOT THE POISON, UNLESS TOXIC EXPOSURE OBSERVED!!!
    - STABILIZATION OF THE PATIENT IS NUMBER ONE PRIORITY!!!

OVERVIEW of Basic Clinical Management of Suspected Intoxications (CONTINUED)

REVIEW of Basic Workup of a Suspected Intoxication (“Malicious” OR “Accidental”/continued):
- D.A.M.N.I.T.
  - **D** = Degenerative
  - **A** = Anomaly
  - **M** = Metabolic
  - **N** = Nutritional/Neoplastic
  - **I** = Infectious/Inflammatory/Idiopathic
  - **T** = Traumatic/Toxic
• POISONING SHOULD BE SUSPECTED WHEN:
  o DIRECTLY OBSERVED TOXIC" EXPOSURE/"TOXICANT X" IN VOMITUS OR GI TRACT
  o Sudden death/Similar clinical signs in MULTIPLE animals
  o Rapid onset of afebrile syndrome or sudden death of a previously healthy animal.
  o Signs of unknown etiology/Other causes ruled out
  o Recent change in diet or environment
  o Neighborhood feuds/Love gone bad/Pet owner often certain of “CULPRIT”
  o Very small, young, mean, noisy, annoying, and/or stupid animals!!!
  o Might be a “Darwinian phenomenon” OR “aliens”, “bikers”, “local meth labs”
  o IF “OBVIOUS” INTOXICATION, GO IMMEDIATELY TO EMERGENCY Rx!!!

• ONCE ANIMAL STABLE/DIAGNOSIS STILL UNCERTAIN: CONTINUE STEPWISE WORKUP
• List plausible toxic/Some not so toxic differentials
• Most likely FINAL “Toxic” diagnosis and WHY IS IT “TOXICOSIS X”
  o Not always possible to CONFIRM Dx/Looking and acting like “Dx” might be sufficient.
• Helpful to know toxic mechanism(s) of action (MOA) of “Toxicant X”
  o Good correlation of “Toxicant X"MOA with Problem List supports “Toxicosis X" Dx
  o Can incorporate knowledge of toxic MOA into successful treatment plan
• “Toxicosis X” successfully treated/Diagnosis of “Toxicosis X” confirmed
  o Sometimes not possible to do both/”Toxicosis X" Dx confirmed by laboratory testing

DETAILED clinical management of a suspected intoxication, including Rx/Dx
EMERGENCY Rx/TREATMENT of suspected intoxications
• REMOVE THE ANIMAL(S) FROM THE SOURCE OF “TOXICANT X”!!!
  o MIGHT BE REMOVAL OF THE SUSPECTED “TOXICANT X" SOURCE FROM ANIMAL!!!
    ▪ Baths for cutaneous exposures to “Toxicant X” (especially the paws of cats)
    ▪ IF “X” eaten, Emesis/GI lavage or containment/Activated charcoal ± Cathartics
  o House/Garage/Kennel/Yard/Junk management
• IMMEDIATE VETERINARY CARE!!!
  o TREAT PATIENT NOT THE POISON, UNLESS “TOXICANT X" EXPOSURE OBSERVED!!!
  o STABILIZATION ABCs
    ▪ Airway/Breathing/Circulation/Depression/Excitation/Fever/Hypothermia
    ▪ Supportive care
• Decontamination/Antidotal therapy AND/OR Lipid Infusion for specific Intoxications
  o Decontamination is ANOTHER way to separate the source from the animal!!!
  o Depends on route of exposure/Stage of intoxication/Specific antidote (IF available)
    ▪ Bath for cutaneous exposures to “X”/Emesis+ IF NO contraindications (“X” eaten)

DETAILED clinical management of a suspected intoxication, including Rx/Dx (CONTINUED)
Some “guidelines” for stabilization ABCs
• Ensure that the Airway is patent
  o Awareness of obstructions/bronchoconstriction
• Establish normal Breathing
  o Awareness of breathing problems/impaired gas exchange
• Correct Circulation deficits
  o Fluid/Electrolyte/Acid-base imbalances + various anemias with different etiologies
• Control Depression of CNS
  o Correct metabolic disturbances/neurotransmitter imbalances
• Control Excitation of CNS
  o Do nothing if very mild
  o Correction of electrolyte imbalances and possible glucose deficits
  o Anticonvulsant medications
• Bring down Fever
  o Avoid use of NSAIDs for toxicant-induced hyperthermia
• Treat Hypothermia
  o No ice water baths

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Supportive care

- Maintenance of vital functions/fluid therapy
  - Might also be used in stabilization
- Antibiotics/Analgesics/Diet modifications/Client communication/Client education

Patient stabilized/Dx uncertain → fine-tuned clinical signs/clinical circumstances reassessment

- Might be onsite/animal side in clinic/remote by telephone, text, or ???
  - Access to both premises and animals is ideal!!!
- A thorough and accurate history is often the key!!!
  - Prevents the chasing of many wild geese AND “innocent” parties!!!
- Information might be relayed by phone or be secondhand.
  - The accuracy of such information might be questionable OR “slightly” exaggerated!!!
- Asking the right questions is extremely important!!!
  - Might need to ask the same question several different ways!!!
  - Might need to seek out the individual really in the know OR the “CULPRIT”!!!
- A good physical examination is of critical importance and may need to be repeated!!!
  - It is perfectly ok to use a stethoscope and a thermometer!!!
- So is careful observation of clinical signs/circumstances!!!
  - Direct observation of patient might yield very different information!!!!
  - Impacted by scheduling/lighting/finances/crowds/decomposition/”karma”!!!

CONFIRMING diagnosis of suspected intoxications (NOT ALWAYS POSSIBLE)

- DIRECT OBSERVATION OF “TOXIC” EXPOSURE/”TOXICANT X” IN VOMITUS OR GI TRACT
- PRETTY MUCH TRUMPS EVERYTHING ELSE IF “TOXICANT X” IS IDENTIFIABLE
- BRING IN CONTAINERS/LABELING/MSDS/ANY AVAILABLE DOCUMENTATION
- MIGHT BE ALL DONE WITH Dx/EXCEPT ”LEGAL” CASES REQUIRING Dx CONFIRMATION

“Common” small animal veterinary intoxications

- Antifreeze
  - Poison
  - Patients
  - Pathogenesis
  - Problems
  - Plan
- Chocolates, Energy Drinks, and Sugar-free Gum
  - Poisons
  - Patients
  - Pathogenesis
  - Problems
  - Plan
- Mouse, Rat, Gopher, and Mole Baits
  - Poisons
  - Patients
  - Pathogenesis
  - Problems
  - Plan
- “Legal” and Illegal Drugs
  - Poisons
  - Patients
  - Pathogenesis
  - Problems
  - Plan

Veterinary poison control center websites

- [http://www.petpoisonhelpline.com/](http://www.petpoisonhelpline.com/) separate tabs for pet owners and veterinarians
- [https://www.aspca.org/pet-care/animal-poison-control](https://www.aspca.org/pet-care/animal-poison-control) for pet owners
- [http://aspcapro.org/poison](http://aspcapro.org/poison) for pet professionals
• Phone apps available and phone consultations available on a fee for service basis

U.S. FDA website providing helpful information for pet food recalls
• http://www.fda.gov/AnimalVeterinary/default.htm
  o Website for Food and Drug Administration
  o Go to Animal & Veterinary tab for veterinary drug-specific information
  o Useful regulatory information on pet food contaminants/recalls
  o Mechanism for reporting adverse drug reactions/pet food-related incidents
  o Other helpful information on veterinary medications
  o Also useful information on human medications
Farm animals (small ruminants, camels and pigs) are increasingly being kept as pets. This means that farm animals may be treated by urban and small animal veterinary clinics. This article will cover important aspects of management and common diseases. In general most diseases of farm animals are as a result of incorrect management and/or nutrition. While these animals are kept as pets, the laws regarding drug use in food animals must be followed. More information regarding appropriate use of drugs in food animals can be found on the Food Animal Residue Avoidance Databank website www.FARAD.org.

**Pigs**

Pet pigs come in a variety of shapes and sizes. The majority of pet pigs are still the Vietnamese Pot Belly pigs that weigh over 100lbs at mature weight. Mini pot belly pigs are supposed to reach a maximum weight of 60lbs and teacup or micro pigs 30lbs mature weight. However, due to breeding, the mature weight of these smaller pigs may not be consistent. Other pet pigs seen in the veterinary clinic are commercial swine breeds and Kune Kune pigs (originally from New Zealand). Occasionally wild hogs may be kept as pigs, however, due to disease risks to humans and domesticated animals this cannot be recommended. Due to the fact pigs are commonly kept as pets, they are often presented to small animal veterinarians. The Food and Drug Administration considers them food animals, and therefore drugs prohibited in food animals should not be administered. The lifespan of pot belly pigs is approximately 15-20 years.

A good source for pet pig drugs and dosages is the Exotic Animal Formulary by Carpenter 4th Edition or the Bayer Compendium of Veterinary Products where one can find drugs labeled for use in pigs. Smaller pigs can be held or restrained and lead using a dog harness. Larger pigs may need to be restrained using a pig board. Snares should never be used on pot belly pigs as their nasal bones can easily be broken.

**Routine care**

As with other species, it is recommended to vaccinate pot belly pigs. We recommend vaccination against Erysipelas, the respiratory diseases (including *Actinobacillus Pleuropneumoniae, Bordatella bronchiseptica, Haemophilus parasuis and Pasteurella multocida*) and leptospirosis. Vaccines require 2 initial doses followed by yearly boosters. Rabies vaccination with IMRab3 is also commonly administered, although the risk of rabies is low in pigs. The dose and route of administration should be followed as per the label. Decreasing the dose in smaller pigs is not recommended. Owners should be warned for adverse reactions and veterinarians and technicians prepared for anaphylaxis, although the risk is very low. Pigs that have access to outside should be wormed regularly for roundworms. Ectoparasites (mange) is common in pet pigs. It is recommended that all pet pigs be castrated or spayed. Castration is important for behavioral control, and spaying to prevent uterine tumors and unwanted pregnancies. Castration can be performed from a few weeks old under local anesthesia and sedation or general anesthesia. Closed castration with skin closure should be performed. Ovariohysterectomy is recommended at around 3-5 months old and is performed under general anesthesia. Depending on the living environment of the pig, foot trimming may need to be performed every 3-12 months. It is important for the owner to initiate this early in life to make is as low stress as possible for the pig (and the technician!). The pig should have access to concrete areas or other rough surfaces to help wear down the hoof. For pigs that require foot trimming in a veterinary clinic, it is recommended to perform this under general anesthesia. Sheep foot trimmers or a dremmel tool work well to trim feet. Pigs ears may need to be cleaned routinely. Use cleaner labeled for dogs on cotton wool or gauze, avoid putting fluid into the ear due to the difficulty in draining the ear, resulting in temporary deafness or head tilt.

**Nutrition**

Good nutrition is paramount in pet pig care due to the ease they can become obese resulting in lameness, skin and eye issues. There is proprietary pet pig food available which should be fed by weight at 1-2% of body weight. The diet can be supplemented with green leafy vegetables and grazing grass. Fruits can be offered in smaller quantities. Human food should not be fed, especially foods high in salt. The body condition and weight should be monitored regularly and the diet adjusted accordingly. It is recommended to feed the youth feed until 18-24 months old, as this formulation has essential nutrients needed for growing pigs (e.g. lysine). After this time the adult or senior diet can be fed. The senior diet has a lower calorie content and may need to be introduced earlier in cases of obesity. Many piglets are weaned early (<4 weeks old) for their new owners to continue to bottle feed and ‘bond’. This often results in a significant dietary change and stress leading to disease. We recommend that piglets are not weaned until over 4 weeks old and appropriate transition to a solid diet occurs. For orphan pigs, pig milk formula may be available. In the absence of this, goat milk formula is appropriate.

Fresh water should be available at all times, especially important in the Summer due to the risk of salt toxicity.
Housing
Pet pigs should always have access to the outdoors, since their rooting behavior can be strong. If kept alone and unsocialized they can develop behavioral issues. They should have adequate shade, fans and pools to keep cool in summer as temperatures above 85°F can be stressful. Pigs are exceptionally clean animals and will not voluntarily urinate and defecate in their ‘sleeping’ area. They can be litter box trained. Exercise is important for mental stimulation and prevent obesity. It must be stressed to future owners that keeping a pet pig, especially as a single indoor pet, requires as much work as a dog. Since pigs are classed as food animals, some home owners associations will not permit them.

Medications
Injections should be given intramuscularly in the neck in all pigs. Intravenous access is best gained by the ear veins. 18 – 22 gauge catheters are appropriate sizes. In old, very small or debilitated pigs where intravenous access is difficult, the intraosseous route can be used for fluid therapy, intravenous medications and euthanasia. A 14-gauge needle can be inserted in the tuba coxae, or an intraosseous catheter used. Due to their single stomachs, oral medications can be administered even in adult animals.

Common diseases
Erysipelas - Younger pigs commonly present with fever initially, the classic diamond skin lesions develop later. Heart and joint disease can occur with chronic infection.

Obesity - a major cause of poor health in pet pigs, predisposing to joint conditions, skin disease and blindness caused by the overhanging ocular fat pad. An ideal body condition is 3/5.

Osseous Chondritis Dessicans (OCD) - This disease is more commonly seen in show pigs that are fed high energy and protein diets. The bone outgrows the blood supply to the cartilage resulting in localized cartilaginous necrosis and bone cysts. The pig usually presents with lameness. Radiographs may provide a diagnosis. Treatment is not always effective, but the protein content of the diet should be decreased. Non-steroidal anti-inflammatories are indicated in the short term. Polysulfated glycosaminoglycans (Adequan) can be used weekly also.

Arthritis - is common in older pigs. and is the most common reason for euthanasia in our clinic. Pigs are naturally clean animals and when they cannot be clean they can become very distressed. Non steroidals, opioids and joint supplements can both be used long term to control pain.

Squamous cell carcinoma - This is a common skin disorder of older pigs with white skin. If white (or spotted/paint pigs) are kept outside, it is recommended to use suncream (non zinc oxide based) as a preventative measure. Usually presents as multiple raised ulcerated areas. Typically unless these areas become infected they do not appear to affect the pig. There are usually numerous lesions making surgical removal difficult.

Diarrhea - Infectious causes are more common in younger pigs. Nutritional diarrhea is not uncommon due to dietary indiscretion. Typically, in nutritional diarrhea, the pig is bright and alert with good appetite.

Dippity Pig - This disease is actually erythema multiforme. It is more common in younger pigs in warmer weather and stressful conditions. It causes acute pain and skin lesions. The condition will spontaneously regress, however antibiotics and steroids can be used. Sunburn can also present with similar signs, therefore a detailed history should be taken.

Skin diseases - many pot belly pigs have dry flaky skin. These can be removed with wet towels and moisturizing lotions applied. Fatty acid supplementation can be used, but care must be taken not to contribute to obesity. Pigs can have lice and sarcoptic mange. Injectable ivermectin is effective for both conditions. Greasy pig disease (Staph. Hyicus) may occur secondary to mange infestations.

Urolithiasis - both males and females can develop uroliths, however obstruction occurs in males. Diagnosis of urolithiasis can be performed by ultrasonography and/or radiographs. Treatment includes both medical treatment with urinary acidification and surgical methods.

Small ruminants
Small ruminants kept in urban environments may actually serve as more than just pets, including milk for human consumption, wool or meat. Therefore, it is important to establish the use of the animal before treatment is initiated and withdrawal times established.

Nutrition
A ruminant’s diet should consist primarily of forage (hay or grass), and supplemented only with a concentrate (grain) feed as necessary to maintain body condition. It is important to remember that body condition must be performed ‘hands on’ in small ruminants and not by sight, especially in wool breeds. All feeding changes should be made gradually, over 10-14 days. This will prevent diseases such as rumen acidosis. In general, most pet ruminants that are not productive (growing, lactating, pregnant or breeding) can maintain their body condition with forage alone. It is recommended that if a higher energy or protein diet is required, this is provided by a commercially available feed formulated for that species. This is especially important for sheep, due to higher levels of copper in other species’ feed. A mineral supplement (loose mineral is preferred over blocks) should be available at all times. Fresh water should be available at all times. Goats in particular, can be picky about drinking water. It is important to encourage them to drink by providing cool fresh water (or warmed water during extremely cold periods) to prevent urolithiasis in males.
Routine management
It is recommended that male pet small ruminants are castrated to prevent undesirable odors and behavior. Pets should be castrated as late as possible (4-6 months old) to allow the urethra maximal development which may help reduce incidence of urolithiasis. At 6 months old some breeds (Pygmies and Nigerian Dwarfs) may be sexually mature. At this age, castration should be performed surgically by a veterinarian under sedation/general anesthesia with systemic or local analgesia. Uterine tumors do occur in older females and a syndrome of inappropriate lactation is common in small breed goats. Therefore, pet female goats and sheep may be ovariohysterectomized under general anesthesia. Essential vaccines for all small ruminants include tetanus and Clostridium perfringens type C and D. This should be performed at 8 weeks old (if the dam was vaccinated 4-6 weeks prior to kidding) and a booster vaccine given 4 weeks later then annually thereafter. Any animal with an unknown vaccine history should be given ‘CD&T’ vaccine in 2 doses to start. Rabies vaccination can also be given. Other vaccines including respiratory, orf, foot rot, caseous lymphadenitis should only be given if there has been documented disease and under the recommendation of a veterinarian

Parasite control
Due to the significant presence of resistant parasites to all classes of anthelmintics, sustainable control of parasites is recommended. In brief, this program means that only those animals that need treating with an anthelmintic (deworming) are given the drug which is most likely to be effective. FAMACHA (ocular mucus membrane color), body condition, fecal consistency, fecal egg counts should be used to determine which animals need treating. Haemonchus contortus (barber pole worm) that causes anemia and Telodorsagia and Trichostrongylus that cause weight loss, bottle jaw and diarrhea are the most common nematodes of small ruminants and camelids. Key points to note are
- Goats and camelids require higher dosages of dewormers than sheep
- When diagnosing and managing parasites a quantifiable egg per gram count (e.g. McMasters) is used rather than a fecal float
- AMDUCA regulations should be followed when choosing an anthelmintic to use and appropriate withdrawal times (according to FARAD) are used
- Oral dewormer should be used. Injectable and pour on formulations are more likely to contribute to resistance.
- More information can be found on the website of the American Consortium of Small Ruminant Parasite Control (www.wormx.info). It is recommended that veterinarians and technicians become familiar with sustainable parasite control as management of parasites has, and is, changing, and clients need to be kept updated.

Foot trimming
Depending on the breed, the animal’s conformation and area where they are kept, sheep, goats and camelids need to have their feet trimmed routinely. Only excess hoof wall should be removed, aggressive foot trimming can result in granuloma formation and lameness.

Shearing
A significant problem in camelids kept in Texas (and the rest of the Southern USA) is heat stress. We recommend that camelids (alpacas and llamas) are sheared twice, once at the start of the warm weather (April) and again in late Summer (August). Veterinarians may be called upon to perform shearing. Wooled sheep need adequate shade and fans during the summer, and shearing before the start of the warm period. Hair sheep can more easily be managed in the climates of the Southern USA.

Common diseases
Some of the more common presentations of disease that are seen in pet small ruminants include inappetance/general malaise diarrhea, bloat, straining to urinate (the owner may recognize this as straining to defecate), and neurological disease. Common diseases include coccidiosis (in younger animals), gastrointestinal parasitism (Haemonchus presenting with anemia and weight loss, or Telodorsagia presenting with weight loss and diarrhea), grain overload (may present with bloat, diarrhea, inappetance), copper toxicity (presenting with malaise, pigmented urine) and polioencephalomalacia and urolithiasis. As a general rule, all sick male castrated animals should have urolithiasis considered as a differential until they pass a normal stream of urine. Copper toxicity is more common in sheep, as they have a lower tolerance level. Sheep that have access to pig, goat, cattle, horse or poultry feed are at risk of copper toxicity. Copper toxicity causes hemoglobinuria, with secondary renal failure and anemia. Due to the common occurrence of rumen acidosis (grain overload) it is advised to pass a tube and check rumen pH (normal is 7) (and protozoal activity under a microscope on low power if possible) in all sick ruminants. Polioencephalomalacia presents with stargazing and blindness (absent menace response but present pupillary light reflex).
One of the biggest, albeit innocent, errors in veterinary management is to appoint a person the head of a technical department and not give them clear goals, capable team members, or the support they need to accomplish the job at hand. Before supervisors appoint a head tech, before the technician accepts the job, or even if you’re already deep into your role, the following points should be considered.

The value of planning and regular communication
An organization on target to succeed is one that has a mission and goals. Mission statements have been discussed for such a long time, but implemented so poorly in most practices, that many veterinary professionals dismiss them as unnecessarily mumbo jumbo with no real relevance to the practice’s day-to-day work. What a shame! Mission statements, when given proper thought and exercised correctly in the practice, can be the beginning of a remarkable success story: a story of you and your team and the journey, effort and outcomes that you not only aspire towards, but achieve. They begin a dialogue on expectation and serve as a sounding board for every management decision in the building. Mission Statements level tyrants because they stand for the place of employment, not the employer. They ground us in what we want to work for, not what we have to do.

I can’t emphasize the value of mission statements enough and a bit of me dies every time I’m lecturing on the topic and the eyes of attendees glaze over. In the presentation in Kansas and San Diego, I’ll do my best do entertain, shout and rant on the topic. In the meantime, please go to www.dvm360.com and search for Bash Halow. In there, you’ll find a link to resources that I’ve built that will help you and your team explore the mission statement and your practice’s reason for being in general.

Mission statements precede another vital and key component of successful leadership: goals. Having outlined in the mission what you one day want to be, the question left hanging in the air is ‘how are you going to get there?’ and it’s a question that every team wishes that you and your employer would agree upon. Most teams falter because leaders have failed to organize the workplace. This prevents team members from working productively with others, and from succeeding as individuals. Your team wants clear goals to work towards so that they can realize their own ambition: to be great at what they do.

Head Techs without headaches are ones that have sat down with their employer and thought through a short list of what needs to be done in the practice in order to move the entire business towards the mission statement. It requires time for everyone to communicate thoughtfully and consistently. Communication is a word tossed about more frequently than the term mission statement, yet it too is never given the kind of consideration it genuinely deserves. In reality, most of us are poor communicators because we assume that if words come of our mouths in a grammatical manner that others will understand our meaning, but this isn’t true. In order for concepts to be understood, you often have to articulate them many times over, in many ways, and you should always be open to the idea that you still may not have made yourself clear.

You and your employer should regularly discuss the mission and how it is expressed in the day-to-day actions of your practice. You should talk about goals, plan together, build action steps, and discuss your progress. If you want to take the icepack off that head and achieve success as a head tech, discuss goals for the practice with the other leaders in the building and agree on how they are to be achieved.

Time heals everything, including the head tech headaches
If you are a head tech with a hurting head, you might blame the lack of time given to you to do the work of leadership. We’re very big on multitasking in veterinary medicine and expect all employees, not just head techs, to keep a few projects on the back burner as a way to justify payroll when the appointment book is looking light. However, great or even good leadership is not something that can be done on the fly. In order to be successful in your role, you have to take thoughtful time to think through how you will lead your team to working upon and achieving practice goals.

And with that comes a responsibility on your part to want to succeed as a leader. Many head techs are stuck with the position, not desiring of it. If you are a leader that lacks passion for the position, both you and your employer should put together a strategy to move you into another role. Because you have no fire inside of you for the position itself, you have no fire underneath you to get its work done.

Action steps
Eager head techs, having met with the other leaders of the practice and in agreement on what the practice’s goals are, should return to their technical teams with the goals themselves and an idea of how they can be achieved. You can bark out orders at this meeting and
make it clear how and when you’d like things done, but if you do, that headache you were worried about may set in again rather quickly. Instead, present the goals to the group and encourage a discussion on the topic as way to engage everyone or even as a way to discover why such goals may have failed in the past. Taking time to hear everyone’s concerns (and occasional digressions) can be tedious, but discussion is an essential part of the thought process and another important component of communication. It’s also demonstrative of the leadership’s respect for the input of the rank and file.

Remember that meetings don’t have to be one-hour long. With the right kind of agenda and stewardship, groups can be taken through a discussion on a topic and come to a resolution on the matter in 30 minutes or less. Short, fast-paced meetings don’t have to mean that a topic is shortchanged. Provided the leader of the group is focused, shorter meetings can energize the group and consequently stimulate thought. They wake up team members and let them know that this isn’t a time for pizza and repose, but active, energetic, invested problem solving.

Leadership training
As Robert Burns opined, ‘the best laid plans of mice and men often go awry’. Even if your practice owner, the other leaders, and you do your very best to get your hospital team focused upon and achieving goals, you will invariably encounter problems. In these moments, everyone will be tested to communicate (there’s that word again), remain patient, stay respectful, and work through problems constructively. In such meetings, you’ll have to practice negotiation and learn how to add input without offending or demoralizing. You’ll have to try to stay positive and professional, despite the real feelings of defeat, frustration, or fear that may be churning inside of you.

Good leaders don’t have to like conflict; in fact, it’s helpful if they despise it. They don’t have to be bossy or extroverted or male or tall or most senior. They have to be focused on the goal and they have to inspire others to pursue it with the same passion that drives them and the other leaders of the practice. Leadership is not a set of must-dos as much as it is a combination of natural talent and the willingness to regularly review the impact and efficacy of one’s actions.

In most cases, employees fail to live up to expectations for logical reasons. Rarely is their uninspired action or lack of action direct insubordination. Good leaders ask questions, listen to answers, and then formulate plans for everyone to move together successfully. Then, if that fails, they try again, and again and so forth until the practice steps forward or until it is clear that the best solution is to end the relationship entirely.

Consequences for not living up to the practice’s Mission Statement, and everything for which it stands (the practice’s goals), must be as ultimate as the Mission itself. If you have a team member who has repeatedly failed to live up to the practice’s expectations and demonstrates an unwillingness to try to change, it’s the leader’s job to terminate the employee. Any member of the team, who insists on putting their own interests before the interests of the practice, is a blight on the practice and the rest of your employees. Shirking your responsibility to address such an employee is probably one of the biggest mistakes you can make and one of the chief reasons why so many head techs’ heads hurt.

If you have an employer who refuses to back you up when taking a stand against willfully toxic and/or uncooperative employees, try engaging him or her in a discussion about the wisdom of their inaction. Even the most short-staffed practice owner is likely to admit that having one bad apple in the barrel risks spoiling a good deal of the others if not the entire stock. Once you agree on this matter, you will be able to formulate a plan for how to change the status quo.

Other resources
Remember that the best way to cure your head tech headache is to nurse yourself. Invariably failure to succeed at leading has something, if not everything, to do with the leader herself. Regularly challenge your leadership actions and ask for feedback from your employees and employer. Review the reams of information available to you on the topic of leadership on the dvm360 website or explore membership in such organizations as VHMA and AAHA.

Push yourself to understand why your head hurts and go to work immediately on one of the big reasons that’s probably responsible for the pain: you. Learning to be a better leader is not something you should eschew, rather understand that growing in your leadership capabilities means growing as an individual and becoming a valuable asset to your company, your family, your community, and this world.

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Cytology for Technicians
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Cytology key facts
1. One of the most valuable tools in Veterinary Dermatology.
2. Immediate information on presence of infectious organisms (cocci, rods, yeast, fungal spores) and different cell type (mast cells, neutrophils, macrophages)
3. As with everything practice is the key. When first starting to learn try to look at as many areas of the body as possible for normal and abnormal patients. One will not be able to determine abnormal if unable to determine what is normal.

Equipment
1. Tends to be very low overhead
2. Microscope
   a. Ideal to have a 4X, 10X, 40X, and 100X objective
   b. Best to know how to Kohler Illuminate
      i. Good tutorial can be found at www.youtube.com
      ii. Search under Kohler illumination
      iii. Keep the light condenser up when examining a cytology sample and down when examining skin scraping/trichogram-increases contrasts to better identify parasites and hyphae
   c. Usually keep the light source dimmed when not in use so as not to burn out the bulbs as fast
3. Microscope slides
   a. I generally prefer the frosted slides for numerous reasons.
      i. They can be written on
      ii. I use the frosted edge as my focal point so I know what part of the body a sample was taken from
      iii. So you heat fix/and dry the appropriate side
4. Staining station
   a. Modified Wright’s stain like Diff Quik is generally what we use
   b. There is a Romanovsky-type stain but this gives less nuclear detail
   c. All stains should be changed at least weekly and containers cleaned
   d. Cytology of skin and ears should be separated from fecal slides
5. Cotton-tipped applicators
   a. Important for ear cytology
   b. Also can break the cotton swab in half and collect samples from claw fold
6. Clear Acetate Tape
   a. Especially useful on hard to sample areas (interdigital webbing), aggressive animals or very dry samples.
   b. Generally you can just dip the tape directly in the Dif Quik third solution and then examine
7. Spatula
8. Blades for deep and superficial scrapings
9. Oil Immersion
   a. Generally our clinics use Either A or B (difference is the viscosities level). Type A is less messy and less expensive
10. Cover slips
    a. Needed for New Methylene Blue (fungal slides)
    b. I generally don’t use for anything else
    c. Some people prefer for skin scrapings
11. Lighter, coffee warmer, hair dryer (heat source), oil lamp
    a. Controversial
    b. Idea is that it fixes waxy samples to the slide so sample is not lost during staining process
    c. If using a lighter or match there will be black char that develops on the bottom and will need to be wiped off.

Sampling technique
1. Many different ways to obtain samples and it depends on your comfort level
2. Ears:
   a. One cotton tip applicator per ear inserted into the vertical ear canal.
b. Roll the sample then onto the slide
   i. I generally put the left ear on the left side of the slide or the frosted edge of the slide
   ii. I place the right side on the side of the slide (away from the frosted edge)

3. Claw folds:
   a. Either a cotton tip applicator (broken in half) or the spatula/edge of the slide scraped parallel to the claw fold and then rolled/pressed onto a slide

4. Body folds
   a. A cotton tipped pressed into the fold
   b. Then the sample rolled on the slide

5. Pustules, crusts, moist/greasy lesions
   a. Directly press the slides up against lesion in a single motion
   b. Crusts: Best to sample the area underneath the crusts
   c. For pustules and papules best to "pop" the lesion with a 25-gauge needle then press the slide on the ruptured lesion.

6. Dry/Scaly lesions/Difficult to reach areas (interdigital)
   a. Clear acetate tape is best

**Examining the sample**

1. Always examine at 4X first to find out where you should go down to higher power
2. Focus down on the areas with most amount of nuclear stranding and inflammation seen.
   a. Intracellular organisms= pathogenic= true infection.
   b. Organisms without the presence of inflammation is more indicative of either non-pathogenic bacteria, bacterial overgrowth or failure to select the infected area for sampling.
3. Be sure to always manipulate the fine focus to be able to differentiate between melanin granules and bacteria

**Sample interpretation**

**Infectious organisms**

**Yeast**

1. The most common yeast organism found on samples from dogs and cats is Malassezia pachydermatis,
   a. Classic “foot print” or "peanut" shape of a budding yeast organism.
   b. 3-5 µm in diameter
   c. Yeast organisms are usually found adherent to corneocytes.

**Bacteria**

1. Staphylococcus Pseudointermedius
   a. Infection is the most common bacteria isolated from infections of the ears and skin of dogs and cats
   b. Cocoid bacterium, 0.5-1.5 µm in diameter and is often found in pairs.
2. Pseudomonas aeruginosa
   a. Rod-shaped organisms, approximately 0.3-0.8 microns wide by 1.0-1.2 microns long
   b. Short chains.
   a. Harmless very large filamentous gram-negative saprophytes
   b. Inhabit the oral cavity of a variety of warm-blooded vertebrates 6-8 µm long and 2-3 µm wide.
   c. Presence is likely associated with licking

**Fungal spores**

1. Demonstrated with either exfoliative or aspiration cytology.
2. Dermatophyte spores often appear as round spheres, usually about twice the size of cocoid bacteria
3. Surrounded by a capsule that limits staining and gives "halo" appearance of a clear halo.

**Fungal hyphae**

1. Uniform 2-3 µm wide filamentous structures often with poor staining characteristics

**Parasites**

1. Cytology does not tend to pick up these organisms but can be picked up on scrapes
2. Best under 4X power
3. Demodex, sarcoptes and notoedres
Documenting findings

1. Ensure you are giving the doctor an accurate picture of the slide. Standardize this interpretation of the slide so there is no confusion on severity of the condition.

Cells

Degenerative neutrophils
1. With cocci = pyoderma

Intact Neutrophils:
1. Sterile process like PF

Eosinophils
1. Inflammation secondary to parasites
2. Hypersensitivity reactions such as (food allergies or atopy)
3. Pemphigus foliaceus
4. Foreign body reaction (such as free keratin within the dermis),
5. Ear cytology with topical reactions

Mononuclear macrophages
1. Chronic or deeper lesions
2. 2) Phagocytic cells and often contain effete neutrophils, red blood cells, bacteria, fungal elements
3. Depending on the severity of the wound or lesion contamination, macrophages will often be markedly vacuolated and degenerate.

Epithelial cells
1. Epithelial cells encountered normally on exfoliative cytology are corneocytes (non-nucleated keratinocytes)
2. Nucleated keratinocytes are normally found in samples from mucous membranes and associated with abnormal turnover of the layers of the skin

Acantholytic cells
1. Lower layer keratinocytes that have lost their adhesion to neighboring keratinocytes
2. Found in pemphigus foliaceus lesions,
3. Large, round cells with a central nucleus
4. Stain with a dark blue periphery
5. Found in rafts and amount well preserved neutrophils and/or eosinophils

Artifacts and other findings

Melanin granules
1. Can also be mistaken for rod-shaped bacteria
2. Found in and around keratinocytes
3. Refractory brown to black color and are very even in size.

Pollen
1. Multiple shapes and sizes and is often confused for fungal elements.
2. Pollen tends to be dramatically geometric and is usually not present in large numbers

References
Veterinarians and staff should work as a team to introduce cat owners to the concepts of normal feline behavior, reward training, and establishing healthy social relationships. This can be complemented by handouts and a list of reading and DVD recommendations. In addition, the clinic website should be populated with links to guide owners to the sites you recommend. Cat owners given educational material are at reduced risk for relinquishment.1

Behavior problems in cats
In a study of 1177 cats 55% of owners reported at least one problem they would want to improve with anxiety most common at 16.7% followed by scratching 16%, feeding problems 11%, aggression 11%, inappropriate urination and spraying 8%, and defecation in the home at 5.1%.2 In a recent study of 277 cats, 61% engaged in one of 6 behavior problems with aggression to owners at 36%, urine soiling 24%, intercat aggression 21%, aggression to visitors 14%, stool soiling 13% and aggression to outdoor cats,12%. Yet only 54% of owners reported these signs to their veterinarian and only about 25% of veterinarians ask.3,4 In a survey of over 1200 cats adopted from shelters, scratching, digging and chewing were reported in 25% of cases in the first week which increased to 28% after one month. Energy level increased (14% to 28%) as did biting and growling. Only shy, fearful and hiding reduced.5 Therefore behavior counseling of new cat owners is essential for problems to be improved and reduced.

Feline behavior and development
Cats might be categorized as a) sociable, confident, and easy going or b) timid, nervous and aggressive. While genetics, especially paternal, has a strong influence this is modified by environment especially prenatal (including the health, nutrition and environment of the mother) and the early postnatal (including maternal effect, handling and socialization).6,7 Kittens separated from their mother and hand raised by 2 weeks of age are more fearful and aggressive toward people and cats, more sensitive to novel stimuli, learn more slowly, and develop poor social skills. Kittens receiving early gentle handling by humans develop healthier social relationships, have accelerated development and are less fearful.

Preventive counselling
Educating new kitten owners on normal behavior, cat communication and body language, socialization, reward based training and enrichment can help to prevent undesirable behaviors. When introducing a new cat the use of pheromones (Feliway) might lessen the stress (improve appetite, less marking). The new cat should be given its own safe, enriched housing area, and introduced gradually to other pets and the rest of the home while insuring positive outcomes. Feliway multi-cat might be more appropriate for multi-cat homes. In a recent study a significantly higher percentage of owners that received no counseling sought advice (46% vs. 4%) or reported behavior issues 10 months later compared to those that had counseling visit of 25 minutes with a veterinary behaviorist at 2-4 months of age. Control cats had more vocalization and climbing problems (furniture, curtains), were more noise sensitive and more likely to solicit attention when the owner was resting. Cats in the treatment group were more likely to greet on homecoming and less likely to react negatively to body handling.8

Socialization and encouraging positive outcomes
Since the upper end of the sensitive period is 7 to 9 weeks, every effort should be made to socialize prior to this age. Treats and toys can be used to make social interactions, handling and stimulus exposure positive. Learning should be reward based to increase behaviors that are desirable. Clicker training can be particularly useful for immediate timing of rewards. Punishment should be avoided as it leads to fear and avoidance.

Environmental enrichment
All animals require an environment that is physically and mentally stimulating and that meets their behavioral needs. Animals kept in a restricted environment may not have adequate opportunity to engage in their full behavior repertoire. The indoor feline environment should therefore provide for food, water, elimination (litter), scratching, elevated perches, comfort and security and opportunities for enrichment including social and object play, exploration and reward training. When sufficient outlets are not available, the pet may engage in behaviors that are undesirable to the owners or develop stress induced health and behavior issues. Many common behavior concerns including scratching, over-exuberant play, chasing, climbing, attention seeking, nocturnal behavior, soiling and vocalization, can be prevented, managed, or resolved by providing appropriate outlets.

- Social time with owners and other cats plays an important role in enrichment and in maintaining healthy social relationships. Prey type toys attached to a wand or rope can be dangled and moved to stimulate hunting which might normally occur as many as 40 times or more a day.
- Reward train what you want the kitten to learn. When giving toys, treats or catnip reward and train what is desirable and associate words to cue the interactions (communicate).
- Feeding toys and object play: In nature cats hunt, capture, and kill multiple prey a day. As an alternative to hunting, cat owners can offer multiple small meals in toys that require rolling, batting, chasing, pawing or chewing to release food. Food can also be scattered or hidden to encourage exploration.
- Outlets for scratching, climbing and perching should also be provided.
- Outdoor access: While outdoor access may be undesirable or impractical for some homes, training the cat to be comfortable on a leash and harness or the use of cat proof enclosures can provide a practical and safe option for outdoor enrichment.

**Elimination**

Since cats generally prefer to eliminate in a substrate in which they can scratch and dig, an indoor box with commercial litter is usually effective. Litter type, number, size, type and location of boxes, and adequated cleaning are needed to establish regular litter box use.

**Management and safety**

Strategies may also be needed to prevent access to areas where problems might arise by cat proofing, blocking off areas or confinement training. The use if an unpleasant substrate such as an aluminum pan with water, sticky tape or carpet runner with nubs can be used to deter use of areas.

**Comfort and privacy**

Cats should be provided with opportunities to perch, rest, sleep and hide, and sufficient space to be able to allow them make choices when and where they want privacy. A normal feline time budget might allocate 44% to sleep, 22% rest and 15% grooming.

**Neutering**

Androgen influenced behaviors can be reduced or eliminated by neutering.

**Kitten kindergarten**

Kitten classes are valuable for socializing kittens, exposing them to a variety of novel stimuli, and teaching good manners. Offering these classes in a veterinary clinic help the kittens (and owners) develop positive associations with the veterinary clinic and staff. Topics include positive body handling, reward training, litter box management, enrichment, administering medication, handling, harness and crate training, the perils of punishment, and sessions to socialize and play.

**The feisty feline**

**Scratching**

Scratching serves a variety of purposes including marking, nail maintenance, and stretching after rest and may increase in response to stress. Owners should provide a scratching post at or near the cats preferred scratching location with a substrate and structure that is appealing, ideally one that is similar to what the cat is presently targeting. Placing catnip, or treats in the area, rewarding / clicker training and the use of feline interdigital pheromone (available in Europe) will encourage the use of the post. In a recent study of over 4000 cats, cats that were rewarded were more likely to use the preferred post and punishment did not affect frequency of inappropriate scratching. While carpet was the most frequently offered substrate, rope (sisal) was used most often (33%) followed by carpet (25%) and cardboard (18%). Cats over 10 preferred carpet (25%), rope (23%) and cardboard (20%). Most cats 9 or less preferred cat trees with multiple levels (76%) followed by vertical posts (69%), horizontal (50%) and hung on the wall (6%). Cats preferred posts that were narrower < 3 feet, and shorter <3 feet.

Inappropriate scratching was reported by 52% of owners, 65% at least once a day and 35% multiple times daily and was unrelated to number of cats or sexual status. Cats obtained from breeders had least inappropriate scratching (38%) and those from shelters most (54%). Inappropriate scratching was associated least with rope covered posts. Cat trees with two or more levels were associated with least inappropriate scratching (55%) with those hung on the door associated with most inappropriate scratching (74%). Inappropriate scratching was less with posts >3 feet (55%) compared to smaller posts (65%). Of cats that were declawed 8.5% were in the front and .9% front and rear, primarily to prevent damage (44%), followed by injury to people (18%) and injury to other pets (11%). Twenty nine percent of cat were declawed when obtained.

Cats that continue to scratch inappropriate targets in response to stress might benefit from applying Feliway to these areas. To prevent further scratching at a site, the scratching post might be placed directly in front of the area, access to the area can be blocked, the cat confined away from the site, or environmental deterrents used. Outdoor access may reduce indoor scratching but there is no evidence that it improves inappropriate scratching.

**Play / predation**

When aggressive play is directed at the owner, the primary focus should be to redirect the predatory play to appropriate outlets such as toys attached to ropes or wands or that can be tossed to stimulate chasing, pouncing and biting at times of day when the behaviors are most likely to arise. When hunting, cats engage in multiple short bouts of chase, may have a daily catch of 8-10 mice, with excursions lasting up to 30 minutes. A cat may spend 14% of its day hunting, 3% traveling and 2% feeding. Therefore it is not surprising that
some cats, will engage in multiple stalk, chase and bite sessions toward owners and other cats. However, perhaps surprisingly in one study cats that hunted and those that spent more time outdoors were reported to have more behavior problems.3

Play with toys can provide an alternative outlet for hunting behavior as well as an opportunity for social interaction and training. Toys with play like characteristics (size, movement, odor) will solicit play.11 However after two to three bouts of chase with a specific toy, the cat may lose interest but remain in a heightened state of arousal. Within 5 minutes play with a novel toy was dis-inhibited and more intense with 25 to 45 minutes needed till intensity is reduced.11 Therefore at least one or two additional toys should be offered for novelty, and the cat offered an alternative activity such as a food filled or catnip toy or a small meal to keep it focused, allow the cat to settle and perhaps simulate prey capture. In one study owners that played with their cat in more than 5 minute bouts a day had fewer problems.3 Owners should diarize when play attacks arise so that they might schedule pre-emptive play or confine the cat with alternative forms of enrichment (e.g. food filled toys, cardboard box to explore).

Since satiation reduces the motivation to hunt and play, feeding multiple small meals might also reduce play predation.12 Training the cat to engage in desirable behaviors on cue (e.g. sit, go to your mat) provides the owners with a communication tool to direct the cat into alternative acceptable behaviors if the owners can identify the body language of impending attack. If signs and situations for aggression are recognized the owner can throw treats to redirect and countercondition the cat. Leaving a bell attached or a leash and harness to inhibit and redirect the cat might also be helpful. While getting a second “compatable” cat could result in new issues, it can be an effective solution for some cases.

Social relationships
When adopting a new cat, it should be confined to its own enriched housing area within the home, so that it might be gradually introduced to other cats, people and increasingly more of the home while insuring positive outcomes. Over time, some cats develop strong bonds with other cats, while some require very gradual introduction and an ongoing need for space, privacy and restricted interactions.

Petting aggression
This aggression may be inhibited or intense. What is most confusing to some owners is that the sequence may begin with the cat seeking physical contact. However, after a variable period of time, the cat may become agitated and bite. This may be related to the cat’s level of arousal, previous unpleasant experiences with handling and restraint or even a sensitivity to touch. Owners must learn to recognize feline body language to identify the earliest signs of anxiety, as well as when and where problems might arise and the type and length of petting the cat will tolerate. While recognizing and respecting the cats limits, giving rewards during petting and ceasing before any signs of anxiety can gradually countercondition a longer response.

Summary
Understanding normal feline behavior and development is necessary to adequately meet the kittens needs. Many of the most common undesirable behaviors in cats can be effectively addressed by understanding and meeting these needs. Preventive counseling is effective in preventing many of the common feline undesirable behaviors. Therefore, veterinarians and staff should be proactive in providing behavior advice and resources for client education.

Web resources
Ohio State University: environmental enrichment
American Veterinary Society of Animal Behavior: www.catvets.com
International Society of Feline Medicine: www.icatcare.org
Catalyst Council: catalystcouncil.org
AAFP and ISFM feline environmental needs guidelines: http://bit.ly/14uWTCB

References / suggested reading
6. Lowe SE, Bradshaw JW. Ontogeny of individuality in the domestic cat in the home environment. Anim Behav 2001; 61; 231
Common Emergency Procedures You Must Know  
Justine Lee, DVM, DACVECC, DABT  
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Veterinary technicians are vital in performing and assisting with common emergency room procedures. Veterinary technicians should feel comfortable obtaining intravenous (IV) access (e.g., peripheral or central catheter placement, jugular cutdown), placing nasoesophageal (NE) or nasogastric (NG) tubes, placing nasal oxygen, performing nebulization and coupage on dyspneic patients, and tube feeding neonates. Likewise, veterinary technicians should feel comfortable assisting veterinarians with more advanced emergency room procedures such as thoracocentesis and decontamination of the poisoned patient (e.g., emesis induction, administration of activated charcoal, gastric lavage).

Venous access
The use of indwelling IV catheters is commonly used in veterinary medicine. These can be placed in either peripheral (e.g., cephalic, lateral saphenous) or central (e.g., jugular, medial saphenous) locations. Peripheral catheter placement can typically be performed with minimal physical restraint. For central catheter placement, light sedation is recommended. Regardless, aseptic technique should be used for indwelling catheters; with sterile technique, venous access can be maintained for days. Indwelling catheters can be used for:

1. Fluid therapy administration
2. Drug administration
3. Blood transfusion product administration
4. Measurement of central venous pressure (CVP) - central
5. Administration of drugs that are perivascularly irritating - central
6. Hyperosmotic fluid administration (e.g., total parenteral nutrition, > 5% dextrose solutions, etc.) - central
7. Venous access for blood draws and clinicopathologic testing - central

For a step-by-step approach on IV catheter placement, the reader is referred to:[1]  

Nasoesophageal and nasogastric tube placement
Placement of NE or NG tubes are commonly performed in the emergent or critically ill patient. These temporary feeding tubes can be beneficial as they can allow for the following:

- Emptying of gastric residual volume
- Administration of a liquid enteral diet (e.g., Clinicare)
- Trickle feeding post-operatively to stimulate enterocyte health and gastrointestinal motility
- Administration of liquid medications (e.g., antibiotics, antacids, anti-ulcer medication, etc.)
- Administration of oral water to aid in hydration of the patient (particularly in patients with heart disease who are at risk for volume overload).

In order to perform NE or NG tube placement, the following steps should be followed:

1. Organize all your supplies so they are readily available in an organized fashion (feeding tube, suture, 20-gauge needle, proparacaine, e-collar, empty syringe, sterile water).
2. Apply 1-2 drops of proparacaine into the designated nostril.
3. Pre-measure the designated feeding tube from the last rib of the patient to the nostril.
4. Mark the line with a permanent marker.
5. Use sterile lubrication to lightly lubricate the tip of the NE tube.
6. Have someone firmly restrain while you, with intentional, rapid, short, fast motions, insert the nasoesophageal (NE) tube into the nostril, directing it ventromedial.
7. Insert in as rapidly as possible to the pre-marked line.
8. Using a 20 gauge needle, make a pinch bite at the lateral nares, avoiding the eye.
9. Feed suture material in through the needle tip, and extend it fully through the hub. Rapidly remove the needle, and suture a Chinese finger trap around the NE tube. Repeat this at the lateral zygomatic arch (avoiding the eye) and tie another suture into this area to secure the NE tube.
10. Apply an Elizabethan-collar on the patient immediately.
11. Aspirate the NE tube – you should get negative pressure. If you do not, you may be in the trachea. Caution should be used, as negative pressure does not definitive indicate that you are in the esophagus (you could be lodged up against the end of a bronchus). REMINDER: There usually isn’t any gas in the esophagus (think chest radiographs)!
12. Using a 20 mls syringe filled with air, rapidly bolus in the air into the NE tube while simultaneously ausculting over the stomach (right sided). You should auscult for “gurgling” of the stomach simultaneously.
13. Verify by administering 5 mls of sterile water into the NE tube – any dyspnea, coughing, or tachypneic should prompt immediate radiographs or removal of the tube.
14. Always verify the placement of a tube by performing a lateral radiograph, which should including the neck. Verify that the tube is in the esophagus, ideally sitting just caudal to the cardiac silhouette. When in doubt, repeat or add contrast to verify placement.
15. Calculate resting energy requirements and feed accordingly [2].
16. Appropriately mark the NE or NG tube with white tape that states “NG/NE” so it is not confused with a nasal oxygen cannula.

**Nasal oxygen cannulas**
The use of nasal oxygen cannulas can be easily placed in the medium to large sized dog. The author does not recommend placement of nasal oxygen cannula in cats, as they are obligate nasal breathers. Note that small dogs and cats can be easily treated in an oxygen cage, while larger dogs cannot fit in many oxygen cages. As a result, the use of nasal oxygen cannulas can help provide oxygen supplementation. The benefit of intranasal oxygen therapy is that it is generally well tolerated, provides excellent patient accessibility (e.g., the patient isn’t “locked” in an oxygen cage), and can deliver a fraction of inspired oxygen (FiO2) of 40-60% (at 50-100 ml/kg of oxygen).

In order to place a nasal oxygen cannula, the following steps should be followed:
1. Organize all your supplies so they are readily available in an organized fashion (red rubber catheter, suture, 20-gauge needle, proparacaine, e-collar, oxygen tubing).
2. Apply 1-2 drops of proparacaine into the designated nostril.
3. Pre-measure the red rubber catheter to the media canthus of the eye.
4. Mark the line with a permanent marker.
5. Use sterile lubrication to lightly lubricate the tip of the red rubber catheter
6. Have someone firmly restrain while you, with intentional, rapid, short, fast motions, insert the red rubber catheter into the nostril, directing it ventromedial.
7. Insert in as rapidly as possible to the pre-marked line.
8. Using a 20-gauge needle, make a pinch bite at the lateral nares, avoiding the eye.
9. Feed suture material in through the needle tip, and extend it fully through the hub. Rapidly remove the needle, and suture a Chinese finger trap around the red rubber catheter.
10. Apply an Elizabethan-collars on the patient immediately.
11. A Christmas tree adaptor often needs to be used on the end of the red rubber catheter to connect to the oxygen tubing.
12. Appropriately mark the red rubber catheter with white tape that states “O2” so it is not confused with an NE or NG tube.

**Performing nebulization and coupage**
The use of nebulization and coupage is important in the critically ill patient. Nebulization provides a fine mist of liquid droplets as part of aerosol therapy and aids in humidifying pulmonary secretions, stimulating coughing, loosening secretions, and enhancing clearance by the mucociliary escalator (so pulmonary secretions can be expectorated out).[3] Nebulization is indicated for upper respiratory conditions such as pneumonia, upper respiratory infections, tracheobronchitis, etc.) and as part of tracheostomy care [4]. Coupage, rotation of the patient, and limited exercise (e.g., encouraging walking) can help enhance expectoration and mobilize airway secretions. The use of nebulization and coupage should ideally be performed every 4-6 hours in the pneumonia patient.

**Tube feeding neonates**
Neonates often present to the emergency room for “failure to thrive.” Once the neonate has been stabilized (e.g., hydrated, euglycemic, normothermic, etc.), oral-gastric feeding should be implemented. A 5 or 8 French pre-measured red rubber feeding tube can be used. To perform this, measure from the tip of the nose to the last rib; mark the tube with a small piece of white tape (as this will be the maximum distance to pass the tube). The tube should be passed down the left side of the mouth; note, a gag reflex may not be present until 10 days of age.[5] Ideally, the appropriate species milk replacer should be given. The use of oral water or dextrose can also be given via the feeding tube if needed. The fluid should be warmed to near body temperature prior to administration, and administered slowly over several minutes.[5] After delivery of fluid, the tube should be kinked prior to withdraw to prevent secondary aspiration pneumonia. This is a procedure that is easy to perform; pet owners can also be taught to perform this for unique situations (e.g., if nursing off the mother is contraindicated (e.g., eclampsia, rejection), if the neonate has a weak suckle, with a severe cleft palate, etc.). NOTE: The normal stomach volume of the neonate is estimated to be 50 ml/kg; the patient should be fed every 2-4 hours and overfeeding should be avoided [5].
Performing a thoracocentesis in dogs & cats
A thoracocentesis is often life-saving, and should be performed immediately in any dyspneic patient that is suspected of having pleural effusion. The thoracocentesis should be performed cranial to the rib, as the blood vessels and nerves lie caudal to the rib (“hiding” behind the rib). Thoracocentesis should be performed at the 7-9 intercostal space (ICS) to avoid the heart (3-5 ICS) or liver (caudal to the ninth ICS). The patient should be shaved, scrubbed, and prepared for sterile technique. The use of a 3-way stopcock, extension tubing, an appropriately sized needle or catheter, and syringe should be used to collect air or fluid. Appropriate sterile collection tubes should be available for sample collection for cytology and/or culture purposes.

Inducing emesis in the poisoned patient
Emetic agents work by causing local gastric irritation, stimulating the central nervous system (CNS) chemoreceptor trigger zone (CRTZ), or a combination of gastric irritation and CNS stimulation.[6,7] Emetic agents are not effective if an antiemetic such as ondansetron or maropitant has been previously administered. Currently, the only home recommendation for dog owners is hydrogen peroxide, while veterinary-prescribed emetic agents include apomorphine hydrochloride (dog) and dexmedetomidine (cat) or xylazine hydrochloride (cat).[6,7]

In the dog, hydrogen peroxide works by local irritation of the oropharynx and gastric lining, which results in a gag reflex. It is usually recommended for oral administration by the dog owner when transportation to a veterinary clinic is delayed. Only a 3% hydrogen peroxide solution should be used, as higher concentrations can potentially be corrosive to the GI mucosa. Adverse effects associated with use of hydrogen peroxide as an emetic agent include irritation to the GI tract, gastric dilatation and/or volvulus (dogs), and potential for aspiration pneumonia.[6,7] Hydrogen peroxide is not a reliable emetic in cats and its use generally is NOT recommended in this species. In addition, cats can develop profound clinical signs from the administration of hydrogen peroxide including profuse foaming from the mouth and severe hemorrhagic gastritis.

Apomorphine hydrochloride is a centrally acting emetic agent that is highly effective in dogs. Administration results in stimulation of the CRTZ, quickly followed by emesis. Adverse effects associated with apomorphine administration are prolonged emesis and ocular irritation when administered subconjunctivally.[6,7] Apomorphine should not be used in cats, as it is not considered to be effective. Apomorphine should not be used when there has been ingestion of medications that result in compounding of symptoms (e.g., respiratory or CNS depression) or with antidopaminergic drugs (e.g., metoclopramide) that prevent emesis from occurring.

Dexmedetomidine and xylazine hydrochloride, alpha adrenergic agonists, are centrally-acting emetic agents that are used as emetic agents in cats. The use of apomorphine and hydrogen peroxide are not recommended for cats, as they are ineffective or can result in severe adverse effects (e.g., hemorrhagic gastritis), respectively. Xylazine does not reliably produce an emetic response in dogs, and thus is not recommended in dogs as an emetic agent. Adverse effects associated with alpha-adrenergic drugs include bradycardia, sedation, tremors, and respiratory depression.[6,7] Thawley and Drobatz found that dexmedetomidine resulted in emesis approximately 80% of the time in cats, as compared to only about 44% of the time in cats with xylazine.[8] Alpha adrenergic agonists should not be used in cats that have ingested medications (e.g., other alpha-adrenergic agonist drugs) or products that may result in compounding of bradycardia, respiratory depression, sedation, or CNS depression symptoms.[6,7]

Methods that are not recommended for emesis induction include digital induction of emesis, syrup of ipecac, liquid soaps, dry mustard powders, and salt. Digital induction of emesis often results in physical injury to the pet owner (dog bite), or injury to the pet’s throat and soft palate. Syrup of ipecac has historically been recommended to induce emesis, but is no longer the standard of care. Its cardiotoxic potential and tendency to result in prolonged vomiting, lethargy, and diarrhea have caused it to fall out of favor in both human and veterinary medicine.[6,7] Soaps, mustard powders, and table salt are not reliable as induction agents and may be detrimental (e.g., resulting in further complications such as hypernatremia of the patient).

Performing gastric lavage for poisonings
Gastric lavage is a labor-intensive procedure, but is life-saving with certain toxicants. While emesis induction can be safely performed in the majority of poisoned patients (e.g., in asymptomatic patients, with recent ingestion within the past 1-2 hours, etc.), some toxicants warrant the use of gastric lavage. These are typically deadly ingestions such as:
- Organophosphates/carbamates
- Cholecalciferol (found in rodenticides, psoriasis creams, and multivitamins)
- Baclofen (a centrally acting muscle relaxant)
- Macrocyclic lactones (e.g., ivermectin, moxidectin)
- Deadly medications (e.g., calcium channel blockers, 5-FU, etc.)
- Any dose that is approaching the LD50 of the drug

The goal of gastric lavage is to remove gastric contents when emesis induction is unproductive or contraindicated. For example, an obtunded, comatose patient presenting with baclofen toxicosis is at high risk for aspiration pneumonia if emesis induction is performed. In this situation, protecting the airway with an inflated endotracheal tube (ETT) and gastric lavage is safer. Rarely, complications of gastric lavage may occur, and include risks of sedation, secondary aspiration pneumonia (once extubated),
mechanical injury (to the mouth, oropharynx, esophagus, stomach), or respiratory effects (e.g., hypoxemia secondary to aspiration, hypercapnea secondary to sedation, etc.).[5]

Contraindications that exist for gastric lavage include[5]:

- A corrosive agent, where esophagus or gastric perforation can occur with orogastric tube placement (e.g., drain cleaners, oven cleaners, ultra-bleach, batteries, etc.)
- Petroleum distillates or hydrocarbons (e.g., gasoline, kerosene, motor oil), which may be easily aspirated due to its low viscosity.
- Sharp objects ingested (e.g., sewing needles, knives, etc.)

Not knowing more about activated charcoal

After an appropriate history, triage, physical exam, and initial decontamination procedures have been performed in the poisoned pet, the next step is the administration of activated charcoal (AC), if appropriate. Activated charcoal should not be given to the poisoned patient when the toxicant does not reliably bind to AC (see below) or when it is contraindicated to administer AC (e.g., salt toxicity, poor gag reflex).[5] In addition, symptomatic patients who are at risk for aspiration pneumonia should not be administered AC orally. Finally, the administration of AC with a cathartic should be cautiously used in dehydrated patients due to the potential (albeit rare) risks for hypernatremia secondary to free water loss in the GI tract.[5]

When administering AC, it should ideally be given within ≤ 5 minutes of ingestion to be most effective. In veterinary medicine, this is almost impossible due to driving time (to the clinic), lapsed time since ingestion, time to triage, and the amount of time it takes to physically deliver AC (e.g., syringe feeding, orogastric tube). As a result, administration of AC is often delayed for up to an hour or more. As time since ingestion is often unknown (e.g., pet owner coming home from work to find their pet poisoned), decontamination (including emesis and administration of AC) is often a relatively benign course of action, provided the patient is not already symptomatic. As always, when administering any drug, it is important that benefits outweigh the risks, and that complications be prevented when possible. In veterinary medicine, administration of AC with a cathartic as long as 6 hours out may still be beneficial with certain types of toxicosis, particularly if the product has delayed release [e.g., extended release (XR) or sustained release (SR)] or undergoes enterohepatic recirculation.[5, 6] While human medicine has moved away from administration of AC with poisoned patients, the aggressive use of AC in veterinary medicine is still warranted, as this is often our last line of defense when it comes to adequately decontaminating our patients. Certain modalities of therapy—e.g., antidotes [such as fomepizole, pralidoxime chloride (2-PAM), digoxin-specific antibody fragments], plasmapheresis, hemodialysis, mechanical ventilation—along with financial limitations of pet owners, limit our ability to treat poisoned pets aggressively as compared to human medicine. As a result, the continued use of AC in veterinary medicine is still warranted as a first line of defense therapy. Current recommended dosing for single dose AC is 1–5 g of AC/kg with a cathartic (e.g., sorbitol) to promote transit time through the GI tract.

Conclusion

Veterinary technicians should feel comfortable performing or assisting in these common emergency room procedures, as they can be life-saving for the emergent or critically ill patient.

References

Today technicians can pursue further training and specialization in many avenues including dentistry, anesthesia, internal medicine and emergency and critical care. The possibilities are seemingly endless for technicians who wish to further their education and expand the depth of their career.

One such avenue for extra training and knowledge is in the field of pain management. This is an exciting new avenue for technicians (and veterinarians as well) to help strengthen not only the practices bond with the animal but the clients relationship as well. Pain management has become an important specialty area in human medicine and that has inevitably led to a greater awareness of pain in animal companions. Pet parents want the best for their furry family members and that includes top-of-the-line treatments for pain management.

It was once thought that animals did not experience pain in the same way that humans do. But research supports that if a procedure is thought to be painful to us, it will also be painful to our fuzzy friends as well, even though they may go to great lengths to hide it from us. Therefore proper pain management must be offered to all patients. In addition to pain medications (analgesics), many clinics are now offering complementary treatments like physical rehabilitation, acupuncture and laser therapy to treat pet pain.

In veterinary medicine we now know that pain is best managed through an interdisciplinary approach and that effective pain management can be best achieved through cooperation, sharing knowledge, and the collective wisdom of veterinary professionals from many disciplines. The International Veterinary Academy of Pain Management is the cornerstone of knowledge and advancement in the field of pet pain management. They currently execute a program for veterinary professionals to gain advancement in the field of pain management.

The IVAPM’s current program will lead to the title of Certified Veterinary Pain Practitioner (CVPP) for veterinarians and licensed technicians; Certified Animal Pain Practitioner (CAPP) for physical therapists and physical therapist assistants with certification in canine rehabilitation. This is an exciting opportunity for veterinary technicians in particular. It is IVAPM’s strongly held view that the CVPP and CAPP recognition is well within the scope of any veterinary professional to attain, and the IVAPM encourages all to consider its merits and begin the application process.

The certification program is intended to emphasize the value of the many disciplines capable of enhancing patient comfort and quality of life, and to facilitate an understanding of the modalities not necessarily in the technician’s current area of familiarity. These areas include:

- Analgesic drug therapies
- Analgesic adjunct therapies
- Physical rehabilitation methods
- Complimentary alternative therapies such as acupuncture and massage

Obtaining the CVPP/CAPP provides the stage upon which all professionals committed to promoting, enhancing, and advancing pain management in animals may interface. It is the foundation upon which the veterinary profession can build the most effective multidisciplinary pain management team. By using a multimodal approach the CVPP can use analgesic drug therapies along with physical therapy techniques such as massage, hydrotherapy and acupuncture to create a multidimensional pain management plan to help the patient achieve the desired outcome.

After obtaining a CVPP, the veterinary technician can work together with pet owners and veterinarians to provide the best pain management plans for patients. CVPP’s can best be used in the following 3 ways:

1. The CVPP can assess the patient’s current status and pain management regime and together with the owner create a pain management plan specific to that patient for the best overall outcome.
2. CVPP’s can also help in the acute pain management setting helping clinics to create protocols for post-operative pain scoring and proper analgesic techniques for acute surgical pain.
3. The CVPP also acts as an important “point person” that the owner can contact and relay information about the pet’s progress. The CVPP can then use this information to make changes to the analgesic plan as necessary to ensure the best outcome for that patient.

By obtaining your CVPP you can also help in the continuing education of your clients and staff. Many veterinary professionals who have obtained the CVPP designation go on to write magazine articles, teach online courses, and lead seminars on various pain management topics.

There are so many opportunities for the veterinary technicians when it comes to pain management. Technicians can further their education and become certified in acupuncture, massage therapy, reiki and various other pain management modalities.
By reaching the goals associated with a Certified Veterinary Pain Practitioner, veterinary technicians can increase patient safety and comfort, increase the overall morale at your practice, educate clients on the importance of proper pain management, and increase client compliance in this area.

If you are a veterinary technician with an interest in pain management please consider the CVPP/CAPP path. If you have any questions about this process, please visit the IVAPM website at www.IVAPM.org or 615-301-3040 by phone.
The role of veterinary technicians in developing an anesthetic and analgesic protocol for critical patients is a complex task. The veterinary technician must work together with the clinician and other team members to ensure that the critical patient is properly evaluated and cared for. Before administration of any anesthetic and analgesic agents, each patient must have a physical exam. This exam should include (but is not limited to) a chest auscultation to assess cardiovascular and respiratory function, an ECG, temperature, blood pressure, femoral pulse evaluation, and pulse oximetry readings. In some cases it is also important to obtain lab values such as a PCV/TS, blood glucose, electrolyte panel, creatinine level, and blood gases. Once the clinician develops a clearer picture of overall patient status, they can assign an ASA rating, and an anesthetic and analgesic protocol can be administered. Proper protocols will change with each patient and the type of surgery or treatment needed as well as perceived pain. It is important to note that if a patient is thought to be in pain, analgesics should NOT be withheld. This lecture will discuss four common critical patients that often need anesthesia and analgesia.

The urethral obstruction patient
Cats with urethral obstruction often present in pain and distress. They often require immediate and rapid urethral catheterization. Treatment should begin with assessing the patients’ electrolyte and hydration status and checking for any arrhythmias. Hyperkalemia is a common finding in urethral obstruction patients. This can lead to ECG changes such as a wide QRS complex and absent or flat P waves. Often the T wave is peaked or tented. (Fossum, 2007) Hyperkalemia and acidosis may require additional drug therapies such as calcium gluconate, insulin given with a concurrent dextrose drip, sodium bicarbonate (Perkowski, 2000) Although calcium gluconate does not alter potassium values, it does stabilize cell membranes, allowing time to reduce the potassium levels and minimize the cardio toxic effects of hyperkalemia. (Cummings, 2014)

An IV catheter should be placed in all critical patients, especially the urethral obstruction patient. Cats will often need analgesics as well as sedation for placement of urinary catheter. Analgesia can be achieved with buprenorphine or hydromorphone. Propofol may be used as an induction agent prior to general anesthesia. Also, if the patient is in the early stages of the disease ketamine can be used in conjunction with diazepam for induction of general anesthesia. If the patient arrives laterally recumbent or critically ill, they may not require chemical restraint. In these patients, urethral catheterization can often be achieved with an opioid analgesic combined with a sacrococcygeal block using bupivacaine. (Campoy, 2013)

Post urethral obstruction, the patient must be monitored closely for hydration status, electrolyte imbalance, and analgesic therapy. In cats without evidence of chronic or acute kidney disease, NSAIDs can be administered to provide analgesia and decrease urethral inflammation. In cases where an NSAID is contraindicated, the clinician may use a therapeutic laser to help reduce the inflammation present post catheterization.

Gastric dilatation volvulus (GDV)
GDV is characterized by stomach distention and a clockwise rotation. This condition is considered a surgical emergency. Patients presenting with GDV are often large breed canines with deep chests. Presenting problems include restlessness, abdominal pain, unproductive vomiting, dyspnea, and distention of the abdomen. Because most GDV patients present in some form of cardiogenic shock, it is important that all team members be on hand as multiple events need to be synchronized to ensure maximum patient comfort and survival. The dilated stomach obstructs blood flow through the caudal vena cava, while the increase in gastric pressure decreases blood flow through the portal vein. (Benett, 2010) Initial treatment after physical examination will include IV fluid therapy via either a jugular catheter or large bore catheters in each cephalic vein. Gastric decompression by trocarization is recommended to improve ventilation before general anesthesia is initiated. (Bennett, 2010) Pre-medication as well as analgesia may be achieved with an opioid such as fentanyl, oxymorphone or hydromorphone. In animals that present as very ill, opioid doses can be reduced. Anesthesia can be induced using an opioid and benzodiazepine such as midazolam. Alternatively, etomidate combined with a benzodiazepine can be used. These combinations have very minimal impact on the cardiovascular system as opposed to other induction medications like propofol. Intra operatively inhalant anesthesia can be kept to a minimum by using a constant rate infusion (CRI) of fentanyl/lidocaine/ketamine. This combination will not only provide multimodal analgesia but also lidocaine has the added benefit of being an anti-arrhythmic should ventricular arrhythmias develop. The FLK CRI can be continued in the post-operative period to maintain a steady state of analgesia. Post-operatively, the patient can be transitioned to IV buprenorphine at the clinician’s discretion.
Hemoabdomen/splenectomy

The hemoabdomen patient often presents with signs of hypovolemic shock (pale mucous membranes, rapid heart rate, weak or “thread” pulses, etc.). Often this can be secondary to neoplasia (or a ruptured splenic mass. Before proceeding with the splenectomy patient, attempts should be made to restore the patients’ tissue perfusion and oxygen delivery before general anesthesia. Hypovolemic patients often have simultaneous RBC and protein loss so colloids and other blood products are often needed preoperatively. (Cummings, 2014)

Hemoabdomen patients proceeding to surgery can be treated similar to the GDV patient. Pre-operatively an opioid will provide analgesia. Induction can be achieved with an opioid and benzodiazepine combination. The patient should also receive concurrent pre-administration of oxygen via facemask or nasal catheter. Etomidate can also be used for induction combined with a benzodiazepine. Agents such as thiopental or propofol are not recommended due to their common side effect of vasodilation.

Again as with GDV patients, a FLK CRI can be a useful adjunct to minimize inhalant anesthesia. The FLK CRI can be continued in the post-operative period to maintain a steady state of analgesia.

Dystocia

The patient presenting with dystocia and requiring caesarian section (CS) must be handled very carefully to ensure the safety of the dam as well as fetuses. In CS patients anesthetic requirements are often reduced because of increased progesterone levels. There is also a reduced functional residual capacity of the lungs due to the pressure of the intra-abdominal volume of the fetuses. Patients that are not overly anxious or stressed should have an IV catheter placed, abdominal shaving and pre-oxygenation before drugs are administered.

If selecting an opioid for pre-medication a mixed agonist/antagonist such as butorphanol may be preferred to minimize fetal respiratory and CNS depression. (Norkus, 2010) A longer acting pure-mu opioid can be administered upon fetus removal to provide analgesia to the mother. Induction can be achieved using a low dose benzodiazepine (<0.15mg/kg) followed by propofol or etomidate. (Norkus, 2010) Alfaxalone, although not yet available in the United States, looks to be a promising induction agent for sick and debilitated patients such as these. Mask induction is not recommended due to the side effects and exposure to the staff.

If staff are so trained, an opioid/local anesthetic epidural can be a very effective analgesic tool that can dramatically reduce the need for inhalant anesthetics.

Trauma

Anesthesia and pain management of the trauma patient can be most challenging to the veterinary staff. Many body systems can be affected and concurrent and multimodal therapies are often needed. For the trauma patient, anesthesia should not be initiated until vital organ function has been stabilized. The trauma patient must have a patent airway. The clinician should ensure that circulating blood volume is maintained in order to provide tissue perfusion and oxygen delivery to vital organs. (Wadell, 2010)

The goal with pre-medicating trauma patients is to provide analgesia as well as reduce the overall amount of induction agent needed. Agents that are reversible (opioids& benzodiazepines) are preferred to agents that are not reversible (acepromazine, ketamine). An opioid analgesic such as fentanyl is an attractive option in the trauma patient as it is rapidly cleared from the body very quickly, which can help facilitate a neurologic examination. During induction patients should be pre-oxygenated. Induction can be achieved using an opioid/benzodiazepine combination. In some cases this may not be enough to intubate and a small amount of propofol is necessary to facilitate intubation. In cases where increased intra cranial or intra ocular pressure is not a concern a ketamine/diazepam induction may be an attractive choice as it allows rapid intubation and will provide some analgesia.

Post-operatively trauma patients must have vigilant nursing care constantly assessing their cardiovascular, respiratory, and pain level. Multimodal CRIs provide constant analgesia without the “peaks and valleys” effect seen with some intermittent dosing of analgesics.

Implementing a pain scoring system can help your clinic effectively titrate analgesics to fit your patients’ needs. The University of Colorado offers a species specific color chart available for download. These handouts can be placed in recovery and treatment areas to help train technicians and staff to recognize various pain behaviors.


Working together the veterinary team can implement an anesthesia and pain management protocol to help ensure the comfort and safety of any patient walking through your doors.

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928
Pain Management and Profitability: Stop the Hurt
Tasha McNerney, BS, CVT, CVPP
Veterinary Anesthesia Nerds
Glenside, PA

There are many reasons for providing appropriate analgesia to your patients, and thankfully for the overall economic health of a clinic pain management can also boost your practices bottom line.

Providing appropriate or even advanced analgesic techniques not only provides an economic benefit to your practice, but it also improves overall staff morale and job satisfaction. Technicians and nurses are able to identify painful patients and if they are allowed to play an active role in the alleviation of a patients’ pain, they have an overall greater sense of purpose at the clinic and increase in job satisfaction. Technicians who are proud of the level of analgesia provided for acute and chronic painful patients often stay at their place of employment longer.

Pet owners are also more educated and expect that their pets pain will be taken seriously and treated appropriately. Whether providing an analgesic regime to treat acute post-surgical pain, or developing a chronic pain plan for a senior osteoarthritis patient, clients have come to expect effective analgesic options. And, many clients are willing to pay appropriately so their beloved pet is not in pain.

When looking at the cost of analgesics, any of the drugs used are relatively inexpensive. The opioid analgesic Morphine for example is one of the cheapest yet most effective analgesic options available. It can be used as a pre-medication prior to surgery, or for added revenue to the practice and increased pain control during surgery it can be titrated to the patient via constant rate infusion.

Offering specialized analgesic options such as constant rate infusions or anesthetic local blocks can provide additional revenue as the clinic should charge appropriately for the supplies needed and for the expertise and additional training of veterinarians or technicians. Technicians can be trained to administer highly effective local blocks including epidurals. An effective epidural will not only improve patient comfort, but also cut down on the use of inhalant anesthetic during the surgical procedure as well as additional rescue analgesics often needed during surgery. If appropriate local and nerve blocks are used during each surgical procedure and charged a small appropriate fee, the practice will see a considerable difference when that small fee is multiplied by the potentially hundreds of surgeries that are done each year.

Some of the simplest ways to provide increased patient comfort and increased revenue without a big investment are as follows
1. Provide regional or nerve blocks to every surgical patient
2. Start using constant rate infusions for pain relief
3. Have a member of your team be the captain of the Pain Police and send them for additional training in analgesic options.
4. Become a member of the IVAPM. www.IVAPM.org

By implementing some simple changes and setting an appropriate in clinic point person, your practice will be known as the go to for the best level of pain management.

Resources
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Often times pet parents are faced with the decision whether or not to put their pets under general anesthesia for a surgical or dental procedure. They are also often worried about whether or not their beloved pet will be in pain afterwards and how they will be able to best manage this pain. Veterinary team members are often the ones responsible for explaining things to the client to alleviate some of the fear associated with post-operative recovery and pain management. Veterinary team members should also be prepared to discuss chronic pain conditions such as osteoarthritis.

**Reception**
When a client calls the clinic, the receptionist is the front line for obtaining information from the client. Receptionists can ask questions to help get a clearer picture of the painful condition either chronic or acute. Some examples include:

1. How long has your pet been experiencing pain?
2. Does the pain appear to be localized to one area?
3. Is your pet licking or biting at a certain area of the body?
4. Has your pet stopped eating/grooming/jumping/running?
5. Does your pet take a long time to stand after sitting or sleeping?

**Technicians/nurses**
Taking the time to get a complete history will be the key to ensuring proper pain management. Be prepared to talk with clients about acute pain after surgery. Orthopedic procedures and dentistry that have required multiple extractions however, may require a longer recovery time. Also after surgery patients may be unsteady on their feet and unable to walk up or down stairs. If their pet usually sleeps upstairs in bed, it’s important to set up a comfortable area on the first floor for their pet to rest in when they come home, or be prepared to carry their pet up and down the stairs! Also explain to the client that some post-operative pain medications such as opioids may make their pet nauseous and whiny; if this happens they should call the office.

Almost every surgical and dental procedure will involve some level of pain. Talk with the client about the expected level of pain and how this pain will be treated. Also talk with the client about the need (if any) for post-operative physical therapy. Orthopedic procedures greatly benefit from post-operative physical therapy such as hydrotherapy, massage therapy, and low level laser therapy. Also, many pain medications have to be administered orally. If it is difficult for clients to give their dog or cat a pill, there are now other drug delivery options to ensure pets stay comfortable in the recovery period.

**Doctors**
The doctors play a very important role in piecing the information together to create the best pain management action plan. By combining patient history with a thorough physical exam a concise and effective pain management plan can be created. The most important aspect of managing pain is to work with a multi-modal treatment protocol. The principle of multimodal therapy is to use analgesic drugs and physical therapy modalities that target several different steps of the pain pathway, allowing more effective pain control with fewer side effects.

**NSAIDs** remain the mainstay of therapy for chronically painful patients. Their principal mode of action is to block prostaglandin production by binding and inhibiting cyclooxygenase (COX). The result of this effect is mainly a reduction in inflammation.

**Opioids** are useful in a variety of painful conditions (though they may have limited effectiveness in some forms of neuropathic pain). Opioids may be particularly useful for chronic pain management, as they are available in oral and transdermal versions.

**NMDA receptor antagonists** are often used as adjunctive drugs (i.e. in combination with other analgesics) to improve the control of pain. Intense and/or chronic painful stimuli result in changes in the central nervous system’s response to input, leading to an increase of pain intensity. NMDA receptor antagonist drugs help to control and treat this “amplification”. Amantadine is the most commonly used oral NMDA receptor antagonist. It was originally developed as an antiviral compound, and has also been used to treat Parkinson’s disease in humans.

**Gabapentin** has been used for many forms of chronic pain, though its best application may be for neuropathic pain. Gabapentin is an anti-convulsant medication with significant adjunctive anti-hyperalgesic action. Gabapentin is commonly used in conjunction with opioids for analgesic treatment options in post-amputation patients.
Tricyclic antidepressants (TCA’s) have been used in humans and animals as adjuncts to other analgesics (especially opioids) for chronic pain. They act to inhibit serotonin and norepinephrine reuptake, though they may have other analgesic effects as well (including possible actions at opioid receptors and on nerve transmission).

Non-pharmacologic therapies for chronic pain management include acupuncture, electroacupuncture and, laser therapy and pulsed magnetic field therapy. Hydrotherapy is also a useful treatment option for patients that have IVDD or osteoarthritis. Often the best course of treatment is to combine physical therapy with pharmacological therapies.

Kennel/support staff
Kennel staff also play an important role in recognizing patients in pain. Because patients who are boarding for long periods of time, kennel staff may notice limping, sensitivity when an area is touched, patients that are uncomfortable and unable to rest, or a change in body language. If the kennel staff notices signs of pain in boarding patients, they should alert the nurses or veterinarians so that the owner can be notified and proper pain management plans can be enacted.

When all staff members work together, we can create a pleasing pain free environment for our patients. Everyone wins!

Another way you can ensure your patients are receiving the most comprehensive pain management plan is to become an IVAPM (International Veterinary Pain Management) member.

The IVAPM is an organization that seeks to educate and promote pain management for animals worldwide. It also provides continuing education in the area of pain recognition and treatment. IVAPM members can work toward certification in the management of animal pain. To find a CVPP or IVAPM member in your area, visit the IVAPM website at www.IVAPM.org

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The Art of Communication:
You Say More than You Think
Dani McVety, DVM
Lap of Love
Lutz, FL

The competence to mitigate pain and suffering by treating diseases and/or symptoms in animals without the ability to communicate both the intentional and potential outcomes of such treatments such that trust and rapport is gained with the client is akin to expecting to know how to ride a bike by simply reading an article about the physics of balance and rotational force. Without the belief and trust that the client and the doctor have the same desired outcome, even if that desired outcome is simply the comfort of the pet, trust and rapport will not be established and the client may not accept the treatment plan that you, as a doctor, went to medical school to learn.

As veterinarians we have two parties to serve in almost all areas of medical interaction; the owner/client and the patient. (Shelter medicine is the only exception to this rule, as treatment of animals in a shelter setting rarely include an owner.) In human medicine, the client and the patient are generally the same entity. Even in pediatric medicine, the parent is the guardian of the child, not the owner of that child. The parent generally has the levity to make decisions, but if that decision is not in the best interests of the child (as reasonably determined in a court of law), then the parent will in fact lose the ability to make decisions for that child. In fact, it took a groundbreaking case in 1984 (In re Guardianship of Barry, 445 So.2d 365 (Fla. 2d DCA 1984)) to determine that a parent can serve as proxy for their dying infant child’s wishes, allowing the removal of life support in this case. In veterinary medicine, however, our clients served as proxy for their pet’s wishes in almost every interaction they have with a veterinarian; from the decision to amputate a limb, chose surgical versus medical treatment, and even the removal of “life support” is a common path that the veterinarian must walk the client through on behalf of the pet. Legally, the clients are in fact owners of the patient and our communication and established rapport with that owner is imperative if we are to gain the trust such that our medical knowledge will be put to use for the betterment of the pet and/or the treatment of a disease. Learning how to gain that rapport is where the rubber meets the road!

In this interactive talk, we will use real-world examples and demonstrations to illustrate various aspects of nonverbal communication. We will discuss the 4 major types of clients, how to adjust your body language to gain and maintain rapport and trust with them, and how to compensate for problems that may arise. Attendees will be able to immediately identify these clients and implement different ways of nonverbally communicating by both reading their clients’ body language more accurately and changing their own to reach the desired outcome.

Important topics we will discuss include:

1. Identify the 4 major types of clients.
2. How to start the conversation: tone of voice to use, where to stand or sit.
3. How to address client concerns and outbursts.
4. Specific words to use (and not use) for different types of clients.
5. How to react and what to say when things do not go perfectly.
A positive outlook can go a long way in your everyday life, as well as within your practice. Unfortunately, many of us have been inundated with negativity from some of our coworkers. We have all worked with that team member who starts the day complaining they are having a bad day and that’s why they may be exuding pessimism all day. This is a very toxic approach to the workplace as it can spread throughout the team, creating an epidemic of unhappiness, and make everyone’s lives much more stressful, as well as their days longer.

**What IS a toxic work environment?**
The enemy of positivity in the workplace is an environment of toxicity. Toxic work environments often have chronically high stress levels, whether that be due to the work being consistently intense, or a fear-based culture. Employees may also have a poor work/life balance, where work is eating into their personal lives and enjoyments. There may be a general lack of enthusiasm and loyalty to the practice. The expectations by the supervisors may be unrealistic, causing team members to feel as though they are constantly failing. Dysfunctional leaders can also create toxicity by bullying the team, poorly communicating, and/or being unwilling to help and communicate.

**The repercussions**
Being part of a workplace consumed by negativity has a detrimental effect on the employees, and ultimately the success of the practice as well. Employees often develop stress-related physical and mental illnesses such as gastrointestinal upset, anxiety, depression, cardiovascular disease, migraines, and musculoskeletal problems. In a 20 year study, published in 2011, researchers at Tel Aviv University found a startling connection between the relationship between the workplace and the individual’s risk of death. Fifty-three individuals had died by the conclusion of the study in 2008. These individuals were significantly more likely to describe their work environment as hostile compared to those who survived.

**Adjusting your own perspective**
Fortunately, there is a lot that is in our own power to change and promote positivity. You ultimately choose how to react to certain circumstances. Although it’s not our natural response, taking on an excessive workload or dealing with a difficult team member/client is much easier when approached with a positive attitude. Remember it’s never a good idea to allow other’s baggage to live rent-free in your head.

We push through our lives with all goals meeting a larger goal of achieving happiness. Many team members see work as a necessary evil (and hurdle) to getting closer to that goal. It’s a much healthier outlook to view work as another opportunity to strive for inner happiness as you spend 40 plus hours a week there. Gratitude is also a great way to reframe your outlook amidst negativity at work. Try to remind yourself of what you are thankful for on a daily basis, especially during the most stressful moments.

Posture can also play a role in your own perspective of the day. Keeping your posture upright can help improve confidence levels and overall physical health. If you feel well-rested and energetic, you are better able to cope with difficult days.

Self-care is very important in combating negativity around you. Giving yourself time around your workday for something that you enjoy can greatly improve your mental state. That optimistic perspective may also rub off on your less than happy teammates.

Many of us in veterinary medicine are in a field that utilizes our strengths and ignites our inner passion. Keeping the big picture in mind and continuing to work towards a greater good can help conquer the more difficult days.

**Creating an overall positive atmosphere**
Outside of controlling our own responses to negativity, we can also help battle toxicity and grow a more positive practice.

Taking the approach of being energetic, charismatic, and people-oriented can help diffuse the group’s stress. If an employee, or even a client, seems stressed at the beginning of the day, try to go out of your way to help them out. Pick up extra tasks, and ask them if they need to talk. If a team member is in a very bad mood, it may be best to take the day off. Team members should not be taking “mental health” days regularly, but a stressed or burned-out employee may need the time once in a blue moon. You’ll find that the day flows much better and the team is more productive (even being short-staffed) without the negative aura. If the behavior continues regularly, then coaching by a manager needs to come into play.

Infusing positivity by allowing team members to have a place to look at a family photo, to decorate for the holidays, or even to add plants, is wildly beneficial. Encourage the team to take breaks when they are overly stressed or emotionally overwhelmed, and go for a short walk or listen to an uplifting song.
Reward systems aren’t just for your pups in the clinic. Team members love to get recognition as well, and it greatly improves morale. Whether it’s a friendly contest to book the most dentals or sell a certain product, or simply a submission box for kudos from fellow team members, a reward system is a must. The rewards can be small too, a $5 or $10 gift card, or ordering pizza for the staff. The benefits to the practice far outweigh the minimal cost.

Practices should also be assessed for dysfunctional relationships. Negativity often grows from clique culture, along with insincerity, favoritism, and grudge-holding. Make it clear to new hires that this behavior is unacceptable and will not be tolerated. With current employees, agree to set a new pattern and eliminate these behaviors. If still present over time, coach employees, and even make them go home for the day if they continue to exhibit this unprofessional conduct.

There’s no arguing that converting toxicity into positivity is a difficult, gradual process in damaged practices. It is not impossible though. With a strong emphasis on keeping team members satisfied and happy, the practice will see greater output and success as well.
Taking advantage of marketing opportunities in your veterinary practice will not only help your practice grow but also can give you a chance for personal growth. Marketing is a great creative outlet and a nice way to switch things up in the sometimes cut and dry life of a technician working on the floor. Thanks to our ever growing technologically advanced world, there are many new marketing outlets available. These modern strategies allow for a quick way to expand your outreach into the community.

Inbound versus outbound

It’s helpful to understand the difference between inbound and outbound marketing strategies. Inbound is comprised of your modern tactics, including (but not limited to) reaching out via social media, blogging, viral videos, and search engine optimized (SEO) website text. Outbound marketing is more of the “old school” approach, also sometimes referred to as “interruption marketing”. The idea in outbound marketing is to reach out to the largest group of people possible, but it lacks direction and can be considered aggressive. Outbound marketing techniques include advertising (billboards, newspapers), cold calling, and direct mail. Since the advertising business is trying to push out their message to the general public in as many ways as they can, in the hope of reaching some interested individuals, this can be a very expensive approach.

Inbound marketing has a more sophisticated approach, connecting with potential clients through content the viewers would seek by themselves. Originally, this approach may have only accessed younger individuals, but in today’s world, even our grandmothers are on social media. Another advantage of this form of marketing is that a company can interact with their clientele and create a more casual and engaging, as well as less aggressive, exchange.

Social media

When your practice decides to explore marketing options through social media, it’s important to understand your potential audience through the various outlets. For example, Facebook users are mostly under 30 years old, with individuals educated minimally at a college level. While Facebook is very popular with women, it has a decent male user database as well. Twitter is more balanced when it comes to gender, but mostly consists of users in their late teens to late twenties. Twitter’s users are mostly college graduates, similar to Facebook. Instagram is comprised of more female than male users, with a similarly aged audience as Twitter. While Instagram users tend to have some college experience, they are not mostly college graduates like the previously discussed outlets. Lastly, Pinterest is mostly made up of women, from a wide age range, and only about a third are college graduates.

In the world of veterinary medicine, Facebook is a great option for diving into the world of social media. To be successful on Facebook, it does take a certain level of effort and finesse. Your practice must post relevant material for pet parents in order to regularly appear in their feeds. If you are not precise in what you choose to post, it is possible you are wasting precious time trying to reach out to an audience who isn’t listening.

The stories that tend to draw people in include patient stories, asking engaging questions, and celebrations. Patient stories are usually very popular- be sure to get the owner’s permission, and post the case with a picture. Questions for your veterinary clients are also a successful attention-getter: ask questions like what their pet is doing for the upcoming holiday, or ask your audience to caption a funny animal photo. Celebrations draw a lot of “likes” as well- whether it be a team member’s professional accomplishment, a pet-related holiday, or a patient’s last chemo treatment.

Instagram is the next best social media option for veterinary practices. It focuses on sharing photos with followers, which is what pet parents love to see on your feeds. It’s a great opportunity to share success stories and funny pet photos.

Blogging

Another great way to reach out to potential and current veterinary clients is through blogging. It’s easy enough to keep up, and also cost-effective. This method helps establish your practice as an authority in the arena of veterinary medicine, and builds trust. Blogging can serve as an easy method of creating viral material that reaches a large audience. If pet parents feel the information posted is worthwhile, they often share this on social media which automatically widens your viewer base. This modern marketing initiative helps initiate conversations with pet owners by allowing for commentary on your posts. If the material is educational and considered valuable by your audience, you will also instill trust and credibility within the pet parent community.

Search engine optimization (SEO)

Your practice can put forth as much online marketing efforts as you like, but with a limited audience, your efforts will go unheard. Search engine optimization (SEO) helps lengthen your message’s tentacles across the internet. SEO effects visibility through free
methods (even better!). The idea here is that the earlier and more frequent a site appears in a pet owner’s search, the more viewers you will obtain.

Some of the methods for SEO include editing content, HTML and coding to increase the site’s significance to searched keywords. Content can be edited to include keywords that are frequently searched for, which can increase traffic to your site. Sites also can be promoted by increasing backlinks and inbound links (a hyperlink back to your site from another site). Cross linking between pages of a website can provide an increased number of links and help widen your audience. Content should be frequently updated as well so search engines will regularly direct searchers back.

**Videos**

Incorporating videos into a practice’s marketing campaign helps add another fun and engaging element for pet parents, and can ultimately increase visibility online. You can take the approach of sharing fun pet-related videos that you find, as well as making your own, which can be educational or simply endearing or humorous. The key is to share and create videos that evoke emotion and portray an image or viewpoint that is in line with that of your practice. Videos are often more likely to be shared, so they are much more likely to become viral. It is important to already have your website and social media platform up to date and well established before you begin to push videos. You wouldn’t want potential clients to stumble upon your Facebook account or website and find outdated or minimally engaging material.

**Review sites**

Another element of a practice’s online presence is review sites. In today’s world, consumers can easily access reviews and ratings from others on a multitude of review forums. While we all would like to find nothing but positive reviews under our name, when negative commentary is added, it is imperative to take action quickly. It’s very important to start out by responding to any negative posts, and do so with sincerity. This allows the public to see that you do care how you are perceived by the community, and want to do right by your clients and patients. Reach out to any upset clients directly to discuss the situation further. Prior to that conversation, be sure to research the situation so that you are knowledgeable during your talk and know if your practice truly was in the wrong. Afterwards, if the hospital was at fault, be sure to hold a coaching session with the employees working with the client to ensure that future clients receive a better experience.

Also remember to encourage those clients who rave on about your hospital at discharge to submit a review on the review site of your choice. Many review sites even have signs, along with a unique QR code, which you can post in your hospital for clients to easily find you (and hopefully provide you with a public kudos!). Links to your listing on a review site can be included on your practice’s website as well as within the email signature.

**Developing a strategy**

The key to jumping into the waters of the modern marketing age is to start with a budget. It will cost money to develop a useful, attractive website, and even the free options offer enhancements at a minimal cost that are worth exploration. The rule of thumb is usually 3-5% of your net sales will go to marketing efforts.

The following steps are developing a plan and mapping it out on a calendar for the year. Before publicizing your practice, you should be comfortable with your message. Your message should be direct, consistent and relatively straight-forward.

It is also worthwhile to consult with a marketing professional or company that has experience with veterinary practices. With the right guidance and approach, your practice will outshine the competitors with ease.
One of the hardest struggles any working adult will face is the balancing act between work and life. Most in the veterinary field are very passionate about their work, and along with commonly working long hours and emotionally tumultuous shifts, the struggle is even more intense.

Step Away! Technology as one of the culprits
In today’s world, we are never completely turned “off”. We are generally just an arm’s length away from our phones, making us available via text, call or email all hours of the day. Especially those in managerial and ownership roles may feel obligated to check their work emails regularly at home and respond while they should be off-duty. In a 2011 study performed by the University of Toronto, published in the Journal of Health and Social Behavior, researchers found that women who were contacted by their supervisors, coworkers or clients frequently also reported higher levels of psychological distress. Men were less effected, which they found was due to women harboring more guilt when responding to work matters at home.

Moving on up and other big life changes
Another component of the stress is that our Generation Y workforce is moving into management positions much quicker than their older counterparts. This generation is very ambitious, and utilizes technology heavily, which allows them flexibility outside of work, but also allows for them to be constantly available. Moreover, when you have younger individuals in management, they feel the work/life balance burden even more as they are getting married, starting families, and still trying to grow themselves as people.

This brings us to the emotional experience of returning to work after having a baby. More women nowadays want to continue working for financial and personal reasons. Your attempt at a balanced life becomes a lot more difficult when you have an unpredictable, demanding little being in the midst. New mothers need not fret, there are many useful techniques for dealing with this challenge.

Letting go of your perceived guilt for taking a break for yourself is the first step. Allow other family members, friends or your spouse, to help out with the baby while you get time to yourself. Having time to rejuvenate- whether that be spent exercising, reading a book or going out with some friends, helps make you a more well-adjusted person all around. It’s easier to give your all to your child, family, work and other components of your life when you are taking care of yourself.

Reaching out to other new parents can also help make you feel less alone in this new endeavor, and can even help provide some useful life tips. During the difficult spots of parenthood, being able to talk to other parents who probably have been there as well, can be reassuring.

If your old schedule is simply not working with your new family, it is always worth discussing with your supervisor. Try to also give some time after you initially return from maternity/paternity leave to settle back in, and make these big decisions when you are less emotional. Thankfully, within veterinary medicine there are a multitude of career options offering different schedules. If working in a clinic is no longer a good fit, it may be worth taking a leap into management, education, or exploring opportunities with veterinary insurance and product companies, or research.

Delegation is a healthy habit almost everywhere- at work, and even at home. Although many of us can realize when it is necessary to delegate tasks at work, we hesitate in the home environment. Getting help from a spouse, roommate or even your child (if they are old enough) can help manage the chaos and your stress level. A lot of times one parent does not see how frazzled and exhausted the other one has been until it is discussed. One of the best pieces of advice for a reduced stress life- don’t be afraid to ask for help.

The consequences of an imbalanced life
Not taking time for yourself and becoming overworked can wreak mental and even physical havoc on your body. When you work long hours and never take time for yourself and your family, you become mentally depressed, helpless, and stressed. Physically you may begin to exhibit psychosomatic symptoms (when mental stress manifests in physical ailments), such as headaches, stomach ulcers, hypertension and chest pain.

The more exhausted and unfulfilled individuals become, the more they withdrawal from enjoyable activities and hobbies, along with relationships. A mental switch is turned to think that personal life is getting in the way of work life, when it’s actually the opposite.

Everyday life functions can be affected such as appetite and sleep. As you may guess, if you allow yourself to get to the point you cannot appreciate life outside of work, it is important to remember that your work will indeed suffer as well.
Getting out of the funk

In order to balance your work and life, you must take an active role in ensuring your own mental and physical health. Positive coping skills can be broken down into the following categories: family, emotional, professional, spiritual, social and recreational.

It’s very important to have a support system, whether it be in the form of blood relatives, friends, or a partner. These individuals help to provide an outsider’s perspective on how we are dealing with certain obstacles, and are also there for support.

Emotional coping skills include being aware when you begin to exhibit signs of burnout and stress and being able to separate yourself from aspects of work that are activating a negative emotional response. This may be due to compassion fatigue or simply spending too many hours at work and not enough time for your own life.

Professionally you can cope better with difficulties at work when you get help and advice from coworkers, and rejuvenate yourself with continuing education opportunities.

You don’t have to go to church to take advantage of spiritual coping methods. Setting aside time for reflection and meditation can help refresh your spiritual side. Getting in touch with the world around you and letting go of your problems for a few minutes every day can help put life in perspective. Those issues that seemed so important and carried a lot of weight in your mind suddenly seem insignificant.

Spending time with friends and your neighbors is great way to enrich your life and separate yourself from your own thoughts. Getting involved in volunteer opportunities can also add a higher sense of purpose.

Lastly, recreational coping is a fun and exciting way to cope with life’s stressors. Starting up a new hobby or activity can help make you feel more well-rounded and is a great stress reliever.

Time management

Identify what areas of your life are most important to you and discover how much time you spend on each aspect. This exercise will help you decide where to make cuts and where to devote more of your time. Most of us will discover we are spending more time on work or work-related thoughts than we should.

Adjusting your own self expectations and learning to say “no” to work-related activities that are intruding on your personal time is an important step. Teach yourself not to feel guilty for taking time for yourself or for needing a change in your schedule at work.

Ultimately, we have to perceive our lives as chapters. The work/life balance struggle will be a constant mental project and an ever-changing process. You will go through periods where you spend more time developing your career goals, and other periods where you need to back off and spend more time and effort on other aspects of life, such as family. The key is taking the time to access your life and make sure you are happy with where your journey is taking you.
Diagnostic Tests for Canine GI Disease: What is Available?
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History and physical examination
A technician plays a vital role in communication with pet owners, and this includes taking an initial history on why the pet is coming in to the hospital. This may include speaking on the phone and encouraging the visit, or taking the owner in to an exam room and hearing about what made them schedule the visit. This is the first opportunity for the owner to explain the signs they have been observing with their dog, and as such is an excellent opportunity to start establishing a problem list and a list of differential diagnoses. The value of this initial history cannot be overstated, as what the technician relays to the veterinarian is often a stepping-stone towards more targeted questions for the owner.

Baseline lab work
A serum chemistry panel, complete blood count, and urinalysis are unlikely to provide a definitive diagnosis for a dog with chronic gastrointestinal disease (such as inflammatory bowel disease), but this is an opportunity to rule out many other underlying causes that could be contributing to the clinical signs. Acute or chronic kidney disease, chronic hepatitis, acute liver toxicity, acute or chronic pancreatitis, and pyelonephritis are all diseases that can initially present very similarly to primary gastrointestinal disease, and these can often easily be excluded on initial lab work.

Certain classifications of intestinal disease can also be identified on routine blood work as well, such as hypoalbuminemia. Dogs with hypoalbuminemia and chronic diarrhea, normal liver function, and negative protein in their urine can be characterized as having a protein losing enteropathy, a more severe and chronic type of infiltrative GI disease. Hypercalcemia, hypocholesterolemia, hypoglycemia, and elevated potassium can be suggestive of hypoadrenocorticism (Addison’s disease), which will often present with waxing and waning chronic GI clinical signs.

Radiographs
Dogs with chronic regurgitation will often be presented for evaluation of “vomiting”, as many owners are not aware of the difference between the two. This is a classic case of how valuable a clinical history can be, as the diagnostic approach is vastly different for these two problems. If the patient is experiencing regurgitation, thoracic radiographs should be taken immediately to investigate for megaesophagus, as well as for aspiration pneumonia which frequently accompanies regurgitation.

Abdominal radiographs are an insensitive tool for primary infiltrative gastrointestinal disease, but can be done to help exclude other causes of similar clinical signs. Hepatomegaly or microhepatica can be seen and are both suggestive of primary hepatic disease. Chronic partial bowel obstruction (stomach or small intestine) can be seen as could a cranial abdominal mass effect that might be causing vomiting.

Radiograph contrast studies provide limited, insensitive information but can be indicated in some cases. Use of an appropriate amount of barium (15mL/kg) is vital for this test to be consistent and reliable. Administering an inadequate volume will affect standardized motility times and can lead to a false diagnosis. Additionally, strict adherence of timing of follow-up radiographs will also help improve the diagnostic utility of this test.

Abdominal ultrasound
Ultrasonography can be a useful test for evaluating the integrity of the gastrointestinal tract. Wall layering and overall wall thickness can be evaluated, identifying both diffuse and focal lesions. Lumen distention and the presence of an acute obstruction can also be appreciated with ultrasound, helping to confirm a presumed obstruction secondary to a mass or foreign object. When the normal structure of wall layering (mucosa, submucosa, muscularis, serosa) is no longer present, neoplasia should be a top differential. Mesenteric lymph nodes can also be evaluated, which are important because they drain the GI tract. In a cat with a diffusely thickened muscularis layer and enlarged mesenteric lymph nodes, for example, small cell GI lymphoma should be high on the differential list.

It should be noted that skill of the ultrasonographer needs to be considered when interpreting ultrasound images. There is a steep learning curve when performing ultrasounds and images can be under- and over-interpreted to a fault in some cases. For instance, it should be known that a small intestine that appears normal on ultrasound can still be markedly inflamed with lymphocytes and plasmacytes consistent with inflammatory bowel disease. On the other hand, a diffusely distended small intestine with fluid in the lumen can represent ileus secondary to many other underlying diseases, and does not necessarily mean there is an obstruction.
**Stool analysis**

A fecal exam for ova and parasites should be one of the first tests performed on a dog that is presented with diarrhea. Standardization of fecal prep in-hospital, or use of an outside diagnostic lab, will help improve the quality of results and increase the ability to get a diagnosis if parasitic disease is present. In addition to a routine fecal flotation, there are other tests that can be performed on the stools.

ELISA testing can be performed on stool samples for infectious diseases including Parvo virus and Giardia. These tests are rapid and inexpensive and can be done cage side, helping to reach a diagnosis faster. In cats, the Parvo SNAP test can be used to test for feline panleukopenia. An extensive PCR panel (DNA testing) can be done on stool as well, which tests for various infectious and bacterial diseases including Clostridium, Campylobacter, Salmonella, etc. I advise taking caution when interpreting this type of test, however, as false positives can occur as well incidental findings. A dog with mild chronic diarrhea that tests positive for Coronavirus, for example, likely has a different underlying disease causing the diarrhea.

Fecal cytology is another simple test that can be performed in-house. This test is generally done to look either for blood in the stool or to quantify Clostridium spores. While a broad collection of different bacteria is normal for a fecal cytology, seeing a large population of a single bacteria type (especially Clostridium spores) should be considered abnormal. Similar to the fecal PCR test, caution should be taken to avoid over-interpreting these results; there are many causes of bacterial overgrowth so this is generally a symptom and not the definitive diagnosis.

A rectal scrape can be performed, primarily looking for intracellular pathogens such as Histoplasmosis. This is a different test from a fecal cytology, as aggressive scraping of the rectal wall should be done to obtain cells in the mucosal layer which may contain the organisms.

Fecal occult blood, on rare occasions, can be a useful diagnostic test. To obtain the most accurate results, the patient should ideally be temporarily on a vegetarian diet (meat proteins can cause false positives) and the stools should be evacuated normally. The act of performing a rectal exam to obtain the feces may be just enough to cause a false positive. The purpose of this test is to try and identify microscopic intestinal bleeding. In lieu of feeding the vegetarian diet, a first test could be submitted and if it comes back as negative then no further action may be needed.

**The GI panel**

Cobalamin and folate are commonly measured in dogs with chronic GI disease. These B vitamins are absorbed at different areas in the small intestine (ileum and duodenum, respectively) and their serum concentration can give insight as to the presence / absence of intestinal malabsorption. The patient should be fasted prior to the blood draw for these samples.

In addition to the vitamin panel, TLI and SPEC cPL can also be measured on this panel. Exocrine pancreatic insufficiency (EPI) is an uncommon cause of small bowel diarrhea and weight loss in dogs but is typically simple to diagnose by measuring serum TLI. The SPEC cPL test is the most accurate test available for diagnosing canine pancreatitis (although there are still many false positives and negatives with this test). The SNAP cPL is a cage-side test that has lower specificity compared to the SPEC cPL but can be used as a screening test to rule out the disease. It is important to understand the limitations of this test and to not over-interpret a weak positive, as many other diseases can contribute to this result.

**Intestinal biopsies**

When a definitive diagnosis cannot be determined with the above testing, taking intestinal biopsies should be the next step. For most cases of chronic enteropathy with presumed diffuse intestinal disease GI endoscopy is my preferred method. This allows the clinician the ability to visually inspect a portion of the GI tract, including esophagus, stomach, duodenum, colon, and possibly ileum, as well as to take multiple biopsies from each of these locations. There is low morbidity with this procedure and the patient can generally go home the same day of the procedure, reducing cost and stress on the patient.

If endoscopy is not available to the clinician or if a focal mass is identified on ultrasound then an abdominal exploratory surgery should be performed. This gives the clinician the opportunity to palpate the entire length of intestines and take multiple representative pieces of tissue for histopathology. If the surgery was performed for a suspected foreign body and no evidence of an obstruction is seen, then biopsies should still be taken prior to closing the abdomen.

The diagnostic approach to gastrointestinal disease can be time consuming and complex but if done systematically with appropriate communication it can be a rewarding experience for both the patient and owner. It is important to remember that therapeutic trials (such as diet and antibiotics) are equally important diagnostic tools that can be used in chronically affected dogs, and to remember that tests done with negative results can be just as important as positive results.
Inflammatory Bowel Disease (IBD) is a common diagnosis in our companion animal patients. With only a small number of exceptions, companion animal patients with IBD are chronic cases that will likely require frequent visits for veterinary care. Many pet owners also suffer from chronic gastrointestinal diseases, and on occasion the similar sounding diagnoses, such as IBD and IBS (Irritable Bowel Syndrome) will cause clients to anthropomorphize the diagnosis of their pet. The purpose of this presentation is to describe the common disorders we call “IBD” in canine and feline patients, and to compare these diagnoses with the more common inflammatory and non-inflammatory gastrointestinal diseases of human beings.

On one level, IBD is fairly easy to understand. Even the name of the condition, Inflammatory Bowel Disease, tells us essentially what is happening. This initially simple situation, however, hides a myriad of subtle differences and altered prognoses. In companion animals, the term IBD encompasses several different histopathological diagnoses. The most commonly diagnosed form of IBD in the dog and the cat is lymphocytic/plasmacytic. As the name suggests, this disease is diagnosed by detecting the presence of increased numbers of lymphocytes and plasma cells (the cells that produce antibodies) in the wall of the intestine. In most areas of the country, lymphocytic/plasmacytic IBD (LP-IBD) is the major form of IBD diagnosed, accounting for about 70% of diagnoses when histopathology is available. The next most common form of IBD in companion animals in most areas is eosinophilic, again referring to the presence of abnormal numbers of eosinophils. Eosinophils are often thought to represent a strong immune response or allergic reaction, possibly to gastrointestinal parasites. Eosinophilic IBD is actually fairly common in the Rottweiler dog, and in this breed it is a common cause of protein losing enteropathy.

Between them, LP-IBD and eosinophilic IBD account for about 85-90% of all diagnoses of IBD in dogs and cats. Other rarer forms are occasionally encountered, often these are diseases with a more severe presentation and a worse prognosis. For these other conditions, please refer to Table One in “Chronic enteropathies in the dog”, elsewhere in these proceedings.

Why do animals get IBD?
For diseases that are remarkably common in our patients, we know very little about how they come about. In part, this reflects the great difficulty of obtaining samples from the intestinal tract of animals. What we do know, though, is that dysregulation of the gastrointestinal tract immune system, and also dysregulation of the microbiome (bacterial populations) in the gastrointestinal tract both seem to play an important part in developing this disease. As mentioned above, LP-IBD is the most common histological type of IBD we diagnose, and it is thought that this process is driven by increased immune responsiveness, either to proteins in the diet, bacteria that are present in the lumen of the intestine, or possibly even to components of the intestinal wall itself. Just why some individuals develop IBD and others don’t is unclear, but we do know that there are breed predispositions (for instance, German Shepherd Dogs, Rottweilers, Basenji, Shar Pei, pure breed cats), which suggests that there is probably a genetic basis to the disease, but just what this basis is is unknown at this time. Similarly, in humans, people with specific major histocompatibility (MHC) grouping antigen types are more prone to development of gastrointestinal inflammatory diseases.

Many dogs with gastrointestinal inflammation, particularly those with lymphocytic/plasmacytic disease, are either intolerant of or allergic to some component in the diet, commonly the animal shows an intolerance to the major protein source of their diet. Because many dogs and cats receive essentially the same diet day in day out for years, the gut is exposed to large amounts of this same protein source over and over. This might be one reason why chronic gastrointestinal disease is becoming so common in our patients, as most animals have far less variety in their diet than their owners do.

Eosinophilic IBD suggests that gastrointestinal parasitism may be present. Dogs and cats are frequently infected with gastrointestinal parasites, and these parasites are often cryptic (ie, hard to diagnose). With many of the parasites of companion animals reinfection is common, particularly when yard soil is heavily contaminated with parasite eggs and larvae. For this reason, animals with eosinophilic IBD are often treated with broad-spectrum anthelmintic drugs such as fenbendazole. Because of the high larval loads that some pets are exposed to in their environments, it may take several treatments and changes in husbandry to completely eliminate parasitic infections. Successful control of parasite infestations is a very important part of an integrated approach to control of IBD in dogs and cats. In some cases, dogs and cats may be having an excessive immune response to bacteria that are present in the gastrointestinal tract.

What are the symptoms of IBD in dogs and cats?
With so many different disease processes that can end up being diagnosed as IBD, it is no surprise that the clinical signs of IBD in dogs and cats are very variable. In the cat, IBD seems to most commonly affect the stomach and the first part of the small intestine, so...
vomiting, loss of appetite and diarrhea (usually quite mild) are common clinical signs. In many cases owners are not aware that their cat has diarrhea, as the severity of the change is mild and many owners use clumping and dehydrating cat litters that dry out the fecal material before the owner notes that it was a little soft. Owners of indoor/outdoor cats are even less likely to be aware that their cat has diarrhea, and the only thing that they might complain about is loss of appetite, vomiting, and sometimes weight loss.

Dogs also develop IBD that affects the stomach (chronic gastritis), but more marked disease in the small intestine and the large intestine is more common in the dog. As owners are more likely to be exposed to the dog’s feces (on walks and in the yard), it is far more common for dog owners with IBD to present their dog with a complaint of diarrhea. If the large intestine is involved (i.e., colitis), the dog may be having accidents in the house, or asking to go out incessantly through the night. Weight loss and reduced appetite can be seen in some dogs with IBD, but these are less common or secondary complaints compared to the diarrhea in most cases.

What is the situation in humans?
Gastrointestinal inflammatory disease in humans is typically one of two diseases, ulcerative colitis or Crohn’s disease. Sometimes it is impossible to differentiate between the two diseases, this is then referred to as “intermediate colitis”.

Ulcerative colitis affects the large intestine, and as the name suggests, is associated with the formation of ulcers and chronic inflammatory lesions. The involvement of the large intestine leads to quite marked diarrhea, often containing blood. Severe bleeding can occur, sometimes to the point of requiring transfusion. Human patients with ulcerative colitis might eventually need to undergo surgery and removal of large sections of the large intestine. Surgical removal of the affected colon can actually cure some human patients with ulcerative colitis. Dogs and cats can develop somewhat similar conditions, ulcerative colitis is occasionally diagnosed in cats, and some forms of colitis in dogs can be quite severe, but these are unusual manifestations of these diseases in our canine and feline patients.

Crohn’s disease tends to be found in the ileum, ileocolic junction and the large intestine, although all parts of the GI tract can be affected. The inflammation in Crohn’s disease often features large numbers of neutrophils and the formation of microabcesses. This type of pathology is actually fairly rare in our small animal patients, who as discussed above tend to develop lymphocyte and plasma cell infiltration, and who tend to have inflammation present throughout the intestinal tract, although it may be more prominent in some areas (such as the stomach and duodenum in the cat, or the large intestine in the dog). Crohn’s disease can lead to gastrointestinal tract obstruction, and some patients will need surgery to remove these obstructing areas. Unfortunately, surgery is not curative for Crohn’s disease in humans. Intestinal obstructions or loss of intestinal mobility in dogs and cats with inflammatory bowel disease is very uncommon, so our patients are far less likely to need to have surgery for their disease (although surgical biopsies are often indicated in their work-up).

Irritable Bowel Disease, or IBS, is actually more common in human beings than either Crohn’s disease or ulcerative colitis. Some estimates put the number of human patients with Crohn’s disease at 27-48/100,000 people (0.02-0.04%); ulcerative colitis at about 1/600 people (0.16%), while IBS may be present in up to 15% of humans, with many of these cases going undiagnosed. A client who hears that their pet has IBD and says “oh, I have that too” is far more likely to have Irritable Bowel Disease than either Crohn’s disease or ulcerative colitis, and if they do have either of the two main inflammatory diseases they are likely to know them by their more common names.

IBS in humans is a quite different disease to the inflammatory bowel diseases. Most notably, inflammation is not a major part of this disease, instead the major symptoms are of increased or abnormal gastrointestinal motility, and often a strong sensation of visceral pain. Humans with IBS may have diarrhea, constipation, or both. In recent years it has become increasingly understood that the neurotransmitter serotonin is an important regulator of the gastrointestinal motility. Some of the drugs used to treat IBS in humans alter serotonin metabolism or serotonin receptors. Interestingly, many commonly prescribed antidepressant medications in humans also alter serotonin metabolism, and many of these drugs can lead to gastrointestinal upset and/or marked worsening of IBS in humans. IBS is also thought to have a psychological component, certainly stress and anxiety can lead to worsening of IBS in human patients. Stress mitigation is a very important part of the management of IBS in humans.

Do our patients get IBS as well?
The occurrence of conditions similar to IBS in dogs and cats is an area of some controversy. Most veterinary gastroenterologists would consider this type of diagnosis to be unusual or rare, many feel that IBS does not exist in small animal patients. One problem with this position is that we have very little knowledge of what the normal speed of movement of the intestinal contents should be in dogs and cats, and what we do know is based on complicated measurement techniques that are not feasible for most practices. Thus, we may miss the presence of markedly abnormal GI motility in some of our patients. Opposing this view, we do know that anti-inflammatory steroids help many of our patients with inflammatory bowel disease, where these drugs are not typically helpful in human patients with IBS.
How do we manage IBD in dogs and cats?
The management of IBD in dogs and cats can be a long and drawn out process, it is important that clients understand that there will be periods where their pet will show worsening of clinical signs, and that their pet may not ever be entirely “cured” of this condition.

A central part of the management of many animals with IBD is dietary modification. Many of these patients are intolerant to the major proteins in their diet, and a change to a new diet will often be recommended. It is important that the new diet is one that contains proteins that the pet has not previously eaten, and during the initial treatment period the pet must eat only the new diet, no treats, snacks or human food are allowed! The new diet is fed exclusively for at least 14 days. If the pet is going to show improvement with the new diet, it should be obvious within that time. Pets failing to respond to their first elimination diet trial should be switched to another new diet, at this point it is important to emphasize to the client that it is important to try and get to the underlying cause of their pet’s disease, and that finding a dietary intolerance or allergy might allow drug-free therapy. If the pet fails the second diet trial, it is usually time to switch to drug therapy. The mainstay treatment for IBD in both dogs and cats is glucocorticoids such as prednisone or prednisolone given orally. Most cases (about 75%) will show a rapid improvement once the glucocorticoids are started, but the doses necessary will cause some dogs and cats to show Cushingoid side-effects, at least in the first several weeks of therapy. Over time, the doses of steroids can typically be tapered down to low doses on every other day.

During this period, it is important that clients understand that most animals with IBD can be managed, and most will eventually be able to have their symptoms controlled. Unfortunately, though, some animals will be more difficult to manage and more aggressive therapies, such as higher level immune suppressive drugs, will be necessary for some patients. It is also important that clients understand that their pet is likely to have flare-ups or reappearance of clinical signs, some times within only a few months of starting their therapy. For some animals, the benefit of therapy is a reduction in the severity or frequency of their clinical signs, they may not be entirely cured. Thus the successful management of animals with IBD requires good, open channels of communication between the pet owner, technical staff of the practice and their clinician.

Further reading
The following publications give good overviews of IBD in companion animals and IBS in people. The internet is full of information as well, but much of it is anecdotal, ill-considered or just plain wrong, the sources listed below are considered reputable.

References

FamilyDoctor.org
National Digestive Diseases Information Clearinghouse
http://digestive.niddk.nih.gov/index.htm
Animals with pancreatic inflammatory disease present to us with widely varying clinical signs and widely varying degrees of illness, which can make the treatment of dogs and cats with pancreatitis both challenging and frustrating. In animals with severe acute pancreatic inflammatory disease, successful management relies on careful assessment, monitoring for and anticipating complications, and diligent nursing care. The purpose of this presentation is to review the approach to therapy of animals with differing forms of pancreatic inflammatory disease. As we will see, the approach to the management of cats can be quite different to the management of dogs, due to the different disease processes in cats and their requirement for high dietary protein intakes.

The dog with uncomplicated acute pancreatitis

Dogs with acute pancreatitis are usually presented for vomiting, lethargy, and possibly collapse and abdominal pain. When the pancreas becomes inflamed, digestive enzymes and inflammatory cells within the pancreas itself and in the fat and mesenteries surrounding the pancreas set up a dramatic inflammatory response. This dramatic inflammation within the abdomen is essentially a form of chemical peritonitis, and is exquisitely painful. When examining dogs with pancreatitis, they will often show abdominal splinting, and may grunt or whine when the cranial abdomen is palpated. These dogs will often have been vomiting for some time, and may continue to vomit when they present to the clinic.

The main clinical signs in a dog with uncomplicated acute pancreatitis are due to the electrolyte and fluid losses caused by the vomiting, the abdominal pain, and ongoing nausea. All of these aspects need to be addressed in therapy of these cases.

Fluid losses are often greater than we would anticipate based on examination of skin tent or mucus membrane moistness. With the inflammation in the abdomen, the gastrointestinal tract can stop moving normally, and these stagnant areas of ileus will accumulate fluid, which is not then available in the circulation. For this reason, intravenous fluid therapy is an important part of the therapeutic plans for these patients. Replacement of the lost fluids expands the blood volume, increasing cardiac output to better perfuse the organs. Maintenance of pancreatic perfusion is very important, as ischemia in the pancreas can perpetuate the inflammation and necrosis. Usually, with cases with marked clinical signs but as yet little in the way of complications, aggressive fluid therapy with standard crystalloid fluids (Normosol-R®, Lactated Ringer’s Solution) is appropriate. The fluids should be supplemented with potassium. Ideally, electrolytes are checked on admission and every 6-8 hours of therapy, with dose adjustments made to the potassium.

Control of vomiting and nausea is important, this is achieved with antiemetic drugs such as metoclopramide, ondansetron or dolasetron, or maropitant citrate. Of these, metoclopramide is the drug that we as a profession have the greatest experience with, and most clinicians will have the greatest comfort using. Routine dosing with metoclopramide is by IV injection every 8 hours, but the use of a continuous rate infusion (1-2 mg/kg/24 hrs) often results in less side effects, and can be very effective. In our practice, we tend to use maropitant as our first line antiemetic. We find it highly effective in many patients for both anti-nausea and apparent pain control, while the once daily dosing (IV or SC) is convenient. Typically, we will start with maropitant, and if vomiting or apparent nausea continues, we will add dolasetron, given IV once daily. In some patients we need to promote gastrointestinal motility, for these we will add in a metoclopramide CRI, followed by erythromycin and ranitidine if extra pro-kinetic effect is needed.

Pain control is absolutely critical to patient comfort and recovery from this condition. With the pre-existing circulatory and gastrointestinal compromise, NSAIDs like carprofen or firocoxib can be quite risky, with an unacceptably high risk of gastrointestinal ulceration and renal failure if the patient’s fluid status has not been properly addressed. Narcotic pain control is recommended.

Traditionally, clinicians have instituted “pancreatic rest” in dogs with acute pancreatitis, with the belief that this would reduce ongoing damage to the pancreas. We now know that this is probably unnecessary, and most likely delays the recovery of sick patients. As long as the dog has stopped vomiting for a period of at least 8-12 hours, which often has already happened by the time the patient is admitted for therapy and stabilized on fluids, I am happy to offer food and water. Usually we will offer small meals of lower fat dog foods, as dietary fat does seem to contribute to the development and perpetuation of pancreatitis in many dogs. Dietary fat also delays gastric emptying, the longer a meal is in the stomach or upper small intestine the greater the chance there is for vomiting.

Bacterial infection plays little to no role in uncomplicated acute pancreatitis, and antibiotic therapy is generally not indicated. Most dogs with uncomplicated acute pancreatitis recover with adequate fluid therapy and pain control. They will usually need at least overnight hospitalization, however many can be discharged within 24-36 hours. Once the dog is willing to eat, urinating appropriately and body temperature is normal, plans can be made to discharge the patient.

Dogs with complicated severe acute pancreatitis

The major potential complications of severe acute pancreatitis are the development of acute renal failure, transient diabetes mellitus (which may become diabetic ketoacidosis), severe systemic vasculitis leading to edema, particularly in the lungs, and disseminated
intravascular coagulation. In animals with complicated pancreatitis of high severity, the replacement and maintenance of circulating fluid volumes, attention to plasma colloid oncotic pressure, and the promotion of oxygen delivery to the tissues are all critical to successful therapy. Dogs with severe acute pancreatitis have a form of circulatory shock with many similarities to septic shock, and the clinical approach to these two forms of shock is essentially identical.

Animals with pre-existing severe inflammatory disease, hypoalbuminemia and multiple organ failure as a result of acute pancreatitis are beyond the therapeutic and management capabilities of most veterinary hospitals, and typically require referral for intensive care if treatment is desired. The prognosis for dogs with acute pancreatitis once they have developed this extent of metabolic derangement is guarded to grave, with mortality rates greater than 75% in some studies.3

Complicated acute pancreatitis, and for that matter any other severe disease process, is a significant metabolic stress to the patient. Nutritional support becomes crucial to the management of severe cases of acute pancreatitis, however it does raise the problem of how do we go about withholding oral intake while attempting to meet the caloric needs of the patient. In human medical practice severe acute pancreatitis patients are often maintained on total parenteral nutrition (TPN). A small retrospective study of nutritional support of dogs with acute pancreatitis has described the use of TPN in dogs with severe acute pancreatitis.4 Another possible route for nutritional support is the through a surgically or endoscopically placed jejunostomy tube.5 These procedures are intricate and time consuming, with the requirement for scrupulous aseptic technique, and thus are probably best performed in dedicated critical care facilities.

Over the last several years, our attitude to management of pancreatitis patients has changed quite a lot. One of the biggest changes has been in the institution of early oral nutrition in pancreatitis patients. In our practice, we are generally comfortable to start offering food if the patient has not vomited for >6 hours. We will recommend some sort of assisted nutrition (such as a naso-esophageal tube or even esophagostomy tube) if the patient is unwilling to eat.

In the special cases where the dog has diabetic ketoacidosis and pancreatitis, our typical approach is to treat these dogs as if they are “typical” ketoacidotic patients, including insulin and dextrose therapy, and feeding as soon as possible. Initial fluid therapy is with isotonic saline at shock rates, after at least an hour of volume expansion with high rate crystalloid fluids we recheck blood glucose, urea and electrolytes, often these parameters will have improved markedly just with the volume expansion. Either intermittent intramuscular regular insulin or regular insulin CRI’s are administered following typical protocols for the management of diabetic ketoacidosis, as described elsewhere.6 When the blood glucose has fallen below 200 mg/dL, food and water are offered. In these cases, the pancreatitis often appears to be secondary to the diabetic ketoacidosis, and management of the fluid, electrolyte and endocrine complications of the DKA leads to resolution of the pancreatitis.

Dogs with this level of disease will invariably require close and scrupulous care. They are often non-ambulatory, and will need close monitoring of their bladder and bowel functions. Cages must be kept clean and dry, both for patient comfort and to maintain fluid line hygiene. Pain control is just as important in these cases as in the less complicated forms, even though the patient may not be showing overt signs of pain due to their dullness or recumbency.

The dog with chronic pancreatitis

Chronic pancreatitis in dogs is often treated in an outpatient setting. Dogs with chronic pancreatitis still experience abdominal pain, often manifested as the “prayer posture”, and often show periods of inappetence or occasional vomiting. Most dogs with chronic pancreatitis have mild disease, and may vomit occasionally, but they usually are not dehydrated or hypovolemic as is seen with the more severe acute pancreatitis cases. Dogs with chronic pancreatitis are managed by diet change in most cases, with the emphasis being on providing a diet with the lowest fat content the dog will tolerate.2 In most dogs, a low fat diet regime and avoidance of table scraps or human food will usually result in a reduction of the frequency that they show clinical signs and resolution of abdominal pain. Some breeds, particularly the Miniature Schnauzer, are at higher risk for the development of chronic pancreatitis and new owners should be counseled on the clinical signs of this condition and the need to avoid high fat foods. In some cases, where fat restriction has not resolved clinical signs, glucocorticoids at low doses or cyclosporine may be necessary to control inflammation.

Dogs with chronic pancreatitis will sometime experience more severe flare-ups of their disease, and might need hospitalization with fluid therapy and narcotic pain control as described above.

The cat with pancreatitis

Cats always have to be different, and with pancreatitis they are definitely different to the dog in many ways. For a long period we believed that cats basically did not get pancreatitis, as the acute severe presentations with vomiting, fever and dramatic abdominal pain were and are rare in cats. We now know that cats have been hiding their pancreatitis from us, and many cats that present for veterinary care for sporadic vomiting, inappetence or generally “feeling off” actually have pancreatitis. Astonishingly, in a large study of cat pancreases at necropsy, the overall prevalence rate for chronic pancreatitis was 67% in ill cats, and even more remarkably, 45% in normal cats.7 Far from being uncommon, it is now apparent that pancreatic pathology, and particularly chronic pancreatitis, is very common in the domestic cat.
As well as hiding their pancreatic disease from us, cats tend to have a quite different form of pancreatitis to dogs. In the cat, most cases of pancreatitis feature lymphocytic infiltration and fibrosis. Breakdown of the cells of the pancreas and release of pancreatic enzymes are not present, or are of only minor importance. This means that fat restriction, the mainstay of treatment for chronic pancreatitis in dogs, is less likely to be effective in the cat. In many cats, the pancreatitis is a part of a complex of inflammatory diseases affecting the abdominal organs, such as IBD and cholangitis. For many cats, dietary modification to a novel protein source diet, as we do for IBD, seems to help control the clinical signs of pancreatitis as well. The author’s preference is to place cats with chronic pancreatitis on higher protein, novel protein, lower carbohydrate diets where possible. Cats with chronic pancreatitis may develop diabetes mellitus, either due to loss of their islet cells with ongoing inflammation, or the development of insulin resistance with obesity and stress. Early dietary interventions to prevent obesity and avoid the development of diabetes mellitus often also seem to assist in the control of these cat’s chronic pancreatitis and other inflammatory abdominal diseases.

Pain control, maintenance of adequate nutritional intake in cats with inappetance, and maintenance of hydration are all critical to success. Most cats with chronic pancreatitis presenting to companion animal practices are able to be managed on an outpatient basis, but the owner should be counseled on the need for close monitoring of food intake and the possibility of worsening of the disease which may require hospitalization for fluid therapy and assisted nutrition.

References
Providing veterinary care to exotic pets is challenging and rewarding, offering the veterinary technician the chance to work with a wide variety of species. There is an inherent challenge for those working with these pets given the large variety of species presented for care. Often the standard organization of a veterinary hospital is not ideal for exotic pets and special accommodations must be made. More time may be required to perform the physical assessment and diagnostic evaluation of exotic pets due to the need to obtain an in-depth history and the sometimes fragile state of these patients. Critical care is an integral part of disease management in zoological species and a high mortality rate is inherent in exotic animal emergency medicine. The nature of these species and the fact that they are fairly recently domesticated dictates that they hide their illness until they have decompensated. Owners need to be aware that their pet is in serious condition but by providing nursing care, you provide them with the best chance for their pet’s recovery. Additionally, front office and support staff must be knowledgeable about exotic pet care and should receive training regarding their handling and husbandry to optimize service of an exotic animal practice.

Natural history and husbandry
It is important to educate your team regarding species identification and the natural history of the species you’re seeing. Certainly years can be spent learning all there is to know about every species kept as a pet, but a general working knowledge of those commonly kept will lend confidence to your client interactions. In addition, knowing the natural history of the species presented to you will help you identify the husbandry requirements of that animal when kept in captivity. The goal in keeping any exotic pet is to maintain it in an environment that is as close as possible to that it would be living in if it were free ranging. Often the presenting problem of an exotic pet is related to deficiencies in the environment or their diet. Being familiar with the husbandry requirements of a species will help you identify potential problems and give insight in implementing treatment recommendations. It will also allow you to help your clients determine if that particular species would make a good pet for them based on their circumstances.

History
A complete history is of paramount importance in providing you with all the information needed to adequately assess the health of the exotic patient. Developing a pre-printed history form will facilitate this process by identifying problems in husbandry to help focus your exam and make specific care recommendations. Additionally, if concerns regarding the patient’s husbandry are addressed prior to the onset of problems, you have provided your client with excellent preventative health care for their pet. Sample history questions should include:

- How long have you owned this pet?
- Where did you acquire this pet?
- What is this pet’s diet (be specific)?
- Are vitamin supplements or medications used for this pet?
- Is water provided for this pet? In what manner?
- How is this pet housed? Describe any cage furnishing the pet has access to.
- What substrate is used in the pet’s enclosure?
- How often is the cage/enclosure cleaned?
- What is the temperature & humidity of the pet’s environment?
- What light is provided for the pet (be specific)?
- What other animals have your pet been exposed to?
- Have you recently added any new pets to your household (within the last year)?
- What problem is your pet currently experiencing?

Preparedness
Many exotic pets requiring hospitalization and nursing care are presented in critical condition. As with domesticated species, the time to prepare for emergencies is before the emergency occurs. Work with your care staff to create a plan to respond to medical emergencies. Most of the supplies and equipment used for emergency care of traditional pets can be used to care for zoological species, but some additional supplies will be needed. Prepare a space in your facility that is ready to care for exotics. Include small gauge needles and small syringes, small endotracheal tubes, and drug doses for exotic species in your crash cart. Organize and hold training sessions to practice emergency techniques on exotic species, using cadavers or models to hone your skills and those of your
support staff. Every good team has members that know their role in an emergency and are prepared to perform it. Make sure your team members are ready.

**Triage examination**
Patient assessment is part of any good triage examination whether the patient has just arrived for an emergency or it is being evaluated after a night of hospitalization and care. It is important to visually assess the patient prior to an actual physical exam. Many zoological species are stressed when in unusual situations and are intolerant to handling and the stress it induces. As animals in critical condition may decompensate from a simple physical examination, decisions must be made as to how much the animal can tolerate at any given time during therapy. It may be necessary to perform diagnostic testing and treatment administration in a staggered fashion, allowing the patient recuperation time between handling periods. Some animals benefit from sedation or anesthesia to minimize perceived stress.

**Basics of nursing care**
Supplemental heat is critical for patients that have lost or do not have the ability to thermo-regulate their own body temperature. This is especially true of avian and reptilian species. Providing an external heat source reduces the physiological stress on the animal during hospitalization care. This can be done with the use of pediatric or veterinary incubators. Often, used pediatric incubators in good working order may be obtained from human hospital surplus stores. Alternatively, radiant heating lamps may be attached to cage doors or placed over aquaria to provide ambient heating when an incubator is unavailable. In this instance, it is important to ensure that the patient and potentially flammable materials are not in direct contact with the lamp as burns and fires are possible. Heating pads and heating discs are good alternatives for providing supplemental heat for patients that are ambulatory and do not need constant thermo-regulation. Temporary heat may also be provided with hot water bottles or rice filled socks that area heated in the microwave. This is especially useful post anesthesia or surgery. A non-ambulatory animal should never be left on a focal heat source without constant monitoring as they may become hyperthermic and not be able to move away from the source to cool down. Additionally, caution must be used when using heating pads for rodent species as they may chew on the power cord leading to electric shock.

Nutritional support must be provided to hospitalized patients, especially those with high metabolic rates where anorexia rapidly results in cachexia. The caloric requirements of the patients must be calculated and those requirements met on a daily basis through self or supplemental feeding. As in domestic species, metabolic rates are measured in kilocalories per day (kcal/day). The basal metabolic rate (BMR), or metabolic rate at complete rest, equals the patient’s weight measure in kilograms multiplied to the 0.75 power times a species coefficient (K). The species coefficient takes into account the type of animal’s metabolic rate. Once the BMR is calculated, the maintenance energy requirement (MER), the metabolic rate based on activity), can be determined by multiplying the BMR by the activity level of the patient (1-2, 1.5 for convalescing animals, 2 for growth).

- BMR kcal/day = K x Weight kg \((0.75)\)
- MER kcal/day = 1.5 x BMR
- K values (placental mammals-70, marsupial mammals-48)

Once the number of kilocalories needed for MER are determined, the amount of food needed to meet that requirement can be calculated and divided over the day’s feedings. The best way to determine if a patient is receiving appropriate nutrition is to weigh the patient daily on an accurate gram scale. Supplemental feeding can then be adjusted as appropriate.

**Nursing care of ferrets**
Ferrets are very resilient and can generally recover well from critical situations. Caution is warranted as even the nicest ferret will bite when uncomfortable or ill. Public health protocols developed for domestic canines should be implemented in response to bites from ferrets. Aggressive or uncooperative ferrets may be restrained for short procedures by providing anesthesia or sedation. A sedation protocol using midazolam (0.5 mg/kg) and butorphanol (0.2 mg/kg) has been effective in the author’s experience. Isoflurane or sevoflurane gas anesthesia administered by mask is also commonly used.

**Hospitalization considerations**
A standard hospital cage is appropriate for ferrets and a litter box should be provided with paper based litter. A towel or hide box is also appreciated as these animals will burrow. Water should be provided in a bowl or sipper bottle, depending on what the ferret is accustomed to, and a high quality kitten food or ferret food should also be provided. Have the owner provide the food that the ferret is used to as some are finicky eaters. If the ferret is not eating on its own, syringe feed a gruel carnivore diet (Eukanuba Recovery, Science Diet A/D, chicken baby food, Oxbow Critical Care for Carnivores, Lafeber Carnivore diet) at a rate of 12-20 ml every 4-6 hours. Syringe feeding may be enhanced by scruffing the ferret and placing the catheter tip at the corner of the mouth to administer small amounts of food at a time.

**Fluid administration**
The cephalic vein is most often used for IV catheterization. A 24 gauge catheter can be placed in the cephalic vein of most ferrets unless dehydration is pronounced. The skin over the vein should be nicked prior to placement to avoid barbing the catheter as ferret
skin is very tough. The catheter is then secured using tape and bandaging. IO catheter placement is typically performed in the proximal femur using an appropriately sized hypodermic or spinal needle. The skin is shaved and aseptically prepared in a routine manner. It is best to anesthetize the ferret prior to this method although a local anesthetic block may also be used. Heparinized saline should be used to keep the catheter patent. Fluids can be administered by SQ, IV, or IO routes. SQ fluids are administered in the intra-scapular area. A large gauge needle should be used as ferrets find this route to be uncomfortable and rapid infusion of warmed fluids is preferred. Fluid requirements are 60-70 ml/kg/day. Fluid therapy instituted for treatment of shock should follow protocols developed for domestic felines.

**Drug administration**
The injection sites for drug administration are similar to those used in domestic pet care. Subcutaneous injections are generally administered in the intra-scapular area; intramuscular injections are administered in the quadriceps muscle mass on the cranial thigh. Oral administration of drugs follows the same technique used to syringe feed a ferret.

**Nursing care of rabbits and rodents**
These species are exquisitely sensitive to stress. Even when therapy seems to be progressing well, these animals can become compromised quickly. As such, supportive care is critical to their management. When stressed or in pain, rabbits and some rodents (guinea pigs, chinchillas) will not eat, leading to gastrointestinal problems (stasis, etc.). These complications can often be more critical than the presenting problem. Rabbits and some rodents (guinea pigs, chinchillas, hamsters, degus) have hind gut fermentative digestion which makes them extremely sensitive to certain antibiotics. Avoid using any beta-lactam (penicillins, cephalosporins), tetracycline or macrolide antibiotics with oral administration. Isoflurane anesthetic restraint can be administered by mask or chemical sedation induced if immobilization is required for a brief period of time. Be sure to monitor respiration and heart rate while anesthetized.

**Hospitalization considerations**
A standard hospital cage or aquarium with lid is used when hospitalizing rabbits and rodents depending on the size and species of the patient. Newspaper or towel bedding is appropriate. Hide boxes are appreciated by many of the small rodent species. Litter boxes should be provided to rabbits using a paper based litter. Food and water should be made available at all times. Water should be provided in either a bowl or bottle depending on which the animal is used to. Most small mammals are sensitive to heat, thus heating pads can be used for supplemental heat under a towel but be sure the animal can move away from the heat source should they becomes over-heated. Supplemental heat is not necessary unless the patient is hypothermic. Pellets, hay, and fresh salad greens may be used to tempt the anorexic rabbit or large rodent to eat. Seed mixes and pellets are generally offered to small rodents. Do not give seeds to large rodents or rabbits. If the patient is not self-feeding, syringe feeding should be implemented using herbivore gruel diets (Oxbow Critical Care for Herbivores, blenderized pellets, canned pumpkin, and vegetable baby food). Small mammals are fed 50 ml/kg/day divided into feedings every 6 hours. Syringe feeding of rabbits and large rodents (guinea pigs, chinchillas) is facilitated by placing the patient on a flat surface wrapped in a towel “burrito”. A small syringe (1-3 ml) filled with gruel is introduced into the mouth caudal to the incisor teeth. This gap between the incisors and cheek teeth (diastema) is an area through with the syringe can safely pass. The food is then introduced into the caudal oral cavity, allowing the animal to chew and swallow before the process is repeated.

**Fluid administration**
A 24 gauge IV catheter can be placed in the cephalic veins of rabbits, chinchillas, and some guinea pigs. Additionally, IV catheters can be placed in the marginal ear vein of rabbits. For small rodents and patients where IV access is compromised, IO catheter placement is indicated. IO catheter placement in rabbits, guinea pigs, and chinchillas performed using the proximal tibia. IO catheter placement in small rodent species is most easily accomplished using the proximal femur. IO catheters may be left in for 3-5 days. IO catheter placement should be performed with the patient sedated or anesthetized. Fluids can be provided by SQ, IV, or IO routes. Maintenance fluid requirements for most small mammals range between 60-100 ml/kg/day, with the average rate being 70 ml/kg/day. Shock fluid rates are 90-100 ml/kg/hr as needed. These fluids can either be given as constant rate infusion or as intermittent boluses. A syringe pump is extremely useful in fluid therapy administration for small mammals.

**Drug administration**
SQ injections are given in the intra-scapular area. Use caution to avoid injecting into the cheek pouches which may extend into the intra-scapular area in some rodents (hamsters). Guinea pigs and rats typically object to subcutaneous fluid administration. Intramuscular injections are administered into the quadriceps muscles on the cranial thigh or the semimembranosis-semitendinosus muscle mass on the caudal thigh. Oral drug administration is performed in the same manner as syringe feeding.

**Resources**
Anemia: It’s Not Only About Bleeding
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Anemia, most accurately described, is a deficiency in the blood’s oxygen carrying capacity due to a reduction in the circulating red cell mass. Measurement of total red cell mass requires specialized testing and is difficult to accomplish in clinical practice. Measurement of PCV, HCT, hemoglobin (Hgb), and RBC count are more common methods in the assessment of erythrocyte content of blood. Thus, anemia is commonly defined as a reduction in these values, and occurs when the rate of red blood cell loss or destruction exceeds the rate of production. Anemia is caused by various diseases, many of them resulting in the patient requiring immediate attention.

Functional role of red blood cells
Erythrocytes or red blood cells (RBCs) exist mainly to transport oxygen obtained through the lungs to body tissues. The cell’s functions are devoted to optimizing oxygen delivery, with all of its potential energy directed towards maintenance of enzymatic and hemoglobin function, as well as cell integrity. The general structure of RBCs involves a cell with no nuclei or organelle and no ability to produce proteins. Therefore, a mature red blood cell must have its full set of proteins to function appropriately. The RBC’s oxygen carrying capacity is very much dependent on hemoglobin, an iron-porphyrin-protein complex. Hemoglobin is synthesized during the erythrocyte’s development to its mature form. The molecule is a tetramer of heme groups working in cooperation to load and unload oxygen molecules. The percentage of heme groups bound to oxygen molecules is characterized by the oxygen-hemoglobin dissociation curve, visualizing the relationship between partial pressure of oxygen in the blood vessel and oxygen binding. Affinity of hemoglobin to oxygen can be affected by various factors, such as temperature, presence of hydrogen ions (pH), and presence of 2,3 diphosphoglycerate (2,3 DPG), altering the ability for transfused RBCs to deliver oxygen in specific conditions.

Erythropoiesis
RBCs are produced through a process called erythropoiesis. Hematopoietic stem cells are progressively differentiated into numerous precursors, eventually expelling their nuclei and developing into reticulocytes, which develop into erythrocytes in 3-4 days. This process is regulated by a hormone called erythropoietin (EPO), which signals for the production of new RBCs. EPO is a glycoprotein produced mainly by the peritubular interstitial fibroblasts in the kidney, though the liver contributes to a small amount of production in anemia. Autocrine production of EPO by erythroid progenitor cells has also been observed. There is a normal amount of erythropoiesis, or basal erythropoiesis, which replaces RBCs living out its life span, maintaining the total red cell mass in a normal range.

Upregulated erythropoiesis in response to an increased EPO production stimulated by inadequate oxygen carrying capacity and resultant hypoxemia and hypoxia (leading up to a 1000-fold increase in severe hypoxia) is called stress erythropoiesis. Any cause for anemia, such as hemorrhaging or hemolysis, can trigger EPO production with a noticeable increase in serum EPO level observed within minutes. Reticulocytes are seen within 3-5 days after EPO level is increased, and take another 3-4 days to mature into erythrocytes. Patients seen within 3 days after the blood loss are said to be in the “pre-regenerative” state with no increase in reticulocyte count. If the blood sample of an anemic patient is showing increased reticulocyte count, the patient has a “regenerative” anemia. Patients without an increase in reticulocyte count after 5 days of ongoing anemia may have “non-regenerative” anemia.

Normal RBC lifespan
Canine RBCs have a normal life span of 100-115 days, while feline RBCs normally live 73 days. RBCs are taken out of circulation as age-related damage occurs. Age-related damage includes compromise in rheological properties due to membrane deformability loss, immunologic removal through IgG binding and opsonization, reduction in antioxidant defenses leading to denaturing of hemoglobin, and compromise to membrane structure through peroxidation of the phospholipid bilayer (oxidative damage). These damaged cells are removed by the macrophages of the mononuclear phagocyte system, involving the spleen, liver and bone marrow.

Types of anemia
RBC loss
One of the most common causes of anemia is an increased rate of RBC loss. Blood can be lost through internal or external hemorrhaging. Internal hemorrhage can involve blood loss into the internal spaces, such as peritoneal, retroperitoneal, pleural, pericardial, and gastrointestinal spaces. There are numerous causes of internal and external hemorrhage. Trauma, surgical accidents, and ruptured neoplasms can cause physical damage to vessels resulting in acute or gradual hemorrhaging. Coagulation factor deficiencies, thrombocytopenia, and thrombocytopenia may render a patient unable to prevent bleeding from normal damage to the
vasculature. Parasitism can lead to external hemorrhage (fleas, ticks, lice) or internal hemorrhage (Ancylostoma, Uncinaria).

Gastrointestinal ulcers and hemorrhagic gastroenteritis are GI specific sources of hemorrhage. Anemia due to hemorrhaging is usually regenerative.

RBCs lost internally may be placed back into circulation through the lymphatic system, or removed from circulation by macrophages, and their components are recycled. Plasma, RBCs, and its iron content cannot be recovered with external hemorrhaging. Hemorrhage leads to a reduction in red cell mass as well as a compromise in perfusion, leading to reduced oxygen delivery. Dogs have a total blood volume of 78-88ml/kg, while cats have a total blood volume of 62-66ml/kg. Blood loss exceeding 20% leads to significant hypotension, and a loss exceeding 30% will lead to hypovolemic shock and possible death.

Treatment of RBC loss through hemorrhaging is directed at maintaining oxygen delivery through providing adequate perfusion and oxygen carrying capacity. Fluid therapy to address hypovolemia is warranted to ensure perfusion to vital organs. Restoration of a normal blood volume will not be possible without addressing the source of the hemorrhage. Locating the source of the blood loss may be more obvious in the case of external hemorrhaging, but may prove to be a challenge if internal. Hemorrhaging from trauma, surgical accidents, and ruptured neoplasms are often stopped through surgical intervention. Coagulation factor deficiencies warrant replacement through appropriate plasma products. The cause of thrombocytopenia and thrombocytopenia should be addressed with appropriate therapy, and platelet transfusions administered if the hemorrhaging is life threatening. Correction of hemostatic disorders should ideally be accomplished before surgical intervention, if warranted. Ectoparasite and endoparasite infestations will require the appropriate anti-parasitic. Gastrointestinal ulcers will require removal of causes and supportive care.

If the patient is showing a rapid decline in lab values related to red cell mass (PCV, HCT, or Hgb) or showing clinical signs of hypoxia due to the anemia, a red blood cell transfusion is warranted. Clinical signs of anemia may vary slightly due to the cause of anemia. In general, any form of anemia is associated with paleness. Patients who have compromised delivery of oxygen will exhibit signs of weakness, exercise intolerance, lethargy, fatigue, and sometimes collapse. Their mentation may be dulled due to brain hypoxia. In acute anemia, a prolonged capillary refill time (CRT) due to peripheral vasoconstriction to shunt blood to vital organs may be seen (Patients with chronic anemia will show a normal CRT). Tachycardia, tachypnea, and bounding pulses without other explanations also point towards compromised oxygen delivery.

**RBC destruction**

Various types of defects in RBCs can cause an increased rate of destruction leading to anemia. Anemia due to hemolysis results in lowered red cell mass and subsequent reduction in oxygen carrying capacity without significant changes in plasma volume. Hemolysis can be intravascular (destruction of RBC within the blood stream), extravascular (phagocytosis by macrophages in the spleen, liver, bone marrow, and lymph nodes), or both. Intravascular hemolysis will result in the presence of free hemoglobin in the plasma, leading to hemoglobinemia and hemoglobinuria (when renal threshold is exceeded). Hemoglobinuria leads to tubular necrosis resulting in acute kidney injury in humans, and poses similar concerns in veterinary medicine. Jaundice may be seen in patients with RBC destruction rate exceeding the liver’s ability to process bilirubin. In addition, the presence of red blood cell fragments may trigger disseminated intravascular coagulopathy. Extravascular hemolysis can lead to splenic enlargement, though other intravascular signs of hemoglobinemia, hemoglobinuria, and jaundice are not seen. Hemolytic anemia is usually regenerative.

Genetic defects of red blood cells, though rare, can cause hemolytic anemia. Elliptocytes, stomatocytes, and pyruvate kinase defect lead to reduced life span due to abnormalities in RBC membrane and shape. Spectrin deficiency and phosphofructokinase defect lead to reduced life span by increasing the fragility of RBCs.

Hemolysis is most commonly caused by acquired RBC defects, resulting in direct membrane injury or osmotic lysis. Exposure to chemicals and drugs that cause Heinz body formation will lead to removal of these red cells from circulation through the phagocytic system or cause direct lysis. Causes of Heinz body formation include toxins contained in food (onion, garlic, propylene glycol), drugs (acetaminophen, vitamin K1 and K3, benzocaine), and chemicals (copper, naphthalene, skunk musk, zinc). Cats are more prone to Heinz body formation, but are also more forgiving towards red cells containing Heinz bodies, allowing for a longer survival time. Because of this, feline RBCs may show Heinz bodies without anemia. Cats can develop Heinz bodies when exposed to propylene glycol, and are more prone if inflicted with diabetes mellitus, lymphoma, or hyperthyroidism. Cats with diabetes mellitus or hepatic lipidosis can develop hypophosphatemia which also can cause hemolysis. Phosphate supplementation is recommended if a phosphate level below 0.5mmol/L is seen. Intraerythrocytic parasites such as Babesia canis and Cytauxzoon felis, can lead to hemolytic anemia as well.

Hemolysis may be caused by antibody or complement response to the surface antigens of red blood cells by the patient’s own immune system, termed immune-mediated hemolytic anemia (IMHA). Extravascular hemolysis can result from an immunoglobulin G (IgG) mediated type II hypersensitivity (cytotoxic) reaction. Phagocytic loss of RBC membranes reduces the surface area of the RBC, leading to formation of spherocytes (RBCs that have lost the biconcave structure). Gross agglutination of red cells may also be seen. If the immune response is initiated by factors such as cancer, drug administration, or infection, the hemolytic anemia is considered to be secondary IMHA. Passive acquisition of anti-red cell antibodies through blood transfusions andcolostrum can cause an IMHA as well. The latter results in a phenomenon called neonatal isoerythrolysis, where anti-red cell antibody is passively acquired by a
nursing neonate, resulting in the destruction of red cells. When no causative agents can be identified, the hemolytic anemia is considered to be primary IMHA, or auto-immune hemolytic anemia (AIHA).

Changes in rheology and passage of RBCs through narrow vessels can cause mechanical and shearing damage to the membranes. Hemoglobinemia and hemoglobinuria result as this is a form of intravascular hemolysis. Fragmented schistocytes and keratocytes are seen on blood smears as an indication of mechanical damage. Patients with cardiac disease, severe heartworm infection, hemangiosarcoma, patent ductus arteriosus, and any other causes of altered blood flow and microangiopathy may show signs of fragmentation of RBCs. DIC can be a cause of fragmentation, and at the same time precipitate DIC.

Efforts in treatment of hemolytic anemia are directed towards removing the cause of the hemolysis, and supporting oxygen carrying capacity as needed. Genetic disorders typically cannot be completely resolved, though patients with disorders leading to hemolysis through the mononuclear phagocytic system may benefit from a splenectomy. Exposure to chemicals inducing Heinz body related hemolysis should have the source removed (change in diet, surgical removal of the ingested copper or zinc material). Some toxins may have antidotes such as acetylcysteine in acetaminophen toxicity. Therapy for mechanical injury induced hemolysis is directed at the underlying cause. IMHA is treated with immunosuppressive agents such as glucocorticoids, cyclosporine, mycophenolate, azathioprine, and intravenous immunoglobulin.

Regardless of the cause of the hemolysis, when the anemia leads to clinical signs of hypoxia, oxygen carrying capacity is supplemented. Packed RBC (pRBC) transfusions are typically the ideal choice as hemolytic anemia does not cause blood volume loss. pRBC will provide oxygen carrying capacity while minimizing the volume of transfused product, reducing the chances of fluid volume overload. In the case of IMHA where finding compatible blood or simply judging compatibility may be difficult, hemoglobin-based oxygen carrier solution (HBOCS) administration may be beneficial.

Decreased production

Anemia can result from a reduced production of red cells as well. One cause for reduced red cell production is a decreased level of EPO, leading to reduced erythropoiesis. Patients with chronic renal disease often become anemic as EPO production by the kidneys are diminished. Other factors such as uremic toxins leading to a lowered red cell half-life, hemorrhagic loss due to GI ulcers, increased bleeding tendencies due to platelet dysfunction, inhibition of iron store release, suppression of erythropoiesis by the parathyroid, and reduced nutrient intake may also contribute.

Suppression of response to EPO is another cause of reduced production. In the presence of chronic inflammatory disease such as chronic infections, chronic immune conditions, and malignant cancers, or in acute inflammatory diseases, red cell production is reduced. This is attributed to an increased production of hepcidin by hepatocytes during inflammatory disease, which inhibit the iron exporting action of ferroportin in macrophages and enterocytes. This reduces the iron available for erythropoiesis. In addition, inflammatory mediators (tumor necrosis factor-α and interleukin-1) released from leukocytes reduce surface EPO receptors on erythroid stem cells, leading to suppression of erythropoiesis.

Dysfunction of the bone marrow may be another cause for reduced RBC production. Irradiation, toxicities, viral or bacterial infections, and administration of certain drugs can result in marrow aplasia, leading to a lack of marrow stem cells. Myelophthisis, or marrow suppression secondary to marrow infiltration by tumors can displace or inhibit production of hematopoietic cells. Both of these situations result in a pancytopenia. In FeLV infections in cats or immune-mediated erythroid stem cell destruction in dogs, erythrocyte precursor cells are specifically reduced in number, leading to red cell aplasia.

When nutrients required for producing the signaling system for erythropoiesis and functional erythrocytes are deficient, anemia will occur. Folic acid, vitamin B12, cobalt and intrinsic factor (a glycoprotein aiding in absorption of vitamin B12) deficiency can result in a dysfunction of DNA and RNA synthesis, leading to production of erythrocytes of abnormal shape and size. These abnormal cells are destroyed in the bone marrow, thus never making it into circulation. Administration of drugs that antagonize folate (methotrexate for malignant tumors), inhibit folate metabolism (sulfonamides), and deplete folate concentrations (phenobarbital) are potential causes of malformed erythrocytes. A genetic disorder in Giant Schnauzers, Beagles, and Border Collies involving selective malabsorption of vitamin B12 has been reported and lead to a non-regenerative anemia. A deficiency in iron results in production of erythrocytes with a reduced concentration of Hgb, or lead to delay in red cell production resulting in anemia.

Treatment for non-regenerative anemia consists of supportive care while the underlying disease process is treated. Infectious and toxic causes may be alleviated over time, yet neoplastic and genetic causes typically have no complete resolutions. Ineffective erythropoiesis due to nutrient deficiency can be alleviated through supplementation. In the case of decreased EPO levels, such as chronic kidney disease, EPO may be administered to promote erythropoiesis. If the anemia reaches a point of clinical signs of hypoxia, administration of red cell products or HBOC solution may be beneficial.
CPR: The RECOVER Guidelines
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In June of 2012, the Reassessment Campaign on Veterinary Resuscitation (RECOVER) published the first evidence based guideline for veterinary cardiopulmonary resuscitation (CPR). The initiative was launched after considering the difference in success rates of CPR between human (20%) and veterinary (6-7%) settings, with the human counterpart having established evidence-based guidelines through the American Heart Association. There are definitely physiologic and anatomic differences between human patients and veterinary patients, but one would expect a comprehensive, evidence-based treatment strategy on execution of CPR to improve the outcome through optimization of the CPR protocols. The RECOVER initiative was carried out through the involvement of over 80 experts from the American College of Veterinary Emergency and Critical Care (ACVECC) and American College of Veterinary Anesthesia (AVTA) of multi-national background evaluating published studies available to answer clinical questions organized into 5 different subtopics to arrive at a consensus guideline. This groundbreaking effort not only produced a guideline that is now utilized all over the world to refine CPR practices, but has also injected fuel into the drive towards evidence-based practices in veterinary medicine, and sparked many other movements in the process.

Evidence in CPR

Many clinical questions asked in 5 different “domains” of (1) Preparedness & Prevention, (2) Basic Life Support, (3) Advanced Life Support, (4) Monitoring, and (5) Post-Cardiac Arrest Care were answered to confirm or disprove existing beliefs, provided new knowledge, and also allowed us to identify gaps in the knowledge available to come to definitive answers.

The guideline emphasizes importance in early initiation of CPR as a key factor in successful outcome. Preparation for swift intervention when a patient going into cardiac arrest can be accomplished through thorough training of the staff in both didactic (knowledge) and psychomotor (physical) aspects of CPR. CPR drills simulating the arrest and response allows staff members to better understand the sequence of events and potential turns the event may take. Periodic refresher training sessions at least every 6 months is recommended. Preparation of the facility through setup of a crash cart in a central location, which is regularly checked for stock with a detailed checklist will allow for easy access to supplies and equipment required for CPR. Cognitive aides consisting of the CPR algorithm, drug dosage charts, CPR priority checklist should be readily available in the emergency area, with the staff trained on their usage prior to the event, helping adherence to proper protocol.

Swift intervention is better made when cardiopulmonary arrest (CPA) is recognized quickly, and CPR initiated. Assessment of the patient for CPA should be performed in no more than 10-15 seconds through a standardized approach. If CPA is even suspected, chest compressions should be started right away since any delay can significantly reduce the chance of success, accurate assessment of a lack of a pulse is difficult without taking a long time, and performing compressions on a patient that is not in CPA brings very little harm. In an inpatient situation, clear identification of patients at risk of CPA to the staff should allow for earlier recognition.

Basic life support

CPR starts with provision of basic life support (BLS) as the priority, and most important aspect of CPR. The mnemonic CAB is now used to describe the priority order of circulation, airway, and breathing, because breathing is not helpful in oxygen delivery if circulation of blood has ceased. Evidence points towards delay in initiation of compressions leading to lower success rates in CPR. In regards to compressions, there were no differences seen between right and left lateral recumbency. Chest compressions should be performed to 1/3 to 1/2 of chest width (which takes quite a bit of force for large animals, while moderation may be required for smaller patients) at a rate of 100-120 compressions per minute while allowing full chest recoil in between. The compressions should be focused at the highest point of the chest for dogs with normal conformation, over the heart for keel-chested dogs, and over the sternum in flat-chested dogs (such as some bulldogs). Small dogs and cats should have compressions performed over the heart, and compressions may be performed with a circumferential or two-handed technique. The use of a metronome, songs, or other methods of keeping the rate consistent to recommended rates is useful. Even when compressions are executed properly, it may only produce about 30% of normal cardiac output, which illustrates the need for swift and proper compressions during CPR. Interrupting of compressions significantly reduces the forward flow created through consistent application of compressions, and is best avoided. Compressions should not be stopped to auscultate the heart, check for pulses, assess the patient, or place an endotracheal tube for a full 2 minutes per cycle of compressions. 10-15 seconds in between 2 minute cycles should be used for assessment of the patient, and compressions resumed promptly if no change in CPA is seen. The compressor should be switched between cycles as well, to prevent physical fatigue as 2 minutes of repetitive compressions is physically demanding.
The airway should be verified to be patent, and any obstructions dislodged. Endotracheal intubation should be performed without interruption of compressions, and ventilations performed approximately at 10mL/kg tidal volume (or 20cmH2O of pressure if no spirometer) at 10 breaths per minute with an inspiratory time of 1 second. Hyperventilation is best avoided to prevent vasoconstriction from low CO2 levels leading to poor cerebral perfusion. Mouth-to-snout ventilation may be used if supplies for endotracheal intubation are not available. In the case of single person CPR, 2 short breaths in between 30 chest compressions is recommended.

**Advanced life support**

With basic life support provided, the attention of the CPR team should be directed to providing advanced life support, including monitoring, drug therapy, and electrical defibrillation. The two forms of monitoring that prove useful during CPR is the electrocardiogram (ECG) and end-tidal carbon dioxide (ETCO2) monitoring. Pulse oximeters and oscillometric or Doppler blood pressure monitoring is not effective in assessment during CPR due to movement and poor perfusion state. The electrocardiogram is also prone to motion artifacts during compressions, making interpretation difficult. Regardless, specific tracings may be seen during or in between compression cycles, guiding therapy. Asystole, pulseless electrical activity (PEA) and ventricular fibrillation (VF) are notable arrhythmias seen in CPR.

Capnography, or measurement of CO2 in the breaths coming out of the patient is monitored easily in a patient that is endotracheally intubated. ETCO2 measurement is the most reliable form of monitoring for effective compressions since the level of CO2 measured correlates to the level of perfusion the lungs are receiving, given there is no severe pulmonary pathology. ETCO2 levels higher than 10-15mmHg during CPR was observed to give a higher chance of return of spontaneous circulation (ROSC). Upon ROSC, ETCO2 increases significantly as perfusion to the lungs are re-established, and can be used as an indicator of ROSC.

Drugs can be administered intravenously (IV) or intraosseously (IO) during CPR, and access should be established without interruption of compressions. Vasopressors, parasympatholytics, anti-arrhythmics, reversal agents, IV fluids, and alkalinizing agents are used in specific situations during CPR. Vasopressors are indicated for use in CPR regardless of ECG readings to increase systemic vascular resistance and optimizing perfusion through the reduced cardiac output. Epinephrine, an alpha-1, beta-1 and beta-2 adrenergic agonist causes vasoconstriction, and is given at a low does (0.01mg/kg) initially, and at a high dose (0.1mg/kg) with prolonged duration of CPR. Vasopressin is an alternative that may be used in place of epinephrine at 0.8U/kg. Both vasopressors are given every other cycle of compressions due to its half-life.

Atropine has traditionally been given as an anticholinergic and a sympatholytic drug. There is minimal evidence indicating benefits of atropine administration during CPR, though there is also no evidence of harm. Atropine is given at 0.04mg/kg IV or IO at the initiation of CPR or as soon as IV or IO access is established, with redosing performed every other cycle of compressions. Anti-arrhythmics may be useful in ventricular fibrillation (VF) that does not respond to electrical defibrillation. Amiodarone at 2.5-5mg/kg IV or IO is recommended, with lidocaine at 2mg/kg slow IV or IO being a secondary option. Reversal of any anesthetic or analgesic drugs seems reasonable though no evidence is seen. Opioids can be reversed with naloxone (0.04mg/kg), benzodiazepines with flumazenil (0.01mg/kg), and alpha-2 agonists with atipamezole (0.1mg/kg) or yohimbine (0.1mg/kg), each IV or IO.

Intravenous fluids may be beneficial if the patient is known or is suspected of hypovolemia to help restore intravascular volume and perfusion, but is unlikely to be of any benefit (and may even be detrimental) to those that are euvolemic or hypervolemic. Corticosteroid administration may have been traditionally performed, though evidence suggests more potential harm than benefits, discouraging its use. Sodium bicarbonate administration is considered in patients with prolonged CPA (10-15 minutes) to counter effects of metabolic acidosis which is likely to be present.

Electrical defibrillation is useful in patients with VF and has been associated with a higher rate of ROSC. Electrical defibrillation delivers an electrical shock to the heart “resetting” the myocytes and allowing them to resume a more orderly conduction and contraction pattern. Monophasic and biphasic defibrillators are available on the market. Biphasic models are recommended over monophasic because of the higher success rate and less damage caused by a lower current used. Defibrillation should be performed in between compression cycles to minimize interruptions and allow for recharging of the defibrillator should repeated discharges be necessary.

**Post-resuscitation care**

The survival to discharge rate of a patient that successfully achieves ROSC is quite low, reported to be 16% in one veterinary study. The final outcome has a multitude of factors including underlying disease, the cause of CPA, and damage to tissues sustained during and after CPR. Post-resuscitative care is directed towards respiratory optimization performed through monitoring and providing adequate ventilation and oxygenation, hemodynamic support with IV fluids, vasopressors, and inotropes as indicated, and neuroprotective therapy consisting of seizure control, permissive hypothermia, and intracranial pressure control. Optimization of the respiratory, cardiovascular, and nervous systems allows the best chance for patient life to continue while the underlying disease is treated.
Non-medical aspects

Even when patients at risk of CPA are identified ahead of time and the team is prepared with the appropriate facilities to perform CPR, administration of CPR can be quite chaotic. The aim is to bring as much organization and order to the chaos as humanly possible. One of the biggest factors to keeping the order is the organization of a team. There are several roles to be established ahead of time in training for any one person to be comfortably able to fill all roles necessary. The roles needed are: CPR leader, compressor, ventilator, record keeper, drug handler, and the veterinarian. The CPR leader should be identified at the beginning of CPR, so assigning of subsequent tasks can begin immediately. Staffing permitting, the CPR lead should be freed from tasks aside from assigning and keeping the team organized. Compressor and ventilators provide the compression and ventilation, and may make sense to alternate with each other between compression cycles if staffing is limited. The record keeper should keep a detailed medical record during CPR, and this task is facilitated with a CPR record form. The drug handler will prepare and administer drugs in most cases. The veterinarian ideally is not fulfilling any of these roles, being able to focus on the patient and making judgments on whether CPR efforts should continue, decisions on drug administration, communication with the owners, and any medical interventions that are necessary for the patient.

Communication during CPR is also vital to inn organizing the effort and preventing mistakes. Closed-loop communication, performed through the person making a request addressing an individual clearly by name, the addressed individual repeating back their understanding of the request, the request being fulfilled being announced, and the requestor acknowledging the completion.

Keeping the communication loops closed each time may feel awkward if it is not used on a regular basis, but contributes to very organized communication allowing everyone on the scene to stay on the same page on the status of the CPR. Double checking each other on tasks being performed is also possible, preventing the preventable mistakes.

Debriefing is another form of communication that is hugely beneficial for the team, regardless of the outcome. After the conclusion of CPR, every member should participate in a 5-15 minute debriefing session discussing the CPR. The discussion will be lead by the CPR lead, discussing the following points:

1. What went well with this CPR session?
2. What could we have done differently?
3. Are there any goals we can set for ourselves for future CPR sessions?
4. Are there any serious concerns you would like to bring up?

Debriefings will bring your team even closer together as a functional unit. This also provides opportunities for staff members to express any stress they may have faced in a productive and constructive manner, and a chance for better understanding of the event that passed. Debriefing is intended for us to be able to think towards bettering our effectiveness in CPR, providing each individual patient the best possible chance of recovery and positive outcome. Bring your open mind, active listening, and participation to each of these debriefings. Commend each other on what was done well, regardless of the outcome. Discuss what could be done differently to perform CPR better. Every opinion is important, and should be discussed in a professional manner. Being open in communication requires trust and willingness to give and take feedback without bias and being personally affected.

Has RECOVER made a difference?

In the first year since the implementation of the RECOVER guideline into our CPR training protocol, a total of 54 CPR efforts (35 dogs, 18 cats, 1 chinchilla) have been made at the author’s practice (data collection for the second year is ongoing). The average age was 9 years old, with variable reasons. The average duration of CPR was 11 minutes, with the shortest effort lasting 1 minute and the longest 32 consecutive minutes (one effort lasted 47 minutes total, with intermittent ROSC). The ROSC rate was 24.1% (13 of 54), with a survival-to-discharge rate of 3.7% (2 of 54). The average duration of CPR effort achieving ROSC was 9.5 minutes (high: 27, low 1). One of the patients who were eventually discharged was suspected to have experienced hyperkalemia related cardiac arrest from urinary obstruction, while the other was suspected to have suffered from severe metabolic acidosis and potential over-supplementation of potassium as insulin doses were reduced without a change in KCl content of IV fluids (diabetic ketoacidosis patient). Comparison with previously published statistics unfortunately does not yield a significant difference at our practice. The staff, however, feels better prepared for the ultimate emergency, and feels confident the best chances are provided for each of our patients.

Other effects of RECOVER

RECOVER has brought on some indirect changes to the veterinary field in addition to providing standardization of CPR protocols. The Academy of Veterinary Emergency and Critical Care Technicians (AVECCT) is in the process of using similar evidence grading methods used by the RECOVER initiative in creating evidence-based nursing guidelines, inspired by the initiative. Evidence-based veterinary medicine (EVBM) has gained significant momentum since RECOVER guidelines were published, and while this could be temporally a coincidence, there is no denying the initiative adds significant weight to the importance of EVBM. ACVECC is now offering a college sanctioned veterinary CPR BLS training program through Veritas, offering certification for both didactic and psychomotor training. The training program, in the long term is anticipated to reach the public. As the immediate next steps, an
advance life support course is being designed, and a trainer certification program is also in the works. Advancement in the field of veterinary emergency and critical care has been accelerated due to the RECOVER initiative.
Respiratory distress is a very common form of emergency in veterinary medicine. The primary role of the respiratory system is to oxygenate and control CO2 levels in the blood. Inability for patients to properly oxygenate blood and saturate hemoglobin (hypoxemia) will lead to inadequate delivery of oxygen to the tissues (hypoxia). In a hypoxic state, cellular energy production is shifted primarily to anaerobic metabolism, resulting in lactic acid buildup and acidemia (metabolic acidosis). In addition, insufficient alveolar ventilation will lead to an elevation in the arterial CO2 level, or hypercapnia. Hypercapnia leads to respiratory acidosis, decreases cardiac contractility, and depresses diaphragmatic function. Both of hypoxemia and hypercapnia, when allowed to persist, will lead to the demise of a patient and swift assessment of respiratory compromise is required for appropriate treatment.

Initial assessment and treatments
Assessment of a patient starts with external physical signs. Patients presenting with signs such as tachypnea, increased respiratory effort, and open-mouth breathing are clearly in trouble. Exaggerated movement of parts of the body surrounding the physical construct of the airway such as flaring nostrils, lip movement with respiration, sucking in and out of the skin under the chin and thoracic inlets, and paradoxical abdominal movement are all signs of significant effort. An orthopneic position, characterized by open-mouth breathing, extending of the head and neck, sitting up sternal, and abduction of the elbows in the effort to open up the airway as much as possible, is another external sign of respiratory distress. If the patient progresses to being unable to hold themselves up, going into lateral recumbency with no improvement in respiratory signs, the patient may be experiencing fatigue and facing imminent arrest.

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Assessment and treatment of a patient in respiratory distress poses a dilemma, as swift determination of the patient’s problem is required, yet they may be compromised such that the stress of diagnostics and treatment may push them into respiratory and cardiac arrest. These patients are in a very fragile state, and initial efforts are aimed at improving the patient’s ability to breathe while minimizing stress and deterioration in respiratory status. Providing oxygen supplementation through flow-by, mask, induction chamber, or cage would be one of the first lines of therapy to alleviate distress.

Patients in respiratory distress are often very anxious, which often makes the patient even more dyspneic. A light sedation with small doses of benign sedative such as butorphanol may be beneficial to help ease anxiety. The staff working on the patient should conduct themselves in a calm and quiet manner yet maintaining swiftness. A calmer environment will not only benefit the patient, but may benefit a worried owner. The presence of the owner can either be beneficial or detrimental to the patient, and staff directing attention to calming a panicked client (and successfully doing so) may also help the patient.

Diagnosis and treatment such as physical examination, radiographs, blood work and IV catheterization may have to be held off until the patient is more relaxed and breathing better. Evaluation of a patient’s respiratory problem begins with external visualization of their breaths. The manner in which a patient breathes is adapted to the method requiring the least work of breathing. An obstructive breathing pattern, involving a prolonged inspiration (upper airway) or expiration (intrathoracic lower airways), will be observed in patients with narrowed airways. A restrictive breathing pattern, involving shallower but tachypneic breathing, will be observed in pleural disease or reduction of lung compliance. Abdominal effort may be seen with patients with compromised lungs. Certain conditions (anemia, metabolic acidosis, and pain, for example) can cause “non-respiratory look-alikes”.

Auscultation is a valuable skill in early detection and detection of change in lung states. Stertor (snoring), wheezes (whistling), and stridor (high pitched noise) can indicate different upper airway issues. Crackles indicate fluid in the alveoli, such as in pneumonia or pulmonary edema. The location lung sounds are present or absent in helps indicate causes as well. Cardiogenic pulmonary edema often begins near the heart (perihilar region), and aspiration pneumonia often originate in the cranioventral lobes. Absence or decrease in lung sounds in the caudal and ventral fields may indicate pleural effusion, while dorsal fields may be due to pneumothorax. While abnormalities in auscultations do not lead to a diagnosis, it serves as an indication for further diagnostics.

If a pleural space issue like pleural effusion of pneumothorax is suspected, performing thoracocentesis to evacuate the fluid or air can provide diagnostic information and therapeutic treatment simultaneously. The staff should be prepared to perform endotracheal intubation and provide positive pressure ventilation (PPV) if the patient does not stabilize with initial treatment and deteriorates.

Types of respiratory emergencies
Upper airway problems
Upper airway issues leading to respiratory distress are common in veterinary medicine, and can involve various causes. Laryngeal paralysis is common in older, larger breed dogs, with a higher prevalence in males. A loss in innervation of the cricoarytenoideus dorsalis muscle leads to atrophy, preventing the artenoid cartilage from being abducted. The laryngeal opening is narrowed, leading to an increase in airway resistance. Causes may be congenital or from trauma, neuromuscular disease, neoplasia, hypothyroidism, or...
idiopathic. Affected dogs exhibit inspiratory stridor, exercise intolerance, ptalism, and a change in their bark. Laryngeal paralysis can cause severe respiratory distress and collapse depending on severity. Emergency stabilization involves endotracheal intubation. Once stabilized, medical management may be possible with the goals of minimizing stress, excitement, and exposure to high environmental temperature. Surgical treatment is most effective, involving unilateral lateralization of the arytenoid cartilage, or laryngeal tie-back. Chances of aspiration pneumonia are increased in dogs undergoing this procedure. Cats rarely present with laryngeal paralysis, though a study suggests laryngeal paralysis a differential for cats with dyspnea, inspiratory stridor, coughing/gagging, or a change in voice, with complete resolution through unilateral lateralization.

Brachycephalic syndrome results from anatomical abnormalities seen in brachycephalic breeds leading to upper airway narrowing or obstruction. Stenotic nares, enlarged tonsils, and elongated soft palate require the breed to create larger negative pressure to breathe normally, creating a narrowed airway from hyperplasia of the airway beyond the nares. A further increase in negative pressure may evert laryngeal saccules and collapse the larynx or trachea. Pulling of air through the narrowed airways can further cause inflammation and edema, placing the patient in further respiratory dysfunction. Surgical intervention through widening of the nares, resection of the palate and everted laryngeal saccules, and removing the tonsils is the recommended treatment. In addition, upper airway obstruction may occur due to lodging of a foreign body, neoplasia, or the formation of nasopharyngeal polyps. Any upper airway dysfunction or obstruction can lead a patient to present with respiratory distress. Secondary complications such as non-cardiogenic pulmonary edema, heat stroke, and aspiration pneumonia may be seen.

Pulmonary edema is a common cause of respiratory distress in dogs and cats. Accumulation of extravascular fluid occurs in the alveoli and pulmonary parenchyma due to increased hydrostatic pressure or increased permeability in the pulmonary vasculature. Patients present with respiratory distress and have poor oxygenation. The reduced oxygenation is due to a ventilation-perfusion mismatch (V/Q mismatch) because the presence of fluid in the alveoli leads to compromised ventilation.

Left sided heart failure can lead to pulmonary hypertension, causing cardiogenic pulmonary edema. In cardiac disease, fluid retention and an increase in blood volume is seen as a compensatory mechanism for lowered cardiac output. The chronic increase in blood volume leads to an increased hydrostatic pressure (because of congestion) in the pulmonary vasculature, resulting in pulmonary edema. Patient with cardiogenic pulmonary edema may show signs of coughing, exercise intolerance, and may have a heart murmur. An echocardiogram may be performed to confirm cardiac disease and pulmonary hypertension. Fluid volume overload through fluid therapy is a possible cause of cardiogenic pulmonary edema, especially in patients with cardiac or kidney disease. Both cardiac and kidney disease can be asymptomatic, so patients on fluid therapy should be closely monitored for signs of fluid overload.

Non-cardiogenic pulmonary edema can occur from increased permeability within the lung tissue through damage to the microvasculature or alveolar epithelium. Electrocution, seizures, strangulation, pulmonary thromboembolism, and chemical exposure are all potential causes. Illnesses associated with systemic vasculitis such as sepsis and systemic inflammatory response syndrome (SIRS) are also associated with non-cardiogenic pulmonary edema.

Patients with pulmonary edema are treated with oxygen to alleviate hypoxemia. Patients that are unable to maintain an arterial partial pressure of oxygen (PaO2) greater than 60mmHg despite oxygen therapy may require endotracheal intubation and PPV. An arterial blood sample and a blood gas analyzer are required to obtain a PaO2 measurement. Placement of an arterial catheter is beneficial in serial monitoring of PaO2. PPV is also indicated if the arterial partial pressure of CO2 (PaCO2) is greater than 60mmHg. A venous sample is acceptable in measuring CO2 levels (PvCO2 for venous) and is typically within 5mmHg from arterial values. Some patients may have a positional “preference” in their ability to oxygenate and ventilate, with sternal recumbency usually being most beneficial.

Medical management of the cause of pulmonary edema is warranted in conjunction with respiratory support. Diuretics are administered to reduce pulmonary capillary pressure and reduce preload through reduction of blood volume. Furosemide is a commonly used diuretic due to its rapid onset. In addition to its diuretic effect, furosemide may have further beneficial effects of pulmonary vasodilation and bronchodilation. Hemoconcentration resulting from reduced intravascular volume increases the plasma colloid osmotic pressure, helping to remove fluid from the alveoli. Nitroprusside and glycerol trinitrate are vasodilators that may be used as an additional method in reducing hydrostatic pressure. Bronchodilators such as terbutaline may also be used, and fluid therapy restricted. Chances of resolution depend heavily on the cause, and treatment for the patient’s specific underlying disease is required.

Pleural space disease
When the pleural space which normally serves to create negative intrathoracic pressure during breathing is filled with material which normally do not exist, normal breathing is compromised. The material may be various types of fluid, air, or even organs. The pleural space being occupied by these abnormal substances will cause the lungs to collapse and prevent adequate inflation, leading to a decrease in tidal volume, total vital capacity, and functional residual capacity. The lung volumes lead to hypoventilation, which can result in hypoxemia and hypercapnia.

Accumulation of fluid in the pleural space is called pleural effusion and can be of various type. Hydrothorax, or accumulation of transudate can be a result of reduced plasma colloid osmotic pressure, increased hydrostatic pressure, increased vascular permeability,
or neoplasia. Transudate is defined as effusion containing TP < 2.5g/dl and total nucleated cell count (TNCC) < 1500/µl. Effusion with TP between 2.5 and 7.5 g/dl and TNCC between 1000-7000/µl is considered to be modified transudate. Effusion with TP > 3.0g/dl and TNCC > 7000/µl is defined as exudate.

Feline infectious peritonitis (FIP), caused by a coronavirus can cause exudative or modified transudate effusion with yellow to straw-colored, viscous fluid with a high protein but low TNCC. Pyothorax is an accumulation of purulent exudate in the pleural space. Causes include bacterial infection due to migrating inhaled foreign objects, penetrating trauma to the chest wall, pneumonia, migrating plant material, and iatrogenic causes. Patients with pyothorax are typically treated with supportive care, antimicrobial therapy, and chest tube placement for intermittent lavaging with physiologic saline. In some cases, surgical exploration of the chest cavity to remove the source of the infection may be chosen. Accumulation of pink or white, milky, chylous effusion is termed chylothorax. The opaqueness is a result of a high triglyceride concentration. Potential causes include cardiomyopathy, congestive heart failure, pericardial disease, thoracic duct obstruction or rupture, lymphosarcoma, thymoma, and lung lobe torsion. Patients with chylothorax are typically managed by removal of the effusion through thoracocentesis. Hemothorax can result from coagulopathy, trauma, neoplasia, lung lobe torsion, pulmonary thromboembolism, and thymic hemorrhage. Iatrogenic causes are also possible, from procedures such as thoracocentesis, thoracostomy, and intrathoracic biopsy.

An open pneumothorax can result from penetrating thoracic trauma. Closed pneumothorax can occur due to damaged lung parenchyma, trachea, airway, esophagus, mediastinum, or diaphragm. Traumatic pneumothorax is the most common type of pneumothorax, caused by blunt force trauma such as automobile accidents or falling from heights. Gradual accumulation of air and pressure due to the lesion acting as a one-way valve results in a tension pneumothorax. A tension pneumothorax is life-threatening; increased intrathoracic pressure causes cardiovascular depression through reduction of venous return, leading to shock. Immediate thoracocentesis, and in persistent tension pneumothorax, a thoracostomy tube may be placed to continuously evacuate air out of the chest cavity. This is accomplished with a continuous suction device, or a one way valve. The patient is monitored for subsequent occurrences of dyspnea, indicating a return of pneumothorax. If a closed pneumothorax does not resolve in 3-5 days, surgical exploratory is warranted.

Diaphragmatic hernia may occur due to trauma or could be congenital. The degree of dyspnea varies depending on the degree of herniation, presence of concurrent pleural effusion, and presence of thoracic injuries. Surgical treatment is warranted when diaphragmatic hernias are seen, and should be performed immediately if any organ torsion or strangulation is suspected. Prognosis is good in patients receiving surgical intervention within 24 hours.

Pneumonia is the inflammation of the pulmonary parenchyma caused typically by an infectious agent which enters the airway. Aspiration pneumonia is caused by inhalation of contaminated material leading to an infection. Patients with aspiration pneumonia may present in respiratory distress and exhibit signs like coughing, weakness and collapse, pyrexia, cyanosis, and purulent nasal discharge. Lung sounds will be loud, and crackles may be heard. Abnormal sounds more often than not can be localized in the cranioventral areas. Treatment will consist of antimicrobial therapy, oxygen therapy and mechanical ventilation if necessary. Nursing interventions such a nebulization and coupaging may be instituted, though human evidence relating to a faster recovery from pneumonia has not been seen.

Patient monitoring
During the treatment of patients with respiratory compromise, the patient should be closely monitored on three different aspects; oxygenation, ventilation (carbon dioxide elimination), and the degree of respiratory effort. In addition to the visible respiratory effort and auscultation, different instrumentation and blood analysis can give insight to the progression of the patient’s recovery.

Oxygenation
A physical sign seen in patients with severe hypoxemia is cyanosis, or a blue color to the mucous membranes. Cyanosis becomes apparent when there is more than 5 g/dL of deoxyhemoglobin present in the blood. An average hemoglobin level in dogs is approximately 13-17 g/dL, and in cats is approximately 10-14 g/dL. This means the oxygen saturation of hemoglobin will be a significantly decreased level on average of 61-70% for dogs and 50-64% for a cat before cyanosis is seen. Patients presenting with cyanosis is severely compromised in their DO2 and requires immediate attention.

Oxygenation can be better gauged through measurement of PaO2, serving as an indicator of pulmonary function measured through arterial blood gas analysis. A patient with normal respiratory function breathing room air will have a PaO2 of 80-100mmHg. PaO2 of less than 80mmHg qualifies as hypoxemia, and less than 60mmHg is considered severe hypoxemia.

Pulse oximetry allows non-invasive measurements of the percentage of oxygenated functional hemoglobin in the arterial bloodstream. The saturation of oxygen measured by pulse oximetry (SpO2) closely reflects SaO2 and can be used to estimate the PaO2 level. The oxygen-hemoglobin dissociation curve expresses the relationship between SaO2 and PaO2. A SaO2 of 95-98% corresponds to a PaO2 of 80-100mmHg. A SaO2 below 90% indicates a PaO2 of less than 60mmHg. Pulse oximetry has its limitations, including false reading in the presence of significant levels of dysfunctional hemoglobin species (methemoglobin, carboxyhemoglobin), inconsistent readings with movement, poor perfusion, anemia, and pigmented skin. Interpretation of
oxygenation and pulmonary function can performed by calculated values called the PaO2:FiO2 Ratio (PF ratio) and alveolar-arterial (A-a) gradient.

**Carbon dioxide elimination**

CO2 is a metabolic byproduct of energy production. The body normally maintains control of CO2 levels to control the pH level of the body. An accumulation of CO2 causes an increase in levels of carbonic acid, increasing the levels of dissociated hydrogen ions, resulting in an acidic environment. A reduction in CO2 level will lead to a decrease in hydrogen ions, leading to a more basic environment. This effect is called respiratory acidosis and respiratory alkalosis, respectively.

The amount of CO2 eliminated by the body depends on the movement of air in and out of the alveoli to perform gas exchange, or ventilation. Room air contains about 0.04% CO2 (0.3mmHg), and the replacement alveolar gas with fresh room air will promote diffusion of CO2 out of the blood stream into the alveoli, which in turn gets expired out of the lungs and airway. A normal CO2 levels within the blood is approximately 35-45mmHg in dogs, and 30-40mmHg in cats, and can be measured by blood gas analysis (PaCO2 if arterial or PvCO2 if venous). The difference in PCO2 in the pulmonary capillaries and alveoli create a pressure gradient required for gas exchange (high to low; high in the capillary, low in the alveoli).

PCO2 is largely influenced by the amount of air that can be moved in and out of the alveoli, or alveolar ventilation. Reduced ventilation leading to high PCO2 is called hypoventilation (>45mmHg) and occurs in cases of respiratory depression (suppression of respiration due to drugs, neuromuscular disease, CNS disease), inability to expand the lungs (pleural space disease, compromise to chest walls), or increased resistance to breathing (narrowed airway). Hyperventilation and subsequent low PaCO2 can be seen in patients with increased RR due to anemia and hypoxia. In metabolic acidosis, compensatory increase in respiratory effort and hyperventilation is often seen, countering the metabolic acidosis effect with respiratory alkalosis. This occurs because the presence of hydrogen ions will stimulate the respiratory center of the brain to increase respiratory efforts.

The PaCO2 can be estimated by measurement of End-tidal CO2 (ETCO2). The CO2 content in the gas present at the probe at the end of expiration is measured to obtain this value. The ETCO2 in normal cardiovascular and respiratory situation, is within 5mmHg of the PaCO2. The ETCO2 is most easily measured when an endotracheal tube is placed in a patient (anesthetic procedure or mechanically ventilated patients, for example). There are nasal tubes and masks available allowing for less invasive ETCO2 measurement.
Vascular access is a critical element in providing treatment for patients presenting in an emergency and requiring continued care during hospitalization. Fluid and blood product infusions, drug administration, and blood sampling are a few key functions different methods in vascular access can serve. Placement of short term peripheral catheters are commonplace in veterinary care even outside of emergency and critical care. There are additional methods in providing vascular access such as central venous catheter placement, intraosseous catheterization, and arterial catheterization, each presenting their own advantages and disadvantages while serving their purpose.

**Peripheral venous catheters**

Peripheral catheterization is commonly performed in everyday veterinary practice, whether it is to administer IV fluids or an avenue for IV injections used for induction of anesthesia. Any catheter that is placed in the periphery that is too short to reach a central vessel is considered a peripheral catheter. Some of the major advantages to using a peripheral catheter includes the ease of placement, minimal discomfort to the patient, and the inexpensive nature of the catheters themselves. In addition, they can be placed quite rapidly with relatively minimal restraint, reducing the stress on the patient. In comparison to central venous catheters, peripheral catheters are less likely to cause complications such as hemorraging, thrombosis, and catheter induced infections.

Commonly used location of insertion are the cephalic vein, saphenous vein, and more rarely, pedal, auricular, and jugular veins. There is a common misunderstanding that a jugular catheter is by default a central line, though this is not necessarily true since whether a catheter is considered central or not is based on the position of the tip of the catheter. The cephalic vein is catheterized with ease in most patients, as the vein is easily visualized and remains straight for the length of the limb. There are, of course patients with short limbs making catheterization difficult, but this is likely true for all other sites available on such a patient. The saphenous vein can be an alternative catheterization location, though catheters placed in this location are more prone to occlusion through patient positioning. Pedal veins are viable options for larger dogs, though the degree of discomfort seems to be greater. Auricular veins can be more accessible for patients with short limbs, though stabilizing the catheter after placement is often a challenge.

Peripheral catheters are most often placed percutaneously with the vein visualized and/or palpated for the location. In cases of the skin seemingly too tough for dry or smooth insertion, or if fraying of the catheter has occurred, creating a relief hole with a separate needle and placing the catheter through the hole is appropriate. In some emergency situations, the vein may not be visible or palpable, in which case a cutdown can be opted to be performed. A venous cutdown involves creating an incision over the anatomic location of the vessel to visualize the vein directly. When a catheter is placed through cutdown, suturing of the incision as well as bandaging of the catheter site is necessary.

While a simple task that is routinely performed, there are several complications associated peripheral venous catheters. The first of which is phlebitis, or the inflammation of the vascular walls, causing inflammation of the vessel and surrounding tissues. A patient suffering from phlebitis will show redness, swelling, and pain on palpation. Phlebitis can be caused by mechanical damage if the catheter moves around, infection through the insertion site, or through irritation from injection of hyperosmolar fluids (high concentration dextrose, propylene glycol based drugs, etc). When evidence of phlebitis is seen, the catheter is recommended to be replaced in a different location. Thrombosis, or formation of clots, around the catheter through the hole is appropriate. In some emergency situations, the vein may not be visible or palpable, which impedes normal blood flow. Another common complication is subcutaneous fluid infiltration which occur due to the catheter being outside of the vein, leading to infusion of fluid under the skin. This could be from improper placement of the catheter to begin with, or the catheter pulling out of the vein with skin movement. Infection of the catheter insertion site can also occur. Lastly, catheter embolism, or fragmentation of the catheter and subsequent embolism can occur either from a faulty catheter, catheter damage from the stylette, or accidental cutting when the tape is being cut.

Because these complications can occur, regular maintenance of the catheter insertion site is warranted. A typical protocol calls for inspection of the insertion site every 24-48 hours, and whenever complications are suspected. Whenever a complication is suspected, the catheter should be replaced. Routine replacement of IV catheters after 72-96 hours has not been shown to be beneficial in preventing phlebitis or catheter induced blood stream infections, and replacement should be based on clinical signs of complications.

**Central venous catheters**

In contrast to peripheral venous catheters, central venous catheters have their most distal end terminate in a central vessel within the chest cavity (cranial or caudal vena cava). Central venous catheters can be inserted through the jugular vein, or be inserted peripherally through the saphenous vein (given you have the correct length catheter to reach the vena cava).
Central venous catheters are preferred over peripheral venous catheters when the patient requires infusion of highly osmolar fluid (high concentration dextrose, parenteral nutrition solution, for example), or infusion of multiple incompatible fluids are desired (a multi-lumen catheter will be used in this case). Additional function of central venous catheters include measurement of central venous pressure, sampling of central venous blood tests, serving as a sampling catheter, long term venous access, and prevention of contamination by substances that can commonly contaminate peripheral catheters (vomitus, diarrhea, drainage/discharge).

Various types of central venous catheters exist. They may be through-the-needle or over-the-needle, and single-lumen or multi-lumen. In general, over-the-needle catheters are preferred as there is no hardware left on the catheter assembly that requires maneuvering during the wrapping. Multi-lumen catheters are generally preferred over single-lumen catheters as it adds flexibility in fluid administration, giving the ability to infuse multiple solutions simultaneously without worries of incompatibility. If the effort and discomfort to the patient in placing a central venous catheter is already being made, giving ourselves the option of utilizing a multi-lumen catheter is a sound decision, as the patient is likely to require multiple infusions, and the cost between a single-lumen and multi-lumen catheter is small.

A commonly used technique in the placement of a central venous catheter is called the Seldinger Technique, or the guidewire technique. A typical protocol and steps involved in the placement of a central line is provided (Figure 1). The Seldinger technique is advantageous because it allows for a smaller catheter to serve as the introducing catheter, making placement in smaller patients or hypovolemic patients easier. In addition, the guidewire can be utilized when a catheter needs to be replaced by refeeding a sterile guidewire through the catheter, allowing a new catheter to be guided back into the vessel.

Similar complications to those in peripheral venous catheters can occur with central venous catheters. Due to its invasiveness, the catheter is more prone to blood stream infections, and an appropriate maintenance protocol (examination of insertion site once every 24-48 hours) is required.

**Intraosseous catheterization**

Intraosseous (IO) catheters, are needles or cannulas inserted into the bone marrow. The marrow vessels, unlike peripheral vessels, do not collapse due to the protective osseous coating it has as a part of the structure of the bone marrow. This makes the bone marrow an effective avenue for fluid and drug administration even during circulatory collapse. The effectiveness of blood components and crystalloids is equal to central or peripheral venous access. Some drugs and fluids may have a higher duration of action and peak effect when given IO during shock, though onset of peak effect may be slower without pressurized infusion.

When IV access is difficult or taking longer than a few minutes, an IO catheter should be considered, and included as a part of a standard fluid resuscitation protocol. An IO catheter is often placed in under a minute, at a very high success rate. Once an IO catheter is placed and fluids given to provide better vascular volume, another attempt at IV access should be more likely to be successful. Other situations which make IV access extremely difficult, such as peripheral edema, obesity, status epilepticus, and generalized burns may be other indications for IO access.

Blood values obtained from IO catheters are found to be comparable to central or peripheral venous samples, aside from potassium, glucose, and acid/base values.

IO catheter placement is contraindicated when fractures are present in the bone. This, along with previous IO catheter placement in the same bone, can lead to extravasation of infusions. In the case of previous IO catheterization, a larger size needle may be inserted in the same hole effectively plugging the existing catheterization site. Use of a completely different bone is also a solution. Placement of IO catheters through infected tissue, or in pneumatic bone in avian species should be avoided.

Any sterile needle long enough and sturdy enough to bore through the cortex and easily contained by the marrow of a specific patient is suitable. 18-22ga needles are often used for rodents, neonates, and even adult cats, small breed dogs. Bone biopsy needles are used in adult cats and dogs. IO catheter drills and catheter guns are contraptions to aid IO placement.

Considerations to make regarding the catheterization site include ease of access and placement, possibility of interference in procedures, and patient comfort (though not a major concern during an emergency situation). No site is shown to be better than the other in terms of efficacy of infusions. The viable sites are (1) Tibia – Medial surface of proximal tibia, tibial tuberosity (point slightly distally to avoid growth plate, which is a major longitudinal growth front for the tibia) (2)Femur – Trochanteric fossa (3) Humerus – Greater tubercle (4) Hips – Wing of ilium, ischium. Any angle of insertion is acceptable as long as the needle tip is within the marrow, though certain angles of approach may be easier for different sites.

Advancing the needle through subcutaneous tissue and periostuem may be painful. In stable patients, subcutaneous lidocaine block may be used to attempt to prevent pain. Many patients requiring immediate IV/IO access will likely have altered mentation preventing them from feeling or responding to pain. In these situations, the speed of placement will be prioritized over initial pain control.

The pressure build up when an infusion is started is known to cause pain in people. The same is assumed for non-human species. Removing a small amount of marrow, and infusing lidocaine over a minute or so will help alleviate this initial pain. Once again, this is likely not our primary concern in cases where seconds to minutes matter in the resuscitation of the patient.
Chances of complications with IO catheters are low, but present. Infection are rare, especially when the catheter is removed as soon as it is unnecessary. Extravasation of fluids, compartment syndrome (pressure buildup damaging muscle, nerve, blood vessels), bone fracture, and fat embolism (unknown clinical significance) are among other complications that might arise.

Figure 1: Central venous catheter placement

Central line placement

**Equipment needed**
- Guide-wire catheter kit
- Suture
- Needle Holder
- Thumb forceps
- Clippers
- Surgical scrub
- Sterile gloves
- Drape
- #11 blade or large needle
- Sterile gauze
- Heparinized saline flushes
- Bandage material
- X-ray request

**Preparation**
- Determine if sedation is required, and administer prior to gathering of supplies to allow time. If transdermal analgesic cream is to be used, clip and apply first.
- Clip area over the jugular, 2-4 inches by 2-4 inches depending on patient size.
- Pre-measure the distance from the intended insertion site to the 4th intercostal space.
- Aseptically prep the area in a circular, surgical scrub motion.

**Procedure**
1. Wash hands and don sterile gloves.
2. Drape the prepared area.
3. Place the over-the-needle catheter included in the kit into the vein, oriented towards the heart. (Note: in some cases, you will not see a flash in your stylet hub, in which case check with syringe)
4. Insert the guidewire into the catheter and feed forward to the premeasured length.
5. Remove the catheter over the guidewire, taking care to not contaminate the guidewire.
6. Place the plastic dilator over the guidewire, and insert into the vessel through the skin. Tenting the skin, spinning the dilator, and creating a relief hole will help facilitate the dilation process.
7. Remove the dilator over the guidewire after leaving in the vessel for a few seconds.
8. Place the catheter over the guidewire and feed into the dilated opening into the vessel. Be sure to observe the guidewire exiting out the catheter port and hold it in place to prevent loss of the wire.
9. Once the catheter is fed to the pre-measured length, remove the guidewire.
10. Aspirate port with a syringe to confirm blood flow, then flush with heparinized saline. Attach needleless ports. Repeat for each port to confirm patency. (Note: Aspirating first prevents injection of air).
11. Secure the catheter with suture using the suture wing(s).
12. Cover the insertion site with non-stick gauze pad or adhesive dressing, and bandage.

**Placement procedures**

After the site is chosen:
1. The hair on the potential insertion site should be clipped and the area disinfected. Be mindful of the balance of the time you have versus the time it takes for a thorough scrubbing.
2. A stab incision can be made to allow for easier access to the insertion site.
3. Firm and steady pressure should be applied to the bone with an alternating clockwise and counterclockwise rotation (being careful not to slip on the bone surface).
4. Once the initial hole is started with the needle, the pressure can be increased as the same motion is made, to bore faster through the cortex.
5. When the needle pushes through the cortex, a sudden loss of resistance will be felt.
6. Proper placement of the catheter can be confirmed in a few ways:
   a. Firmness of the catheter insertion – There will be very little wiggle in the needle when properly inserted through cortex.
   b. Movement with the limb – Catheters in the cortex will make the same movement as the bone it is inserted in. If the catheter moves independently of the bone, or vice versa, the catheter is likely placed improperly.
   c. Aspiration – Bone marrow should be able to be aspirated in a properly placed catheter, though this is less likely to occur in an older animal.
d. Saline Infusion – If placed correctly, saline should be able to be pushed without much resistance. If a large backpressure is encountered, the needle is likely to be in the cortex, or pushed against it. Rotation of the needle 90-180 degrees may alleviate this if the needle opening is simply pushed up against cortex. If the infusion results in subcutaneous elevation of tissue (subcutaneous “bleb”), the catheter is improperly placed.

7. Once proper placement is confirmed, the catheter is stabilized via tape or suture on tape wings. Some IO catheters will come with suture wings.

8. Proper catheter care is required with bandages applied when possible. A properly maintained IO catheter may be kept for 72 hours with minimal chance of complications. Typically, IO catheters are replaced by IV catheters within 2-12 hours after initial fluid resuscitation.
Despite the increase in use of blood products and transfusion therapy becoming more commonplace, there are many questions surrounding their use. Many of these questions are being answered by a combination of emerging veterinary and human evidence further clarifying already known concepts, with others confirming long time suspicions and teaching us something new.

Can RBCs be given through an infusion pump?
Whether there is an optimal method of red blood cell transfusion administration has been a point of discussion. Recommendations from infusion pump manufacturers are variable in ability to transfuse blood products. The criteria used to make these recommendations are also uncertain without an established standard or regulatory body in validating these claims. An ideal method of administration should allow one to administer the red cells at a consistent and specified rate while not affecting the integrity, survival time, and oxygen carrying capacity of the red blood cells.

Studies evaluating the effect of various administration methods on the integrity of blood cells exist. The majority of these studies investigate the in vitro effect of infusion pumps, measuring the degree of free RBC content (free hemoglobin, potassium, lactate dehydrogenase, bilirubin) and osmotic fragility. The results vary from observing significant increases to insignificant increase in values, while transfusions with red cells with longer storage time resulting in a larger increase of hemolysis markers than those with shorter storage times. The variability in results, in addition to the anecdotal evidence of patients benefiting from RBC transfusions administered with infusion pumps are a cause for varying opinions.

A study assessing in vivo survival time of RBCs infused with various infusion methods, compared the use of gravity flow, volumetric peristaltic pump, and syringe pump in autologous transfusions in dogs. Blood was collected from 9 healthy dogs, washed, and separated into 3 portions labeled with different densities of biotin. These labeled red cells were transfused through either gravity flow with a 170-260 µm filter, volumetric peristaltic infusion pump with a 170-260 µm filter, or a syringe infusion pump with an 18 µm aggregate filter at 2mL/kg/hr. Blood was sampled from test subjects at day 1, and every 7 days until day 49, measuring the proportion of red cells with biotin labels through flow cytometry. Additional in vitro testing was conducted, measuring plasma hemoglobin and osmotic fragility testing.

Labeled RBCs infused through gravity flow, volumetric pump, and syringe pump were detectable in 100% (8/8), 50% (4/8), and 14.3% (1/7) samples, respectively post-transfusion. The quantity and half-life between RBCs infused by gravity flow and volumetric pump that were detectable (4/8) were not different. The RBCs infused via syringe pump detected at 24 hours post transfusion was no longer detectable at 7 days, indicating complete removal of those cells from circulation sometime between 24 hours and 7 days post transfusion. There were no differences seen in in vitro values examined.

The study concluded that delivery of RBCs with a syringe pump and microaggregate filter is associated with significant decrease in in vivo survival time. Volumetric pump delivery was associated with a 50% probability of loss of transfused RBCs within the first 24 hours, and gravity flow allowed for highest chance of RBC survival. The reason behind this difference is speculated to be the mechanical shear damage to the RBC membranes when transfused through the microaggregate filter, causing preferential removal of damaged cells upon entry into the circulation and exposure to the mononuclear phagocytic system. Though unconfirmed, there is a potential for microclots to have formed in the blood during resuspension in sub-room temperature plasma, which placed a higher degree of shearing stress on the RBCs going through the filter, causing this effect. Early denaturation and oxidation of hemoglobin due to the mechanical stress induced by syringe pump and volumetric pump methods, leading to IgG binding to the red cell surface and removal from circulation, is another possible cause for early removal.

There were other limitations to this study such as the reduced half-life of transfused RBCs when compared to our current knowledge of dog erythrocyte lifespan (43 days in study vs 104-110d) attributed to the insult to RBCs during the biotinylation and processing. The use of biotin for RBC detection itself is not without worries of immunologic removal through anti-biotin antibody production, though previous studies have observed no development of such antibodies. Small sample sizes limiting the power of the results is a common limitation in the veterinary field, and this study is no exception. The results are most relevant to exact methods used in the study, and we can only make speculations on alternate setups to remove the use of microaggregate filters with the syringe pump (use of an in-line pediatric 170-260 µm filter or extraction of blood through a 170-260 µm filter administration set into a syringe, for example).

The authors of the study recommended against using a syringe pump with 18 µm aggregate filters in the light of the results of their study, though considering the limitations, drastic changes to clinical protocols was not stated to be necessary. The current best practice considering this evidence would be to administer blood products via gravity flow for larger volume, higher flow rate transfusions as
Transfusions such as in felines. A similar study performed with feline blood stated their observation of RBC survival time being unaffected by the syringe pump method.

**PRBC has an expiration date of 42 days(?)**

Current practices in blood banking involve the usage of APS and additive nutrient solution which are labeled for 42 days of storage. More recent evidence gathered over the past decade indicates stored red blood cells to have impaired RBC survival, reduced efficacy as an oxygen carrier, and even incite adverse effects in the recipient causing mortality and morbidity. These changes are seen as early as 7 to 14 days into storage, and involve a collection of biochemistry, biomechanical, and oxidative changes to the RBC and storage solution, all collectively referred to as “storage lesions”.

Mature RBCs lack mitochondria and rely on glycolysis for ATP production, leading to a lowered pH. ATP production is reduced by the acidic environment, combined with depletion, leads to decreased RBC membrane integrity. Lowered pH also affects 2,3 diphosphoglycerate (2,3 DPG) level reducing hemoglobin’s effectiveness as oxygen carriers, though this effect is reversible and not significant in cats. Hemoglobin in longer stored RBC products contain free hemoglobin and microparticles that scavenge nitrous oxide (NO) upon transfusion and cause a vasoconstrictive effect impairing blood flow, stimulate coagulation, induce oxidative damage, and cause proinflammatory effects. Microparticles, which are vesicles that have budded off of cellular components, induce proinflammatory and procoagulant effects. Stored RBCs show morphologic changes to echinocytes and spherocytocytes leading to a loss of deformability and impairment in normal flow through capillaries. Oxidative damage leads to increased hemolysis and methemoglobin formation decreasing viable RBC count and oxygen carrying capacity.

There are many complicated mechanisms in play during RBC storage. To summarize the effects, storage lesions can lead to impaired RBC survival, reduce the efficacy of RBCs as oxygen carriers, and induce adverse effects such as arrhythmias, thrombosis, systemic inflammation, transfusion-related acute lung injury (TRALI), acute respiratory distress syndrome (ARDS), hypotension, and multiple organ dysfunctions. These changes occur as early as 7-14 days into storage, making supplying our patients with safe transfusion products a realistic challenge. Clinical impact of storage lesions is a topic of ongoing investigation while blood banks strive to balance provision of fresher products and minimizing wasting.

**First transfusions are “free”**?

Compatibility testing for canine blood transfusions has traditionally been omitted in the interest of swift transfusions and financial considerations. This comes from the a widespread notion that the “first transfusions are free for dogs”; intended to state that canine RBC transfusions can be given without blood type matching (without typing the donor or recipient) or cross matching yet be performed without signs of immunologic complications, namely acute hemolytic transfusion reactions or anaphylaxis. This statement is made with the understanding that the most clinically significant dog erythrocyte antigen (DEA) is DEA 1, responsible for inciting acute hemolytic transfusion reactions when preexisting alloantibodies for the antigen is present. In 98% of the population, these antibodies are not present, so the first mismatched transfusion will only result in sensitization of the immune system to the antigen, leading to the development of antibodies over a course of approximately 4 days. This leads to a delayed hemolytic transfusion reaction, often asymptomatic as long as the patient has overcome the initial incident of anemia, or clinical symptoms of anemia as well as bilirubinemia and bilirubinuria may arise.

Given the asymptomatic or mild nature of clinical signs, many have accepted this reason to forgo compatibility testing. However, the sensitization will lead to an acute hemolytic transfusion reaction in subsequent mismatched transfusions, resulting in hemolysis of transfused cells and likeliness of anaphylaxis. By omitting compatibility testing, we run the risk of priming a patient for such reaction in the next transfusion which may be handled similarly if the patient’s transfusion status is not noticed. A medical team may be placed in a situation where the transfusion status of the patient may be unknown (pet brought in by pet sitter who thinks there had been no transfusions, or adopted dogs who “probably” has not had a transfusion). In the case a patient presents with risk of imminent death from anemia, this practice may be justified with the knowledge of the risk. Blood typing of all blood donors and stocking of DEA 1 negative blood is highly recommended for use in these situations to avoid sensitization of the patient to DEA 1. If there is any uncertainty in the transfusion history of the patient, cross matching is appropriate as erythrocyte antigens aside from DEA 1 exist with limited knowledge on consequences from patients sensitized for these miscellaneous antigens (some reports of AHTR exist).

Transfusions of canine RBCs without compatibility testing are not “free”, and certainly have the hidden costs of DHTTR and sensitization. Cats possess alloantibodies for the RBC antigens foreign to them (aside from the very rare type AB cats), leading to hemolytic transfusion reaction even with first exposure.
DEA 1 negative is the universal blood type?
The concept of “universal” blood type indicates a blood type that can be given to any member of the same species without expectation of an immunologic reaction related to blood type mismatches. Because DEA 1 is the one antigen we know most about and leads to AHTR when mismatched for the second time, blood from DEA 1 negative dogs can be given without sensitization of DEA 1 negative and DEA 1 positive recipients, and often is considered as “universal”. There are, however, other RBC antigens such as DEA 3 through 8, dal, and other less known antigens confirmed to exist which can lead to sensitization when mismatched transfusions occur. Therefore, a donor should be tested negative for every RBC antigen we are capable of testing in order to truly considering it “universal”. This creates a challenge as 98% of the canine population is positive for DEA 4, and a donor negative in DEA 4 is virtually impossible to find. Fortunately this is not a clinical issue since the recipient is likely DEA 4 positive as well, allowing the blood types to match. Another challenge lies in our current inability to routinely test for DEA other than 1, 4, and 7 through a reference lab due to a lack of testing anti-sera (and anything aside from DEA 1 not available as in-house kits), preventing complete typing of our donors and timely testing of our recipients. Given our knowledge of additional RBC antigens, we should consider DEA 1 negative, 4 positive, 7 negative blood type as the “least antigenic”, and type our donors for all DEAs we are capable of, given finances permit it. DEA 1 negative can be considered safe blood to use from anecdotal evidence as reports of hemolytic transfusion reactions are rare, and cross matches should detect incompatibility issues arising from repeated exposure to the less known erythrocyte antigens. Cats have no universal donors, though type AB cats may receive transfusions from both type A and B donors.

Are blood transfusions between different species possible?
Common knowledge dictates that blood product transfusions should be between members of the same species in order to prevent immunologic consequences. Despite this “common” fact, one may be surprised to hear an ongoing research to test interspecies transfusions, or xenotransfusions. Early experiments in blood transfusion in the 1600’s document a human patient receiving sheep blood, and showing no signs of reaction (at least on first exposure). Porcine red blood cells with modified antigens have been a topic of research in compatibility as human blood substitute. In the veterinary field, feline blood is consistently in short supply, especially for patients with the rare blood type of B. Type B cats can only be transfused with type B blood as introduction of a small volume of type A blood will result in an acute hemolytic reaction and anaphylaxis. In addition, even for type A patients, blood supply may be short causing delays or inability to obtain blood products in a timely manner as the patient suffers life-threatening anemia. In these situations, veterinarians have attempted to use canine blood as a source of blood as it is more readily available, and can easily tolerate the small volume donations.

There is limited amount of evidence available from a few studies conducted on canine to feline transfusions. The results of the studies concluded felines do not possess naturally occurring alloantibodies against canine erythrocytes. Compatibility testing methods such as slide-agglutination test and cross-matching only revealed agglutination on the minor-crossmatch. Of the total of 62 transfusions performed between the various studies, 5 cats showed signs of mild reactions, with tachypnea and pyrexia within 24 hours of the start of transfusion. Development of antibodies against canine RBCs were seen 4 to 7 days after the transfusion, indicating the transfusion led to sensitization of the immune system to the foreign antigens. Because of this, the life span of the transfused RBCs was approximately 4 days due to delayed hemolytic transfusion reactions while feline to feline transfusions allow RBCs to last 30 days. Subsequent transfusions resulted in anaphylaxis and were fatal in 66% of documented cases.

While transfusion of dog blood to a feline patient is not the best solution to supplementing oxygen carrying capacity, it may be justifiable when faced with imminent death of the feline patient and without blood. A responsible medical team would discourage dog to cat transfusion and consider the method for situations where the patient 1) has no source of compatible cat blood (Type B cat with no stocked blood, donor, or nearby hospital with stock, for example) or hemoglobin based oxygen carrier solutions, 2) is imminently going to pass away without a transfusion or compatible blood will not be obtained soon enough (truly dying animal), 3) is expected to benefit from a short term oxygen carrying capacity gain, and 4) the owner understands risks and consequences. The method of xenotransfusion should not become a common practice and effort for practices to maintain a good source of cat blood should always be pursued without considering canine blood as “backup”.

Premedication reduce chances of reactions?
Premedication, or administration of antihistamines, glucocorticoids, or antipyretics in anticipation of immunologic complications to counter histamine and inflammatory mediators and suppress the effects, have been a traditional practice in transfusion medicine. While there is no veterinary evidence available addressing the efficacy of this practice, there are a number of human studies observing no difference in incidence of type I hypersensitivity reactions (allergic reaction) or febrile non-hemolytic transfusion reactions (FNHTR). Some clinicians reason that administration of premedication potentially masks early symptoms of immunologic complications delaying required interventions for treatment, advocating against it. Evaluation of the difference in severity between recipients with premedication or without premedication has not been performed, and remains a question whether this reasoning is valid. Human evidence is unfortunately not always directly translatable into veterinary practice, though expectations of similar
physiological mechanisms exist. Studies evaluating effects of premedication and efficacy in prevention of hemolytic transfusion reactions are not apparently available, and the theoretical benefit is no justification for forgoing proper compatibility testing.

Is warming of blood products necessary?
Warming of blood products in the interest of prevention hypothermia in the recipient is a consideration during blood product administration. Concerns for hemolysis of erythrocytes when warming during transfusion exist, and studies point towards little to no difference in markers for hemolysis in vitro when blood is warmed to typical body temperature. However, at non-emergent administration rates, blood reaching the patient through the line placed in a room temperature environment is easily at room temperature upon reaching the patient, and will not contribute to a significant decrease in body temperature. In the case of rapid transfusions of large volumes into small patients, warming of the blood may be indicated with care taken to be evenly warmed to 35-37°C and not exceed 42°C close to the patient to minimize loss of heat. Hypothermia is also a documented complications related to massive transfusions. Aside from these situations, in many cases warming effort directed at the patient is most effective in treating hypothermia without causing risk of damage to the RBCs.

Is plasma indicated for use in hypoproteinemia? Parvoviral enteritis?
Plasma contains many proteins of interest, namely hemostatic proteins, albumin, and immunoglobulins. Hypoproteinemia, specifically hypoalbuminemia, occurs in many critically ill patients with protein-losing disorders including protein-losing enteropathies, protein-losing nephropathies, liver failure, trauma, burn wounds, etc. This leads to a loss of intravascular colloid osmotic pressure (COP), and subsequent consequences. Administration of plasma products (fresh frozen plasma, frozen plasma, or cryosupernatant) have been used as a method in supplementing albumin for COP. However, the amount of plasma required to raise the patient’s albumin level by 1g/dL is approximately 40-50mL/kg. This is equivalent to 1.1L of plasma (9.5 units) for a 50# patient. The amount of plasma required to make a significant difference in the measurable level of albumin is both cost prohibitive and pose a large immunologic risk to the patient. Whether increasing the albumin level to a normal value (>2g/dL) will lead to increased chances of a positive outcome is still unclear, and difficult to advocate.

Similar concepts can be applied to the usage of plasma products derived from survivors of parvovirus infection. Clinicians have theorized that transfusion of plasma containing antibodies against canine parvovirus (CPV) will aid in recovery from CPV infections. A study evaluating use of a single dose of plasma containing CPV antibodies in its efficacy versus saline placebo saw no significant difference in reducing clinical signs, viremia, or speeding recovery. The volume used in this study (12mL) may be a limitation to the efficacy of the compared treatment, though the amount of plasma required for an adequate dosage of antibodies is unknown, and is likely to be at similar or higher levels of dosage for albumin supplementation. Thus, same concerns prevent use of plasma in this manner.
Shock has Two Faces: 
The Keys to Perfusion
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Shock has two faces. Shock is defined as an inadequate production of cellular energy, very commonly brought on through forms of circulatory failure. Hypovolemic (inadequate circulating blood volume), cardiogenic (inability for heart to create forward flow), distributive (loss of systemic vascular resistance), and obstructive (obstruction of large vessels, sometimes not considered its own category) shock describe one aspect of inadequate delivery of oxygen (DO₂) leading to inadequate production of cellular energy. Shock despite proper circulatory function arises from metabolic dysfunction due to inadequate substrate supply or dysfunctional metabolic mechanisms. Oxygen is a very important component in carrying out aerobic metabolism, a vastly more efficient and sustainable method of energy production than anaerobic metabolism which takes place with the absence of oxygen. Lack of oxygen being delivered to tissues from inadequate oxygen content in arterial blood lead to what is known as hypoxemic shock, and may occur from varying reasons.

Oxygen in energy production
The importance of maintaining adequate DO₂ lies in the difference of the amount of adenosine triphosphate (ATP) produced in the presence and absence of oxygen. ATP is considered the “currency of cellular energy”, providing energy for cellular processes required to maintain life as phosphate groups are cleaved off resulting energy release and formation of adenosine diphosphate (ADP) or adenosine monophosphate (AMP). ATP is involved in cellular signaling, DNA and RNA synthesis, muscle contraction, cytoskeletal maintenance, active transporting, and many other cellular functions. There is a finite amount of ATP available within a body, and a constant recycling of ADP and AMP into ATP is required to keep up with energy demands. In the presence of oxygen, 38 ATP molecules are generated from metabolism of a single glucose molecule undergoing oxidative phosphorylation occurring in the mitochondria. In contrast, a single glucose molecule yields two ATP molecules through anaerobic metabolism. The presence of oxygen is imperative in efficient energy generation.

Provided there is adequate functional hemoglobin levels and normal respiratory function, DO₂ is dependent on the ability to circulate the oxygen containing blood to tissues requiring oxygen, called perfusion. The mathematical expression of DO₂ is: DO₂ = CaO₂ x CO. Oxygen contained within blood exists in two forms; dissolved in the plasma and bound to hemoglobin. The amount of oxygen dissolved in plasma depends on the partial pressure of oxygen (PaO₂), with 1 mmHg creating enough tension to result in 0.0031mL of dissolved O₂ per dL of plasma. Each gram of hemoglobin is able to theoretically carry 1.39mL of O₂ when fully bound with oxygen, making up a significant portion of oxygen content of blood. In reality, there are portions of dysfunctional hemoglobin lowering this to approximately 1.34mL. In addition not every hemoglobin molecule will be fully bound to oxygen in every situation (SaO₂, or arterial oxyhemoglobin saturation) adding some variability. With all of these considerations in mind, the resultant formula to quantify DO₂ is the following, expressing the impact lowered hemoglobin concentration and saturation of the hemoglobin will have on overall delivery of oxygen: DO₂ = [(1.34 x Hgb x SaO₂) + (0.0031 x PaO₂)] x CO.

In animals without disease, DO₂ is significantly above oxygen consumption (VO₂), supplying a very comfortable buffer of available oxygen for energy production. This buffer allows for sudden changes in oxygen demand through changes in cellular metabolic rate or reduction in CaO₂. When DO₂ is significantly compromised (termed critical oxygen delivery), tissue hypoxia results and increased lactate levels and lowered pH are seen. The oxygen extraction ratio can also be used to express the level of oxygen consumed in relation to DO₂: O₂ER = VO₂/DO₂. Higher oxygen consumption or lower DO₂ leads to a higher ratio. The normal O₂ER value is approximately 0.2, though different organ systems have varying O₂ER (normal O₂ER of the heart is 0.6, making it more sensitive to hypoxemia). A normal DO₂, VO₂, and O₂ER in dogs were observed to be 790ml/min/m², 164ml/min/m², and 0.205, respectively in one study. Another couple of studies cite a normal DO₂ of 20-25ml/kg/min and observed critical oxygen delivery levels of 8-11ml/kg/min regardless of the cause (anemia, hypoxemia, and cardiac tamponade). A patient is said to be in hypoxemic shock when Hgb, SaO₂, or PaO₂ levels are low enough for DO₂ to reach this critical oxygen delivery level. In clinical settings, measurement of specific values such as CO and VO₂ (though can be estimated) are rather difficult, and we utilize this concept in determining when a patient is suspected to be in hypoxemic shock rather than making direct comparisons.

Tissue perfusion
The maintenance of normal blood pressure and tissue perfusion depends on adequate CO, and systemic vascular resistance (SVR). The most common form of reduced DO₂ and shock occurs secondary to reduction in CO or SVR. CO is can be reduced through a loss of intravascular volume, leading to hypovolemic shock. Hypovolemic shock can be caused by many situations leading to hypovolemia, such as internal or external hemorrhaging, fluid loss through vomiting, diarrhea, polyuria, exposed subcutaneous surfaces (burns, bit wounds) and/or reduced water intake. A loss in circulating blood volume leads to a diminished venous return and preload to the heart,
Reducing the stroke volume (SV). A significant degree of reduced CO due to decreased SV is compensated for through an increase in heart rate (CO = SV x HR). SV itself is improved through increased contractility, or a more forceful contraction of the heart to eject a larger volume of blood. Reduced blood flow to the kidneys stimulates the renin-angiotensin-aldosterone system, increasing production of aldosterone leading to sodium retention, increasing plasma osmolarity and encouraging shifting of fluid to the intravascular compartment. Increased antidiuretic hormone (vasopressin) also promotes water retention, reducing urinary fluid loss. While vasoconstriction does not directly add to intravascular volume, its occurrence increases systemic vascular resistance, improving blood pressure and circulation of the reduce blood volume.

Patients faced with hypovolemia initially show signs of compensatory shock, involving tachycardia, normal prolonged capillary refill time (CRT), normal to pale mucous membranes, tachypnea, and cool extremities. Pulse quality and blood pressure may be mostly normal, and subtle depression in mentation may be seen. As intravascular volume continues to be lost, compensatory mechanisms will be unable to adequately maintain proper perfusion, and later signs of shock such as pale mucous membrane, prolonged CRT, poor pulse quality, depressed mentation, and hypotension will be seen. Uncorrected poor perfusion will lead to organ ischemia, leading to organ failure and death.

CO can be significantly affected by cardiac dysfunction as well. Congestive heart failure from cardiomyopathy can reduce contractility of the heart or reduce end-diastolic volume, decreasing SV and subsequently, CO. Cardiac arrhythmias may lead to improper filling, ejection, and effectiveness of the heart to create CO. Cardiac tamponade, occurring when effusion fills the pericardial space creating external pressure on the myocardium significant SV. Certain drugs may have cardiovascular depressant effects or cause myocardial conduction defects, leading to reduced CO. Any cardiogenic cause leading to reduced CO and resultant shock is called cardiogenic shock.

Distributive shock is another form of shock characterized by an inappropriate distribution of blood flow and volume. One example can be considered vasodilatory shock, resulting in profound vasodilation causing “relative hypovolemia” and a reduction in SVR, leading to reduced BP and poor perfusion. Systemic inflammatory response syndrome (SIRS) and septic shock (SIRS due to an infectious cause) involves vasodilation caused by cytokine and other inflammatory mediator secretion leading to a hyperdynamic phase involving hyperemic mucous membranes, bounding pulses, fever, and tachycardia. As the hypoperfused state is allowed to persist, myocardial damage leads to reduced cardiac output, and clinical changes to the patient to more classic signs of shock. Tachycardia, pale mucous membranes, prolonged CRT, cold extremities, poor pulse quality, and depressed mentation will be signs of significantly impaired perfusion.

Patients suffering from gastric dilatation-volvulus (GDV) will have a distended stomach compressing the intra-abdominal vessels (caudal vena cava, portal veins, and splanchnic vessels), impeding venous return to the heart leading to a reduced CO. This is considered obstructive shock by many (while many others consider it a form of distributive shock), where major blood vessels are occluded or carry reduced blood flow contributing to poor CO. The cause of shock in GDV is actually multi-faceted, since the occlusion of major vessels leads to portal hypertension and splanchnic pooling, leading to effusion of intravascular fluid into the abdominal cavity and interstitium, contributing to hypovolemia. Additional fluid loss may also occur due to vascular injury to gastric vessels as it is stretched, and repeated vomiting. Many disease processes involve different causes of shock occurring in varying in degrees, leading to the cumulative effect of reduced CO and DO₂.

Monitoring
The effectiveness of treatment can be determined through physical parameters as well as laboratory values. Physical perfusion parameters consist of mentation, heart rate, pulse quality, mucous membrane color, CRT, core to extremity temperature gradient. A patient in shock will have dulled mentation, increased heart rate (bradycardic in decompensated shock), poor pulse quality, pale mucous membrane (hyperemic if early vasodilatory shock), prolonged CRT, and a significant difference in core vs extremity temperature. These physical parameters should be monitored as shock is treated to ensure signs of poor perfusion are alleviated as therapy is continued. Except during compensatory shock, hypotension would be present and thus blood pressure should be monitored for changes. If hypotensive, initiation of therapy should be aimed to increase to a normal ranges (MAP 70-120mmHg). Blood pressure may be measured indirectly via Doppler, oscillometric monitors, or directly through an arterial catheter and pressure transducer setup.

Hypoperfusion of tissues and inadequate oxygen delivery results in anaerobic respiration. Anaerobic respiration is performed in hypoxic situations, leading to hyperlactatemia, and resultant metabolic acidosis. Normal lactate level dogs and cats is 0.5-2.0 mmol/L. Elevated lactate measurement indicates significant lactate production overwhelming the liver’s metabolic clearance rate. Serial lactate measurements as fluid resuscitation is performed will allow monitoring of changes in the lactate level. A swift decrease in lactate level during fluid resuscitation serves as a positive prognostic indicator in patients with shock.

Other assessment tools such as central venous pressure may help guide fluid therapy and monitor fluid balance in a patient as fluid therapy is continued. Mixed venous oxygen saturation (SvO₂), or oxygen saturation of hemoglobin at the pulmonary artery (after maximal oxygen extraction), will be decreased when DO₂ is decreased. Since pulmonary arterial catheters are not commonly placed in veterinary medicine, central venous oxygen saturation (ScvO₂) can be used as an indicator for SvO₂, as the values parallel each other

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closely, and a reduced ScvO₂ typically correlates to a reduced SvO₂. Urine output serves as an indicator for adequate renal perfusion, and will be greater than 1ml/kg/hr when blood flow is adequate. Urinary catheters are placed in critical care patients for the purpose of urine output monitoring. In the absence of renal disease, urine specific gravity may give clues as to adequate fluid infusion rate as well. Cardiac output itself can be monitored through advanced modalities such as thermodilution or lithium measurement, though not readily available in veterinary medicine.

**Therapy**

Treatment for shock will vary depending on the underlying cause. Hemorrhaging may require surgical intervention, or treatment of coagulopathies. Treatment of sepsis is a very intensive process including early antimicrobial administration. Cardiomyopathies may require various anti-arrhythmics, anti-hypertensives, or inotropics. These are only a few examples. Regardless of the underlying cause, there is a general strategy that can be applied to treating patients in shock which is aimed at reversing the restoration of tissue perfusion and preventing progression of shock while the underlying causes are treated.

Obtaining vascular access is one of the first steps in restoring cardiovascular stability in states of shock. A larger diameter, shorter catheter will cause the least amount of resistance for fluid boluses to be administered and recommended. Many of these patients, however, will have a compromised cardiovascular system often making placement of intravenous catheters difficult. Placement of a jugular venous catheter or intraosseous catheters may be more readily possible compared to placement of peripheral venous catheters and should be considered very early in attempting vascular access.

The first line of therapy is isotonic crystalloid therapy in all forms of shock aside from shock arising from cardiomyopathies (adding intravascular volume in congestive heart failure will exacerbate the congestion). A IV crystalloid bolus dose of 20ml/kg may be given, and the patient re-evaluated for further need. Crystalloids are thought to remain in the intravascular space only for a short amount of time (25% remaining approximately 30 minutes after infusion), and may require re-dosing at this point. In the case of hemorrhaging, hypotensive resuscitation, keeping the MAP approximately 60mmHg, may be beneficial in preventing exacerbation of bleeding. Infusion of crystalloids is aimed at replacing lost intravascular volume, or adding intravascular volume to combat relative hypovolemia caused by vasodilation.

Synthetic colloids such as hetastarch and tetrastarch provide higher osmolarity than crystalloids, allowing better retention and even causing shifting of fluid into the intravascular space, increasing intravascular volume and better tissue perfusion. In human medicine, there was recently a warning issued regarding the use of hetastarch and it being linked to renal injury. While human kidneys and canine/feline kidneys seem different in terms of sensitivity to insult, hetastarch is now recommended to be used with caution. Natural colloids are available in the form of albumin contained in plasma or albumin concentrate. Plasma may be used to supplement albumin levels in hypoalbuminemia. Hypoalbuminemia may result due to protein losing enteropathy or nephropathy, septic peritonitis, trauma, burns, and any other pathologies causing protein loss. However, the dose required for this particular use is 20-25mL/kg to achieve an increase of 0.5g/dL in plasma albumin. For example, a 25kg patient with an albumin level of 1.0g/dL will require 1000-1250mL of plasma to regain a low normal plasma albumin level of 2.0g/dL. In addition, this is not taking into account ongoing loss from the patient’s pathology. Use of plasma in this manner will pose a higher transfusion related complication risk, be an inefficient use of plasma, and will be at a significant cost to the owners. Serum albumin concentrate is a better source of albumin.

Human serum albumin (HSA) has been used in canine patients with hypoalbuminemia. However, these infusions have a significant chance of an immunologic reaction as human albumin differs from canine albumin by 20% of its amino acid sequence. Previous sensitization to human albumin and subsequent acute hypersensitivity reactions are especially a concern when repeat doses are necessary. A study found presence of anti-albumin antibodies in dogs without prior exposure to human serum albumin, which was hypothesized to be from prior vaccinations involving production in bovine albumin cultures. Canine specific albumin has recently been produced as a commercial product, observed to increase serum albumin levels in the recipients with a low chance of immunologic complications. The most recent published study indicated albumin administration in dogs with septic peritonitis to have improved albumin level, Doppler blood pressure values, and colloid osmotic pressure measurements, as well as a comment on the association between albumin transfusion and survival. A connection between an improvement in serum albumin level and ultimate survival continues to be a topic under investigation.

Hypertonic saline (7% compared to 0.9% in normal saline) possesses higher osmolarity than crystalloids, which provides a hyperosmolar shifting of fluids into the intravascular space upon injection. The effect of hypertonic saline has a fast onset though the effect is also short lived. There may be additional beneficial effects such as reduced endothelial swelling, modulation of inflammation, and increased cardiac contractility. A mixture of hypertonic saline and synthetic colloids given simultaneously has been seen to improve hemodynamic status better than each given individually. Blood products such as pRBC and plasma may need to be administered in cases of severe hemorrhaging or coagulopathy. Blood products are not recommended to be used solely as volume replacement due to potential immunologic and non-immunologic complications. RBCs are warranted for existing or anticipation of clinically significant anemia due to the rate of hemorrhaging seen. Plasma is useful in replacing coagulation factors. Hemoglobin
based oxygen carrying solutions such as Oxyglobin, if available, will allow replacement of oxygen carrying capacity as well as providing colloidal effects without risk of immunologic complications.

When efforts in providing better intravascular volume are not sufficient in restoring adequate perfusion, vasopressor and inotropic therapy is required. Vaspressors function to provide vasoconstriction improving perfusion by increasing SVR. Vasopressors such as dopamine, norepinephrine, phenylephrine, epinephrine, and vasopressin may commonly be used. Inotropes such as dobutamine improve cardiac output through increasing myocardial contractility.

As with many conditions, successful treatment of shock depends on early recognition, assessment, and swift response and treatment of shock. Quick determination of the cause of impaired perfusion will allow for the appropriate fluid resuscitation strategy and medical management. Technicians play a large role in providing the monitoring of the patient as therapy is performed, through frequent monitoring of physical perfusion parameters and working in conjunction with the veterinarian to provide additional measures.