Dogs and cats with respiratory diseases can be challenging to anesthetize. During induction of general anesthesia and recovery the animal should be closely monitored and being ready for complications may save the patient’s life. During general anesthesia oxygenation via pulse oximeter and ventilation via capnograph should be considered. Animals with respiratory disorders may have problems exchanging oxygen at the alveolar/pulmonary capillary level and oxygen supplementation may be required during general anesthesia and pre- and post-procedure.

For these patients it is paramount minimizing stress during induction and recovery, pre-oxygenating, securing the airway after induction as soon as possible, ventilating, and monitoring closing during and after the procedure.

1. Upper airway disorders
   a. Brachiocephalic Syndrome
      Characterized by stenotic nares, elongated soft palate, everted laryngeal saccules, and hypoplastic trachea. Redundant soft tissue in pharyngeal and laryngeal areas may cause difficulty breathing. These patients may be challenging to intubate and severe complications may present during extubation and recovery. Prepare your equipment before sedating the dog and be ready to induce and intubate if the animal has trouble breathing. Opioids and acepromazine can be used for premedication. Have a wide selection of endotracheal tubes (ETTs), since these patients may take a much smaller tube that you would expect due to their hypoplastic trachea. This author’s preference is to induce these animals using propofol. This drug will cause apnea, but will also abolish the swallowing reflex allowing for a faster intubation. Plan of having extra injectable anesthetic agents, in case the procedure requires extubation and re-intubation of the animal. Before induction, have suction ready and plan of delivering oxygen flow-by after induction but before intubation, if a laryngeal exam is required. To do this, an insufflation line connected to an oxygen source can be taped to the blade of the laryngoscope (Fig 1).

   Figure 1. Insufflation line connected to the common gas outlet of the anesthesia machine and taped to the blade of a laryngoscope to use during laryngeal exams

   At the end of the anesthetic event, extubate after the animal is completely awake. These dogs usually tolerate the ETT in place very well. Have and induction agent ready (i. e. propofol) just in case the animal stops breathing after extubation. Monitor closely for complications during the next few hours and have an ETT 0.5-1 size smaller than the one you used for the procedure tide to the cage or close by, just in case you need to re-intubate should complications arise.

   b. Laryngeal paralysis
      Usually diagnosed in older medium/large breed dogs (i.e. Labradors and Golden Retrievers). Some dogs may present with severe difficulty breathing. Administration of acepromazine (0.01-0.02 mg/kg IV or IM) and oxygen supplementation may be beneficial before doing any diagnostics. Pre-oxygenation and rapid control of the airway is very important. Regurgitation should be avoided during before and after the anesthetic event, since these animals are at high risk of aspiration pneumonia. Use antiemetic and prokinetic drugs preoperatively (i.e. maropitant and metoclopramide). This author prefers avoiding full mu agonist opioids and alpha-2 agonists before induction to decrease the chance of vomiting. These drugs will also interfere with the laryngeal exam. After induction with propofol, administer O2 flow-by (see Fig 1) and wait until the animal breath again to perform the laryngeal exam. Doxapram 0.5-1 mg/kg IV can be used to stimulate the respiration and facilitate the exam. Anesthesia can be maintained with isoflurane or sevoflurane in O2. This author prefers to use ketamine and lidocaine CRI during the procedure and low dose of fentanyl CRI if needed. Be ready to extubate the patient after the tie-back procedure is done to examine the larynx. To do so, have propofol in case the animal gets light when extubated. Use a new ETT to re-intubate. Minimize stimulation during recovery. Ideally the patient should be pain-free, not dysphoric and awake enough to stay in sternal recumbency or standing. During extubation keep the animal in sternal and head up to decrease the chance of aspiration.

c. Tracheal collapse
   In small and toy breed (i.e. Yorkshire Terriers). Can be life-threatening. Avoid stress and use acepromazine (0.01-0.02 mg/kg IV or IM) if the animal is anxious. Oxygen supplementation should also be considered. Secure the airway as soon as possible after induction
and monitor closely after the anesthetic even. If the dog has severe difficulty breathing in recovery, consider inducing general anesthesia again and secure the airway.

d. Rhinoscopy and rhinotomy
The obstruction can be unilateral or bilateral (open mouth breathing?). Cats usually have difficulty breathing through their mouth, so they can be very severe distress if bilateral occlusion is present. Both procedures (rhinoscopy with biopsies and rhinotomy) can be very painful. Systemic analgesia and local blocks (i.e. maxillary block) should be considered. These animals may bleed a lot through their nose during the procedure and in recovery. Monitoring packed cell volume, total protein, and blood loss is recommended. During the procedure, pharyngeal packs are usually used. Remove them before recovery. Monitor animals in the post-operative period for blood loss and pharyngeal/tracheal obstruction.

2. Lower airway disorders
a. Infiltrative and chronic obstructive diseases
These animals may need to be anesthetized for diagnostic procedures related to their disease or for unrelated causes. Common pathologies are feline asthma, atelectasis, neoplasia, lung contusion, pulmonary edema, and pneumonia. It is important to minimize stress and provide oxygen supplementation when possible. It is important to protect the airway when possible. In small size animal, when bronchoscopy is required, removing the ETT may be necessary. In this case it is paramount to supplement oxygen (use flow-by or working channel of the scope) and monitor SpO2 and color of mucous membranes. Be ready to re-intubate if complications arise. Recovery should be quiet and stress/noise should be avoided. Oxygen supplementation should be considered

b. Lung lobe torsion
This is considered a surgical emergency. The animal (usually dogs with narrow and deep chest) may present in respiratory distress and depressed. The torsion is generally associated with chronic respiratory disease, trauma, neoplasia, and chylothorax. The venous circulation and the bronchus become obstructed, but the arterial flow persists. This causes severe congestion of the lobe with possible edema and pleural effusion. The patient may need to be sedated and a chest tube may need to be placed. During surgery, when the chest is open, the negative intrathoracic pressure is lost and the patient will require supported ventilation. Thoracotomies are painful procedures, make sure that the patient receives sufficient analgesia during surgery and in recovery. Systemic drugs such as opioids, alpha-2 agonists, ketamine, lidocaine can be use and local techniques should be considered (i.e. intercostal block or epidural injection). When the chest is closed, make sure to aspirate the air from the thoracic cavity until you reach negative pressure. Monitor in recovery for respiratory distress due to complications, such as pneumothorax, and pain.

3. Extrapulmonary diseases
a. Pneumothorax and pleural effusion
Sedation may be required if the animal presents in respiratory distress (acute onset). Provide oxygen and perform a thoracocentesis to evacuate the fluid or gas. If there is a big amount of fluid or air in the pleural space, place a chest tube. If tension pneumothorax is present, place a chest tube. See “Lung Lobe Torsion” for management during surgery and recovery

b. Diaphragmatic hernia
The animal can present in respiratory distress (acute/traumatic onset) or it can be asymptomatic if chronic or congenital. Part of the abdominal content is relocated in the thoracic cavity, compressing the lung and causing atelectasis. If possible, pre-oxygenate and prepare the patient for surgery before induction (if this doesn’t cause stress to the animal). Induction of anesthesia should be rapid and mechanical ventilation should be used. When anesthetized, the animal should be placed in sternal recumbency or in lateral with the herniated side down. When the abdomen is open, the chest is open too, since they communicate though the hernia. Use intermittent positive pressure ventilation if you haven’t started yet. If the diaphragmatic hernia is chronic, there is higher risk of morbidity/mortality due to reperfusion injury and prolonged pulmonary atelectasis. Lidocaine CRI can be used as an analgesic and as a free radical scavenger during surgery. A dose of corticosteroids can be administered before removal of abdominal contents from chest. The lungs should be re-expanded very slowly.

c. Other extrapulmonary diseases
Chest wall injuries, neurological diseases, obesity and abdominal distention can all cause respiratory dysfunction. Minimizing stress, providing oxygen support and intermittent positive pressure ventilation may be required for these diseases. Close monitoring, especially oxygenation (SpO2) and ventilation (end tidal CO2) are recommended. Titrate drugs to effect, especially if central neurological disease is present and use drugs that can be reversed. These patients should be monitored closely during recovery.
Main components of the anesthesia machine

The anesthesia machine consists of four main parts:

1. A source of carrier gas. This gas is oxygen (O2), but nitrous oxide (N2O) can be added to it. Different sources of O2 can be used:
   - E-cylinders. They contain 660 L with a capacity pressure of 1900 at 70°F. E-cylinders are commonly mounted on anesthesia machines to transport patients under general anesthesia. H-cylinders. They contain 6900 L with a capacity pressure of 2200 at 70°F. H-cylinders are placed on dollies or chained to the wall.
   - The following formula is used to calculate the amount of O2 left in an E-cylinder:
     \[ \text{Volume (L)} = \left(\frac{\text{pressure on gauge (psig)}}{1900 \text{ (psig)}}\right) \times 660 \text{ (L)} \]
   - The oxygen bank. It is comprised of several H-cylinders connected to the pipeline system.
   - Liquid O2. O2 does not liquefy at ordinary ambient temperature regardless of the pressure applied. It becomes liquid at approximately -182°F (critical temperature) when a pressure of about 50 atm (critical pressure) is applied. One cubic foot (about 28 L) of liquid O2 will produce approximately 24,080 L of gaseous O2 at ambient temperature (one volume of liquid O2 produces 860 volumes of gaseous O2). This is the most economical way to store O2 in hospitals where large amounts of this gas are used. Once the O2 is in its gaseous phase, it is distributed via the pipeline system.
   - Oxygen concentrators. They use the pressure swing adsorber technology, which increases the O2 concentration by adsorbing nitrogen into a molecular sieve and allows O2 and trace gases, especially argon, to pass through. They can reach concentrations of O2 between 90–96% and they can be used when compressed or liquid O2 are not available to generate O2 to supply the pipeline system.

2. A pressure regulator, which converts variable high gas pressure from cylinders to constant lower pressure suitable for the anesthesia machine. Regulators in the pipeline systems decrease the pressure to 50 psig, while regulators connected to E-cylinders decrease it to 45 psig. A second regulator further decreases the pressure to 16 psig to isolate the flowmeter from any fluctuations in the pipeline pressure.

3. A flowmeter, which measures and indicates the flow of the carrier gas and enables precise control of O2 (or N2O). The flowmeter is positioned downstream from the O2 source and upstream from the vaporizer(s). The gas enters the bottom and exits at the top of the glass tube. When the gas goes through the tube, it raises an indicator (float). A scale, associated with the glass tube, indicates the rate of gas flow in mL/min or L/min. A flowmeter indicator should be read at the top, except for a ball-type float, which is read at the center.

4. A vaporizer, which converts a liquid anesthetic (e.g. isoflurane, sevoflurane, and desflurane) into its vapor, and adds a specific amount of this vapor into the carrier gas. Modern vaporizers are concentration-calibrated (meaning that the vaporizer delivers a concentration that is close to the setting on the vaporizer dial), variable-bypass (meaning that after the carrier gas enters the vaporizer, part is directed to and part bypasses the vaporization chamber), temperature, flow, and back pressure compensated (meaning that the vaporizer has a means to compensate for fluctuations of temperature, flow of carrier gas, and back pressure in order to keep the delivered inhalant constant), and are positioned out of the circuit (meaning that the vaporizer in positioned outside and upstream from the breathing system).

Other components that are part of the anesthesia machine are

1. Safety devices for oxygen pressure and flow. The safety device can be an alarm or a mechanism that cuts off the supply of all other gases (e.g. N2O) when the pressure of oxygen flow reaches dangerously low values.
2. Flush valve. This valve delivers high flow of O2 (35 to 75 L/min) directly into the common gas outlet and bypassing the vaporizer. The flush valve is used when the operator wants to decrease the amount of inhaled anesthetic present in the rebreathing system and in the anesthesia machine (e.g. at the end of the procedure or in case of emergencies). It should never be used with a non-rebreathing system, due to high risk of overpressurizing the patient’s respiratory system.
3. Common gas outlet. It represents the interface between the anesthesia machine and the breathing system. This outlet is the site from which gases that have passed through the flowmeter and vaporizer (or flush valve) exit the anesthesia machine.
Breathing systems
A breathing system is connected to the anesthesia machine, to allow proper delivery of carrier gas (O2 or O2 with N2O) and inhaled anesthetic to the patient.

The main functions of the anesthesia machine and the breathing system are:

1. To provide O2 to the patient. The fraction of inspired O2 (FiO2) should never be less than 30%. In veterinary medicine, an FiO2 close to 100% is normally used. When N2O is used, the FiN2O is 60-70% and the FiO2 is 40-30%.
2. To deliver inhaled anesthetic to the patient. Inhaled anesthetics used in veterinary medicine in United States are isoflurane, sevoflurane, and, less frequently, desflurane.
3. To support ventilation. General anesthesia induces hypoventilation, due to the depression of the respiratory center. It is important to ventilate for the patient and, even if the animal is breathing on his own, one breath per minute or every other minute is recommended. This ensures delivery of O2 and inhalant, and stimulates the release of surfactant, which prevents atelectasis. Ventilatory support can be achieved by squeezing the reservoir bag or by using a mechanical ventilator.
4. To remove carbon dioxide (CO2) from the patient. When a rebreathing system or circle system (see below for definition) is used, the CO2 is removed by a chemical absorbent. The most commonly used in veterinary medicine is soda lime. When a non-rebreathing system or Mapleson system (see below for definition) is used, the CO2 is removed by the high O2 flow rate (100-200 ml/kg/min). If lower flow rates are used, CO2 may be rebreathed by the patient.
5. To remove waste inhaled anesthetic agent from the work environment. The use of a scavenging system is required by the Occupational Safety and Health Administration (OSHA). There are active and passive scavenging systems.

Types of breathing systems

Rebreathing systems or circle systems
As a general guideline, pediatric circle systems are used for patients between 5 and 10 kg, and adult circle systems for dogs weighing more than 10 kg. However, choosing the size of the circle system may be influenced by the species, the practical availability of equipment, the type of ventilation used, and the veterinarian’s preference. The universal F-circuit is a type of rebreathing system, where the inspiratory tube is placed inside the expiratory tube (co-axial configuration). Advantages of this system include compactness (important when the veterinarian needs to work in the patient’s mouth) and moderately increased inspiratory heat and humidity. Disadvantages include increased gas flow resistance and work of breathing, and difficulty in detecting possible leaks in the inner tube.

The inspiratory and expiratory tubes are usually corrugated (except for the inspiratory tube of the universal F-circuit). Corrugations prevent inhalation of dust coming from the CO2 adsorbent, inhalation of moisture, water, and condensation that gets trapped in the grooves of the tubes, and they reduce the likelihood of obstruction if the tubes are bent. The breathing tubes come in different length. Longer tubes do not increase mechanical dead space (volume of gas re-breathed as the result of use of mechanical devices, such as endotracheal tube, gas sampling line adaptors, etc.), but they will only add to the overall volume of the rebreathing apparatus.

Mechanical dead space ends at the point where inspired and expired gas streams diverge (the Y-connector).

The gases flow only in one direction in the rebreathing system. This is guaranteed by the correct function of the one-way valves (the inspiratory one-way valve opens during inspiration, while the expiratory valve is closed, and the opposite occurs during expiration). On inspiration, gases exit the reservoir bag and travel through the inspiratory one-way valve, the inspiratory tube, and the Y piece to the patient. On expiration, the exhaled gases enter the Y piece, the expiratory tube, and the expiratory one-way valve. Gases may enter the reservoir bag before or after going through the CO2 adsorbent canister. The excess gases exit the breathing system through the pop-off or adjustable pressure limiting (APL) valve and move into the scavenging system. The APL valve should be kept open when the patient breathes on his own, since it is a safety device that prevents dangerous rise of the pressure in the breathing system and in the animal respiratory system. It can be partially or completely closed when the operator delivers a breath to the patient, but it should be reopen after the breath. It should be completely closed when mechanical ventilation is initiated (mechanical ventilators have their own pressure release valves and an open APL valve will cause leakage in the system).

The reservoir bag provides compliance in the system during exhalation and provides a means for assisted or controlled ventilation. Commonly used sizes in small animals are 0.5, 1, 2, 3, and 5 L. The volume of the bag should exceed the patient’s inspiratory capacity, therefore a spontaneous deep breath should not empty the bag. The bag size should be about 6 times the patient’s tidal volume (Example: a 30 kg dog has a tidal volume of 300-600 ml, since the tidal volume is 10-20 ml/kg. For this patient a 2 or 3 L bag is appropriate).
The CO2 chemical adsorbent removes CO2 from the rebreathing system preventing inhalation of this gas by the patient. Before using the rebreathing system, the operator should always confirm that the adsorbent is functional. Some CO2 absorbents have indicators that change color when the absorbent is expended. Most of them, though, return to their original color after their use. The best way to determine if the CO2 absorbent is fresh is to crush a couple of granules between the fingers. If the granules crumble easily, the CO2 absorbent is fresh, if they are hard it is time to change the CO2 adsorbent. The lifespan of CO2 absorbents varies based on O2 flow rate used, size of the animal, and size and number of canisters (some anesthesia machines have a double canister), but in general it lasts about 6 to 8 hours.

The circle systems can be used as a closed system or semiclosed system. The only difference is that in the close system the O2 flow rate has to meet the O2 metabolic demand of the patient (4-10 ml/kg/min, depending on the patient’s body weight and body surface area, temperature, state of consciousness, and type of anesthetic). In the semiclosed circle, the most used in veterinary medicine, the O2 flow rate is set at 22-44 ml/kg/min. This means that we use a closed system for a 30 kg dog, the O2 flow rate should be 0.2-0.3 L/min. If we use a semiclosed system for the same patient we will turn up the O2 flow rate to 0.7-1.3 L/min.

Advantages of rebreathing systems include reduced cost related to inhaled anesthetics and decreased environmental pollution thanks since O2 and inhalants are rebreathed by the patient, decrease of loss of heat and moisture from the respiratory system, and large buffer for barotrauma if the APL valve is inadvertently left closed, thanks to the volume of the CO2 adsorbent canister, reservoir bag, and other components. The main disadvantages include greater resistance to breathing (especially due to the one-way valves and the CO2 adsorbent), maintenance of the CO2 adsorbent, longer time to change the concentration of inhalant delivered to the patient, bulkier design, and high number of connections (one-way valves, CO2 adsorbent canister, rebreathing tubes, etc.) where leaks might develop.

**Non-rebreathing systems or Mapleson systems**

As a general guideline, these systems are used in patients weighing less than 5-7 kg. They could be used for bigger patients, but the cost and the amount of waste gases will increase, since the O2 flow rate should be 100-200 ml/kg/min. This means that a 20 kg dog, on a non-rebreathing system, should receive 2-4 L/min of O2. These systems use no chemical adsorbent for CO2, but depend primarily on high fresh gas flow rates to flush exhaled CO2 from the system.

Mapleson systems comprise 6 configurations and some of them include modifications of their configuration. The classification of these 6 groups is based on the location of the patient’s connection, scavenging system, APL valve, and reservoir bag within the breathing system. A commonly used non-rebreathing system in veterinary medicine is the Bain system or Mapleson D and its coaxial modification. The inspiratory tube is generally smooth and smaller than the expiratory tube (which is corrugated) and it connects directly to the common gas outlet of the anesthesia machine. This allows for rapid changes in the concentration of inhalant anesthetics delivered to the patient.

Advantages of non-rebreathing systems include simple design and easy to use, can be easily cleaned and sterilized, lightweight and compactness, few moving parts and less chance of developing leaks, relatively inexpensive, do not require CO2 adsorbent, and changes in inhaled anesthetics can be rapidly achieved. The main disadvantages include high flow rate of fresh gas, which promotes patient’s heat and moisture losses, increases cost and environmental pollution, and higher risk of barotrauma if the APL valve is inadvertently left closed.
There are several tests that can be used to check the anesthesia machine and rebreathing systems for leaks, some of them tests different parts of the machine and others only test for 1 or 2 components. Below is a list of most commonly used leak tests.

1. **Anesthesia machine tests**
   These tests can identify leaks in the anesthesia machine (flowmeter, vaporizer, machine tubing…) but do NOT test the breathing system components (breathing tubes, reservoir bag, unidirectional valves, canister…).
   
   a. **Negative pressure test or universal test**
   You will need a squeeze bulb leak tester (Fig. 1). This can be bought or easily made using a sphygmomanometer bulb and reversing the air inlet valve in the bulb. When reversed, the valve will pull air from the anesthesia machine creating a negative pressure (see below step-by-step procedure).
   
   The bulb is then connected to a short tubing with a 15-mm connector (can use an endotracheal tube adaptor) at the other end.
   - Make sure the O2 flowmeter is turned off. If the anesthesia machine has a minimum mandatory flow, close/disconnect the O2 source.
   - Disconnect the fresh gas line and connector from the common gas outlet (you will not test the breathing system components with this test!)
   - Connect the 15-mm connector to the common gas outlet of the anesthesia machine.
   - Squeeze the bulb several times, until it stays collapsed.
   - If the bulb stays collapsed for 10 seconds, there is no leak.
   - If the bulb inflates, there is a leak.
   - Repeat these steps with the vaporizer on.

   ![Figure 1. Squeeze bulb leak tester connected to the common gas outlet](image1)

   b. **Positive pressure test**
   This test can only be used for machines that don’t have a minimum mandatory flow. The pressure used during this procedure CANNOT increase beyond the prescribed limits (see below step-by-step procedure), since there is only little room for compression in the machine tubing and high pressures can damage flowmeters and other components.
   - Connect a pressure gauge manometer to the common gas outlet. You can use the manometer from a sphygmomanometer (Fig. 2).
   - Slowly open the flowmeter until the manometer reads 30 cmH2O (22 mmHg).
   - Turn flowmeter off.
   - If the indicator of the manometer stays at 30 cmH2O, the machine passes the test.
   - If the indicator moves back to a lower pressure, there is a leak.
   - To quantify the leak slowly turn on the flowmeter until the indicator of the manometer stays still at 30 cmH2O.
   - The flow of O2 that allows to maintain this pressure represents the leak.
   - If this leak is 50 ml/min or less, the machine still passes the test.

   ![Figure 2. Pressure gauge manometer connected to the common gas outlet](image2)

   c. **Fresh gas line occlusion**
   - Set O2 flow at 50 ml/min.
   - Kink the fresh gas line.
   - The flowmeter indicator should move downward (no leak)
   - If the flowmeter indicator doesn’t move, the machine does not pass the test.

2. **Combination of breathing and machine leak tests**
   a. **Retrograde fill test**
   - Close the APL valve and the patient port of the breathing system using your hand.
   - Fill the reservoir bag using high O2 flow by opening the flowmeter or using the flush valve.
   - Pressurize the system to 30 cmH2O (pressure gauge manometer).
Turn the flowmeter of is used it to fill the reservoir bag and pressurize the system.
If the indicator of the manometer stays at 30 cmH2O, the machine passes the test.
If the indicator moves back to a lower pressure, there is a leak.
To quantify the leak slowly turn on the flowmeter until the indicator of the manometer stays still at 30 cmH2O.
The flow of O2 that allows to maintain this pressure represents the leak.
If this leak is 350 ml/min or less, the machine still passes the test.
To test for leaks in the vaporizer, repeat these steps with the vaporizer on.
To release the pressure open the APL valve and do not remove your hand from the patient port. Reasons:
You will also test that the APL is working
You won’t release inhalant in the room (remember you test the machine with the vaporizer on as well)
You will prevent a quick drop of pressure in the anesthesia machine (better if this occur gradually)
Dust from the CO2 absorbents will not go into the rebreathing corrugated tubes and, potentially, into the patient’s airway.

When a leak is suspected, the components of the machine and the breathing system should be checked, following the route of gas travel. A leak can be located by placing some alcohol and the hands and moving them over the components while the O2 is flowing. Alternatively, soapy water can be sprayed over the components while the O2 is flowing. Bubbles from the soapy water will reveal the leak (and you also cleaned the anesthesia machine!).

b. Squeeze bulb test
- Make sure the O2 flowmeter is turned off. If the anesthesia machine has a minimum mandatory flow, close/disconnect the O2 source.
- Remove the reservoir bag and connect a suction bulb with a 22-mm connector to the reservoir bag mount.
- Close the APL valve and the patient port of the breathing system using your hand.
- Squeeze the bulb repeatedly until the pressure reaches 50 cmH2O.
- If the pressure drops from 50 cmH2O to 30 cmH2O in 30 seconds or longer, the leak is acceptable.

3. Unidirectional valve tests
These tests only check for competency of the unidirectional valves (inspiratory and expiratory valves). The rebreathing tubes can be removed before performing these tests.

a. Valve tester
You will need a bulb with a 22-mm female connector for this test.

Inspiratory valve
- Compress the bulb and connect it to the inspiratory port.
- The bulb should immediately reinflate.
- When you compress the bulb (still connected to the inspiratory port), you should meet firm resistance.
- Expiratory valve
- Connect the bulb (inflated) to the expiratory port
- Squeeze the bulb.
- The bulb should collapse easily and should remain deflated.

b. Pressure decline method
You will need a second reservoir bag to preform this test.
- Place one reservoir bag on the reservoir bag mount.
- Place the second bag on the inspiratory port.
- Close the APL valve.
- Pressurize the system to 30 cmH2O using the O2 flush.
- If the reservoir bag on the mount remains inflated, the expiratory valve is competent.
- Now open the APL valve.
- If the reservoir bag on the inspiratory port remains inflated, the inspiratory valve is competent.
Local Anesthetic Blocks for Dental Procedures
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General anesthesia or heavy sedation is required to perform these blocks. Needle size and volume of the local anesthetic injected varies from location and size of the animal. Generally 25- to 30-gauge, 12-25 mm long needles are used. Bupivacaine or lidocaine are usually selected and volumes injected are between 0.2 and 2.5 mL. Always calculate the maximum dose you can inject and do not exceed that limit. For bupivacaine stay under 1.5-2 mg/kg in dogs and cats, for lidocaine 6 mg/kg in dogs and 2-3 mg/kg in cats. Once the needle is placed close to the nerve that needs to be desensitized, always aspirate to make sure you are not injecting the local anesthetic in a vessel before performing the block.

Inferior alveolar nerve block
This block desensitizes the ipsilateral lower lip, mandibular teeth and associated soft tissues. It can be performed using 2 approaches:
1. Intraoral; the mandibular foramen is palpated just caudal to the last molar while the mouth is kept open (use mouth gag to protect the operator hand). The needle is directed ventrocaudally on the medial side of the mandible aiming towards the angle of the mandible. Stay as close as possible to the mandible to avoid the lingual nerve.
2. Extraoral; with the animal in lateral recumbency, the uppermost mandibular foramen is palpated intraorally (use mouth gag). The needle is placed close to the foramen by inserting it through the skin perpendicular to the ventral margin of the mandible and on its medial side. Stay as close as possible to the mandible to avoid the lingual nerve.

Maxillary nerve block
This block desensitizes the ipsilateral upper lip, skin of the nose, mucosa of soft and hard palate, maxilla including the teeth and associated soft tissues. This block can be performed using 3 approaches:
1. Intraoral; the animal’s mouth is kept open (use mouth gag) and the needle is inserted caudal to the last molar perpendicular to the hard palate.
2. Subzygomatic; the needle is inserted through the skin perpendicular to the median plane of the head. The point of insertion is ventral to the zygomatic arch and between the caudal aspect of the maxilla and the coronoid process of the mandible.
3. Infraorbital; after identification of the infraorbital foramen via palpation (dorsal to the 3rd maxillary premolar, rostroventral to the eye) a thin needle is inserted in the infraorbital canal to reach the caudal position and exit the maxillary foramen where the local anesthetic is deposited. Higher risk of damaging neurovascular structures in the canal.

Infraorbital nerve block
The identification of the infraorbital canal can be done via palpation (see “Maxillary Nerve Block, infraorbital approach” for location). The needle is inserted through the skin (transcutaneous approach) or the mucosa (transmucosal approach) with the syringe parallel to the median plane of the head. The area desensitized by the block depends on placement of the needle and the volume used. If the local anesthetic is placed outside of the canal, only the ipsilateral skin of the nose and the upper lip are desensitized. If the drug in placed in the canal (by inserting the needle in the canal and/or by increasing the volume and gentle pressure with the finger on the injection site) some premolar, canine, incisor teeth and associated soft tissues will be desensitized as well.

Mental nerve block
This block is performed with the animal in lateral recumbency with the side to be blocked facing up. The middle mental foramen can be identified by palpating the root of the second premolar in dogs and the area between the canine and third premolar in cats. A transcutaneous or transmucosal approach can be used. The needle is placed between the finger palpating the foramen and the lateral aspect of the mandible and the local anesthetic is injected. This block only desensitizes the rostral lower lip. If the needle is placed inside the mental canal, the rostral inferior alveolar nerve can be blocked desensitizing also premolar, canine, and incisor teeth.

Palatine nerve block
This block is performed when only the mucosa of the soft and hard palate need to be desensitized. If other structures have to be blocked, such as maxilla and upper teeth, a maxillary block should be performed. The animal is positioned in dorsal recumbency with the mouth open (use a mouth gag). The needle in inserted midway between the palatine midline and the dental arcade at the level of the last premolar. A small volume is injected when preforming this block to avoid ischemic injury and pain after the effects of the local anesthetic wears off. Usually only 0.1 mL of local anesthetic is injected in small patients and 0.2-0.4 mL in larger animals.
**Microanatomy of peripheral nerves**

The nervous system is classified in central and peripheral nervous system. For the purpose of these proceedings on local anesthetic drugs and techniques only the anatomy of peripheral nerves will be discussed.

Peripheral nerves are made of several components. The outermost structure of the nerve is the epineurium, made of dense and irregular connective tissue. The epineurium holds together the vessels that supply the peripheral nerve and multiple fascicles, small bundles of nerve fibers. Each fascicle is surrounded by the perineurium, a protective structure composed of connective tissue with laminar arrangement. Each nerve fiber contained in the perineurium is surrounded by a protective sheath of connective tissue called endoneurium. When peripheral nerves are temporarily blocked with local anesthetics, the drug is injected in the vicinity of the nerve. This technique is known as regional or nerve block anesthesia.

Most of the peripheral nerves are composed by different types of nerve fibers. These nerve fibers are classified based on presence of myelin, diameter, and conduction velocity.

The A fibers are large, myelinated, and have high conduction velocity. They modulate muscle and reflex activity (α) muscle tone (γ) and transmit information about pressure and touch (β). The Aδ fibers are responsible for transmission of touch, pressure, and fast, sharp, well-localized pain. The group B is composed by small, myelinated fibers, which are responsible for modulating the autonomic functions. The C fibers are small and non-myelinated and transmit slow, dull, and non-well localized nociceptive information (pain). As general rule, when local anesthetics are used for regional anesthesia, fibers with smaller diameters are affected first. Most local anesthetics will cause sensory blockade before peripheral motor blockade. This difference may not be noticed if larger volumes or high concentrations of local anesthetics are used.

**Pharmacology**

Local anesthetics are divided into amino-esters and amino-amides, based on their chemical structure. Examples of amino-esters are cocaine, benzocaine, procaine, chloroprocaine, and tetracaine. Examples of amino-amides are lidocaine, prilocaine, etidocaine, mepivacaine, bupivacaine, levobupivacaine, ropivacaine, and articaine. Amino-esters are hydrolyzed by cholinesterase in the plasma and liver (cocaine is an exception, because it undergoes significant liver metabolism), while amino-amides are metabolized in the liver by microsomal enzymes.

Local anesthetics are weak bases, with pKa (dissociation constant or negative logarithm of the dissociation constant) between 8 and 9. When the pH of the environment equals the pKa, the drug is 50% dissociated (or ionized, or charged) and 50% non-dissociated (or unionized, or neutral). Unionized drugs are more lipid soluble and will cross the cell membrane. If the pH of the environment increases (becomes more alkaline), weak bases like local anesthetics dissociate less and will present more unionized drug, which is more lipid soluble and will pass through the cell membrane.

Once the unionized form is in the cytosol, it has to be ionized again to be able to block the Na+ channels. These channels have to be in the activated-open conformation to allow binding with local anesthetics. Once this happens, the local anesthetic will change the channel conformation into inactivated-close, which will prevent the Na+ from entering the cell and, consequently, will block the action potential of the nerve fiber (and transmission of the signal). This phenomenon is known as frequency-dependent block, meaning that repetitive stimulation of nerve fibers increases the binding affinity of the receptor site for local anesthetics and facilitates the development of neural blockade.

The chemical structure of local anesthetics determines the properties of the drug. Lipid solubility, as mentioned above, influences penetration through the nerve membrane, but it also promotes sequestration of local anesthetics in lipid soluble compartments, such as myelin. Compounds that are more lipid soluble have a longer onset and duration of action compared to less lipid soluble drugs. Lipid solubility is directly correlated with the potency of local anesthetics. The pKa also influences the onset of the local anesthetic, since it will determine how much of the compound will be unionized (and more lipid soluble) in a given pH. Drugs with higher lipid solubility also show higher degree of protein binding. The “free” (unbound) drug is the active form, which can block Na+ channels. Local anesthetics with high degree of protein binding are metabolized slower and have longer duration of action.

Mixing two local anesthetics has become common practice. The main theoretical advantage is to decrease the onset and increase the duration of action by mixing a local anesthetic with short onset and another with long duration. Unfortunately, this is not the case. When two drugs are mixed together, the pKa of the mixture in unknown and the onset and duration are unpredictable. In addition, a 50:50 mixture will have half strength concentration of each drug. This may influence the property of both local anesthetics, by decreasing the onset and shortening the duration of action (1,2). Due to the lack evidence showing the advantage of mixing different
local anesthetics, it is recommended to choose only one drug per block based on pharmacokinetics and pharmacodynamics of the local anesthetic and the type of block and procedure performed.

**Adjuvants**

Adjuvants are often added to local anesthetics, to increase the duration and the analgesia of the block. The most commonly used are: epinephrine, sodium bicarbonate, opioids (especially buprenorphine), and alpha2-agonists (dexametomidine).

Epinephrine causes vasoconstriction when used for regional anesthesia, which decreases bleeding in the surgical field, decreases systemic absorption of the local anesthetic, and increases the duration of action. The usual concentration of epinephrine is 5 μg/ml or 1:200,000. Market preparations of local anesthetics containing epinephrine have a lower pH than plain solutions or solutions freshly prepared. The pH of 2% lidocaine and 0.5% bupivacaine are 6.78 and 6.04, respectively. When epinephrine is freshly add to these drugs, the pH becomes 6.33 and 5.99, respectively. In market preparations of 2% lidocaine with epinephrine and 0.5% bupivacaine with epinephrine, the pH is 4.55 and 3.73, respectively. Decreasing the pH of the solution will increase the percent of the ionized form of the drug and, consequently, slowing the onset of action. When epinephrine is used in regional block anesthesia, it is important to avoid injection of the solution in terminal arterioles, which can cause necrosis of the supplied area, and intraneural injection, which can cause ischemic nerve injury.

Sodium bicarbonate is added to local anesthetics to increase the pH of commercial solutions. Although local anesthetics are weak bases, the pH of their commercial solutions rages between 3.9 and 6.7. Alkalinizing the solution increases the unionized fraction of the drug, which shorten the onset and increases the duration of action. Unfortunately, local anesthetics cannot be alkalinized to pH values greater than 6-8, because this cause precipitation of the resulting solution. Increasing the pH to these values only increased the unionized fraction by 10%. Modifying the pH of the solution has also the advantage of decreasing discomfort on injection.

Opioids have been mixed with local anesthetics to increase the duration and enhance the quality of the regional block. Buprenorphine is commonly used, due to its long duration on peripheral μ receptors and its local anesthetic-like mechanism of action involving Na+ channel block (3). There are few studies in people that showed that buprenorphine enhances analgesia following sciatic nerve block (4) and, when combined with bupivacaine for minor oral surgery, provides a 3-fold increase in the duration of postoperative analgesia when compared to bupivacaine alone (5) This has not yet been documented in veterinary medicine.

Alpha2-agonists, and more specifically dexametomidine, can be mixed with local anesthetics to enhance duration and sensory analgesia. It has been shown that in rats dexametomidine prolongs the duration of sciatic nerve block when combined with either ropivacaine or bupivacaine (6-9). In people when dexametomidine is added to either levobupivacaine or ropivacaine, it shortens the onset and increases the duration of axillary brachial plexus block (10-12) and prolongs postoperative analgesia after cleft palate repair when mixed with bupivacaine for palatine nerve blocks (13).

**Toxicity**

Signs of systemic toxicity caused by local anesthetics can be seen if high plasma levels are achieved. This can happen if toxic doses are administered to the patient and/or if a local anesthetic, such as bupivacaine, is accidentally injected intravenously. In general neurological signs manifest first. Unbalanced excitation (i.e. nystagmus, muscular twitching, and seizures) can be seen due to the depression of cortical inhibitory pathways, followed by generalized depression of the central nervous system (CNS) resulting in coma and respiratory arrest. The first CNS signs may be difficult to identify when the patient is under general anesthesia. If the awake patient shows neurological signs, oxygen administration, intubation and assisted or controlled ventilation, and treatment for seizures (benzodiazepines, propofol, levetiracetam) should be initiated. Cardiovascular (CV) toxicity is usually seen after seizures, with the exception of bupivacaine, the most cardiotoxic local anesthetic. CV signs are characterized by depression of contractility and conduction velocity through the heart. An overdose of lidocaine usually results in hypotension and bradycardia, whereas bupivacaine and ropivacaine can induce sudden CV collapse or ventricular dysrhythmias that are refractory to treatment. Depending on the CV signs, intravenous fluids, vasopressors, inotropes, anticholinergics, CPR (cardiac massage, bretylium, magnesium, defibrillation) should be considered. Administration of 20% lipid emulsion (4 ml/kg bolus, followed by 0.5 ml/kg/min for 10 minutes) is also recommended.

**References**


Case 2

Signalment:
Buster, 6-year-old, miniature Schnauzer, intact male, body weight 8 kg

History
Buster becomes tired soon after he starts exercising

Physical exam
- T: 100.1° F  P: 210 bpm (irregular) RR: 20 bpm
- Buster is BAR and overall calm
- Auscultation
- Lung field is normal
- Cardiac auscultation → irregular rhythm
- Blood-work within normal limits

Presenting complaint and plan
Buster needs a dental prophylaxis and extraction of a fractured 104 (canine)

1) Before anesthetizing Buster, you decide to perform and ECG. What do you think (ECG provided during presentation)?

2) Do you proceed with anesthesia or do you reschedule the procedure?

Anesthetic protocol:
Pre-medication
Morphine 0.5 mg/kg IM
Acepromazine 0.02 mg/kg IM
Induction
Propofol 4-6 mg/kg IV to effect
Maintenance
Sevoflurane in 100% O2 (adult rebreathing system)

3) What else could you do to prevent and manage nociception and pain caused by this procedure?
4) During the procedure the heart rate increases up to 200 beats per minute (it was 140 beats per minutes before the procedure). Look at the ECG (PowerPoint). What do you think?

5) Would you treat this arrhythmia? If so, how?

6) What about Buster’s capnograph? What is your diagnosis?

7) What is the technology used by capnography and how many types of capnographs can you name and what are their advantages and disadvantages?
Why is it so critical to understand body postures in dogs? There are several reasons why this is an important topic with any discussion of dog behavior. By understanding how dogs communicate we can diminish the amount of miscommunication that occurs between people and dogs, it can help us better predict future behaviors in the dogs we interact with, understanding how dogs communicate can help reduce the incidence of dog bites, and it can increase the enjoyment people can have in their relationships with their dogs.

Behavior evolves just as body type evolves. Behavior can change over time as a dog learns what behaviors work in a given situation and which do not. As a result the successful behaviors will flourish while those that are less successful will tend to fade. This evolution can be seen in the individual animal by observing body posture since this is the principle means by which dogs communicate.

The eyes, ears, tail, mouth and overall posture can give us the best indications of what dogs are trying to communicate. These structures can convey relaxation, anxiety, tension, or confidence and by understanding the subtleties of their expressions, much ambiguity can be eliminated.

Because aggressive can greatly influence the bond and attachment we have with our pets, an understanding of the progression of aggressive responses can help in minimizing exacerbation of problem behaviors. The “Ladder of Aggression” serves to provide a good model of how aggressive behavior can develop from relatively benign “calming signals” to more overt aggressive displays culminating in snapping and biting.
Aggression is the most common behavior problem presented to veterinary behaviorists followed anxiety related disorders (separation anxiety, phobias). Traditionally, dominance aggression is most often diagnosed, especially when evaluating owner directed aggression. As a result of the label “dominance” being applied in these cases, owners were often directed to establish themselves as higher ranking over the dog through the use of a variety of physical means (punishment, alpha rolls, leash hangs, pinch and shock collars, etc.). Escalation of aggressive responses often followed this approach. By examining the situations in which the aggression occurs, body posture exhibited by the dog and evaluating the early history of the behaviors it becomes evident that not all aggression is related to a question of dominance hierarchy. In many, if not most, of these cases a definite fear component seems to be the driving force behind the aggressive displays. This presentation is meant to clarify terms, differentiate possible diagnoses of aggression and offer thoughts on treatment of fear associated aggression.

Aggression is a normal canine behavior when displayed in the proper context. As a tool, aggression is utilized by dogs for a variety of purposes such as acquisition of food, defense of resource (food, territory, mating access), establishment of pack hierarchy, and self defense when threatened. In addition, submissive displays (averting stares, exposure of the underbelly, urination and retreat) are often utilized when a dog is presented with an overwhelming threat. If these signals are not recognized, a subordinate individual may be forced to rely on aggression (growling, barking, snarling or biting) as a last resort.

When examining these behaviors in the context of human-canine interactions, several factors must be considered. Do dogs and humans communicate in the same manner? While both are social species, methods of exchanging information differ. Often submissive signals are missed by observers not familiar with canine body language. As a result, dogs may be put in a position to use aggression when more subtle signals of submission are missed. Over time, learning can occur such that some dogs will totally abandon these submissive cues and instead more quickly elect to utilize these more offensive strategies to alleviate perceived threats.

Secondly, when punishment is used by humans as a means of exerting dominance, fearful dogs may be forced to respond aggressively while more confident animals may see the use of punishment as an incentive to engage in a so-called “arms race”. This involves raising the bar by showing higher and higher degrees of aggression in response to ever increasing levels of punishment. In addition, punishment is often applied in the inconsistently creating an increased anxiety in the fearful animal. Not knowing whether to expect reward or punishment, conflicting emotions result lowering the threshold of reactivity and increasing the chance the dog will resort to the use of aggression.

It also appears that fear can be highly inherited so that fearful, anxious or timid parents can produce a higher number of similarly behaved puppies in a litter. Combine this genetic component with the previously described communication breakdown and the true meaning of nature and nurture can be seen. In addition, failure to positively socialize during the sensitive period (up to 14 weeks of age) results in the genetic prophecy of fearful behavior being fulfilled.

**Diagnosis**

Body posture at the time surrounding the aggressive episode can be most valuable in determining etiology. Typical signs include:

- Tail dropped or tucked
- Ears laid back
- Dorsal Piloerection (evidence of arousal and non-specific for fear)
- Weight positioned over hind legs, head and neck lowered
- Gaze dorsally or via sideway glance at target
- Autonomic responses (urination, defecation, anal sac expression)
- Lip retraction (Vertical)

This may be the early presentation in a younger dog. Over time, the body language may suggest a more confident dog as it learns to deal with its fear and anxiety by adopting a more offensive strategy:

- Tail raised
- Ears forward
- Piloerection
- Weight shifted forward with head raised
- Staring directly at target
- Lunging at or chasing target
In a fearful animal, the target is often an unfamiliar person or can be a very familiar person when conflict exists. It can be sometimes seen where an initially offensive aggressive dog can revert to a more defensive body posture if the threat does not retreat or is sudden and overwhelming.

The situation often also helps determine etiology.

A typical presentation where fear is induced and has the potential to result in aggression includes:

- Approach from a stranger while on leash walk (leash can transmit owner anxiety, prevents escape by the dog, and also prevents canine specific communication in cases of Interdog aggression).
- Situations where persons are bitten on the hand while reaching toward the dog
- Being bitten on the backside or caudal thighs/feet (common with herding breeds)
- Secondary to punishment by strangers or owners
- Commonly seen with strangers entering the home or moving suddenly
- Young, mobile, active children. Unpredictability breeds anxiety in the dog and can cause biting to prevent movement.

Abuse can cause fearful behavior but commonly is displayed as fear toward a specific trigger as opposed to more generalized responses.

Dominant behavior over another individual normally is not seen until a dog reaches social maturity (12-18 months) whereas fearful behavior is often seen very early (at times as early as 8 weeks of age). Body postures associated with dominance are usually more offensive in appearance, they never have an early defensive presentation and is often associated with control of resources (food, space, items) or secondary to attempts to direct the animal’s behavior (commands, pushing, wiping feet, approaches, etc.). Dominant animals can also attempt to block movement of individuals. Dominant behavior can be very calculated and purposeful whereas fear responses are much more sudden and reactionary.

The successful use of aggression in a defensive situation can become a learned behavior. Over time, this response can be used in similar situations with greater confidence. As a result, the aggression can be displayed with increasing efficiency.

The principles of reinforcement and conditioning apply to the use of aggression.

Need to know the situation in which the aggression is occurring and the past history of aggressive behavior in order to make a proper diagnosis. Aggression is not static. Constant interaction of genetics and environmental influences can determine behavior at any one point in time.

**Conflict aggression**

- Often Diagnosed as Dominance Aggression
- Often show submissive posture. Not confident.
- Ambivalent body language (wagging tail while growling). May show “remorse” after aggression.
- Conflict occurs when put in confrontational situation or when cannot predict interaction.
- Dog learns to use aggression to get out of uncomfortable situation and is reinforced
- Owner directed aggression can occur in fear based situations:
  - Inappropriate use of punishment
  - Attempt to create owner: canine dominance structure in household
  - Inconsistent interactions

**Treatment of fear based and conflict behavior**

The basis of treatment is to remove exposure to inciting stimuli, utilize counter-conditioning/desensitization and at times prescribe anti-anxiety medication.

**Removing stimuli – can be accomplished in several ways:**

- Response Substitution - Discontinue all forms of punishment. Focus instead on distraction and redirection of inappropriate behavior to more appropriate responses which can be reinforced.
- Head Halter – Can be used to help facilitate response substitution with the use of an indoor drag leash. Head halter decreases arousal and allows safe, efficient, non-emotional interruption of problem behaviors.
- Avoid reinforcement of the behavior by withdrawing in response to aggression or giving positive attention (telling the dog, “it’s all right”).
- Have unfamiliar people ignore dog at first greeting to allow more time for the dog to assess the situation without feeling threatened.
- Identify any fear inducing triggers and avoid. For example, if house has several young children, isolating dog can avoid potentially negative interactions.
- Increase consistency of owner and dog interaction. Always give a command, wait for a response and reward.
- Avoid inconsistent, casual interactions by ignoring all attention seeking behaviors. Punishment should never be used.
Often called “Nothing in Life is Free” or “No Free Lunch”

**Counter conditioning**
Counter Conditioning is the proactive relaxation techniques in all environments that the dog will be in without presence of offending stimuli.

Make use of a palatable treats made available by visitors (while still ignoring dog) as a means of accomplishing Classical Conditioning (associate visitors with positive results).

**Desensitization**
By using fear inducing triggers that gradually increase exposure while asking for, and rewarding, relaxed behaviors taught during the counter conditioning phase. Examples would be people entering the home or approaches from strangers or unfamiliar dogs.

**Medication**
The use of medication addresses anxiety issues which can accompany fearful behavior. Anti-anxiety medications are indicated when the degree of anxiety is great enough to interfere with the ability to learn as behavior modification techniques are applied. Common side effects include sedation, anorexia, gastrointestinal disturbances, increased aggression and anxiety.

**Typical anxiolytics include**

**Tri cyclic antidepressants (TCA’s)**
- Clomipramine (Clomicalm) 2-4 mg/kg BID
- Amitriptyline (Elavil) 1-3 mg/kg BID-TID

**Common side effects include**
Sedation, anorexia, gastrointestinal disturbances, increased aggression, anxiety and drug tolerance.

**Selective serotonin reuptake inhibitors (SSRI’s)**
- Fluoxetine (Prozac) 1 mg/kg SID
- Paroxetine (Paxil) 1 mg/kg SID

**Common side effects include**
- Sedation and anorexia
- Long half life results in delay (6-8 weeks) to effect

**Benzodiazepines (BZD’s)**
Benzodiazepines are contraindicated due to potential for disinhibition of fear and possibly heightening the aggression.

**Conclusion**
Aggression, even when directed at owners, should not be automatically classified as Dominance Related Aggression. Often, the origin is a fear based response directed at unfamiliar people or, when conflicting signals are displayed by the dog’s owners, can also be directed toward more familiar people. Understanding the animal’s history and body language can be valuable in making the correct diagnosis. Treatment can include avoiding trigger stimuli, utilizing counter conditioning and desensitization (after grading the stimuli) as well as adding appropriate medication where indicated.
Medical causes – LUTD
- Cystic Calculi
- Crystaluria
- Bacterial Infection
- Neoplasia
- Interstitial Cystitis
- Viral, Stress Induced, Idiopathic

Medical causes – PU/PD
- Chronic Renal Failure
- Diabetes Mellitus
- Pyometra
- Estrus
- Hyperthyroid

Medical causes – fecal abnormalities
- Inflammatory Bowel Disease
- Dietary Intolerance
- Gastrointestinal Parasitism
- Neurological or Locomotion Abnormalities

Minimum database
- Urinalysis
- Urine Culture if indicated by U/A or blood work (ex. If Azotemic)
- CBC
- Chem. Profile
- Total T4

The goal in making a behavioral diagnosis is deciding between: marking vs. toileting

Minimum behavioral database
- Location of elimination and substrate - Marking typically occurs on vertical surfaces vs. horizontal
- Along walls, center of room, near windows or doors - Marking can often occur along perimeters
- Personal items vs. flooring - Horizontal marking can occur on personal items
- Type of elimination - Stool vs. urine (domestic cats do not mark with stool)
- Volume of urine - Marking commonly associated with small volumes
- Length of time problem has been occurring (Chronic vs. acute) - Can give an indication of prognosis
- Began as adult or kitten - Marking usually begins as kitten ages (after successfully using the litter box)
- Frequency of housesoiling incidents - increased frequency can be seen with marking behavior
- Number/Types of surfaces - marking commonly involves multiple surfaces
- Number of litter boxes and location – (Rule of Thumb: 1 box per cat + 1 and boxes should be separated in space to increase number of “core areas”
- Type of box - Covered vs. Uncovered
- Liners Used
- Size of box
- Litter types used (scented vs. unscented, clay vs. clumping)
- How long were the litters used
- Cat’s response to each litter
- Cats in household
  - Number of cats in household - Increased marking with increased # of cats
Correctly ID problem cat - Use of fluorescein and non-toxic crayons

- Relationship between cats
- Access to outdoor animal activity - Territorial marking near viewing areas
- Changes in household (people and pets)
- Routine change in the home prior to onset of problem
- Previous treatments and results

**Behavioral causes**

**Toileting issues**

- Substrate Preference - Cats will strive to reach proper substrate material.
- Substrate Aversion - Unacceptable litter type and can also occur secondary to LUTD or de-claw
- Location Preference - Cat finds an alternate location that it prefers in place of where litter box is located. Could be an area where cat feels safe or prefers secretive elimination.
- Location Aversion - Cat may have been frightened in the litter box area or had been attacked by another cat in the home while using the litter box.

**Marking behavior**

- Vertical Marking (Spraying) - Typical Posture with tail raised, quivering and urine projected in a horizontal fashion
- Horizontal Marking - not as common. Characterized by depositing urine on personnel items
- Middling (Fecal Marking) not suspected to occur in domestic cats.

**Characteristics of marking**

- Small Amounts of Urine
- Deposited on vertical surfaces (spraying) or on personal items (horizontal marking).
- Locations - No commonality of surface types (carpet, tile, wood, etc)
- Litter Use - Normal frequency of litter use. There is typically no issue with acceptance of litter. Remember, marking is for communication purposes.
- Elimination Posture - Spraying (tail raised and quivering)

**Treatment options**

- Toileting Issues
  - Place Litter Box in Cat’s Preferred Location - consider placing a litter box in this area in order to determine if the problem is location-related.
  - Litter Trial - Offer several litter choices and record frequency of use of each.
  - Confinement with Preferred Litter - The goal is to increase the likelihood of the cat re-acclimating to the litter of choice
  - Prevent Access to Soiled Areas
  - Enzymatic Cleaners (Anti- Icky Poo, KOE)
  - Litter Box Care
    - scoop daily
    - open litter boxes
    - no liners
    - clean with hot water only
    - 3-4” of unscented litter
  - Appropriate Number of Litter Boxes - 1 box per cat plus 1 additional and distributed around the home.

It is important to gradually reintroduce cat to living area after proper interval of confinement. Slowly increase access to increased number of areas of the home. Be sure to provide additional litter boxes (with the preferred litter) in those areas to increase the likelihood of the cat using the box with the proper litter material.

**Treatment options**

**Marking behavior**

- Treat as for Toileting Issues - Evidence suggests that, even for marking behavior, proper litter management (#of boxes, dispersed throughout the home, proper litter cleaning protocol) can increase the tendency to utilize the litter box for elimination
- Medication
  - Clomipramine – 0.25-0.5 mg/kg bid
  - Fluoxetine – 0.5-1.0 mg/kg sid
- More effective, safer and less recidivism rates as compared to Diazepam and Buspirone
• Treatment Options
• Feliway – synthetic Feline Facial Pheromone. Apply to marked areas and prominent spots in the home. Available as a spray or a plug-in diffuser.
• Provide alternate marking opportunities
  o scratching posts or scratch boxes (in a proper location)
  o scratching combs (Cat A Comb)
• Manage relationship issues in the home - Address aggression issues between cats (indoor and outdoor) as well as relationship with human members of the household.
Resource Guarding:
What’s Mine is Mine and What’s Yours is Mine
John Ciribassi, DVM, DACVB
Chicagoland Veterinary Behavior Consultants
Carol Stream, IL

The focus of the discussion

- Which individual in a dyad (pair of animals) is considered to be dominant in the relationship?
- What criteria is used to make that determination (acquisition of resource vs. defense of resource)?
- Does aggression over the control of resources equate with dominance based aggression?

“Dominance: the assertion of one member of a group over another in acquiring access to a piece of food, a mate, a place to display, a
sleeping site or any other requisite that adds to the genetic fitness of the dominant individual…” E.O. Wilson from Sociobiology: The

Resource holding potential
“……examples of “aggressiveness” are far more likely to represent long-term differences in subjective resource value.” Hurd PL.
“Resource holding potential, subjective resource value, and game theoretical models of aggressiveness signaling.” J Theor Biol. 2006

“Dominance is a concept found in traditional ethology that pertains to an individual’s ability, generally under controlled conditions, to maintain or
regulate access to some resource.” Karen Overall (“Clinical Behavioral Medicine for Small Animals” Mosby 1997. pg. 115

“Relative dominance is usually tested by giving two dogs access to one bone. The dog that gets possession is considered the
higher-ranking dog.” Katherine Houpt (“Domestic Animal Behavior for Veterinarians and Animal Scientists” Iowa State U. Press
1982 pg 65)

“…a single bone was brought in, shown to the puppies, and laid between them….”
“…we defined a completely dominant animal as one that kept possession of the bone the majority of the time and was able to repossess it at will.” John
Paul Scott and John L. Fuller (“Dog Behavior: The Genetic Basis” The University of Chicago Press 1965 pg. 156)

“The dominant dog shows a self-assured gait, a large, confident body posture, raised head, raised ears, large eyes and curled lips, all in different
intensities and combinations depending upon the degree of dominance, superiority, or self-confidence.” Roger Abrantes (“Dog Language” Wakan
Tanka Publishers 1997 pg. 93)

“…Once everyone knows his place, the alpha male need only move to ward a lower-ranking male to have that individual hurry out of the way or

Equal opportunity tests (EO tests)
“In equal opportunity tests (EO tests), both members of a pair had equal chance to seize the bone when it was tossed into the arena” Beach, Beuhler
and Dunbar (“Competitive behavior in male, female, and pseudohermaphroditic female dogs.” J Comp Physiol Psychol. 1982 Dec;96(6):855-74)

Established possession tests (EP tests)
“During an EP test, the loser of the preceding EO test was given possession of the bone before the former winner was returned to the test arena” Beach,

“…for a meaningful formal test of dominance, and to rule out differential motivation as a confounding factor contaminating the results, both animals
must be motivated equally for the same resource.” Wendy van Kerkhove (“A Fresh Look at the Wolf-Pack Theory of Companion-Animal Dog Social
Behavior” JOURNAL OF APPLIED ANIMAL WELFARE SCIENCE, 7(4), 279–285)

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Possessive aggression
Aggressively guarding or maintaining control of a valued object (bone, chew item, stolen items or food, etc.). Guarding is considered
to be normal behavior but can increase with opportunities for learning or can be exaggerated as a consequence of fear or defensive
behavior/conflict.
“…food guarding was the most common circumstance for bites to familiar children (42%) and territory guarding for bites to unfamiliar children (53%). Behavioral screening of the 103 dogs examined revealed resource guarding (61%) and discipline measures (59%) as the most common stimuli for aggression.” Reisner IR, Shofer FS, Nance ML; “Behavioral assessment of child-directed canine aggression.” Department of Clinical Studies, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA 19104-6010, USA.

- Food Guarding
- Resource Guarding
- Possessive Aggression

These are all terms describing the use of aggressive behaviors to maintain possession of valued items.

The aggression can be directed towards humans or other animals.

Items can include anything which motivates an individual animal. In companion dogs these can be:

- Food
- Bones
- Rawhide
- Stolen Items

Possessive Aggression

The sphere of guarding (critical distance in which a dog may react to approaching individuals) can increase over time to the point of the animal guarding a space that the valued object is contained within.

The behavior can be seen concurrently with Conflict Aggression and Territorial Aggression.

Punishment or forced removal of items or food can increase the likelihood of the animal escalating aggressive displays to maintain control of items. This fear based response can result in the aggressive guarding of benign items that may not contain the same value as the original objects possessed by the dog.

The aggressive behaviors can be directed to both familiar and unfamiliar individuals when the appropriate circumstances exist to motivate the guarding response.

Fear based body postures may be present initially but over time, as the dog learns the value of using aggression, body language may appear more confident.

Other possible diagnoses

- Disease Conditions - Is there a medical condition causing the dog to use aggression to prevent pain inducing activities
- Conflict Related Aggression - Does the aggression extend to other situations where the dog is using aggression to have an individual cease certain activities
- Dominance Related Aggression - Does the dog displace another individual from a valued resource?

Medical examination

Always begin with having the animal evaluated medically and appropriate testing should be performed. Conditions which cause pain or conditions which increase appetite may result in an increase in food acquisition and guarding behaviors.

Treatment

- Avoid known triggers (secure food, control access to toys and highly valued items, isolate during feeding and feed small meals)
- Consistent periods of play and exercise
- Avoid confrontation over retrieval of objects
- “Nothing in Life is Free” routine in order to increase consistency of interactions and put control of resources in owner’s hands
- Provide alternate items and activities, especially at high risk times, to substitute for the animal focusing on other valued items
- Trade for valued items that must be retrieved
- Utilize a leash and head collar to facilitate redirecting the dog’s behavior when needed

Once the level of tension has reduced between the dog and owner, if desired, the owner can work on teaching:

- “Drop It” and “Leave It” commands for managing object possession
- Desensitization to the presence of the owner around the food bowl in order to manage food guarding behaviors

Possessive Aggression is typically managed and controlled and not cured. As with most forms of aggression, the only guarantee can be made with a recommendation of euthanasia. Short of this option, the owner is always accepting some degree of risk.
Symptoms of anxiety, distress or panic exhibited when animals are left alone. Separation anxiety can be characterized by pacing, drooling, vocalization, destruction, and elimination which are not related to other behavioral disorders. All or some of these behaviors can be present.

**Behavioral symptoms**
- Monotonal Vocalization/Barking - Typified by barking and whining which begins soon before or after departure and persists for a large percentage of the time the dog is alone. Often is reported to the owners by neighbors.
- Inappropriate Elimination - Depositing of urine and/or stool in various locations around the home (as opposed to in a single, consistent location). Only occurs when the dog is alone or perceives that they are alone. Stool may be abnormal in appearance (is commonly mucoid).
- Destructive Behavior - Characterized by damage to exit points from the home (doors and windows) or destruction of personal items (pillows, clothing, remote control units). Confinement in a cage often escalates the destruction and can result in injury to the animal (tooth or toenail fracture for example)
- Hypersalivation - Is often considered to be highly suggestive of separation anxiety when the behavior is restricted to those times when the dog is alone or perceives to be alone.

**Data collection**
- Physical Examination
- CBC
- Chemistry Profile
- Thyroid Profile
- Urinalysis
- Fecal Exam

**Behavioral history - who, what, when, where**
Who is present at the time of the behavior (is the pet alone or are there people present), before the behavior begins (departure) and afterwards (arrival).

Who is the primary caretaker of the animal and how does the pet interact with this person (follows the person or is willing to be voluntarily separated from that person)

Describe the behavior. What does the pet do when alone? Videotaping the dog’s activity when alone can help to verify whether the pet appears anxious (panting, pacing, etc)

When does the behavior occur? Is the pet alone or does it perceive to be alone (while owner is sleeping or in the shower, for example). Or does the pet have full access to the owner when the behavior occurs.

Where does the behavior occur? Are the behaviors directed toward exit points or are there multiple locations vs. single locations in the home.

**Previous history**
- Age of onset and character of the behavior at onset
- Changes in the pet’s environment at onset such as a move, work schedule change, or loss of a house member
- Treatments attempted previously and outcome

**Medical differential diagnosis**

**Hypersalivation**
- Dental Disease
- Oral Foreign Body
- Oral Toxin
- GI Distress
- Medical Differential Diagnosis

**Vocalization - any condition resulting in pain**
- Otitis
• Osteoarthritis
• Dental Disease
• Severe Dermatitis
• Etc

Inappropriate elimination
• Lower Urinary Tract Disease
• Diabetes Mellitus
• Cushing’s Disease
• Renal Failure
• Colitis
• Inflammatory Bowel Disease

Behavior differential diagnosis

Hypersalivation
• Only known behavioral cause of hypersalivation is anxiety, most commonly separation anxiety

Vocalization
• Territorial Behavior
• Attention Seeking Behavior
• Hyperactivity
• Play Behavior
• Behavior Differential Diagnosis

Destructive behavior
• Normal Puppy Behavior
• Exploratory Behavior
• Food Acquisition Behavior

Inappropriate elimination
• Failure to Housetrain or Loss of Housetraining
• Marking Behavior

Co-morbidity
• High probability of dogs with noise phobia or thunderstorm phobia to also have separation anxiety
• If any of these conditions are present in a pet, carefully evaluate the animal for the other conditions

Treatment
The overall goals of treating separation anxiety are to reduce dependence on the owners.....

Attention seeking behavior
Owners should not respond in ANY way to the pet’s attempts to get attention from them by such behaviors as barking, whining, jumping up, pawing, etc. They should not look at, talk to or touch their dog at these times. Expect the behavior to initially get worse and more physical.

Departure and arrival routine
Have the owners ignore the dog for 30 minutes prior to leaving home. This is meant to prevent inadvertent reinforcement of anxious behavior as they prepare to leave.
  Ignore dog upon arrival until it is relaxed

Arrival routine
The owners should not interact with their dog when they arrive home until the pet is completely calm.

Distraction at departure
Use a Kong Toy stuffed with a treat, or some similar product, at the time of departure. This is meant to distract the dog away from the act of the owners departing from the home. The toy should be given approximately 5-10 minutes before departure.

Use of punishment
The owners should not use physical or verbal punishment in response to destructive behavior or elimination. These behaviors are symptoms of anxiety and punishment, especially after the fact, will increase the level of anxiety.

Uncoupling departure cues (habituation)
This refers to making a list of activities the owners perform prior to leaving home which signals to the pet that they are leaving and results in the dog getting more and more anxious. These activities are then performed at times when there is no intention of leaving the home.
Indoor relaxation exercises
Have the owners train the dog to assume a calm, relaxed behavior during gradually increasing periods of separation. This is commonly done when moving casually from room to room.

Graduated departure exercises
Have the owners train the dog to assume calm, relaxed behavior during gradually increasing periods of separation as they leave the home. They may need a “bridge” cue to signal “safe” departures.

Exercise
Consistent exercise in the form of walks and play can serve to reduce anxiety by decreasing the dog’s focus on the owner’s departure from the home.

Anti-anxiety medication
The judicious use of medication can decrease the overall level of anxiety and enable the pet to respond better to the behavioral tasks just outlined.

**Clomipramine**
- A Tricyclic Antidepressant (TCA) that functions primarily to elevate the levels of serotonin and norepinephrine in the synaptic cleft of brain neuropathways
- 1-4 mg/kg bid
- Allow at least 2-4 weeks for onset of action
- Expect sedation and anorexia as common side effects. Increased anxiety, aggression and hepatic disturbances are less common
- Preliminary CBC/Chemistry Profile and Thyroid Panel pre-treatment
- CBC/Chemistry Profile 4 weeks post-treatment
- Allow 2-3 months on the medication with the behavior being relatively normal
- Begin weaning by decreasing the dose by 25% every 3-4 weeks until off the medication or when symptoms return. Then return to the previously effective dose.

**Fluoxetine**
- Fluoxetine is a Selective Serotonin Reuptake Inhibitor (SSRI). Only has an effect on Serotonin and not on other neurotransmitters
- 1-2 mg/kg SID
- Allow at least 6-8 weeks for onset of action
- Expect sedation and anorexia as common side effects. Increased anxiety, aggression and hepatic disturbances are less common
- Preliminary CBC/Chemistry Profile and Thyroid Panel pre-treatment
- CBC/Chemistry Profile 4 weeks post-treatment
- Allow 2-3 months on the medication with the behavior being relatively normal
- Begin weaning by decreasing the dose by 25% every 4-6 weeks until off the medication or when symptoms return. Then return to the previously effective dose.

**Benzodiazepines**
- These are typically used in Separation Anxiety to treat panic behavior seen at time of departure to help ease the transition
- Diazepam (Valium)
- Alprazolam (Xanax)
- Clorazepate (Tranxene)
All have short onset, short half-lives and are used in conjunction with TCA’s and SSRI’s

**Trazodone**
- It is a serotonin agonist at 5HT1A receptor and a weak serotonin reuptake inhibitor.
- It is unclear which of these effects is responsible for the reduction in anxiety that occurs with its use.
- 1-3 mg/kg dose either as needed or up to 3 times per day
- Begin at the low end of the dose range for 3 days then increase dose gradually as needed
- Can be used along with an SSRI or TCA but use carefully to minimize possible side effects
- drowsiness, nausea/vomiting, headache and dry mouth, dizziness, constipation, urinary retention
- Hypotension, tachycardia, syncope, arrhythmias

Factors effecting outcome
- The older the patient at the time of onset or presentation, the poorer the prognosis
- Multiple diagnoses will decrease the prognosis
- The ability of the owners to follow through on recommendations
- The ability to administer medication and the patient’s response to that medication
- The living situation of the owners (neighbor complaints or degree of damage to the home)
Sibling Rivalry: When Roommates Come to Blows
John Ciribassi, DVM, DACVB
Chicagoland Veterinary Behavior Consultants

Risk factors

Household instability
- One or more dogs in household achieving social maturity (1-3 years)
- New pet or person added to home
- Illness in one or more pets in the home
- Pet returning from an absence
- History of one or more dogs in the home of having poor early socialization with dogs (genetics, early health issues, inadequate exposure)
- Anxiety related condition(s) in one or more dogs in the home (Separation Anxiety, Noise Phobia, CCD, General Anxiety, Fear Based Aggression, Conflict Aggression)
- Medical condition causing irritability (Otitis, Dermatitis, etc.)
- Deprived environment (fewer than ideal resource load; food, resting areas, owner interaction
- Same-sex pairs in the home.
- Most commonly females. Particularly in spayed females
- Young dogs being added to a household or dogs rehomed to a household are more likely to initiate fights

Typical history
- Often between two specific dogs even in a multiple dog household (>2 dogs in the home)
- Various stimuli
- Excitement in the home (greetings, passing through narrow openings, territorial barking, laughter or arguing in the home or running through the home)
- Resources (food, owner attention, toys, space) – recognize the relative value of the items to each individual dog in the household (Resource Guarding Potential)
- Hierarchy conflicts – behaviorally appropriate dogs are similarly motivated to maintain or acquire access to similar resources.
- Competition can be over one specific person in the home
- Owners undermine appropriate social structure between the dogs
- Aggressor may persist in attacks even if victim offers proper deferent signaling

Differential diagnosis
- Medical conditions
- Dominance Hierarchy – Resource Related
- Anxiety Related
- Redirected aggression
- Play Related Aggression

Differential diagnosis
Commonly seen with newly introduced housemates
- Fear Based Aggression
- Territorial Aggression

Typically increased social contact between housemates diminishes the likelihood of these interactions. However, socially inept dogs may show a reduced inability to adapt to prolonged exposure and continue to display behaviors more common with contact between unfamiliar dogs.

Medical conditions
Any condition which causes increased pain or irritability can increase the likelihood of an aggressive response between dogs
- Otitis Externa
- Osteoarthritis
- Dermatitis
Dominance hierarchy – Resource related
If there is equal motivation between dogs in a household over the acquisition or holding of a resource we can see an escalation of aggression between those individuals. Commonly a factor between intact males in the same household.

Equal opportunity and established possession testing

Anxiety related
- Behaviorally inappropriate dogs
- Do not adequately recognize normal signaling in other dogs (deference cues such as lip licking, yawning, turning away, moving away or exposure of underbelly, for example)
- Excessively reactive. More likely to target another dog in the home in situations characterized by high arousal (exposure to excitement stimuli)
- Can have poorly inhibited bites

It is critical to recognize, in these instances of aggression between dogs in the same household in which the attacker is socially inappropriate, the victim’s quality of life may suffer greatly. These dogs are doing everything they know how to diffuse the aggression and communicate deference or submission to the attacker but the attacks persist.

Stress escalates when the individual has minimal control over the outcome of a situation. This chronic stress results in continued activation of the Hypothalamic Pituitary Axis and thus prolonged cortisol exposure for the victim.

Redirected aggression
- The victim of the attack is the secondary target. The attacker cannot access the primary focus (another dog passing the home, for example, and then targets the other dog in the home which is more available.
- Can result in extreme fear in the victim, who can respond in a likewise aggressive manner thus escalating or maintaining the aggressive relationship between the dogs

Play based aggression
- Typically occurs between younger dogs
- Bites are usually inhibited so that significant injury does not occur
- Frequent reversal of roles during fights such that each dog will take turns showing dominant displays (mounting or biting over the dorsal aspect of the neck, for example)
- If excessive, can escalate to more serious encounters necessitating the owners to intervene

Fear based aggression
- Fearful animals may elect to utilize aggressive responses in order to manage or cope with stressful situations involving new dogs in a household
- May be initiated by the newcomer or the resident dog
- Depending on the age and experience of the fearful animal you may or may not see typical fearful signs (tail tucked, cowering, ears down and back, etc.) Dogs with a longer history of fear based aggression may have abandoned these postural strategies due to perceived ineffectiveness and now depend on aggression as a better coping response.

Territorial aggression
- Resident dog responds to newcomer by preventing access to valuable space.
- May be the home itself, certain areas of the home, the yard or valued sleeping areas.

Prognosis
- The likelihood of a successful outcome is good if both dogs are behaviorally appropriate, if resources can be identified, and the resources can be adequately managed.
- Prognosis is poor if one or both dogs are behaviorally inappropriate (anxiety or fear is a component of the behavior), particularly if response to medication is inadequate
- Prognosis is also poor if aggression occurs immediately whenever dogs come into sight of one another…..

Diagnostic evaluation
- Physical Exam
- Neurologic Exam
- CBC, Chemistry Profile and Thyroid Screen
- Further labs as indicated by basic work up

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Questions
- Household composition
- When aggression began
- Frequency
- How are resources managed between the dogs
- How do dogs interact outside of aggressive episodes
- How do fights occur. Give examples from most recent to previous fights as well as description of earliest fights.
- How do the fights resolve
- Are there injuries

The most important question is which dog, if any, is acting appropriately in the interactions. In this way, the attention can be centered on the correct dog. That may be changing the response of the dog acting inappropriately in the relationship or, if both dogs are appropriate, managing the resources in the household.

Treatment
- Manage resources (food, toys and attention) – “dogs are not best thought of as a pack in a home environment. They are best thought of as roommates who need to learn to share”
- Identify all situations which trigger aggression and avoid these triggers or separate the dogs at these times
- Safety
- Provide owners with means to break up fights (head collars with drag leashes, blankets, air horns, water, instruct in removing dog by pulling on rear legs)
- Isolate pets when unsupervised
- Address triggers (food, toys, resting areas, access to owners)
- Feed dogs separately
- Do not leave toys out but apportion them as needed
- Deny access to elevated surfaces and have dogs resting remotely away from owners (on mats or dog beds, for example)
- Basket Muzzles

These can be used whenever there is a higher likelihood of aggression between the dogs where the owners are not as likely to be able to quickly intervene. Can result in increased comfort for the owner in knowing the dogs are at least safe from severe injury.
- Separation with gates or tethers
- Used when dogs cannot be closely supervised
- NILIF or "SIT" protocol
- Goal here is to increase the dog’s attention to the owner for direction
- Regular periods of basic training (clicker training)

By increasing the dog’s level of responsiveness it allows the owner better ability to direct their dog’s behavior and therefore having them show less focus on each other. A good recall is important in that it gives the owner the ability to call the dogs away in potentially problematic situations.
- Have owners ignore BOTH dogs if owner attention is causing hierarchy issues between the dogs

The goal here is to reduce the value of the owner as a resource for either dog. Increased owner attention to either dog (as opposed to trying to figure out which dog is higher ranking with respect to this particular resource) can escalate the owner’s value and thus increase conflict and also elevate emotionality in the home (problematic for the behaviorally inappropriate dog).
- Support higher ranking dog?

There are several problems with this approach
- Difficulty for owners to identify accurately
- Owners may be reluctant to demote an older, favored dog
- Dogs who are behaviorally inappropriate may not be signaling correctly and thus owners red these dogs incorrectly thus favoring a dog who is showing aggression at the wrong times and putting the victim in a difficult situation
- The aggression in the household may not involve hierarchy at all

Response substitution (operant counter conditioning)
- This involves interrupting the dog and then redirecting to more appropriate sets of behaviors (that the owners have been rehearsing with the dog on a regular basis in non-distracting situations) and reinforcing those behaviors.
- Does not reinforce the aggression since the dog is being relocated and not reinforced until it complies with a request to perform an alternate behavior. We are conditioning a behavior that is counter to the problem behavior.
**Counter conditioning and desensitization to graded triggers such as sounds in the environment**

If there are triggers which can be identified as causes of the aggression, and the intensity of these triggers can be adjusted, the owners can gradually expose the dog(s) to the trigger at slowly increasing levels (desensitization) while asking the dog to perform more appropriate competing behaviors (counter conditioning).

**Example: Door bell triggering excessive greetings and resulting aggression.**

**Reintroduction**

In some cases dogs have to be separated for an extended time while owners work on getting consistent responses from each dog separately and each dog learns it will receive positive rewards for attending to the owner. This would be needed if the dog’s cannot be in each other’s company without immediately reacting.

Once each dog is responding well separately from each other, then they can be reintroduced on walks. First at a comfortable distance while going through training individually then gradually decreasing the distance between them as they adjust.

**Treatment**

If treatment proves to be unsuccessful, other options include:
- Rehoming
- Permanent Separation of the dogs
- Euthanasia (particularly if one of the dogs is behaviorally inappropriate)

**Should dogs “fight it out”?**

In one study, 42% of dog fights did not require intervention to break them up.

However, if there is a history of injury to either of the dogs involved in fighting, it would be inappropriate to allow them to continue to fight without intervening. The injuries demonstrate that the dogs have been unable to arrive at a mutually beneficial agreement over partitioning or resources. If the fights are motivated by fear or anxiety in behaviorally inappropriate dogs, they will be incapable of regulating the level of violence and injuries are likely.

In these cases, owners need to learn how to safely break up fights

**Options in breaking up dog fights**
- Wheelbarrow the attacker by picking up the rear legs and lifting while moving back and to the side
- Compressed air or citronella
- Water
- Sudden noises such as with pot lids
- Board to wedge between the dogs
- Blankets or cushions
- Leashes attached to both dogs (with or without a head halter)

**Drug therapy**
- ONLY if one or both dogs are abnormal in terms of fear/anxiety
- SSRI (05-2.0 mg/kg SID)
- Fluoxetine
- Sertraline
- Paroxetine
- Selegiline if Canine Cognitive Dysfunction (1 mg/kg SID)
- As Needed Options
- Clonidine (0.01-0.05 mg/kg 1-2 hours before needed or up to tid)
- Trazodone (3-5 mg/kg 1 hour before needed up to tid)
- Benzodiazepines (not indicated in fear based aggression due to the possibility of disinhibition).

**Pre-treatment blood work**
- CBC/Chemistry profile/Thyroid profile

**Post-treatment blood work (4-8 weeks post onset of therapy)**
- CBC/Chemistry profile
  - Pheromones (Adaptil)
  - Neutraceuticals such as Anxitane

**Surgery (if hierarchy related)**
- Castration

**Client education**
- Discuss canine body posturing and communication methods
- Regular communication with client to enable adjustment of treatment plan

**Prevention**
- Add dogs to home of different genders and ages
- Regulate access to resources
- Castration to help prevent intermale aggression
- Proper socialization
- Puppies stay with litter until about 8 weeks of age
- Socialization classes between 8-14 weeks of age and reward based obedience class at around 4-6 months of age
Aggression is one of the most common complaints presented to veterinary behaviorists. According to the Centers for Disease Control, approximately 4.5 million people are bitten by dogs each year in the United States. One study has indicated that approximately 41% of dogs had growled, snarled or snapped at a familiar person at some time in the dog’s life.

Classification and labeling of aggression is in a continuous state of flux as behaviorists continue to understand the underlying motivation and emotional states involved in aggressive encounters. Because canine communication occurs along a continuum, which includes aggressive displays and responses, understanding and treating aggression in dogs can be misunderstood and mismanaged. Although the labels for the aggressive response may differ within the behavioral community, the treatment often results in improvement and safe management.

In order to create an appropriate treatment program for a patient, it is important to identify the type of aggression through identification of whom the aggression is directed toward and what the underlying motivation and/or situation is. It is best to first identify whether the aggression is human-directed or animal-directed. Then determining an accurate diagnosis will depend on the circumstance, the body language of the patient and the motivation. Motivations for an aggressive response may include play, fear, pain, protective, territorial, resource-related, predatory, lack of impulse control, conflict-related and/or redirected response.

Diagnosis should always include a thorough medical evaluation as well as a behavioral evaluation. A behavioral evaluation is enhanced with direct observation of the behavior, but this is not always feasible due to safety considerations. Utilizing an aggression screen and the behavioral information from the history (vocalization, posture, context, and target of the aggression) will lead to a diagnosis. It is also important to remember that pets are complex creatures and often will have multiple diagnoses.

Counseling owners on aggression requires a solid knowledge base of both normal and abnormal behavior, basic learning principles and a thorough understanding of psychotropic medications. Liability concerns should be addressed when working with clients with an aggressive patient, including but not limited to utilizing informed consent forms.

The goals of counseling an owner with an aggressive animal are to obtain an accurate history, identify the motivation for the behavior, determine a prognosis for the likelihood of future aggressive events and educate the owner on safe management of the patient. Prognosis may be based on six factors: 1. The ability/willingness of the owner to modify the situation; 2. The ability/willingness of the owner to modify their own behavior; 3. The size/strength of the patient; 4. The severity of bites/aggressive events; 5. The underlying motivation of the patient and 6. The predictability of the aggression.

Basic tools for treatment of canine aggression identification of motivation and triggers, behavior modification techniques, environmental modification techniques (including modifications for safety), meticulous record keeping (including teaching owners to journal), +/- surgery, +/- medication and thorough follow-up.

The above principles will be discussed and demonstrated through case discussions.

References available upon request.
In an era where people have demanding schedules and desire quick fixes, many clients come to their veterinarian for a pill to “cure” their pet’s behavior problem. For many patients, use of a medication as part of the behavioral treatment plan is essential. As more behavioral drugs become available to the veterinarian, it can be difficult to discern which medication is appropriate for usage in a particular patient. This seminar will give a very brief introduction to behavioral pharmacotherapy. In order to have a thorough understanding of psychopharmacology, it is best to pursue in-depth continuing education.

Appropriate use of behavioral medications requires: 1. an appropriate diagnosis and 2. an understanding of the medication’s effects, onset to efficacy, side effects and interactions with other medications. The goals of using psychotropic medications are multifold: 1. to decrease emotional reactivity when the stimulus cannot be controlled; 2. to potentiate behavioral therapy (decrease time to improvement); 3. as synergism with behavioral therapy (to obtain an increased level of improvement); and/or 4. to treat a pathology responsible for behavior that requires pharmacological intervention. Studies have shown that using behavioral medications can lead to a quicker outcome and a higher level of improvement.

Many drugs used in veterinary behavior have limited controlled studies. Veterinarians are often required to make decisions on application of behavioral drugs without the benefit of scientific evaluation for a particular indication or a label for the condition. Many drugs have been transferred from usage in the human psychiatric community but may have different side effects and toxicities than humans, some of which are not yet known. Some behavioral drugs also have potential for human abuse. State laws also vary regarding requirements for usage of controlled drugs.

It is important to remember that the brain and all synapses are in a dynamic state. The actual response that occurs is a result of an infinite number of factors in the individual brain. Therefore, the response of a behavioral drug may not necessarily be the expected effect of the drug. The existing chemical and physiologic state of the neuron and synapse as well as the resting tone of the neurotransmitter system will influence the ultimate response to the drug.

Because few psychotherapeutic drugs are labeled for use in veterinary medicine, most behavioral drugs are extra-label use. Requirements for extra-label use from the Animal Medicinal Drug Use Clarification Act 1994 (AMDUCA) include:

- Establishment of a valid client-veterinarian-patient relationship
- Behavioral history must be taken
- Veterinarian must establish diagnosis
- Veterinarian must keep up with literature
- Owner must be informed of extra-label use
- Owner should sign informed consent
- Record must be kept of drug, condition treated, species of animal treated, dosage and duration

The selection of a drug requires accurate diagnosis of the behavior problem and knowledge of the existing data regarding use of that behavioral drug for a particular diagnosis. It is recommended that selection of the drug include consideration of whether a drug is labeled for that condition. First choice should be a drug labeled for that condition in that species (i.e. clomipramine or fluoxetine for separation anxiety in dogs). If there is no behavioral drug labeled for that condition in that species (i.e. no behavioral drugs are labeled for usage in cats, birds, etc.), then the second choice should be a drug labeled for that condition in another species. Third choice should involve a drug labeled for the condition in humans. Selection of a particular drug should also include assessment of the animal’s health (consider contraindications for health status or concurrent medications), the cost of medication, owner compliance, (dosing frequency, mode of administration, bitterness of drug), expected time to efficacy, side effect profile and your experience with that particular drug.

A medical assessment is recommended (in addition to the behavioral history) prior to prescribing a behavioral drug. Since many of the behavioral drugs cannot be stopped “cold turkey” without significant side effects, it is important to know the metabolic status of the individual prior to starting the medication. The minimum database recommended prior to starting a medication is a complete blood count, chemistry profile, +/- thyroid profile, and +/- urinalysis.

It is imperative that follow-up be conducted regarding efficacy of the medication and health of the patient. Many clients do not understand that it can take weeks for the medications to have effect. Additionally, the dosage may need to be adjusted in order to achieve full efficacy or reduce side effects. A typical recheck schedule may involve:

- 1-2 weeks (by phone, e-mail or in-office): Discuss side effects, if any. If fast-acting medication, evaluate efficacy. If some benefit, may increase dosage. Review behavior modification recommendations.
- 4-6 week (in office): Evaluate efficacy of slow onset medication. Change dosage if necessary. Review behavior modification recommendations.
- 10-12 week (in-office): Assess efficacy of full behavior treatment plan.
- 6-9 months (in-office): Evaluate possibility of weaning off medication (if feasible).

Some patients may be difficult to medicate either secondary to aggressive behavior or because of fearful or unruly behaviors. Compounding behavioral drugs into palatable treats is often recommended to increase compliance. Transdermal formulations of behavioral drugs have not been shown to be effective in behavioral treatment. Blood levels of behavioral drugs administered transdermally can be significantly lower than expected or produce unpredictable levels due to application. Slow-release forms that have been formulated for humans can also vary according to species and may give unpredictable blood levels as well.

When selecting a behavioral drug for usage with a behavioral disorder, the first decision must be whether an immediate-acting drug (such as a benzodiazepine) is indicated and/or a maintenance drug (such as a TCA or SSRI) will be beneficial. Behavioral drugs affect neurotransmitters at presynaptic sites, postsynaptic sites or within the synapse itself. Tri-cyclic Antidepressants (TCAs) are inhibitors of neurotransmitters serotonin and norepinephrine. They also have antihistaminic effects, anticholinergic effects and are α-1 adrenergic antagonists. The effects of TCAs vary by drug, with clomipramine being the most serotonergic of the TCAs typically used in veterinary medicine. Most of the TCAs have a very bitter taste and can be difficult to medicate. All TCAs have a slow onset of action (2-4 week latency to behavioral effect). The TCAs can be given once or twice daily. TCAs are metabolized in the liver and clearance occurs through the kidneys. TCAs need to be stopped with gradual withdrawal.

Indications for usage of TCAs can include anxiety, fears and phobias, compulsive disorders, affective aggression (TCAs that have a serotonergic effect), urine marking, and depression. Side effects of TCAs vary between drugs, but can include sedation, constipation, diarrhea, tachycardia, cardiac arrhythmias, blood pressure changes, interference with memory, anxiety, restlessness/agitation, sleep disorders, fatigue, headache, ataxia, urinary retention and/or lowered seizure threshold. TCAs should not be used in combination with MAOIs or sympathomimetics. Examples of TCAs commonly used in veterinary behavior include clomipramine, doxepin, amitryptiline and imipramine.

SSRIs (Selective Serotonin Reuptake Inhibitors) inhibit serotonin reuptake at the presynaptic site allowing serotonin molecules to act for longer periods of time. SSRIs can also have a slow onset of action (1-4 week latency to effect). SSRIs are metabolized through the liver and excreted through the kidneys. Most SSRIs have a long half-life.

Indications for usage of SSRIs are anxiety disorders, compulsive disorders, fears and phobias, urine marking and/or aggression. Side effects of SSRIs are fewer than TCAs and can include: sedation, gastrointestinal (such as anorexia, nausea and diarrhea), anxiety, agitation, insomnia, aggression, tremors, mania and seizures (rare). SSRIs should not be used with MAOIs or cimetidine. When combining an SSRI with another behavioral drug, caution should be used. SSRIs can be used cautiously at lower doses in combination with TCAs, buspiron and tryptophan, but combinations of serotonergic drugs can result in serotonin syndrome. SSRIs are also competitive inhibitors of several cytochrome P450 enzymes and may result in elevated levels of SSRIs or another medication if those pathways are used concurrently. SSRIs may also alter the metabolism of benzodiazepines. SSRIs commonly used in veterinary behavior include: fluoxetine, paroxetine, sertraline, fluvoxamine and citalopram.

Trazodone (a Serotonin 2A Antagonist and Reuptake Inhibitor - SARI) has been a more recent addition to veterinary medicine. Trazodone has been used to treat anxiety and depression in humans and as well as an aid in sleep. Indicates for usage include post-surgery confinement, hospitalization, anxiety as a solo agent and in combination with other psychotropic medications.

Many patients benefit from a combination of medications. Combining medications requires a thorough understanding of the individual medication, the potential for increased side effects and changes in metabolism and the risk for serotonin syndrome. Serotonin syndrome is a potential complication of serotonergic drugs. It is a rare, life-threatening complication that can occur when central 5-hydroxytryptophan (5-HT) receptors are overactivated. Serotonin syndrome can be a result of inappropriately combining medications or overdosing a particular serotonergic drug. The result is mental and neuromuscular changes or death (mortality rate in humans is 11%). Symptoms of serotonin syndrome may include tachycardia, tachypnea, agitation, anorexia, hyperpyrexia, hypertension, diarrhea and/or seizures. The only treatment for serotonin syndrome is symptomatic treatment.

References available upon request.
Many veterinary staff avoid discussing behavioral issues with clients because they are unfamiliar with the terminology of learning theory. A basic understanding of learning theory is imperative for working effectively with animals with and without behavioral issues. Every interaction that occurs with an animal results in learning. It is important that veterinary professionals understand the science behind how animals learn (both deliberately and “accidentally”) and that we understand how to teach them new information.

Learning is a process whereby an organism is changed. It can be seen as an external behavior or may be a change in an internal process. In animals, we typically monitor learning by behavior. The process of learning depends on the animal’s perception, the animal’s memory and the animal’s ability to categorize events as similar or dissimilar to previous events. The animal’s behavioral response (or evidence of learning) depends on the animal’s physical ability to perform the response, the animal’s motivation, the animal’s opportunity for response and whether learning truly occurred.

**Learning terminology and techniques**

**Classical conditioning**
Classical conditioning is the pairing of a neutral stimulus (that has no pre-existing meaning for the animal) with another stimulus, which results in a learned (conditioned) response. These can be either positive or negative results. The most recognized example of classical conditioning is the salivary reflex in Pavlov’s dogs. Pavlov’s dogs began to associate the arrival of food with various unconditioned stimuli (such as bells ringing) and began to salivate with the ringing of the bell.

**Operant conditioning**
Operant conditioning involves learning that a consequence (positive or negative) occurs with an action. In this situation, the animal causes the results—what the animal does is critical to what happens next. The likelihood of a behavior increases if it is reinforced or decreases if it is punished (see above).

It is important to remember that dogs and cats are learning all the time—intentionally or unintentionally. In the real world of dog behavior, classical and operant learning can occur simultaneously. For example, even though your dog associates the sound of the can opener with food and begins to salivate at the sound of it (classical conditioning), he is then rewarded for coming to the food bowl with the food (operant conditioning).

**Reinforcement**
A reinforcer is a “reward”. Reward implies that it is good and that both the giver and the receiver consider it a reward. If this is true, reinforcing a behavior will increase the likelihood of its occurrence.

**Positive reinforcement (+R)**
- This involves adding something the animal likes to increase the likelihood that the behavior will recur.
- Example: You say “sit”, your dog sits” and you give him a treat. The treat serves to increase the likelihood of the response in the future.

**Negative reinforcement (-R)**
- This involves the removal of something unpleasant when a behavior is performed which increases the likelihood that the behavior will recur.
- Example: You ask your dog to “sit” while pushing on his rump, your dog sits, and you stop pushing on his rump. This will also increase the likelihood of the behavior occurring in the future.

**Punishment**
Punishment will result in a behavior occurrence decreasing with the presentation or removal of something undesirable. Unfortunately, punishment is often not accurately or effectively used by humans and can cause an increase in anxiety, fear and aggression.

**Positive punishment (+P)**
- This involves the presentation of something undesirable (negative) when a behavior is performed. Because the consequence is negative, the behavior will decrease. +P must occur within seconds of the behavior to be effective. +P is rarely used in treatment of behavior disorders in animals because: 1. Humans are very poor at timing +P; 2. Anxiety or fear may be associated with the individual delivering the +P; 3. +P does not provide an alternative behavior for the animal; and 4. +P may increase aggressive behavior.
- Example: Dog jumps on you, you kneel him in the chest. (Note: this is not recommended because many dogs who are punished learn to fear the punisher or shut down in training – see above).

**Negative punishment (-P)**
- This involves the removal of something good as a consequence for the behavior, which causes the behavior to decrease.
- **Example:** Puppy is playing too roughly, owner stands up, leaves the area and stops play; puppy’s rough playing therefore decreases.

**Desensitization (DS)**
Systematic desensitization involves gradually exposing an animal to a stimulus at a low level so that it does not evoke an undesirable response. The goal is to work on replacing an undesirable response with a more appropriate response (such as a relaxed, calm response rather than an anxious, fearful or excitable response). Note that this is a systematic exposure, starting at a low level and slowly increasing the level of exposure over many learning sessions.

**Flooding**
Flooding is used in some situations to treat low level fears by exposing the individual continuously to the stimuli that causes them anxiety until the fear is extinguished. This is not typically used with animals because it may worsen the condition rather than improving it in some circumstances.

**Counterconditioning (CC)**
Counterconditioning is often used in conjunction with desensitization (DS/CC). This involves teaching the animal a response that is incompatible with the response previously given in a situation. For example, you may teach a dog to stop barking at the door using DS/CC by using a low-level doorbell stimulus (the sound muted until the dog does not respond) while teaching him to sit in a calm manner when the sound occurs. Systematically, over many sessions, the intensity of the doorbell stimulus is increased.

**Generalization**
Generalization occurs when animals begin to transfer the information they have learned in a particular setting to other settings. This often requires training in multiple setting with varying degrees of distraction before generalization begins to occur.

**Extinction**
Extinction occurs when a reinforcer is withheld from an animal. Over time this will lead to the elimination of the behavior. It should be noted that many animals will exhibit an “extinction burst” following removal of the reinforcer, in which case the behavior will intensify as the animal tries harder to get the reward before the behavior extinguishes.

References available upon request.
Incorporating treatment of behavior problems into a busy veterinary practice is often avoided due to a lack of time or knowledge. Behavioral treatment can be done efficiently with preparation, a strategic plan and appropriate usage of the full veterinary team.

The initial approach to a behavioral issue with a client/patient should be equivalent to the triage of a medical crisis. The client will typically contact either the receptionist or the technician with the problem or may casually mention the problem during a routine visit. When receiving this information, the staff member should initially determine whether the client is in crisis; all staff members should be trained to recognize the signs of a behavioral crisis. Although many behavior problems develop over time and gradually worsen, some behavior issues may be critically urgent when the client brings them to the attention of the clinician.

Typical signs of a behavioral crisis may include injury to people (such as a bite), injury to other animals (such as a fight between dogs in the home) or injury to the patient (such as a severe storm phobic who has jumped through a window). An additional sign of a crisis is a client who is discussing possible relinquishment or euthanasia. Critical situations should be scheduled for an immediate appointment with the veterinarian to determine the appropriate approach to the problem. It may be warranted to board the patient at the hospital for 24 or more hours in order to allow the client relief from the risk of physical harm and/or allow the client relief from mental or emotional stress.

Once the situation has been triaged, the veterinarian can begin to differentiate whether a primary medical diagnosis has resulted in the problem behavior or a primary behavioral diagnosis is responsible for the crisis. This may involve a series of diagnostics to rule out medical issues prior to addressing the behavioral problem. A minimum data base for many behavior problems includes a thorough and rigorous physical examination, including an evaluation for neurological function and sources of pain, as well as a full metabolic screen. Once the medical factors have been ruled out, it can be determined whether the patient needs an in-clinic behavioral evaluation, needs to be referred to a qualified professional trainer or technician or needs to be referred to a qualified behaviorist (a DACVB or CAAB). This may be determined through information gathered in an interview with the client or may be more efficiently gathered in a written history form completed by the client.

If a behavior evaluation is scheduled with the clinician at your hospital, the consultation should be scheduled to allow adequate time for evaluation of the problem. The charges should also be consistent with the time necessary to diagnose the behavioral condition and create a treatment plan. A diagnosis is achieved through meticulous history taking and observation of the pet and its interaction with the owner(s). It is common to have multiple behavioral diagnoses when working with a behavior patient; patients rarely present with a single behavioral complaint.

Once the problem(s) have been diagnosed, they may then be addressed with a repertoire of tools used in treatment of behavioral diseases in animals. Typically, a combination of behavioral modification, environmental modification and +/- pharmacological intervention is used in the animal’s treatment program. Veterinarians in private practice often shy away from behavioral medicine because of time restraints. A qualified veterinary technician can be an integral part of the process in history taking, demonstrating behavior modification techniques and usage of behavioral tools as well as implementation of the treatment plan and follow-up.

In order to be an effective behavior team member, the veterinary behavior technician must have appropriate knowledge of learning theory, training techniques and tools, and commonly prescribed behavioral medications. The successful veterinary behavior technician must also be an empathetic listener, as many behavioral issues can be emotionally exhausting for clients. The veterinary behavior technician must also be able to communicate well with the veterinarian to ensure that the treatment plan is consistent with the veterinarian’s recommendations.

Many veterinary staff avoid discussing behavioral issues with clients because they are unfamiliar with the terminology of learning theory. A basic understanding of learning theory is imperative for working effectively with animals with and without behavioral issues. Every interaction that occurs with an animal results in learning. It is important that veterinary professionals understand the science behind how animals learn (both deliberately and “accidentally”) and that they understand how to teach them new information. This will enable veterinary professionals the ability to create plans to change undesirable behaviors to preferred behaviors and to understand why behavioral treatment plans work/do not work.

Owner compliance with a behavior treatment plan is improved when the client is given written instructions, receives follow-up from the clinician/technician and understands an appropriate time line for improvement. A treatment plan is a fluid and changing model that should be adjusted as the patient begins to respond to treatment. Some behavioral issues may improve slowly while others are quickly and easily managed. It may be important to help the client realize that treatment of behavior problems are often seen as being “managed” rather than “cured”. Access to a support system through the veterinary staff enables clients to more easily handle the chronic nature of managing a behavioral patient.

References available upon request.
A significant number of dogs which are surrendered to a shelter each year are relinquished due to behaviors that can be classified as “unruly” (such as jumping up, mouthing, vocalizations, destructive behaviors and nocturnal activity). Unruly behaviors can be frustrating both to the owners and to the veterinarians who attempt to efficiently help clients with these issues. Medical issues should always be eliminated as possible causes of unruly behaviors before diagnosis of a behavioral problem. Understanding how to manage these behaviors will benefit both veterinarians and clients.

The initial behavioral approach to many unruly behaviors should include an evaluation of the current daily budget for the pet. This includes an evaluation of the provided opportunities for exercise and cognitive enrichment. Additionally, an evaluation of the structure and predictability of the daily schedule for the pet may help illuminate areas where undesirable behaviors may be inadvertently rewarded. Often the approach to unruly behaviors is focused on the inappropriate behavior or treating the behavior as a “dominance issue” when a better approach may be to satisfy the pet’s needs physically and psychologically while focusing on a desired behavior as a replacement behavior.

Jumping-up in canines is a normal behavior that can occur during play or greeting. In a large breed dog, it can be a serious issue and result in injuries to owners and visitors. There are many techniques that pet owners have often tried prior to seeking help; some of these techniques may have accidentally rewarded the jumping behavior. Appropriate management may involve use of a head halter, withdrawal of attention (both verbal and physical) and/or response substitution.

Mouthing in canines can occur during play, during greeting and possibly during manipulation of the dog (such as during grooming or while fastening on a leash). Management of mouthing behaviors may involve desensitization and counterconditioning to handling and manipulation, withdrawal of attention and redirection to an alternative oral behavior.

Excessive barking in canines can be a crisis for some owners. Barking can be motivated by many different factors such as territorial issues, excitement, attention-seeking, food-soliciting, conflict, fear, play, group-facilitation, separation anxiety, cognitive decline and/or reinforcement. Management of barking involves identifying the trigger and/or motivation and removing the trigger, if possible. Response substitution and extinction techniques may be used, as well as training tools such as citronella anti-bark collars.

Destructive chewing may be a normal behavior for many puppies and adolescent dogs. It may also be a result of exploration, play, scavenging, hunger or attempts to escape. Separation anxiety or noise phobias should be ruled out as potential differentials. Management may involve ensuring the dog has adequate opportunities for chewing, adequate exercise, “dog proofing” the home and/or confining the dog. Medical causes may also be a contributing factor in some dogs, often geriatric.

Nocturnal activity in cats is often a behavioral problem in young, energetic cats, but may also be an issue in geriatric cats with cognitive dysfunction. Management of young cats involves active play during the day with moving objects, preventing access to sleeping areas during the day and possibly providing a playmate.

Excessive vocalization may be a problem in many oriental breeds of cats, but may also be a result of attention-seeking behavior, hunger, territorial arousal and/or cognitive dysfunction. Management may involve identifying the owner’s response and removing reinforcement of the behavior in many situations. Additionally, rewarding appropriate quiet behavior is often beneficial.

Scratching is a normal feline behavior which can be destructive to the home environment. It is typically a social message (both visual and chemical) which is displayed by five weeks of age. Management involves providing a scratching area that is desirable to the cat. The scratching area should preferably be at least 30 cm off the ground, be taller than the cat when he/she stands on the hind legs, should be located next to the sleeping area and/or an area which has previously appealed to the cat. A texture that the cat finds attractive (such as sisal or jute) should be used. A kitten or cat can be trained to the area.

References available upon request.
An essential component of behavior consultations includes gathering information from the client, engaging the client in the treatment plan, and ensuring that the client is making progress toward a goal throughout the patient’s treatment. Many clients are apprehensive when entering into behavioral treatment for their pet as the unknown can be discomforting. Training of veterinarians often concentrates on the interaction with and the comfort of the pet while ignoring the human-human interactions. The creation and implementation of a treatment plan for a behavioral patient is only effective if the plan is followed through. The human component of the treatment plan is essential and is dependent upon the client’s willingness for compliance. Clients arrive at a behavioral consultation with a goal in mind. Sometimes the goal is vague, such as “I would love my dog to behave better” or “I want my dog to stop destroying my house.” Often the goal is additionally steeped in emotion. Individuals often select a goal based on the perceived outcome rather than the actions that are necessary to achieve the goal. The task of the veterinarian is to define and refine the goal so that it is achievable.

Motivation can be defined as the desire to perform voluntary actions to achieve a desired outcome. Motivation is dependent on both the difficulty of the task and the perceived reward and has been shown to be highest with a combination of identified goals and feedback. When a client is considering how to spend their time and energy, the outcome of that decision is affected by the concept of a goal. A goal may be an experience, an end-state, or an outcome. Goal setting requires identification of both delayed (Distal Goals) and immediate (Proximal Goals or Subgoals). Subgoals provide an immediate source of information regarding progress toward the distal or ultimate goal. Completion of subgoals provides information regarding whether the ultimate goal is appropriate, may supply feedback that provides motivation, and/or may influence the individual’s sense of self.

Research in the field of human motivation has identified a number of reasons why individuals are able to achieve goals. Highly competent people tend to set difficult but attainable goals, create subgoals as steps to their ultimate goal, structure their environment for success, self-evaluate to determine where they are in the process of achieving the goal and learn how to create positive outcomes. Goals are most effective when they are specific, as this allows the individual to identify whether the goal is being achieved and allows for appropriate feedback to occur. Goal-specificity results in increased planning and increased effort from the individual. Precision of how and when the subgoals will be achieved increases the probability of success. For example, outlining a subgoal as “train your pet for 10 minutes once daily in the evening after dinner” allows a client to identify whether or not they are achieving success as opposed to “work on training when you can.”

Highly competent people also identify task strategies that help them achieve a goal. This may include “if-then” plans (known as implementation intentions). In the example of an aggressive pet in the home, creating an if-then plan with the owner will allow them to have a safety management plan in place for situations in which aggression may occur. Evidence indicates that up to 95% of goal-directed behavior can become and often is unconscious or automatic; therefore, establishing the automatic response through structured techniques will help create appropriate automatic behaviors. Simulations are effective in helping individuals prepare an automatic response in a situation in which the behavior needs to be quick and effortless. Airplane pilots utilize simulations to become proficient at handling emergencies and automatize their response. Simulations may be beneficial with behavior clients and patients and can be implemented with models.

Emotions can interfere with both setting goals and achieving goals. When working with a family during a behavior consultation and implementation of a treatment plan, different emotions experienced by each family member contribute to the success or failure of the plan. For example, in a home where there has been a bite to a family member, the victim may be experiencing fear while at the same time is still highly bonded to the pet. Another family member may be angry at the pet and want immediate removal of the pet from the home. This allows the goal to be “framed” (i.e. changes the perspective from which the goal is viewed). Identifying the emotional influence on goal achievement involves accurate reading of nonverbal information (such as body language and eye contact) and engagement of all family members in the consultation.

Goal shielding is a technique that prevents other goals or behaviors from interfering with the desired goal. This may occur through creation of operant thoughts (such as cognitive maps, scripts and plans). Operant thoughts are mental attempts to try out different strategies to problem solve. A cognitive map is a visual or graphical representation of the individual’s belief system. Creation of a cognitive map may be assisted through use of interview techniques. Open-ended questions and closed-ended questions will move individuals in varying directions while creating their cognitive map. The technique of motivational interviewing provides guidance on interactions with clients to empower the client while making them an active participant in the goal-setting and achieving process. Empowerment can occur through creation of scripts (creation of memories of behavioral sequences necessary to complete an activity) and plans for goal achievement.
Individuals who do not achieve goals may be thwarted from achievement by a fear of failure or ridicule or regret that the individual may feel if he/she does not achieve the goal. The client’s motivation in this situation is to protect their self-esteem rather than achieve the goal. Empathic listening by the veterinarian will allow the client to express and address their fears and concerns. Often clients feel they have a lack of guidance or feedback to help them achieve a goal. For some clients, the change in their behavior (or their pet’s behavior) does not appear important or necessary and/or they are frozen in ambivalence (if the list of pros for change equals the list of cons). A lack of ability is rarely the cause of individuals not achieving goals.

Studies have shown that individuals who are told “do your best” do no better achieving a goal than if they had no goal at all. Whether we are working with a client managing a disease (such as treatment of a diabetic patient) or working with a behavior patient, helping clients create a functional plan is essential to the success of veterinary treatment.

References available upon request.
Adapt, Improvise, and Overcome: Interesting Large Animal Ruminant Cases
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This presentation will review some unique and challenging cases. These cases include the following: a case of albendazole toxicity in alpacas, spastic paresis in a pygmy goat, a few cases of severe preputial stricture in bulls, and other cases of reproductive failure in bulls.

Albendazole toxicity in alpacas
Three crias presented for fevers of 24 to 48 hours duration. The crias were a 2 month old female, a 6 months old male, and a 7 month old male. The fever was ~105-107°F. The owner administered flunixin meglumine and oxytetracycline prior to presentation and had a history of lethargy and diarrhea. Physical examinations of the three crias revealed that the crias were depressed with harsh lungs sounds; all crias were febrile and had diarrhea. Diagnostics performed included complete blood counts (CBC), chemistry profile, fibrinogen, fecal examinations, BVDV testing, and +/- fecal cultures. The female cria was positive for tapeworms while the 2 male crias were negative for parasites. All crias were negative for BVDV via PCR from buffy coat, and fecal cultures were negative, as well. Complete blood counts revealed severe leukopenia, specifically neutropenia, in all crias with WBC counts of 4200, 2030, and 180; serial CBCs showed a regenerative response. Treatments for the crias consisted of florfenicol antibiotic (n=3), praziquantel anthelmintic (n=1), IV fluids (n=1), flunixin meglumine (n=2), and Filgrastim (n=1). Filgrastim is a human granulocyte colony-stimulating factor (G-CSF) that acts on hematopoietic cells to stimulate proliferation and differentiation. In the one cria that received Filgrastim, the WBC count increased from 2030 to 6910 following 2 days of treatment (one vial = $240). One animal died soon after presentation, and the remaining 2 animals were discharged with no explanation for the severe neutropenia. After speaking to the owner (for the fourth time), he “remembered” giving albendazole and numerous other anthelmintics to his young crias; albendazole was given >5 times the label dose for at least 5 days in most cases. Albendazole toxicity causes bone marrow hypoplasia with 9 previous cases reported in alpacas (Gruntman, 2006). Three to four months following administration of the toxic doses of albendazole, the affected alpacas began to “blow their fiber” with large foci of alopecia.

Spastic paresis in a pygmy goat
Spastic paresis is rare, but has been reported in 3 pygmy goats between the ages of 1-2 years (Baker et al, 1989) and in Czechoslovakian in a Saanen goat in 1973. Clinical diagnosis is based on physical examination. Successful treatment of spastic paresis in these goats included tibial neurectomy (n=4) and desafferentation of the dorsal spinal roots (n=1). The goat that presented underwent bilateral, tibial neurectomy. Tibial neurectomy was performed by dissecting between both parts of the biceps femoris m. and identifying the tibial nerve. Successful identification of the tibial nerve is confirmed with electrostimulation of the nerve and appropriate muscle stimulation. Once the nerve has been accurately identified, the nerve is transected. Within 24 hours of surgery, the goat began walking normally and one leg and much improved on the second leg. The owner reported a normal gait in the goat 3 months post-operatively.

Severe preputial stricture in bulls: an interesting repair
A 10 year old Brahman bull presented for a history of preputial injury which had been treated conservatively by the rDVM. The owner and rDVM became concerned about the bull when he noticed that the bull was having difficulty urinating. On presentation, the bulls had a preputial swelling approximately 6 inches proximal to the preputial orifice. A stricture was palpated inside the preputial orifice, and necrotic tissue was removed which allowed the bull to urinate. Owner wanted to do everything possible to save the bull so a resection and anastomosis of the prepuce was performed. Seven days post-operatively, another stricture was detected at the surgical site. Four weeks later, and second resection and anastomosis of the prepuce was performed, and four days later, the prepuce began to stricture down yet again. So, a plastic tube was inserted into the preputial cavity to help prevent further stricture formation. The plastic tube was maintained within the preputial cavity for fifty-five. Once the tube was removed from the preputial cavity, the prepuce continued to stricture. Therefore five days later, surgery was performed to create a preputial stoma. A two inch stoma was created between the ventral prepuce and skin of the sheath to create a marsupialization from the prepuce to the skin. A penrose drain was sutured to the penis and exited through the stoma. The bull was discharged with 60 days of sexual rest. Following this time, the owner exposed the bull to a cow and saw the bull breed the cow. The cow was only exposed to this bull, and the rDVM confirmed that the cow was pregnant; the cow delivered a healthy calf. This procedure has been performed in 3 bulls with the following results: a bucking bull that went on to buck without event, the Brahman bull mentioned above with a calf on ground by natural service, and a Santa Gertrudis bull that has had semen collected through his stoma.
Fetal mummification in a Brahman cow

A 5 year old Brahman cow that was primarily used as an embryo donor presented for a possible fetal mummification. A 7 day embryo was transferred on May 24th. She was confirmed pregnant approximately 4 weeks later on June 20th with an estimated calving date of March 3rd. Upon passing her due date, the cow was palpated by the rDVM and diagnosed with a possible mummified fetus (~7 month fetus). Upon presentation, the physical examination was normal except for the presence of a presumed fetal mummy of approximately 6-7 months of age. The fetus was difficult to reach via transrectal palpation. Due to the potential value of the fetus, additional diagnostic tests were performed to confirm the diagnosis of fetal mummification. These additional tests included the following: transabdominal ultrasonography, testing for pregnancy specific protein B with the BioPRYN® blood test, and fetal electrocardiogram.

The fetal echocardiogram was performed by directing the leads across the cow (negative lead at mid-level jugular groove, positive lead at inguinal region between stifle and abdomen). This technique would allow for detection of both the dam and the fetus’ heart rate and rhythm at the same time. Based on ECG, ultrasound, and eventually a BioPRYN® test, the cow was confirmed to be pregnant with a fetal mummy. Based on the size of the fetal mummy, luteolytic drugs and colpotomy were ruled out as possible treatment options.

Because of the value of the cow, the owners elected for surgical removal of the fetal mummy with the cow under general anesthesia.

References available upon request.
The use of antimicrobials has been a conventional therapy in treatment of uterine infections in cattle. However, the use of antibiotics has not been without controversy. Debate continues regarding antimicrobial efficacy, effects on future fertility, risk for bacterial resistance and residues. The proper use of antimicrobials to treat uterine infections must first begin with an appropriate diagnosis and thorough understanding of the immunology of the uterus, the pathophysiology of uterine infections, and the properties of the various antimicrobial agents that may be used therapeutically.

**Intrauterine therapy**

A variety of antibiotics and antiseptics have been infused into the uterus of cows to treat postpartum infections. Intrauterine antimicrobials are used in order to achieve high concentrations at the site of infection but are usually unable to penetrate any deeper than the endometrium. The intrauterine use of antimicrobial agents is controversial as some have found intrauterine treatment to be beneficial while others have found these agents to have no effect or a detrimental effect. The bovine uterus is an anaerobic environment. Thus, antibiotics that are chosen for intrauterine infusion must be active in the absence of oxygen. Additionally, most antibiotics depress the activity of uterine neutrophils and interfere with uterine defense mechanisms. Thus, one must carefully evaluate the evidence regarding intrauterine antimicrobial use and carefully consider both the advantages and disadvantages associated with therapy.

Historically, intrauterine use of antimicrobials has been a common therapy for treatment of uterine infections. Antimicrobials that have been reportedly used for these infections include tetracycline, penicillin, cephapirin, chloramphenicol, Lugol’s iodine, gentamycin, spectinomycin, sulfonamides, nitrofuransone, povidone iodine solution, urea, and chlorhexidine. Although, most of these compounds are not approved for intrauterine use and have no published withdrawal times. There are also reports that intrauterine infusion of antibiotics causing drug residues in milk. In addition, regulatory guidelines must be adhered to in cases of extralabel use of antimicrobials in food animals. Intrauterine therapy is considered an extralabel use, and thus may be prohibited for many antibiotics, particularly in the United States.

The organisms that cause most postpartum infections are usually sensitive to penicillin. However, bacterial contaminants present within the uterus during the first several weeks postpartum produce penicillinase which makes penicillin useless if used locally in the early (less than 30 days) postpartum period. By 30 days postpartum, the contaminant bacteria are usually eliminated and intrauterine treatment with penicillin is more likely to be effective. Other factors may also affect the efficacy of intrauterine antibiotic therapy. Uterine lochia present during uterine infections contains organic fluids and debris that can render certain antibiotics, such as sulfonamides, ineffective.

More recently, oxytetracycline has been the antimicrobial that is commonly used for intrauterine therapy. However, one study indicated that most isolates of *A. pyogenes* are resistant to oxytetracycline. This study also showed that large doses of intrauterine oxytetracycline did not affect the frequency of isolation of *A. pyogenes*. In addition, oxytetracycline as well as Lugol’s iodine are quite irritating and are reported to cause coagulation necrosis of the endometrium. Although some studies indicate an improvement in reproductive performance with the use of intrauterine oxytetracycline, it has been speculated that this improvement may be due to local prostaglandin production due to chemical irritation of the endometrium.

In general, intrauterine infusion of antimicrobials has generally failed to show any increase in reproductive performance. Although, two large field studies evaluated the use of cephapirin benzoate in cows with clinical endometritis and reported some improvement in reproductive performance. However, other studies indicate no improvement in reproductive performance when evaluating intrauterine administration of cephapirin benzoate. The appropriate use of intrauterine antibiotics to treat uterine infections still remains controversial as only a limited number of studies indicate the efficacious use of intrauterine antibiotics.

**Intrauterine antiseptics**

Numerous antiseptics have been used to flush and lavage the postpartum bovine uterus with iodine and chlorhexidine solutions being most commonly used. Many of these solutions are quite irritating to the endometrium and are thought to stimulate endogenous prostaglandin release. One study showed that the incidence of retained fetal membranes and endometritis was reduced in cows that received 500 mL of 2% Lugol’s iodine immediately after calving and again 6 hours later. However, this study did not evaluate the future reproductive performance of these treated cows. Another study evaluated the use of 50 to 100 mL of 2% povidone iodine solution in the uterus one month postpartum and found that the reproductive performance of normal cows was not improved and that the treatment was detrimental to the fertility of cows with endometritis.
Systemic antibiotic therapy
Cattle with metritis often suffer from moderate to severe illness. These cattle are often septic and present with fever, depression, and anorexia. A variety of antibiotics have been recommended for parenteral use in cattle suffering from uterine infections. Penicillin or one of the synthetic penicillin analogues and ceftiofur are two of the most common antibiotics used systemically in cattle suffering from metritis. Systemic use of oxytetracycline may not be efficacious because of the difficulty in achieving the minimal inhibitory concentration (MIC) required for *A. pyogenes* in the uterine lumen. However, one study reported clinical improvement of cattle suffering from metritis with the use of tetracycline at 10mg/kg.

Ceftiofur is a third generation cephalosporin that has broad-spectrum activity against gram negative and gram positive bacteria. Ceftiofur (when administered parenterally) is reported to reach all layers of the uterus without causing violative residues in milk. Ceftiofur is approved in the United States for systemic administration to lactating cows affected with metritis. A subcutaneous dose of ceftiofur at 1mg/kg in post-partum cows results in a concentration of ceftiofur and its active metabolites in plasma, uterine tissues, and lochia at a higher MIC than required for most of the common pathogens involved in metritis. One study demonstrated that ceftiofur administered at 2.2mg/kg daily for 5 days was effective in treating cows with metritis. Another study supported these findings and showed ceftiofur administered at 2.2 mg/kg once daily for 5 days is as effective for treating metritis as procaine penicillin G or procaine penicillin G with intrauterine infusion of oxytetracycline.

Because of the reported lack of efficacy and potential detrimental effects of future fertility, intrauterine infusion of antibiotics is not a favored treatment for most cases of metritis. Certain systemic antibiotics have demonstrated their effectiveness at treating uterine infections in cattle. Thus, most cases of metritis, especially cows that are toxic, should be treated with systemic antibiotics such as penicillin or ceftiofur.

Conclusion
There are no antibiotics currently approved for intrauterine administration. Intrauterine infusion of antibiotics leads to contamination in milk and tissues for which appropriate withdrawal times have not been ascertained. In addition, the assays used on farm to detect antibiotics in milk may not be accurate. Although some studies indicate a positive response to therapy with the use of intrauterine antimicrobials, most studies do not show an improvement in reproductive performance or clinical signs of disease when comparing intrauterine antimicrobial therapy and systemic antibiotic therapy. This information, in conjunction with concerns regarding uterine or endometrial damage and withdrawal times following the use intrauterine antimicrobials, suggests systemic antibiotic therapy as the best treatment for many cases of cows with uterine infections.

References available upon request.
Bovine trichomoniasis is a sexually transmitted disease caused by the extracellular protozoa *Trichomonas foetus*, an obligate parasite of the reproductive tract of the cow and the folds on the mucosal surfaces of the bull’s penis and prepuce. Infected bulls are often asymptomatic carriers of *T. foetus*. However, these infected bulls are capable of transmitting the organism to a cow during coitus. Infections in cows cause endometritis, cervicitis, vaginitis which may result in early embryonic death, abortion, pyometra, fetal maceration, or infertility. The major economic losses associated with *T. foetus* are due to: 1) reduced calf crop due to early embryonic loss or abortion, 2) reduced weaning weight due to delayed conception, and 3) culling and replacement of infected cattle. Due to the inability to use efficacious drugs, such as the nitrimidazoles, for control and prevention of *T. foetus* infections in food animals, most control efforts have targeted identification and elimination of positive bulls, systemic immunization of cows and bulls, and management strategies to prevent introduction of the organism into the herd.

**Pathogenesis in the female**

**Life cycle**
The life cycle of *T. foetus* is thought involve two forms 1) a tear-shaped trophozoite form and 2) a pseudocyst form. The trophozoite is 10-25μm long and possesses three posterior flagella, one anterior flagellum and an undulating membrane. Trophozoite multiply asexually through binary fission. Pseudocysts usually appear as a result of unfavorable conditions; although, a small percentage of pseudocysts exist under normal conditions. Pseudocysts occur when *T. foetus* trophozoites round up and internalize their flagella in response to assorted stimuli. The pseudocyst form lacks a protective cyst wall and does not represent a true cyst form. Trophozoites of *T. foetus* are transmitted between cows and bulls during coitus and remain in the genito-urinary tract where they multiply by longitudinal binary fission. Under stressful conditions trophozoites will internalize their flagella and replication of the nuclei and other cellular structures will occur, resulting in a multinucleate pseudocyst form. When conditions become desirable once more, mononucleate trophozoites will bud from the pseudocyst. In bulls, infections are usually chronic and asymptomatic and often persist for the life of the animal. Infected cows will initially experience vaginitis which may or may not resolve spontaneously. In some cases, endometritis can occur resulting in complete sterility. Tritrichomonas infections may also result in fetal loss during pregnancy.

**Transmission**
Cows become infected with *T. foetus* primarily through coital exposure with an infected bull. Subsequently, a mild vaginitis occurs that may go undetected. The organism gains entry into the uterine lumen via the cervix during estrus. Colonization of the entire reproductive tract with *T. foetus* occurs within 1 to 2 weeks. Although, contaminated semen or contaminated insemination equipment may also be minor sources of infection. Penetration of the vagina is seemingly necessary because swabbing the vulvar area with high numbers of organisms does not result in vaginal or uterine infection. Infected cows conceive but infection causes endometritis, cervicitis, or vaginitis which results in death of the conceptus within the first half of gestation, abortion, pyometra, fetal maceration, or infertility. These infected cows usually remain infertile for a period of 2 to 6 months. In heifers, the duration of infection is reported to be as short as 95 days or as long as 22 months. *Trichromomonas foetus* has been detected in the reproductive tract for 13 to 28 weeks after experimental infection in heifers.

**Consequences of infection**
*T. foetus* organisms arrive in the female reproductive tract concurrently with spermatozoa. However in most cases, fertilization occurs in spite of the presence of the pathogen. In vitro studies have demonstrated that fertilization and early embryonic development to the hatching stage (8-10 days) are not significantly affected by simultaneous culture with *T. foetus*. Conceptus deaths most commonly occur between 50-70 days of gestation. Therefore, the majority of pregnancy loss is during the fetal period (>42 days of gestation). Although unusual, occasional abortions can occur of fetuses greater than four months of gestation. Conceptus deaths most commonly occur within 1 to 2 weeks. Although, contaminated semen or contaminated insemination equipment may also be minor sources of infection. Penetration of the vagina is seemingly necessary because swabbing the vulvar area with high numbers of organisms does not result in vaginal or uterine infection. Infected cows conceive but infection causes endometritis, cervicitis, or vaginitis which results in death of the conceptus within the first half of gestation, abortion, pyometra, fetal maceration, or infertility. The proportion of pregnancies conceived towards the end of the breeding season. Most producers do not recognize a problem in the early breeding season as conception occurs normally. The conceptus in most infected cows typically survives long enough to release sufficient interferon tau to prevent the prostaglandin F2α-mediated lysis of the corpus luteum. Fetal death in infected cows occurs between 7 to 10 weeks of gestation. Death of the conceptus during the early stages of pregnancy results in a prolonged listeriosis interval. Due to abortions and subsequent immunity, the distribution of pregnancies is unusually skewed with a higher proportion of pregnancies conceived towards the end of the breeding season. Although in many progressively managed herds with a limited breeding season, the bulls may no longer be available by the time the cow aborts and clears the infection. Therefore, *T. foetus* infection in a herd may go unnoticed until the time of pregnancy diagnosis when a high percentage of females are diagnosed not pregnant. Pyometra, along with abortions, may be the first physical signs of *T. foetus* infection in a herd, but are thought to occur in less than 5% of infected cows. Pyometra results as the corpus luteum of pregnancy is maintained with a large purulent response which may cause damage to the uterine endometrium.
Most infected cows will clear the organism and develop short-lived immunity of 6 months to one year. However, carrier cows do occur and are capable of spreading the protozoa. In the case of carrier cows, a very small percentage of cows (<1%) in infected herds have been shown to remain infected throughout pregnancy and into the following breeding season. Thus, the carrier cow has the potential to be quite devastating to control efforts and emphasizes that control programs must focus on the entire herd, not just the bull. Pathologic changes have been reported in several late-term, *T. foetus* aborted fetuses. The placenta had focal or diffuse invasion of the chorionic stroma by *T. foetus* as seen on hematoxylin and eosin (HE) stained sections of placentas. There was also evidence of a moderate inflammatory cell infiltrate comprised mostly of mononuclear cells. Six of eleven fetuses that were examined had bronchopneumonia with identifiable trichomonads in the airways. Another examination of late term abortions associated with *T. foetus* described a necrotizing enteritis and pyogranulomatous bronchopneumonia with tissue invasion by trichomonads. The exact mechanism that leads to the death of the conceptus is not fully understood. Although, cytotoxic and hemolytic effects by *T. foetus* on mammalian cells have been described.

The preputial cavity of the bull provides an ideal environment for *T. foetus* as the organism localizes in the preputial smegma of the epithelium of the bull’s penis and prepuce. The organism does not penetrate the epithelium and does not cause any observable gross pathology or affect semen quality or libido. Histological changes are subtle at first with an increase in the number of neutrophils in the nonkeratinized, stratified squamous epithelium of the glans penis and preputial epithelium followed by an infiltration of lymphocytes and plasma cells penetrating into the intraepithelial area which coalesce in the subepithelium to form lymphoid nodules. The duration of infection with *T. foetus* for bulls is not clearly understood. There are two theories regarding this debate: 1) transient infection and 2) chronic carrier state. Bulls with the chronic carrier infection of *T. foetus* rarely clear the infection regardless of time. The pathophysiology of infection regarding the carrier state in mature bulls in not fully understood. *T. foetus* infections in bulls less than 3-4 years of age are more likely to have a transient infection. Younger bulls may not efficiently transmit the organism to a noninfected cow unless the sexual contact occurs within minutes to days of breeding an infected cow. Thus, transmission of *T. foetus* by a young bull is thought to be more passive, mechanical transmission as compared to transmission in older, chronically infected bulls. Nonetheless, any bull exposed to a *T. foetus* infected cow as a result of natural breeding is capable of becoming chronically infected, regardless of age.

**Immunity**

In the female, *T. foetus* induces inflammation of the mucosa of the vagina, the cervix, the endometrium and the oviducal mucosa. In the first one to two weeks post infection, neutrophils and eosinophils predominate; however, this is followed by a moderate to severe mononuclear infiltration of lymphocytes and plasma cells. Subepithelial and periglandular lymphoid nodules resembling lymphoid follicles begin to develop at almost six weeks post infection. In addition, there is also an apparent degranulation of mast cells between six to nine weeks post infection. *T. foetus* specific IgA and IgG1 antibodies are detectable in uterine and vaginal secretions by the fifth to sixth week post infection. The IgA antibodies do not kill the organisms but may be responsible for immobilization and agglutination of parasites as well as preventing adhesion of the organisms to the mucosal surfaces. The IgG1 antibodies are presumed to facilitate complement mediated lysis of the parasites as well as opsonization and enhanced phagocytic killing by neutrophils or macrophages. Immunity following natural infection and clearance of *T. foetus* is short-lived with females becoming susceptible within a year, in time for the following breeding season. Because *T. foetus* is an extracellular pathogen, the immune response from the host is predominately humoral and the result of the short-lived immunity. The uterine mucosal inflammation that is seen with infection may allow systemically derived IgG and complement to gain access to the lumen of the uterus and, thus, clear the organism. A relative lack of IgG from the vagina or possibly blocking of IgG effects by vaginal IgA binding of organisms may help explain the carrier state that can be seen in infected herds. Although specific immunoglobulins have been detected in small amounts in preputial smegma by some researchers, there seems to be no effective acquired immunity to *T. foetus* in the mature bull.

**Diagnosis**

The comparison of diagnostic assays for detection of *T. foetus* infections has primarily focused on the bull. Collection of *T. foetus* samples from bulls involves recovering the organism from the preputial cavity of the bull. Several techniques have been described for collection of diagnostic specimens in the bull and include a dry pipette technique, a wet pipette technique a douche technique and a swab technique. While the douche method is preferred in Europe, the dry pipette technique is most commonly used in the United States. Regardless of which technique is used, it is generally recommended that bulls be given 2 weeks of sexual rest prior to sample collection in order to allow accumulation of the organism on the bull’s penis and prepuce and a greater chance of recovery.

Isolation of *T. foetus* from the female is reported to be less sensitive when compared with techniques used for bulls. In one study, the InPouch™ TF system (BioMed Diagnostics, Inc; White City, OR) was more effective than Diamond’s medium (88% versus 68%) in detecting heifers that had been experimentally infected with *T. foetus*. The accuracy of prevalence in the cow most likely depends on the timing of sampling relative to exposure. The immune response in females begins to eliminate the infection within 8 to 10 weeks after exposure in unvaccinated females. Therefore, cultures from females are best performed before the infection is possibly eliminated by the immune response.
Sample handling is also crucial for accurate detection of T. foetus. When evaluating temperature and media type it has been found that when laboratory of field isolates were cultured in Diamond’s medium or InPouch™ TF, all cultures were positive for T. foetus when maintained for up to 4 days at either 22° or 37°C. However, samples maintained at 4°C or less resulted in inconsistent sensitivity. It is important to remember that time, temperature, type of isolate, and type of media all have an effect on the sensitivity of T. foetus culture.

Microscopic evaluation of cultured organisms is not sufficient to differentiate T. foetus from nonpathogenic intestinal or coprophilic trichomonads (Pentatrichomonas hominis, Simplicimonas moskowitzi, Tetratrichomonas spp., etc). Therefore, several conventional and real-time polymerase chain reaction (PCR) assays have been developed for the definitive diagnosis of trichomoniasis, and this methodology has demonstrated some advantages over culture. However, accurate PCR results are directly related to the quality of the sample, which can be affected by transport condition parameters such as temperature and time of transport to the laboratory. There have been a number of issues that have limited the sensitivity of various conventional PCR assays for the detection of T. foetus. These problems include DNA degradation, accumulation of inhibitory compounds, sample contamination, and unexpected amplification products. One study demonstrated a decrease in sensitivity of PCR testing with samples that were stored for 5 days or more. However, PCR was in 100% agreement with culture as long as the PCR was performed within 24 hours of the sample being submitted.

A more recent study evaluated the effect of different simulated transport conditions on samples containing T. foetus for the diagnosis of trichomoniasis using culture and quantitative PCR (qPCR). This study demonstrated that transport temperatures of 4–20°C for 1–3 days before culture reduced or temporarily inhibited parasite replication but maintained viability. Samples tested by either culture or qPCR would have been expected to give positive results. However, diagnosis of trichomonads by both methods was negatively affected when specimens were maintained at transport temperatures of 42°C for 24 hours or more. This study emphasizes the importance of ensuring that clinical samples arrive to the diagnostic laboratory within 24–48 hours and of avoiding temperature transport conditions above 37°C in order to achieve an accurate diagnosis of T. foetus. The effects of high incubation temperatures on culture and real-time PCR for T. foetus have also been evaluated following inoculation into the InPouch™ TF system. This study showed that T. foetus was detectable at microscopically in inoculated pouches incubated at 37°C regardless of exposure time (1, 3, 6 and 24 hours), whereas those samples incubated at 46.1 °C detected T. foetus only after 1 and 3 hours of incubation. T. foetus was detected in samples incubated at 54.4°C after only 1 hour. Testing using real-time PCR for all inoculated media samples (37°C, 46.1°C, and 54.4°C at 1, 3, 6 and 24 hours) produced positive results for all inoculated media samples. This study suggests that samples collected for culture alone should be protected from high temperatures.

Prevention and control
One complicating factor with bovine trichomoniasis in the United States is the lack of effective treatments with U.S. Food and Drug Administration approval. Historically, the most successful treatment for bulls with trichomoniasis was systemic treatment with nitromidazole derivatives. Currently, the use of nitromidazole derivatives is illegal in food-producing animals in the U.S., and no effective alternative treatments are available. The lack of effective, approved therapies for bovine trichomoniasis emphasizes the need for appropriate preventive and control measures. Prevention of trichomoniasis includes the following recommendations: 1) avoid movement of animals (co-grazing, leasing of bulls, good fences); 2) utilize artificial insemination, if possible; 3) use a defined breeding season and cull all non-pregnant females after the breeding season; 4) purchase virgin bulls and heifers as replacements; 5) test all bulls for T. foetus prior to introduction into the herd and maintain a young population of bulls; and 6) breed purchased cows and heifers in a separate herd.

Once T. foetus has been confirmed in a herd, there are additional measures that should be considered in order to “clean up” the herd. These measures include 1) testing and culling all infected bulls and purchasing T. foetus negative bulls; 2) intense management of bulls so that smaller breeding units are used and bulls are bred to the same cattle until trichomoniasis is under control; 3) create high and low risk herds; and 4) vaccinate all herd females with an approved T. foetus vaccine. Vaccination is an important aspect of any control program as it has been shown to reduce pregnancy wastage associated with T. foetus infection in cattle herds. Currently, TrichGuard® (Boehringer Ingelheim Vetmedica, Inc.) is the only commercially available vaccine licensed by the USDA for the control of trichomoniasis in the United States. TrichGuard® is a proprietary vaccine that is a Freund adjuvant killed T. foetus-derived vaccine that requires two doses subcutaneous injections administered 2 to 4 weeks apart with the last injection to be given 4 weeks prior to the breeding season. One study compared pregnancy and calving rates between beef heifers vaccinated with TrichGuard® and control heifers after heifers were exposed to T. foetus infected bulls and intravaginally inoculated with a large number (10 million) of T. foetus organisms. At calving twice as many vaccinated heifers calved when compared to control heifers (61% versus 31%). Thus, the vaccine appeared to offer at least some protection against T. foetus. More recent studies have confirmed these findings, as well. In addition, Palomares, et al. concluded that vaccination of heifers with TrichGuard® significantly increased the levels of IgG antibodies to the T. foetus surface antigen in serum, vaginal secretions, and uterine fluid; these antibody levels remained elevated through days 43, 75, and 182, respectively.
Conclusion
Trichomoniasis can be an economically devastating infection in cattle herd with losses due to reduced calf crop due to early embryonic loss or abortion, reduced weaning weight due to delayed conception, and culling and replacement of infected cattle. Carrier females and concerns with diagnostic sampling and testing have made the control of trichomoniasis in cattle even more complex. Control and prevention of *T. foetus* infections in cattle must focus on identification and elimination of positive cows and bulls, systemic immunization of cows and bulls, and management strategies to prevent introduction of the organism into the herd.

References available upon request.
Lameness accounts for tremendous production loss in the cattle industry and has been identified as a particular concern in animal welfare. Cattle are relatively stoic animals and often do not show lameness until significant pathology is present. This discussion will explore anatomic and management relationships with common orthopedic conditions in cattle. Additionally, diagnostic and therapeutic options will be reviewed.

**Stifle injuries**

Stifle injuries are common in cattle and one or more structures may be involved. Of the common injuries rupture of the collateral ligament produces the least degree of lameness. Cattle with this condition are slightly lame and the injury may be easily diagnosed by watching them walk away from you. There is medial-to-lateral instability and the stifle will deviate either medial or lateral, toward the affected side when the animal is full weight-bearing. Restrain the animal and place fingers of one hand on the medial aspect of the stifle joint while abducting the lower limb. If the medial collateral ligament is torn there will be excessive joint space while the leg is abducted. Place the fingers of one hand on the lateral aspect of the stifle and adduct the lower limb to examine for excessive motion if the lateral collateral ligament is torn.

Meniscal tears cause the next most severe lameness in cattle. The most common injury is similar to other species in that the posterior horn of the medial meniscus is injured more commonly than the lateral meniscus. With acute injury there will be noticeable lameness and there may be evidence of joint effusion. The injury appears to occur more commonly in heavy muscled beef bulls than in other cattle. There may be an audible or palpable “click” during the weight bearing portion of the stride. The mass of the animal usually precludes palpation of the classical anterior drawer sign as may be detected in dogs. However, many beef cattle will tolerate flexion of the affected limb whereby the veterinarian may be able to detect excessive motion in the stifle joint and perhaps grating of bony surfaces due to loss of articular cartilage.

The third common and most severe stifle injury is rupture of the anterior crucial ligament (ACL). This injury causes marked lameness and usually obvious joint effusion. The animal is very reluctant to bear weight on the affected limb. These injuries are discussed together as they all appreciably shorten the productive life of cattle. Animals with only collateral ligament tears develop degenerative joint disease due to joint instability and abnormal wear of joint surfaces. Animals with meniscal tears do likewise with the added risk of suffering cruciate ligament tears due to the atrophy of leg muscle that frequently rapidly accompanies this injury and more severe loss of stability of the stifle joint. Animals with cruciate ligament tears suffer severe joint instability, rapid muscle atrophy and frequently quickly suffer meniscal tearing and loss of articular cartilage.

Therapy for any of the above conditions consists of confining the animal to a stall or small paddock that is level and free of mud for 6 – 8 weeks. Bulls with anterior cruciate ruptures should not be used for breeding for a minimum of 6 months. Animals with this injury usually do not return to soundness and have permanent muscle atrophy on the injured limb. Analgesics are not recommended during the acute phase of the injury as animals so treated may use the limb excessively and sustain additional trauma to the joint. However, anti-inflammatory agents may prove beneficial after a few months convalescence to assist a bull through a breeding season.

Alternatively, application of a Walker Splint on the affected limb may improve longevity in bulls with ACL ruptures. This device immobilizes the limb for 6 weeks preventing motion and additional soft-tissue damage in the joint while simultaneously allowing fibrosis of the joint capsule. Negative effects of the splint are the additional expense incurred and the degree of pressure necrosis in the flank inherent with this type splint. Following removing the splint the bull should be confined to a stall for an additional 2 – 4 weeks as the bull regains muscle tone on the immobilized leg. He should not be used for breeding for a minimum of 6 months from the original injury.

**Emergency treatment & first aid**

A thorough physical examination should be conducted on all animals suspected of having a fracture prior to the decision for treatment. However, the patient first must be made safe from continued trauma. Often, injured cattle are recumbent when examined. The animal should be allowed to remain recumbent until the physical examination has been conducted and an initial fracture assessment done. Adequate colostrum ingestion by newborn calves is critical to pre-operative preparation of the patient and success of the procedure. If colostrum ingestion is unknown, serum IgG should be determined or total protein measured. Calves that are well hydrated and have a serum protein of less than 5.5 g/dl should be considered to have poor colostral antibody transfer and receive a plasma transfusion before attempting fracture repair.

Temporary stabilization of limb fractures may be performed prior to moving the animal or attempting to get the animal to stand. As a general rule, fractures below the level of the mid-radius or mid-tibia may be temporarily stabilized with splints or casts. In my experience, field stabilization of fractures proximal to this level should not be attempted. These efforts often result in the creation of a
“fulcrum effect” at the fracture site and result in increased soft tissue trauma, damage to neurovascular structures, or compounding of the fracture. Cattle with these fractures should be carefully loaded into the trailer and allowed time to lie down before beginning transport.

Two splints or a cast may be used for temporary stabilization of the fracture. Two boards or pieces of PVC pipe that is cut in half and placed at 90° to each other (i.e. caudal and lateral aspect) create a stable external coaptation. A padded bandage is placed on the limb, splints and positioned and elastic tape applied firmly. The injury should be centered within the coaptation with as much support proximal and distal to the injury as possible. All external coaptation devices should extend to the ground. Injuries that occur distal to the carpus or hock should have splints placed to the level of the proximal radius or tibia. For injuries proximal to the carpus or hock and distal to the midradius or midtibia, the lateral splint should extend to the level of the proximal scapula or pelvis.

Treatments

Walking block

Because cattle have two weight-bearing digits, a cow may stand one digit during convalescence of the opposite digit (phalangeal fracture). A wooden or rubber block can be applied to the sole of the healthy digit. A walking block is most suitable for management of P1, P2, and P3 fractures of a single digit. These animals should be confined to a small pen or paddock for 6 to 8 weeks while the fracture heals. If the block remains on for 6 weeks after application, it should be removed.

Casting

Half-limb or low-limb casts can be used for immobilization of phalangeal fractures and for distal metacarpal or metatarsal physeal fractures. The cast is placed from a point immediately distal to the carpus or hock and extend to the ground with the foot included in the cast. The dewclaws and the top of the cast should be padded but only stockinette is placed on the remainder of the limb. If thick padding is placed over the entire limb, the padding quickly becomes compressed which allows room for the limb to move within the cast and displacement of the fracture to occur. Full-limb or high limb casts are used for fractures that occur at or proximal to the midmetacarpus or metatarsus but distal to the midradius or midtibia. Full-limb casts are placed in a similar manner as half-limb casts, but the bony prominences of the accessory carpal bone, styloid process of the ulna, calcaneous, and medial and lateral maleolus of the tibia must be padded.

Physical restraint, sedation, or anesthesia can be used as deemed necessary to facilitate placement of the cast. Maintaining alignment, both craniocaudal and lateromedial, of the limb during application is essential. Tension on the limb during casting may be achieved by placing wires through holes drilled in the hoof wall and applying tension. Tension should be placed so that the hoof is positioned in a normal to slightly flexed position. The thickness of the cast is usually based on clinical judgment. Casts that are 6 to 8 layers thick are usually adequate for calves weighing less than approximately 300 pounds. However, adult cattle may require casts as much as 12 to 16 layers thick. Casts on the hind limbs must be made thicker because of the stress concentration by the angulation of the hock. Incorporating two metal rods at 90° to each other can increase the strength of the cast for extremely large animals. A walking bar (U-shaped bar placed under the hoof and incorporated into the cast) concentrates loading forces into the cast and away from the distal limb, but the foot should always be included in the cast.

Casts may be maintained in calves for up to 6 weeks without being changed. Scheduled cast changes at 3-week intervals may be required for rapidly growing calves or for calves that become lame during convalescence. Physeal fractures usually heal within 4 weeks, but nonphyseal fractures often require 6 weeks to reach clinical union in calves. Fractures in adult cattle may heal within 8 to 10 weeks, but often require 12 to 16 weeks for clinical union to occur. Radiographic union of the fracture (defined as bone union with resolution of the fracture line) is not seen for weeks to months after clinical union (defined as sufficient bridging callus to allow weight bearing without additional support to the limb) has been reached.

Thomas splint +/- cast

Use of a Thomas splint and cast combination is appropriate for fractures distal to the elbow or stifle. The length of the splint should be measured while the animal is standing and by using the normal limb for measurements. An appropriate splint is chosen or constructed, and the patient is placed into lateral recumbency (using rope restraint, sedation, anesthesia, or a combination of the three). The fracture is reduced and a cast applied from the distal metacarpus or metatarsus to the level of the proximal radius or tibia. The splint is placed on the limb, the foot is attached to the base of the splint by drilling holes in the hoof walls and wiring the foot to the splint base, and casting tape is used to attach the cast to the splint frame. The limb cast should be firmly attached to the splint frame to prevent rotation of the limb along the splint during ambulation. The hoop of the splint must be firmly placed into the axilla or groin to allow maximal weight transference. Therefore, the hoop must be heavily padded. Cattle having a Thomas splint-cast must be assisted to stand for 3 to 5 days until they learn how to rise under their own power. Also, these patients must be checked several times daily to ensure that they have not lain down on top of the splint. Often patients are not able to rise after lying down on the splint and life-threatening rumen bloat may occur if they remain trapped for a prolonged period.

Closed versus open fractures

Overall, closed fractures without damage to the blood supply to the limb have a good to excellent prognosis for healing in cattle. The prognosis for success is less for older cattle or cattle of high body weight. Open fractures have a guarded prognosis for healing in
cattle. The success rate depends on the severity of soft tissue damage, the bone affected, the age of the patient, the duration and degree of contamination of the wound, and the economic limitations placed on fracture management. Prolonged antibiotic therapy is indicated, and open wound management is preferable to enclosing the wound within a cast. Mature cattle are better able to overcome bone infection associated with open fractures than young calves. Often, mature cattle having an open metacarpal fracture are able to heal and return to productivity after thorough cleaning of the wound, administration of antibiotics, and application of a full limb cast. However, young calves with similar injury are prone to septic non-union or delayed union.

Treatment and prognosis for specific fractures

Pelvis
Fractures of the ileum or sacroiliac junction are the most common pelvic fracture in cattle. These injuries occur because of falls during mounting or on slippery flooring. Fractures of the ileum or sacroiliac junction respond well to stall confinement. Occasionally, ileum fractures become open, with bone projecting through the skin. Infection rapidly becomes established and bone sequestration occurs. Surgical removal of the fracture fragment of the ileum is indicated when sepsis or debilitating lameness is present. Internal fixation of ileum fractures is rarely indicated, but may be requested for cosmetic reasons. These fractures may be repaired by application of a bone plate, but reduction of the fracture may not be possible when the fragment is severely displaced.

Humerus
Non-articular, minimally displaced fractures of the humerus are best treated by stall confinement. In cattle, open reduction and internal fixation of the humerus with bone plates often causes permanent radial nerve paralysis. Use of an intramedullary interlocking nail may allow rigid fixation with minimal risk of radial nerve injury. The prognosis for healing the fracture with stall confinement is good, but the prognosis for return to normal productivity is guarded. Severely displaced or articular fracture of the humerus requires attempted internal fixation, but the prognosis is poor for return to normal productive use.

Radius and ulna
Closed fractures of the distal physis of the radius may be treated by a full limb cast and has a good prognosis for success. Fracture of the mid-radius and ulna require use of a Thomas splint-cast, transfixation pin-cast, or bone plate. The prognosis for healing is good, but significant contralateral limb injury may occur in animals treated by Thomas splint-cast. Transfixation pinning and casting and bone plating have good to excellent prognoses with minimal risk of permanent injury to the contralateral limb. Where applicable and economical, I prefer to treat fractures of the radius and ulna using transfixation pinning and casting.

Femur
Femur fractures most often occur in calves during forced extraction for dystocia. Femur fractures are occasionally found in adult cattle after falling during mounting or on slippery flooring. The femur is generally difficult to radiograph in adult cattle. Most femoral fractures are readily diagnosed by physical examination. Femur fractures in mature cattle have a grave prognosis for success because of their body weight and an inability to reduce the fracture. Therefore, euthanasia is elected. However, some selected femur fractures may respond to stall rest for 8 to 10 weeks. Contracture and swelling of the heavy muscles of these animals serve to reasonably splint the injured bones.

In calves, stack pinning of the femur has a good prognosis for success. Open reduction of the fracture is performed, and 2 to 5 intramedullary pins are placed into the femur. If large cortical defects are present, then an external skeletal fixator may be applied in addition to the intramedullary pins. These fractures are usually healed by 6 weeks after surgery. Sepsis is the most common reason for failure of fracture healing.

Tibia
Although fracture of the tibia has been seen as a result of forced extraction during dystocia, tibia fractures are usually caused by trauma. Fracture of the distal physis of the tibia may be treated with a full limb cast, but these fractures are common. Fracture of the middle portion of the tibia may be treated by Thomas splint-cast, transfixation pinning and casting, or use of a bone plate. Thomas splint-casts have a good prognosis for bone healing, but have a high rate of injury to the contralateral limb. Transfixation pin-casts and bone plates have a good to excellent prognosis for healing, and minimal problems with contralateral limb injury.

Metacarpus and metatarsus III/IV
Fractures involving the metacarpus (MC) or metatarsus (MT) III/IV are the most common fractures to occur in food animals. These injuries often occur as a result of forced extraction during dystocia. Closed fracture of the distal physis of the MC or MT may be treated using a half limb cast. Closed fracture of the middle portion of the MC or MT may be treated with a full limb cast. Open fractures in mature cattle may be treated by thoroughly debriding, cleaning, and flushing the wound, applying a full limb cast, and administering antibiotics for 10 to 14 days. In valuable cattle and young calves, open fractures are best treated by use of an external skeletal fixator and daily wound care until the wound is healed. Bone sequestra are often associated with open fractures of the MC and MT. Bone healing may not occur until sequestra have been removed. If prolonged sepsis has been present, cancellous bone grafts may be required to facilitate bone union. The optimal site for
harvesting cancellous bone grafts are the wing of the ileum and the proximal tibia. Implantation of antibiotic impregnated bone cement beads into a septic wound will provide prolonged local release of antibiotics and may accelerate resolution of osteomyelitis.

**Phalanges**

Closed fractures of the phalanges may be treated by application of a 2.5 to 3.5 cm height block to the sole of the healthy digit. Confinement to a stall or small pen is recommended for 6 to 8 weeks.

**References**

References are available upon request.
Etiology
Urinary calculi may occur in both sexes but is far more common in males. The composition of the calculi is more variable in small ruminants than cattle, and the anatomy of the goat urethra makes clinical management more frustrating. Feedlot steers, show steers, and animals that are kept primarily as pets are often castrated at an early age. Castration before six to nine months of age does not allow the urethra to develop to its full diameter. Therefore, these animals are at an increased risk for developing urolithiasis. Unlike calculi formation in animals that are kept on range pasture, calculi that develop in steers and small ruminants are usually multiple and may be present at more than one location along the urinary tract. The mineral composition of the calculi is dependent on both geographic region and diet. The most common compositions of calculi are calcium apatite, struvite, and phosphatic. The most common sites for urethral obstruction are the sigmoid flexure and the vermiform appendage at the distal tip of the urethra in small ruminants. The diet should be adjusted to increase fiber and water intake and to acidify the urine (ammonium chloride at 300 mg/kg orally). Much of this presentation will focus on urolithiasis in small ruminants.

Diagnosis
The early diagnosis of urolithiasis is important in determining treatment options and prognosis. These animals will often display a stretched out stance and may flag the tail while making repeated attempts to urinate. These signs are often mistaken by the owner as signs of constipation. Other signs of urolithiasis may include abdominal discomfort, dribbling urine, lethargy, depression, anorexia, abdominal distention, preputial swelling, and gritty precipitates on the preputial hairs. A chemistry profile may reveal an increase in BUN and creatinine, normokalemia or hyperkalemia, hyponatremia, hypochloremia, increased CPK and AST, and possibly academia. A complete blood count may show a leukocytosis with a left shift. Ultrasoundography may show a distended bladder and/or urethra to the level of the calculi.

Medical management
Medical management includes fluid support with correction of any alterations in electrolytes, the use of anti-inflammatory drugs (flunixin meglumine at 1 mg/lb IV), and antibiotics for cystitis. Sedation of animals that present for obstructive urolithiasis may be necessary to facilitate physical examination and extension of the penis. Acepromazine hydrochloride may be given at 0.05 to 0.1 mg/kg intravenously or intramuscularly with a slaughter withdrawal of 7 days. Diazepam may also be given at a sedative dose of 0.1 mg/kg intravenously. There is no data available for meat withdrawal for diazepam but a 30 day meat withdrawal is considered adequate. A lumbosacral epidural with 2% lidocaine at a dose of 1mL per 10-20 pounds of body weight (do not exceed 15 mL total volume) may also be used to help extend the penis. In small ruminants, once the penis has been extended simple excision of the vermiform appendage may provide immediate relief from urinary obstruction. If an obstruction existed at the vermiform appendage, urine will immediately begin to flow from the urethral opening. Urethral patency has been reported to be restored in 37.5% to 66% of cases using this method. However, rarely does a single calculus cause an obstruction in small ruminants. Therefore, obstruction at this site or at another location along the urinary tract is likely. Catheterization in ruminants is very difficult due to the presence of the urethral diverticulum, and surgery is usually the best therapeutic option. Another option in certain cases is to use infuse Walpole’s solution (pH 4.5) into the bladder to help break down uroliths and crystals. This procedure should be performed under general anesthesia and utilizes ultrasound-guided transabdominal cystocentesis.

An 18 gauge, 4 inch needle is used to remove urine from the bladder. The bladder is then lavaged with 30-60 mL of Walpole’s solution and then removed. An additional 30-50 mL of Walpole’s solution is infused into the bladder in similar manner and left in bladder. In some cases urine flow resumes in 24-36 hours with normal voiding occurring in 3-5 days. Some animals may require a second cystocentesis in 2-3 days.

Surgical management
Several surgical options exist for obstructive urolithiasis in ruminants. The options include cystotomy, tube cystotomy, bladder marsupialization, perineal urethrostomy, penile amputation, and urethotomy. The most common procedures performed in small ruminants are the perineal urethrostomy, cystotomy, tube cystotomy, and bladder marsupialization. Penile amputation and ischial urethrostomy are most commonly performed in bulls. All of which will be discussed in further detail.

Cystotomy
Cystotomy and tube cystotomy offer the longest survival and return to breeding function. However, the cost of cystotomy may limit its use to pets and breeding animals. The animal is anesthetized and placed in dorsal recumbency. The skin is clipped and aseptically
prepared. A right paramedian incision is made that is 2 to 3 cm off midline and extends cranially approximately 6 cm from the teats. The urinary bladder should then be identified and stay sutures placed at either end of the cystotomy site for greater stabilization of the bladder. The bladder is then opened and the bladder emptied and lavaged to remove all calculi. Normograde and or retrograde urethral flushing with an isotonic solution can be attempted. Once the urethra is cleaned of all visible stones, the cystotomy and abdominal incision are closed. However, if the urethra can not be cleared with 3 to 4 attempts of urethral flushing, then a tube cystotomy should be performed.

**Tube cystotomy**

Placement of a Foley catheter into the bladder and exiting through the ventral abdomen allows for continual drainage of urine. By routing urine flow through the catheter, the urethra is allowed to rest in order to decrease inflammation and promote healing. A small skin incision is made lateral to the paramedian incision and inserts the catheter subcutaneously, where it enters the abdomen and then the bladder. A purse-string suture is placed in the bladder wall to position the Foley. A small stab incision is made in the middle of the purse-string and the balloon end of the Foley catheter is placed into the bladder, after which the purse-string suture is tightened. After inflating the Foley catheter with saline, the bladder is tacked to the body wall with minimal tension. A one-way valve can be made from a finger of a latex glove and placed over the end of the catheter to create a type of Heimlich valve which helps decrease the incidence of ascending infections. The celiotomy site should be closed in three layers with an absorbable suture, and the subcutaneous tissues and skin closed in routine fashion. Sutures can be removed in 10 to 14 days. Clamping the catheter should begin on the fourth day after surgery to allow for normal urination. This should be done in a dry stall and with increasing duration until a full-stream urination is achieved. Normal urination should occur for 1 to 2 days before the catheter is deflated and removed. The Foley catheter should not be removed before day 7 after surgery to reduce the chances of urine leaking from the bladder. This bladder defect is allowed to heal spontaneously.

**Perineal urethrostomy**

Perineal urethrostomies have been performed in many sheep and goats and in young or lightweight cattle, but surgical failure, poor long-term survival rates due to strictures, and decreased reproductive function limit this to a salvage only procedure. The animal may be sedated heavily, given epidural anesthesia, or placed under general anesthesia. The perineal area is scrubbed and aseptically prepared. A skin incision is then made on the midline between the scrotum and the anus, and the retractor penis muscles and the penis are then identified. The penis is then exteriorized and rotated so that the dorsal blood supply can be ligated. As much of the penis as possible should be freed from the surrounding tissue in order to place the urethra in close proximity to the skin. The urethra is incised, and the urethral mucosa and tunica albuginea are sutured to the skin with as little tension as possible. The urethra is then sutured with a non-absorbable, monofilament suture in a simple interrupted pattern. The skin may then be closed in two layers with a non-absorbable, monofilament suture in a simple interrupted pattern. A Foley catheter can be placed into the urinary bladder for 3 to 4 days, and the animal placed on systemic antibiotics (procaine penicillin G 22,000 IU/kg IM BID) before surgery and for 3 to 5 days post-operatively. Sutures can be removed in 10 to 14 days. These animals can no longer be used for breeding. If strictures develop after surgery, bladder marsupialization may be performed. Prognosis for long-term survival after urethrostomy is guarded to poor because of stricture formation.

**Penile amputation**

Amputation of the penis may be indicated following rupture of the urethra in steers, bulls, rams, bucks, and wethers. Due to urine contamination of the peripenile elastic tissue at the site of the rupture, penile amputation may allow these animals to be salvaged for harvest after several weeks of healing. With the animal restrained in a squeeze chute, an epidural should be administered and the perineum should be prepared for aseptic surgery. The skin incision should allow for the penile stump to be directed caudoventrally to that urine flow will be directed at an angle between the hocks and tail. At the beginning of the anterioventral curvature of the perineum, at 12 cm skin incision should be made ventrally on the midline. The incision is then deepened through the subcutaneous and dense connective tissue between the semimebranosus muscles to expose the paired retractor penis muscles. Continue dissecting deep between the retractor penis muscles to locate the penis. Grasp the penis firmly and apply traction caudally and dorsally to bluntly dissect the penis from the surrounding tissue. Dissection may be necessary unless advanced necrosis is present. Once the penis is exteriorized, the retractor penis muscles should be ligated and transected as far proximally as possible. If possible, the dorsal vessels may be dissected free of the penis without transection to preserve the nutrient blood supply to the distal portion of the penis and prevent sloughing of the penis (when minimal necrosis is present). The penis should then be transected with a scalpel 5 cm distal to the dorsal apex of the skin incision. The urethra should then be generously spatulated. It is not necessary to suture the cut end of the CCP in steers. However, there may be hemorrhage from this cavernous tissue in bulls when erection is stimulated. Wedge excision of the end of the stump with suture closure will minimize hemorrhage in bulls. Suture the penile stump to the skin with nonabsorbable, monofilament suture. The suture should be placed through the skin and body of the penis and exit through the skin on the opposite side of the incision. The second limb of the suture should then be placed through the skin, under the penis and exit the skin on the
original side of the incision and tie. This suture will prevent the penile stump from retracting into the incision. Using #2-0 chromic gut, closely spaced simple continuous sutures should be placed around the incision in the urethral mucosa to reduce hemorrhage from the CSP during urination. Hemorrhage from the CSP may still be a problem in bulls and large steers during urination. After suturing, a 15 cm length of 1 cm diameter latex tubing should be placed inside the urethra and fixed into place with a single suture through the tubing and penile stump. This tubing serves as a stent to compress the CSP and reduce hemorrhage in the early postoperative period. The tubing should be removed in 5 days. Systemic antibiotics should be administered for 5 days postoperatively, and the patient monitored for hemorrhage from the penile stump and for the ability of the animal to urinate. Sutures may be removed in 10 days. Stenosis of the urethral opening is one potential complication.

Ischial urethrostomy
The urethral diverticulum located on the dorsum of the urethra just inside the ischial arch makes bladder catheterization extremely difficult in ruminants. Ischial urethrostomy is used primarily to bypass the diverticulum and allow introduction of a catheter into the urinary bladder to provide urine egress. This may work as a salvage procedure for steers to reach acceptable slaughter conditions. This procedure may also be used to divert urine from the distal urethra when attempting surgical repair of urethral fistulae or urethral tears in bulls. In addition, this procedure may also be used in cases with ruptured bladders. Because the rupture typically occurs on the dorsum of the fundus, this indwelling urinary catheter provides urine drainage which prevents bladder distention. With the animal restrained in a squeeze chute, an epidural should be administered and the perineum should be prepared for aseptic surgery. A 10 cm vertical incision should then be made on the midline of the perineum beginning 5 cm below the anus. The incision should be deepened through the dense fascial place beneath the subcutaneous tissue to expose the paired retractor penis muscles. Next, bluntly dissect between the muscles and identify the bulbospongiosus muscle. The urethral groove should then be palpated immediately deep to the bulbospongiosus muscle. The incision should then be made along the median raphe of the muscle through the CSP and into the urethra which is easily identified by its smooth mucosal surface. There may be quite a bit of hemorrhage from the CSP. A pair of hemostats may then be inserted into the urethra in both directions in preparation for inserting the urinary catheter. A 10 French male dog catheter with sterile lubricant applied should be introduced into a Foley catheter to act as a stylet for insertion of the catheter into the urethra. Select a Foley with a 30mL cuff and of the largest diameter that will pass into the urethra, usually a 20 to 28 French in adult bulls. The lubricated catheter should then be inserted into the urinary bladder and the cuff inflated with sterile water. Be careful not to overinflate the cuff such that pressure necrosis of the bladder may occur. Place a one way valve on the end of the catheter to prevent aspiration of air into the bladder.

Laser lithotripsy
Laser lithotripsy has been successfully used in establishing urethral patency in a steer, goats and pot-bellied pigs. The procedure consists of passing an endoscope and laser fiber retrograde in standing bulls with a pudendal nerve block or in recumbent animals under general anesthesia. The endoscope may also be passed through an ischial urethrostomy site distally to the level of the calculus. Once the laser (holmium:yttrium-aluminum-garnet or Ho:YAG) reaches the calculus, it is then centered on the calculus and fired in a pulsatile manner with urethral flushing in between firing until the calculus fractures into fragments small enough to dislodge with hydropulsion or retrieve with a wire basket catheter. It is reported that previous chemolytic treatment in animals with surgical urinary diversion results in uroliths with uneven surfaces that fracture readily.

References available upon request.
Bovine respiratory disease (BRD) is the most common and costly syndrome afflicting beef cattle after weaning. A basic understanding of the disease syndrome is important to design a treatment and prevention program. Many bacterial pathogens associated with BRD are normal flora that can be isolated from the upper respiratory tract of healthy cattle. Other disease syndromes relevant to the cow-calf farm including reproductive pathogens, may also be found in animals without clinical signs. Contributing factors such as animal immune status, pathogen load, organism virulence, and environmental conditions influence disease severity. Managing for a single disease causing agent or risk factor will not eliminate disease from the population. The complete animal management program must be evaluated to maintain hope of diminishing disease impact.

BRD diagnosis
Timely identification of clinically ill animals is critical because the best treatment protocol is ineffective if severe damage occurred prior to treatment. Recognition of disease is an art, not a science. The keys are systematic pen and animal appraisal, and diagnosis evaluation. Differentiation of specific diseases often depends on the epidemiology of the case presentation in the affected population.

Typical signs of respiratory disease include: anorexia, depression, animal isolation, increased respiratory rates, nasal discharge, coughing and diarrhea. A consistent method for evaluating pens and individuals within the group is important for accurate, timely identification of disease. Cattle are herd animals and considered prey in the predator-prey relationship of wild animals. In nature, predators feeding on the herd will pick out the weakest animals that may be easier to catch; therefore, the instinct for a sick calf is to blend in with the herd and not be found. Domesticated cattle have this instinct and try to avoid appearance of illness when possible.

A study of feedlot steers revealed that although only 35% of the animals were treated, 72% had pulmonary lesions present at slaughter. The pulmonary lesions were directly associated with a significant reduction in ADG during the feeding period. One of the most remarkable findings of the study was that 68% of the untreated steers had pulmonary lesions. This indicates that visual evaluation was inadequate to prevent significant production losses attributable to respiratory tract disease.

We should evaluate pens with these facts in mind. Walking or riding into the middle of the pen and trying to identify a sick animal is often fruitless unless the animals are very ill.

BRD prevention
Immunizations at arrival are commonly used for prevention of BRD. Products utilized often include viral antigens and may also include bacterial antigens. Research has illustrated differing levels of efficacy among specific antigens, and the veterinarian should have a realistic expectation of the risk reduction associated with vaccination at feedyard arrival.

Population-level BRD treatment and prevention strategies may most efficiently be employed soon after cattle arrive to the stocker or feeder operation; however, utilization of these tools is based on an accurate estimation of the population risk for BRD. Immediately prior to and after feeding is a good time to evaluate a pen for clinical illness. Animals exhibiting anorexia can be identified and animal movement toward the bunk can be used to assess locomotion or potential signs of lameness. All individuals within the pen should be viewed to assess for potential signs of illness.

BRD Risk profiling
Individual pens need to be managed differently based on cattle type and length of time they have been on the farm. Appropriate labor should be allocated to ensure adequate evaluation of cattle in the highest risk category and time frame. Risk categories are based on several factors that influence overall risk for morbidity and mortality including: distance traveled, age, weight, gender, and previous vaccination status of the cattle. Cattle that arrived in the last 2-3 weeks are at highest disease risk and these cattle may need more frequent observations. Treatment records and necropsy results also dictate pens needing a concentrated effort.

Summary
Operations differ significantly in management techniques and health programs. Critical control points should be identified for each farm and used to ascertain the biggest areas for potential improvement to allow proper allocation of resources. A customized wellness program couples medicine and management to minimize the negative impact of disease.

References / Suggested reading

Clinical diagnosis is one of the most common and important tasks completed daily by veterinarians. Adding value to client herds through diagnostic tests is common, and test results should be interpreted considering expected disease prevalence and economic consequences. The value of adding new diagnostic methods to the existing system is dependent on the characteristics of the current diagnostic test as well as the expected change in specificity and sensitivity with the addition of the new assessment methodology.

Clinical decision process

The clinical decision process often utilizes diagnostic tests as a method to generate information for determining treatment and management plans. Diagnostic tests are often evaluated in terms of sensitivity and specificity which is a useful starting place, but clinical decisions based on test results should also be influenced by expected positive and negative predictive value of the test. The positive and negative predictive values of a diagnostic test are influenced by test sensitivity, test specificity, and prevalence of the disease (or predicted probability that an individual animal or herd has the disease). The positive predictive value provides a probability that the positive test is truly positive, and conversely the negative predictive value provides a likelihood that a negative test animal is truly negative. Often the practitioner may choose to believe one side of the test (example: a test with high positive predictive value that is positive is likely true; while the same test with a low negative predictive value that is negative may not be accurate).

Using the negative and positive predictive values allows veterinarians to incorporate clinical judgement in the test evaluation process and quantify the effect that changes in disease prevalence could have on test or case outcome. Additionally, the economic consequences of misdiagnosis (false positive or false negative) is often not equally distributed and the optimal solution may be influenced by the expected economic consequences. The economic consequences of misdiagnosis may even vary by the expected prevalence (as this drives the overall rate of misdiagnosis in each category). Thus, the decision to retest or further evaluate test positive/negative animals is based on the expected consequences of an incorrect classification (false positive / false negative) vs. the expected cost of accurate diagnosis (true positive / true negative). Several online and decision tools are available for practitioners to calculated the expected positive and negative predictive values as well as the expected economic consequences of disease.

Summary

Clinical diagnosis is a key daily task for veterinary practitioners and using specific criteria for evaluating test results in differing situations can provide increased overall clinical diagnostic accuracy. Decisions often need to be made with imperfect information, but a framework can be developed to guide the process by including the known characteristics of the diagnostic modality, the expected prevalence (or predicted probability) of disease, and the economic consequences of misdiagnosis (false positive and false negative). Using this framework, clinical decisions can be augmented and improved over time as new information is collected.

References

Cow-Calf Reproductive Profiling for Success  
(Parts 1 and 2)  
Brad White, DVM, MS  
Kansas State University  
Manhattan, KS

Key points

- Monitoring reproductive success is an important service to cow-calf clients and visualizing the reproductive history of the herd can be a useful methodology to monitor progress.
- Graphing the calf birth rate by 21-day period over the calving season provides valuable information when diagnosing reproductive problems, evaluating herd economic performance, and optimizing breeding program success.

Evaluation of reproductive success in the cow-calf herd

Reproductive success is a key component of the successful cow-calf herd. Reproductive parameters should be evaluated in light of the herd's production constraints and overall goals. To identify areas for improvement, veterinarians can compare numbers from the herd of interest to standard production targets. The goal is to create a reproductive profile of the herd which contains information regarding the breeding season, maintenance of pregnancy, calving patterns, and the weaning percentages. A key statistic to evaluate is the number (or percent) of calves weaned per female exposed to the bull in the previous breeding season. Our common target for this number is approximately 85-88%. If the herd is below this number, then further investigations should be initiated to determine potential causes of reproductive loss.

Our goal for pregnancy rate after a 60 day breeding season is 95% or greater; while, we may not recommend an intervention or actions unless the pregnancy rate drops below 85%. It is important to realize that pregnancy rates will fluctuate some on an annual basis and may vary based on the age of the cow herd and the current nutritional status. If pregnancy rates are lower than expected, this indicates potential reproductive problems; however, this value does not indicate where the problem is occurring. Typically reproductive problems can be divided into three major areas: female problems (cows not cycling, cows in poor body condition), male problems (bulls with traumatic injuries, bull infertility), or infectious reproductive diseases (viral or bacterial). To assist the herd in overcoming reproductive issues, it is important to utilize the herd reproductive profile to narrow down the potential issues for evaluation.

Front end loading is important to maintain a 365-day calving interval with the majority of the cow herd. By managing average herd age, body condition score, and calving time frame, a producer can keep cows calving on a 365-day or less calving interval. This enhances lifetime productivity of the cow herd and increases economic results to the producer. While the average length of post-partum anestrus is around 60 d, this is variable among herds and in individual cows within the herd. The 90th percentile for length of post-partum anestrus (or number of days at which 90% of the herd would be expected to be cycling) is almost 80 days (or near the maximum days so that a cow can breed back and calve every 365 days. Any problems can cause cows to experience longer post-partum anestrus periods.

Evaluation of the length of the breeding season and distribution of calves born within the calving season can be a valuable diagnostic tool for the cow-calf practitioner. Ideally, we will have 60-65% of cows calve in the first 21 days of the calving season. Another 25% will calve in the second 21-day period followed by 10% of the herd calving in the last 21 day period. This leaves 5% open at the end of a 60 day breeding season. Some breeding seasons may be longer, but a rule of thumb is that in cycling females, we'd expect about 2/3 (66%) of eligible females to conceive during each 21 day period.

Modifications to this ideal calving pattern (either increases in length or changes in distribution) may result from changes in management or disease problems. These patterns may be indicative of specific problems and are useful to narrow the differential list into more discrete categories (e.g. failure to conceive, male problems, pregnancy loss). Using the pregnancy histogram as a diagnostic tool provides the practitioner a cost efficient method of generating a prioritized differential diagnosis list.

Summary

Reproductive success is critical to the beef cow-calf herd and the veterinarian plays an important role in identifying problems in this area. Using the histograms describing the calving pattern can be useful to help herds optimize production as well as diagnosing potential problems when reproductive rates are less than ideal.

References

Adding value to client operations is a key component of many veterinary practices. Practitioners strive to provide the best information to clients in a timely fashion. Often data are collected in the field and require some interpretation to provide useful information to clients. Several tools exist to help in this process. Each client also has somewhat unique situations or restrictions which may limit their ability to control disease: online portals are available to assist veterinarians in providing decision support and customized recommendations for their clients. Veterinarians have several opportunities to add value to client herds by using online tools and providing additional information.

**Mobile apps**

Mobile applications allow field data collection and interpretation for practitioners. The advantage of having the data present on phone or portable tablet is that immediate feedback can be provided to the client. Several mobile apps exist that can be useful for practitioners including a pregnancy analysis app.

Optimal pre-weaning calf health starts with the planned breeding season. Creating a well-defined, relatively short calving season leads to groups of calves that are more manageable from a health stand point because they are at similar age / immunological states. The risk of disease changes with calf age and in a herd with a prolonged calving season managing these risks becomes problematic. A short calving season allows more efficient of grouping calves by age to manage risk status. Tightly grouping calves by age also allows the ability to apply interventions, such as vaccinations, at an appropriate time period to the greatest number of calves. In this case, the production goal of a tight calving season with most calves born early in the season, aligns well with the health management goal of preventing disease in pre-weaned calves. The timing of the breeding season should be planned to match resource availability allowing cows to breed back in a reasonable period and maintain annual calving.

**Online CONSULTs**

The CONSULT (Collaborative, Online, Novel, Science-based, User-friendly, Learning Tools) system has created several modules to help practitioners work through recommendations with their clients. Each tool is built using expertise combined with published literature to generate a series of recommendations for specific diseases and different situations. The CONSULTs model a phone call between a producer and an expert on the specific disease or syndrome: the CONSULT provides a recommendation and the producer can select if they can follow the recommendation or not. The selection by the producer then influences subsequent recommendations. Final results include a customized series of recommendations for each farm.

Initial consults have been created for Trichomoniasis and Bovine Viral Diarrhea:

- [http://www.trichconsult.org/](http://www.trichconsult.org/)

Working through the CONSULTs with clients can facilitate communication on effective disease control measures for specific syndromes on each operation.

**Summary**

Several mobile apps and online tools are present to help practitioners add value to client herds through decision process. Practitioners can apply these tools using field data collection and provide immediate feedback to enhance client communication.
Managing and Treating Bovine Respiratory Disease in Beef Calves
Brad White, DVM, MS
Kansas State University
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In many situations, the infrastructure of the beef industry limits the ability to prevent BRD before it occurs and health care providers must work on managing BRD cases to limit potential outbreaks. Optimal BRD management is based on accurate diagnosis and timely application of appropriate therapy. The BRD management plan can be evaluated based on case outcomes, but these outcomes need to be evaluated over time to determine potential changes in treatment protocols.

Diagnosis of BRD
Therapeutic response is influenced by the timing and accuracy of BRD diagnosis. The most common method for diagnosis is based on visual appraisal of animal clinical signs, and this method has been shown to have relatively low diagnostic accuracy. Cattle illustrate behavioral changes associated with illness, but some of these behavioral changes may be difficult to identify early in the disease process. Finding diseased cattle early in the process is an important aspect of applying appropriate therapeutic regime.

Making sure that cattle initially identified as ill are truly diseased is also an important part of the diagnostic process. Research has shown that improving the specificity of BRD diagnosis is economically viable in the feedyard setting. BRD diagnostic specificity can be improved by applying multiple tests in series and modifying the case definition as appropriate. Commonly measured parameters, such as rectal temperature, may provide some information, but the ability to predict case outcome is limited. However, incorporating additional information regarding the case (clinical illness score, case history, etc) may be useful in generating more accurate predictions of case outcomes. Care should be taken to create a consistent case definition to allow appropriate application of therapeutic products and evaluate case outcomes.

Population therapeutics
Population-level BRD treatment and prevention strategies may most efficiently be employed soon after cattle arrive to the stocker or feeder operation; however, utilization of these tools is based on an accurate estimation of the population risk for BRD. The population risk for BRD can be estimated based on a combination of subjective and objective variables including: historical records, previous history of vaccinations or preconditioning, implementation of management procedures such as castration, arrival weight of calves, the distance traveled to the feedyard. However, this baseline data may not predict the exact risk of the population, but can serve as a guideline of when to implement treatment at arrival.

Metaphylaxis has been shown to decrease the overall morbidity risk compared to negative controls, and the rule of thumb estimate is to expect a 50% reduction in morbidity and mortality compared to non-treated controls. However, research has illustrated that the impact of metaphylaxis varies by the type of protocol selected and the specific outcome of interest. Several factors should be evaluated to determine the appropriate situations to apply metaphylaxis to incoming cattle.

Summary
Bovine respiratory disease is the major illness encountered by calves in the post-weaning production phase and appropriate therapy is dependent on accurate diagnosis. A case definition with accompanying management strategy should be designed for each situation. Evaluation of case outcomes can be used modify and improve the diagnostic and therapeutic strategies in specific situations.

References / Suggested reading
Pre-Weaning Calf Health
Brad White, DVM, MS
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Key points
- Calf wellness prior to weaning requires a systematic approach to reducing disease challenge and building calf immunity.
- The herd calving pattern influences the ability to prevent and control pre-weaning disease.
- Some diseases (e.g. calf scours) are best controlled by managing pathogen exposure.
- Building and managing immunity is important for controlling some syndromes (e.g. bovine respiratory disease).
- Combining knowledge of disease and management factors can facilitate building complete preventative health plan.

Introduction
Pre-weaning disease in beef calves can cause significant problems in cow-calf herds. Several diseases are relatively common (e.g. calf scours and bovine respiratory disease, BRD), but these syndromes do not impact all herds equally. While some disease may be present at a low level in many herds, other herds may deal with outbreaks of disease in pre-weaning calves. The objective of this presentation is to describe best practices for preparing a preventative health program with the goal of optimizing calf health prior to weaning.

Optimizing health management includes managing both the disease challenge and the level of immunity in pre-weaned calves. The specific disease syndrome and epidemiology of risk factors influence where focus should be placed for the preventative health management program. The herd health program can be customized to individual operations to decrease the disease presence in the herd.

Calving management
Optimal pre-weaning calf health starts with the planned breeding season. The calving season in the current year is highly influenced by the previous calving season as post-partum interval influences potential rebreeding times. In other words, herds have reproductive momentum and the calving pattern and length of season tends to be similar year to year unless actions are taken to modify this pattern. Herds can have a negative or positive momentum and this can influence the risk of disease.

Creating a well-defined, relatively short calving season leads to groups of calves that are more manageable from a health standpoint because they are at similar age/immunological states. The risk of disease changes with calf age and in a herd with a prolonged calving season managing these risks becomes problematic. A short calving season allows more efficient of grouping calves by age to manage risk status. Tightly grouping calves by age also allows the ability to apply interventions, such as vaccinations, at an appropriate time period to the greatest number of calves.

In this case, the production goal of a tight calving season with most calves born early in the season, aligns well with the health management goal of preventing disease in pre-weaned calves. The timing of the breeding season should be planned to match resource availability allowing cows to breed back in a reasonable period and maintain annual calving.

Managing calf scours
Calf scours is a common pre-weaning disease and this syndrome can be caused by a variety of pathogens. Most of the etiologic agents are transmitted by fecal-oral transmission and disease results when the pathogen burden on the environment is greater than the calf’s ability to immunologically clear the pathogen. Calves born early in the season may be exposed to a relatively small pathogen burden, but when calves become sick they shed large amounts of pathogen into the environment. Thus, on a herd level, most outbreaks of calf scours occur after sufficient calves have been infected to create an environment where pathogen levels are high. One of the mainstays of calf scours control is managing cattle movement and environment in a manner to decrease potential pathogen exposure when calves are in a high risk period.

Pre-weaning bovine respiratory disease (BRD)
Bovine respiratory disease (BRD) prior to weaning can cause significant problems in cow-calf herds. Most herds have few problems with this syndrome; however, some herds have outbreaks of disease resulting in significant losses. Several research projects have identified common risk factors associated with potential pathogen exposure (e.g. contact with calves new to the herd or stocker calves during the pre-weaning phase). Decreasing maternal immunity as calves age may also play a role in outbreak development as most cases occur between days 80-120 of age.

Producers have the opportunity to immunize calves early in life and this may help decrease the incidence of disease. Research on both viral and bacterial immunizations have illustrated the benefits of these vaccines at decreasing the level of respiratory disease in the herd. However, most research has been performed on cattle at high risk for BRD when entering the feedyard. Managing both the environment and calf immunity are keys to preventing problems with pre-weaning BRD.
Summary
Pre-weaning health management is an important aspect of the cow-calf production system. Creating a systematic plan that includes calving season management, decreasing disease exposure, and increasing immunity is key to optimizing pre-weaning calf health.

References and further reading
Reproductive success and optimal performance in cow-calf herds is influenced by disease prevention, replacement heifer, and bull management. Disease prevention is based on managing both the disease challenge and building an appropriate level of immunity. Bull management is also an important part of the reproductive equation by performing breeding soundness exams and selecting the appropriate number of bulls for the herd. Overall success of the replacement heifer program is based on preparing the heifers for the initial breeding season, selecting the subset of heifers with the greatest chance of reproductive success and managing the breeding program. Combined, these decisions influence overall reproductive productivity of the herd.

Preparing productive loss
Optimizing reproductive success in cow-calf herds relies on combining appropriate immunization and biosecurity practices with the current production system management techniques in the herd. The goal of the immunization program is to match herd immunity to the risks faced. Modifications to the immunization program include not only selection of appropriate antigens, but also matching the timing of immunizations to the time of greatest disease challenge in the environment.

Importing cattle into a cow-calf operation represents a potential source of disease exposure to the resident herd. This risk can be limited through diagnostic testing and an effective quarantine program. The diagnostic tests selected for the operation depend on goals of management, current on-farm disease status, and other state/federal regulations. Prior to implementing any test as a part of import procedures, the veterinarian and owner should decide how test results will impact future decisions (if tests are positive, what will be done with the animals?) Using diagnostic tests as a screening tool to reduce risk of disease introduction from new imports is most effective in diseases with a carrier state.

Bull management
Breeding soundness examinations are commonly performed in yearling bulls. Research at Kansas State has evaluated risk factors associated with failing the initial breeding soundness exam and also the potential to predict if the bull will eventually pass the exam. Selecting the appropriate number of bulls for the herd is critical, and even in multi-sire pastures, the distribution of pregnancies is not evenly dispersed among all bulls. After bulls have entered the breeding herd, an in season evaluation can help identify problems early enough to intervene prior to the end of the breeding season.

Replacement heifer: Preparation for initial breeding season
Preparing heifers to breed early in the breeding season is critical as the timing of calving at the first calving influences the overall lifetime productivity of the replacement heifer. Calving early in the calving season increases calf weaning weight due to age and increases the heifer’s chances of re-breeding. The post-partum interval in heifers is the time required from calving to potential rebreeding. The post-partum interval or anestrus period in heifers can range from 80-120 days; therefore, heifers that calve early in their first calving season have more time to return to estrus and become pregnant earlier in the subsequent breeding season. Calving early has been shown to increase longevity in the herd and increase overall lifetime productivity.

Preparing for the initial breeding season begins with adequate record keeping when the heifers are born and continues through the initial breeding. Heifers that were born earlier in the calving season have a greater chance of attaining puberty in a production system designed to have heifers calve at approximately 24 months of age. Recording heifer birthdates may be useful to identify heifers born early in the calving season. At weaning, weight of the heifers can be evaluated to determine the optimal nutrition program to be sure heifers attain the ideal target weight by pre-breeding.

The goal is to have the heifers reach puberty before the initiation of the breeding season. The onset of puberty is influenced by weight, age, and breed. Age and breed are typically threshold parameters and once a minimum age for a specific breed is attained, greater age is not valuable. Heifer weight is one of the primary drivers of puberty onset and heifers from most breeds reach puberty at 55-65% of mature body weight. The heifers should be managed so that the majority of heifers are pubertal at the start of the breeding season.

Examination of heifers prior to initiation of the breeding season can provide information on the pubertal status of the group and identify potential problems that should be removed from the group. This examination is typically done when heifers are yearling age and near enough the breeding season to provide an accurate prediction of the breeding status of the heifers. The pre-breeding exam
may occur anywhere 2 to 6 weeks prior to breeding. Performing the pre-breeding soundness exam 6 weeks prior to breeding allows time for management changes (nutrition / ration changes) to be implemented; however, the greater time from the actual breeding means potentially less accurate depiction of the reproductive status of the group. Performing the pre-breeding soundness exam 2 weeks prior to breeding provides an accurate depiction of replacement heifer status, but does not allow time to initiate any management changes.

The pre-breeding heifer soundness exam typically consists of several assessments of each heifer combined to make an overall determination of the cohort status as well as identify individual problem heifers. Collected information typically includes body weight, body condition score, age (if known), a reproductive tract score, and a pelvic measurement. These data can be combined in a variety of manners, and we have found using the Ready, Intermediate, Problem (RIP) categorization system as a useful tool.

The RIP categorization allows placing heifers into discrete categories based on the combination of measurements assessed at pre-breeding. Heifers in the RIP category have BCS > 4, are at 55-67% of mature body weight, are cycling and have normal pelvic shape with > 130 sq cm of pelvic area. Heifers in the intermediate category have BCS > 4, are at 50-60% of mature body weight, are non-cycling, and have normal pelvic shape with > 130 sq cm pelvic area. Heifers in the problem category may have immature or problem reproductive tract (pregnancy or free martin), or abnormal shaped or very small pelvic area. Heifers in the Ready category are deemed as ready to breed immediately, Problems should be culled, and Intermediate may eventually be good breeding stock, but are not currently ready.

In herds close to breeding (within 2 weeks), the target is to have 85-90% of heifers in the Ready category. For heifers farther from breeding (6 weeks), the target is to have 65% in the Ready category with the assumption of a homogenous group that the Intermediate heifers will begin cycling by the start of breeding. Problem heifers should be a minimal component of the group and should be culled.

Summary
Replacement heifer management is key to long term beef cow-calf reproductive success, and the metric for a successful program is the percent of heifers that have their second calf in the first 21 days of the primiparous calving season. A successful reproductive program includes replacement heifer management, a disease prevention program, and appropriate bull management.

References / Suggested reading
People often acquire a dog for exercise or play. Some of the common reasons for visits to the veterinarian result from injuries during exercise or play! On the other side of that coin is the intention to exercise and play is often greater than the actual amount of time spent doing it. A consequence of inactivity is the development of obesity and lack of conditioning that can lead to injuries and medical conditions like osteoarthritis.

Whether you are providing advice for how to safely exercise for a canine athlete or working with a client to get their pet dog into shape, there are several components of fitness that should be considered. Just like humans should check with their physicians prior to starting a new exercise program, veterinarians should be consulted on whether a type of exercise is safe and appropriate for a dog. The first consideration is overall cardiovascular and respiratory health. A dog with a heart condition will have a much different exercise plan than a dog with normal cardiac function. Dogs with either congenital or acquired airway disease, particularly conditions like brachycephalic syndrome or laryngeal paralysis will also need to tailor their exercise program to minimize risk of overheating. For dogs that are overweight or obese, the process of weight loss will require dietary management and an exercise program that helps burn calories while minimizing the impact of the bones and joints, swimming is a good example of low impact cardiovascular exercise. The dog’s conformation may also dictate the types of exercises that it can safely perform. For example, a chondrodystrophic dog like a dachshund will not be able to jump as high as a nonchondrodystrophic dog like a Papillon even if they are both the same height at the shoulder.

The 5 components of a balanced exercise program include flexibility, proprioception, strength, balance, stamina. The important part of exercise that ties it all together is the mental exercise of the dog learning new behaviors. Prior to any exercise the dog should warm up by trotting for 5-10 minutes. Included in this time can be changing direction, going in concentric circles in both directions. Transitions from sit to stand, stand to down and sit to down will also be low impact warm up. A vigorous rub-down or massage at the end of the exercise is a great way to end the warm up period. The benefits of warming up include getting the blood (and therefore oxygen and nutrients) flowing to the muscles and increasing temperature of the muscles. As muscle tissues are warmed they increase the force of contraction (through more efficient enzymatic function and enhanced ATP generation) and speed of relaxation. This increases power, speed and reduces the risk of overstretching. Warm tissues (muscles, ligaments, tendons and fascia) are more elastic, providing greater range of motion and decreasing the risk of strains and sprains. In addition, neural pathways are primed for the subsequent activity. A warm up is an active process and does not include passive stretches.

Flexibility is a key component of fitness and is accomplished through active stretches. The goal of active stretching is to get the muscles, tendons, ligaments, fascia and joints lubricated so that injury risk is reduced. Active stretches of the spine are valuable in increasing movement and protecting the spine. Side bending with the use of lure to bring the dog’s head to its shoulder and then its hip and then the hind foot is an easy way for an untrained dog to increase flexibility. Be sure that is performed on each side! This exercise can also be diagnostic for areas that are particularly tight. Dogs can also weave between the owner’s legs or do figure 8’s to get the side bending Dogs naturally tend to stretch by doing a play bow, this stretch incorporates the triceps and shoulders, spinal extension and stretching of the hamstrings. The play bow can be incorporated into a regular routine and even placed on cue. Having the dog place his front paws on an elevated surface or wall targets the lumbosacral spine and hip flexors. This exercise is important for dogs that will be repeatedly up on their back legs (like search dogs). Having the dog wave or perform a “high five” helps actively stretch the muscles of the forelimb. These active stretches can be done every day and ideally should be performed prior to any exercise.

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. Just like a gymnast learns to land on a balance beam, proprioception can be learned and connections between neurons can be strengthened, and the number of synapses increased. Dogs are often describe as “front wheel drive” and are notorious for not recognizing where their back feet are. Teaching a dog to put all 4 feet in a box increases hind end awareness. Having a dog “target” an object with its back feet or learn to walk backwards all increase proprioception. Proprioception and balance are interrelated. Balance incorporates strength and body awareness and is invaluable 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. Once a dog is taught to place all 4 feet in a box the next step is to make the box smaller and smaller which requires the addition of core strength to maintain his balance. This type of work will allow the dog to be able to perform complex tasks for longer periods of time without fatigue and maneuver on unstable surfaces safely.

Strength training or anaerobic exercise stimulates predominantly Type II muscle fibers. With training, the local effect is an increase in glycogen storage in the muscle fibers to allow increased glycolysis (the process by which Type II muscle fibers create
energy to contract.) 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. Typically that external force is the weight of its own body, although sometimes resistance is applied like in weight pull or tugging, or underwater treadmill. Most strength exercises begin at a very basic level and will target the front limbs, the hind limbs, the core or multiple areas. To increase the difficulty of a strength exercise it is possible to increase the duration a posture is held, the number of repetitions of the posture or change the surface to unstable or inclined. For progression in difficulty, only one parameter should be changed at a time and nothing should be changed until the dog is able to perform the exercise with good posture. Examples of strengthening exercises for the trunk and neck include: sit up and beg, diagonal leg stands, roll over, crawl, 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; sit-to-stand on a hill (facing left right and up); high tugging, and jumping.

Stamina is sometimes referred to as endurance, however most of our pets and even most canine athletes do not come close to the classic endurance athlete, the sled racing dog. Stamina is the amount of effort exerted over a period of time. Most dogs work in bursts of energy. Stamina is influenced by physical conditioning, which incorporates cardiovascular, respiratory and musculoskeletal input. Exercises that build stamina 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).

Stamina is built through aerobic exercise in which the dog is trotted for at least 20 minutes (or swims continuously for 5 minutes). Dogs running in a sprint will be performing anaerobic exercise and accumulate lactate. This phase only lasts for the first few minutes, when dogs convert to an efficient aerobic metabolism. With increasing duration of exercise, dogs convert from utilizing carbohydrates to burning fat for energy. The ideal gait for stamina is a trot (diagonal limbs move together), this balanced gait works the limbs evenly. Because repetitive concussive forces of trotting can lead to joint injuries, the best surface for extended periods of trotting are soft, forgiving surface like dirt, wood chips, or a rubberized track. This type of repetitive impact activity should be restricted to dogs that have full closure of their growth plates (12-18 months of age). Land treadmills can be useful but have some limitations. First for growing dogs, they should be used as a walk to minimize bone and joint concussion and acclimate the dog to the exercise. When dogs are worked at a trot or faster the length of the tread should be at least 2.5 times the length of the dog or else the dog will shorten its stride to accommodate the tread. The treadmill requires less muscle activity than running on land, therefore, treadmills should be a part of a balanced approach to stamina and not be the only exercise for athletes that require significant stamina in their sport (mushing, field work, and herding). Swimming is a great way to build stamina with minimal impact on the bones and joints. As a stamina exercise, swimming should be continuous activity for at least 5 minutes rather than repeated fetches into the water. Dogs have variable swim styles and may not equally utilize their front and rear limbs, so the strength building may need to be balanced out with targeted exercises. The underwater treadmill, while often utilized in a rehabilitation program provides some advantages for conditioning. The treadmill ensures that all limbs are being utilized so that the cardiovascular benefits are coupled with a balanced strengthening plan. Depending upon the amount of water in the treadmill tank, you can vary the buoyancy and resistance.

Rest and recovery is essential to preventing injury. At the end of exercise a cool down should consist of a trot, followed by a walk, to aid the body in preventing lactic acid build up in the muscles.
Who are our common canine athletes?

Canine athletes constitute a special population of dogs. Similar to human athletes, there are professional and amateur athletes who train regularly for competition or working careers. Mirroring their human counterparts, there are also weekend warriors. Competitive dog sports are one of the fastest growing segments of the pet market. Although this list is not comprehensive, it includes some of the most common sports or those in which injury or performance issues may be common. Canine agility first appeared in the UK in 1978 and was rapidly adopted by US dog lovers. Agility is the fastest growing canine sport and places unique demands on the dog and the handler. Agility requires bursts of speed, control, jumps and often rapid deceleration down an A-frame, dog walk or teeter. Flyball is a relay race where teams of 4 dogs compete by racing over 4 jumps, hit a spring-loaded box releasing a tennis ball and returning over the jumps. These dogs require power over the jumps and coordination in order to hit the box and grab the ball in one smooth motion. The impact of slamming into the box while turning has potential for a variety of injuries. Disc dog competitions are made up of disc tosses with the goal of number of catches in a set time, creativity and choreography of the catches and sequences or distance catches. The events pairing a dog and handler involve speed, turns, thrust and aerial moves, sometimes with forceful landings. It is estimated that over one million dogs participate, although most do not actively compete. Lure coursing is a speed race following a plastic lure across 600-800 yards in an open field. These dogs are classic sprinters. A new canine sport of nose-work adapted from the scent detection trials for working dogs has recently swept through the US. This sport is less of a physical sport than a mental and olfactory sport, for competition level, the dogs do need some physical stamina and ability to search up on their hind legs to locate odors that are hidden above nose level. Weight pull competition is a growing sport, especially for the “bully breeds”, although one of the top weigh pull dogs in its weight class is a Pomeranian! These dogs need pure brawn! Field trials and hunt tests attract hunting dogs in a sport that recreates the features of the hunt in controlled and competitive events. These dogs require physical stamina and mental focus. The event may also lead to injuries and environmental exposure. The protection sports (Schutzhund, Ring Sport, French Ring, Mondio Ring) recreate the tasks necessary for police dogs in a competitive arena. These dogs perform obedience which includes specific agility like a retrieve over a 6 ft slanted wall, tracking and several demonstrations of protection including biting and holding a fleeing person (fully equipped with a protective bite sleeve – although that doesn’t keep them from being bruised or knocked down). These dogs are subject to all of the same physical challenges as professional police dogs. Search and rescue dogs are expected to locate missing or trapped persons. The Federal standard for disaster (urban) search and rescue dogs includes obedience, agility (climbing ladders and traversing unstable or elevated surfaces), control at a distance, and rubble search. Wilderness or area search dogs are generally required to be able to locate the source of odor in a large wilderness area (80 acres for some certifications), requiring stamina and an ability to navigate various terrains.

Defining the performance problem

A performance dog client may approach you to evaluate their dog for poor performance. Most performance clients will observe changes in behavior and performance much earlier than an average pet owning client. These keen observations make veterinary assessments more challenging because the problems will be often very subtle! It is important to seriously consider that even if the dog appears normal on routine examination, that there is a concern that warrants attention (it may not be medical etiology, but take the concern seriously and work with the client to ensure that there isn’t a medical component, you may uncover a condition at an early and maybe even more treatable stage).

As with any case, you want to obtain a complete medical history. In that history you will need to understand what the client perceives as poor performance. Can they specifically define the tasks at which the dog is struggling? For example, an agility dog may be having difficulty with jumps, but no problems with contacts or stamina. Further, exploration should determine whether it involves all jumps or only doubles and triples, or only when they are jumping and turning. Does the dog have difficulties throughout the day or only at certain times? Does the dog have difficulty every day? Is the problem limited to competition or is present in training? Does the dog have difficulty jumping in other situations (into the car or off of an elevated surface)? It becomes clear that an understanding of the tasks that the dog performs will be invaluable in evaluating a performance issue. One of the best tools will be to review video of the dog to help characterize the problem. Most performance dog handlers will have video of the dog performing normally; this can be compared to video when the dog is having performance issues. In addition to the clients whose dogs were performing well and developed an issue, you may be consulted by individuals with dogs that were considered to have excellent potential but are not measuring up. If the dog has never achieved top performance, then congenital problems should also be considered along with all of the acquired or delayed onset problems that are in the differential list for a dog that has proven performance. The history should also include standard components of a medical history, with a special emphasis on

- Nutrition: including supplements, changes in diet, changes in appetite, thirst
into obstacles. For any dog that has a detection component to its performance, olfactory impairment should be considered. It is
evident in dogs at rest. Any increase in stridor with exercise warrants a laryngeal examination. Respiratory impairment will also
impact heat exchange!

Components of performance
The ability of an athlete to perform is a combination of the dog’s natural physical ability, its motivation (or drive to perform), its
persistance, the training methods and training frequency. In addition, since most canine performance events are “team sports”, the
performance of the handler must be factored in. Athletes may experience unexpected setbacks in their performance and the challenge
for the clinician is to separate out the canine health factors from the training factors and human factors.

Physical factors that should be considered in evaluating a dog for poor performance involve every organ system! The first and
most obvious question is whether the dog is in appropriate body condition. A canine athlete should have a body condition score of 4-5
out of 9. An over-conditioned dog is carrying unnecessary weight which will compromise heat exchange and contribute to joint
degeneration. An under-conditioned dog may not have the muscle to perform the task at hand, especially if endurance or repeated
exercises are required. Body condition scoring does not provide an insight into the nature of the muscle condition. A muscle condition
score has been developed but is focused on evaluation of muscle wasting. Dogs that lack core strength or muscle conditioning will not
have the defined muscles that we expect of a professional athlete. A healthy musculoskeletal system is essential for all canine athletes.
This includes bones, joints, muscles and nerves. The first component of the exam is to evaluate the spine to confirm that further
manipulation or activity will not compromise the dog. Once the spine is cleared, evaluate posture at a sit and a stand and through
transitions between positions. A thorough assessment of gait is the next step. Pressure sensitive mats for gait analysis are ideal but not
always available. A simple approach commonly used by Dr. Chris Zink for evaluating gait is to use corn starch and yoga mat. Dip the
dog’s feet in corn starch and then walk or trot them over the mat. The footsteps can be evaluated to show disparity in pressure,
distance or tracking. Slow motion video of the dog walking and trotting over cavalettis, up and down stairs, or around cones arranged
in a figure 8 may also reveal subtle abnormalities. After gait observation, careful palpation of each bone, muscle and joint will be
necessary. Reflexes, range of motion and nerve glides are part of a complete examination. Clearly any abnormalities warrant further
diagnostics.

The cardiorespiratory system is a critical component of the canine athlete. The basic evaluation of cardiac performance will include
auscultation, pulse rate and quality, an ECG, blood pressure, electrolytes and hematocrit. Additional diagnostics might include
imaging of the heart (radiographs, echocardiogram) and extended ECG monitoring (Holter). For the canine athlete, the resting heart
rate is expected to be on the low end of normal. Evaluation should be performed at rest and after exercise. Twenty minutes of trotting,
treadmill or fetch should provide an indication of the heart rate and rhythm after exercise, equally important will be the time to return
to baseline. Dogs that are conditioned will have a more rapid return to baseline heart rate, likely within 30 min or less. The respiratory
system should be evaluated by auscultation, pulse oximetry and blood gases to evaluate gas exchange (CO₂ and O₂). Venous blood gas
collected anaerobically and analyzed without delay will give an approximation of CO₂ but will not reflect O₂. Similar to the
cardiovascular system, the respiratory system should be evaluated at rest and after exercise. Subtle restrictions to air flow may not be
evident in dogs at rest. Any increase in stridor with exercise warrants a laryngeal examination. Respiratory impairment will also
impact heat exchange!

Heat intolerance can impact performance. Many factors contribute to heat tolerance. They include the endogenous generation of
heat through metabolism and muscle activity, the exogenous temperature and humidity and the ability of the dog to effectively
eliminate heat (primarily through redirecting blood flow to the nasal and oral cavities and panting). Sudden changes in environmental
temperature may impact heat tolerance. It has been suggested that most dogs require 2 weeks to adequately acclimate to a change in
ambient temperature. In addition, some dogs may not be able to adapt to high environmental temperatures when they spend most of
their time in air conditioning. Veterinarians should be cautioned that elevated rectal temperatures during active exercise in conditioned
dogs can reach temperatures of 108 F (42.2C) without adverse effects; however these temperatures should return to baseline within 20
minutes. It has been suggested that dogs that experience heat stress once will be less heat tolerant in the future, although this
phenomenon has not been well studied.

Metabolic conditions should be considered. Baseline bloodwork should include a complete CBC, chemistry profile and endocrine
testing. In my experience, healthy search and rescue dogs often have lower white blood cell counts than pet dogs.

Special senses may also impact performance. Dogs with a loss or impairment of vision may be reluctant to perform or may crash
into obstacles. For any dog that has a detection component to its performance, olfactory impairment should be considered. It is
challenging to evaluate olfactory function in dogs and often structural brain or nasal disease will need to be ruled out. Although there are numerous causes of anosmia in people, few drugs and diseases have been shown to impact canine olfaction. Training issues need to be considered if there is no clear organic cause. Dogs are excellent at reading human body language but hearing loss should also be evaluated in cases in which a clear cause of performance impairment is not identified.

The greatest challenge is to find that the physical health of the dog is not the source of the performance issue. Some clients will want to exhaust all possible physical causes before considering that the issue is with training or even with their own physical or mental health. It was documented that dogs that responded to 9/11 had more training issues when their owners were suffering from PTSD. The performance dog client can be one of your most challenging, yet most rewarding clients! A thorough and methodical approach will keep them happy and performing for years to come!
Flexing Your Muscles with Therapeutic Exercise
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Physical rehabilitation is an important and growing area of veterinary practice. The perception of rehabilitation is associated with the use of equipment like lasers, therapeutic ultrasound and underwater treadmills. These are valuable components of a complete program; however, they are frequently not available to the client for use at home! If you have ever had physical therapy, you know that the balanced plan that leads to long-term change is one that incorporates a home exercise program.

As in any rehabilitation plan, when considering therapeutic exercise as part of the plan, the first step is to identify the goals of therapy. The goals of therapy for a tetraparetic Dachshund are likely different from those of an agility dog with an iliopsoas strain. This will influence the type of exercises and the intensity of the program. Communication with the client regarding the expectations of the rehabilitation at the onset will help set you up for success. An effective therapeutic exercise program requires a strong commitment from the client. Exercises should be completed on a daily basis and in addition to time invested, may require specific equipment to be built or purchased. The most effective way to establish a home exercise program is to teach the dog the exercise behaviors. Dog training may not be familiar to the client and therefore a major component of the program will be educating the owner on how they can elicit the desired behaviors. Positive reinforcement training is the recommended method of training. There are numerous techniques that will aid in teaching the behaviors. Although clicker (or marker) training is invaluable for precise training, for many clients who are unfamiliar with training techniques, introducing the use of a clicker with the requirement for precise timing and coordination may be overwhelming. If a client and their dog are familiar with clicker training, this is an invaluable tool for teaching therapeutic exercises. The options for teaching the exercises include shaping, luring, capturing, positioning and targeting. Shaping involves rewarding the dog for offering behaviors that are close to the desired behavior and gradually waiting until the behavior becomes closer and closer. Eventually the dog is only rewarded for the complete desired behavior. It is thought that the process of the dog “figuring out” the behavior helps solidify the final behavior. The caveat is that the dog can get frustrated if they do not reach the final behavior with a reasonable amount of effort. Luring is one of the quickest and easiest ways to get a behavior. For example, to elicit a sit behavior, a treat can be shown to the dog and then raised above the dog’s nose; as the dog lifts its head to follow the treat, it will naturally move to a sitting position. For luring to be effective, treat placement is critical. Ideally, the lure is eliminated once the behavior is being performed readily. The lure is then replaced with a reward. The difference between a lure and a reward is that the lure is provided BEFORE (or during) the behavior and a reward is provided AFTER the behavior. Capturing a behavior is taking advantage of the dog’s tendency to spontaneously exhibit the behavior. For example, a dog may perform a bow stretch every time it gets up out of its crate. If the bow is rewarded regularly, the dog may start to offer it. Positioning is the process of physically manipulating the dog to achieve the desired behavior. Positioning should only be used to fine tune exercises, since the process of the dog moving its body is part of the neuromuscular stimulation that is desired with therapeutic exercise. Targeting is a valuable part of training. The dog can learn to touch its nose, paw or other body part to your hand or a target. This allows you to have more variety in your training and is part of an active learning process for the dog. One example of targeting would be to have the dog touch your hand with its paw. This can then be used to move from a simple “shake paw” to an elevated paw (high five) or “wave” depending on the goals of strength and flexibility training.

When starting a therapeutic exercise program there are several components of a balanced plan. The first step is to always include a warm up. The warm up allows increase blood flow to the muscles, joints and connective tissue, increasing mobility and decreasing risk of injury. The specific exercises will include flexibility, strength, stamina, balance and proprioception. Flexibility exercises can be performed on a daily basis, but should always be after a warm-up. Active stretches, where the dog is performing the stretch, rather than being physically manipulated into a stretch are recommended. Proprioception exercises are generally low impact but focus on body awareness and can be done on a daily basis. Strength training should be performed intermittently to allow time for muscle recovery. Strength exercises can be targeted to forelimbs, hindlimbs, core or full body. Balance work is a combination of proprioception, strength and depending on the exercise, flexibility. Stamina or cardiovascular work can be challenging to be done safely. For immature patients or those with arthritis, water work is ideal. This can be accomplished with an underwater treadmill or swimming. For dogs free swimming, the ideal is for extended duration of swimming with engagement of all limbs. Land treadmills are valuable for controlled exercise, but the treadmill length is important to consider. Ball retrieves are commonly used by client as a form of cardiovascular exercise. The sudden stops and turns often associated with chasing a ball can lead to further injury. Following exercise, a cool down period is recommended.

Case studies of dogs will highlight the application of the principles of therapeutic exercise for musculoskeletal injuries, hip dysplasia and reconditioning after severe illness.
Health Hazards and Rehabilitation of Dogs that Play or Work Too Hard
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Working dogs constitute a special population of dogs that are specifically trained to perform tasks that assist people in a variety of settings. These dogs fall into the general categories of service dogs, protection dogs and detection dogs. Service dogs are recognized by the ADA as those that have been individually trained to do work or perform tasks (related to the disability) for an individual with a disability. Guiding dogs are the prototype of service dogs, but numerous other disabled individuals have benefitted from the expansion of service dog tasks. Currently, service dogs can assist people with hearing loss, mobility impairment, post-traumatic stress disorder, autism, diabetes or seizures just to name a few. These dogs have a variety of mental and physical demands to meet. Obesity is a major risk among service dogs. Protection dogs are typically police, military or private security dogs that function to protect property or an individual. These dogs rely heavily on athleticism and bite work. These dogs are typically Shepherd breeds and often have a predisposition for hip and lumbar spine disorders. Their work can lead to a variety of environmental exposures and injuries. Detection dogs work in a wide variety of environments and are exposed to various hazards depending on their specific jobs. Detection dogs that are trained to find explosives are at potential risk of blast injury, as seen with the improvised explosive detection dogs (IED) used with the military, and there have been reports of these dogs ingesting some of the compounds (e.g. cyclonite/C4). Drug detection dogs are at potential risk of ingesting or inhaling the compounds for which they are searching, but also are at risk of gunshot wounds from unhappy criminals or cross fire during police actions. Search and rescue dogs are trained to find live humans and can work in wilderness or urban environments. In wilderness settings, the risks are similar to hunting dogs and include ticks, snakes, porcupines and environmental hazards. In urban or disaster settings, dogs work around collapsed buildings and are exposed to the hazards of unstable surfaces, potential toxic chemicals and other environmental hazards. For most working dogs heat stress is a major concern. Some of the detection dogs, however, work in more controlled environments like the USDA Beagle brigade which works in the airport, screening luggage. Cancer detection dogs typically work in a controlled laboratory setting and the physical risks are limited.

Veterinarians that have the opportunity to work with these dogs recognize the unique considerations in the care of the dogs and the relationship between the dog and the handler. There are multiple differences between a working dog and the typical pet dog patient. The relationship between the dog and handler is unique, in that in some cases the handlers rely on these dogs for personal safety (e.g. explosive detection dogs) and the ability to function (e.g. guiding dogs, mobility assistance dogs). The bond between the dog and the handler is very strong. This relationship often means that the handler will identify a change in behavior long before an obvious physical abnormality is apparent. In addition, the dog will sometimes change their behavior in response to a physical or mental malady in the handler. When examining most working dogs it is important to include the handler in the process. This is especially important in protection dogs where the dog may not be amenable to handling by strangers. For service dogs, it is critical for the handler not to be left without their dog whenever possible. Educating handlers on the impact of any procedures or medications on the dogs’ working performance is also important.

A limited number of studies have reported the occupational hazards of working dogs. For military working German shepherd and Malinois, the top 5 causes of death were degenerative joint disease, neoplasia, spinal cord disease, aging changes and gastric dilatation and volvulus. Early retirement (at < 5 years of age) was often the result of behavioral problems or heat stroke, whereas older dogs retired as a result of arthritis or spinal cord disease. Gunshot wounds are seen in both police and military working dogs. Urban police German shepherds are brought to the emergency service most commonly for musculoskeletal problems or gastrointestinal disorders. Interestingly, back problems are equally represented in pet and police German shepherds. Lameness, however, is more common in police German shepherds. Fitness and conditioning is rarely included in police dog training and has only recently been incorporated into the military working dog program. Given the incidence of lameness in working dogs, incorporation of routine fitness may reduce the incidence of lameness. Incorporation of a rehabilitation plan for those dogs that develop lameness will get the dogs back to work sooner. All German shepherds should be evaluated and guided through a core strength program to help support the lower back and hips, this is especially important in working dogs since lumbosacral disease could be career ending.

In a study of the response to 9/11, search and rescue dogs had limited morbidity, with minor cuts and scrapes being most common. Dehydration is a risk in any working dog that is highly motivated to do their job and is working in a hot or cold environment. These results have been replicated under a variety of disaster response settings, consistently identifying dehydration and minor cuts and lacerations as primary concerns. Urban search and rescue dogs incorporate components of agility in their training that demand proprioception (climbing ladders) and core strength (unstable surfaces). It is possible that this training helps to minimize the seriousness of the injuries during disaster deployments.
Similar to police dogs, where fitness and conditioning are not a routine part of the training, service dogs often retire as a result of orthopedic issues. In addition, behavioral problems, skin allergies and ear disease are common in the service dogs. Cancer is the cause of death in approximately 30% of service dogs and 40% of search and rescue dogs.

Many canine sports have evolved from working tasks. Canine athletes represent a rapidly expanding population of pet dogs. Although this list is not comprehensive, it includes some of the most common sports or those in which injury may be common. Canine agility is a critical component of police and search dog work. Competitive agility is the fastest growing canine sport and places unique demands on the dog and the handler. Agility requires bursts of speed, control, jumps and often rapid deceleration down an A-frame, dog walk or teeter. Shoulder injuries are common in these dogs. More frequently iliopsoas strains are being recognized in these dogs with their explosive movements and risk of hyperextension of a hind limb. Flyball is a relay race where teams of 4 dogs compete by racing over 4 jumps, hit a spring-loaded box releasing a tennis ball and returning over the jumps. These dogs require power over the jumps and coordination in order to hit the box and grab the ball in one smooth motion. The impact of slamming into the box while turning has potential for a variety of injuries. Disc dog competitions are made up of disc tosses with the goal of number of catches in a set time, creativity and choreography of the catches and sequences or distance catches. The events pairing a dog and handler involve speed, turns, thrust and aerial moves, sometimes with forceful landings. It is estimated that over one million dogs participate, although most do not actively compete. Lure coursing is a speed race following a plastic lure across 600-800 yards in an open field. These dogs are classic sprinters. Weight pull competition is a growing sport, especially for the “bully breeds”, although one of the top weigh pull dogs in its weight class is a Pomeranian! There are no published reports of injuries associated with this sport, but similar to power lifters, it can be anticipated that muscle strains, tears, joint and spinal injuries could result. Field trials and hunt tests attract hunting dogs in a sport that recreates the features of the hunt in controlled and competitive events. These dogs require physical stamina and mental focus. The terrain may also lead to injuries and environmental exposure. Injuries are similar to the search and rescue dogs, with foot and musculoskeletal injuries most common. The protection sports (Schutzhund, Ring Sport, French Ring, Mondio Ring) recreate the tasks necessary for police dogs in a competitive arena. These dogs perform obedience which includes specific agility like a retrieve over a 6 ft slanted wall and components of criminal apprehension. Spinal injuries can result from a running bite on a stationary decoy. Muscle and joint injuries are a risk, especially for dogs that are not adequately warmed up prior to exercise. The classic endurance athlete is the sled dog. There has been extensive research with these dogs to determine health and performance characteristics. One of the most common problems that these dogs faced historically was gastric ulcers, this has been greatly reduced through improved management and preventive care.

Common injuries in all types of working and performance dogs include footpad injuries and nail trauma. Some dogs are able to wear booties to protect their pads, but many dogs, especially those required to maneuver on unstable surfaces need the gripping of their toes. Flyball competitors often wrap their dogs’ feet which can protect their pads from abrasions but may alter the mechanics of their jumping and box turn. The shoulder joint is particularly vulnerable in agility and search dogs. There is limited data on the incidence of shoulder injury in police or other working dogs. Carpal injuries are common in dogs that land hard on their front legs. This is a particular risk in dogs competing in flyball and dogs landing hard after jumps or after scaling high obstacles like the slanted wall in Schutzhund. Carpal injuries are also reported in racing Greyhounds and earth dog competitors. Gracilis myopathy appears to be more common in German shepherds, whereas iliopsoas strains are common across all types of athletes.

In addition to the treatment of acute disorders, veterinarians need to be active in the prevention of disease and injury in working dogs. General preventative health care includes appropriate vaccinations. All working dogs should receive core vaccines. It is recommended that search rescue dogs receive Leptospirosis vaccines. There is no evidence that detection dogs or dogs that compete in the sport of flyball are at higher risk for canine influenza (CIV H3N8), although it is rare to find titers to CIV in any of these dogs. Due to the likelihood of travel, flea and tick control and heartworm prophylaxis are important in most working dogs even if their primary residence is not endemic. Prophylactic gastropexy is the standard for all military working dogs and should be considered for working and performance dogs at risk. In general female working dogs are spayed; however, they should not be spayed until after closure of their growth plates. Appropriate diet and nutrition are critical for working and performance dogs. Many service dogs and some working and performance dogs do not get sufficient exercise and are prone to obesity. It is well recognized that obesity contributes to arthritis and can shorten the life of a dog. Encouraging an exercise program that fits with the handler’s disability or lifestyle is also valuable preventive medicine. Research on the best composition of food for different types of working dogs is ongoing. In addition, the timing of feeding may impact work performance.

Once injured it is important to proactively return these dogs to top physical condition. It is recognized that deconditioning will start to occur within 2 weeks of inactivity. Early implementation of a rehabilitation or reconditioning program will serve to get the dog safely back into top performance. Working dogs serve vital roles in our society and it is our duty as veterinarians to provide them with the best care so they can do their jobs well and safely. We also need to be aware of and advocate for humane care of working dogs, helping to recognize and prevent stress, encourage positive training methods and appropriate environmental enrichment.
One of the biggest challenges for athletes of any species is to balance the generation of heat produced during exercise with the removal of heat to maintain a safe body temperature. One of the most consistent problems encountered by search and rescue dogs in deployments to natural or manmade disasters is dehydration. The environments of disasters are typically adverse and availability of water may be limited. This is a similar situation for military working dogs and dogs of the Border Patrol. Some of the canine athletes are also at risk for dehydration during their competitions.

Exercise and exposure to high ambient temperatures (>80°F or 26.7°C) leads to an increased transfer of heat through the bloodstream, more than is conducted through the superficial tissues. When high ambient temperatures advance above this range, temperature sensors in the hypothalamus respond with thermoregulatory pathways to maintain homeostasis. During exercise, dogs exert energy that leads to heat generation. Dogs exercising in high ambient temperature and humidity require increased energy to cool. Heat is eliminated through four major mechanisms; conduction, convection, radiation, and evaporation. When environmental temperatures are above approximately 90°F (32.2°C), evaporation is the most essential mechanism for thermoregulation. The ambient heat and humidity will reduce evaporative heat removal. Humans and horses rely on evaporation of electrolyte rich sweat to dissipate heat produced by exercising muscles. Dogs and several other species have limited capacity to sweat and rely predominantly on panting for heat exchange. Dogs do sweat through their feet and through other small regions, but this does not contribute significantly to either heat management or electrolyte loss. Temperature regulation in dogs is primarily a function of respiratory exchange. In dogs, this occurs through panting by bringing large quantities of air in contact with the mucosal surfaces of the nose and mouth. Because they are limited to evaporative cooling through panting, dogs are vulnerable to heat stress and hyperthermia. Proper brain cooling is critical in exercising dogs. During exercise, the temperature of the brain is lowered, contrasted with a rapid increase in carotid blood pressure. Countercurrent heat exchange between warm arterial blood that is supplying the brain and cool venous blood draining the nose and mouth allows the brain to cool. This heat exchange takes place at the base of the brain, forming a rudimentary carotid rete. This carotid rete is greatest during exercise and works as a protection mechanism against overheating in dogs.

Hydration is critical for effective performance. It contributes to the heat balance in several ways. First to maintain brain cooling, adequate circulating volume is necessary. Second, efficient evaporation from the airways and nasal passages requires hydrated tissues. Third, without adequate circulating volume, perfusion of muscles will be limited and oxygen delivery may be impacted, leading to an increase in anaerobic metabolism, lactate and heat generation. In human athletes, exercise in the heat not only leads to fluid loss but also results in significant electrolyte loss. Numerous industries have researched and promoted oral electrolyte replacement solutions for human athletes. As a result, many dog owners are familiar with and actively consume these products. The logic is: “what is good for me is good for my dog”. This approach however fails to account for the difference in physiology between dogs and humans. The addition of electrolytes in hydration fluids has the potential to increase water loss through diuresis of unneeded sodium or sequestration of fluid in the gastrointestinal tract. There are however, some aspects of electrolyte solutions that may have a benefit in canine athletes. The first and foremost is palatability. Many dogs that are competitive athletes or intense working dogs will override the physiologic signals driving thirst. Use of a highly palatable liquid may help to keep these dogs drinking. There are numerous commercial electrolyte products promoted for hydration in dogs, but there is no data on the safety or efficacy of these products under these conditions. The concern that dogs working in austere environments are unable to maintain hydration and will be at greater risk of heat injury has led to the common practice of “prehydration”. Dogs will be preloaded with subcutaneous fluids prior to working. This practice is implemented by individual handlers as well as veterinarians. The practice has been the topic of much debate. Concerns about the practice include potential for introduction of infection, especially when this is applied in the field. Additionally, the fluids represent added weight that could increase the work load of the dogs. The fluids also can migrate and interfere with harnesses or even limb mobility. There are no reports of safety or efficacy of this technique in the field.

We conducted two separate cross-over studies compared the effects of water versus oral electrolytes versus subcutaneous fluids as pre-hydration strategies. The first study involved Border Patrol dogs screening vehicles at the Texas border in the summer. The second study involved Border Patrol dogs tracking in the desert in the summer. Each dog was evaluated on each of 3 days. They were randomly assigned to receive either water, oral electrolytes or subcutaneous fluids. Dogs were then provided water or electrolyte solution at specified intervals. The dogs were monitored for signs of heat stress, while they worked. Core body temperature, activity and blood and urine were monitored.

The dogs all found the oral electrolyte solution to be highly palatable and consumed more than the groups only offered water.
The most interesting data was that the dogs tracking in the desert reached core body temperatures in excess of 107°F (41.7°C) without adverse effects. It is unknown if these dogs were able to sustain these high working temperatures because of acclimation to the environment or conditioning, which may be more important factors than the approach to hydration.

Recommendations for canine athletes based on this study are the following: Condition your dog, regular athletic challenges will help a dog to adapt and perform in adverse environments. Offer your dog water, if that your dog does not drink regularly you can consider an oral electrolyte solution but be sure it is palatable. Most oral electrolyte solutions have not been tested for safety and results from one product may not translate to others. Although this study did not evaluate dogs that were not accustomed to the high temperatures of the desert, it is recommended that exercise intensity is increased gradually when a dog (or the seasons) transition. In some situations, subcutaneous fluids may be required. For example in disaster or remote settings when water is not readily available it may be advantageous to have the dog “carry its own water” in the form a subcutaneous fluids.

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Working dogs fulfill a variety of essential roles to protect and serve. Veterinarians can play a critical role in maintaining the health and the physical and behavioral performance of these dogs. The convergence of medical knowledge (including behavior) and a thorough understanding of the job requirements of the working dog will aid in providing the best care for both the dog and the handler. Unfortunately, there is limited information in the veterinary world about the unique demands of different types of working dogs. In response to this knowledge gap, the Penn Vet Working Dog Center opened in September of 2012 as a national resource for research, education and training of detection dogs. As part of the research mission, data collection encompasses genetic, behavioral, physical traits of the dogs, training applications and human factors. The ultimate goal is to prepare for future demands and facilitate detection dog research through a breeding and training program that will implement, test, and disseminate the knowledge gained.

Although there are numerous working dog careers and a long history of dogs aiding humans, the focus on harnessing the exceptional olfactory capacity for service to humans outside of traditional hunting careers is relatively recent. Military working dogs have been a vital component of many historical conflicts but really became prominent when the Germans started utilizing them in WWI. In the military, dogs were used for several functions including patrol, messengers, transportation (using sleds or carts) and ambulance dogs. The US did not start utilizing dogs extensively until 1942. Most veterinarians will have limited contact with active military working dogs, but now that retired military working dogs can be adopted, there may be an increase in exposure to these retired dogs. In addition, dogs from the Department of Defense breeding program at Lackland Air Force Base that were unsuitable for military careers are available for adoption. Sensibly, these high-energy dogs are first offered to law enforcement agencies, then former handlers with the last consideration being private citizens. Understanding the jobs that these retired dogs held will help veterinarians understand and anticipate physical or behavioral conditions. Historically, the US had several major job categories for their dogs; some of these duties remain or have been adopted by other organizations or even sporting venues. “Sentry dogs” were tasked with warning and the challenges of mines and trip wires, dogs were starting to be used in the 1940’s to detect these threats. Several thousand dogs were utilized during the Vietnam War to identify these threats. It wasn’t until the late 1960’s that dogs were being used domestically to identify illicit substances and explosives. Currently, most military working dogs are trained to identify either explosives or drugs. Many of these dogs are also dually trained in criminal apprehension. In the 1970’s law enforcement began regularly using dogs for criminal apprehension, tracking and illicit substance detection. The demand for police dogs has had a recent resurgence, as departments recognize the value of the canine officer in detection and law enforcement. The cost of maintaining a police dog (and any working dog), however, is high. Much of that cost is tied up in the initial training of the handler and the dog. Keeping that dog healthy and working is invaluable to the finances and safety of the citizens that support that program.

Military trainers were instrumental in bringing back the skills of the military dogs to the general population in the US. In 1962, a group of trainers and dog club members in Washington State started a Search Dog Committee. Tracking had been the primary focus for human detection, but incorporating some of the lessons learned in the military, this group started to explore “air scenting” in the vast wilderness of the Pacific Northwest. Their success has led to the current proliferation of wilderness and disaster dogs in this country. Search and rescue dogs represent a unique population of working dogs. Typically, they are owned and trained by private citizens (typically volunteers), although some police and fire agencies also have search and rescue dogs. Unlike most police dogs, search and rescue dogs may only be deployed rarely, but when they are called upon, lives are in the balance. Search and rescue dogs
are typically trained to find live humans in either a wilderness (or wide area) or urban (disaster) environment. Another branch of
search is human remains detection, sometimes called “cadaver” dogs, these dogs are trained to find human remains and can be trained
for wilderness (or wide area), urban or water (drownings) search. Some dogs are trained to find both live humans and human remains,
although typically they are trained to give a different indication (alert) for each. The skills required and environmental risks are
related to their search specialty. Search and rescue dogs were launched into the public awareness during the response to 9/11. The total
number of responding dogs is unknown, but it is commonly estimated that approximately 300 search dogs were working at Ground
Zero (plus many more at the Pentagon). The longitudinal study of the health and behavior of these dogs was instrumental in launching
the Penn Vet Working Dog Center. The important news from this study is that the canine 9/11 responders did not develop more
respiratory complications or cancer than search and rescue dogs that did not respond. In general search and rescue dogs that
participated in the monitoring study lived long and healthy lives, with the last dog from 9/11 passing at almost 17 years of age.
Understanding, the impact of working careers and the hazards that these dogs do face will help us provide better medical care and
keep these dogs working!

The majority of graduates of the Penn Vet Working Dog Center work as police or search dogs. These dogs are either donated from
breeders or the result of our breeding program. All dogs begin basic foundational training at 8 weeks of age. Along the way, there are
numerous evaluations of skills for each dog to contribute to the data collection. From the time they enter the program, they are
provided with a variety of experiences that will prepare them for their careers. These can be divided into physical and behavioral
components of their training. The rationale for starting the puppies in a training program as such a young age is that behavioral issues
are the most common reason for a dog to fail out of a working dog program. The dogs at the Penn Vet Working Dog Center are
trained daily, allowing rapid interventions of developing problems (like fear). The dogs have an extensive socialization program,
rooted in the fact that they live with foster families on evenings and weekends when they are not training. At the training center the
dogs are exposed to a wide variety of people, environments, field trips and noises, all in an atmosphere of positive reinforcement. The
physical training focuses on developing a balanced and age appropriate fitness program for each dog. It is well recognized that
working detection dogs have a high incidence of low back and hip problems. From the time they are young we focus on teaching the
dogs about proprioception. The ability of the dogs to safely and securely place their feet on unstable surfaces will help prevent injuries
and allow them to move agilely across a wide variety of terrains. Both police and search dogs are required to climb ladders. Learning
to safely traverse ladders and other obstacles requires training.

A major component of the ability to move safely and protect their lower back involves good structure coupled with core strength.
For most police dogs or dogs from rescue organizations, critical and often predictive information about their lineage is not available.
Once the puppies are old enough, they are also screened for hip and elbow issues. The use of PennHip screening provides a more
quantitative measure and can be reliable at an earlier age than other screening tools for hip dysplasia. Low impact strengthening
exercises are taught as part of the training program. Core stabilization using balance discs is an easy and fun first step and also builds
body awareness. Developing balance and core strength prepares dogs for searching in a rubble pile of a collapsed building. With
developing puppies, the only exercises for cardiovascular training are low impact, like underwater treadmill or swimming. Dogs learn
to walk on the land treadmill but only as a training exercise rather than a cardiovascular exercise. Developing the habit of warming up
prior to exercise helps ensure that the dogs will be able to perform with lower risk of injury. Cool downs after work also become habit.

Although, fitness is a part of our training program, most handlers of working dogs have not even considered fitness in their
approach to training. Handlers spend their time training the dog for task specific behaviors (like searching rubble or biting a decoy).
Educatcing handlers to the value of a fitness and conditioning program should start before any injuries develop. The education starts
with basic care of their working dog and developing a trusting partnership in the success of the team. Handlers should be educated
about basic nutrition and body condition. It is well recognized that a dog with a body condition score of 7 versus a score of 5 has a
shorter lifespan. The length of life as well as quality of movement that are supported by a body condition score of 4 to 5 are valuable
motivators for handlers of working dogs. It is known that lameness is one of the most common injuries in military and working dogs.
Handlers can be educated to incorporate appropriate exercises to strengthen core and targeted muscles to help minimize injury.
Incorporation of a warm up into the routine of most canine handlers will take a major shift of focus, but has potential for important
benefits.

Another major factor in helping handlers keep their dogs working is to educate them on basic first aid and recognition of common
medical ailments. It is surprising how many handlers of German Shepherds are not familiar with the clinical signs of gastric dilatation
and volvulus. Many handlers wish to learn advanced medical techniques, but nothing will serve them better than knowing how to
differential normal physical parameters from abnormal. Teaching handlers how to obtain vital signs will make them better partners in
the active health care of their dogs. One of the major life threatening hazards that working dogs face is heat injury or heat stroke. Most
working dogs have a tenacity and desire to work that overrides normal physiologic signals, in addition they are often required to work
in adverse environments. Educating handlers on signs of early heat stress before it progresses to heat induced injury or life-threatening
heat stroke is invaluable. Traditionally, veterinarians have focused on rectal temperature in heat injury; however, in our field studies it
became clear that the physical signs of heat stress are not predictive of core body temperature. Handlers should be attentive to

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expanded and flattened tongues, shade seeking, excessive panting, squinty eyes and slower or less direct returns of the dog to the handler.

For detection dogs, olfactory function is a vital component of work. We are investigating factors that influence olfaction. In humans several drugs and diseases can interfere with normal olfaction. In dogs there is limited knowledge about factors that can impair olfactory function. Diseases of the nasal passages, like canine distemper and parainfluenza can alter olfaction. There is one study demonstrating that high doses of steroids for a week will also impair olfaction. High doses of metronidazole also may reduce a dogs’ sensitivity to odors. From a veterinary practitioner’s perspective, avoiding any unnecessary medication for working dogs is the safest approach.

In the almost 5 years since the Penn Vet Working Dog Center program began there are several key learning points. The most compelling is how these dogs serve humanity and whatever we can do to enhance their health, performance and welfare is a task worth undertaking. We have learned about the constant medical attention required to keep the dogs happy, healthy and working. We have learned about how they inspire us and the volunteers and students who work with us. The reward of becoming part of the working dog team is unmeasurable.
Antimicrobial Decision Making: Interpreting Culture and Susceptibility Data

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For complicated infections, or in patients at risk for therapeutic failure or developing resistance, identification of the infecting organism most appropriately should be based on appropriately collected cultures with (ideally) tube dilution susceptibility testing to allow assessment of “how” susceptible the isolate is, in general, to other drugs, but specifically to the drug of choice, such that a dosing regimen can be designed for the bug in the patient. Basing antimicrobial selection on C&S data does not guarantee success, just as failing to use C&S as a basis for selection (or selecting a drug characterized by “R” on the data) does not guarantee failure. The “90-60 rule” implies that approximately 90% of infections treated based on C&S are likely to respond if an “S” drug is selected; yet, up to 60% will respond even if an “R” drug is selected. The most likely situations where the latter is true is if the infection is at a site in which drug much higher than that achieved in the test tube (ie, much higher than the minimum inhibitory concentration (MIC)).

To treat or not to treat

The sample must be collected, handled and tested properly. Free, midstream catch samples are unacceptable and even catheterized samples are less than ideal. Just as absence of growth does not indicate absence of infection, isolation of an organism is not necessarily evidence of infection, nor even if infection is present, does the isolated organism represent the infecting organism. Clearly, (properly collected) culture of an organism from a tissue that is normally sterile indicates infection. However, discriminating between normal and infecting flora can be difficult. Pure, vibrant (meaning special media was not needed to coax the growth of the organism) of a large number of colonies are indicators of infection. The isolation of three or more different organisms (including more than one strain of the same organism) may indicate contamination and reculture should be considered. The extent of growth should be strongly considered when deciding to treat. Generally, infection is considered to be present if >10^7 CFU/ml at the site. For C&S purposes, quantitative cultures can be helpful: the urinary tract is not considered infected until >10^5 CFU/ml are present whereas only >10^3 is indicative of infection in the respiratory tract. Use of “urinary” paddles might be considered for samples that are being shipped in order to increase accuracy of identification and numbers. Laboratories may also indicate “heavy, moderate or light” growth; isolates with the greatest amount growth might be targeted. For multiple organisms, that with the greatest growth should be the primary focus of therapy. A call to the diagnostic lab might be prudent before marked financial commitment is put into treating an organism that is not causing infection. This is particularly important if the organisms’ presence is unusual (eg, Lactobacillus sp in urine).

To wait or not to wait?

Frequently, antimicrobial therapy is begun before cultures are collected. This is particularly true and appropriate in critical patients or in patients for which clinical signs are evident and are detracting from quality of life (patient or parent). However, should therapy begin and the choice prove to be wrong once C&S data is received, the original C&S data collected before the drug was begun may no longer be relevant to the patient. The use of the drug may change the pattern of resistance versus susceptibility, or may result in higher MIC (see mutant prevention concentration). A reculture may be indicated at that time, if possible. Certainly, dosing regimens with the appropriate drug should take into account the possibility that some level of resistance has developed toward the indicated drug.

To trust or not to trust

The C&S procedures themselves are fraught with potential errors. For practices that provide in-house susceptibility testing, care must be taken to follow guidelines established and published by (or comparable to) the Clinical and Laboratory Standards Institute (CLASI) or comparable standard-setting agency. Materials, including interpretive standards, should be validated by the appropriate agency. Minor changes in pH, temperature, humidity, etc can profoundly affect results. Personnel should be trained specifically in culture techniques and hospitals that provide this service (as do diagnostic labs) should maintain well designed and adequately collected quality control data to validate their procedures (CLSI indicates control organisms). Pitfalls of susceptibility testing also reflect the drugs selected for testing. Not all companies are interested in establishing interpretive criteria and as such, not all drugs are available for testing. Because automated systems can not accommodate and laboratories (nor clients) can not afford to test all potential drugs used to treat an infection, one drug often is tested as a model for other drugs in the class. For example, cephalothin models first generation cephalosporins, even though it is no longer used clinically. Note that it does not represent cefazolin well, the latter being more effective toward Gram negative (especially E coli) isolates. No single cephalosporin can represent 2nd or beyond generations. Enrofloxacin often represents the fluoroquinolones. In general, cross resistance can be expected among the FQs, although differences in potency do exist (for example, ciprofloxacin is more potent toward Pseudomonas or E coli, but less to Gram positives compared to enrofloxacin). Culture does not take into account active metabolites of some drugs (eg, enrofloxacin converted to ciprofloxacin). Note that if an organism is R to any FQ, FQ should be used only cautiously even if another is “S”. Amikacin is often more effective than
gentamicin toward many organisms, but less effective toward *Staphylococcus* sp. (hence both are often on a report). Note that CLSI interpretive criteria are generated for specific species, and often for specific organisms and specific infections. Human laboratories will use human interpretive criteria, which often are not relevant to animals (eg., ciprofloxacin). Ciprofloxacin (CIP) oral bioavailability in dogs is 30 to 40% of that in humans, and despite its increased potency compared to enrofloxacin (ENR) toward Gram negative organisms, its potential efficacy (MIC<sub>90</sub>) is equivalent to or less for many organisms. Susceptibility data also does not take into account active metabolites, again exemplified by ENR, which is metabolized to CIP: both C<sub>max</sub> and area under the curve (AUC) of bioactivity of ENR may increase up to 50% or more by CIP; as such, C&S data may underestimate efficacy. MIC<sub>90</sub> generally are based on the highest labeled dose, but higher doses might be safely administered for many antibiotics. If recommended doses change, the manufacturer should provide CLASI with updated pharmacokinetic information so that interpretive criteria may change. One of the disadvantages of current susceptibility testing is that the concentrations tested are close to the MIC<sub>90</sub> and thus, does not allow identification of isolates that are very susceptible (that is, MIC are far away from the MIC<sub>90</sub>). As such, drugs may be chosen based on isolates that have already undergone first step mutations (see below).

A final concern relates to the 3<sup>rd</sup> and 4<sup>th</sup> generation (extended spectrum) cephalosporins: they are susceptible to extended spectrum beta-lactamase (ESBL) that tend to be induced *in vivo* but often missed *in vitro*. If CLSI guidelines are followed, resistance to cefpodoxime indicates ESBL being produced, if CLSI guidelines are not followed, therapeutic failure may occur. Carbepenems and clavulanic acid (eg, amoxicillin-clavulanic acid) generally are not susceptible to these enzymes.

**Bridging pharmacodynamic (PD) and pharmacokinetic (PK) data**

So once you have the data, what do you do with it? Simplistically, susceptibility data represents “what is needed” in the patient to facilitate antimicrobial efficacy. Care must be taken with this simplistic approach: susceptibility data is generated from in vitro methodologies, yet it is applied to in vivo (and abnormal) conditions. As long as this caveat is foremost in the clinicians mind, the data can be useful to antimicrobial selection. Note that population susceptibility data can be helpful with empirical selection of antimicrobials (see below).

**Pharmacodynamic (microbiological) data:** what you need

PD data includes data generated both from agar gel disc diffusion (eg, Kirby Bauer: zone diameters) as well MIC tube dilution (MIC) methods of susceptibility testing. What is tube dilution data and why is it so useful? In contrast to disk diffusion, tube dilution methods involve inoculation of a series of test tubes with a standard number of organisms. The test tubes contain increasing concentrations of the drug of interest in two-fold dilutions whose range varies with the drug, reflecting concentrations achieved in the patient for that drug at the recommended dose. Following a standard time, the tubes are evaluated for detectable growth. The test tube that contains the lowest concentration of drug and no visible growth contains the minimum amount of drug necessary to inhibit (not kill) the growth of the organism cultured from the patient (the MIC). Ideally, this concentration must be achieved at the site of infection. Adaptation to computerized/automated systems allow much more accurate testing in short time periods.

For either method of susceptibility testing, simplistically, the likelihood of a drug being effective in the patient is based on whether or not the recommended dose on the label is likely to generate plasma drug concentrations (PDC) that equal or surpass the MIC of the
infecting organism. Diagnostic laboratories indicate the likelihood of susceptibility by the “SIR” letter designation. Understanding the basis of that designation will facilitate antimicrobial selection. The SIR designation reflects whether or not the MIC of the infecting organism is less than (“S”), close or equal to (“I”) or greater than (“R”) the breakpoint MIC (MIC_{BP}) of the drug. CLSI determines the breakpoint, based in part, on peak plasma drug concentrations (C_{max}) of the drug (population data). Because dose and C_{max} varies for each drug (eg, at 20 mg/kg, C_{max} of enrofloxacin is 4 mcg/ml; at 22 mg/kg, C_{max} of amikacin is 65 mcg/ml), the concentrations of drugs tested by the laboratory vary, and the breakpoint will also vary. Thus, one should not compare an MIC for enrofloxacin (eg, 0.25 mcg/ml) to an MIC for amikacin (eg, 4 mcg/ml) and assume the former is better. Rather, “how far” that MIC is from the C_{max} determines how susceptible the isolate is to each drug. Note also that the range of each drug tested is very narrow, leading to “<” on reports. For example, for the culture report below and amikacin, C \leq 4 means no growth occurred in the test tube containing 8 mcg/ml, which was the lowest concentration tested by the lab, so the MIC must be lower than 8 or \leq 4 mcg/ml, (the next lowest concentration). The isolate is susceptible. An MIC of > X is accompanied by an “R” because the organism was not susceptible to the highest concentration tested.

CLASI updates MIC_{BP} (generally yearly) particularly as new data is provided regarding organism susceptibility. Increasing resistance to organisms may lead to changes in the MIC_{BP} such as has recently occurred for amoxicillin and doxycycline. For older antibiotics approved decades ago, originally labeled doses may be inappropriate for all except very sensitive organisms. Again, a good laboratory will follow CLSI guidelines.

Population data
Population data can be used for empirical antimicrobial selection. For example, using an antibiogram which indicates the proportion of isolates resistant vs susceptible to a drug. Similarly, theTarget® Antimicrobial Handbook indicates not only the most likely organisms cultured (but not necessarily pathogenic) from selected sites, but also provides a “scoring” system of susceptibility. For antibiograms (see Auburn University Canine Cumulative Antimicrobial Susceptibility Report) drugs to which >75% or more of isolates are susceptible might be wiser selections. A patient that has not been previously exposed to antimicrobials is more likely to be represented by the “susceptible” isolates whereas an “at risk” patient (eg, previously exposed to antimicrobials, immunosuppressed) may be better represented by the resistant proportion. Likewise, package inserts for newer antimicrobials include susceptibility data (MIC) and as such, can guide not only the selection of a drug, but the design of a dosing regimen. The MIC data on a label may include: 1. the range of MIC for susceptible organisms; 2. the mode of MIC (the most frequently cited MIC); 3. or the MIC_{90} and the MIC_{50}. The data are population statistics; the latter two reflect, respectively, the MIC below which 50% and 90% of the isolates (by genus and species) are inhibited (not killed). However, the MIC_{90} and MIC_{50} should be based on a large number of microorganisms to assure accurate sample representation of the population (ideally >300). Organisms with MIC_{90} that are low are more susceptible than organisms with higher MIC_{90}. Organisms whose MIC_{90} is approaching the C_{max} of the drug (also on a package insert) prudently should not be treated with that drug. See also concentration and time dependency. An example of population MIC data is demonstrated from a fluorinated quinolone package insert. Those organisms most susceptible to the drug have the lowest MIC whereas organisms with higher MIC are less likely to respond.

Pharmacokinetic (PK) data: What you get
The selection of an antimicrobial should be based on the likelihood that therapeutic (effective) concentrations will be achieved at the tissue site. What is needed for therapeutic efficacy for infections is determined largely by the susceptibility (pharmacodynamic data) of the organism. If you have a C&S from your patient with MIC, the MIC for the drug of interest is how much you need. For populations of microbes, the MIC_{90} provides an indication of what is needed. Efficacy of an antibiotic is most likely to occur when the pharmacodynamic data is coupled with what is achieved in the patient. For the clinician seeking to improve antimicrobial efficacy, the further the MIC of the infecting organism is from the C_{max} (or MIC_{BP}) of the drug, the more likely effective concentrations will be reached at the site of infection. If a number of drugs are designated as “S”, the selection of which “S” is best might be narrowed by focusing on those drugs for which the MIC is furthest from the MIC_{BP} or C_{max}. The most susceptible, lowest tier drug should be selected.
Selecting a drug: patient data

Compare the MIC (what is needed) to the peak drug concentration achieved at the recommended (or modified) dose: the higher the Cmax is compared to the MIC, the greater the chance of therapeutic success and the less the chance of resistance. Once the “best” drugs are identified based on C&S, then the list can be narrowed down based on other factors. The same approach can be used for population data.

Package insert data: Using Proteus as an example, comparison of Cmax to MIC90 reveals a ratio 2:0.125 or 16 for marbofloxacin compared to 1.8:1.8 or 1 for difloxacin, using the low dose for each drug. For E. coli, the numbers are 2:0.06 or 33 for marbofloxacin compared to 16 for difloxacin. For either organism, marbofloxacin offers the best ratio. For E. coli, the low does might be acceptable for marbofloxacin, and potential for difloxacin, although the latter might not be prudent. For Proteus, again, the low dose of marbofloxacin might not be prudent, and difloxacin should not be used to treat

<table>
<thead>
<tr>
<th>Drug</th>
<th>MIC BP</th>
<th>MIC BP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin (C)</td>
<td>&lt; 16</td>
<td>&gt; 64</td>
</tr>
<tr>
<td>Amoxicillin (T)</td>
<td>&lt; 25^1</td>
<td>&gt; 0.5</td>
</tr>
<tr>
<td>Amoxicillin (T)</td>
<td>&lt; 16^2</td>
<td>&gt; 32</td>
</tr>
<tr>
<td>Amoxicillin with clavulanic acid</td>
<td>4/2</td>
<td>8/2</td>
</tr>
<tr>
<td>*Ampicillin^4</td>
<td>&lt; 2.5</td>
<td>&gt; 32/16</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>≤ 4</td>
<td>≥ 8</td>
</tr>
<tr>
<td>Carbenicillin</td>
<td>≤ 16</td>
<td>≥ 64</td>
</tr>
<tr>
<td>Cefazolin</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cefotaxine</td>
<td>≤ 8</td>
<td>≥ 64</td>
</tr>
<tr>
<td>Cefoxitin</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cefpodoxime</td>
<td>≤ 2</td>
<td>≥ 8</td>
</tr>
<tr>
<td>Cefazidine</td>
<td>≤ 2</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cefotin</td>
<td>≤ 2</td>
<td>≥ 8</td>
</tr>
<tr>
<td>Cefuroxime</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>≤ 64</td>
<td></td>
</tr>
<tr>
<td>Cefuroxime</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Cefalexin</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>*Chelothion</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>≤ 1</td>
<td>≥ 8</td>
</tr>
<tr>
<td>*Clindamycin</td>
<td>≤ 0.5</td>
<td>≥ 4</td>
</tr>
<tr>
<td>*Difloxacin</td>
<td>≤ 0.5</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>≤ 4</td>
<td>≥ 16</td>
</tr>
<tr>
<td>*Enrofloxacin</td>
<td>≤ 0.5</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>&lt; 0.25</td>
<td>≥ 1</td>
</tr>
<tr>
<td>Florfenicol</td>
<td>≤ 2</td>
<td>≥ 8</td>
</tr>
<tr>
<td>Gentamicin*</td>
<td>≤ 4</td>
<td>≥ 16</td>
</tr>
<tr>
<td>*Imipenem/cilastin</td>
<td>≤ 4</td>
<td>≥ 16</td>
</tr>
<tr>
<td>Kanamycin*</td>
<td>≤ 16</td>
<td>≥ 64</td>
</tr>
<tr>
<td>Lincomycin</td>
<td>≤ 0.5</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Marbofloxacin</td>
<td>≤ 1</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Meropenem</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Meltrondazole</td>
<td>≤ 8</td>
<td>≥ 32</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>≤ 32</td>
<td>≥ 128</td>
</tr>
<tr>
<td>*Orbafloxacin</td>
<td>≤ 1</td>
<td>≥ 8</td>
</tr>
<tr>
<td>*Oxazolin</td>
<td>≤ 2</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Penicillin G</td>
<td>≤ 8^1</td>
<td>≥ 16</td>
</tr>
<tr>
<td>*Piperacillin</td>
<td>≤ 16^2</td>
<td>≥ 128</td>
</tr>
<tr>
<td>*Rifampin</td>
<td>≤ 4</td>
<td>≥ 4</td>
</tr>
<tr>
<td>Sulfadiazine</td>
<td>≤ 2</td>
<td>≥ 4</td>
</tr>
<tr>
<td>*Tetracycline</td>
<td>≤ 16</td>
<td>≥ 128</td>
</tr>
<tr>
<td>*Ticarclin</td>
<td>≤ 16^4</td>
<td>≥ 128</td>
</tr>
<tr>
<td>*Ticarclin with clavulanic acid</td>
<td>64^2</td>
<td>128/2</td>
</tr>
<tr>
<td>*Trimethoprim</td>
<td>≤ 2/38</td>
<td>≥ 128/2</td>
</tr>
<tr>
<td>Sulfamethoxazole</td>
<td>≤ 0.5/0.5</td>
<td>4/76</td>
</tr>
<tr>
<td>*Vancomycin</td>
<td>≤ 4</td>
<td>≥ 32</td>
</tr>
</tbody>
</table>

**Table 2. Selected Pharmacokinetic Data for Antimicrobials**

<table>
<thead>
<tr>
<th>Drug</th>
<th>HL</th>
<th>Dose</th>
<th>Route</th>
<th>Cmax</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin (C)</td>
<td>1</td>
<td>20</td>
<td>IV</td>
<td>65</td>
</tr>
<tr>
<td>Amoxicillin (T)</td>
<td>1</td>
<td>12.5</td>
<td>PO</td>
<td>4.5-6</td>
</tr>
<tr>
<td>Amoxicillin (T)</td>
<td>1-1.5</td>
<td>12.5</td>
<td>PO</td>
<td>4.5-6</td>
</tr>
<tr>
<td>Ampicillin (T)+A29</td>
<td>0.5-1.5</td>
<td>30</td>
<td>PO</td>
<td>10</td>
</tr>
<tr>
<td>Cefotaxine</td>
<td>0.8</td>
<td>50</td>
<td>IV,IM</td>
<td>41</td>
</tr>
<tr>
<td>Cefpodoxime</td>
<td>5.6</td>
<td>5</td>
<td>PO</td>
<td>8.2</td>
</tr>
<tr>
<td>Cefazidine (T)³rd</td>
<td>0.82</td>
<td>30</td>
<td>SC</td>
<td>42</td>
</tr>
<tr>
<td>Cephalaxin (T)¹st</td>
<td>1.3-2.5</td>
<td>20</td>
<td>PO</td>
<td>20</td>
</tr>
<tr>
<td>Chloramphenicol (T)</td>
<td>1.2-2.7</td>
<td>20</td>
<td>PO</td>
<td>23</td>
</tr>
<tr>
<td>Ciprofloxacin (C)</td>
<td>5.3</td>
<td>20</td>
<td>PO</td>
<td>2.8</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>4.6</td>
<td>2.5</td>
<td>PO</td>
<td>3</td>
</tr>
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<td>Enrofloxacin</td>
<td>4.1</td>
<td>20</td>
<td>PO</td>
<td>4.2</td>
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<tr>
<td>Gentamicin (C)</td>
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<td>10</td>
<td>IV</td>
<td>28</td>
</tr>
<tr>
<td>Imipenem/cilastin (T)</td>
<td>1</td>
<td>5</td>
<td>IM</td>
<td>13</td>
</tr>
<tr>
<td>Marbofloxacin (C)</td>
<td>11</td>
<td>2</td>
<td>PO</td>
<td>1.4</td>
</tr>
<tr>
<td>Orbifloxacin (C)</td>
<td>5</td>
<td>2.5</td>
<td>PO</td>
<td>2</td>
</tr>
<tr>
<td>Sulfadimethoxine (T)</td>
<td>13</td>
<td>55</td>
<td>PO</td>
<td>67</td>
</tr>
<tr>
<td>Tetracycline</td>
<td>10</td>
<td>13.75</td>
<td>IV</td>
<td>7</td>
</tr>
<tr>
<td>Ticarclin</td>
<td>1</td>
<td>40</td>
<td>IV</td>
<td>80</td>
</tr>
<tr>
<td>Ticar-clav (T)</td>
<td>1</td>
<td>6</td>
<td>IV</td>
<td>1</td>
</tr>
</tbody>
</table>

C=concentration dependent
T=time dependent
“It is unwise to underestimate an adversary that has had a three billion year evolutionary head start” (Sayers, 2004). The advent of antimicrobial resistance is increasingly limiting therapeutic options in human and resistance to an antimicrobial varies with the species and strain. Among the most adaptable organisms is *E. coli*. Discovered in 1885 by the pediatrician Theodore Escherich, it was originally discovered in neonatal fecal samples. Dr. Erich recognized *E. coli* was acquired at birth and remained with us till death, with strains coming and going. It is the most thoroughly understood microbe, and is critically important as a research tool. Through *E. coli*, we have come to understand such diverse activities as intermediary metabolism, DNA replication and RNA transcription, protein synthesis and genetic recombination. Indeed, recombinant products would not be possible without *E. coli*. *Escherichia coli*, a member of the family Enterobacteriaceae, is a lactose fermenter, causing a distinct color on diagnostic agar. It is the predominant facultative anaerobe (in the normal intestine of both humans and many warm-blooded animals), playing a major role as normal microflora. However, it also is ubiquitous in the environment, as is recognized by its appearance as contaminants in food stuffs. It has or acquires genes that encode for flagella, making it mobile. Its presence in the environment is used as a sentinel of environmental contamination. Referred to as the “cockroach” of microbes because of its adaptability, *E. coli* rapidly divides, potentially doubling its population every 20 minutes. Further, it is highly mutagenic, with spontaneous mutations occurring in of 1 per 100 thousand to 1 per billion new progeny (assume 1 gm of feces contains 100 million *E. coli*) thus assuring opportunity for spontaneous mutation even in the absence of stimuli, such as drugs.

**Resistance**

The gastrointestinal environment is conducive to development of resistance. Environmental microbes maintain an ecological niche by suppressing competition through secretion of antibiotics. As such, commensal organisms are constantly being exposed to antibiotics. However, the microbe producing the antibiotic, as well as surrounding normal flora, are resistant to the antibiotic. Thus, genes for resistance develop along with genes directing antibiotic production and organisms are “primed” to develop resistance. Microflora of the GI tract can serve as reservoir of resistance genes. Exposure to antimicrobials may facilitate survival of isolates that have either spontaneously mutated or acquired resistant through other means. Resistance may be easily conferred to other potentially more virulent organisms. *E. coli* rapidly develops resistance, particularly that associated with multiple drug resistance (MDR) when exposed to selected antimicrobials. More disconcerting, resistance is easily conferred to more pathogenic organisms. In human medicine, *E. coli* has developed resistance to the fluorinated quinolones, beta-lactams, or both: it is among the Gram negative organisms that secrete extended spectrum beta-lactamases (ESBL). Emergence of extended spectrum extended spectrum beta-lactamases (ESBL) is an example of the relentless adaptive nature of microbes toward designer drugs intended to preclude the advent of resistance. The ESBLs are encoded by large plasmids that can confer the information between strains as well as different species of organisms. The gene mutation confers resistance to newer cephalosporins including cefotaxime, ceftazidime and...
Table 1. The percent of E. coli feline and canine uropathogens from throughout the US (2009-2012) resistant to antimicrobial drugs.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Overall (n=1512)</th>
<th>95% CI</th>
<th>Species</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Canine</td>
<td>Feline</td>
</tr>
<tr>
<td>AMX</td>
<td>41.5</td>
<td>39.1-44.0</td>
<td>45.2</td>
<td>30.0</td>
</tr>
<tr>
<td>AMP</td>
<td>49.1</td>
<td>46.6-51.6</td>
<td>52.8</td>
<td>37.6</td>
</tr>
<tr>
<td>CPF</td>
<td>9.3</td>
<td>7.7-11.0</td>
<td>10.5</td>
<td>5.7</td>
</tr>
<tr>
<td>CFT</td>
<td>8.5</td>
<td>7.1-9.9</td>
<td>9.6</td>
<td>4.9</td>
</tr>
<tr>
<td>CFO</td>
<td>12.8</td>
<td>11.1-14.5</td>
<td>14.2</td>
<td>8.5</td>
</tr>
<tr>
<td>CFP</td>
<td>13.0</td>
<td>11.3-14.7</td>
<td>13.7</td>
<td>10.6</td>
</tr>
<tr>
<td>CFZ</td>
<td>8.9</td>
<td>7.5-10.5</td>
<td>10.4</td>
<td>4.4</td>
</tr>
<tr>
<td>CPL</td>
<td>98.9</td>
<td>98.2-99.3</td>
<td>99.0</td>
<td>98.6</td>
</tr>
<tr>
<td>CHP</td>
<td>12.6</td>
<td>11.0-14.3</td>
<td>13.7</td>
<td>9.3</td>
</tr>
<tr>
<td>DXY</td>
<td>100</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ENR</td>
<td>10.5</td>
<td>8.8-12.3</td>
<td>11.69</td>
<td>6.7</td>
</tr>
<tr>
<td>GEN</td>
<td>7.9</td>
<td>6.6-9.3</td>
<td>8.56</td>
<td>6.0</td>
</tr>
<tr>
<td>MRP</td>
<td>1.3</td>
<td>0.7-2.0</td>
<td>1.05</td>
<td>1.9</td>
</tr>
<tr>
<td>TCL</td>
<td>19.4</td>
<td>17.5-21.4</td>
<td>20.79</td>
<td>15.3</td>
</tr>
<tr>
<td>TMS</td>
<td>8.9</td>
<td>7.4-10.3</td>
<td>9.69</td>
<td>6.3</td>
</tr>
</tbody>
</table>

Idexx laboratories. Currently, regional differences in resistance continue to persist. Overall resistance is greatest to cephalaxin (9%) as is demonstrated in Table 1; 100% of isolates are also resistant to doxycycline using the new interpretive criteria established by CLSI. Further, 40% are resistant to amoxiclavulanic acid and 50% to ampicillin (amoxicillin). This latter statistic suggests that for treatment of E. coli, Clavamox® may not have that much advantage over amoxicillin along and this would also be true if treating Enterococcus. However, if treating other organisms, protection against beta-lactamases may be helpful.
Gram positive organisms
Methicillin resistance (MRSA; S. aureus; MRSP; S. intermedius [pseudintermedius] Multidrug resistance is now considered the normal response to antibiotics for Gram positive cocci pneumococci, enterococci and staphylococci. Methicillin resistance (MRSA; S. aureus; MRSP; S. intermedius [pseudintermedius]) is indicated by the presence of the mecA gene, which encodes a mutation in penicillin binding protein (PBP) resulting in formation of PBP2a rather than PBP2. As such, affinity is reduced for the beta-lactam ring, rendering the organism resistant to all beta-lactams. Protectors such as clavulanic acid are ineffective. Detection of MRSA or MRSP on C&S generally is based on resistance to oxacillin, which is more stable than methicillin in disks used for testing. However, increasingly, laboratories are indicating MRS based on absence of susceptibility to any beta-lactam. In our hospital, approximately 25 to 30% of Staphylococcus pseudintermedius express methicillin resistance. Antibiotics and especially cephalosporins are associated with induction, selection, and propagation of MRSA. MRSA in human patients has evolved from a hospital-acquired (HA-MRSA; nosocomial), in which occurs most commonly in patients immunocompromised by disease, drugs, procedures and duration of hospitalization, to a community acquired infection (CA-MRSA), in which otherwise healthy persons are infected, usually in the skin or soft tissue. Crowded conditions, shared items and poor hygiene increase the risk of community acquired infection. Although it is community acquired MRSA strain USA300 that appears to be most commonly associated with increased colonization in dogs and cats, it is USA100, most commonly associated with hospital acquired-MRSA infections in humans, that most commonly is associated with infections in dogs and cats animals. The impact of MRSA in veterinary medicine is increasingly problematic, not only because of its impact on the patient, but the public health considerations. The mec gene has been detected in methicillin-resistant Staphylococcus aureus organisms infecting dogs and MRSA has been associated with infection in dogs. However, MRSA also has been found in up to 4% of healthy dogs, with identification complicated by the need for multiple sampling sites (nasal and rectal or perineal). Infections have been isolated in family members and pets in the same household, but this is likely to reflect transmission from humans to the pet. It is likely that colonization is transient in animals. However, healthy pets have been demonstrated to be potential reservoirs for transmission of MRSA to healthy handlers and a potential health risk to immune-compromised patients (human and presumably other animals in the household). Human colonization with MRSP is unusual. However, MRSP has been reported as a cause of infection in human patients and transmission from pets with pyoderma has been confirmed. It is the very immunocompromised patient that is at risk for MRSA infection acquired from an animal. In such cases, the carrier or infected animal should be removed from the environment until successfully treated for methicillin-resistant Staphylococcus. Glycopeptides such as vancomycin are the initial drugs used to treat MRSA in humans, although increasingly vancomycin resistant staph infections (VRSA) have emerged.

Reducing resistance: The three “DE”s
Regardless of the organism, the most significant mechanism by which bacterial resistance is likely to be reduced is implementation of behaviors that are designed to reduce patient risk such as length of hospital stay, and design, implementation of and adherence to infection control practices. Consider the three DEs: (1) DESCALATE. Because previous antimicrobial therapy is one of the most important factors associated with resistance, approaches which minimize indiscriminant antimicrobial use will be important. Examples of human strategies include improving appropriate antimicrobial use (eg, including less ideal strategies such as strict adherence to prescribed formularies, setting limits on the duration of antimicrobial therapy); potentially reasonable strategies such as requiring prior approval for use of certain antibiotics such that proper use can be verified; and more rationale strategies such as narrowing the spectrum of empiric antibiotics, and rotating the use of antimicrobial drugs on a regular schedule); primary prevention by decreasing length of hospital stay, decreased use of invasive devices, and newer approaches such as selective digestive decontamination and vaccine development. Probably the single most important first step in judicious antimicrobial use and avoiding resistance is questioning/confirming the need for therapy. This is no small task, being fraught with the lack of effective diagnostic aids. Probably the most common-- and least correct mindset is failure to recognize that we are in conflict with our directive of “above all else do no harm” if we use antimicrobials in the absence of infection. De-escalation includes taking actions that stay the hand in reaching for drugs if they are not really necessary. For urinary tract infections, increasingly the need for treating asymptomatic bacteria is questioned. What constitutes an infection may not depend only on the inoculum size (e.g, 1000 CFU/ml) but the presence of clinical signs. The second De is (2) DESIGN. Dosing regimens should be designed to assure that adequate drug concentrations are reached at the site of infection to kill, not simply inhibit, microbial growth. DEAD BUGS DON’T MUTATE! Once the decision to use the antimicrobial is made, efforts should focus selecting a drug to which the bug is most susceptible. A practice-based antibiogram (see above) may be helpful. If an animal has not been exposed to antimicrobials, the chances are improved that any infecting pathogens are among the susceptible isolates. A narrow spectrum is preferred to a broad spectrum drug. Once the drug is chosen, design focuses on the dosing regimen to assure that concentrations adequate to kill the infecting microbe are achieved at the site of infection. This involves not only selecting the drug to which the isolate is most susceptible (and the drug most likely to reach the target site), but also designing the dosing regimen based on the MIC, potentially the MPC and time or concentration dependency of the drug (see parts II and III if relevant). An antibiogram (see figure; top number is % susceptible, bottom number of each cell is number
tested) generated for each practice can support empirical selection of antimicrobial drugs although increasingly, culture is indicated in all but the antimicrobial naïve patient (this includes direct - or indirect through a household member - exposure). (Note that squares without information are drugs which should not be tested toward that bug).

**Detergent**

Hospital strategies include: improving infection control (eg, selective decontamination procedures, prevention of horizontal transmission via handwashing, use of disinfectants, glove and gown use, alternatives to soap, and improving the workload and facilities for health care workers), and identification of specific areas for treatment of potentially infectious agents (ie, bandaging areas that can be easily cleaned). Increasingly “detergent” should be applied to the patient and its home. For example, recurrent infections might be reduced if successful initial therapy is coupled with cleansing of the environment in which the pet is located such that it is not continued to be exposed to the infecting bug. For UTI infections, this may become particularly important in that urine contaminates the environment.
Compounded Medications:  
Getting More Complicated
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Individualized drug therapy increasingly is being recognized as an important aspect of health care for both human and veterinary medicine. Compounding has been defined by the National Association of Boards of Pharmacy (Model State Pharmacy Act) as the preparation, mixing, assembling, packaging, or labeling of a drug or device, as the result of a practitioner’s prescription drug order (or initiative) and based on the practitioner/patient/pharmacist relationship (http://www.iacprx.org/index.html, accessed July 2004). Among the controversies surrounding compounded products is the use of bulk substances. A bulk drug substance is legally defined [21 CFR 207.3(a)(4)] for both human and animals as “any substance that is represented for use in a drug and that, when used in the manufacturing, processing or packaging of a drug, becomes an active ingredient or a finished dosage form of the drug.” In laymen’s terms, any drug or drug preparation ingredient not prepared in an approved finished dosage form is considered to be a bulk substance. Compounding pharmacists stress that the quality of some products is markedly approved when using a bulk substance. The FDA perceives that compounding from bulk substances increases the risk of adversity. A major reason is the lack of quality assurance of compounded products. Among the reasons potentially contributing to poor quality is the bulk substance, the majority of which come from Asia and for which, for some products, contamination has been identified. The availability of bulk substances also facilitates the manufacturing (rather than compounding) of compounded products that several nationally recognized pharmacies implement. A search of internet pharmacies demonstrates that many products continue to be compounded that mimic commercially available approved products.

Compounding is as old as drug use. A major advent in the profession of pharmacy and the science of compounding was the development of drug standards. In the 19th century, the United States Pharmacopeia (USP) began its role in the provision of drug standards, thus assuring strength and purity of drug materials. It maintains this often unrecognized, yet critically important role today; its pharmacopoeia (United States Pharmacopoeia/National Formulary; USP/NF) are the legal standards recognized by the Food and Drug Administration. Compounded products predominated into the twentieth century; as late as the 1930s and 40s, 50 to 60% of human drugs were compounded by pharmacists. However, in the late 19th century, the need for new, therapeutically useful compounds led to the advent of pharmaceutical research, and shortly thereafter, pharmaceutical manufacturing. By the 1950’s, advances in manufacturing technology led to the mass production of drugs, causing pharmacists to largely become dispensers, rather than compounders, of drugs. The 1980’s and 90’s were accompanied by a resurgence in compounding in human medicine for a variety of reasons. The history of veterinary compounding has paralleled human compounding. The cost of approval of an animal drug surpasses $15-20 million. The economic return of animal drug approval is not surprisingly is low (generally well below $100 million); subsequently, the financial incentive to pursue animal drug approval compared to human pharmaceuticals is much less. Further, because of cost differences, veterinarians often will prescribe a human or human generic drugs. Despite the fact that it should not be, compounded preparations are often prescribed because they can be cheaper. The issues with veterinary compounding include are not necessarily encountered in human compounding. Unfortunately, animal care givers, veterinarians and pharmacists often are unaware of these differences.

Definition and regulations for compounding
While the FDA does not regulate the act of compounding, it does regulate the product. The advent of pharmaceutical manufacturing in the early 1900s increased human exposure to drugs, and thus the risk of adverse drug events. In the late 1930’s, over 100 persons died after being treated with sulfanilamide prepared in a toxic vehicle. The resultant public outcry was instrumental in the passage of the 1938 Food, Drugs and Cosmetic Act (FDCA) which addressed drug safety. In 1962, the FDCA was amended to include the assurance of drug efficacy in the mandated activities of the FDA. As in 1938, compounding nor animal drugs were specifically addressed; further, animal drugs were not addressed. It was not until 1968, with amendment of the FDCA by the Animal Drug Amendment, that animal drugs were distinguished from human drugs. This amendment provided for the formation of the Bureau (later renamed to Center) of Veterinary Medicine (CVM) within the FDA. The mission of the CVM, as mandated by Congress, was (is) assurance of both animal and public health resulting from drug use in animals. Regulatory actions of the FDA are delineated in Congressionally approved acts or their amendments. The regulations (“rules”) established for implementation of the FDCA and its subsequent amendments are published in codified form in the Code of Federal Regulations (CFR), which is available for public review. To facilitate understanding of the regulations by FDA staff, and to a lesser degree, industry and the public, the FDA may publish Compliance Policy Guides (CPG) for each set of regulations. The CPG direct FDA regulatory actions but are not legally binding, and are open to interpretation by the FDA.
Compounding of human drugs

Compounding of human drugs was not specifically addressed in either the original FDCA or its 1962 amendment. However, the FDA is empowered to regulate any drug (or any product intended to be used as a drug) and interprets a compounded drug to be an unapproved, new drug. As compounding increased toward the end of the 20th century, FDA regulation of the human drug compounding was specifically addressed in 1997 with passage of the Food and Drug Administration Modernization Act (FDAMA). This Act, which does not apply to veterinary medicine (compounding of animal drugs addressed below), included Section (503A) entitled “Pharmacy Compounding. However, in order to protect consumers, the act also attempted to provide the FDA with criteria by which inappropriate compounding could be identified and subsequently regulated. The intent of FDAMA was “to ensure continued availability of compounded drug products as a component of individualized therapy, while limiting the scope of compounding so as to prevent manufacturing under the guise of compounding.” These included the amount of drug product compounded in anticipation of need, whether or not the compounding of the drug was individual-patient driven, and, because it was perceived by the FDA as an indication of manufacturing of inappropriate amounts of a compounded product, it prohibited their advertisement. However, this aspect of the law was subsequently challenged by the pharmacy profession, based on infringement of the second amendment (right of free speech). Ultimately, the US Supreme court agreed and because the advertisement portions of the laws could not be easily separated from the remainder of the law, the entire Pharmacy Compounding section of FDAMA was invalidated. The CPG for FADAMA stated that compounding of human drugs from bulk substances will be tolerated as long as an approved finished version of the drug exists. Ironically, with the invalidation of the Pharmacy Compounding section of FDAMA, while gaining the right to advertise their expertise, pharmacists have subsequently failed to gain protection of their right to compound human drugs (Gibbs 2002), including compounding from bulk substances. Despite the lack of legal protection, the CPG for FDAMA as currently written do indicate tolerance of the FDA toward compounding from bulk substances if an approved version (human) of the bulk substance exists.

Compounding of veterinary drugs

Pharmacists may not be aware of regulatory differences between animal and human drugs (and do not realize that CPG are not legally binding). As such, they often assume that compounding of animal drugs from bulk substances is legal as long as an approved animal version of the drug of interest exits. In contrast to compounding of human drugs, federal regulation of veterinary compounded veterinary drugs is specifically and legally allowed by the Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994 (21 C.F.R Section 530; http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcr/cfrsearch.cfm?fr=530.41). As the animal counterpart to FDAMA, it amends the FDCA. The major benefit of this act to the veterinary profession was legalization of the already common practice of extra-label drug use (ELDU) in animals [(Sec 512 (a) (4)], as long as the conditions stipulated in the regulations are met. AMDUCA stipulates that compounding must be performed by either a licensed veterinarian or pharmacist (thus assuring the rights of both professions to compound) in the context of a valid veterinary client-patient relationship and that no approved dosing form or concentration of the drug (human or animal) commercially exists for the treatment of the diagnosed condition.

The interpretation and implementation of compounding regulations of AMDUCA are delineated in CPG 608.400: Compounding of Drugs for Use in Animals. The 2003 CPG describes those activities not considered to be compounding. These include mixing, reconstituting or other acts [on the drug] that are performed in accordance with the approved labeling provided by the manufacturer. Thus, any modification in the finished dosing form of the approved drug that is not specifically delineated on the drug label (which includes its accompanying package inserts) is considered by the FDA as compounding. The FDA considers any compounded product (human or animal) to be a new, finished (that is, ready for administration) drug, and, because it undergoes no federally-mandated approval process, an unapproved drug. The FDA assumes that both public and animal health potentially are put at risk if compounded drugs are administered to veterinary patients since the drugs are not accompanied by “adequate and well controlled safety and effectiveness data,” particularly if not compounded in “adherence with pharmaceutical chemistry and current good manufacturing practices” (CPG Section 608.400). The FDA anticipates that compounded products may cause adverse reactions or contain potentially harmful excipients, and that the unscientific assignment of withdrawal times to compounded food animal products may lead to potentially harmful tissue residues. Accordingly, the laws (eg, AMDUCA), regulations and CPG that address compounding of animal drugs focus (although not exclusively) on protection of human (public health) safety.

Several sources of active ingredients are used for compounded animal drugs. Legal sources are limited to FDA-approved finished forms of either animal or human drugs; the FDA makes no distinction as to which (animal versus human) is the preferred source. Because no other source is legalized, all other sources are considered by the FDA to be illegal, including non-FDA approved finished drug products obtained outside of the United States and bulk substances. Whereas AMDUCA regulations specifically state that ELDU of drugs compounded from an approved animal or human drug is permitted, (21 C.F.R Section 530), it further states that “nothing (in [Part 530]) shall be construed as permitting compounding from bulk drugs.” This statement emphasizes that the law and its regulations do not address compounding from bulk drugs (ie, compounding from drugs is not legalized and thus, according to the
FDA, is illegal). It was included in the law, in part, because compounding from bulk substances is perceived by the FDA to place humans at an increased risk to inappropriate drug residues. It is this statement that is the focus of challenge by the pharmacy profession as it seeks Congressional action to change the FDA’s interpretation of compounding from bulk substances in animals. Confusion has surrounded the issue of compounding of animal drugs; this reflects, in a part, wording of the law. The 2003 CPG specifically state that AMDUCA does not permit veterinarians to compound unapproved, finished drug products from bulk drug substances, unless the finished drug is not a new animal drug. Because any compounded animal drug is a new (yet unapproved), animal drug, then no circumstances exist in which compounding from bulk substances is allowed (except for bulk substances delineated in Appendix A of the CPG).

In the 2003 CPG, compounding actions considered for regulatory action by the CVM include violations that may result in harm to public health (eg, involves compounding for food animals) are most likely to be regulated, followed by compounding that may harm animal health. Further considerations include if: 1. The health of the animal being treated with the compounded drug is not threatened and if suffering or death is not likely to result from failure to treat with the compounded product. 2. Compounding is done in anticipation of prescriptions, unless in limited amounts as indicated by a prescription issued in the confines of a veterinary client-patient-relationship. 3&4. Compounding is performed using drugs prohibited for extra-label use in either food or non-food producing animals or from drugs with a restricted distribution system (drugs whose use is restricted by the FDA, such as thalidomide). 5. Compounding occurs from drugs that are not approved (human or animal, including bulk drugs) unless the product is specifically addressed for regulatory discretion by the FDA in Appendix A (see below). 6. Compounding involves the use of commercial scale manufacturing equipment (implying the manufacture of large amounts of drug products, in anticipation of need, and thus not patient driven). 7. Compounding occurs for third parties with subsequent resell to individual patients. While resale of compounded products is considered illegal, some State Boards of Pharmacy allow, while others do not (see below), “for office use” products which are intended for short-term dispensing to animals (clients) when prescription availability is precluded (eg, weekends or evenings). 8. Compounding is not in compliance with applicable state pharmacy laws. 9. Compounding results in piracy, that is, the compounded product mimics an FDA-approved (human or animal) product which is commercially available in finished dosing form and appropriate for treating the patient. Importantly, this guideline indicates that cost is not a justifiable reason for use of a compounded product that replaces a more costly, commercial product. Piracy of commercially available pharmaceutical animal products is prolific (particularly equine products but is a marked financial disincentive for pharmaceutical manufacturers to pursue approval of animal drugs. Indeed, some manufacturers that would have pursued approval of a generic animal drug product have chosen to offer compounded versions of the products instead, a decision that is illegal if not driven by individual patients. 10. The compounded label does not contain sufficient information as delineated in AMDUCA regulations and 11. In food animals: exclusion of the use of human drugs, avoidance of drug residues and scientific establishment of withdrawal times.

Drug Quality and Security Act (DQSA)

In 2012, an outbreak of fungal meningitis in humans was traced to contaminated injectable glucocorticoids compounded by a pharmacy. This incident, which led to 64 deaths and over 750 illness, redirected Congress towards effort to increase FDA’s ability to regulate compounding. As such, DQSA was intended to correct what the 1996 FADMA was not able to accomplish. High points of the bill, which was passed in 2013, include: exemption of compounded drugs from new drug labeling and track and trace requirements if the drug is compounded by or under the direct supervision of a registered pharmacist and if the compounding takes place in a registered outsourcing facility (currently registered facilities can be found at: http://www.fda.gov/Drugs/GuidanceComplianceRegulatoryInformation/PharmacyCompounding/ucm378645.htm). Some of the differences between an outsourcing facility and a traditional compounder include the following. Traditional compounders are either pharmacists or physicians who receive orders for individuals and anticipatory compounding is limited to individuals patients or the physician. In contrast, outsourcing facilities are considered compounding manufacturers that can compound sterile drugs either with or without a prescription. In regards to the source of active ingredients, traditional compounders can compound from FDA approved drugs, USP monographs or a list of “positive” drugs. In contrast, outsourcing facilities can compound only from the FDA’s drug shortage list, or the FDA’s list of bulk compounds. The bulk compounds must be from a registered facility. Such compounding must be limited to 5% interstate sales or the state of origin must have a Memorandum of Understanding with the FDA. Neither traditional nor outsourcing facilities can compound from withdrawn or removed drugs, or those from the “difficult to compound” list that are considered unsafe or ineffective. To facilitate quality, traditional compounders must follow USP monographs if not compounding from the list. Outsourcers, but not traditional compounders, must follow GMP requirements and are subject to risk-based FDA inspection protocols. The facilities must report to the FDA. Other considerations of the bill include, but are not limited to the following: publication of a list of drugs (generated through an advisory committee) considered to be difficult to compound and thus might reasonably lead to an adverse effects (safety or effectiveness); improves communication between the federal government and state boards of pharmacy in regards to disciplined compounding pharmacies; removes FADMA prohibitions; notes that a compounded product will be considered misbranded if the advertising or promotion of a compounded drug is false or misleading. The
bill also prohibits resale of a compounded drug labeled “not for resale,” or the intentional falsification of a prescription for a compounded drug.

The FDA has deemed that the DQSA does not apply to animals. The Government Accountability Office (GAO) has been given the task of addressing the relevance of the law to veterinary medicine. Currently, animal guidelines are not likely to allow from compounding from bulk unless from a specific list. In response, the AVMA has sponsored a Task Force that will advise Congress. In its most recent report, the AVMA has identified the following subjects related to compounding from bulk as “critical” for discussion/delineation: 1.Adverse event reporting; 2.Labeling/disclosure of the product being compounded to clients; 3.Office stock (to allow up to 14 days work); 4.Drug shortages/unavailability (to allow compounding from bulk and to have a notification system); 5.Compounding from bulk API; 6.Quality assurance testing; 7.Liability; 8.Drug mimics (to disallow); and 9.Compounding in the lab animal/wildlife/zoo/aquaria (removed from restriction).

State regulatory considerations
In addition to federal laws (AMDUCA, etc), all actions related to pharmacy, including compounding, are regulated by State Boards of Pharmacy. However, individual states vary in the applicability of these laws to compounding veterinarians. Most, but not all states, recognize a veterinarian’s right to compound. Many states have specific regulations for veterinary compounding; in their absence, human compounding regulations apply. Rarely, State Veterinary Medical Boards regulate veterinary compounding. The regulations of the states are quite variable. Some State Boards of Pharmacies allow activities that are clearly in conflict with AMDUCA (such as allowing compounding of animal drugs from bulk substances by some states versus removal of veterinarians right to compound by others). In light of the changes in both human and animal compounding CPG, many State Boards of Pharmacy are re-examining their rules and regulations regarding compounding. The National Association of Board of Pharmacies (NABP; www.nabp.net) is a non-regulatory organization that attempts to provide standards and conformity for individual State Boards of Pharmacy. Currently, this association is generating standard regulatory guidelines (within a model Practice Act) regarding many aspects of pharmacy practice, including compounding, which might be implemented among the states. Because the NABP has recognized the increase in veterinary drug compounding, it has begun to address problems and concerns of the veterinary profession such as compounding by pharmacists that are unaware of differences in regulatory philosophy, or “rogue” pharmacists that are indifferent to the regulations. Veterinarians that dispense or prescribe compounded drugs should become aware of the relevant state laws; (http://www.avma.org/issues/drugs/compounding/default.asp). It is noteworthy that since the passage of DQSA, several states have implemented new state laws that are intended to address some of the issues related to compounding. Among the actions are those that address compounding of office stock. According to the AVMA’s State Legislative and Regulatory Affairs Department, currently, 22 states allow veterinary offices to administered compounded products, but specifically prohibit them from dispensing products compounded by a pharmacy; 5 states allow veterinary offices to administered and dispense compounded products, with selected conditions; 11 states allow administration of compounded products, but do no address office dispensing of pharmacy compounded products, and 3 states that have laws and regulations that address compounding in general but not administrating or dispensing by practitioners. Seven states have no laws that address compounding.
Pharmacogenomics: The Role in Drug Interactions and Adverse Drug Events
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Among the animal species, differential response of breeds to xenobiotics is increasingly being understood though the discipline of pharmacogenomics. Pharmacogenomics is the genome wide analysis of genetic determinants of drug metabolizing enzymes, receptors, transporters and targets that influence therapeutic efficacy and safety and drug related phenotypes. It is discriminated from pharmacogenetics is the study of specific defects in specific genetic determinants or genes responsible for specific responses (or gene functions) to drug therapy. Based on systematic searches of variability in DNA sequencing of genes responsible for drug movements, this relatively new discipline will facilitate design of dosing regimens (both drug selection and the regimen itself) for patients such that efficacy might be optimized and toxicity minimized. Genetic variation can influence every aspect of drug disposition (ADME). However, it is important to realized the genetic variation may also alter response to drugs due to differences in drug-receptor interactions (pharmacodynamics) which are not addressed in this manuscript. For example, in humans, polymorphisms have been described for angiotensin-converting enzyme, beta-2 adrenergic receptors, dopamine receptors, and estrogen receptors. Variants have also been associated with differences to anti-infective and cancer drugs.

Among the best described differences are in metabolism due to variation in CYP enzymes (particularly for humans) and transport proteins (efflux and influx), which increasing are being identified in animals as a basis for disease. Multiple drugs serve as substrates for P-glycoprotein, including vinblastine, taxol, digoxin, selected morphine derivatives, loperamide, ivermectin, cyclosporine A, teriflunomide, macrolide antimicrobials and others. Human polymorphisms (genetic variations) in CYP metabolic enzymes have been associated with phenotypic expressions of both therapeutic failure resulting from extremely rapid metabolism of a xenobiotic, and toxic effects, due to decreased metabolism. Polymorphism of the MDR1 gene and P-gp have been reported in humans and are associated with altered disposition and thus susceptibility to adverse events.

Drug interactions occur whenever the action of one drug is modified by the presence of another, concurrently administered drug. Their incidence increases with the number of drugs included in the preparation and with the duration of treatment. Drug interactions may occur prior to or after the drug is absorbed. Factors at the site of drug administration might also interact with a drug, altering its disposition.

Pharmacokinetic drug interactions
Drug interactions that occur inside the body may be life-threatening. Pharmacokinetic interactions occur when one drug alters the disposition of another drug. Each stage of disposition of a drug--absorption, distribution, metabolism, or elimination--can be altered by another drug.

Oral absorption
Drug interactions that have an impact on absorption may also reflect altered metabolism and/or uptake in the enterocyte or liver. Drugs entering enterocytes are subject to metabolism by CYP3A, efflux mediated by P-gp, or passage into the portal system and exposure to metabolic activities of the liver. Cytochrome P450 3A is the most predominant drug-metabolizing enzyme in the intestinal cell and mediates biotransformation of more than half of all drugs currently available (for humans). Interestingly, oral pentobarbital also increases the amount of CYP2C present in the intestinal tract of dogs when administered at doses consistent with that consumed by dogs fed commercial dog foods prepared from animals euthanized by pentobarbital (10 to 60 μg/gm). Transporters such as the multidrug export pump P-gp, facilitate either drug absorption or efflux from the enterocyte, and drugs may act as substrates or inhibit or compete at these proteins. Both proteins (CYP 3A and P-gp) share drug substrates, and interplay between metabolic enzymes and transporters appears to confound the disposition of many orally administered drugs. Poor oral bioavailability may reflect a coordinated action of intestinal drug-metabolizing enzymes and efflux transporters; both drugs and dietary constituents can compete for, or induce or inhibit these proteins. Of the potential drug–nutrient interactions affecting absorption, competition among substrates for transport proteins have probably been the best described. For example, flavonoids (found in grapefruits) are inhibitors of several P-gp substrates, which increases the risk of diet–diet or diet–drug interactions during both absorption and distribution.

The role of efflux proteins in drug absorption can be profound; partnership with CYP3 or other drug metabolizing enzymes further impacts absorption. For example, Mealey et al has begun to characterize the distribution of CYP3 in the intestinal villi in dogs, noting marked variability among animals. P-glycoprotein distribution appears to pattern humans and its impact on absorption in dogs has been demonstrated through the use of a P-glycoprotein inhibitor: when used in combination with drugs effluxed by the protein, oral bioavailability of an antineoplastic agent (doxetaxol) which is a substrate of P-glycoprotein increased 17 fold.

Distribution
Pharmacokinetic drug interactions that alter drug distribution from the central compartment to peripheral tissues usually result from competition for protein-binding sites between two or more concurrently administered drugs, although altered tissue perfusion is also
Pharmacokinetic drug interactions frequently alter the metabolism of a concurrently administered drug. When administering a drug metabolized by the liver, it is wise to anticipate a drug interaction if a second drug also metabolized by the liver is added to therapy. Most of the interactions result from modulation of hepatic (phase I) drug metabolizing enzymes. Induction of drug metabolizing enzymes is a protective mechanism which facilitates excretion of potentially toxic compounds. However, induction is a double-edged sword: although inducers generally increase the elimination of a potentially toxic drug, increased formation of toxic or carcinogenic metabolites may also occur. Most CYP enzymes are inducible, although the response to inducers varies within and among species. Human CYP known to be influenced by inducers include CYP 1A1/2, 2A6, 2C9, 2C19, 2E1, and 3A4. Inducers are substrates for the enzymes; induction generally is dose dependent and often, enzyme specific although more than one CYP may be induced. Inducers may result in significant activity of an enzyme that otherwise is present in very low concentrations or absent. Maximal transcription in response to an inducer generally requires 10 to 12 hrs of exposure to a drug, with the impact generally resolved by 18 hrs (dependent on dose) following discontinuation of exposure to the inducer. However, the impact of the inducer may persist, depending on the rate of CYP degradation. The time required for the CYP to reach a new steady-state concentrations in response to an induced rate of synthesis is determined by its rate of degradation.

Barbiturates are recognized inducers of CYP; indeed, the observation that “tolerance” to hypnotics developed in dogs chronically exposed to barbiturates lead to the recognition of the phenomena of induction. Phenobarbital is one of the most potent microsomal enzyme inducers known and can enhance the hepatotoxicity of other hepatotoxic drugs. Likewise, it increases the formation of and response to pro-drugs and decreases the effects of itself and other drugs metabolized by the liver as clearance of these drugs is increased. The CYP2B9 family, responsible for metabolism of a large number of drugs in rodents, is induced by phenobarbital. Therapeutic doses of phenobarbital have been associated with induction of CYP1A activity. Epileptic dogs treated with phenobarbital may encounter an initial decrease in concentrations after several months of therapy despite no dose change. Other anticonvulsant drugs demonstrated to be impacted (concentrations lowered) by phenobarbital include zonisamide and levetiracetam.

A number of inhibitors of CYP enzymes also have been identified. Some inhibitors are characterized by broad-enzyme inhibition, where as others are selective for a single enzyme. Reversible inhibition is most common, is most often (but not always) competitive, and is transient, resolving when therapy with the inhibitor is discontinued. Like induction, reversible inhibition appears to be dose-dependent. Generally, clearance of a concurrently administered drug metabolized by the liver is decreased, increasing the potential for toxicity or for an exaggerated pharmacologic response. Additionally, pro-drugs (e.g., enalapril, prizomide) are less likely to be activated. Chloramphenicol, imidazole antifungals, and cimetidine are examples of potent microsomal enzyme inhibitors. Co-administration with potentially toxic drugs that are also metabolized by the liver should be done cautiously. Fluorinated quinolones such as enrofloxacin and marbofloxacin can increase theophylline plasma concentrations to toxic levels, presumably due to impaired hepatic clearance of theophylline. The inhibitory effect of erythromycin may reflect inhibition of CYP 3A; although azithromycin is purportedly less likely to inhibit, our lab has documented marked increase in cyclosporine concentrations in patients receiving azithromycin. Drug-induced inhibition of drug metabolism can be used for therapeutic benefit. Both cyclosporine and ketoconazole are substrates and inhibitors of both P-gp and CYP3A. As such, the combined use of the drugs may result in marked prolongation of the half-life of either drug. Our laboratory has documented an elimination half-life of over 150 hours for cyclosporine (normal 4-5 hrs) in dogs simultaneously receiving ketoconazole with the intent of lowering the dose (and thus cost) of CsA while maintaining cyclosporine concentrations. Cimetidine-induced enzyme inhibition has been used to prevent metabolism of acetaminophen in cats into potentially lethal toxic metabolites. Cilastatin inhibits renal tubular drug metabolism of imipenem; the net effect may prolong the half-life of imipenem, but hepato or renal toxicity resulting from metabolites might also be reduced. Nutrition, sex, age, and other factors can influence how drug metabolizing enzymes respond to drugs. Alcohol and 4-methylpyrazole competitively inhibit alcohol dehydrogenase, the drug metabolizing enzyme that converts ethylene glycol to its lethal metabolite.

As in humans, polymorphism in xenobiotic metabolizing enzymes also has been reported in other animals. The science of pharmacogenomics is just now emerging in dogs and cats. The distribution of drug metabolism in dogs may be different compared to that in humans. That breed differences in xenobiotic metabolism may be clinically relevant has been established for some canine breeds. For example, Court et al., (1999) demonstrated that propofol metabolism was about three fold less in Greyhounds compared to...
Beagles. Celecoxib metabolism was attributed to CYP2D15, for which three canine variants were found. Polymorphism also has been described for CYP2C isoenzymes, again in Beagle dogs. Polymorphisms in CYP have also been described in cats: three variants of CYP2E were described by Tanaka et al., (2005), although breeds were not cited. Gender differences also are emerging in CYP.

Most pharmacogenetic variations thus far identified have focused on phase I (especially CYP) drug metabolizing enzymes. However, variability in Phase II can also impact therapeutic success. An example of species polymorphism occurs in cats: they have a pseudogene, rather than a functional glucuronyltransferase gene. As such, acetaminophen and other drugs are not conjugated with glucuronide as they are in other species. Another species differences occurs in acetylation: dogs are deficient in N-acetyltransferase, an enzyme responsible for elimination of sulfonamides. Presumably, shunting sulfanilamide based antimicrobials to phase I metabolism such that the aryl-amine is metabolized to a nitroso molecule can lead to the hypersensitivity reactions that occur in some dogs. An example of a within species polymorphism is variation in the TMPT (thiopurine methyltransferase) enzyme. Variability has been associated with toxicity: low activity increases susceptibility to azathioprine-induced bone marrow suppression. Variability in activity ranges at least 9 fold with Giant Schnauzers characterized by low and Alaskan Malamutes by high activity.

Excretion
Pharmacokinetic drug interactions may alter urinary excretion due to changes in glomerular filtration and/or competition between the drugs for transport pumps that effect active tubular secretion. Competition for either acidic, basic or organic carrier proteins responsible for active tubular secretion usually involves acidic drugs (beta-lactams, sulfonamides, NSAIDs, metabolites of phase II metabolism, furosemide) but might also involve basic drugs (eg, procainamide, dopamine, trimethoprim, opioiods). Probenecid is still occasionally used to prolong the elimination of an expensive penicillin because it competes with the penicillin for a carrier protein. Drugs that alter urinary pH and tubular resorption may also affect renal excretion. Changes in urinary pH conducive to formation of a greater proportion of unionized drug (eg, an acidic urinary pH and an acidic drug) will encourage tubular reabsorption of a drug, thus decreasing its clearance and prolonging its elimination half-life. For example, overdosing of some drugs (eg, strychnine) can be treated by hastening elimination with urinary acidifiers.

Among the most commonly identified drug–diet (nutrient or supplement) interactions that alter drug pharmacokinetics are those reflecting drug metabolism, particularly CYP3A4. Several dietary supplements are known to interact with drugs. These include, but are not limited to, St. John’s wort (induction of CYP3A4, particularly intestinal), echinacea (induction and inhibition of intestinal CYP), ginkgo biloba (induction of CYP219A), and grapefruit (inhibition of CYP3A4 and inhibition of P-gp).
### Interactions between Drugs at Cytochrome P450 Enzymes (Humans)

#### CYP1A2
<table>
<thead>
<tr>
<th>Substrate</th>
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<th>Inhibitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>acetaminophen</td>
<td>ondansetron</td>
<td>insulin</td>
</tr>
<tr>
<td>amitriptyline</td>
<td>propranolol</td>
<td>omeprazole</td>
</tr>
<tr>
<td>clomipramine</td>
<td>theophylline</td>
<td>tobacco</td>
</tr>
<tr>
<td>clozapine</td>
<td>verapamil</td>
<td>fluoroquinolones</td>
</tr>
<tr>
<td>imipramine</td>
<td>warfarin</td>
<td>ticlopidine</td>
</tr>
<tr>
<td>naproxen</td>
<td>zileuton</td>
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</tr>
</tbody>
</table>

#### CYP2B6
<table>
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<tr>
<th>Substrate</th>
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<th>Inhibitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>clophosphamide</td>
<td>phenobarbital</td>
<td>ticlopidine</td>
</tr>
<tr>
<td>clophosphamide</td>
<td>carbamazepine</td>
<td>cimeticidine</td>
</tr>
<tr>
<td>lansoprazole</td>
<td>imipramine</td>
<td>prednisone</td>
</tr>
<tr>
<td>omeprazole</td>
<td>indomethacin</td>
<td>rifampin</td>
</tr>
<tr>
<td>diazepam</td>
<td>primidone</td>
<td>indomethacin</td>
</tr>
<tr>
<td>phenytoin</td>
<td>progestrone</td>
<td>ketoconazole</td>
</tr>
<tr>
<td>phenobarbital</td>
<td>propranolol</td>
<td>lansoprazole</td>
</tr>
<tr>
<td>amitriptyline</td>
<td>warfarin</td>
<td>omeprazole</td>
</tr>
<tr>
<td>clomipramine</td>
<td>peroxine</td>
<td>phenytoin</td>
</tr>
<tr>
<td>naproxen</td>
<td>zileuton</td>
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#### CYP2C19
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</tr>
</thead>
<tbody>
<tr>
<td>cyclophosphamide</td>
<td>cimetidine</td>
<td>fluoxetine</td>
</tr>
<tr>
<td>lansoprazole</td>
<td>imipramine</td>
<td>prednisone</td>
</tr>
<tr>
<td>omeprazole</td>
<td>indomethacin</td>
<td>rifampin</td>
</tr>
<tr>
<td>diazepam</td>
<td>primidone</td>
<td>indomethacin</td>
</tr>
<tr>
<td>phenytoin</td>
<td>progestrone</td>
<td>ketoconazole</td>
</tr>
<tr>
<td>phenobarbital</td>
<td>propranolol</td>
<td>lansoprazole</td>
</tr>
<tr>
<td>amitriptyline</td>
<td>warfarin</td>
<td>omeprazole</td>
</tr>
<tr>
<td>clomipramine</td>
<td>peroxine</td>
<td>phenytoin</td>
</tr>
<tr>
<td>naproxen</td>
<td>zileuton</td>
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</table>

#### CYP2C9
<table>
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<th>Substrate</th>
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<th>Inhibitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSAIDS</td>
<td>glipizide</td>
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</tr>
<tr>
<td>ibuprofen</td>
<td>loxartan</td>
<td>flunoxazol</td>
</tr>
<tr>
<td>diclofenac</td>
<td>amitriptyline</td>
<td>isoniazid</td>
</tr>
<tr>
<td>meloxicam</td>
<td>celecoxib</td>
<td>lovastatin</td>
</tr>
<tr>
<td>naproxen</td>
<td>fluoxetine</td>
<td>paroxetin</td>
</tr>
<tr>
<td>piroxicam</td>
<td>phenytoin</td>
<td>phenbutazone</td>
</tr>
<tr>
<td>tolbutamide</td>
<td>warfarin</td>
<td>proberenicid</td>
</tr>
<tr>
<td>clofibrate</td>
<td>flaxseed</td>
<td>flaxseed</td>
</tr>
<tr>
<td>clofibrate</td>
<td>flaxseed</td>
<td>flaxseed</td>
</tr>
<tr>
<td>clofibrate</td>
<td>flaxseed</td>
<td>flaxseed</td>
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#### CYP2D6
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<th>Substrate</th>
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<th>Inhibitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>carvedilol</td>
<td>encaidine</td>
<td>dexamethazone</td>
</tr>
<tr>
<td>metaprolol</td>
<td>fluoxetine</td>
<td>rifampin</td>
</tr>
<tr>
<td>timolol</td>
<td>flecainide</td>
<td>chlorpromazine</td>
</tr>
<tr>
<td>amitriptyline</td>
<td>lidocaine</td>
<td>chlorpheniramine</td>
</tr>
<tr>
<td>clomipramine</td>
<td>meloxicam</td>
<td>cimetidine</td>
</tr>
<tr>
<td>paroxetine</td>
<td>nortriptilne</td>
<td>cimetidine</td>
</tr>
<tr>
<td>chlorpheniramine</td>
<td>ondansetron</td>
<td>doxurubicin</td>
</tr>
<tr>
<td>chlorpropramidine</td>
<td>propranolol</td>
<td>fluoxetine</td>
</tr>
<tr>
<td>codeine</td>
<td>tramadol</td>
<td>flucloxacine</td>
</tr>
<tr>
<td>codeine</td>
<td>tramadol</td>
<td>flucloxacine</td>
</tr>
<tr>
<td>codeine</td>
<td>tramadol</td>
<td>flucloxacine</td>
</tr>
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#### CYP2C9
<table>
<thead>
<tr>
<th>Substrate</th>
<th>Inducer</th>
<th>Inhibitor</th>
</tr>
</thead>
<tbody>
<tr>
<td>clarithromycin</td>
<td>hydrocorisone</td>
<td>barbiturates</td>
</tr>
<tr>
<td>erythromycin</td>
<td>progesetone</td>
<td>carbamazepine</td>
</tr>
<tr>
<td>(not azithromycin)</td>
<td>testosteron</td>
<td>glucocorticoids</td>
</tr>
<tr>
<td>quinidine</td>
<td>vepine</td>
<td>phenobarbital</td>
</tr>
<tr>
<td>alprazolam</td>
<td>cocaine</td>
<td>phenytoin</td>
</tr>
<tr>
<td>diazepam</td>
<td>dapsone</td>
<td>rifampin</td>
</tr>
<tr>
<td>midazolam</td>
<td>codeine</td>
<td>St. John’s wort</td>
</tr>
<tr>
<td>cyclosporine</td>
<td>dextromethorphan</td>
<td>troglitazone</td>
</tr>
<tr>
<td>tacrolimus</td>
<td>finasteride</td>
<td>pioglitazone</td>
</tr>
<tr>
<td>cisapride</td>
<td>fenetyl</td>
<td>norflaxacin</td>
</tr>
<tr>
<td>chlorpheraseine</td>
<td>ondansetron</td>
<td>verapamil</td>
</tr>
<tr>
<td>amiodopine</td>
<td>lidocaine</td>
<td>amiodopine</td>
</tr>
<tr>
<td>diltiazem</td>
<td>propranolol</td>
<td>diltiazem</td>
</tr>
<tr>
<td>felodipine</td>
<td>quinine</td>
<td></td>
</tr>
<tr>
<td>mifedipine</td>
<td>terfenadine</td>
<td></td>
</tr>
<tr>
<td>verapamil</td>
<td>vincristine</td>
<td></td>
</tr>
<tr>
<td>lovastatin</td>
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### Drug-Herbal/Botanical Interactions

<table>
<thead>
<tr>
<th>Drug</th>
<th>Supplement</th>
<th>Side Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alprazolam</td>
<td>Kava</td>
<td>↑ CNS effects</td>
</tr>
<tr>
<td>Anesthetics, sedatives</td>
<td>St. John’s Wort, valerian, kava</td>
<td>↑ CNS effects</td>
</tr>
<tr>
<td>Antiplaque*</td>
<td>Garlic, Ginger, Cinnamon, Chamomile, St. John’s Wort</td>
<td>Prolonged bleeding time</td>
</tr>
<tr>
<td>Anticoagulants</td>
<td>Feverfew, Bromelain, Danggui</td>
<td></td>
</tr>
<tr>
<td>Antiviral (eg, ribonavler)</td>
<td>Garlic (Allium)</td>
<td>↓ due to↑ CYP 3A4 and P glycoprotein</td>
</tr>
<tr>
<td>Cardiac stimulants</td>
<td>Ephedra, Ginseng</td>
<td>↑ risk of cardiac arrhythmias</td>
</tr>
<tr>
<td>Chlorpropramide</td>
<td>Garlic (Allium)</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>CNS stimulants</td>
<td>Ephedra, Yohimbine, Guarana, Guarana</td>
<td>↑ CNS stimulation</td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>St. John’s Wort</td>
<td>↓ due to↑ CYP 3A4 and P glycoprotein</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>Dextromethorphan</td>
<td>↓ due to↑ P glycoprotein</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Sena, Loricor</td>
<td>Electrolyte disturbances</td>
</tr>
<tr>
<td>Hypoglycemics</td>
<td>Ginseng, Bilberry, Dandelion, Hypoglycemia</td>
<td></td>
</tr>
<tr>
<td>Immunosuppressants</td>
<td>Echinacea, Astragalus</td>
<td>Antagonizes immunsuppressive effects</td>
</tr>
<tr>
<td>Loperamide</td>
<td>St. John’s Wort</td>
<td>MDA inhibition (↑effect)</td>
</tr>
<tr>
<td>Oral drugs</td>
<td>Senna</td>
<td>↓ absorption</td>
</tr>
<tr>
<td>Propranolol</td>
<td>Piperine</td>
<td>CYPIinhibition (↑effect)</td>
</tr>
<tr>
<td>SSRIs</td>
<td>St. John’s Wort, SAMe, Simalayn</td>
<td>↑ risk of side effects, serotoninergic crisis</td>
</tr>
<tr>
<td>Theophylline</td>
<td>St. John’s Wort</td>
<td>↓ due to↑ CYP 3A4 and P glycoprotein</td>
</tr>
<tr>
<td>Theophylline</td>
<td>Piperine</td>
<td>CYPIinhibition (↑effect)</td>
</tr>
<tr>
<td>Thiazide duretics</td>
<td>Gingko</td>
<td>↓ clearance (enhanced effect)</td>
</tr>
</tbody>
</table>
Interactions” in this same proceedings. The role of enantiomers has led to drugs designed to exclude in effective or toxic of cytochrome P450 (CYP450). A more focused discussion of each of these protein classes can be found in the discussion of “Drug Interactions” in this same proceedings. The role of enantiomers has led to drugs designed to exclude in effective or toxic enantiomers. Finally, novel drug delivery systems are emerging as tools to improve drug efficacy and safety. Although most of these advances are occurring in human medicine, translational studies in animals with spontaneous disease may offer guidance regarding their use in veterinary medicine as well. However, it is important to note that this document is not necessarily promoting their use in animals; rather it will focus on those directions that appear to hold most promise in development. Two innovative approaches to drug therapy will be discussed: liposomal drug delivery and designer small drug therapy, with a focus on tyrosine kinase inhibitors.

Liposomal drug delivery
Liposomes are artificially induced lipid (hydrophobic) bilayer vesicles which encapsulate an aqueous (hydrophilic) vehicle containing a medicament (drug or nutrient) of interest. A common method of formation is sonication of biological membranes. Phospholipids comprising the structure are natural, and may contain other materials to facilitate delivery (eg, surfactant or ligands that direct attachment to unhealthy tissues). Different methods of drug delivery occur. For example, medicament may be delivered when the bilayer fuses to other bilayers, such as a cell membrane. Alternatively, gradual changes in vehicle pH will allow gradual release of the drug as it becomes unionized and thus able to penetrate the lipid bi-layer. Liposomes of different sizes can be targeted for phagocytosis by macrophages which then digest the liposome, releasing drug to a targeted cell. Liposomes also have been sued to deliver DNA to a host cell (lipofection). Liposomal technology is sophisticated and complex, with mitigating considerations being the phospholipid used, the homogeneity of particles sizes and its influence on liposomal stability. “Stealth” liposomes have been coated with polyethylene glycol (pegylated: PEG), which is inert and thus allows avoidance of the reticuloendothelial system, resulting in a longer presence in the body. Pegylated liposomes generally contain ligands (eg, monoclonal antibodies or specific antigens) on their surface to facilitate binding to specific cell types.

Liposomal drugs approved in the USA include the antifungal, morphine, estradiol, amphotericin B, an ocular preparation for preventing macular degeneration and several anticancer drugs including cytarabine and doxorubicin. Several of these drugs have been studied in dogs or cats.

Liposomal Amphotericin B and morphine both have been studied in dogs. The liposomal amphotericin B allows higher dosing with less nephrotoxicity (although not less anaphylactoid reactions); doses of 5 mg/kg were well tolerated in dogs (but not 15 mg/kg) IV daily. Liposomal encapsulated morphine has been studied in dogs: concentrations of 4 ng/ml (the recommended therapeutic concentration) were maintained for 96 hours after IV administration of 3 mg/kg SC.

Liposomal doxorubicin also is available as a pegylated liposome (Doxil®). Its small size is postulated to facilitate distribution in otherwise altered vasculature of tumors, surpassing delivery of the regular product. Despite a half-life of 55 hrs in humans (compared to 30 minutes in dogs or 1 hr in humans for standard doxorubicin, its use does not appear to be associated with myelosuppression or myocardial toxicity typical of the free drug. However, pegylation causes preferential concentration in the skin, and subsequent secretion, creating a unique toxicity referred to as hand-foot syndrome. Doxil® has been administered to cats (n=36) with various malignancies (soft tissue sarcoma, carcinoma, osteosarcoma) in an open clinical trial, with 10/32 cats responding (6/13 sarcomas). The study included dose escalation with the final dose being 50% higher (based on free doxorubicin) than the dose tolerated in dogs (1.5 mg/kg every 3 weeks). Side effects observed included mild to moderate localized hyperpigmentation and alopecia, especially of the chin, mild transient gastrointestinal symptoms, and sudden explained death (2/32). In dogs, in a pre-clinical (for humans) study, the dose-limiting toxicity of Doxil® is an ulcerative cutaneous reaction resembling palmar-plantar erythrodysesthesia (PPES) (hand-foot syndrome in humans). The maximum tolerated dose was 1.0 mg/kg IV every 3 weeks. Doxil® use in dogs (n=51) at 0.75 to 1.1 mg/kg IV every 2 weeks (dose escalation study) revealed the drug to be well tolerated except for PPES. Response rate was 25%, including 5/51 complete remissions. A follow-up randomized CCT in dogs with lymphoma (n=41) were treated with vitamin B6 (pyridoxine) or placebo with Doxil at 1 mg/kg IV every 3 weeks. The remission rate was 70 to 75% in both groups with the relative...
membrane receptors activating tyrosine kinases involved with cancer include growth factors (epidermal growth factors [EGFR], have been found in the human genome. Tyrosine kinases can be activated by molecules located at two sites. Examples of cell rather than in response to signals. Serine and threonine do the same function for their respective proteins. Over 90 tyrosine kinases become unregulated if a protein kinase fails to switch off, as may occur with mutations, thus causing the cell to respond constitutively, Examples include monoclonal antibodies that are prepared by immunizing laboratory animals with a purified version of the specific target and generating monoclonal antibodies through clonal procedures. Note that monoclonal antibodies developed for humans are rendered more human-like by replacing “animal” components of the antibody with human components. Such manipulation calls to question the use of human monoclonal antibodies for treatment of canine or feline diseases. Monoclonal antibodies developed for cancer target growth factors

Small drug molecules are those that are sufficiently small to enter a cell and reach targeted intracellular targets. This is in contrast to larger molecules – such as monoclonal antibodies – that are excluded from cell penetration. The advantage of targeted drug therapy using small molecules is that very specific intracellular molecules can be targeted (hence, “designer drug” therapy), including cell signals responsible for cell division, movement, response to external stimuli and cell death. The molecules are generally identified through testing of 1000s of potential candidates using laboratory methods. Among the first small molecules developed were tamoxifen and toremifene, drug molecules that bind to the estrogen receptor; thus preventing estrogen binding that promotes breast cancer. Other examples included aromatase inhibitors that inhibit estrogen synthesis. Among the more recent and prolific small molecules developed to treat cancer are the tyrosine kinase inhibitors (“tinibs”).

Tyrosine kinase inhibitors (TKI)
Tyrosine kinases are molecules responsible for cellular signal transduction. By transferring a phosphate group from ATP to a tyrosine molecule target protein, the serve as “on-off” switches for signals that communicate cell functions. For example, cancer growth may become unregulated if a protein kinase fails to switch off, as may occur with mutations, thus causing the cell to respond constitutively, rather than in response to signals. Serine and threonine do the same function for their respective proteins. Over 90 tyrosine kinases have been found in the human genome. Tyrosine kinases can be activated by molecules located at two sites. Examples of cell membrane receptors activating tyrosine kinases involved with cancer include growth factors (epidermal growth factors [EGFR], platelet derived growth factor [PDGFR], vascular endothelial growth factor [VEGFR:HER2/neu]); these enzymes have an identified role in breast, gastrointestinal, non-small-cell lung and pancreatic cancer. Non-receptor tyrosine kinases located in the cytoplasm transact signals to the nucleus and are responsible for signals controlling cell-cycle function (mitogenesis, mitosis). For example, “Lyn” is a cytoplasmic tyrosine kinase encoded by the Src (“sarc” for sarcoma) proto-oncogene (a normal gene that mutates to become an oncogene). Src genes are associated with a wide variety of cancers: for example, the first src gene identified was from a chicken sarcoma virus. This family of genes located in the nuclear matrix is responsible for cell cycle control. Mutations can result in increased enzyme activity and thus a loss of growth regulation. Another example is the BTK family of tyrosine kinases that are encoded by the pro-oncogene (resulting from chromosomal transposition) Abl that is responsible for chronic myeloid leukemia. Serine and threonine kinases have identified roles in malignant melanoma, colorectal and ovarian cancer. It is important to note that inappropriate tyrosine kinase activity has been implicated in variety of other disorders, including local (eg, chronic inflammatory disease) and systemic (eg, endotoxic shock) inflammation. They play a major role in lymphocyte regulation.

A number of tyrosine kinase inhibitors have been developed in human medicine to treat a variety of cancers: imatinib (gastrointestinal stromal tumors or chronic myeloblastic leukemias), nilotinib (chronic myelogenous leukemia, targeting Abl), sunitinib (VEGFR, PDGFR and others), and gefitinib (EGFR). Tyrosine kinase inhibitors (TKI) are designed to compete with target substrates for tyrosine kinase activity. The more specific the drug for its target molecule, the less the toxicity. Potential toxicities include myocardial damage and protein loss (renal?), the latter monitored at least bi weekly for the first 3 months of therapy and then monthly thereafter (treatment is discontinued until resolved and then re-instituted at a reduced manner).

Mast cell disease in dogs
Mutations in tyrosine kinases (particularly Kit) and enzymes and receptors JAK, PDGFR, Src-kinases, and the histamine H4 receptor have been associated with loss of mast cell activity regulation. In dogs, high grade mast cell tumors frequently are associated with mutations in the c-kit gene, resulting in constitutive phosphorylation of KIT. Imatinib targets several tyrosine kinases, with major

risk of PPES being 4 fold greater in the placebo group. The dose of Doxcil® achieved in the treatment group was 5 mg/kg compared to 4 mg/kg in the placebo group which appeared to contribute to longer remission in the treatment group. Doxcil® administered intraabdominally did not result in statistically significant improvement in dogs with hemangiosarcoma, although the treatment size was small.

Targeted designer (molecular) drug therapy
Targeted drug therapy focuses on a specific molecule that is relatively unique to the cell causing diseases. The term emerged largely in the context of treating cancer with target molecules being those that supported tumor growth or metastasis such as cell growth signaling, tumor blood vessel formation, or promotion of cell death (apoptosis). Some targets are intended to stimulated the immune system to destroy specific cells. Finally, targeted molecular therapy includes delivery of toxic drugs to specific sites. Targeted molecular therapy increases both efficacy and safety. Perhaps not intentionally, terms such as “macromolecules” and “small molecules” are emerging to describe the cellular site of action of these drugs. Macromolecules are large and as such, face multiple barriers before intracellular target molecules are reached. Such drugs may be more effective if targets are located on cell surfaces. Examples include monoclonal antibodies that are prepared by immunizing laboratory animals with a purified version of the specific target and generating monoclonal antibodies through clonal procedures. Note that monoclonal antibodies developed for humans are rendered more human-like by replacing “animal” components of the antibody with human components. Such manipulation calls to question the use of human monoclonal antibodies for treatment of canine or feline diseases. Monoclonal antibodies developed for cancer target growth factors
targets being KIT, PDGFR, and Abl. Using an open clinical trial design, 21 dogs with MCT (5 positive for the c-kit mutation) were treated with imatinib (10 mg/kg daily) for 1-9 weeks (only 6 dogs were treated for longer than 21 days; drug was d/c in the other dogs at owner’s request due to financial burden). 48% responded, including all dogs with the c-kit mutation. Response to therapy (ie, complete remission) could not be correlated with the presence of the c-kit mutation. Some of the dogs expressing clinical signs of mast cell disease (vomiting, inappetance) had been treated with prednisolone prior to TKI therapy with no response in tumor reduction; 5 of these were among the 10 responding dogs suggesting a potential combination effect. Dogs tolerated the drug well; no evidence of liver disease observed in pre-clinical studies at human doses was observed in this study.

In veterinary medicine, two tyrosine kinase inhibitors (TKI) have been approved for treatment mast cell disease in dogs in the USA. Toceranib (Palladia®; a “sister” to sunitinib), is a receptor TKI that targets over 50 tyrosine kinases; its major targets are KIT, PDGFR and VEGFR2 and Flt-3. Palladia® is marketed as an anti-angiogenic, antiproliferative therapy. The package insert indicates a 59% response rate (complete remission to no progression of disease) at 3.25 m g/kg/every other day. Among the side effects are vascular disorders, including severe diarrhea (> 1/100)thromboembolism (COX-2 specific NSAIDs might need to be avoided), gastrointestinal bleeding, “penias” (neutropenia [> 1/100], anemia, thrombocytopenia), hypoalbuminemia (> 1/10) hepatotoxicity or nephrotoxicity. Masitinib (Kinavet®/Masivet®) has been designed to be more selective than imatinib, targeting KIT, PDGFR and Lyn. Presumably, it is less likely to inhibit tyrosine kinases that result in toxicity, thus allowing higher doses. Based on studies in Europe and the USA, masitinib has increased survival rates in dogs with non-resectable tumors, allowing “curative” therapy in some dogs. Control of disease at 6 months is highly predictive of long-term survival, whereas short-term response at 6 weeks was not. Toxicity (based on review of the package insert) may be less compared to toceranib. Note that drug interactions involving metabolism and transport proteins may be an issue with small drug molecules, and perhaps more so than traditional chemicals. Most TKI in humans are transported by multiple transporters, including P-glycoprotein in portals of entry and sanctuaries, as well as tumor cells. metabolized by major CYP enzymes. The package insert for masitinib does indicate that the activity of a number of CYP enzymes (including 2C9, 2D6, 3A4 and 3A5) are inhibited by masitinib, suggesting care should be taken with combination with other drugs. For Palladia®, no CYP enzymes are cited in regards to potential inhibition by the drug, although enzyme inhibition will increase Palladia® concentrations.

Other oncologic indications for masitinib in dogs include T-cell multicentric lymphoma, feline vaccine induced sarcoma, melanoma and as part of combination chemotherapy (eg, vinblastine, gemcitabine, doxorubicin). Non-cancer potential indications for masitinib include atopic dermatitis (particularly dogs not responsive to cyclosporine, glucocorticoids or dogs with severe pruritus; clinical trial ongoing), inflammatory bowel disease, arthritis and feline asthma.

To understand why oclacitinib is showing up for indications other than atopy, we have to back up and look at its mechanism of action.

Oclacitinib

Apoquel® It is a JAK inhibitor. JAK is "just another protein kinase". When cytokines interact with their target cell membrane receptors, JAK enzymes (phosphorylate) activate another site on that same receptor such that the receptor interacts with a third protein, STAT (signal transducer and activator of transcription). STATS then go on to bind to DNA and subsequently cause the transcription of genes that cause inflammation. There are 4 JAKs (JAK 1, 2 or 3 and TYK2), each responsible for activating specific cytokines; cytokines often interface with two different JAKs. What is the role of cytokines in allergies and atopy? Interleukins (ILs) are among the cytokines that are over produced in allergic patients. These include ILs from Th2 cells (ILs-4, 5, 10, 13,31) and those that stimulate Th2 cells (ILs-25, 33). These ILs promote signs of disease: For example, IL4 causes t cells to proliferate, IL-5, eosinophils and B cells; IL-10 mast cells, Th2, macrophages, and B cells; and IL-13, B cells, particularly IgE. IL-31 is in particular is a pruritogenic cytokine, being responsible for skin inflammation. In spontaneous canine atopy, ILs-4, 5 and 13 are increased and in experimental canine atopy, IL-6, 13 and 18 are important (I did not address interferon gamma and TNF alpha, which also play a role). It is likely that a variety of allergic diseases involve these same cytokines.

How does oclacitinib impact allergies through JAK? As a JAK inhibitor, oclacitinib is most potent at inhibiting JAK1, followed by JAK 2. It is much less potent at inhibiting JAK3 and Tyk2. JAK1 transmits signals for ILs 2, 4, 6 13 and 31 (and others) and JAK2, ILs 6, 13 and 31. As such, oclacitinib should be very effective for atopy. However, both JAK1 and JAK 2 transmit signals for other cytokines (ILs7, 9, 15, 21 for JAK 1 and ILs 3, 5, 12, and 23 for JAK 2). As such, we would expect oclacitinib to have effects on other inflammatory allergic diseases for which JAK 1 and 2 are important, but less effective for diseases in which JAK3 and Tyk2 transmit cytokine signals.

A drug similar to oclacitinib has been approved for use in humans to treat rheumatoid arthritis which is associated with IL-6. If the same interleukin is mediating polyarthritis in dogs, it would make sense that oclacitinib should be considered. Atopy, asthma and IBD are often linked together because of the same underlying pathophysiology (different end organ). Asthma involves ILs 4, 5, 9 and 13, so again, assuming cats read the same papers, it is reasonable to think about using oclacitinib for asthma. In humans, IBD involves ILs 2,6, 10 (which is ANTI-INFLAMMATORY), 17, 21, 22, and 23. So, considering the ILs produced in these diseases and the similar potential role in (what I call) chronic allergic inflammatory diseases, the potential role of oclacitinib is pretty exciting.
The most recently approved drug that targets specific small molecules is grapiprant (Galliprant®). Indicated for treatment of osteoarthritis, this drug differs from nonsteroidal anti-inflammatories (which target cyclooxygenase, and newer ones, cyclooxygenase 2) by targeting (agonizing) the prostaglandin E-2 EP4 receptor. All prostaglandin receptors are g-protein coupled receptors which bind to one or more of 9 prostanoid receptors. PgE is the predominant PG interfaces with 4 receptors (hence, PgE receptors). The receptors is located in areas beyond the joint. In the gastrointestinal tract, they are highly expressed in small intestine and colon, providing an anti-inflammatory role (suggesting this drug might be avoided in patients with IBD). EP4 is important in stimulating bone growth, suggesting antagonisms might impair bone healing and it may be important in cancer growth, suggesting its antagonism might be helpful in cancer. EP4 may also protect the heart. Although this drug is categorized by an NSAID, its mechanism of action is clearly different than cyclooxygenase inhibitors because of its increased specificity. Based on assessment of the package insert, selective inhibition of prostaglandin receptor EP4 appears to be associated with better safety compared to co-inhibiting drugs.
For complicated infections, drugs and doses ideally will be based on C&S. Note that the sample must be collected properly (cystocentesis; do NOT use free-catch data). It also must be handled properly: with a doubling time as short as 20 minutes, a small colony count indicative of no infection can rapidly become a high colony count (>10^5 CFU/ml) indicative of infection. Consider using a “paddle” that maintains colony counting capability during shipping. Finally, it must be performed properly: find a laboratory that uses guidelines and interpretive criteria delineated by the Clinical Laboratory Standards Institute and specifically, those for animals. Tube dilution (minimum inhibitory concentration) data is most helpful: the further the MIC is from the drug concentration achieved at the site, the more likely the drug is to be effective. Remember that an “S” designation does NOT indicate that the isolate has not developed resistance, it only means that effective concentration are likely to be achievable in the patient at the recommended dose. The more at risk the patient is for developing a resistant residual inoculum, however, the less confident the clinician should be in using a drug for which the MIC of the infecting organism is approaching the susceptible breakpoint. The more chronic the infection, the more likely the infection is in the deeper layers of the uroepithelium and protected by biofilm and thus the more important the drug be lipid soluble. In addition, the following should be considered.

1. Bactericidal versus bacteriostatic drug: Although reaching for a “cidal” drug is appropriate, the dose must be designed to assure cidal concentrations are reached. This is much easier for a cidal drug. On the other hand, selected bacteriostatic drugs are capable of killing particularly if accumulated [eg, macrolides and lincosamides in phagocytes; urine concentration.

2. The mutant prevention concentration (MPC). Because each isolate in an infecting inoculum has an MIC, the infecting population is characterized by an MIC distribution for each drug. The MIC yielded from C&S is likely to be the median (50th percentile), with those isolates at the low end most susceptible. Those at the high end represent mutant variants and are least susceptible to the drug. Indeed if the infecting inoculum reaches 10^5 to 7 CFU, spontaneous mutations will allow at least one isolate develop resistance to any drug that might be used. The highest MIC of any of the infecting isolates in the inoculum is the MPC, and this is the concentration that must be reached in order to kill the mutants and avoid emergence of a resistant population. Should drug concentrations at the site of infection reach the mutant selection window (the concentration between the MIC and the MPC), because the most susceptible of the isolates are removed, a more resistant population will fill the resultant void. Once the new population reaches a sufficient size, infection may remerge. The new population will be characterized by a higher MIC compared to the original population. A normal, healthy patient may be able to overcome this infection, but a patient at risk may not be. The MPC cannot be predicted by the MIC but in general will be 10 to 100 or more fold higher than the MIC. Accordingly, dosing regimens should be designed to well exceed the cultured MIC (eg, target MIC90).

3. Postantibiotic Effect (PAE) and relationship between MIC and PDC: The PAE is the continued inhibition of microbial growth after a short exposure of the organisms to the drug. The impact of PAE on efficacy can be profound, particularly for concentration-dependent drugs. It is the PAE that allows some of these drugs to be administered at long intervals. The PAE may be absent for some organisms or some patients (e.g., some immunocompromized patients). In general, concentration-dependent drugs appear to exhibit longer PAE. The duration varies with the peak PDC (ie, higher = longer); the PAE is enhanced by combination antimicrobial therapy.

4. Concentration vs Time Dependency. The relationship between MIC and the magnitude and time course of PDC allows drugs to be fall into two categories. Efficacy of concentration dependent drugs,

<table>
<thead>
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<th>Drug</th>
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<tr>
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<td>1</td>
<td>40</td>
<td>IV</td>
<td>80</td>
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C=concentration dependent
T=time dependent
best represented by FQs and aminoglycosides, is best predicted by the ratio of peak plasma drug concentration (Cmax) compared to MIC of the infecting organism (Cmax:MIC). For such drugs, the magnitude of the IQ generally should be 8-10 or higher for more difficult infections (eg, Pseudomonas aeruginosa), or infections caused by multiple organisms. The duration that PDC is above the MIC is less important; in fact, efficacy may be enhanced with longer intervals. For such drugs, a low dose is particularly detrimental. Thus, the highest dose should be used; this is particularly important for fluorinated quinolones (FQ), because resistance is always associated with multidrug resistance. For example, and MIC of 0.25 means 2.5 mcg/ml should be targeted at the site of infection. This necessitates a dose of 15 to 20 mg/kg. However, FQ efficacy is also related to total exposure; as such, twice daily administration of the same high dose might be indicated for organisms already characterized by low level resistance (see MPC below).

For time-dependent drugs (eg, beta-lactams), efficacy is enhanced if PDC remain above the MIC [T> MIC] for the majority (60 to 70% or more) of the dosing interval. For such drugs, simply achieving the MIC is insufficient because PDC (and certainly tissue concentrations) fall below the MIC immediately. With time-dependent drugs, increasing the Cmax:MIC is likely to be beneficial, but choosing a drug with a longer half-life is important: since drug concentrations decrease by 50% every drug half-life, a Cmax:MIC ratio of two will result in PDC below the MIC in one half-life. The dosing interval can be two half-lives. To increase the dosing interval by two more half-lives, the dose would need to be doubled; to add another two half-lives, the dose would have to be quadrupled. For example, for a *Staphylococcus pseudintermedius* with an MIC for cephalxin of 2 mcg/ml (half-life approximates 3 hr), PDC achieve approximately 20 mcg/ml when given at a dose of 25 mg/kg. In one half-life (3 hr), PDC have dropped to 10; in 6 hr, to 5. Thus, a 12 hr dosing interval might be acceptable. However, this assumes all drug in plasma makes it to the tissue and the half-life is 3 hrs. In contrast, for amoxicillin, a dose of 13 mg/kg results in 4 mcg/ml in the plasma; with a half-life of about 1.5 hr, 90% of the drug is eliminated in 4.5 hours (a 9 hr dosing interval) and only isolates with very low MIC could be treated at this dose every 8 hrs. Indeed, CLSI (standard setting organization for C&S) has recently determined that amoxicillin with or without clavulanic acid should not be used to treat soft tissue gram negative infections; care should be taken even for gram positive infections in at risk patients. We recommend 25 mg/kg every 8 hours and only for very susceptible isolates. Choosing time dependent drugs with long half-lives is prudent.

4. Infections rarely are in plasma: Penetrating the site of infection. Water soluble drugs (beta lactams, aminogyclosides) may not reach concentrations in tissues that equal those in plasma. Even in non-sanctuary tissues, assume that only 30 to 50% water soluble drugs (beta-lactams, aminoglycosides) distribution into extracellular fluid. Thus, doses automatically should be doubled if based on MIC in plasma. Dose must be increased even higher for tissues characterized by non-fenestrated capillaries. For example, distribution of amoxicillin (but not imipenem) to bronchial secretions may be ony 30%. For lipid soluble drugs, distribution into tissues tends to be excellent but for “static” drugs, killing concentrations may not be achieved. An exception may be drugs that are accumulated in WBC (such as macrolides and clindamycin). In general, “I” drugs should be avoided because of this concern. Topical therapy will allow high concentrations to be reached with minimal impact on host systemic toxicity or host microbiota. For UTI, infection is in the bladder wall covered by biofilm; additionally, all patients concentration urine. Further, organisms in biofilm are frequently quiescent and thus not very susceptible to even bactericidal drugs. Most “bacteriostatic” drugs are eliminated in via the liver/bile and do not achieve high concentrations in urine. Thus assuming high concentrations in urine precludes the need for higher doses is not prudent if any complication exists.

A marked host inflammatory response mandates the need for cleansing the site; using drugs accumulated in white blood cells might be prudent. Most infections are associated with biofilm with a formidable barrier to drug penetration. Foreign bodies should be removed whenever possible to help decrease the impact of biofilm.
Controlled Substances: What You Really Need To Know!
Mary L. Berg, BS, RLATG, RVT, VTS (Dentistry)
Beyond the Crown Veterinary Education
Lawrence, KS

Whether you are ordering and monitoring the controlled substance in your practice or administering these substances, it is important that you understand the rules and regulations. This knowledge will not only benefit you and your practice but protect you from potential problems.

Responsibility
All licensed veterinarians that conduct any activities with controlled substance are required to be registered with the Drug Enforcement Agency. This includes individuals that purchase, stock, order, and prescribe controlled drugs (CS). Registration is NOT required for a veterinarian that administers and dispenses as an agent of another veterinarian or veterinary hospital. Registrations are to be maintained at the registered practice and must be readily available. The DEA (Drug Enforcement Agency and the State Boards of Veterinary Examiners are responsible for enforcement of the regulations.

Schedules of Drugs
Drugs are scheduled based upon the degree of severity and risk of addiction. Schedule I drugs such as heroin or cocaine are rarely used in veterinary practices. Examples of Schedule II drugs include Fentanyl, Hydrocodone, Morphine. Schedule III include Buprenorphine and Ketamine, Schedule IV include Buphropanol, Diazepam, Phenobarbital and Schedule V drugs include preparations such as Codeine preparations.

Purchasing
The purchaser must have a DEA registration to purchase CS. An individual is prohibited from writing a prescription to obtain stock for the hospital. CS can be purchased from a distributor or a pharmacy. If the drug is a Schedule II drug a DEA Form 222 must be completed and filed with the DEA and kept as records by the purchaser and the supplier.

Record Keeping
A record must be kept each and every time a CS changes hands or is used on a patient. A proper paper trail that shows the path of the CS from supplier to patient is essential and must be kept for at least 2 years and must be readily available. Receipts of all purchases must be kept. Schedule III-V can be kept in the same file. Schedule II drugs must have a DEA Form 222 and kept in a separate file from the others. It would be highly unlikely for a veterinary practice to have schedule I drugs.

All records for CS must include the following data: date of receipt, drug name, dosage form, strength, quantity received, name, address and DEA number of the supplier and recipient as well as the initials or name of all employees verifying receipt. These records can be handwritten, typed or maintained electronically.

Inventories
Inventories should be conducted regularly but must be conducted at least annually. This requires balancing beginning inventories with amounts purchased to amounts used and total on hand.

Receipts and logs are useful in balancing CS. Logs are strongly recommended. There are commercially available logs or clinics can create a log. Logs should be balanced daily and do not replace annual inventory records. The logs must include patient name, address, drug, drug strength, dosage, route of delivery and initials of the person dispensing. The logs must be kept in a bound binder, not a loose leaf folder.

Purchasing
The purchaser must have a DEA registration to purchase CS. An individual is prohibited from writing a prescription to obtain stock for the hospital. CS can be purchased from a distributor or a pharmacy. If the drug is a Schedule II drug a DEA Form 222 must be completed and filed with the DEA and kept as records by the purchaser and the supplier.

Disposal of unwanted or expired drugs should be sent to reverse distributor and records should be kept for two years. Only contaminated drugs can be disposed of on site by the practitioner and a witness.

Transferring of drugs among veterinarians is allowed but must have a transfer of controlled substances form for the supplier and the receiver. Any schedule II drugs require a DEA Form 222.
Security
CS and prescription pads must be locked in a safe or cabinet with double locks. CS should never be left out during the day. Perform background checks on individuals that have access to the CS. Have an SOP in place that every individual in the practice reads and signs. Conduct regular training on controlled substances and have two individuals present at each use. Do not allow visitors access to the drugs and restrict the number of individuals with access to the drugs. Create a code word with external pharmacies. Review logs for falsifications and perform regular internal audits.

Handling loss and/or theft
It is normal to have some loss in liquid CS. The DEA allows for ~10% loss in liquids. If the loss is significant or in a tablets and the clinic is unable to account for the loss a report must be made to the DEA and the state board of Veterinary Examiners within 7 days.

It is illegal for a veterinarian to prescribe drugs to a human or themselves. Doing so is a felony. It is also illegal for a veterinarian to supply CS to animal shelters, animal control officers, and groomers if that drug is to be used as stock drug.

Warning signs of substance abuse include secrecy, moodiness, abrupt changes in behavior, withdrawal from relationships, changes in appearance, financial stress, and disappearance of medications. The possibility that an individual is experiencing substance abuse can be difficult to address but remaining silent is just as serious. Open a conversation with compassion and be vigilant.

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https://www.aaha.org
https://www.avma.org/KB/Resources/Reference/Pages/dea-registration.aspx
www.veterinaryteambrief.com - Signs and Symptoms of Possible Substance Use
Veterinary patients requiring surgery can be broadly classified into 3 categories as follows: patients requiring immediate surgery to prevent imminent death, patients with varying degrees of hemodynamic stability with injuries that require surgical intervention and patients that are stable and can undergo surgery on a semi-elective basis. Patients within the first and last category require little consideration with regards to timing, however patients within the second category present a dilemma. These patients have often suffered severe trauma and may require surgery for injuries such as long bone fractures, pelvic fractures, severe soft tissue wounds or ballistic injuries. The dilemma arises due to the fact that these patients often develop an inflammatory state similar to sepsis that may not be identified early in their evaluation. In its worst incarnation, patients that undergo inappropriately timed surgical intervention are at risk for developing multiple organ failure due to the addition of surgical trauma/inflammation to the already primed immune response.

Human trauma surgery evolved from an early belief that all surgical procedures should be delayed due to the patient’s being “too sick” to an “early total care” approach in which every patient received definitive surgery as soon as possible. While this approach improved outcomes for many patients a small subset were found to be adversely affected. The most current recommendations for human trauma management focus on a “damage control surgery” (DCS) approach in which a patient is taken to early surgery but the procedure may not be definitive. Patients undergoing a DCS approach require re-operation once they are deemed stable enough to receive definitive care. The DCS approach minimizes surgical trauma while maximizing the benefit (i.e. preventing ongoing contamination or hemorrhage).

The pathophysiology of trauma is complex. The immune recognition of tissue damage is necessary to initiate healing and this recognition occurs trough activation of the immune system. When trauma is severe, this local inflammatory response may become systemic and result in a condition that is virtually indistinguishable from severe sepsis or septic shock. Following a traumatic event two main determinates dictate the host defense response. The first and most important is direct tissue damage from the trauma; the second is sequelae of the inflammatory response. Direct tissue injury is determined by the force and impact severity of the trauma and is commonly referred to as the “first hit”. This first hit in turn dictate the severity of the secondary response or “second hit”. Second hits can be considered endogenous (complications arising from the initial injury; i.e. shock, necrosis, etc.) or exogenous (resulting from attempts to treat the initial injury; i.e. surgical trauma, anesthesia, transfusions, etc.).

Intra-abdominal or intra-thoracic organ damage is the most common clinical problem associated with blunt trauma. Hemorrhage associated with damage to the liver, spleen, kidneys or large blood vessels can be significant. Blunt crushing injuries to intra-abdominal organs including the GI tract can be particularly severe due to the amount of mechanical injury that occurs at the time of trauma. Thoracic injuries such as rib fractures, pulmonary contusions and pneumothorax are often accompanied by shock and hypoxemia.

Long bone fractures are generally associated with a large amount of soft tissue injury in addition to the bony injury. When shock is present the extremities are temporarily sacrificed as precious blood flow is diverted to the core and vital organs. This added hypoxia might exacerbate crushing or shearing injuries and increase the possibility of ischemia and reperfusion injury and secondary infections. Humeral, femoral and pelvic fractures can result in large amounts of blood loss further contributing to the duration and severity of shock. In veterinary medicine blunt torso trauma or crushing bite wounds are the most likely types of trauma to result in a systemic inflammatory response syndrome.

Systemic inflammation secondary to trauma results from stimulation of the innate immune system, the branch of the immune system evolved to respond to novel microbes and to mediate both non-infectious and infectious inflammation. When the inflammatory response is stronger than necessary organ damage and dysfunction can result. The classic example is acute respiratory distress syndrome (ARDS) in which pulmonary capillary permeability is increased leading to the accumulation of pulmonary edema. Necessary activation of the coagulation system occurs in parallel with activation of the immune system at the time of trauma to minimize blood loss. Due to the intricate interactions between these two systems, pro-coagulant states lead to further stimulation of the immune system. Ultimately, this stimulation can result in the development of disseminated intravascular coagulation.

Veterinary guidelines are currently lacking, however extrapolating from the human literature it would appear that the worst timing for surgery of polytrauma patients is between days 2-4 post injury. During this time the systemic inflammatory response and immunologic changes are sustained and the immune system is primed to respond to any additional trauma load. In some cases the very need for surgical intervention is being questioned with a growing movement in human trauma management toward non-operative
management of many blunt injuries. Ultimately, the ideal timing of surgical intervention is likely to be highly patient dependent with the clinician using their judgment to find the balance between adequate resuscitation and prevention of complications associated with delayed surgical intervention (i.e. sepsis, SIRS, MODS).

Specific disease categories
Blunt abdominal trauma can result in many injuries including diaphragmatic hernia, hemoperitoneum and axial skeletal fractures. The forces created during these injuries can cause devastating injuries and death with an overall mortality rate of 10-12%. Unfortunately, human and veterinary studies show physical exam findings and blood work results are unreliable for evaluating the severity of abdominal trauma. Diagnostic tests that may impact the decision of surgical intervention have been investigated in human and veterinary studies. Currently in people suffering blunt abdominal trauma, focused assessment with sonography for trauma (FAST) is the preferred imaging modality compared to CT scan for penetrating injuries. The average time to perform this test is 6 minutes with free abdominal fluid being found in up to 45% of patients.

Penetrating trauma, especially bite wounds, are common in veterinary medicine with reported survival rates ranging from 38% to 100%. Evaluating surgical recommendations for penetrating trauma is made more difficult due to the vast difference in severity of trauma and mechanism of trauma. Abdominal impalement injuries may warrant special consideration for emergency laparotomy as extent of injuries, particularly with sticks, may not be immediately evident due to the potential for these foreign bodies to migrate. Human patients with gunshot wounds to the abdomen are often managed operatively but can be successfully managed non-

The most common etiology of blunt trauma is motor vehicle accidents, accounting for up to 90% of cases. One large, retrospective study combined all blunt trauma cases, analyzing data from 200 dogs and found 50% of these patients required some type of surgical intervention, with 8% requiring multiple surgeries (Simpson et al). Polytrauma was seen in 72% of these cases illustrating how difficult it is to characterize these patients into only one trauma subtype (open fracture vs. hemoperitoneum, etc). The most common surgical procedures were orthopedic (63.5%) followed by soft tissue procedures (36.5%). Hemoperitoneum was present in 23% of cases and hernias were present in 5% with only 5% of dogs with hemoperitoneum requiring emergency surgery. The mean number of days from admission to surgery in this study was 2.2 days (+/- 1.7 days). Unfortunately, the timing of surgery in relation to the time of trauma or admission was not analyzed for outcome. There were no significant associations with mortality and the need for surgical intervention, length of surgery, length of anesthesia or postoperative temperature.

Diaphragmatic hernia (DH) is the only specific injury that has been evaluated in the veterinary literature with regards to surgical timing and outcome. The overall mortality rate for DH ranges from 6.3-20%. Early evidence suggested that early intervention (less than 24 hours) was associated with an increased mortality rate. A more recent study specifically designed to evaluate surgical timing and outcome found that 42.6% of patients went to surgery within 24 hours of injury with a survival rate of 89.7% (Gibson). These findings suggest that surgical intervention within 24 hours of DH may not have an adverse effect on survival, contradicting earlier recommendations. Importantly, most of the patients in this study were stabilized at a primary care practice before being referred to a specialty center for surgery. Paradoxically, it may be necessary in some patients to perform surgery to achieve hemodynamic stabilization.

Hemoperitoneum is diagnosed with abdominal effusion has a PCV within 25% of a patient’s peripheral PCV. Many patients likely expire from life-threatening hemorrhage before stabilization can be attempted leading to the paucity of information in veterinary medicine. In one review, arresting ongoing hemorrhage is fourth on a list of initial stabilization goals and can in some cases be managed without the need for surgery (abdominal counter pressure techniques). If a patient cannot be stabilized with volume expansion, blood products and counter pressure, then emergency surgery is warranted. The timing of surgical intervention with regards to traumatic hemoabdomen has not been analyzed, perhaps because the need for surgical intervention appears to be rare. Surgical readiness not only of the patient but also of the facility is vitality important in cases of hemoperitoneum, especially in trauma patients. Specific staffing needs for a decompensating patient (anesthetist, primary surgeon, assistant) must be considered and met before attempting surgical resolution of a hemoperitoneum. Blood products should be available in the operating suite and operative times kept short. This may include rapid clip and prep if catastrophic hemorrhage is occurring. All emergency and pain-related drug dosages should be calculated before induction and the need for ventilation, blood pressure support, and intensive anesthetic monitoring assumed.

Penetrating trauma, especially bite wounds, are common in veterinary medicine with reported survival rates ranging from 38% to 100%. Evaluating surgical recommendations for penetrating trauma is made more difficult due to the vast difference in severity of trauma and mechanism of trauma. Abdominal impalement injuries may warrant special consideration for emergency laparotomy as extent of injuries, particularly with sticks, may not be immediately evident due to the potential for these foreign bodies to migrate. Human patients with gunshot wounds to the abdomen are often managed operatively but can be successfully managed non-

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operatively. Recent work suggests that an initial non-operative approach to patients meeting specific criteria leads to fewer non-therapeutic surgeries without affecting outcome.

Trauma associated specifically with bite wounds has been well reported with survival rates ranging from 73-83%. The degree of tissue damage, especially with bite wounds, is commonly underappreciated by visual exam and traditional radiography. It is recommended to take patients to surgery based on finding injuries to the thorax or abdomen because of the high potential for intracavitary trauma with early exploratory surgery recommended to rule out more serious internal injury. With regards to thoracic trauma, some recommend exploratory surgery for any patient with a flail chest, rib fractures, lung contusion or pneumothorax but the optimal timing of surgery for these potentially unstable patients is unknown; however many clinicians treat these injuries conservatively with great success and one report showed no significant difference in outcome between cases of flail chest stabilized surgically versus those that were not.

Very little clinical work has been done regarding spinal trauma in veterinary medicine. This may be due to actual or perceived poor outcomes causing many owners to opt for euthanasia in this subset of patients. The most common region affected in dogs is the T3-L3 region (20-55%) with up to 45% of patients having concomitant injuries. Approximately 1/3 of spinal trauma patients are euthanized without treatment while 1/3 receive surgical care and 1/3 are managed conservatively. The necessity of surgical intervention is determined by: spinal instability, compression of the spinal cord, continued pain past 48-72 hours of medical treatment, and deterioration in neurologic status.

Traumatic long bone and pelvic fractures from motor vehicle accidents are one of the most commonly encountered problems in blunt trauma patients yet despite a large amount of research in this area, the optimal timing of definitive surgical stabilization is controversial. In veterinary medicine, no definitive guidelines with respect to fracture management in the traumatized patient have been written, leaving it to the surgeon to determine when the patient is sufficiently stable for a potentially long anesthetic procedure. Up to 59-72% of cats with long bone fractures will have concurrent injuries and that identification and management of these potentially life-threatening problems should be the initial focus. Emergency treatments that must be performed on open fractures include: sedation/aesthesia for patient comfort, removal of gross debris, clipping, flushing of wounds, culture procurement, administration of antibiotics, and coverage of the wounds with a sterile dressing. Unfortunately no specific guidelines as to when these fractures should be definitively repaired are in existence.

Ultimately, the application of DCS principles to veterinary patients is an area that has not been explored. While there may be a subset of patients that may benefit from a DCS approach, this is far from proven, and if attempted this limitation should be kept in mind. Whether DCS has been performed or not, the optimal time to operate a trauma patient that required aggressive hemodynamic stabilization is not known.
Oxygen and carbon dioxide are the two major gases present in the blood in times of health. Each molecule is transported by a unique method and the presence of type of molecule affects the transport of the other. Oxygen is transported in the blood in either a dissolved form or bound to hemoglobin. The amount of oxygen that is transported as dissolved gas is extremely small accounting for 0.3% of total blood oxygen, but it is this form of blood oxygen that is utilized to assess pulmonary function. Carbon dioxide can be transported in three different ways. Approximately 7-10% of CO2 is transported as gas dissolved in plasma. A further 20% is transported bound to hemoglobin as carbaminohemoglobin. The remainder is transported in plasma as bicarbonate after being converted from CO2 in the red blood cells.

Based on these principles blood gas samples may be utilized to evaluate patients for pulmonary failure (hypoxemia), ventilatory failure (hypoventilation) or perfusion derangements. Arterial blood samples are collected from peripheral arteries and are utilized to evaluate patients for hypoxemia or ventilatory failure. These samples must be collected and handled anaerobically and processed promptly since exposure to air can generate several artifacts. Venous blood gas samples can be used to evaluate acid/base status, electrolyte profiles and possible perfusion abnormalities.

Analysis of an arterial blood gas sample begins with the arterial CO2 content (PaCO2). This value is an indication of how well the body is able to move gas from the environment into the alveolus and does not reflect lung function. Normal PaCO2 is between 35-45 mmHg. Values below 35 mmHg indicate hyperventilation, which may be due to many causes including: pain, anxiety, metabolic acidosis or hypoxemia. Hypoventilation on the other hand is diagnosed when the PaCO2 is greater than 45 mmHg. Common causes of hypoventilation include: pleural space disease (pneumothorax, pleural effusion, etc.), thoracic wall disease (flail chest, constrictive bandage), primary neurological disease, drug administration (opioids, dexmedetomidine, etc.), upper airway obstruction (laryngeal paralysis, tracheal foreign body) or severe abdominal distension (GDV, ascites, obesity).

Arterial oxygen content (PaO2) on the other hand reflects how well oxygen is able to move into the blood from the alveolus. Normal PaO2 values range from 80-100 mmHg on room air at sea level. A value below 80 mmHg is defined as hypoxemia. There are 5 general causes of hypoxemia: low partial pressure of inspired oxygen (PiO2), hypoventilation, right to left pulmonary shunt, ventilation/perfusion mismatch (V/Q mismatch) and diffusion impairment. Decreased PiO2 is uncommon in veterinary medicine but may result from a malfunctioning anesthetic machine (no fresh gas flow or exhausted CO2 adsorbent) or from living at high altitude. Hypoventilation leads to hypoxemia by reducing the amount of oxygen in the alveolus and subsequently the arterial blood. Right to left shunting occurs when venous blood bypasses the gas exchange regions of the lung and enters the arterial circulation. This mechanism of hypoxemia is seen with structural cardiac diseases (atrial or ventricular septal defects, reverse patent ductus arteriosus), arteriovenous fistula, pulmonary hypertension or atelectic lung tissue. The most common cause of hypoxemia is V/Q mismatch. This is almost always caused by a specific pulmonary parenchymal pathology (pneumonia, pulmonary edema, PTE, hemorrhage).

Although similar V/Q mismatch is not the same as right to left shunting since blood is being exposed to gas exchange surfaces and a small amount of exchange is still occurring. For instance, a single pulmonary capillary may flow past several alveoli, some which are ventilated and others which are not. Diffusion impairment is an extremely rare cause of hypoxemia in veterinary patients but can be caused by any combination of the following three factors: defect in the tissue that inhibits gas diffusion (thickened membranes), decreased equilibration time (fast blood flow) or decreased surface area for diffusion (emphysema).

The A-a gradient is an assessment of pulmonary gas exchange that removes the effect of ventilation and is a useful way to evaluate ventilation and perfusion mismatch. Normal A-a gradient is approximately 5-15 mmHg when breathing room air. This difference is primarily due to normal small amounts of right to left shunting (pulmonary blood vessels, i.e. bronchial, mediastinal etc.) and gravity dependent irregularities of blood flow in the lung. The A/a gradient is determined by subtracting the partial pressure of arterial oxygen from the partial pressure of alveolar oxygen. Alveolar oxygen is determined using the following formula: \[ P_{A}O_2 = \text{Barometric pressure} - 50 \text{ mmHg} \times \text{FiO2} - (P_{A}CO_2/0.8) \] where barometric pressure at sea level = 760 mmHg and fraction of inspired oxygen (FiO2) ranges from 0.21 (room air) to 1.0 (100% oxygen). It is normal for the A-a gradient to increase as patients age. The A-a gradient can increase to approximately 150 mmHg when a patient is breathing 100% oxygen.

Since the A-a gradient becomes less reliable when oxygen is supplemented the PaO2/FiO2 ratio is used to quantify pulmonary function in this setting. This is possible because when FiO2 is greater than 0.50 the PaCO2 exerts a negligible effect on PaO2 and can therefore be ignored. Normal values for PaO2/FiO2 ratio are greater than 500. Values between 300-500 represent mild oxygenating inefficiency; values between 200-300 represent moderate inefficiency and values less than 200 represent severe venous admixture. A rough approximation of the PaO2/FiO2 ration can be made by using the “5 times rule” in which the PaO2 should be roughly 5 times the inspired oxygen percentage assuming normal ventilation.
The importance of effective pain management goes beyond the ethical consideration of ameliorating pain. The presence of pain causes increases in sympathetic tone and catecholamine secretion leading to increased cardiac output, increased peripheral vascular resistance and increased myocardial oxygen consumption. While important for the fight or flight response these changes, if chronic, further impair oxygen delivery to tissues. In addition to the cardiovascular effects of pain, a neuroendocrine response occurs resulting in elevated levels of catabolic hormones including cortisol while a concurrent decrease in the anabolic hormones insulin and testosterone is present. The net result of the neuroendocrine response is the development of a catabolic state characterized by hyperglycemia, type B hyperlactatemia and ketogenesis. Treatment of pain reduces or eliminates these responses, facilitates patient evaluation, improves patient quality of life and leads to more rapid healing and reduced morbidity.

Patients that suffer severe trauma or burns should be considered to be very painful and analgesia should be provided liberally. The best method of pain management in traumatized or burned patients utilizes a multimodal approach. Pure opioid agonists are the mainstay of pain management and should be provided early in the course of treatment. Opioid medications have no ceiling effect meaning that there is no maximum dose at which further administration would not be expected to affect a result. In most cases administration should be continued throughout the duration of hospitalization. While opioids are capable of depressing respiratory drive in veterinary patients this is rarely clinically relevant. If there is concern over a patient’s ventilatory drive then the dose of opioid can be reduced or broken into aliquots and administered to effect.

The dissociative agent ketamine has some analgesic properties, particularly for superficial pain. More importantly perhaps are the n-methyl D-aspartate (NMDA) antagonistic properties of ketamine that may alter spinal modulation and prevent or ameliorate central sensitization (wind-up). In order to derive the full benefit of ketamine in regards to sensitization prevention it should be administered for at least 24 hours as a constant rate infusion (5-15 mcg/kg/min). The use of ketamine alone is not effective for deep or visceral pain and can result in behavioral changes, therefore ketamine should almost always be administered in conjunction with an opioid. When discontinuing ketamine for patients that have been on a CRI for longer than 24 hours a gradual reduction of the infusion rate over several hours is used to prevent behavioral changes associated with abrupt discontinuation.

Clinically, the α2-agonists are most commonly used for sedation however their use for pain management is becoming more common. Their short half-lives and reversibility make them theoretically attractive options in many trauma patients but careful consideration should be taken prior to their use since clinically relevant cardiovascular side effects are possible. Animals in shock or with significant heart disease (especially diseases causing decreased systolic function) should not be administered α2-agonists. A constant rate infusion is often employed to most effectively use this class of drugs as a component of the pain management protocol. When used at rates of 0.5 to 2 mcg/kg/hr, dexmedetomidine can result in anxiolysis and analgesia without significant cardiovascular side effects.

Regional or local anesthesia should be considered as an adjunct to traditional, systemic analgesic methods. The use of regional or local anesthesia often requires coordination with the surgical team since these interventions often require general anesthesia or sedation to be performed. Placement of diffusion catheters for repeated administration of local anesthetic medication should be considered at the time of surgery if applicable; their use in contaminated wounds (bite wounds) should be avoided however. Epidural administration or placement of an epidural catheter can provide excellent pain management for animals with pelvic limb or pelvic ring trauma. If sacral fracture or sacro-iliac luxation is present or the landmarks used for epidural administration are disrupted then epidural administration should not be performed. Local anesthetic drugs can also be used systemically with IV infusion of lidocaine being useful as both an analgesic adjunct and a pro-motility agent for management of ileus.

Both blunt trauma and burn injury are capable of inducing a very pronounced inflammatory state. The use of non-steroidal anti-inflammatory drugs can be very beneficial in these patients however, animals that are severely traumatized or are in shock should not be administered NSAIDs until all tissue perfusion has normalized and is not anticipated to change abruptly. Patients with severe soft tissue injury or crush injury are at risk for development of myoglobinuria and renal failure. Any animal that has pigmenturia or evidence of renal insufficiency should not receive NSAIDs. The decision to use NSAID medications should be deliberate and if case selection is appropriate then NSAIDs may prove to be a very useful analgesic adjunct. Due to the risk of systemic side effects including further impairment of the already compromised immune system in these patients, animals that have suffered severe trauma or burn injury should not receive corticosteroid medications unless specifically indicated.

Many animals that suffer severe soft tissue injury require repeated sedation events on concurrent days or even multiple sedation events within the same 24-hour period. Patients that are going to require repeated sedation should be identified early in the course of hospitalization and measures should be taken to coordinate their care. Following repeated sedation with the same anesthetic medications it is common to see recovery times become more prolonged and patients require larger doses of drugs to achieve the same...
level of sedation due to the development of tolerance. It is good practice to alter the sedation protocol every few days to prevent development of tolerance or toxicity with repeated dosing such as reported with propofol administration in cats. Additionally, animals that are sedated serially are at risk for nutritional deficiency due to repeated fasting. Efforts should be made to sedate the animal at the same time every day to allow for the most opportunities to provide nutrition.
Historically, hemostasis has been divided into two categories: primary hemostasis, which is platelet mediated and secondary hemostasis that is mediated by soluble clotting factors. Normal hemostasis begins with damage to the endothelium leading to exposure of tissue factor and collagen. When this tissue injury occurs local vasoconstriction takes place decreasing blood flow to the damaged vessel. Platelets become activated and bind to subendothelial collagen and von Willebrand’s Factor (vWF). These platelets change shape and release granules to attract other platelets resulting in the formation of a temporary platelet plug. Concurrent with the formation of the platelet plug, soluble factors are activated primarily via the tissue factor pathway (extrinsic) with the ultimate formation of a fibrin meshwork that stabilizes the platelet plug and provides lasting hemostasis.

Testing the hemodynamic system in a bleeding patient begins with obtaining a comprehensive history. Specifically, any individual history or lineage history of bleeding that required interventions should be elucidated; this includes need for blood product transfusion, bandaging or surgery. A thorough physical exam should be performed with extra attention paid to perfusion parameters such as mucous membrane color, pulse quality, capillary refill time and respiratory rate. The site of bleeding should be evaluated to determine if the cause is not due to a hemostatic defect (i.e. loss of blood vessel integrity). Primary hemostatic disorders often result in development of petechia, ecchymoses or gastrointestinal bleeding whereas disorders of secondary hemostasis often lead to hematoma formation or cavitary bleeding (hemothorax, hemoabdomen, pericardial effusion).

Readily available methods to evaluate primary hemostasis include platelet count and buccal mucosal bleeding time (BMBT). Thrombocytopenia can be caused by decreased production, increased consumption or destruction. Automated platelet counts are generated by in house CBC analyzers however, agglutination (platelet clumping), poor sample quality, giant platelets and red or white blood cell fragments may all cause erroneous results. Manual platelet estimates can be performed by evaluating a direct blood smear on 100x magnification in the red cell monolayer. Each platelet observed at this magnification is equal to approximately 15 x 10^3/ul platelets. The number of platelets in several high-powered fields (HPF) is averaged and the total multiplied by 15 x 10^3/ul to obtain an estimate. When performing a manual platelet estimate it is imperative that the feathered edge be carefully evaluated for the presence of platelet clumps. Normal platelet counts range from 170 – 400 x 10^3/ul.

Buccal mucosal bleeding times are performed on patients with a known normal platelet count and coagulation profile. In theory BMBT evaluates platelet function and platelet interaction with subendothelial components such as vWF and is not affected by disorders of secondary hemostasis. Unfortunately the BMBT test is highly operator dependent, poorly reproducible and insensitive for detection of mild bleeding disorders. For the best results a commercially available spring loaded device is used for incising the oral mucosa of the upper lip. The patient is gently restrained and the lip is inverted to expose the buccal mucosa. The test device is placed flat against the mucosa and deployed resulting in 2 incisions of equal length and depth. Blood is gently blotted away using a circular filter paper taking care not to disturb the forming clot. The final time is measured from the time of device deployment until cessation of bleeding. Normal BMBT time is 2-4 minutes.

Secondary hemostasis is typically evaluated by measuring the prothrombin time (PT) and the activated partial thromboplastin time (aPTT). The prothrombin time evaluated the tissue factor (extrinsic) and common pathways (factors VII, X, V, prothrombin, fibrinogen) and is unaffected by platelets or intrinsic factors. The test is run on citrated blood that is exposed to thromboplastin, phospholipids and calcium. Since factor VII has the shortest half-life of the vitamin K dependent coagulation factors (II, VII, IX, X), PT is particularly useful as a screening test for rodenticide exposure and should be performed 2-3 days post exposure. Importantly, a normal PT does not definitively rule out factor deficiency since prolongation typically does not occur until greater than 75% of factor activity is lost.

Activated partial thromboplastin time evaluates the intrinsic and common pathways (XII, XI, IX, VIII, V, X, prothrombin, fibrinogen). The aPTT is also run on a citrated blood sample exposed to phospholipids and calcium. In combination with a normal PT the aPTT can be useful as a screening test for hemophilia A (VIII) or B (IX). Unfortunately, prolongation of the aPTT with normal PT also occurs with factor XII deficiency which does not lead to clinical bleeding and is relatively common in cats. Activated partial thromboplastin time is subject to the same limitation as PT in that 75% of factor activity must be lost before significant prolongation of the test time occurs.

Ultimately, definitive diagnosis of either an inherited or acquired factor deficiency must be made by specific factor assays in which individual factor activity levels are measured and compared to a standardized normal. Specific factor assays exist for most soluble coagulation factors and von Willebrand’s factor. Perhaps the most commonly assessed soluble factor is fibrinogen, which can be measured by several different desktop analyzers. Hyperfibrinoginemia can occur with inflammation, stress, infection or disseminated neoplasia. Hypofibrinoginemia may be congenital, acquired (hemodilution, consumption, DIC, sepsis), or due to decreased hepatic synthesis.
Fibrinolysis is more difficult to evaluate directly without performing viscoelastic testing which has limited availability and is therefore not very practical. Indirect tests of fibrinolysis include measurement of fibrin degradation products (FDP) and D-dimers. Fibrin degradation products are produced when fibrinogen or fibrin is cleaved by plasmin. Elevation of FDPs may be an indication that the fibrinolytic system is active although elevated FDP may occur in conditions with hyperfibrinoginemia. D-dimers are a unique type of FDP that is specific for mature clot breakdown. Elevation of D-dimers is therefore indicative of degradation of a mature clot.
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 hypoproteinemnic 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 a 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/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 \times \text{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 fluoroquinolone 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, hemoabdomen 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%.
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 animal, E\textsubscript{r}CO\textsubscript{2} should not be used as the sole confirmation of endotracheal intubation in CPA animals. Since E\textsubscript{r}CO\textsubscript{2} 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\textsubscript{r}CO\textsubscript{2} levels can predict the likelihood of success with E\textsubscript{r}CO\textsubscript{2} level of less than 15 mmHg and 20 mmHg suggesting worse prognosis in dogs and cats respectively. Once ROSC occurs E\textsubscript{r}CO\textsubscript{2} will undergo a rapid and sustained rise as the heart more efficiently delivers CO\textsubscript{2} 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.
Extraction Pearls: 
How I Try and Make Extractions Easier 
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Extraction decisions
Sometimes it is easy to decide when to keep a tooth and when to extract, but at others time, the choice is not as obvious. Of the three criteria to evaluate, examining the tooth in question is the first step. If periodontal attachment loss is greater than 50% or the pulp is compromised or there is extensive tooth resorption, then it is typically best to remove. If periodontal disease is moderate, then you consider the relative importance of the tooth and if the disease around it can impact a more strategic tooth. For instance, if either the fourth premolar or second molar adjacent to the large mandibular first molar can compromise the health of that important tooth, it may benefit the patient to extract the smaller tooth, thus giving better access to treat the adjacent surface of the first molar. The same would apply to the mandibular third incisor or even the maxillary third premolar. If the decision is still up in the air, the health of the patient is to be considered: any patient with an ongoing systemic issue (heart murmur, diabetes, renal disease) would likely benefit more from an extraction that will remove the source of infection in one visit, as compared to extended anesthetic times and more frequent procedures. And third, consider the owner: if an advanced periodontal procedure or root canal is to be done, are they willing to consider the additional expense, and be committed to thorough home care and regular re-treatments? If not, then again, extraction may be optimal.

Requirements

Equipment
- Periosteal elevator – Molt #2 and Molt #4 – for elevating flaps
- Means of sectioning –
  - High speed handpiece/unit is preferable, but sectioning teeth can be done with a lowspeed unit, just have someone dripping water on the site for cooling
  - Set up regular maintenance schedule, including daily oiling
- Sectioning burs – replace regularly, they get dull quickly
  - 700L – dog teeth
  - 699 – cat teeth
  - #2 or #4 – round burs for alveoloplasty
- Dental elevators
  - Winged, not too thick – to fit in the PDL space
  - SHARPEN on a regular basis, even during the procedure
  - If used and sharpened regularly, they will wear down and will need to be replaced
- Dental luxators – thinner, more delicate – be careful not to bend
- Extraction forceps – small breed
- Blade – 15C
- Suture – 4-0 to 5-0 poliglecaprone
  - Reverse cutting for dogs
  - Tapered for cats
- Magnification – better posture

Pain management
Apply general principles of surgical pain management to every dental patient, even if not performing extractions. Pre-operative analgesia with opioids, alpha 2 agents, and NSAIDs when appropriate, with post-operative dispensing of NSAIDS, opioids. Peri-operative regional, local and splash blocks can minimize the amount of general anesthesia used, help keep the patient more stable, and provide better post-operative analgesia for a smoother recovery. While lidocaine and bupivacaine can be mixed, if surgery sites are identified early in the procedure, use bupivacaine alone if it can be administered 20 minutes prior to extraction. Bupivacaine with 1:200,000 epinephrine premixed will provide longer analgesic effect and reduce bleeding. Watch total dose, not to exceed 1 mg/kg for cats and 2mg/kg for dogs.

Regional blocks can be very effective when placed accurately and not causing damage. Adequate training should proceed any attempts on patients, as nerve damage can result. If a regional block cannot be placed (infraorbital on brachycephalic, inflamed purulent tissue, etc), then at least place a linear local block in the alveolar mucosa above/below the tooth, and you can place additional material directly at the site when open (splash block). Caudal maxillary blocks method.
Radiographs
Extractions are one of the major reasons to use intraoral radiographs, particularly when challenging procedures are encountered. Preoperative radiographs should be closely evaluated to determine the presence and condition of the periodontal ligament (PDL), as this is the structure that elevation attempts to impact. If there is no periodontal ligament space, indication of ankylosis or even tooth/root resorption, then elevation will not go as planned. Radiographs will also alert you to abnormal root structure (or number), and if there is any compromise to the jaw strength. Radiographs will not always be decisive in evaluation teeth with compromised pulps, so use transillumination and examination to assess those teeth. Post operative radiographs are a good medical and legal record, to show the correct tooth was extracted completely, without any complications (root tip, fractured jaw).

Steps of extractions
Flaps
With few exceptions (very loose incisors, premolars where envelope flaps are sufficient), most extraction sites benefit from full thickness mucoperiosteal flaps with releasing incision(s).

- Flap design – broad base, not directly over bone defect if possible
  - Extend releasing incision just past mucogingival junction, into alveolar mucosa
  - Maxillary canine – two releasing incision
  - Maxillary fourth premolar – one releasing incision mesially (rostral)
  - Mandibular canine – T- or Y- shaped distal incision, mesial incision
    - Follow the ‘path’ of the root – angled lingually
    - Elevate buccal flap completely
    - Elevate lingually to expose distal aspect of root
  - Maxillary first molar – if extracted on its own, a flap will not be reasonable
- Flap elevation and release
  - Debride gingival margin before elevating – cut 1-2mm away
  - Periosteal elevation to lift full thickness flap off of bone – past MGJ
    - Only elevate as far as you need for adequate access
  - Use blade or iris scissors to snip the fibers of the periosteum on the under side of the flap

Alveoloplasty/sectioning
- Maxillary Canine
  - Make a groove at mesial and distal aspects of the root – place for elevator – to the widest part of the root, then connect across
- Mand Canine
  - Remove bone from buccal, distal and lingual surfaces, as well as a groove at the buccal-mesial aspect
- Multi-rooted teeth
  - Shave away buccal bone until furcation is visualized
  - Using crosscut fissure bur – section from furcation through the crown
    - Max fourth premolar – one cut from furcation into developmental groove; second cut from furcation mesially to remove ‘diamond’ shaped piece of crown
      - Access to furcation between two mesial roots now visible, section those two apart
      - Mand first molar – section from furcation to just past mesial crown, but not at too much of an angle
      - Max molars – section palatal root away from two buccal roots, then separate the two buccal roots

Elevation – the goal is to fatigue the periodontal ligament to the extent that the tooth can be elevated from the socket
- Advancing the sharpened tip of the dental elevator down the root, in the periodontal ligament space, with rotational hold, is the best force to use
- Elevating in between crown portions with the fulcrum of force below the alveolar ridge – teeth may break
- Elevate tooth/section against adjacent tooth – make sure that tooth is very stable
- Gently grasping the tooth/segment with the extraction forceps and putting rotational force can help fatigue the ligament and/or tell you where you need further elevation
- If there is no movement and Radiographically the PDL was healthy, remove more buccal or interseptal bone.
  - In the maxilla, additional buccal bone removal is reasonable (window washer movement of the bur on the bone surface)
  - In the mandible, particularly of small dogs, preserve as much buccal bone as possible (cortical bone)
    - To access adjacent roots, remove one first, then remove the cancellous bone that was in between the roots to get better access for elevation without having to remove buccal bone
- Once fully elevated, radiograph to confirm
Finishing

- Elevate the lingual/palatal mucosa once the tooth is gone for better exposure for alveoloplasty and to facilitate suturing
- Smooth any rough edges of the alveolar bone (alveoloplasty)
- Curette any debris or infected tissue from the alveoli
  - Determine if any bone graft material is needed
  - Small breed dog – mandibular canines and first molars, incisor?
- Osseconductive or promotive?
- Scarify any epithelial edges
- Simple interrupted, bite through palatal, lingual mucosa first, then buccal flap

Complications

One of the most important resources in performing extractions is a load of patience. As soon as you lose focus or are distracted, that’s when you hear the ‘crack’. If that sound is a root tip breaking off, go through these steps to manage the situation:

- On radiographs – was the PDL intact and healthy
  - Elevation should continue – more bone may have to be removed
    - Buccal bone removal at maxillary teeth – ‘shave’ the cortical bone away to expose the root further
    - Mandibular teeth – try to preserve buccal bone, but remove the cancellous bone that was in between the teeth for better access
    - Palatal root – dig a trench around the root and make sure there are no overhangs
  - If there is any periapical bone loss (and the pulp is dead or infected), the root HAS to come out
    - Avoid aggressive elevation toward the apex – the root could punch through into the nasal cavity or mandibular canal
    - Work the root tip from side to side – use a root tip pick
- If the root tip goes into the nasal cavity or mandibular canal, every effort should be made to remove it THEN! – this is your best chance to remove it while it is still loose and not encased in scar or fibrous tissue
  - Take radiographs at several angles to localize where the tip is
  - Open the hole it pushed through even more (watch for important vessels)
  - If you can gently grasp it without damaging other structures, attempt to do so – but it will usually move further away
  - Once the hole is wider than the root tip without overhangs, uses copious water to flush the area, and adjust the head to allow ventral drainage
  - Many times you won’t even see the tip flush out – so re-radiograph often.

If you hear the big ‘crack’ – the jaw breaking – hopefully you had pre-operative radiographs and have told the owner that the jaw could be fragile. If this is a pathological fracture due to extensive periodontal disease, it will be a difficult area to stabilize, as the affected teeth usually have to be extracted anyway. Sometimes a partial rostral mandibulectomy is the best option for the patient.

Tooth resorptions

The term Tooth Resorption (TR) is now used to describe any level of root and/or crown erosion or loss due to a variety of processes. While this is most commonly seen in cats, dogs can also exhibit signs of TR. The ‘typical’ tooth resorptive lesions that are diagnosed are those in cats, frequently in the premolars (mandibular third premolar) where radiographically it appears as if the root is being turned into bone. This odontoclastic lesion is a Type 2 TR, and should be distinguished from the less common Type 1 inflammatory lesion. The inflammatory lesions may appear similar to odontoclastic lesions in the physical appearance of the crowns (some crown loss with gingival tissue growing into the defect), but radiographs will show roots with intact periodontal ligament space(s) and intact roots, other than where the resorption is taking place. If this type is diagnosed, careful extraction of the entire root(s) is necessary.

If the radiograph shows root structure that is not distinct, with no clear periodontal ligament (PDL) space (as the root is being converted into bone, the PDL space is obliterated), and if there is no indication of apical bone loss or infection, then a modified extraction technique may be appropriate. While some of these roots can still be gently elevated, if the PDL is damaged, elevation will not be able to fatigue the ligament for extraction. If this is the case, after radiographic evaluation and initial attempts at elevation result in the crown breaking off, the modified technique may be done: remove the remainder of the crown and coronal aspects of the root (if possible), and smooth the alveolar bone before suturing the gingival closed. These areas should be radiographed post-operatively, the client should be informed that there was intentional root retention of the resorbing roots, and that the patient should be monitored for any persistent inflammation in the area.
Post-operative
Most patients benefit from appropriate pain medications, and some may require antibiotics after the oral surgery. Depending on the extent of surgery, a softened diet may be needed, and in rare instances, supplemental feeding may be needed. Active tooth brushing may be delayed for two weeks, until the oral recheck, but oral rinses and gels may be used immediately post-operatively to help with tissue healing and antimicrobial needs.

Summary
With the right equipment, training and patience, extractions in practices can be successful surgical procedures with minimal complications. Often these patients will clinically be much healthier once the infection in their oral cavities have been managed with extractions.
Feline Dental Support Group:  
How Many Ways Can Cats Get Rid of Teeth  
Heidi Lobprise, DVM, DAVDC  
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For any practitioner who treats cats, it seems like those creatures are experts at trying to get rid of their own teeth! Two of the most common dental dilemmas, tooth resorption and stomatitis, continue to frustrate general practitioners and specialists alike.

“Decades ago, in a galaxy very close, it was once believed that the world of veterinary dentistry would be able to fully comprehend, understand the causes and find the cures to all oral and dental ailments that befall our feline companions. It is now 2016, and while we continue to explore the many facets of unique oral dilemmas our feline patients may encounter, many questions are still not fully answered; though we have managed to rename many terms!”

Stomatitis
Aka – Lymphocytic-plasmacytic stomatitis (LPS), feline chronic gingivostomatitis (FCGS) and Feline Gingivostomatitis, this syndrome may have many names, but there is a base of definitions to be found on the website of the American Veterinary Dental College. Officially - inflammation of the mucous lining of any of the structures in the mouth; in clinical use the term should be reserved to describe wide-spread oral inflammation (beyond gingivitis and periodontitis) that may also extend into submucosal tissues (e.g., marked caudal mucositis extending into submucosal tissues may be termed caudal stomatitis).

Inflammation limited to the gingiva (gingivitis) may be the only indication of early periodontitis, but when inflammation extends past the mucogingival line into the alveolar mucosa and even into the vestibular space, this alveolar mucositis generally signifies a more advanced process. While inflammation in the oral cavity can involve any group of tissues, from buccal mucositis to glossitis and palatitis, one of the most important regions to inspect is the mucosa of the caudal oral cavity. This region, bordered medially by the palatoglossal folds and the fauces, dorsally by the hard and soft palate and rostrally by alveolar and buccal mucosa, is often the site of significant to extreme ulceration and proliferation of tissues.

In a consensus statement from the AVDC and the European Veterinary Dental College, stomatitis can be divided into two types: Type 1 – cases with alveolar and labial/buccal mucositis/stomatitis only, or Type 2 – cases with caudal mucositis/stomatitis, with or without alveolar and labial/buccal mucositis/stomatitis. Identification of caudal mouth involvement is important, as these are the case that are generally most difficult to manage.

Distinguishing from periodontal disease
While early cases of stomatitis may resemble inflammatory periodontal disease, and stomatitis may be a different in a case of severe periodontal disease, the refractory or non-responsive nature of the syndrome also plays an important role in the diagnosis and determining the prognosis. In early management of these cases, a thorough effort should be made to provide optimal oral conditions to monitor the patient’s response to treatment.

Phase I treatment includes a complete dental assessment, including radiographs, as well as thorough cleaning (scaling), root planing and polishing. If any teeth are identified with attachment loss (gingival or bone) or tooth resorption, complete extraction with appropriate alveoloplasty and gingival closure should be performed. Attention should also be paid to other antigenic stimulation, including discussing hypoallergenic diets, appropriate food and water dishes and removal of any environmental contaminants. Antibiotics may be used judiciously to maximize the patient’s response, but are not to be used as a stand-alone therapy. While corticosteroids have been used extensively in the past for patient comfort, they not only can make a non-responder, but can have unwanted side effects. As practitioners become more comfortable with using other anti-inflammatory products, early use could help these patients, particularly in early cases.

At the time of recheck, in 7 to 10 days, a patient with (fairly) simple gingivitis or periodontitis show be showing a response to treatment with appropriate healing of extraction sites and decrease in the level of inflammation throughout the mouth. It is very important for these patients to receive whatever level of home care the owner is capable of providing, and regular, routine professional care should be administered to monitor the case closely.

Refractory – Chronic – Disease
While some descriptions may just use the presence or absence of caudal mouth inflammation to categorize cases of stomatitis, others recognize that earlier cases may not yet have caudal involvement. The aspect of these Type 1 patients is to recognize the lack of response to conservative or Phase I therapy. Persistent inflammation with labial/buccal mucositis/stomatitis will often progress to include the caudal oral cavity, when the term stomatitis is generally accepted. There is wide variation to the range of this syndrome, from patient to patient, and even different time frames for the same patient.

Once identified, it is important to be able to somewhat quantify the level of disease, both for initial diagnosis, and to evaluate any response to treatment, whether clinical or in therapy evaluations/trials. The Stomatitis Disease Activity Index (SDAI) evaluates four aspects of owner observation, including appetite, activity level, grooming behavior and perceived comfort (or lack thereof). The
veterinarian then scores specific areas on a 0 to 3 basis for the level and extent of inflammation. These scores are then computed to assign a number to the degree of disease. The patient’s dental chart should be thorough, indicating the level of plaque and calculus, the degree and extent of inflammation, especially caudal mouth, any missing teeth or retained roots. Photos should be taken of all regions described, particularly the caudal mouth.

If, after the Phase I therapy, no response is seen within 7-10 days, the client should be counseled that early caudal mouth extractions (CME) will provide the best level of relief for their pet. In one study, patients with CME needed no further treatment, and an additional 37% only needed low levels of inflammatory support. No significant difference was seen between cases of CME or FME (full mouth extraction). (Jennings 2015)

**Extractions**

Full details of extractions will not be provided here, but some salient points are offered. Depending on the practitioner’s level of skill and equipment, CME can be scheduled as one or two surgical procedures. Multi-modal analgesia should be provided from the first, including appropriate opioids, regional and local blocks and post-operative medications. Unless complicated, in this author’s experience, removal of the teeth provides significant relief for the patient and with reasonable levels of surgical pain management, the patients return to function and eating quickly.

Working quadrant by quadrant, full elevation of buccal gingiva and mucosa, past the friable edges, allows visualization of the teeth, furcations and alveolar bone. A small 699 crosscut fissure bur is ideal for sectioning feline teeth, and initial removal of alveolar bone. Careful elevation, starting with a thin, flat elevator or luxator and then using small winged (sharpended) elevators can help loosen the tooth segments. Gentle elevation with small breed extraction forceps should remove the entire root. The lingual and palatal mucosa are then elevated away from the alveolar ridge. It is important to remove all rough bone edges, debriding down to healthy bone in a fairly aggressive alveoloplasty. The alveoli should also be debrided. Closure with 5-0 monofilament should be done with no tension. While a continuous pattern will leave least number of knots, any knot failure would be a detriment. Simple interrupted or an interrupted cruciate pattern can be used to close the extraction sites.

**Other therapy options**

There is a very long list of medications that have been used in attempt to manage feline stomatitis, with great variability in response. In a European study with FCV (Feline Calicivirus) positive cats, non-responders to FME, transmucosal administration provided comfort and reduced inflammation in some of the patients. (Hennet 2011) Subcutaneous and intraslesional administration has also been described. Mesenchymal stem cell therapy has been initially investigated in a number of non-responsive patients with 5 of 7 having complete resolution. (Arzi 2016) Long term NSAID use in cats has been studied with other diseases, so its use in stomatitis may be able to play an adjunctive role.

Corticosteroids are used frequently in general practice to provide some level of relief from inflammation for patients, though often with a gradual decrease in effectiveness. Cyclosporine has also been studied to look at its effect on this syndrome that demonstrates an inappropriate immune response, to effect in some patients. (Lommer 2013) There is no general consensus of the impact of laser therapy, but removal of significant proliferative tissue can be beneficial to some patients, when used with other treatment.

**Summary**

A key point is to realize that these cases can be quite frustrating to everyone involved, and particularly the patient. Efforts should be made to find the best combination of care that will make individual as comfortable as possible, which often entails extensive extractions. While generally thought to be more prevalent in cats starting around 7 years of age, a trend seems to be including younger patients in the group that require early extractions.

**Tooth resorption (TR)**

In the name game race, this group of lesions beats out ‘stomatitis’! From feline caries to neck lesions to cervical line lesions and Feline Odontoclastic Resorptive Lesions (FORLs), the identification and understanding of these pathological changes has changed over the years. Our management approach has also changed – no more glass ionomer kits for repair!

The AVDC has again provided guidance for the classification and definitions of these lesions, basing the TR type on their radiographic appearance. Knowing that these are progressive lesions, the staging looks at the levels of severity, from which treatment decisions can be made. Generally speaking, the broader terms of tooth resorption (TR) can be applied to any type of resorption – external or internal, replacement or inflammatory, in dogs or cats, or any species.

**Tooth resorption types**

TR Type 1 is identified radiographically if a focal or multifocal radiolucency is present in the tooth. Other portions of the tooth will be otherwise of normal radiopacity, and a normal-appearing periodontal ligament (PDL) will be identified. This type of resorption typically occurs in response to an inflammatory process such as periodontal disease. This inflammatory resorption can be seen particularly when the periodontitis has caused the loss of gingival tissue and bone, exposing the neck of the tooth and the root. The exposed hard tissue now is susceptible to demineralization and erosion by the surrounding inflammatory processes. The remaining root structure that is still protected by alveolar bone will generally have a healthy and visible PDL space.
TR Type 2 is identified by the narrowing or disappearance of the PDL in at least some areas, often starting at the apical area and progressing coronally. There will be decreased opacity of part of the tooth, as the odontoclastic process continues. This replacement type of resorption takes place as odontoclasts attach to the lacunar surface of intact dental tissue, typically protected by connective tissue. Once the surface cementum and underlying dentin are resorbed, reparative bone-like or cementum-like tissue covers the evacuated spaces. The PDL is destroyed as bone cells basically try to convert the root into bone.

Often these lesions go unnoticed until they progress up the root into the crown, where defects are then filled in with inflamed gingiva or granulation tissue at the neck of the tooth. If left un-addressed, the lesion will progress until structural loss of the crown is eventually covered with healing gingiva.

TR Type 3 description covers those teeth that have features of both Type 1 and Type 2. There will be areas of normal and abnormal (narrow or lost) PDL space. Focal or multifocal inflammatory radiolucent areas may be distinct from decreased opacity in other regions of the tooth, including the roots.

**TR stage**

While this is a progressive disease, the rate of progression can be highly variable among patients, and even among different teeth in the same patient. Personal clinical observations have sometimes noted active progressive disease in young cats with multiple lesions as compared to older patients with limited lesions and a slower progression.

TR Stage 1 – or mild dental hard tissue loss (cementum or cementum and enamel) would be a very challenging level to identify clinically. While it certainly occurs, it is often not appreciated until progressing to TR Stage 2. This second stage will exhibit moderate dental hard tissue loss, to included loss of dentin that does not extend into the pulp cavity. Radiographically this may be assigned to a tooth exhibiting loss of PDL space and initial root conversion into bone.

Once the pulp cavity is involved, a TR Stage 3 classification should be applied. While most of the tooth will retain its structural integrity, the extent of the lesion with deep dental hard tissue loss will necessitate its extraction. Further progression with extensive hard tissue loss, or TR Stage 4, will include teeth that have lost its integrity and structure, but with some crown or root tissue still remaining. Subcategories include TR4a (crown and root equally affected) TR4b (crown more severely affected) and TR4c (root more severely affected). The final stage – TR5 – there are only remnants of dental hard tissue, visible only as irregular radiopacities. The gingival covering signifies complete healing, and is a sign that the body has completed its effort to get rid of the tooth.

**Management**

One aspect of handling TR lesions is early identification. In the exam room, an effort should be made to visualize the mandibular third premolar, typically the first tooth affected by resorption. If even one indication (loss of tooth structure, ingress of gingival or granulation tissue, complete loss of the crown) is present, the entire dentition needs to be radiographed. These patients exhibit pain when the teeth are palpated (under anesthesia), and extractions are typically needed.

Intraoral radiographs are absolutely essential in managing tooth resorption lesions. Close evaluation of the periodontal ligament space is necessary in order to determine the type of resorption – inflammatory (Type 1) or odontoclastic (Type 2). No matter what the extent of resorption, if the PDL space is intact and visible radiographically, the entire root/tooth must be elevated. This can be challenging if the resorption has damaged the structure of the tooth, making it fragile and more prone to fracture during extraction. All apical portions must be completely elevated, as bacteria may have entered the pulp cavity during resorption/inflammation. Complete extraction is even more critical in cats with concurrent stomatitis.

On the other hand, if radiographs show Type 2 odontoclastic or replacement resorption with loss of the PDL and conversion of the root into bone, a modified approach may be taken. Routine steps of regional/local block, an envelope flap, sectioning the tooth and starting gentle elevation should be started. Occasionally an involved root will actually elevate completely out of the socket. Typically, though, there will be little indication of fatiguing of the PDL, and the crown section will often snap off. As long as there is no visible PDL and no evidence of apical bone loss, it is generally acceptable to finish the extract by smooth any rough alveolar edges, elevating the gingival margins, and suturing the site closed (cruciate). It should be noted on the record, and the owner informed, that a modified extraction technique was used (MET), with intentional root retention (IRR) and that you will continue to monitor (CTM) the site. Any remaining root should undergo continuing resorption, and complications are very unlikely, but radiographs are essential to determine the MET is the appropriate therapy for that tooth.

One slight difference might be encountered with Type 2 resorption of canine teeth. Sometimes they are slower to progress (not always), and may be preserved with extensive root involvement as long as the crown is stable and pain-free. Doing the initial elevation will seldom result in the ‘snapping’ off of the crown, so an actual ‘crown amputation’ may be necessary, removing just enough structure to be able to close the gingiva at the site. Pulverization of roots is not recommended.

**Summary**

While tooth resorptions may have many different presentations, the odontoclastic feline version by far is the most common type encountered. Intraoral radiographs are an absolute necessity when managing these lesions for the best care for our patients.
References
Johnston NW. An updated approach to chronic feline gingivitis stomatitis syndrome. Veterinary Practice 2012 Vol. 44, 5; pp34 -38
Intraoral Radiographs:
Taking and Reading Tips
Heidi Lobprise, DVM, DAVDC
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There are many ways to teach and take dental radiographs; the author’s preference is to have the patient in lateral recumbency and slightly adjust the head position using towels, depending on the image needed. Others prefer dorsal and ventral recumbency for taking radiographs - determine what works best for you and your staff.

Parallel

While a parallel technique (film and object parallel with x-ray beam perpendicular) would be ideal to minimize distortion, most areas of the oral cavity do not lend themselves easily to this positioning. The only region where the film can be placed parallel to the teeth is that of the mandibular premolars and molars, with a corner of the film pressing into the intermandibular space. The most mesial (rostral) roots and teeth may not be visible on this view, as the film may be limited by the mandibular symphysis, but aiming the radiographic beam from a slightly rostral oblique position may allow these roots to be imaged.

Bisecting angle technique

For the rest of the teeth in the oral cavity, a parallel positioning is not possible, so, a film is placed as close to a parallel plane to the object (root or tooth) as possible. Remember to place the film so the roots will be imaged, not necessarily the crown. One option is to use a bisecting angle technique for these films by aiming the beam at a line that bisects the angle formed by the long axis of the object (tooth) and the film.

Modified technique

Another way of determining beam position is to first line up the beam (or similar object such as a 2-inch roll of tape) perpendicular to the film. This would result in an image that is too short (shadow of a tree at noon). Next, line up the beam perpendicular to the root (tooth); this image would be too long (shadow of a tree at daybreak). Then, split the difference between these two positions, and the resulting image will be approximately the same size as the object, thus minimizing the distortion (and the beam will be perpendicular to that bisecting line mentioned earlier). Helpful devices, such as connecting two tongue depressors with a pushpin, and using a roll of tape to visualize where the beam will travel, can help you determine the two positions (perpendicular to film; perpendicular to tooth), so you can aim the beam halfway between the two. This perspective will also help you make appropriate adjustments to an image; if you want to make the image shorter, move the beam to a position more perpendicular to the film.

Challenging radiographs – the cat quick 6-7

- With the cat in lateral recumbency (e.g. – left side down), take the first image of the mandibular premolars and molar with a parallel technique.
  - If the mesial (rostral) root of the mandibular third premolar does not show, adjust the x-ray head further ventral and forward
- Take an image of the lower canines and incisors: roll the tongue back into the pharyngeal area to keep the sensor in place better; use the modified technique
- Take an image of the upper canine and incisors with the sensor ‘wide’ across the palate
  - If you need to isolate the right canine tooth apex better, come slightly off midline
- Take an image of the maxillary premolars
  - Place the sensor up against the palate
  - Using a tape roll, visualize where the beam would be, if aimed directly perpendicular to the teeth: you will not be coming directly laterally to the maxilla, but slightly from in front
  - Then visualize where the beam would be perpendicular to the film
  - Split the difference
  - The zygomatic arch will always be in the way – if you elongate the image by moving the x-ray beam more perpendicular to the teeth, the arch ‘moves’ a little more out of the way.
- Using a clear feline mouth gag (cut part of a tuberculin syringe); place the sensor under the head on the left side (extraoral); the left maxillary premolars will be placed nearly flat on the sensor in this position.
  - Using the tape roll, and angled from the back of the head, look across the arch at an oblique/angle, until you see the palatal surfaces of the left maxillary premolars without the right premolars superimposed over them
• Make sure the sensor is placed far enough forward and dorsal that the angled beam will go through the teeth and hit the plate.

• 5 of the 6 films are done!
  o Adjust the cat to left lateral recumbency and take the left mandibular premolars

**Challenging dog radiographs**

• Maxillary incisors – in most dogs with a normal head shape, then ventral portion of the nares will be lined up with the base of the xray cone when positioned
  
• Maxillary canine apex – palpate where the apex is positioned by running your finger up the buccal jugae to the tip (it is usually somewhere over the second premolar
  o Place the sensor centered at the maxillary second premolar
  o Adjust the xray beam from midline to a slight oblique so the canine is not superimposed over the premolars in the image; make sure it is centered on the spot where you palpated the canine apex

• Maxillary molars – with a skull or model, observe how the molars are in a different ‘line’ than the premolars
  o Place the sensor in the mouth lined up with the two molars (usually angled in a palatal direction)
  o Aim the beam almost directly onto the sensor (just a slight adjustment)

• Mandibular canines
  o If you place the sensor across both lower second premolars and aim the beam perpendicular to the sensor, you will have both canine apices for good comparison

• Mandibular premolars
  o Since the symphysis restricts the sensor from going far enough forward to get a true parallel image of the first and second premolars, adjust the beam to come from in front of and below the teeth to ‘push’ them onto the image (or take it extraorally)

• Brachcephalic dogs
  o Use extraoral shots as is done for cats

**Troubleshooting radiographs**

• Teeth are too long, or the apex is not on the film
  o Place the sensor deeper into the palate – you want to see the roots, not the crown
  o Adjust the beam to be more perpendicular to the film – ‘shortens’ the teeth

• Teeth are too short
  o Adjust the beam to be more perpendicular to the tooth – ‘enlongates’ the teeth

• Image shows unexpected bone loss (and crowns are burnt out)
  o Decrease time of exposure; if at lowest time, move xray cone an inch or two away from object

**Identification of teeth or region**

Most digital intraoral radiography software systems have precise ways of taking images to correspond with the appropriate teeth. While this is very helpful in record keeping, if anesthetic time needs to minimized, or if images are unlabeled or mislabeled, it is important to be able to identify a tooth or structure in any image taken.

With actual digital films, part of this identification process deals with how the film is placed in the mouth. A dot is embossed on the film (through the packet), so the raised dot faces the xray beam source. In reviewing hard films, placing the film so the raised dot is facing you orients the image in the same way as digital films are viewed, as if you are looking onto the outward surfaces of the patient. Having models or skulls are helpful guides when starting out, until you become familiar with structures, including the differences between maxillary and mandibular images.

With either digital or actual films, there are a few quick steps to take to be able to identify what teeth are being viewed:

• First, orient or rotate the film/image until the roots are pointing in the appropriate direction
  o Maxillary roots pointing up
  o Mandibular roots pointing down

• If the teeth imaged are incisors or canines – “Shake hands”
  o The patient’s right is on your left, and vice-versa

• If the teeth images are premolars and molars -
  o Ask – “which way is the nose?”

• If the nose is to the right – it is the right side, and vice-versa
  o It is VERY important to only rotate the image digitally – NEVER “FLIP”
  o Flipping the image – horizontally or vertically – reverses right and left
However, for images taken with the sensor or film placed extraorally:
  o Then right and left are reversed
  o This should be noted on the film/image that it was taken extraorally

Know normal
By reading lots of films/images, you will become more familiar with normal structures of the oral cavity. Superimposition of the nasal cavity, the mandibular canal, foramina and osseous structures such as the zygomatic arch can complicate evaluation of the films. An apex of a tooth superimposed over a less dense structure, such as the nasal cavity or mandibular canal, may give the impression of a wider periodontal ligament space, or even bone loss. This chevron effect should be verified by imaging the tooth on the opposite side, or taking multiple views at different angles. Further evaluation for tooth vitality, such as transillumination, can provide additional input. Imaging both sides can also help identify lucencies that may appear as lesions that are actually mental foramina. Adjusting technique and angles to ‘move’ the zygomatic arch away from maxillary premolars can allow you to visualize certain tooth portions better.

Evaluation of periodontal bone
In the evaluation of periodontal disease, it is important to be able to assess the extent of periodontal bone loss, as well as the type of bone loss. This information, along with probing depth and visual assessment, will give a complete picture of the staging of the disease for that tooth or region, and will guide treatment decisions. Each tooth in a patient’s oral cavity can have a different bone loss pattern, and the pattern can differ from root to root of the same tooth.

  o Stage of disease – with each subsequent stage of disease, there is an increase in the percentage of attachment loss, which included bone
    o Stage 1 – no attachment loss
    o Stage 2 – up to 25% attachment loss
    o Stage 3 – 26 to 50% attachment loss
    o Stage 4 – greater than 50% attachment loss

  o Type of bone loss
    o Crestal bone loss – initial loss of the rounded alveolar crest in between teeth
      ▪ There is typically little periodontal pocket formation
    o Horizontal bone loss – bone loss proceeds in a linear fashion across a tooth or several teeth
      ▪ If accompanied by gingival recession, roots can be exposed, and even the furcations of multi-rooted teeth, with variable extents of soft tissue pockets that will be suprabony
      ▪ If there is no gingival recession, the horizontal bone loss will result in the formation of soft tissue or suprabony pockets
    o Vertical bone loss – bone loss extends down the length of a root or roots
      ▪ This will form an infrabony pocket that can be challenging to access without gingival flaps or surgery
      ▪ If the vertical bone loss extends to the apex of a root, the infection will enter the root canal system at that point and infect the pulp, eventually killing the pulp
        ▪ This may lead to endodontic or apical bone loss of additional roots of a multirooted tooth

Endodontic disease evaluation
There are several ways to assess the health of the endodontic system: if the pulp is exposed by fracture, resorption or caries, treatment (extraction or root canal) must be performed, even in the absence of radiographs signs or lack of transillumination. Discolored teeth should likely be considered to be non-vital, though transillumination may help in the evaluation. The absence of radiographic signs does not mean the tooth is vital, as osseous changes may be very subtle, may take extended periods of time to occur, or may be missed. When present, however, radiographic signs are confirmation of pulpal compromise and can also be used to determine the best course of therapy.

  o Apical bone changes – apical periodontitis
    o If the periodontal ligament at the apex is wide, this may be an early indication that infection or compromise is present
    o The typical ‘mushroom’ area of bone loss – often termed an apical abscess – won’t be found in every case, and in theory, cannot be termed an abscess unless histopathology or culture is done. Some lesions could be sterile granulomas
    o Chronic lesions may also show resorption of the root itself
- Significant changes would decrease the likelihood that an endodontic treatment would be successful, so extraction may be needed.

- Canal width – normal aging changes includes a narrowing of the pulp canal as the dentinal walls increase in width with a healthy pulp and odontoblasts
  - A wide canal, in comparison to a relatively more narrow canal of a similar tooth, may indicate the pulp became non-vital at some time in the past (the tooth stopped growing)
    - This comparison is used to assess teeth that have sustained injury (pulpitis) or have been treated (vital pulpotomy) to make sure they continue to mature
  - Internal resorption – irregular areas of wider canal
    - Indicative of an inflammatory process occurring in the pulp – likely non-vital or compromised

- Combination periodontal and endodontic diseases
  - Type 1 Perio-endo lesion – an initial endodontic lesion at the apex extends up the root length coronally until it reaches the base of the sulcus (J-shaped)
  - Type 2 Perio-endo lesion – an initial periodontal lesion (deep infrabony pocket) extends down the root to the extent that the infection reaches the apex of the tooth and the infection compromises the pulp; a periapical bone loss pattern may occur on other roots of multirooted teeth
  - Type 3 Perio-endo lesion – concurrent periodontal lesion and endodontic lesion – either separate or eventually coalescing

**Tooth resorption**

While classically thought of as feline odontoclastic lesions (FORL), the term tooth resorption (TR) refers to any resorptive or erosive lesion of the hard tissues of the teeth (enamel, dentin, cementum), internal or external, dog or cat. Both the type and extent of resorption should be determined radiographically. (AVDC Website)

- Severity of resorption
  - Stage 1 – mild dental hard tissue loss (cementum or enamel)
  - Stage 2 – moderate dental hard tissue loss (cementum or cementum and enamel with loss of dentin) that does not extend to the pulp cavity
  - Stage 3 - deep dental hard tissue loss (cementum/enamel/dentin) – extends to pulp cavity but most of the tooth retains its integrity
  - Stage 4 – extensive dental hard tissue loss, extends to the pulp cavity, most of the tooth has lost its integrity
  - Stage 5 – Remnants of dental hard tissue are visible only as irregular radiopacities and gingival covering is complete (usually odontoclastic)

- Types of resorption
  - Type 1 – focal or multifocal radiolucency is present in the tooth with otherwise normal radiopacity and normal periodontal ligament space
  - Type 2 – there is narrowing or disappearance of the periodontal ligament space in at least some areas and decreased opacity of part of the tooth
  - Type 3 – features of both 1 and 2
The extent of periodontal disease you might encounter in patients can vary from patient to patient and even from tooth to tooth in the same patient. From minimal inflammation and no attachment loss in Stage 1 Periodontal Disease to the beginnings of attachment loss (up to 25%) in Stage 2, then deeper pockets (up to 50% attachment loss in Stage 3) and even compromised teeth (greater than 50% loss) in Stage 4, you must be able to tailor the treatment to the problem. Beyond the dental cleaning, being able to provide advanced periodontal management for your patients is not only good medicine, but good business. By adding simple instruments, materials and skills to your dental armamentarium, you can identify and treat those teeth that may have been extracted in the past.

**Therapy goals**

When looking at periodontal disease, therapy is determined by a number of factors, such as the stage of the disease, the involved tooth, the client’s commitment and the desired outcome. There are several goals to set, including removal of all debris or biofilm (plaque, calculus), keeping the maximum amount of attached gingiva, minimizing attachment loss and minimizing the pocket depth. Certainly, all foreign material, from bacteria to desquamated cells must be removed from the tooth surfaces and pockets in order to attain healing. Since the attached gingiva is the first line of defense against bacteria, a minimum of 2-3 mm is necessary to protect underlying tissues, as the looser alveolar mucosa doesn’t afford that protection. The inability to halt attachment loss will eventually lead to tooth loss. Minimizing pocket depth is related to attachment loss, but is a more specific parameter, because pocket depth in itself directly affects the ability for effective home care and maintenance, and deeper pockets can harbor more virulent strains of bacteria. There are even times where excessive gingiva will be removed to decrease pocket depth (hyperplastic gingiva) or the gingiva will be sutured further down the root (apically repositioned flap) for the same effect. Attachment loss without pocket formation occurs when gingival tissue and bone is lost at the same time, exposing the roots and even furcation areas.

The ability to take intraoral radiographs is essential, in order to determine the extent and characteristics of bone loss. With recession of gingiva and bone across several roots and/or teeth, a horizontal bone loss pattern will often result in exposed roots. With a deeper osseous loss down one root surface, an infrabony pocket may result from the vertical bone loss, and specific therapy may be needed to address that specific defect. These deeper pockets are more difficult to treat and maintain, and anaerobic infections may persist.

**Attachment loss – Treatment decisions**

In evaluating teeth at either end of the spectrum – minimal disease with stage 1 or 2 teeth, or extensive stage 4 disease – the decision process is pretty straightforward. With stage 3 periodontal disease affected teeth – there is more of a challenge to decide whether to extract or try to save. The extent and type of attachment loss is a part of the decision process, as is the consideration of the relative importance of the tooth itself. Major teeth (canines, carnassials) will often be considered for advanced procedures, and adjacent, smaller teeth that are contributing to the infection should be considered for extraction, as their removal will allow better access to the strategic tooth. By extracting the middle tooth in the middle of three rotated, crowded premolars can often enhance the health of the remaining two teeth.

If the attachment loss results in root exposure with minimal pocket formation, professional cleaning and home care may be easier. Any involvement of the furcation puts the tooth at higher risk, due to challenges of continued care. If a pocket is present, it should be thoroughly evaluated: how deep is it? is it suprabony or infrabony?

Patient health status is also evaluated: patients with systemic disease would like benefit more from extraction with the immediate removal of the infection, and a decreased anesthetic time. Clients also are involved in the decision: advanced periodontal therapy requires excellent home care and more frequent professional visits.

**Advanced periodontal therapy**

**Moderate pocket depths**

With suprabony pockets (soft tissue only) of up to 5 mm in depth, evaluate not only the pocket, but the amount of attached gingiva left. If there is 7mm of attached gingiva due to inflammation or gingival enlargement, a simple gingivectomy/plasty can immediately reduce the pocket depth to a more manageable level. A 12-fluted bur on a high-speed hand-piece is extremely helpful with minor trimming. If there is minimal gingival enlargement and only 2-3 mm of attached gingiva, then closed root planing and placement of a periocutec can provide excellent care for the defect.
**Root planning/subgingival cleaning**

This is by far the most important aspect of periodontal therapy. If the debris is not thoroughly removed from the pocket depths, the disease will remain and intensify. The rounded tip of the curette, and it’s rounded back, makes it ideal for subgingival therapy, as opposed to the sharp tip and back of a hand scaler. Certain ultrasonic scalers are modified for subgingival treatments, but most are not. If root surfaces are exposed, or if the pocket depth is less than five mm, closed root planing and subgingival curettage may be performed. Using a curette subgingivally with overlapping strokes in horizontal, vertical and oblique directions, root planing removes calculus, debris and necrotic cementum to provide a clean, smooth surface. Root planning that is too aggressive can damage the root, so take some care. The curette can also be angled slightly to engage the gingival surface for removal of diseased or microorganism-infiltrated tissues, but again, not too aggressively. When pocket depth exceeds 5 mm, or other pathology exists, more invasive procedures are warranted.

**Perioceutic therapy**

In moderate pockets of up to 5 mm in depth (and generally deeper than 2 mm), once the area is debrided, placement of a local perioceutic gel containing doxycycline hyclate can not only provide a direct antibacterial affect against any remaining bacteria, but the anticolagenase activity can help “rejuvenate” the soft tissue of the pocket. The combination of the cleaning and therapy can often help reduce the pocket depth in moderate situations.

Once mixed, the tip of the cannula is gently placed to the depth of the treated pocket, and the material is slowly inserted into the pocket, until a small amount extrudes from underneath the gingival edge. By using light digital pressure on top of the gum, and by gently scraping the cannula tip on the tooth surface, the cannula can be removed without taking the gel with it.

The gel firms up on its own within a minute or two, or a drop of water can be placed on the material to speed up the process. Once firm, the visible material should be gently packed into the pocket, using an instrument such as a W-3, or beaver tail instrument. The owner should be instructed not to brush for about a week in the region (gels and solutions are recommended), nor to pick at the ridge of material that may become visible (light yellow-brown). The material is biodegradable and does not need removal. Sometimes periodontal sealants can be placed after a procedure.

**Surgical periodontal therapy**

Many standard pieces of equipment and supplies can be used, including scalpel blades (15C works well), scissors (sharp/sharp for gingival remodeling), and sutures (usually absorbable, from 3-0 to 5-0). It is important to have other equipment as well for unique oral situations, including periodontal curettes for scaling root surfaces and periosteal elevators (Molt No. 2 or No.4) for elevating gingival flaps. For minor gingivectomy/gingivoplasty, a 12-fluted bur on a high-speed hand-piece can be helpful.

When pocket depths exceed 5 mm but remain above the level of the bone, a simple envelope flap allows access and improved visibility for open curettage and root planing. That deep of a pocket will usually lead to a consideration of extraction, unless the tooth is a strategic one (canine tooth, carnassial tooth). Exposing the area with a gingival flap (scalpel blade inserted into the sulcus, sometimes with a releasing incision, and elevation with a periosteal elevator) allows thorough evaluation and debridement. The flap can then be sutured back into place, or to a position further apical on the root, more directly over the bone, to reduce the pocket depth.

If the pocket extends down between the root and alveolar bone (infrabony defect) inadequate therapy can lead to even further attachment loss and even tooth loss. Just cleaning the area will often lead to the soft tissues (gingival epithelium, gingival connective tissue) growing back into the defect faster than the more important supportive tissues of the periodontium (alveolar bone, periodontal ligament). Placing bone graft material and barrier membranes can actually help exclude the soft tissue and allow bone to grow back into the defect (guided tissue regeneration).

If an adjacent, smaller tooth is involved in the area of attachment loss, its extraction is sometimes the best way to get access to the larger, more strategic tooth’s surfaces. The releasing incision is made away from the tooth being treated, allowing a complete attached gingival coverage of the treated site. Extraction of the middle of three crowded teeth also allows better exposure and treatment of the remaining teeth.

**Specific conditions**

**Mandibular canines and incisors**

The mandibular incisors are frequently affected by periodontal disease and bone loss, especially in smaller dogs. It is tempting just to wiggle out a loose tooth, and that will remove the primary source of the disease, but leaving the involved, less healthy soft tissues can continue to impact adjacent teeth, especially the mandibular canines. The bone loss between the mandibular third incisor and canine can result in a persistent deep soft tissue pocket (with some intrabony extension) once the incisor is gone. A deep soft tissue pocket may also be present around the mandibular canine if the tooth is not fully erupted, as gingiva cannot attach to the enamel that is still below the gum line. Persistent pockets here can predispose the canines to additional periodontal disease with anaerobic plaque bacteria present.

In order to minimize these pockets, the soft tissue linings often have to been excised, and the level of the gingival margin may have to be moved further apically down the tooth. A wedge excision of the tissue from the mesial margin of the canine (the surface closest to the midline of the symphysis) helps remove the excess and granulomatous tissue, and can minimize the pocket depth if the height is
reduced (if sufficient attached gingiva remains). With partially erupted teeth, the wedge incision may not be enough: the attached gingiva may have to be elevated past the muco-gingival junction to release the flap at the level of the looser alveolar mucosa. This way the flap can be repositioned further apically on the tooth and secured with sutures, revealing more of the crown and decreasing the pocket depth. In other teeth, trimming the gingiva or securing the margin further apically will actually expose more root surface, but root exposure is simpler to keep clean that a root within a pocket.

**Mandibular first molar**

Any attachment (bone) loss at the mandibular first molar deserves attention. Advancement of bone loss at this tooth is one of the most common reasons for pathological fracture of the mandible. Bone loss at the mandibular fourth premolar or second molar, particularly if vertical bone loss has started at the first molar, is sufficient reason to extract the smaller tooth to provide access to treat the first molar more effectively. For best periodontal treatment, a releasing flap is made at the furthest margin of the adjacent tooth to be extracted, with the gingiva elevated to facilitate extraction, and thus exposure of the affected root of the first molar. Any pocket lining or granulation tissue in the region should be removed, and the area scaled until healthy root and bone is exposed. If there is an intrabony pocket around the first molar, a bone graft material can be placed, as well as in the alveolus of the extracted tooth. At the very least, the disease tissue should be removed, the root cleaned thoroughly, and the gingiva sutured closed around the first molar.

**Maxillary premolars**

In smaller dogs and brachycephalic breeds, maxillary premolars can often be crowded, sometimes with significant rotation that stack them up on each other. The lack of healthy bone in between these teeth predisposes them to additional periodontal attachment loss, and it can be challenging to keep them healthy. While some propose prophylactic extraction of any rotated and crowded maxillary premolars, in most patients, regular examination and cleaning can alert the practitioner to those that may require extraction. Often, the ‘middle’ tooth in a series of three teeth can be extracted to improve the condition of the two adjacent teeth. Special attention should be paid to the maxillary third premolar, for if the distal root is crowded between the two mesial roots of the fourth premolar, the third premolar may need to be sacrificed.

As a strategic tooth, it is often worth it to provide additional effort to preserve the health of the maxillary fourth premolar. In smaller dogs, it is critical to evaluate the status of the periodontal tissues around the palatal root. It is often so small, that a 3-4 mm pocket with bone loss can completely envelope the root, compromising the entire root. In fact, an infraorbital swelling in a small dog with an intact (not fractured) fourth premolar should lead a close examination of the palatal root.

**Maxillary canines**

Periodontal bone loss at the palatal aspect of maxillary canines can lead to oronasal fistulae, once a deep pocket extends past the level of the palatal bone. Once formed, the fistula is nearly impossible to correct, so extraction is necessary. Chronic fistulation can be challenging to close, as every breath puts tension on the sutured flap. Prevention of fistulation is critical, so careful evaluation of the palatal (and mesiopalatal aspect) of the maxillary canine is important. If a moderate pocket is formed, closed root planning and a perioexceltic may help stop the progression. If an intrabony pocket has formed, there may an opportunity to provide advanced periodontal treatment for guided tissue regeneration to build back the lost bone before the fistula is formed.
Good medicine is good business
All of the areas of dental care that have been discussed obviously provide better medical care for our patients. With a focus on growing that business, dentistry can be a substantial part of your practice’s business as well. There are always challenges to making that happen, and a big challenge can be implementing the great ideas you might have.

Smart goals
The SMART acronym is often used in business applications, but not as well known in the veterinary field:

S – Specific
M – Measurable
A – Attainable
R – Realistic
T – Timely

If you really want to make changes in a practice, you need to select 1 or 2 specific goals that you want to accomplish, and you can only see if you were successful if that goal can be measured. Of course, the outcome needs to be realistic and attainable, but you need to make sure that you set a time limit on reaching that goal as well. Specific goals may be doing more dental procedures (how many more? Compared to what?), taking more radiographs, getting better at extractions (training). One important aspect may be building the value of what you provide for your patients, as this can help overcome many client objections.

Overcoming objections
Two of the most common objections most owners have for dental procedures are the cost and the risks of anesthesia. In the past, anesthesia concerns likely held more importance, but with advances in anesthesia and analgesia management, these risks can be minimized for most of our patients. Appropriate pre-operative patient screening allows us to customize the anesthetic protocols to minimize risk, and use of multi-modal analgesia, including local and regional blocks, can help keep anesthetic doses to a minimum. Advanced patient monitoring and supportive care helps protect these patients during the procedure and close post-operative monitoring gets them through to discharge.

Unfortunately, these advances in anesthetic care have driven the costs of good dental care up, as well as the costs of intraoral radiology and advanced procedures. Practicing a good standard of care does not come cheaply, but providing sub-standard care (including anesthesia-free dentals) can cost our patients their health.

Providing a consistent message about the benefits of good oral care from all clinic team members is important – from the veterinarian to the technician/nurse to the front office staff. We know the medical benefits of the procedures provided, and should be able to confirm this to the client. Sometimes having a ‘pet-parent testimonial’ – a note from a previously reluctant owner – about how good dental care impacted their pet’s health – can help another owner.

Build the value
Part of a client’s objections, especially the monetary ones, come from a lack of appreciation of everything that is provided with dental care. Building the appreciation of the value you provide may start when the ‘phone shopper’ calls in to see how much a ‘dental’ is. Be sure to invite any clients to visit your clinic for a complimentary dental examination, and give them a tour of your facilities to show off your dental capabilities. Part of this visit underscores the education you provide about how many problems can be present in the oral cavity, and how many problems may never be discovered until the complete dental exam under anesthesia.

In the exam room, alert the client to obvious problems – even beyond plaque and calculus – like broken, chipped or discolored teeth, or tooth resorptions in cats. This ‘red flag’ check can be your opportunity to insert two important words – PAIN and INFECTION. While they may be aware of the oral odor their pet has, they may not realize how much the bacteria can be affecting their pet. You can even discuss the impact oral disease can have on systemic health. This is a great time to bring in that ‘pet-parent testimonial’ – the story where the dog felt like a puppy again after getting its oral problems treated.

Another great way to build the value and educate clients is the use of photos. While there are some very good brochures and handouts that help explain the stages of periodontal disease and tooth resorption, nothing hits closer to home than pictures of their own pet’s teeth. Pictures in the exam room (or while they are boarding) can be printed so they can take them home. Before and after pictures the day of the procedure are often quite amazing. Even pictures of the external surfaces of the teeth combined with the appearance of the radiographic structures can help build the value of the importance of taking dental radiographs. A dental photo album following all the steps of a dental procedure at your practice can demonstrate how much goes on during “Fluffy’s Day at the Dentist”.

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**Building compliance**

With all of the education we provide, why are there still issues with compliance? Studies have shown that it takes several ‘touch points’ to get clients to comply with recommendations. Consistent messaging from all staff members is important, and even the wording of recommendations can play a part. Stronger messages of how important the care is for the patient, and detailing the steps of the assessment, radiographs, cleaning and polishing can be more impactful that suggesting a ‘dental’ might be helpful in the next few months.

Consistent recording of the level of disease and other problems is essential in providing recommendations for the patient. Those with significant disease (stage 3 to 4 periodontal disease, non-vital teeth or tooth resorption) should have the procedure scheduled before they leave the practice. If not, they should be called in the next two days to get it scheduled. Mild to moderate disease may not need immediate treatment, but a reminder card or call should be made in the near future to schedule it. Similarly, any patient receiving therapy should be placed into a reminder system, with appropriate time frames for re-calls: 12 months for stage 1 treatment, down to 3-4 months for more advanced cases.

Compartmentalizing some of the costs can help with compliance also. When the patient has been identified as needing therapy, getting the blood work at the time of exam, and dispensing appropriate antibiotic and pain medication serves two purposes. First, it can help defray some of the costs over the two visits (exam and procedure). Second, if the blood work is done, following up with the dental work is a logical step.

In fact, screening blood work and diagnostics (senior care, etc), can easily lead to compliance to recommendations for dental care. Once the blood work is done (and is within appropriate health limits), an important initial step is completed, driving the conversation for dental care. In reverse, the need for dental care may allow you to provide appropriate senior screening for patients that might have not had it otherwise. Occasionally, such pre-operative screening may also uncover previously undiagnosed diseases that can now be managed appropriately. In some clinics, having the blood work done can provide a small discount or incentive for the dental work.

**Home care compliance**

As challenging as compliance for dental recommendations may be, compliance for home care is likely much lower. We should still encourage our clients to provide appropriate home care for their pets, and we should continue to be their best resource for education, and products. Puppy and kitten training should include some discussion on a lifetime of oral care, including professional exams, professional care, and home care. Training to brush should be a gradual process, and alternatives to brushing should be covered as well.

After every dental procedure, a two week recheck can allow you to monitor the recovery of the patient – and also to hear directly from the owner about how well their baby is doing! A toothpaste taste test at this visit (for dogs) gives the opportunity to show how you can ‘pet-alize’ their pet’s home care, using their favorite flavor (or the owner’s favorite, if the dog doesn’t choose), and appropriate brushing device. Discussion should also include appropriate chewing devices (nothing too hard), as well as diets and other products.

**Clinic challenges**

Once you have the clients complying with your recommendations, now you have to handle some challenges within the practice. Three common areas of hurdles include space, equipment and training. Space issues may not have much flexibility, and may determine some choices on equipment (dental and radiographic units on stands, wall-mounted?). Equipment choices are extremely variable, with many levels of investment, but some means of high-speed dental equipment and dental radiography are absolutely essential. Substantial improvement in your instrumentation may be as simple as sharpening your instruments (periodontal curettes and dental elevators) on a regular basis (daily, not monthly).

Staff training is the foundation for a strong dental practice. Without the basics for both veterinarians and technicians/nurses, providing any level of care will be frustrating at best, and sub-standard at the worst. Continuing education is one of the best investments you can make for your patients’ care.

**Scheduling and treatment plan challenges**

Inherent in the challenges of providing good dental care is the fact that there can be so many hidden problems in the mouth that aren’t discovered until the complete examination under general anesthesia with probing and radiographs. Estimates for costs and time of procedure can be ‘guestimated’ at the time of examination, but that is only the beginning. It will be necessary to be able to contact owners during the procedure, and you should inform them that the problems found can substantially change the treatment plan or estimate.

In the work flow of most practices, the technician is providing the bulk of the work, from cleaning and polishing to taking radiographs. It is often helpful to determine at the beginning of the procedure if there will be additional therapy needed, such as extractions that will need the veterinarian’s participation. An initial ‘red flag’ check (after the patient is stabilized and on monitors) can identify many of these problems right away: deep periodontal pockets (palatal aspect maxillary canine or fourth premolar, at either end of the mandibular first canine, between mandibular third incisor and canine), any broken or discolored teeth, tooth resorption lesions. If these are found, they should be marked on the chart, and early placement of local/regional blocks and radiographs can be
taken to confirm if surgery is needed. One quick assessment of broken, worn or discolored teeth is using transillumination (shining a bright light through the tooth) to determine if it is vital (pink pulp apparent) or non-vital (dark or diffuse).

Even with the best planning and exams, it is not uncommon to find substantial disease that may extend the anesthetic time needed for any one patient. Multiply that by the 3 to 4 procedures scheduled for that day, and it can be problematic. Clinics can set up scheduling guidelines based on the level of disease estimated at the time of exam (stage of periodontal disease, or obvious extractions needed), and determine what time frame (or segment) might be needed for that patient. For example, if a clinic can do up to 4 procedures a day, but has 7 ‘segments’ set aside, and a stage 4 patient takes up 4 of those segments, then the remaining procedures only have 3 more segments in which to schedule.

And in those patients where the unanticipated level of disease would either cause a prolonged anesthetic time (or if there are issues during monitoring), it is not unreasonable to consider staged therapy. Being able to do the majority of the cleaning and removing most of the cause of the infection (including very simple extractions) can provide enough initial treatment for the patient to support them until the additional oral surgery is performed 2 to 3 weeks later. Not only can this provide healthier tissue for the more advanced procedure that is needed, but it can help avoid prolonged anesthetic time and give some flexibility in an over-booked schedule.

**Challenging procedures**

In building your dental practice, once you’ve overcome the obstacles of reluctant clients and scheduling nightmares, you will encounter disease that may be beyond your presumed capabilities. Certainly, if there is a referral practice in your area, suggesting a referral is always an option, and if the owner declines, make a note of this in the record. Continuing to build on your dental expertise through training and experience, having the right (and sharpened) equipment, and keeping basic principles in mind, you can likely take on challenges with reasonable results. Dentistry is a practical field, and while advanced procedures can be done to save teeth, sometimes extraction is an option. Your end goal is to provide your patient with a functional oral cavity without pain. That non-healing fracture or oral mass may require a partial mandibulectomy, but knowledge of basic anatomy and surgical skills can help you through that also. The great blood supply of the oral cavity helps tremendously with healing (though challenging during the procedure).

**Summary**

The potential to grow your dental practice is nearly unlimited – from concentrating on expanding the scope of dental procedures to increasing client compliance through education. You can provide a lifetime of care for your patients, and with early intervention and preventive care, many believe you can enhance not only the quality of life of your patients, but you can extend their lifespans as well.

It is essential to support the practice’s effort by obtaining and maintaining appropriate equipment and providing training for the team members. That training will include education of all clinic personnel, in order to support the best messaging for your clients. Working as a team with setting specific goals can help drive the changes you need to build your dental practice. And the good business of dentistry will provide good medicine for your patients.

**References/Recommended reading**


It is important to understand that ear disease is only a symptom (no more specific than “pruritus”). As Dr Flemming Kristensen stated “A patient showing ear problems is a dermatology case until proven otherwise”. It is appropriate therefore to approach the diagnosis of ear disease just as you would for any other skin disease.

Obtaining a detailed history is an important first step in trying to identify the underlying cause of the ear disease. Specific questions that should be asked include:

1. When did the symptoms first occur? This is an important question, because many owners will only tell you when this current episode of symptoms occurred, not the very first time it occurred;
2. Other than the problem the owner presents the patient for, you must ask all owners if the dog has EVER had problems with excessive licking, scratching, chewing, biting or rubbing. Has the dog ever had ear problems before this episode? If so, when, with what medication and what was the response to treatment;
3. Where does the dog live- indoor, outdoors, both? Describe the environment, especially the outdoor environment;
4. Is the dog on heartworm and flea preventative? If so, what product, how often is it administered and is it year round or seasonal?
5. Are there any other pets in the household? If so, what kind and are they symptomatic. If they are cats, do they go outside? 
6. Are any of the humans in the household showing “new” skin problems? If so, what kind;
7. Do they board the dog, take him to obedience school, training or to the groomers? If so, when was the last time? 
8. Do they know if the parents of the dog or any siblings have ear or pruritic skin problems? If so, what was done and what was the response? 
9. What does the dog eat? 
10. How do the ears seem today- is today’s presentation the best, worse or average since the problem began?
11. Do you notice if the symptoms were better, worse or no different or not sure between the different seasons.

After reviewing signalment and thoroughly questioning the owner, the next step is to do a complete physical examination – be sure to note any constitutional signs that may be present that could explain the ear problem (eg fever associated with pemphigus, lethargy associated with vasculitis, etc)

This is followed by a complete dermatologic examination. Because ears are really just skin attached to the skull many diseases that affect the ears frequently will be affect the rest of the skin and vice versa. Therefore even when a dog is presented only for otic pruritus you still need to examine the rest of the body. And the opposite also holds true, when a dog is presented for truncal pruritus be sure to do an otic examination.

In order not to miss an abnormality, an otic exam should be done in a systematic manner beginning with the pinna. You should note any alopecia, erythema, ulceration, crusting, scaling or swelling. Then palpate the canals for pain, calcification or thickening. This is followed by an otoscopic examination of the ear canals. Due to the curve in the external ear canal, the ear canal must be straightened in order to see the horizontal canal and the tympanic membrane. This is accomplished by placing the tip of the cone of the otoscope in the opening of the external ear canal. As you advance the cone is proximally you need to pull the pinna laterally (outward). By “stretching” the pinna laterally into a straight line horizontally the ear canal becomes straight and allows examination of the horizontal canal and the tympanic membrane

The presence, degree and location of inflammation, ulceration & proliferative changes should be noted (i.e. cobblestone hyperplasia). Describing the size of both the vertical and horizontal canals along with the type, location and quantity of debris or exudate should also be included in the medical record. Next it should be documented whether the tympanic membrane is visualized. If it is not, then note why the membrane is not seen- is it due to swelling in the ear canal, the presence of a ceruminolith or is there debris in the proximal horizontal canal obstructing the view? Sometimes it is because the animal is too painful to allow deep examination of the ear canal. If you can visualize the tympanic membrane (TM) you need describe if it is normal in appearance or not. Changes that may be noted include discoloration or bulging.

It is important to then evaluate for concurrent middle or inner ear disease. This is because dogs with chronic recurrent otitis externa (OE) may have concurrent otitis media (OM). This step may require heavy sedation or general anesthesia. Evidence of middle ear involvement include a ruptured TM or an abnormal appearing TM (i.e. thickened, change in lucency (opaque), bulging or discolored).
Horner’s syndrome (damage to sympathetic innervation); keratoconjunctivitis sicca (damage to the parasympathetic component of the facial nerve) and facial nerve paralysis may be present in cases of OM due to the close association of the respective nerves to the middle ear. Deafness may also be present with OM.

Some veterinarians will have their staff collect ear cytology samples prior to examining the ear (as a time saver) but this makes it more difficult to evaluate the true appearance of the ear canal. Debris may be pushed into the horizontal canal thereby limiting visualization of the tympanic membrane due to the compacting of debris in the canal.

Now diagnostics and treatment needs to be pursued. The first step is to identify and treat the primary (underlying) cause(s) of the ear disease. These would include:

1. Parasitic (including Demodex, Otodectes, Sarcoptes);
2. Foreign bodies;
3. Hypersensitivities (atopy- NOTE OE may be the ONLY symptom in 3-5% of the environmentally triggered atopic dermatitis cases and it may be UNILATERAL!!; it may be seen in cutaneous adverse food reactions where it too may be the ONLY symptom in up to 20% of the cases and also may be unilateral or flea allergy dermatitis. In cases of FAD there should be involvement of the posterior 1/3 of the body in addition to the OE;
4. Allergic or irritant contact dermatitis;
5. Endocrinopathies, keratinization or sebaceous gland disorders leading to an altered lipid layer in the epidermis, alteration in normal keratinization or glandular function; idiopathic seborrhea is there such a disease?);
6. Autoimmune or immune mediated diseases (eg pemphigus complex, vasculitis- note these diseases involve the pinna >>> canals);
7. Zinc responsive dermatosis (will involve more than the pinna);
8. Juvenile cellulitis;
9. Immunosuppressive diseases (distemper, FeLV, FIV, parvo virus);
10. Neoplasia (adenoma, adenocarcinoma) ;
11. Dermatophytosis (affects the pinna rather than the ear canal).

In addition to identifying the primary cause, secondary factors must be addressed if possible. Secondary factors don’t cause ear disease but increases the risk of developing ear disease and may make successful treatment more difficult. Secondary factors are:

- anatomical factors (eg- long pendulous ears in the Basset Hound or stenotic ear canals in Shar Peis);
- excessive moisture in ears (swimming);
- iatrogenic trauma (plucking hairs from the ear canals, cleaning ear canals with cotton tip applicators).

Lastly perpetuating factors must be identified and treated. These factors don’t initiate the problem, but will cause the disease to continue, even with the elimination of the primary factor, once it has been established until these factors have also been addressed. Perpetuating factors include:

1. Bacteria (coci most commonly *Staphylococcus intermedius* (acute infections), beta hemolytic streptococci and rods most commonly *E. coli, Pseudomonas* spp (chronic infections); *Proteus* spp, *Klebsiella* spp and *Corynebacterium* spp);
2. Fungi (*Malassezia pachydermatis* (which may cause a hypersensitivity reaction so that small numbers may be significant) ;
3. Progressive pathological changes;
4. Otitis media;
5. Contact hypersensitivity/irritant;
6. Treatment errors (most commonly due to under treating the infection).

Laboratory tests are a necessary component to the proper workup of a case of canine ear disease. CBC, serum chemistry profile, urinalysis, skin scrapings, fungal culture, endocrine testing and skin biopsies may be necessary depending on what the differential diagnoses are for that patient.

Cytologic examination of a roll swab sample should be performed on any exudate. The numbers & type of bacteria, yeast and inflammatory cells should be quantitated. In cases of OE the question of what is an abnormal number of organisms, per oil field, has not been settled. Depending on the study, cutoff numbers, per oil immersion field, that differentiates between normal and abnormal ears ranges from >1 Malassezia to >4 Malassezia and from >1 bacteria to >10 bacteria. It is the author’s opinion that the number of organisms present to be considered significant is not just a “number”. The author doesn’t perform cytology on normal ears – it is only done if the ears that are inflamed or have exudate. Therefore ANY organism seen will be considered significant and will be treated as part of the therapy regardless of the number present. As for follow-up cytoplogies, the only time cytology is performed during therapy is when the ear is not clinically improving OR if the initial cytology had rods. If there is a mixed population of organisms present at the initial examination without rods and the ear is clinically normal at the recheck examination, follow-up cytology is not performed.

Bacterial culture and susceptibility (c/s) should only be rarely, if ever, performed in cases of OE. If a c/s is performed, it should be done in conjunction with cytology. One reason that the author doesn’t perform cultures in OE cases is that with a culture the
susceptibility is based on systemically achieved antibiotic levels (measured in microgram/ml) not topically. Since topical medication has a 1000 fold higher concentration (milligrams/ml) the resistance reported on the culture can’t be extrapolated to topical therapy.

Other concerns include poor reproducibility of c/s results when culturing the ear. In a study where two samples were taken for bacterial c/s from the same location in the external ear canal of dogs who had otitis externa, there were different bacterial isolates identified 20% of the time and the same isolate with different susceptibility patterns another 20% of the time. Eleven percent of the P. aeruginosa isolates had different susceptibility patterns. A second study took triplicate samples and sent the samples to 3 different laboratories. There were 18 samples that had Pseudomonas spp. Identified. All three laboratories only agreed on the presence of Pseudomonas in 15 (83.35) of the ears while 2 agreed on 2 (11.1%) of the samples and on one occasion (5.5%) only 1 laboratory identified Pseudomonas but none of the samples had identical patterns of antibiotic susceptibility. A 3rd study was performed in which duplicate samples were sent to the same lab. Seventy percent of the Pseudomonas aeruginosa had different susceptibility profiles.

There are a few possible reasons for these discrepancies. These include:
1. Multiple strains with different susceptibilities
2. Single strain with heteroresistances

In both of the cases the selection of which colonies are selected to be tested for susceptibility may vary from technician to technician. A 3rd study was performed in which duplicate samples were sent to the same lab. Seventy percent of the Pseudomonas aeruginosa had different susceptibility profiles.

These results should give you great pause as to the reliability of cultures. The author will only take a culture in cases of OE when there are proliferative changes present AND there are numerous rods present on cytology AND the dog has failed to respond to empirical antimicrobial therapy. This is a very uncommon scenario. This approach is supported by a study in which the author evaluated if there was any correlation between topical antibiotic selection, in vitro bacterial antibiotic sensitivity and clinical response in 16 cases of canine otitis externa complicated by Pseudomonas aeruginosa. For these cases empirically selected topical antibiotic therapy was dispensed after collecting bacterial cultures from the affected ears. All dogs had Pseudomonas aeruginosa isolated on culture. In 10 cases, the antibiotic selected was deemed to be resistant based on the culture, yet 8/10 responded to the selected antibiotic. One of the 10 resistant cases needed to have a second antibiotic selected to successfully treat the infection. This supports the observation that there is no value to performing cultures in cases of canine otitis externa.

The MIC (broth microdilution technique) method is the “gold standard” for culture technique therefore if a c/s is submitted, the MIC method should be used to determine the susceptibility of the organism(s) rather than the disc diffusion method (Kirby-Bauer). This is because the disk-diffusion susceptibility test (DDST) is only semi quantitative. This means that the drug concentration achieved in the agar surrounding the disc can be roughly correlated with the concentration achieved in the patient’s serum. It will only report the organism’s susceptibility (susceptible, intermediate or resistant) based on an approximation of the effect of an antibiotic on bacterial growth on a solid medium. Tube dilution (MIC) is quantitative, not only reporting SIR but also the amount of drug necessary to inhibit microbial growth. The MIC is reported as the amount of antibiotic (in µg/ml) necessary to inhibit 90% of the tested bacteria (the lowest concentration in the tube that is clear). This allows a clinician to not only decide susceptible or resistant but also the proper dosage and frequency of administration of the antibiotic. Note that if the MIC for the bacterial isolate is reported to be susceptible, there is a greater likelihood of successful treatment (cure) than if the isolate was classified as resistant. Treatment failure is still possible due to other drug or patient factors such as the location of the infection and the immunologic status of the host. If the MIC value is in the intermediate category, therapy with this drug at the usual dose will likely be unsuccessful in establishing a cure. However, successful therapy is possible when doses higher than the label dose is used or if the drug is concentrated in the affected organ (eg urine) or is used topically (ear). If the MIC is in the resistant category, treatment failure is more likely because of resistance mechanisms or inadequate drug concentrations. Lastly not only does the MIC method indicate susceptibility, but it also implies the relative risk of emerging resistance and thus the need for a high dose.

The other limitation to the Kirby-Bauer results in regards to Pseudomonas susceptibility is the discrepancy between it and MIC. In two studies, Kirby-Bauer underestimated P. aeruginosa sensitivity to enrofloxacin (when compared with MIC) whereas in 2 other studies Kirby-Bauer overestimated enrofloxacin susceptibility. Since Pseudomonas infections is one of the most common reasons cultures are performed in cases of otitis externa, enrofloxacin is a commonly used antibiotic for this infection, this inability to properly identify susceptible vs resistance to enrofloxacin is an important limitation in using Kirby-Bauer testing.

With the information gathered above, the treatment is directed toward the primary cause(s) (eg parasiticidal treatment, food trial, intradermal testing and allergen specific immunotherapy, etc) and perpetuating factors. Ear cleaning is performed in the clinic with a bulb syringe, AuriFlushTM system or by retrograde tube flushing (under anesthesia). If on the initial examination the ear canals are swollen and painful, ear cleaning may not be performed on the first visit, preferring to use topical glucocorticoids (GC) and systemic GC for 10-14 days to decrease the swelling. Once the swelling has decreased it will be much easier to examine the ear canals and visualize the TM.
Cleaning agents contain substances that soften and emulsify wax and lipids. This initial cleaning is necessary in order to remove debris that may interfere with the effectiveness of topical agents and to reduce inflammatory debris (bacterial toxins). The author doesn’t usually have the owner do cleaning after the initial exam since it seems that many owners have trouble with just medicating the ear, let alone cleaning too. Many of the cleaners have a low pH leading to discomfort if used in an inflamed ear. A study comparing 2 ear cleaners (original formulation and then a new formulation) noted that in 38% of the cases with the old formulation and 37.5% of the cases with the new formulation dogs had a moderate to marked avoidance to having the cleaner instilled. This behavior was believed to be due to either a reaction to the ear cleaner or just overall animal irritability. Also the base in the otic ointments/suspensions (mineral oil, liquid paraffin) acts as a ceruminolytic agent. In addition, a recent study calls into question whether any of the ear cleaners have any ceruminolytic activity. In this study the ceruminolytic activity of 13 ear cleansers was evaluated using a standardized synthetic cerumen (SSC) that mimics the composition and texture of canine cerumen. Of the tested products only Cerumene®, Epiotic® and Vet Solutions Ear Cleaner® are available in the US. The test products were incubated with mild agitation for 20 min with 500 mg of SSC previously compacted at the bottom of a test tube. Ceruminolytic activity was then assessed by quantifying the SSC removed by decantation. Overall, Otoclean® (OT) was most efficacious, reaching an activity of 86–90% followed by Netaural® (NET) with a 39%, Specicare® (SP) with a 23% and Cerumene® (CE) with an 8% ceruminolytic activity. None of the other products displayed any ceruminolytic activity. It was concluded that, in the experimental conditions used in this study, only 1/13 products had significant ceruminolytic activity. Please note that the company that manufactures OT funded this study A follow up study by Robson, et al using Australian and US products revealed that 15/24 cleaners had <5% efficacy while only 6/24 ear cleaners had >80% efficacy-none of which are available in the US.

There is frequently a discussion of the otoxicity of agents put into ears. Remember that it is inner ear damage, specifically vestibular and/or cochlear damage that occurs with ototoxic agents, not middle ear damage. In order for a drug to cause damage to the inner ear it must either get to the inner ear hematogenously or by traveling thru the middle ear and entering the inner ear thru the vestibular (oval) or cochlear (round) window(s).

In humans because ofloxacin otic solution (Floxin Otic®) is the only topical agent to be labeled by the U.S. Food and Drug Administration (FDA) for use when the tympanic membrane is perforated, oral antibiotics have traditionally been used in this situation. However, according to otolaryngologists because the risk of cochlear damage with the use of other topical medications seems quite small, perforation alone is not an indication for oral antibiotics.

The opinion of this author is that the concern for otoxicity due to topical medications is overstated. This position is supported by a consensus panel on reviewing the use of ototopical antibiotics. In their report they stated “There have been very few irrefutable cases of otoxicity reported (after proper use of a topical otic preparation). Under many circumstances, it is difficult to separate the underlying disease process, which is also known to cause otoxicity, from ototopical drug use.” They go on to state “For more than 40 years, the most common treatment has been aminoglycocide combination drops. A longstanding debate over the safety of these drops centers on otoxicity. Even though the theoretical risk exists, there have been few reported cases in the literature, considering the millions of doses given”.

The author has only seen one ototoxic reaction that was suspected to be due to a topical agent and in that case the TM was intact! Therefore, agents are chosen more for their effectiveness than the concern about otoxicity, especially since there are very few agents that have been proven to be safe in cases of a ruptured TM. It is more important to get rid of the infection than to avoid (effective) drugs because of otoxicity concerns. Also, just because the TM is intact doesn’t mean that the barrier function is complete, therefore, even in the presence of an intact TM it is possible to get drugs into the middle/inner ear.

After ear cleaning topical agents are dispensed. The author prefers ointments over drops because of the impression that ointments get the drugs to the region of the tympanic membrane better than drops do (this may be a volume issue more than the formulation- it has been reported that it takes 1.0 cc of medication to get down to the TM in a medium sized (40 pound) sized dog - personal communication). The other advantage of ointments is that the base vehicle in the otic ointments (mineral oil/liquid paraffin) acts as a ceruminolytic agent.

Most topical products contain a combination of glucocorticoids, antibacterial and antifungal agents. Antibacterial agents used topically include:

1. Broad spectrum agents (gram positive and negative organisms) –
   a. Aminoglycocides
      i. Decreased effectiveness in an acidified ear
      ii. Inactivated by purulent debris (so they must be put in a clean ear)
   b. Neomycin
   c. Gentamicin – note injectable water based gentamicin is non toxic even if the dog has a ruptured tympanic membrane- this has not been studied when using commercial ear products that contain more than just gentamicin.
   d. Silver sulfadiazine - inactivated by purulent debris so they must be put in a clean ear. It needs
to be compounded to a 1% solution
   a. Spectrum also includes yeast
   b. Inactivated by purulent debris so they must be put in a clean ear

2. Narrow spectrum agents (gram negative rods) – most are reserved for resistant gram negative infections
   a. Polymyxin B - inactivated by purulent material
   b. Fluoroquinolone – decreased effectiveness in an acidified ear
      i. Never a first line choice
      ii. EnroFloxacin
      iii. Orbifloxacin
   c. Extended-spectrum penicillins (anti- Pseudomonas penicillins)
      i. Susceptible to beta lactamase
      ii. Penetrate Pseudomonas cell wall better than other antibiotics
      iii. Increase gram negative activity but less activity gram positive and anaerobes compared to other penicillins
      iv. Carboxypenicillin
         a. Ticarcillin
   d. Aminoglycocide
      i. Amikacin and tobramycin
         a. Gram negative bacteria (including some Pseudomonas) have less resistance to amikacin or tobramycin then gentamicin or neomycin
         b. Decreased effectiveness in an acidified ear
      c. Inactivated by purulent debris so they must be put in a clean ear

Antifungal agents used include thiabendazole (anecdotally reported to have poor efficacy against Malassezia- is it volume related?), nystatin, clotrimazole 1%, miconazole 1 or 2%, posaconazole 0.1% and ketoconazole 1 or 2%

When gram negative organisms are present treatment of OE should include EDTA. To understand the action of ethylenediaminetetraacetic acid (EDTA) solution we need to review some microbiology. A capsule surrounds bacteria. Under the capsule is the cell wall that contains peptidoglycans. Under the cell wall is the cytoplasmic membrane (plasma membrane, cell membrane). The cytoplasmic membrane surrounds the cytoplasm and nuclear body. Gram negative have 2 additional layers. The outer most is the outer cell membrane that lies between the capsule and the cell wall. The outer cell membrane is composed of lipopolysaccharides. The other additional layer is between the cell wall and cytoplasmic membrane, called the periplasmic space. This space contains a variety of enzymes and other proteins that help digest and move nutrients into the cell. Gram positives do not have the outer cell membrane (and therefore no lipopolysaccharides) but do have a thick layer of peptidoglycans in the cell wall (vs. gram negatives which only have a thin layer). Note the peptidoglycans are the site of action for beta-lactam antibiotics.

Topical EDTA solution has a direct bactericidal action against bacteria by chelating metal ions important for the integrity of the bacterial cell wall. EDTA also stimulates the release of outer cell membrane lipopolysaccharides (LPS), proteins, and other cell contents. The end result of these actions is the leakage of cell solutes leading to cell death and better drug penetration and antimicrobial activity. Note - since EDTA stimulates the release of LPS from the outer membrane it is less effective at inhibiting gram-positive than gram-negative bacteria because gram-positive bacteria lack an outer membrane.

Pseudomonas bacteria have an efflux pump that is mediated by the MEX gene. This protein pumps the drugs out the bacteria, rendering the antibiotic ineffective. EDTA blocks this pump thereby allowing the antibiotic to accumulate in the bacteria.

To maximize its bactericidal activity it is essential for EDTA to be in an environment with an alkaline pH. Appropriate pH (8.0) is maintained by combining it with buffers such as tromethamine (TRIS) hydrochloride. This alkaline pH also decreases the bacterial MIC for an aminoglycocide or a fluoroquinolone. It is therefore useful to use TrisEDTA prior to instilling either of these antibiotics. Two commercial veterinary preparations are available - TrizEDTA®, (Dechra) or Tris Flush® (Sogeval). The ear canal should be filled with the solution prior to instilling the topical antibiotic (15-30 minutes before is ideal). This is done q 12 hrs. EDTA is used primarily for treatment of otitis externa and/or media caused by gram-negative organisms especially Pseudomonas.

A product made by Dechra, TrizChlor® contains 0.15% chlorhexidine in addition to the trisEDTA. The combination of these 2 ingredients is beneficial due to the synergistic effect between EDTA and chlorhexidine. The addition of the chlorhexidine extends the antimicrobial spectrum to include cocci in addition to the rods. There are 2 studies that support the effectiveness of this combination. The limitations of these studies are they are in vitro studies and they used a 30 minute contact time. Whether these results can be repeated in vivo has not been studied. Since the author uses this product in combination with other topical agents, it is impossible to draw an accurate conclusion.
In regards to safety of the chlorhexidine in otic products, a study reported the effects of instilling 0.2% chlorhexidine into the ear canals of dogs with experimentally ruptured tympanic membranes. In this study, 0.2% chlorhexidine was instilled in greyhound’s ear canals bid for 21 days. At the end of the study there were neither clinical vestibular signs nor BAER changes noted. THIS DOESN’T APPLY TO CATS!!!. A study instilling 0.05% chlorhexidine once every other day for 3 treatments into the middle ear of cats concluded that even this concentration of chlorhexidine may cause hearing loss in a cat. The authors did a subsequent study in which they evaluated vestibular effects of infusing chlorhexidine into the middle ear of cats. That study concluded that exposure of the middle ear to even dilute concentrations of chlorhexidine (0.05%) were likely to cause vestibular disturbances.

Any otic cleaner that contains EDTA-Tris would be appropriate to use when otitis externa/media is complicated by both rod shaped bacteria and Malassezia. Some contain ketoconazone. An unanswered concern about using ketoconazole chronically as a maintenance treatment is whether (when?) resistance will to ketoconazole will develop. Also acidifying the ear canal is one of the best treatments/prevention for Malassezia otitis and these products alkalinize the ear.

GC’s are an essential component of topical treatment. Successful treatment of OE frequently requires topical GC and in fact the author has seen cases resolve where the only change in therapy was the addition of topical GC. GC are antipruritic, anti-inflammatory, decreases glandular secretions (cerumen), decreases pain and swelling and decreases hyperplasia- all properties that can help restore the normal barrier function to the epithelium of the ear canal. When using topical GC it is best to begin with the most potent form and if GC are needed long term go to less potent (and less side effects) forms (in decreasing potency-mometasone>betamethasone= hydrocortisone aceponate > fluocinolone> triamcinolone>dexamethasone> prednisolone> hydrocortisone). Note- even though hydrocortisone aceponate is classified as an intermediate potent glucocorticoid, equal to that of betamethasone 17-valerate, it has an improved benefit/risk ratio due to its decrease incidence of skin atrophy. REMEMBER topical steroids are systemically absorbed and can lower thyroid hormone concentrations; elevate liver enzymes, suppress the hypothalamus- pituitary-adrenal axis and even cause pu/pd.

The author has rarely used systemic antibiotics when treating OE. This approach is supported by the previously mentioned consensus panel who stated “In most cases of uncomplicated AOE, topical antibiotics are the first-line treatment choice. There is no evidence that systemic antibiotics alone or combined with topical preparations improve treatment outcome compared with topical antibiotics alone”.

In addition systemic antibiotics increase the risks of adverse effects and enhancing the environment for the production of resistant organisms. In humans it has been reported to increase the time to clinical cure and do not improve outcomes compared with a topical agent alone in uncomplicated otitis externa. In humans systemic antibiotics are recommended to be used only when the infection has spread beyond the ear canal, or when there is uncontrolled diabetes, immunocompromise, a history of local radiotherapy, or an inability to deliver topical antibiotics.

Systemic antibiotics or antifungal agents are used only if otitis media with bacteria, other than Pseudomonas (see below about Pseudomonas), or Malassezia are present on cytology, compliance and follow up has been good and topical treatment has been unsuccessful (very rare occurrence). Once again this approach is supported by the consensus panel (for humans) in which they state “The initial therapy o otherwise normal, healthy patients with CSOM (chronic suppurative otitis media)... should consist of ototopical drops and thorough cleaning of the canal.”

Empirical choices for cocci include cephalosporins, amoxicillin–clavulanic acid, clindamycin and potentiated sulfas. Empirical choices for rods include cephalosporins, amoxicillin–clavulanic acid (use TID vs. BID for gram negative organisms) and potentiated sulfas. Fluoroquinolones should be reserved for culture-proven resistant gram-negative rods. The antifungal agents that the author prefers include ketoconazole (5 to 10 mg/kg sid, given with food to enhance absorption), fluconazole (10 mg/kg sid), anditraconazole (5 mg/kg sid).

If the OM infection is due to Pseudomonas it is unlikely that systemic antibiotics will be useful. This is because systemic administration of antibiotics, including the fluoroquinolones, can’t exceed the MIC for P. aeruginosa in the ear canal. Since P. aerugiosa is the most common pathogen associated with OM in dogs, systemic administration of antibiotics will only select for more resistant organisms. Since it has been documented in humans that high drug concentration may be achieved in the middle ear when topical antibiotics are used, in cases of OM, topical treatment is the author’s mainstay therapy.

Systemic glucocorticoids are used if the ear canals are edematous, ulcerated and/or stenotic. Even proliferative changes may decrease with steroid administration since secondary edema may be present. Prednisone at 0.25-.50 mg/# bid for 7-14 days is dispensed and a reassessment is made in 7-14 days. At that time if the canals are completely open and the ulcers are healed, the prednisone can be discontinued. If the ears are better but not normal then make a clinical decision is made whether to maintain or decrease the dose for another 7-14 days. Again reassessment should be done in 7-14 days. If the ear canals are not opened by this second recheck, a total ear canal ablation with a bullae osteotomy would most likely need to be performed.

Specific scenarios

1. Acute otitis (and/or infrequent) externa treatment overview. It is important to differentiate whether this is a first time occurrence, a recurrence or an unresolved infection. The only way to know this is to do follow-up examinations on
ALL cases of OE. Remember that the absence of symptoms is not synonymous with resolution of the disease. This means that owners are unable to determine whether the infection is resolved and the dog must be rechecked. If this is the first episode, discuss the possible predisposing, primary and perpetuating causes and foreshadow that additional testing may be necessary in the future. In this situation, begin with eliminating easily diagnosed primary causes (foreign bodies, parasites, masses, etc). During the examination be sure to evaluate the status of the tympanic membrane. Perform a cytology to identify secondary infections. Treatment should be directed toward both the infectious component and the inflammatory component. Treatment should be for 7-14 days, unless using Easotic® (Virbac). At the end of the treatment, while still on therapy, a recheck examination should be performed!! Traditionally once the OE has clinically resolved the author has treated cases for an additional 7 days. More recently the author has begun to use a product with a unique delivery system- Easotic® (Virbac). When using this product, the dog is only treated for 5 days, in contrast to the 7-14 day schedule as previously mentioned, and then rechecked. The author has found this product to be very effective- most likely due to better compliance. Unless contraindicated, a topical GC containing product should be used as part of the therapy. The author prefers ointments over drops when treating otitis externa. Since all the otic ointments contain steroids and an antimicrobial agent, the author uses a combination product.

a. In cases of an acute infection there are a variety of products that are effective and would be appropriate to dispense (note most products will contain a combination of 3 of these drugs- antifungal, antibiotic and steroid). Typical ingredients include miconazole, polymyxin B, prednisolone, nystatin, neomycin sulfate, thiostreropon, triamcinolone acetonide, gentamicin, hydrocortisone aceponate, betamethasone valerate and clotrimazole.
b. The only time this is altered is if there are heavy rods or just rods present, which is very rare in this scenario. In that case the author would use TrisEDTA, silver sulfadiazine and either gentamicin or polymyxin B (see below- Pseudomonas)
c. If the dog is painful, systemic GC and analgesics (tramadol, gabapentin and/or Tylenol with codeine) are added to the treatment.

2. If initially TM the is not visible due to swelling of the ear canals oral prednisone ½-1mg/#/day for 10-14 days will be added to the topical treatment. Because of the potency of fluocinolone or mometasone, Synotic® (fluocinolone with DMSO) and/or Mometamax® (mometasone) will be included in the therapy. Many times an analgesic is added as previously described (NO NSAID!).
a. A recheck examination will be performed in 10-14 days. If the TM is visible and the swelling resolved, then only the prednisone can be stopped. All the other treatment should be continued.
b. If the TM is not visible but the swelling has resolved, then an ear lavage via FEVO under general anesthesia should be performed.
c. If at the 10-14 day recheck the TM is not visible and the swelling has NOT resolved, continue the prednisone for another 10-14 days and then recheck.
   i. If the ear canals are still narrowed at the next recheck, perform (or refer) a total ear ablation with a bullae osteotomy.

3. In cases of chronic (recurrent and/or unresolved) otitis externa, it is essential to determine if it is recurrent or unresolved. If it is unresolved is it because of owner compliance? If it is poor compliance then this problem must be resolved! If it is recurrent (or unresolved with good owner compliance) in addition to the above, a very aggressive search is performed to identify and treat the primary, perpetuating and secondary factors. Treatment should be for a minimum of 30 days. As above, GC will be an important component of therapy.
a. If there is only yeast, then the depending on what products have already been used, consider using clotrimazole 1%, miconazole 2%, 0.1% posaconazole or 2% ketoconazole lotion compounded with dexamethasone 0.1%.
b. If cocci are the only organism present then use gentamicin, mupirocin or 5% cefazolin (1 gm vial mixed with20 cc Triz-Edta plus).
c. If rods +/- cocci are present then use- Triz-Edta (+/- chlorhexidine if cocci are present) along with gentamicin or polymyxin B and silver sulfadiazine
d. Because of the association of the use of fluoroquinolones and the development of MRSA, and E.coli, the author rarely uses fluoroquinolones for the treatment of otitis externa. This concern is supported by many different sources. In the BSAVA “Guide to the Use of Veterinary Medicines” it discusses the prudent use of antimicrobial agents. In regards to all fluoroquinolones (FQ) it states “that in all species fluoroquinolones and third- and fourth-generation cephalosporins should be used judiciously and never considered as first-choice options”. The concern with using FQ is that, according to information from the CDC website, “a major
limitation of fluoroquinolones is that resistant mutants can be selected with relative ease, leading to relapse and treatment failure”. In addition it has been observed that there is a significant association between total fluoroquinolone use within human hospitals and percentage of S. aureus isolates that were MRSA and between total fluoroquinolone use in the community and percentage of E. coli isolates that were fluoroquinolone-resistant E. coli. Association between fluoroquinolone exposure and the induction of mecA-positive S. aureus (MRSA) and the increase in the resistance index for methicillin resistance has been noted. Lastly it has been widely reported that there is an association between FQ use and clinically significant MRSA i.

The only time the author will use enrofloxacin or orbifloxacin is when the infection has failed to respond to the author’s aggressive therapy. The author prefers the later product due to the inclusion of steroids in the lotion. If using the former, dexamethasone should be added to achieve a final concentration if 0.1% dexamethasone.

Pseudomonas infections are especially challenging because of Pseudomonas’ intrinsic multidrug resistance (MDR). Many of the clinically relevant resistance mechanisms in Pseudomonas aeruginosa are attributed to synergy between its outer membrane that has a very low permeability to drugs and the presence of an active drug efflux pump (MEX). Because of the intrinsic MDR, Pseudomonas infections successful treatment must be aggressive before other resistance develops.
Atopic Dermatitis:
The New Paradigm and How it Changes the Way We Treat It
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The understanding of canine atopic dermatitis (cAD) has changed dramatically over the last several years. This has lead to a change in our therapies. It is now accepted that the pathogenesis of cAD involves not only an immunologic component but also a barrier dysfunction. This disruption of normal barrier function leads to increased allergen penetration and sensitization. Thus, the therapeutic approach has changed from addressing only the immunologic abnormality (hypersensitivity) to one that also includes managing the barrier dysfunction. It is essential that before treatment for cAD is begun that the proper diagnosis has been established. It is recognized that the diagnosis of cAD is a clinical diagnosis made by ruling out other pruritic diseases. Criteria that can help establish a diagnosis of cAD are:

1. Onset of signs under 3 years of age
2. Dog living mostly indoors
3. Glucocorticoid-responsive pruritus
4. Pruritus sine materia at onset (i.e. alesional pruritus)
5. Affected front feet
6. Affected ear pinnae
7. Nonaffected ear margins
8. Nonaffected dorso-lumbar area

If the dog meets 5 criteria - there is a specificity of 79% (21% false positives) and a sensitivity of 85% (15% false negative)
If the dog meets 6 criteria - there is a specificity of 89% (11% false positives) but the sensitivity decreases (more false negative) to 58% (42% false negative)

In the author’s experience an easier and very accurate modification can be used as follows. It is called the “One minute atopic dermatitis test”. If you have a pruritic dog, you can be fairly certain that he/she has cAD ectoparasites and infection (bacterial, yeast/fungal) have been ruled out. There are exceptions to the rule but they are very uncommon and suspicion for these diseases would be raised based on signalment, history and physical findings. Note that serum testing/intradermal testing and/or food trials may be needed to ID flare factors of cAD but are not used to diagnose cAD. Whether these additional tests are necessary depends on the severity of the cAD.

Treatments of the pruritic dog

There are a variety of therapies for the symptomatic relief of pruritus in dogs with atopic dermatitis which will help the disease at that moment. These treatments will do nothing to prevent recurrence, this can only happen if the underlying cause is identified and treated. You will be more effective long term in treating the pruritic patient if you find the “due to” rather than just treat the symptom (pruritus). This lecture is focused on new therapies for the treatment of cAD.

If a specific diagnosis for the pruritus has not been established after the initial diagnostic tests have been performed and infection is present it is best to treat the infection for 14-21 days and then re-evaluate how much pruritus remains. DON’T use GC, oclacitinib or lokivetmab. Treat what you know and see what is left. If you used the previously listed drugs and also the infection at the same time it would make interpretation of response to therapy impossible (was it the antipruritic drug or the antibiotic/antifungal therapy that resolved the pruritus?).

If the pruritus has resolved after only treating the secondary infections and/or ectoparasites it means that the ectoparasites or the secondary pyoderma/Malassezia dermatitis was the major trigger of the pruritus at this time. This secondary infection was due to one (or more) of the following:

1. Ectoparasites
2. Seasonally triggered environmental allergen induced atopic dermatitis and the season has changed
3. Nonseasonally triggered environmental allergen induced atopic dermatitis that is not symptomatic when the infection is absent (threshold theory)

4. Environmental allergen induced atopic dermatitis that is triggered by a cutaneous food reaction that is not symptomatic when infection is absent (threshold theory)

5. An endocrinopathy (hypothyroidism, hyperadrenocorticism)- remember these are only pruritic when there is a secondary bacterial infection or Malassezia overgrowth.

If pruritus continues after treating the secondary infections and a specific primary disease has not been not been established through physical examination and laboratory testing, or there was not a secondary infection to begin with the next step is a therapeutic ectoparasiticidal treatment (if it has not been previously performed). Glucocorticoids or oclacitinib may be used during the first week or 2 of the ectoparasiticidal treatment but they need to be stopped a couple weeks before rechecking so that it can be determined whether it was the medication or the ectoparasiticidal treatment that resolved the pruritus. If the pruritus has continued in spite of treating for infection and ectoparasites a food trial should be instituted. A home cooked diet is the gold standard and owners should be encouraged to use this diet rather than commercial diets. It is beyond the scope of this lecture to discuss the many reasons that this is true but to summarize the high point- commercial foods contain ingredients that are not labeled. In addition commercial diets have only been shown to have a 50% negative predictive value (poor at ruling out the disease).

Both the diet trial and a therapeutic ectoparasiticidal treatment can be done simultaneously. If they are done simultaneously and there is a positive response you can do a food “challenge” to determine which therapy was effective. By going back and feeding the original diet and seeing if the pruritus resumes you will be able to determine the underlying cause. A short course of GC or oclacitinib at the beginning of the therapeutic trial may be done as long as you have eliminated the presence pyoderma, Malassezia, demodex and dermatophytes.

At the end of these steps, if the pruritus has resolved w/o the concurrent administration of GC or oclacitinib, you have identified your primary cause and can treat accordingly. If the dog has residual pruritus then the dog has environmental triggered atopic dermatitis.

**Treatment of canine atopic dermatitis- overview**

As previously mentioned it is now recognized that canine atopic dermatitis has both an allergic component and a barrier dysfunction component both of these should be addressed in your treatment.

Treatment options for dogs with atopic dermatitis include - (please note that these therapies are used as a preventative so they should be instituted before clinical signs recur):

1. **Good skin care**
   a. Restore barrier function
   b. Protecting the skin
      i. Wiping the dog off after coming in from outdoors
      ii. Clipping the hair coat to a short length (10 or 15 blade) which helps to decrease exposure to and contact with environmental triggers (allergic and irritant).
      iii. Clothing all the time and boots outdoors
   c. Bathing with a hypoallergenic veterinary shampoo that contain moisturizers or barrier repair ingredients (eg shampoo that contain phytosphingosine) weekly
   d. Follow the bath w/a humectants or barrier repair product
      i. In humans moisturizers are best applied w/in 2 minutes after finishing the bath for maximum effect
   e. Fatty acid supplementation- try an omega 3 product for 3 months and if there is no improvement, try a product with a combination of an omega 3 and 6
      i. Omega 3
      ii. 18 mg/kg of EPA daily
      iii. Omega 6/3- double the bottle dose OR
      iv. High fatty acid diets
   f. Bathing is helpful to decrease antigen load and bacterial colonization

2. **Identify and prevent/manage the triggers (ectoparasites, food, infection (bacterial/Malassezia)**
   a. If the dog has environmental triggered atopic dermatitis, allergen specific immunotherapy (ASIT) is appropriate if the symptoms are present for more than 2 or 3 months/year and is severe enough to need corticosteroids or cyclosporine for symptomatic control. ASIT may be administered either subcutaneously or sublingually. See comments at the end of this article
   b. If the dog has a food trigger- avoid those foods
   c. Good flea control especially if the dog has flea bite hypersensitivity

3. **During acute flares- treating infection and inflammation is necessary.** Therapy would include antibiotics, antifungals and glucocorticoids along w/the above recommendations

4. **Treatment options for symptomatic relief of dogs w/atopic dermatitis w/o secondary infection are**
a. Glucocorticoids
   i. Advantages – quick, effective against both pruritus and inflammation including ear canal disease, inexpensive. Appropriate for short term use or when finances restrict other options
   ii. Disadvantages- numerous – well known

b. mCSA
   i. Advantages-
   ii. The author uses this drug in dogs with uncomplicated atopic dermatitis if the dog is moderately to severely pruritic and I want to avoid steroids (due to side effects or owner preference) and has failed to respond to oclacitinib
   iii. There may be a 4-6 week delay before seeing full effectiveness so you can give glucocorticoids during the first 3 weeks to help keep the dog comfortable during this lag time.
   iv. Side effects in dogs are very limited and are primarily GI. Other side effects reported include cutaneous papillomatosis and hyperplastic gingivitis. In order to minimize the most limiting factor of CSA (vomiting) I use Cerenia® or zofran (0.5-0.75 mg/kg) 30 minutes before administering mCSA. I do this for the first 4-7 days and administer Atopica® with a meal.
   v. An important drug interaction is ketoconazole (KCZ). KCZ inhibits the enzyme responsible for CSA metabolism (cP450 3A4) thereby increasing concentrations and prolonging elimination of CSA. Because of the cost of CSA, coadministration with ketoconazole has been used by some authors in DOGS. This combination with KCZ can lower the amount of CSA that needs to be administered. Doses suggested are 2.5 mg/kg of CSA and 7.5 mg/kg of KCZ sid. Please note failure to respond to this combination doesn’t mean that a full dose of CSA will be ineffective. The author therefore rarely begins therapy w/this combination. Note recently the author has seen resistant cutaneous Malassezia infections. Is this due to the indiscriminate use of KCZ orally and topically?
   vi. Dosage is 5 mg/kg sid on an empty stomach. There may be a 4-6 week delay before seeing full effectiveness so you can give GC during the first 3 weeks to help keep the dog comfortable during this lag time.
   vii. Side effects in dogs are very limited and are primarily GI. Other side effects reported include cutaneous papillomatosis and hyperplastic gingivitis. In order to minimize the most limiting factor of CSA (vomiting) I use Cerenia® for the first 4 days and administer Atopica® with a meal.
   viii. Drug interactions – the most important is ketoconazole (KCZ). It inhibits the enzyme responsible for CSA metabolism (cP450 3A4) thereby increasing concentrations and prolonging elimination of CSA. You need to be aware of this when treating Malassezia with KCZ if the dog is also on CSA
   ix. You can use this drug interaction to your advantage by using a combination of CSA and KCZ- using 2.5-5.0 mg/kg of CSA and 5-7.5 mg/kg of KCZ

c. Oclacitinib
   i. During inflammation, a variety of mediators such as cytokines, chemokines, and neuropeptides are released into the microenvironment by Th2 lymphocytes. (note in dogs w/AD there is an increase in the number of Th2 lymphocytes) Cytokines convey their information by binding to specific receptors on the cell membrane to induce a biologic response. The cytokine receptors are transmembrane receptors composed of multiple subunits. On the intracellular portion of each receptor subunit are one of 4 JAKs – JAK1, JAK2, JAK3 and TYK2.
   ii. Afferent nerves, in close proximity to the inflammation that are responsible for pruritus are activated by these mediators. They transmit signals that travel along unmeylinated C nerve fibers and are received by the dorsal root ganglia (DRG) within the dorsal horn of the spinal cord. The signal finally reaches the brain and affects regions involved in pruritus. Adjacent afferent nerves are stimulated (axon reflex) when the peripheral nerve endings of the affected area release neuropeptides (e.g., substance P, calcitonin gene-related protein, CGRP) and neurotropins (e.g., NGF). These mediators can also modulate inflammatory responses as well as directly triggering vascular responses in the skin. In the skin, cytokines regulate acute and chronic processes such as neuronal itch stimulation and inflammation.
   iii. After a cytokine binds to its cell membrane receptor it triggers specific intracellular pathways. One such intracellular pathway is the Janus kinase (JAK) pathway. Cytokines implicated in allergic skin disease (such as Interleukin IL-31 and IL-4) bind to their receptor on the cell membrane and activate the JAK pathway. JAKs activate intracellular proteins called Signal Transducer and Activator of Transcription (STAT) to induce gene transcription and biological responses
iv. What types of proteins or functional changes are produced by activation of the AK/STAT pathway? Some are 1) ↑ IgE production 2) lymphocyte proliferation 3) ↑ cytokine production 4) cytokine receptor expression 5) ↑ chemokine production 6) pruritus

v. Oclacitinib is a JAK inhibitor with more selectivity to block JAK1 than JAK2, JAK3 or TYK2. It blocks the activation and function of cells that use the JAK1 enzyme as a part of the cytokine receptor. The result is a decrease in the activity of pro-inflammatory and pruritogenic cytokines that use JAK1 such as IL-2, -4, -6, -13, -31.

1. The organ systems that are affected by the inhibition of JAK1 mainly are the epidermis, lymphocytes and the peripheral nervous system.
2. Inhibiting JAK 1 inhibits the production of IL 31. IL31, which is made principally by activated Th2-type T cells, induces production of several chemokine involved in inflammatory skin disease. These chemokines are not only involved with inflammation but also recruit to the skin IL 31 producing T cells thereby amplifying inflammation and pruritus.
3. IL 31 receptors are also present on nocieptive neurons in the dorsal root ganglion. Currently it is unclear whether IL 31 induces pruritus by directly modulating the function of sensory neurons or stimulating keratinocytes, which may induce a yet unknown keratinocyte-derived mediator that subsequently activates unmyelinated C fibers in the skin.

vi. In a review of 200 dogs treated in the author’s practice, the drug was effective in adequately controlling pruritus in about 75% of the dogs with environmental induced atopic dermatitis when used per label instructions.

vii. The dosage is 0.4-0.6 mg/kg bid x 14 then sid. Some dogs will have their pruritus increase when the dose is changed from bid to sid. Before adjusting the medication be sure to collect your minimum data base to evaluate for bacterial pyoderma, Malassezia dermatitis and ectoparasites. If the dog has uncomplicated atopic dermatitis and sid oclacitinib is inadequately controlling the pruritus the author will do the following step wise adjustments

viii. If the dog is not responding at all (or minimally) to sid then increase the dose if possible (the chart accompanying the drug has a some dogs receiving the low end of the dose while others are at the high end)

ix. In those cases that it is effective but it is not lasting all day, take the daily dose and divide it into 2 doses. The doses don’t have to be the equal – if the dog is on 1 ½ pills daily you can do 1 in the am and ½ in the pm.

x. If the above doesn’t work and you have not used modified cyclosporine you should do so

xi. Regardless of the response to oclacitinib – identifying and treating the underlying cause is the best course of action rather than just masking the symptoms.

xii. Because this drug blocks the neurogenic component of pruritus other pruritic skin diseases (pyoderma, flea allergy, scabies) may also respond to this medication. This emphasizes the importance of a thorough dermatologic examination and a minimum data base of skin scrapings and cytologies. Pruritic dogs, whether or not they are given oclacitinib, should have flea control therapy instituted.

xiii. The author is concerned cases may not have a thorough evaluation before dispensing oclacitinib and that dogs may have these other pruritic diseases present but not addressed. The author will dispense oclacitinib in the same situations as mCSA except if the dog needs instant, predictable relief, mCSA would not be appropriate due to the lag effect, while oclacitinib would be effective. Before dispensing oclacitinib the author discusses the following with the owners

1. Identifying and treating the underlying cause is the best long term therapy
2. The author has used this drug for 6 years with no serious side effects noted.

xiv. Lokivetmab (Cytopoint) is an injectable formulation containing a caninized monoclonal antibody (mAb) against interleukin-31 (IL-31). These mAb remains in circulation for several weeks. It exerts a therapeutic effect by binding to and neutralizing soluble IL-31, thus inhibiting pruritus and reducing skin lesions. Like other naturally-occurring antibodies and antibody-antigen complexes, elimination is via normal protein degradation pathways.

i. It is administered by a subcutaneous injection and is repeated monthly, as needed.

1. Some dogs may need it less than every 30 days
2. May be effective even in dogs that failed to respond to oclacitinib

ii. It is for DOGS only
iii. Long term safety and efficacy has yet to be determined. One of the issues is will dogs develop antibodies to the product since it not 100% canine autobody (contains 10% mouse).
   1. To reduce immunogenicity in dogs recombinant DNA technologies are used to engineer the antibodies to be over 90% canine in structure
   e. Antihistamines/tricyclic antidepressants – there are a variety of antihistamines available that may help mildly pruritic dogs.

5. ASIT
   a. May be administered either subcutaneously (SCIT) or sublingually (SLIT)
   b. SCIT
      i. Has a long term track record of safety and efficacy
      1. Extremity rare for life threatening reactions
      a. Will see less serve reactions that need injection modification (localized swelling, increase in pruritus, etc)

6. SLIT
   a. Recently sublingual immunotherapy (SLIT) has become available to veterinarians for the treatment of canine atopic dermatitis (cAD). The author has some reservations about the use of this therapy for cAD.
   Recognizing that SLIT has been used for many years in Europe for the treatment of human asthma we can review the information that is available in that species. The vast majority of studies and protocols in humans are for rhinitis/asthma and NOT atopic dermatitis. A review in human medicine (2006) found the following –
      i. Dosing summary
         1. The studies included doses that varied by 30,000-fold
         2. Frequency of dosing varying from daily to weekly
         3. Duration of treatment varying from 2 months to 5 years
   Their conclusion was that SLIT is an effective treatment (for rhinitis or asthma) but it was unclear what the proper dose, treatment schedule and overall duration of treatment was to be effective.
   Other review articles found that the cumulative monthly dose varied between 0.017 and >500 times the customary subcutaneous maintenance dose. In addition that each manufacturer uses its own standardization, formulation, and administration schedules. In a review of SLIT for human atopic dermatitis the authors could only find 1 double blinded, placebo controlled randomized study (DBPCR). That study evaluated the efficacy and safety of SLIT using house dust mite containing drops. They concluded that for mild–moderate disease there was significant improvement but there was no improvement in cases of severe disease. But it went on to say that standardized treatment was essential to ensure therapeutic efficacy. They used 80 umg protein concentration/day once daily with instructions for the patients to keep the drops under the tongue for 1–3 minutes and then swallow. Note in this study the treatment group had a total efficacy rate of 77.78% (cured + marked improvement) vs. 53.85% in the control group. These were statistically significant but look at the placebo effect! The other important finding was that during the first year of immunotherapy there was no difference between placebo and SLIT response and the difference was only noticeable at 2 years. In 2015 there was a systematic review to evaluate the evidence supporting the use of SLIT for hAD. They could only find 5 studies to fit their criteria. They found that in 4/5 studies there was an improvement in AD but in 2/4 there was a substantial placebo effect making the true effect of SLIT difficult to determine. They found serious shortcomings such as lack of control group, lack of randomization and data analysis was not by intention to treat. The group graded 1 of the studies to have moderate quality, 2 to have low quality and 2 to have very low quality.
   As you review the studies in veterinary medicine concerning SLIT and eAD you will note that all studies except for 1 have the same very serious limitations- they are open studies, there are no placebo groups and the studies only applies to mite sensitive dogs. Also the studies state that there are statistically significant changes in CADESI and PVAS but doesn’t state if this translated into CLINICAL improvement- for example pruritus may go from +10/10 to a +7/10- which may be statistically different but not clinically different. In the 1 DBPCR study that has been done to date in veterinary medicine, they found that overall the percentage of dogs that improved >40% were 50% in the control and 66% in the active group. Once again look at that placebo response! Two problems with this study- 1 they don’t state if the response rate is statistically different and also the criteria that has been establish states there must be at least a 50% improvement in pruritus to be considered clinically significant- so why did that study use a 40% cutoff?
   Lastly, things that give the author great pause about this whole subject is that there are some companies that refuse to tell the veterinarian what is in the SLIT formula that they expect us to give to our patients. In addition the different antigen companies are using different strengths in their SLIT (one company offers a dilution of 20,000 pnu or 40,000 pnu whichever you want – but doesn’t give guidelines how to chose), different volumes and different frequency (sid vs bid). So how can they all be effective? Discussion about dermatologist who formulate their own SLIT in their hospitals also reveals a lack of standard protocols. The author uses SLIT in very limited, specific situations such as when owners are absolutely adamant that they won’t give SCIT and won’t bring the pet in
for you to give the injection, an animal that has had a severe reaction to SCIT or if the animal fails to respond to SCIT after 1-1½ years. I tell the owner that we really don’t know how successful this method is but that it is very safe to try.

Summary from the ACVD task force on AD

Treatments of acute flares of canine atopic dermatitis

1. Identification and avoidance of flare factors:
   a. Identification and elimination, whenever possible, of allergenic flare factors (fleas, food and environmental allergens)
   b. Evaluation of use of antimicrobial therapy if clinical signs of infection or colonization with bacteria or yeast are present on the skin or in the ears

2. Improvement in skin and coat hygiene and care:
   a. Bathing with a nonirritating shampoo

3. Reduction of pruritus and skin lesions with pharmacological agents:
   a. Treatment with topical glucocorticoids, especially for localized lesions, as needed to control signs
   b. Treatment with oral glucocorticoids, especially for widespread or severe lesions, as needed to control signs

Treatment of chronic canine atopic dermatitis

1. Identification and avoidance of flare factors:
   a. Dietary restriction-provocation trials in dogs with nonseasonal signs
   b. Implementation of an effective flea control regimen in areas where fleas are present
   c. Performance of allergen-specific intradermal and/or IgE serological tests to identify possible environmental allergen flare factors
   d. Possible implementation of house dust mite control measures, if relevant and feasible
   e. Evaluation of use of antimicrobial therapy if signs of infection or colonization with bacteria or yeast are present on the skin or in the ears

2. Improvement in skin and coat hygiene and care:
   a. Bathing with a nonirritating shampoo or an antiseborrheic/antimicrobial shampoo, depending on the skin lesions seen
   b. Dietary supplementation with essential fatty acids

3. Reduction of pruritus and skin lesions with pharmacological agents:
   a. Treatment with topical glucocorticoids or tacrolimus, especially for localized lesions, as needed to control signs
   b. Treatment with oral glucocorticoids, cyclosporine or subcutaneous interferon, especially for widespread or severe lesions, as needed to control signs. These agents would not normally be combined together.
   c. Use of steroid-sparing agents, such as essential fatty acids, Chinese herbs and antihistamines, if glucocorticoids are being used as a long term treatment option.

4. Implementation of strategies to prevent recurrence of signs
   a. Avoidance of known flare factors, as identified above
   b. Consideration of preventive pharmacotherapy, if feasible and relevant
   c. Implementation of allergen-specific immunotherapy, if feasible. This can be used alongside all the above treatment options in an attempt to provide long term amelioration of the aberrant immune response

5. When should you consider antihistamines, steroids, cyclosporine, Oclactinib, lokivetmab, ASIT, SLIT for the pruritic atopic dog without infections (should of course do good skin care and flea prevention as mentioned above)
   a. Each of the treatments have advantages and disadvantages which I will try to summarize below
   b. Antihistamines/EFA
      i. I would use in mildly pruritic dogs
      ii. Safe, inexpensive, available OTC

iii. No age restrictions—very few contraindications
iv. Possibly effective in mildly pruritic dogs
v. No advantage of using second generation antihistamine (eg Loratidine)
vi. For optimal efficacy, this class of drugs are best used as preventatives before a flare occurs—not during or after it—and they should preferably be given on a continuous daily basis.

c. Steroids
i. I would use if limited budget and the dog has seasonal symptoms without concurrent infection
   1. Appropriate for acute flares of uncomplicated atopic dermatitis (pruritus only)
ii. Inexpensive, rapid onset and very effective
   1. Only drug effective for decreasing the swelling in ear canals
iii. Should have cbc, serum chemistry profile and a urinalysis if on steroids for >6 months
iv. May be effective for managing otitis
v. Effective as a transition drug during the lag phase of CSA or the lag phase of ASIT/SLIT
vi. Numerous short term and long term side effects
   1. Oral and topically administered steroids are also detrimental for the epidermal barrier due to multiple mechanisms including decreased lipid synthesis, decreased epidermal proliferation and differentiation and decreased production of antimicrobial peptides
   6

   
   d. Cyclosporine
i. Consider if Oclacitinib is ineffective and the dog is a chronic steroid dog or doesn’t tolerate steroids
ii. Long track record of use
iii. No age restrictions
iv. Can be used concurrently with steroids
v. Can be used in diabetics
vi. Effective in 50-60% of the cases
vii. Because of the slow onset of action
   1. Need to have concurrent steroid or Oclacitinib for the first 3 weeks or so
   2. Slow onset of action of makes them unsuitable for managing acute flares of AD
viii. Relatively safe—can predispose to gingival hyperplasia and systemic fungal infections (rare).
   1. Predisposes to papillomas
ix. May cause GI side effects—especially initially—use cerenia or zofran for first week or so
x. If on >6 months, should have cbc, serum chemistry profile q 6-12 months depending on dosing and frequency of administration
xi. May be expensive if needed in large breed dogs—at full dose daily
   1. May be able to decrease frequency of administration to q 48 hrs or even 2 times weekly
a. May be able to lower the daily dose if less frequent administration is not adequate
xii. Ineffective in treating or preventing otitis

   
   e. Oclacitinib
i. I use if a dog needs instant relief and I am trying to avoid steroids (preparing for IDT, hx of chronic steroids, etc)
ii. If I am going to treat an elderly dog who has atopic dermatitis (not enough time for ASIT to be effective)
iii. Labeled for allergic dermatititis not just environmental allergen induced atopic dermatitis
iv. If I am going to treat a dog long term with symptomatic therapy only
v. Rapid onset, minimal side effects
vi. Can be used in diabetics
vii. Can be used prn for pruritus
viii. More expensive than steroids
ix. Limited long term studies but appears to be well tolerated
   1. Predisposes to papillomas


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x. Weight gain w/o polyphagia or change in caloric intake
   1. May be because of inhibiting STAT -
      a. In humans STATs tend to promote lipolysis in mature adipocyte- so inhibiting
         STAT will prevent lipolysis
xii. Ineffective for treating or preventing otitis
   1. Should not be used in animals <12 months of age, or pregnant or lactating animals

f. Lokivetmab
   i. For dogs where owners can’t/won’t give oral medication
   ii. For dogs that need instant relief and steroids or olacitnib is contraindicated (pyoderma, demodicosis, age, etc) or has been ineffective
   iii. Rapid onset
   iv. No age restriction
   v. Can be used in combination with any other therapy
   vi. Parental administration= no need for owner to medicate
   vii. Minimal side effects but limited time frame
   viii. ONLY label for atopic dermatitis
   ix. Expensive especially in large dogs
   x. Needs to be repeated every 14 (off label) to 60+ days
   xi. Ineffective for treating or preventing otitis

g. ASIT
   i. Safe, long track record
   ii. Can cure/long term remission
   iii. Avoids oral administration
   iv. However, owners need to learn to give injections or take the dog to the veterinarian
   v. Can prevent otitis, pyoderma and recurrent Malassezia
   vi. Can be used in combination with any other therapy
   vii. Maybe more expensive than other options

h. SLIT
   i. Consider if the dog has had a reaction to ASIT, owner has needle phobia or dog has failed ASIT
   ii. Safe
   iii. May be effective in cases where ASIT failed
   iv. No good EBM studies documenting effectiveness
   v. No long term studies
   vi. Has to be administered sid or bid
Canine Cutaneous Adverse Food Reactions: Diagnosis and Treatment
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Initially, the ACVD task force on canine atopic dermatitis (cAD) defined cAD as a genetically-predisposed inflammatory and pruritic skin disease, most commonly associated with IgE antibodies to environmental allergens. cAD has now been recognized as a multifaceted disease associated with exposure to various offending agents such as environmental and food allergens. This author believes that the later definition should be used when discussing cAD.

It is important to remember that there are many other causes for pruritus in the dog other than cAD such as ectoparasites, cutaneous neoplasia (epitheliotropic lymphoma), bacterial pyoderma, etc. so canine pruritus is not always due to cAD.

In veterinary medicine the criteria for diagnosing cAD has evolved over time. Historically 1 of 2 sets of criteria have been used for making the diagnosis of cAD. The problem with these previous criteria is the former was never validated while the later had a limited sample size. The most current guideline was proposed by Favrot. Please note that before applying these criteria to a pruritic dog, other causes of pruritus, such as ectoparasites or infectious causes, need to be ruled out. You shouldn’t use these criteria alone to make a diagnosis of cAD. History, physical examination, diagnostic testing and response to treatment should also be included in your evaluation.

The criteria used to establish a diagnosis of cAD include:

1. Onset of signs under 3 years of age
2. Dog living mostly indoors
3. Glucocorticoid-responsive pruritus
4. Pruritus sine materia at onset (i.e. alesional pruritus)
5. Affected front feet
6. Affected ear pinnae
7. Nonaffected ear margins
8. Nonaffected dorso-lumbar

Using these criteria, if 5 criteria are matched, and ectoparasites and infectious causes have been ruled out, the sensitivity and specificity are about 85% and 79% respectively. This means that using only these criteria, a wrong diagnosis will be made about 20% of the time.

Once you have established a diagnosis of cAD it is important to identify triggers that may cause the cAD to flare up. Triggers include:

1. Environmental allergens
2. Food allergens
3. Ectoparasites
4. Infectious (bacterial, *Malassezia*)

This lecture is going to focus on food allergens as the trigger.

Food allergy (FA) is recognized as a potential cause of various dermatological and gastrointestinal (GI) signs in the dog and cat. The exact incidence of FA is unknown. However, the term “allergy” is often used indiscriminately. Acquaintance with exact terminology is important when dealing with FA.

An adverse food reaction (food sensitivity) as defined by the American Academy of Allergy and Immunology and the National Institute of Allergy and Infectious can be divided into two categories: immunological and non-immunological reactions. Food allergy

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Common confounders of dietary elimination trials contain the antigens soy, pork, and beef. J Am Anim Hosp Assoc 50(5): 1340-51


Regardless of what type of diet is used to diagnose CAFR there are several potential pitfalls to avoid. A common mistake made during food trials is using flavored heartworm preventative. This was reported in an abstract in which there were 12 dogs with natural occurring CAFR to either soy or corn. The author fed a flavored heartworm preventative (Interceptor) fed to each dog. This preventative contained pork liver and soy. A clinical score (CS) was assigned based on the severity of skin and otic disease. After 1 pill 10/12 dogs had an increase in CS. In 5/12 dogs the values peaked on day 2 post challenge while in 5/12 dogs it occurred on day 5.

Another problem is the use of supplements or medications during the food trial. In a study the authors tested 7 supplements for the presence of soy, pork, or beef antigens. Three were flavored OTC products and 4 were veterinary therapeutics. All OTC test products produced ELISA results in agreement with their ingredient lists. ELISA testing of veterinary therapeutic products did not agree with either their ingredient lists or product inserts because of the presence of other ingredients not listed. In 1 product, the "artificial beef flavor" was made using pork liver and 1 arthritis product listed "natural flavors" which was determined to be a spray-dried digest derived from pork liver. Another potential problem identified was administering supplements/medications that were in gelatin capsules. This is because the gelatin is derived from beef or pork. This lead the authors to recommend that veterinarians contact manufacturers of oral therapeutics prior to prescribing them during a dietary elimination trial to determine the other ingredients in those products that may not be listed on the ingredient list or product insert.
Mislabeling is not limited to supplements. A study\textsuperscript{15} was done using 12 dog foods (eleven novel protein diets and one hydrolyzed diet) from five different manufacturers, both international and Italian, for potential contamination by animal origin ingredients that were not mentioned on the label. The food was analyzed using both the official method (microscopy) to identify bone fragments of different zoological classes (mammalian, avian and fish) and by polymerase chain reaction (PCR) for the identification of DNA of animal origin. In 2/12 samples the results of both analyses match the ingredients listed on the label. In the remaining 10 samples, microscopy detected bone fragments from 1 or 2 unlabeled zoological classes. In 6/10 samples, there were undeclared avian fragments, 5/10 had fish and 4/10 had mammalian fragments. In two samples, microscopy analysis identified a contamination that would have otherwise passed unobserved if only PCR had been used. However, PCR identified the DNA of undeclared zoological class in 2 samples. The conclusion by the authors was that dogs might fail to respond to commercial limited antigen diets because such diets are contaminated with potential allergens. Both PCR and microscopy analysis are required to guarantee the absence of undeclared animal sources in pet foods. Lastly a study by Okuma et al collected 52 commercial dog and cat food products from southern California and on line. They tested the foods for the presence of eight meat species (bovine, caprine, ovine, chicken, goose, turkey, porcine, and equine) using real-time polymerase chain reaction (PCR).\textsuperscript{16} Of the 52 products, 31 were labeled correctly, 20 were potentially mislabeled because they either (1) contained meat species that were not included on the product label (16) and/or (2) did not contain meat species that were included on the product label (7) - note some food had both problems. One food contained a non-specific meat ingredient that could not be verified. Pork was the most common undeclared meat species detected. There was also a trend to substitute lower cost ingredients, such as poultry meats, for higher cost ingredients, such as beef and lamb. These studies support the position that before ruling out an AFR, a novel protein home-made diet trial should be performed.

A retrospective study added additional evidence to support the statement that a homemade diet is superior to commercial diets in diagnosing CAFR\textsuperscript{17} In this retrospective study reporting CAFR in cats, the author evaluated cases presented to a dermatology referral service for possible CAFR. Seventeen cats were diagnosed with having CAFR. Home prepared elimination diets were completed by 16 cats; 8 cats with a final diagnosis of CAFR failed to respond to a minimum 6-week commercial hydrolyzed protein diet but did respond to the home-made diet. Of the 13 cats in which their final dietary management was reported, 6 cats could not tolerate any commercial dry foods, but did tolerate select canned foods; 7 cats could consume commercial dry foods, with 4 maintained on commercial hypoallergenic diets and 3 with other commercial restricted protein diets.

As previously discussed, an appropriate elimination diet should contain 1 new, highly digestible protein or a diet that contains hydrolyzed proteins. Ideally a homemade diet (HMD) should be fed. This is the type of diet the author uses. A HMD consists of one novel protein and one novel carbohydrate. The protein usually is rabbit, venison, goat, ostrich, emu or alligator. White or sweet potatoes, oats, quinoa or rutabaga are appropriate carbohydrate sources. It is mixed 1 part meat and 3 parts carbohydrate and the dog is given 1-2 cups of the mixture/10#. HMDs should not include ANY other ingredients. The dog must not ingest any other food, treats, tidbits, etc. including items used to hide medication in. Avoiding gelatin capsules should be attempted. This may be difficult because some medications only come in a capsular form (e.g. modified cyclosporine). The problem with HMDs is that they are nutritionally inadequate for growth and maintenance therefore they are not using in growing dogs or for long term maintenance. Because they are not very calorically dense most animals will lose weight on these diets. If a dog has a body score of 4/9 or less, this author does not use a HMD. Although a HMD is not nutritionally balanced nor complete, supplements are not necessary, nor used, during the short test period. When a HMD is given during a prolonged time, it is recommended to consult a veterinary nutritionist to formulate a balance diet.

Although the gold standard for diagnosing CAFR is a HMD there are circumstances where the author will use a commercial diet instead. Examples include owners who will not cook for the dog, if the dog doesn’t tolerate HMDs (typically because of weight loss but some dogs will become lethargic on them or have GI disturbances). They are not fed to growing dogs.

Commercial novel protein diets (NPDs) can be used to diagnosis CAFR and can be used long term to maintain a dog with CAFR. A variety of NPDs are available for dogs. These diets are readily available but do not have a 100% negative predictive value (false negatives occur 25-50% of the time). Several studies have demonstrated the problems associated with NPD. In the first study, they fed dogs with proven CAFR either venison/rice, chicken/rice or catfish/rice commercial dog food.\textsuperscript{18} When fed the venison dog food 85% of the dogs with CAFR reacted while 52% and 47.5% reacted to chicken and catfish dog food respectively. More recently 3 of 4

\textsuperscript{16} Okuma T.A., Hellberg R.S. Identification of meat species in pet foods using a real-time polymerase chain reaction (PCR) assay. Food Control, 2015; 50: 9

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over the counter (OTC) dog foods that didn’t list soy on their ingredients list had soy identified via ELISA testing. More disturbing
was the study that reported 3 out of 4 OTC dog foods that specifically stated “NO SOY” had soy found when ELISA testing was
performed. Note that in the same study 2 of 3 hydrolyzed soy diets had intact soy identified.

Commercial hydrolyzed protein diets (HPDs) contain proteins that been enzymatically hydrolyzed to smaller molecules. This
reduces the MW of the original protein which leads to a decrease in the antigenicity and allergenicity of the protein. This means that
the molecules are too small to evoke a cross binding between IgE on the surface of the mast cell. This prevents degranulation of the
mast cell and IgE-mediated (Type I) hypersensitivities. This is a key point because if the CAFR in that dog is not caused by IgE (which
is believed to be the more common scenario) but by some other mechanism (e.g. type IV which is a T cell driven disease) the size of
the molecule doesn't not matter and the diet will be ineffective. The optimal MW of a protein hydrolysate in dogs has not been agreed
upon. Hand et al states that an ideal molecular weight of less than 10,000 Daltons, while Cave states that if the protein size is reduced
to less than 6,000 Daltons in size, it should reduce binding to IgE and increase digestibility. However, Verlinden et al. states that
peptides over 4,500 Daltons could still can start the immunologic reaction which contributes to the allergic reaction. In addition,
diets are only partially hydrolyzed. This means that only a percentage of the protein is hydrolyzed- there is still some intact
protein remaining. In the humans, peptides with a MW as low as 3000 Da are still capable of an allergic reaction. Free AA are not
allergenic, but are not suitable in foods because of their bitter taste, high osmolarity (leading to diarrhea) and very high costs. As with
the NPD, HPD are not able to diagnose CAFR in all dogs- they probably miss about the same percentage as the NPD.

Recently a study evaluated whether the molecular weight of peptides present in three different hydrolysed foods [Royal Canin
Ultimino (Royal Canin; Aimargues, France), Purina HA (Nestle Purina PetCare; Meaux, France) and Hill’s z/d Low Allergen (Hill’s
Pet Nutrition; Sophia-Antipolis, France)] matches with the producers’ claims and if peptides are recognized by IgE. The results
showed that in all 3 diets there were several high molecular-weight (HMW) proteins ranging from 15 kDa to 60 kDa. This study
further demonstrates that even highly hydrolysed hypoallergenic foods may still contain immunoreactive HMW proteins. Further
studies are required to determine the clinical relevance of these findings but it is a concern that these HMW proteins may impact the
success rates of some elimination diets.

Regardless of which diet is used there are a few points to discuss. The first issue is how long to feed the diet. The following is
based on the best evidence available as of December 14, 2014 and is information gathered from 209 dogs with CAFR. After 3 weeks
on the diet approximately 50% of the dogs will have achieved a complete or marked reduction (>50%) of pruritus. After 5 weeks 85
% of dogs had responded partially or completely and by 8 weeks >95% had responded. Less than 5% needed 9-13 weeks.
Information gathered from 40 cats with CAFR revealed that it took approximately 4 weeks (50% of the cats), 6 weeks (80%) and 8
weeks (90%) on the diet to achieve a remission. As in dogs, by 13 weeks 100% had either partially or completely responded.
Remember if at any point the pruritus has completely resolved, the diet can be “challenged” at that time, there is no need to extend the
special diet any further.

Veterinarians are frequently asked by owners which ingredients cause the most reactions? The answer depends on the study. In
265 dogs reported collectively by 12 different studies, beef, dairy products, and wheat accounted for two thirds of reactions.
Reactions to corn, pork, rice, and fish were rarely reported in dogs. In the April 2013 issue, Veterinary Dermatology a letter to the
editor reported the most common ingredients causing CAFR in 330 dogs- beef, dairy, chicken and wheat accounted for 78% of the
reactions. Of 56 cats reported collectively by 10 studies, beef, dairy products, and fish accounted for 80% of reactions.

More recently a literature search (limited to 1985-2015) for canine or feline food allergy in CAB Abstracts and Web of Science
revealed that of the 297 dogs included in the selected studies the most frequently reported food allergens involved in dogs were beef
(102 dogs, 34 %), dairy products (51 dogs, 17 %), chicken (45 dogs, 15 %), wheat (38 dogs,13 %) and lamb (14, 5 %). Other less
commonly reported offending food sources were soy (18 dogs, 6 %), corn (13 dogs, 4 %), egg (11 dogs, 4 %), pork (7 dogs, 2 %), fish
and rice (5 dogs each, 2 %). In cats the food sources most frequently causing CAFR in were beef (14 cats, 18 %), fish (13 cats, 17 %),
chicken (4 cats, 5 %), wheat, corn and dairy products (3 cats each, 4 %) and lamb (2 cats, 3 %). Egg, barley and rabbit were also
reported as offending allergens in individual cats.

20 Willis-Mahn, C., et al. (2014). ELISA testing for soy antigens in dry dog foods used in dietary elimination trials J Am Anim Hosp Assoc
The problem with any of these retrospective studies is that the offending allergens listed reflects pet feeding habits in the preceding decades, and these allergens could change once new pet foods become fashionable and used more frequently. Note that many owners believe that food additives (dyes and preservatives) are common causes of food allergy in dogs, yet there has not been even 1 published case report documenting this.

Maillard reactant products are formed when proteins are cooked with carbohydrate. They can increase or decrease the allergenicity of proteins, depending on the food component. This phenomenon may explain the apparent increase in allergenicity of proteins in commercial pet foods compared to fresh proteins. Because of this, the author suggests that when preparing the HMD, the protein and carbohydrate should be cooked in separate pots.
Protocols are useful in helping to diagnose and treated many different disorders. Part of any good protocol should be a minimum data base (MDB). In addition to signalment, history, etc in veterinary dermatology, laboratory testing should be a component of this data base. Just as you may have a standard set of tests for diarrhea you should have a standard set of tests for dermatology cases. Because practitioners get busy, sometimes collection of this minimum data base is overlooked. By training technicians to perform the tests this potential problem can be avoided. Instructing technicians to perform these tests on every pruritic animal ensures that this will be done on every case.

Tests can be separated into immediate and delayed tests. For a pruritic dog or cat all the immediate tests should be performed. Which of the delayed tests should be performed will vary based on the results of these tests.

Immediate tests include
1. Skin scrapings **
2. Impressions smears **
3. Ear cytologies ** if ear disease is present
4. Fine tooth combing **
5. Hair plucks/trichograms

Delayed tests would include
1. Skin biopsies
2. Woods lamp and fungal culture
3. Bacterial culture and susceptibility
4. CBC, serum chemistry profile and urinalysis
5. Adrenal function tests
6. Thyroid profile
7. Dietary elimination food trial
8. Intradermal testing (or serum testing) and allergen specific immunotherapy

** Component of MDB

Equipment
The equipment needed is very basic and include
1. #10 scalpel blade- dulled by scratching the frosted part of a glass slide
2. Mineral oil
3. Frosted glass slides and cover slips
4. Clippers
5. Microscope
6. Minitip culturettes
7. Needle and syringes
8. Woods lamp +/- derm duet
9. Punch biopsy
10. Lidocaine/bupivicaine/sodium bicarbonate

Skin scraping
Let’s begin with skin scrapings. Before performing skin scrapings you should ask the following questions
1. What technique do I use (broad superficial or deep scrapings or both)
2. Where do I need to skin scrape?
3. What lesions should be scraped?

The answers to these questions depend on which parasite you suspect. If you suspect a superficial mite (Sarcoptes, Notoedres, Demodex gatoi (cats), Demodex cornei (dogs) Cheyletiella) then broad superficial scrapings should be performed. Deep skin scrapings should be performed when Demodex canis or cati is suspected. (Table 1)

When performing superficial scrapes be sure to scrape from appropriate areas. For Sarcoptes you will be more successful if you scrape pinnal edges, the elbows, ventral chest and hocks. In addition any popular, crusted or erythematous lesion should be scraped.
For any of the superficial mites, broad scraping should be performed. Remember that mites associated w/hypersensitivity (eg *Sarcoptes*, *Cheyletiella*) may be difficult to find due to their low numbers so be sure to take multiple (10-15) sites. In contrast to demodex, all scrapes can be placed on 1 or 2 slides because the quantity of mites present is not important, they are either found or not.

When performing a deep skin scrape for demodex (this applies mostly to dogs) there are a few pitfalls to avoid. By avoiding these errors the diagnosis and your management of demodex will improve.

These include

1. Failure to squeeze the skin prior to scraping. Squeezing helps express the *Demodex* from the hair follicles
2. Failing to record location of scrapes;
3. Failing to record numbers & stages present;
4. Failing to record whether the mites are alive or dead;
5. Failing to clip hair at skin scrapings sites (if it is a recheck appointment, the hair may be regrowing preventing proper sample collection);
6. Failure to squeeze the skin prior to scraping to try
7. Failure to recognize that lesions that are granulomatous & fibrotic, especially on the paws may have demodex that are hard to demonstrate on skin scrapings and a skin biopsy may be necessary to diagnosis;
8. Failure to sedate dogs if the feet are to be scraped
9. Failing to scrape hyperpigmented areas even if they are not alopecic;
10. Failing to scrape areas with comedones even if they are not alopecic
11. Failing to scrape if a dog only has greasy seborrhea (especially along the dorsum). A long body type of demodex mite has been identified (*Demodex injai*). This mite lives in the sebaceous glands of the dog's skin, and thus, is commonly associated with "greasy coats" rather than the moth eaten or pustular appearance that we are used to seeing.
12. Failing to take broad superficial skin scrapes even if demodex is the only parasite you suspect. There is a short bodied demodex mite (*Demodex cornei*), which lives on the surface of the skin layer. Note that there may be a low number of these mites found because of the superficial location of the mites allowing removal by the animal.

**Cytology**

Cytologic examination is another very commonly performed procedure in dermatology that should be performed on any dog or cat presented w/skin or ear disease. Cytology is used to identify the presence (and/or type) of:

1. Bacterial or fungal organisms (*Malassezia*);
2. Neoplastic cells;
3. Inflammatory cells;
4. Abnormal cells (eg acantholytic keratinocytes associated w/pemphigus foliaceus)

When the skin is scaly, a superficial skin scraping can be useful. A very small amount of mineral oil is placed on a #15 scalpel blade to help keep the scale on the blade once it has been collected. The lesion is scraped a few times, and the material collected is placed on a microscope slide, stained (see below about staining samples), and examined microscopically at 40X and 100X.

Direct smears can be collected by a variety of ways.

Impression (touch) smears are useful when there is an erosion, ulcer, crust, moist or greasy lesion. To perform an impression smear, a slide is firmly applied to a lesion and, in most cases, is then gently moved back and forth a few times to increase the yield. Some people will use slides that are “sticky” on one side. These slides are reported to increase the yield of sample collected but the author finds that a standard slide works quite well. The slide is then processed and examined as described below.

If the lesion is fluid filled (eg pustule, papule) but is too small for a fine needle aspirate, “lance” the lesion with a 25 gauge needle, gently squeeze the lesion and then do an impression smear of any material expressed. When sampling crusts, lift the crust and rub both the underside of the crust and the surface of the skin.

Roll smears (swabs) are used when it would be difficult to get a slide into the affected area. This could be the face fold, the interdigital space on cats and small dogs and the ear canals in all dogs and cats. A cotton tipped applicator is gently rubbed back and forth across the lesion and then the material from the applicator stick is rolled back and forth on the slide. If the lesion is scaly, applying a small amount of mineral oil to the swab can help with collection. The sample is rolled onto a microscope slide, stained and examined as previously described.

A fine needle aspirate is performed when a solid or fluid filled mass or lesion is present. A 22-25 gauge needle attached to a 12 cc syringe is placed into the lesion and suction is applied by pulling back the plunger of the syringe (½ to ¾ of the way). The syringe plunger is pulled back and released a few times. Don’t aspirate aggressively enough that you get blood contaminating the sample (you should not see blood in the hub of the needle). After aspirating one spot, stop aspirating and redirect the needle in the mass w/o pulling out and repeat the aspiration. This can be repeated 2 or 3 times on each sampling attempt. The needle is disconnected from the syringe, the syringe is filled w/air and the needle is placed back on the syringe. The material is then ejected from the needle by
compressing the plunger. If the lesion is a fluid filled you only have to pull back far enough to get a sample into the syringe. Note-
measuring and noting the location of the masses is valuable for monitoring progression and/or response to treatment.

Regardless of the collection technique (except when using the tape prep) historically the author would heat fix the sample, using a
cigarette lighter, and then wait a minute or so to allow it to cool. The slide was then stained w/a modified Wright stain (Diff Quik®).
There are 3 jars in the Diff Quik® kit. The first jar is a fixative containing methanol, the second contains buffered xanthene dye,
which stains the cells and organisms red and the third contains a buffered thiazine dye (methylene blue) which stains the cells and
organisms purple. After drying, the slide would then be examined.

A more rapid and equally effective method is to bypass the fixative step and the second step (eosin) and directly go to the
3rd step using the methylene blue only. It doesn’t appear to hinder the identification of bacteria, yeast or inflammatory cells except for
eosinophils. If using the tape prep I will put a drop on stain on the slide and then place the tape, sticky side down, over the stain
and examine.

Ear cytologies are performed to identify mites, infectious agents and inflammatory cells. A cotton tip applicator is used to collect
the samples prior to instituting therapy. Results of the cytology help direct appropriate therapy (presence of infectious agents would
indicate the need for antimicrobial therapy). I will also perform ear cytologies during therapy if either the ear(s) are not responding to
treatment OR if there were rods or WBC’s on the initial cytology regardless of the appearance of the ear. If the initial cytology
revealed yeast and/or cocci and the looks normal at the recheck examination I don’t cytology it since I don’t expect to sterilize the ear
canal- in fact the treat for eliminating certain bacteria (eg enterococcus) may be just discontinue the antibiotic and allow restoration of
the normal microbiome.

A few tips when examining your sample.

1. For skin cytologies
   a. For bacteria look in 10 fields and record a range (eg 0-5, 5-10, 10-20 etc) – be sure to note if they are cocci or
      rods, if WBC’s are present or not and if the bacteria are intracellular or extracellular
   b. For Malassezia look in 20-25 fields (unless they are ID sooner). Report them as negative/+0 if NO
      Malassezia is found, +1 if 1 or 2 organisms are found (total #) in all the fields examined and there were never
      more than 1 in a field, report a +2 if there are more than 1 organism in a field or 1 organism q 3-4 oil fields –
      treat any case w/a +2 and consider treating even if +1. In fact the ACVD now recommends either reporting
      Malassezia as either present or absent.

2. For ear cytologies
   a. There is no universal agreement as to what are normal number of cocci or Malassezia from an ear cytology
      i. Because the host reaction to the organism is as important as the number, ANY organism seen in a
diseased ear will be treated as part of the therapy regardless of the number present
   b. Inflammatory cells or rod shaped bacteria are never present in a normal ear.

Fine tooth combing
Combining of the hair with a fine tooth comb (“flea comb”) is a method that can be useful in finding fleas and other ectoparasites (ticks,
lice and Cheyletiella). You may also detect military lesions on cats that were not appreciated on your physical examination.

Trichogram (“hair plucks”)
Veterinarians are frequently presented w/animals that have hair loss. In establishing the diagnosis of the hair disease, signalment,
history (constitutional signs present or not?) and physical examination (eg pot belly, enlarged liver, etc) are important components
in establishing a diagnosis. There are times that even w/this information the cause of the alopecia has not been established. A
trichogram, which is a microscopic evaluation of plucked hairs, may be a useful tool to help identify the underlying cause.

If the alopecia is post traumatic (pruritus) or due to fragile hairs (eg dermatophytosis) the distal end of the hairs will be broken (or
if the dog/cat gets haircuts). If the hair loss is spontaneous (eg endocrinopathy) the tips are tapered.

Hair plucks can also be useful in ruling in (but not ruling out) demodicosis. Other ectoparasites may also be identified such as
Cheyletiella or lice.

 Follicular cast can also be identified w/hair plucks. Follicular casts refers to the accumulation of keratin debris that adheres to the
hair shaft as it extends out of the hair follicle. This finding indicates a follicular keratinization disorder which occurs w/vitamin A
responsive dermatosis (rare- but if occurs would be a Cocker Spaniel most likely), follicular infections (demodex, dermatophyte,
bacterial), Malassezia dermatitis, sebaceous adenitis, endocrinopathy (hyperadrenocorticism, hypothyroidism) or primary seborrhea
such as ear margin seborrhea.

Skin biopsies
Skin biopsies are an easily performed outpatient procedure. The author will perform a skin biopsy for:

1. Any skin disease that is not responding to what should be effective therapy;
2. Any skin disease that may be potentially neoplastic;
3. Any skin disease that may be a cutaneous marker for a systemic disease (e.g., hyperkeratotic footpads associated with metabolic epidermal necrolysis);
4. Any skin disease that may be autoimmune or immune mediated;
5. Any nodular disease;
6. Any skin disease that appears unusual;
7. Any skin disease that requires expensive or potentially toxic therapy

The 2 methods used to biopsy the skin are the punch technique and the elliptical, incisional biopsy.

For punch biopsies, the author usually will use a 6 mm punch biopsy instrument. When using this instrument, DO NOT include normal tissue in the sample—only the lesion. If biopsying the edge of a lesion then perform an incisional biopsy.

The author uses elliptical, incisional biopsy with a scalpel blade for lesions that are alopecia, ulcerated, erosive or are suspected to involve the subcutaneous tissue (e.g., panniculitis). For subcutaneous lesions, a punch sample may not get subcutaneous tissue and therefore may miss important lesions. This type of biopsy has one end of the sample in normal tissue and 1 end in the middle of the abnormal. The biopsy should be elliptical and request the laboratory to section the sample from tip to tip. This technique allows the evaluation of the formation of the lesion—from normal to very affected skin—it allows a “story to be told” about the lesion.

Sites should NOT be shaved or scrubbed prior to collection since this may remove very valuable information. The hair may be partially clipped to visualize the lesion better, but in order to avoid traumatizing the skin, at least ¼ inch length of hair should remain.

**Bacterial cultures**

In the past, bacterial cultures were not frequently performed in dogs with skin disease since *Staphylococcus intermedius* was the most common bacterial pathogen and had a predictable susceptibility profile. Unfortunately it isn’t that simple any more. *Staphylococcus intermedius*, *Staphylococcus pseudointermedius*, *Staphylococcus lugdunensis* or *Staphylococcus delphini* *Staphylococcus schleiferi* subsp. *Schleiferi*, *Staphylococcus schleiferi* subsp. *coagulens*, and *Staphylococcus aureus* all w/variable susceptibilities (methicillin resistant, multidrug resistant, combination) are now associated w/pyoderma in dogs. The need for bacterial culture and susceptibility testing in the dog or cat has become more frequent. Indications for bacterial culture would include the presence of:

1. Nodules;
2. Deep draining tracts;
3. A bacterial infection of the skin (confirmed by identifying intracellular bacteria and degenerative neutrophils) that fails to respond to appropriate antibiotic therapy;
4. Suspicion of an uncommon bacterial infection (atypical mycobacteria, nocardia, actinobacillus);
5. Suspicion of an anaerobic infection (gas pocket formation);

A few tips when dealing w/a bacterial culture (see table 1 for more details)

1. Use a Mini-Tip Culturette (Becton Dickinson Microbiology Systems) to pin point the sample
2. Taking samples from 2 or 3 lesions (if possible) will increase the likelihood of identifying all pathogens
3. Do cytology concurrently
4. When selecting a lesion to culture – from best to worse - pustule >papule>crust>epidermal collarette
5. If you are sampling a crust- lift the crust and swab the underside of the crust and the surface of the skin under the crusts with a the culturette.
6. For an epidermal collarette: lift the edge of the collarette- if you are not able to do this then clip the hair w/scissors to expose the collarette then take a the culturette swab and gently roll it across the collarette 3 to 4 times.
7. Have the lab do susceptibility testing use the tube dilution (MIC) rather than disc diffusion (Kirby-Bauer)

**Wood’s Lamp examination and fungal culture for dermatophytes**

Dermatophyte infection is a common problem in cats and young animals of all species. Proper collection of the specimen is critical in identifying this infection. The first step is to examine the animal with a Wood’s lamp. You should let the Wood’s lamp warm up for at least 10 minutes, and then shine the light on the hair coat looking for apple-green glow to the entire hair shaft. Remember crusts may glow as may some topical medications. A positive test is suggestive of dermatophytes, but you need to culture the hair to confirm this. Please note that a negative test does not rule out dermatophytosis, in fact you should only use the lamp to guide in selecting hairs to pluck for culture not as a tool to rule out dermatophytosis.

Prior to collection, the suspected skin lesion should be gently cleaned if grossly contaminated. Mild soap (not antimicrobial) and water may be used. Allow the site to dry before collecting the sample. Using a sterile hemostat, you should pluck the hairs near the base so that you can get close to the bulb. Also scrape a small amount of scale/crust from the edge of the lesions. This will increase the success rate of identifying dermatophyte infections. If there are diffuse lesions or you are screening a cat for infection, a Mackenzie toothbrush method is used. To perform the toothbrush method, take a sterile toothbrush and rub it over the entire lesion.
from the margins to the center. Then take a sterile hemostat and remove the hairs/scale from the tooth brush and inoculate the culture plate.

Once a media is inoculated, close the cover and place the culture plate in a plastic bag or “pencil box” with a sponge to prevent dehydration of the media which can inhibit growth of organisms. In contrast to previous recommendations the sample does not need to be placed in a darkened area and it doesn’t need to be incubated- it should be left at 77-86O F. PUT IT IN A PLACE WHERE IT WILL BE EXAMINED DAILY.

If submitting to a reference lab, just take the sample and place it in a red top tube and send that to the reference lab.

If you are doing the culture in house, be sure to check it DAILY and record the findings. It is important to note when the media changes color w/respect to colony growth. A large amount of growth w/small color change (contaminant) is interpreted differently than a small amount of growth & large color change to RED (dermatophyte). The color of colony is important in determining contaminant vs. dermatophyte, as is microscopic examination of macroconidia. To get the sample for microscopic examination, apply sticky side of clear acetate tape to the culture media where the growth has occurred. Then stain the sample with Lactophenol cotton blue.

By microscopically examining the sample you can speciate the dermatophyte. By speciating the dermatophyte you can tell the source of the infection (see below). This is done by identifying macroconidia. The descriptions of the different macroconidia are available in many text books or on line.  

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Sampling procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pustule</td>
<td>No surface disinfection. Clip hair with sterile scissors (avoid clippers). Lance pustule with sterile narrow-gauge needle. If purulent exudate is visible on the needle, apply to a sterile swab; if not, gently touch exudate expelled from pustule with sterile swab and place in transport medium or sterile container. Sometimes lancing of very small pustules results in haemopurulent exudate, which is still suitable for sampling.</td>
</tr>
<tr>
<td>Crust</td>
<td>No surface disinfection. Use sterile forceps or a sterile needle to lift the edge of a crust. The presence of exudate under a crust indicates an ideal site for culture. Touch sterile swab to exposed skin surface and place in transport medium or sterile container.</td>
</tr>
<tr>
<td>Epidermal collarette</td>
<td>No surface disinfection. Clip hair with sterile scissors (avoid clippers). Roll sterile swab across border of collarette two or three times and place in transport medium or sterile container.</td>
</tr>
<tr>
<td>Papule*</td>
<td>Sampling by biopsy is probably more reliable. Provide local anaesthesia by subcutaneous injection of 2% lidocaine. Clip hair with sterile scissors or clippers. Clean skin surface by a simple wipe with 70% alcohol† (no additional surgical preparation). Allow alcohol to dry. Using a sterile 3 or 4 mm punch and sterile surgical instruments, collect tissue sample and place in sterile container or transport medium. Suture biopsy site. Alternatively, papules may be prepared and disinfected* as above, then sampled by insertion of a sterile needle and culture of emerging or expressed blood or exudate.</td>
</tr>
</tbody>
</table>

*There is no research to show which method is more appropriate. †This method of disinfection is suggested to kill any surface bacteria. However, there is no research to indicate the value or necessity for any disinfection of the skin surface prior to sampling of papules.
Resistant Bacteria are Coming to Your Neighborhood- MRSA, MRSP, and MRSS:
Proper Antibiotic Use in Small Animal Dermatology-
What Should We Be Using? (Parts 1 and 2)
Paul Bloom, DVM, DACVD, DABVP
Allergy, Skin, and Ear Clinic for Pets
Livonia, MI

Superficial bacterial folliculitis (SBF) is one of the most common dermatologic problems diagnosed in dogs. The infecting organism is usually a staphylococcus. In the recent past, successful treatment could be accomplished with a beta lactam antibiotic (a first-generation cephalosporin (e.g. cephalaxin) or a potentiated amoxicillin). Increasingly, methicillin resistant staphylococcus (MRS) is being identified as a cause of skin infections in dogs. The MRS may belong to the species aureus (MRSA), pseudintermedius (MRSP), intermedium (MRSI), or rarely lugdunensis or delphini.

NO member of the beta lactam family of antibiotics will be effective when a MRS is identified. If the laboratory reports methicillin (oxacillin) resistance but susceptible to other members of the beta lactam family of antibiotics you should contact the laboratory to clarify- either the report on the methicillin resistance is incorrect or the reported susceptibility to the other beta lactam antibiotic is incorrect. Complicating the treatment of MRS is that these bacteria are frequently multi-drug resistant (MDR). In a study by Bemis, et al. it was found that more than 90% of the MRSP were MDR. MDR was defined as being resistant to ≥4 antimicrobial drug classes. The cause of the increased frequency of MRSP has not been clearly established but one of the many risk factors for MRS and MDR staphylococcus is the administration of fluoroquinolones. Reducing the administration of antibiotics and particularly fluoroquinolones and 3rd generation cephalosporins may help prevent persistent carriage of MRSA in humans. In study, the hospital recognized that MRSA outbreaks were correlated to the overuse of third-generation cephalosporins for prolonged periods. Additional information about the administration of 3rd generation cephalosporins or fluoroquinolones is discussed below.

The purpose of this lecture is to help stem the rising incidence of MRS in our cases of canine pyoderma.

Bacterial culture and susceptibility (c/s) testing should be performed in cases of poorly responsive (NOT recurrent) SBF. If a deep pyoderma has exclusively rods on cytology, has been treated with antibiotics recently or the dog is systemically ill then a culture and susceptibility test should be performed on the first visit. If a c/s is submitted, the MIC (broth microdilution technique) method should be used to determine the susceptibility rather than the disc diffusion method (Kirby-Bauer). The disk-diffusion susceptibility test (DDST) is semiquantitative in that the drug concentration achieved in the agar surrounding the disc can be roughly correlated with the concentration achieved in the patient’s serum. It will only report the organism’s susceptibility (susceptible, intermediate or resistant) based on an approximation of the effect of an antibiotic on bacterial growth on a solid medium. Tube dilution (MIC) is quantitative, not only reporting SIR but also the amount of drug necessary to inhibit microbial growth. The MIC is reported as the amount of antibiotic (in µg/ml) necessary to inhibit the growth of the tested bacteria (the lowest concentration in the tube that is clear). This allows a clinician to not only decide susceptible or resistant but also the proper dosage and frequency of administration of the antibiotic.

There are limitations to bacterial cultures. In regards to susceptible vs resistant bacteria- if the MIC for the bacterial isolate falls in the susceptible category, there is a greater likelihood of successful treatment (cure) than if the isolate were classified as resistant. The prediction of whether the bacteria will, or will not, respond to treatment is commonly referred to as the “90/60 rule.” The 90/60 rule was derived from the observation that, in general, bacteria treated with antimicrobials to which the strain is sensitive will have a favorable therapeutic response in approximately 90% of the patients. On the other hand, when the bacteria are reported as resistant to the antimicrobial administered approximately 60% of patients will respond to therapy. This rule only applies to immunocompetent

26 Carlene A. Muto, MD, MS; John A. Jernigan, MD, MS; Belinda E. Ostrowsky, MD et al SHEA Guideline for Preventing Nosocomial Transmission of Multidrug-Resistant Strains of Staphylococcus aureus and Enterococcus • Infection Control and Hospital Epidemiology, Vol. 24, No. 5 (May 2003), pp. 362-386
patients with monomicrobic bacterial infections who are treated with a single antimicrobial agent which is administered parenterally in circumstances in which the penetration of drug to the site of infection is predictable.30

Another limitation of bacterial cultures is that the clinical predictive value of a susceptible result is excellent (>90%) while the ability of *in vitro* tests to reliable predict failure is much less (<35%)30

In veterinary medicine, we have no data to confirm or challenge the 90/60 rule.

Why might an antimicrobial fail even if the culture reports the organism as susceptible? There are several possible causes including

1. susceptibility is based on serum concentrations, not tissue concentrations. The discrepancy may occur because the infected tissue may have a decrease in drug penetration due to poor blood flow, localized hypoxia or unfavorable tissue pH.
2. Presence of necrotic tissue or biofilms
3. The number of organisms present at the site of the infection (typically cultures use 10^5 organisms while most infections have 10^7 to 10^10 organisms). For many compounds, the MIC increases as the inoculum increases, perhaps because of a decrease in per-cell antibiotic concentration
4. The dosage and/or frequency of administration of the drug is different than the ones used to calculate the breakpoint
5. The MIC results have a standard deviation of +/- one dilution so that an MIC of 2 can actually be 1 or can be 4.

The intermediate category is not intended to mean "moderately susceptible." If the MIC value is in the intermediate category, therapy with this drug at the usual standard dosage is discouraged because there is a good likelihood that drug concentrations are inadequate for a cure. However, successful therapy is possible when doses higher than the label dose is used or if the drug is concentrated in the affected organ (eg urine) or is used topically. If the MIC is in the resistant category, treatment failure is more likely because of resistance mechanisms or inability to obtain effective drug concentrations that are not toxic to the patient. However, a patient with a competent immune system may sometimes eradicate an infection even when the isolate is resistant to the drug based on the MIC. Lastly not only does the MIC method indicate susceptibility, but it also implies the relative risk of emerging resistance and thus the need for a high dose.

In regards to bacterial skin cultures- to interpret and use a susceptibility test based on MIC requires the following information

1. First confirm that the organism is an expected pathogen from the skin
   a. *Staphylococcus pseudintermedius*
   b. *Staphylococcus intermedius*
   c. *Staphylococcus delphini*
   d. *Staphylococcus schleiferi coagulans*
   e. *Staphylococcus schleiferi schleiferi* -
   f. *Staphylococcus lugdunensis*
   g. *Staphylococcus aureus*
2. MIC of the antibiotic in relationship to the organism. This is reported on the culture results.
3. The breakpoint is the highest plasma concentration of the drug that can safely be achieved in the patient. If the MIC exceeds the breakpoint this means that to inhibit visible growth in the test tube, the drug concentration exceeds what can safely be obtained in the patient’s plasma. The breakpoints for each antibiotic should be available from your laboratory. Currently MSU’s DCPAH website has a breakpoint chart available go to https://www.animalhealth.msu.edu/Sections/Bacteriology/WEBCD.BACT.REF.011.pdf
4. You then look at the culture results and list all the antibiotics that are reported as < X where X is the listed MIC for each antibiotic
5. For the next step, you need to be aware that within a population of susceptible bacteria there is a mixture of strains (heterogeneity). Some of the strains are very sensitive to a given antibiotic while others are less susceptible. The less susceptible ones would be the ones w/the MIC closer to the breakpoint (resistant MIC level). From the list, you made in step 3 you need to rank the antibiotic based on which have the most susceptible bacteria. You do this by calculating the efficacy ratio. This number is the breakpoint of the antibiotic divided by the MIC of the bacteria. The higher the number the more susceptible the bacteria are to that antibiotic.
6. You will need to take the list from step 4 and decide which antibiotic fulfills your needs based on
   a. High efficacy ratio
   b. Ability to penetrate the infected tissue
   c. Side effects of the drug
   d. Ease of administration (consider both route and frequency required)
   e. Cost of the medication
7. If there are no antibiotics w/< X or the ones that do are either too toxic or too expensive you should then list, the remaining antibiotics that are reported as susceptible. From this list, you need to calculate the efficacy ratio.
Remember this number is the breakpoint of the antibiotic divided by the MIC of the bacteria. The higher the number the more susceptible the bacteria are to that antibiotic. For example, you have a staph that has a MIC of 1 umg/ml to enrofloxacin and has a MIC of 4 umg/ml to cephalaxin. Which antibiotic is the population of bacteria most susceptible to? To determine this, you take the breakpoint of enrofloxacin (4) and divide it by the MIC (1) and the efficacy ratio is 4. Doing the same to cephalaxin you get (32/4) 8. Remember the higher the number the more susceptible the bacteria are to that antibiotic. So, cephalaxin would have the highest number of susceptible bacteria.

8. With this list of antibiotics and their efficacy ratio, apply the criteria listed in step 5 to determine the most appropriate antibiotic.

In humans, the MRS organism is Staphylococcus aureus. In animals, the staphylococcus responsible for infection usually belongs to the staphylococcus intermedius group (S. intermedius, S. pseudintermedius, and Staphylococcus delphini). Currently the protocol for identifying MRSA in vitro is to use cefoxitin as the surrogate. The problem is that certain strains of methicillin-resistant S pseudintermedius (any in the SIG?) may be falsely identified as methicillin susceptible, while truly being resistant, if the laboratory uses cefoxitin susceptibility as the indicator. This is because cefoxitin may not induce the mecA gene as reliably in S pseudintermedius as it does in Staphylococcus aureus. It is important that laboratories know this and use oxacillin susceptibility testing for identifying MR S pseudintermedius isolates (all SIG?) instead. To avoid mislabeling MRSP as susceptible the laboratory needs to know that the break point for S pseudintermedius has been lowered from the previous level of 2.0 umg/ml down to 0.25 umg/ml. Why is this clinically important? If you are using a human laboratory, or a local laboratory, they may not be aware of this difference in testing between S. aureus and S pseudintermedius. Because of this, the author strongly recommends using a veterinary laboratory that uses Clinical and Laboratory Standards Institute (CLSI) guidelines.

Recently the effectiveness of clindamycin against MRSA has been questioned. There are 2 genes, msrA and the erm gene family that are responsible for S.aureus’ resistance to macrolides (eg erythromycin). The msrA gene accounts for the resistance to beta lactams and macrolides, while the erm gene codes for macrolides and lincosamides (lincomycin and clindamycin) resistance. The erm gene expresses macrolide resistance constitutively while the clindamycin resistance can be either constitutive or inducible. Constitutive expression means that this gene will be active in the bacteria from the onset and the culture will report resistance to erythromycin and clindamycin. However, if the erm gene is an inducible gene then only if there is a mutation in the erm genes will resistance occur. These mutations occur at a rate of about one in every 106 bacteria. Because most bacterial infections have bacterial populations that are in the range of 107 - 1010 these mutations readily occur, resulting in constitutive resistance. This leads to resistance to clindamycin while on treatment. As the susceptibility pattern to clindamycin of MRSA isolates (or MSSA) possessing only the msrA gene (truly resistant to erythromycin and susceptible to clindamycin) and those that also have the inducible erm gene (truly resistant erythromycin and falsely reported as susceptible to clindamycin) are the same, it is important to distinguish between these phenotypes. Unfortunately, no commercial lab is currently doing any additional testing to identify the erm resistance gene. So, in the meantime resistance to erythromycin may be used as a clue to the presence of this inducible gene. It is best to avoid clindamycin in any MRSA infection if the organism is reported to be resistant to erythromycin. Note this inducible gene has rarely been reported in MRSP.

In the tetracycline family, there is a gene (tet m) that is responsible for bacterial resistance to tetracycline, doxycycline and minocycline. However, there is an inducible gene tet (k) that is responsible for resistance to tetracycline and inducible resistance to doxycycline but the bacteria are not resistant to minocycline. Because of this, minocycline should be tested separately from tetracycline/doxycycline.

Systemic therapy for canine pyoderma is becoming more problematic because of the increasing incidence of methicillin resistant Staphylococcus. To help address this problem topical therapy, either as a monotherapy or as part of polypharmacy, has
become an essential component of managing SPF. Topical therapy may decrease the length of time administering, or eliminate the need for, systemic antibiotics. Since dogs with SBF frequently have atopic dermatitis, bathing will remove problematic allergens, in addition to bacteria from the skin. The limitations of using topical therapy include time constraints of the owner and, if treating a large area, possibly cost. Shampoo ingredients that are effective for treating bacterial pyoderma include chlorhexidine, benzoyl peroxide, ethyl lactate, triclosan and boric acid/acetic acid. In 2 different studies, chlorhexidine was the most effective ingredient.39,40 Silver sulfadiazine has traditionally been used for to treat gram negative bacteria, especially Pseudomonas.41 However it is also effective against some gram-positive bacteria42 including Staphylococcus aureus.

When treating a dog with a SBF, an antibiotic should be administered for at least 21 days, or 14 days past YOUR clinical examination that has determined the infection has resolved, whichever is LONGER. For dogs with deep pyoderma, treat for at least 6 weeks or 21 days beyond clinical resolution, whichever is longer. In cases of SBF don’t use glucocorticoids (GC) when the pruritus is only at the lesions or when the pruritus is only mild at the nonlesional areas. If a dog with a SBF has intense pruritus at nonlesional areas, then a tapering 21 days’ course of prednisone may be dispensed. Using GC in the presence of a pruritic pyoderma makes interpretation of response to therapy impossible (was it the steroid or treating the infection that resolved the pruritus?). It also makes it more difficult to resolve the infection. NEVER use GC in cases of deep pyoderma!!

Before discussing systemic antibiotics for bacterial pyoderma, the author needs to make a few comments about cefpodoxime and cefovecin. Cefpodoxime is an oral 3rd generation cephalosporin effective for most of the staphylococcus infections that occur in dogs. This once a day antibiotic is useful in cases where the owner has difficulty administering medication. However recently, cephalexin has become available as a chewable tablet (Rilexine® Virbac) that should help make administration of cephalexin much easier. The once daily administration and the formulation in a pill rather than a capsule may make it easier for some owners to medicate their dog. Another instance where it may be of use is during a food trial. During this trial, it is best, if possible, to avoid gelatin (animal protein) that is present in capsules. Using cefpodoxime tablets would solve this problem. The author also has an impression that there are fewer intestinal disturbances using cefpodoxime versus cephalexin. However, consider when dispensing cefpodoxime there are some staphylococcus infections that will be resistant to cefpodoxime but susceptible to cephalexin43. Also, the stated higher compliance rate of once daily medication vs twice daily may not be true. Adams et al reported that in their study there was no difference in compliance with once daily versus twice daily dosing44. Lastly there are numerous studies showing that once daily cephalexin at 30-40 mg /kg is as effective as splitting this dose and administering q 12 hours.45,46,47,48,49,50,51 HOWEVER these were not peer reviewed studies so this is NOT my recommendation. However, these studies do suggest that missing 1 dose of

35 Loeffler A, Cobb MA, Bond R Comparison of a chlorhexidine and a benzoyl peroxide shampoo as sole treatment in canine superficial pyoderma The Veterinary Record 2012;170:26 675
38 Drug insert- silvadene- http://www.drugs.com/pro/silvadene.html
39 Rankin SC, O’Shea K, Morris DO;Susceptibility of companion animal isolates of Staphylococcus schleiferi to cephalothin and cefpodoxime Vet Derm: 17;3:214
Cefovecin is a parenterally administered 3rd generation cephalosporin that has tremendous value when used properly (selectively). Cefovecin may persist in the body for up to 5 weeks; therefore, adverse event monitoring should be carried out for a similar amount of time. (note USA insert states reactions may require prolonged treatment due to the prolonged systemic drug clearance (65 days). The drug insert from the New Zealand product states “Prudent Use: It is prudent to reserve third generation cephalosporins for the treatment of clinical conditions which have responded poorly, or are expected to respond poorly, to other classes of antimicrobials including first generation cephalosporins. Use of the product should be based on susceptibility testing and consider official, and local, antimicrobial policies. Indiscriminate use of cefovecin could contribute to the development of antibiotic resistance.”

This author believes that this drug should be reserved for cases where the owner is unable to orally medicate the dog or cat or the animal can’t tolerate oral antibiotics. The concern about using this medication is that after the first injection therapeutic drug concentrations (above MIC) are only maintained for 7 days for S. intermedius infections, while tissue levels persist for up to 65 days. The question is whether this prolonged subtherapeutic blood (tissue?) level will encourage the incidence of methicillin resistant staphylococcus. Will adverse reactions require prolonged treatment due to the prolonged systemic drug clearance? What are the long-term effects on injection sites, especially in cats? How clinically significant is the in vitro finding that cefovecin increases free concentrations of carprofen, furosemide, doxycycline, and ketoconazole. Will drugs with a high degree of protein-binding (e.g. cardiac, anticonvulsant, and behavioral medications) compete enough with cefovecin-binding to create adverse reactions. Most of these questions have not been answered, even by the company.

In the BSAVA Guide to the Use of Veterinary Medicines, it discusses the prudent use of antimicrobial agents. In regards to 3rd generation cephalosporins and for any fluoroquinolones (FQ) it states “that in all species fluoroquinolones and third- and fourth-generation cephalosporins should be used judiciously and never considered as first-choice options”.

The Europeans are also concerned about 3rd generation cephalosporin use and FQ use. The European Medicines Agency states (EMEA/CVMP/215997/2006) “Following advice given by the CVMP Scientific Advisory Group on Antimicrobials (SAGAM), the CVMP agreed the following statements should be included in section 4.5 of the SPC (special precautions for use)” “It is prudent to reserve third generation cephalosporins for the treatment of clinical conditions, which have responded poorly, or are expected to respond poorly, to other classes of antimicrobials or first generation cephalosporins.” and “Use of the product should be based on susceptibility testing and take into account official and local antimicrobial policies”.

The Swedish veterinary medical society published guidelines in 2009 for the use of antibiotics in the treatment of dogs and cats. In this guideline, it is stated very clearly that third generation cephalosporins should only be used to treat infections where there are no other suitable options. It goes on to state that injections with long-acting antibiotics should not normally be used to treat a pyoderma. Specifically, in the guidelines it states that cefovecin should only be used if the treatment is “of the utmost importance” for the animal AND administration of other medications is not possible.

The Norwegian Antibiotics Policy states that “Antimicrobial drugs considered critically important for human health by the WHO: Fluoroquinolones, macrolides, glycopeptides and 3rd and 4th generation cephalosporins, should always be considered last resort and never be prescribed unless c/s dictates that there are no other available drugs that can be used to treat the infection. They go on to state “Long acting drugs or slow release formulations should be used very cautiously as the documentation is lacking with regards to the impact these drugs may represent with regards to resistance development in the normal flora.”

The concern with using FQ is that, per information from the CDC website, “none of the fluoroquinolones are FDA-approved for treatment of MRSA infections. A major limitation of fluoroquinolones is that resistant mutants can be selected with relative ease, leading to relapse and treatment failure”. MRSA strains are especially adept at developing fluoroquinolone resistance, and such resistance is already found among MRSA isolated from patients with CA-MRSA infections. In addition it has been reported that there is a significant association between total fluoroquinolone use within human hospitals and percentage of S. aureus isolates that were MRSA and between total fluoroquinolone use in the community and percentage of E. coli isolates that were fluoroquinolone-resistant

52 Pfizer drug insert – prescribing information for Convenia
It has been noted that there has been an increase in the number of ESBL \textit{E. coli} and Salmonella spp. in the absence of prior exposure to the cephalosporins, suggesting potential coselection and coreistance.\textsuperscript{60} Lastly, even after selection pressure is removed (stopped using the FQ), fluoroquinolone resistance persists.\textsuperscript{61}

Additional concern about both FQ and 3\textsuperscript{rd} generation cephalosporins is that they are both independent risk factors for development extended spectrum beta-lactamase (ESBL) producing bacterial infections.\textsuperscript{62, 63} Extended-spectrum beta-lactamases (ESBLs) are beta lactamases found in \textit{Enterobacteriaceae} (\textit{E. coli}, \textit{K. pneumoniae}, etc) and are a concern in human medicine because they cause serious infections in humans. These bacteria are frequently multi-drug resistant, not only to beta lactam antibiotics, but also to non-beta lactam antibiotics such as aminoglycosides, fluoroquinolones, tetracyclines, chloramphenicol, and sulfamethoxazole-trimethoprim. This wide-ranging resistance greatly limits effective treatment options. The genes encoding this resistance are mediated by plasmids and/or mobile elements which allows horizontal transfer between the same and different species of \textit{Enterobacteriaceae} making wide spread dissemination a concern.\textsuperscript{64} In contrast to FQ and 3\textsuperscript{rd} generation cephalosporins, first generation cephalosporin have not been reported to be a risk factor for such resistance.\textsuperscript{62}

A consensus statement has been released with the purpose of guiding practitioners in the diagnosis, treatment, and prevention of superficial bacterial folliculitis(SPFI).\textsuperscript{65} These guidelines, like the previous guidlines published concerning antibiotic use for treating urinary tract infections,\textsuperscript{66}, are the result of a committee consisting of veterinary internists, pharmacologists, microbiologists and dermatologists. In this article, it is stated that “there is concern among some members of this panel about the potential selective effects of third generation cephalosporins (cef podoxime and cefovecin) on the Gram-negative microbiota, due to their broader spectrum of activity compared with first generation cephalosporins.” The key points are:

1. Identify and treat the underlying cause for SPF;
2. Perform skin scrapings to identify demodex mites;
3. Perform cytology to confirm a bacterial component;
4. If possible, use disinfectants and/or topical antimicrobials as the sole treatment. If this is not possible, at least use topical therapy to shorten the length of time that systemic antibiotics need to be used;
5. Empirical therapy can be done in non-recurrent cases or recurrent cases that have successfully responded to previous treatment. You should select a drug from the list of first tier medications. This list includes (the author will prescribe the drugs in bold):

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a. Clindamycin- Antirobe® 5-10 mg/# sid 67,68,69,70,71
b. First generation cephalosporin
   i. Cephalexin 10-15 mg/# bid-tid
   ii. Cefadroxil – 10 mg/# bid
c. Amoxicillin/clavulanic acid – Clavamox® 10 mg/#bid
d. Potentiated sulphonamides
   i. Trimethoprim/sulfonamide- Tribrisson® 15 mg/# bid
   ii. Sulfadimethoxine and ormetoprim- Primor® 25 mg/# sid on day 1 then 12.5 mg/# sid
e. Erythromycin
f. Lincomycin

6. In cases, which fail to respond to appropriate treatment (dose and frequency) using a first-tier antibiotic, a bacterial culture should be performed. When selecting, an antibiotic based on a culture result, a sensitive second tier antibiotic should ONLY be used if the organism is resistant to all first-tier antibiotics OR the animal can’t tolerate any of the first-tier drugs OR the owner is unable to administer them. The second-tier antibiotics include:
   a. Any fluoroquinolone (FQ)
      i. Please note- when appropriate, a veterinary FQ should be administered rather than ciprofloxacin. This recommendation is based on a study that revealed that there is a very large variation in absorption of ciprofloxacin 72. In this study, it was reported that to get appropriate blood levels you would need to dose ciprofloxacin at 12-52 mg/kg. This is for highly susceptible (low MIC) organisms, not the ones with higher MICs!
   b. Chloramphenicol
c. Rifampin
d. Doxycycline/minocycline

7. 3rd tier antibiotics should never be used without consultation with a specialist – these are vancomycin and linezolid.

Note in regards to 3rd generation cephalosporins (cefovecin, cefpodoxime). In this report, they state there is insufficient evidence for this working group to reach consensus on categorization of these agents as first or second tier drugs (see text under ‘Systemic antimicrobial therapy’ and concerns about selection of ESBL-producing Enterobacteriaceae). Author’s comment- since there is a disagreement on the use of these drugs, why not reserve 3rd generation cephalosporin for cases where first generation would not be appropriate.

Recently Dr Scott Weese wrote an editorial for Clinicians Brief. (CB) 73 He states that “this journal (CB) like Equine Veterinary Journal will avoid articles that recommend extralabel the use of fluoroquinolones and extended-spectrum beta-lactam antimicrobials (eg. third- or fourth-generation cephalosporins). Consideration for their use will occur if there is specific mention of the relevant issues, and evidence supporting that recommendation is provided.”

This author wants to remind the readers that cefovecin is ONLY labeled for the treatment of canine skin infections (secondary superficial pyoderma, abscesses, and wounds) caused by susceptible strains of Staphylococcus intermedius and Streptococcus canis (Group G). Cefpodoxime is ONLY indicated for dogs with skin infections (wounds and abscesses) caused by susceptible strains of Staphylococcus intermedius, Staphylococcus aureus, Streptococcus canis (Group G, β-hemolytic), Escherchia coli, Pasteurella multocida, and Proteus mirabilis. So as stated above other usage should be avoided.

73 Weese JS Prudent Antimicrobial Use in an Antimicrobial-Resistant World. Clinician’s Brief 2015: June; 10-11
Lastly, ACVIM published a consensus statement on antimicrobial use in animals\textsuperscript{74} and they state “fluoroquinolones and later generation cephalosporins possess activity against a wide range of bacteria and potential far-reaching effects on the microbiota. While limiting use of classes such as the 3rd generation cephalosporins and fluoroquinolones is widely accepted and consistent with principles of antimicrobial stewardship...” Again, this reiterates the recommendation of restricting the use of these antibiotics.

Bottom line – we should be very selective when dispensing any antibiotic but especially fluoroquinolones or any third- and fourth-generation cephalosporins in the treatment of canine bacterial pyoderma\textsuperscript{75, 76, 77}. However, to be clear - ALL antibiotics have consequences. This was demonstrated in a report of 173 dogs presented to a dermatology referral practice for treatment of a bacterial pyoderma\textsuperscript{78}. The study evaluated the impact of routine antimicrobial therapy on emergence or resolution of resistant bacteria in a group of 173 dogs presented to a dermatology referral practice for treatment of a bacterial pyoderma. Additionally, it evaluated the prevalence of MRSP colonization after successful treatment of their bacterial pyoderma. In this study skin, nasal and rectal swabs for bacterial culture were collected at the time of referral and after clinical resolution of the pyoderma. Of dogs that initially had an MRSP pyoderma, 26 of 42 (61.9%) were colonized at one or more sites at follow-up, even though the pyoderma had resolved. Of the 60 dogs with a non-MRSP pyoderma on initial presentation, 23 (38.3%) were colonized with MRSP at one or more sites after clinical resolution of the pyoderma.

It is apparent from this information that the older mindset “I wasn’t sure what to do so I put him on antibiotics since they won’t hurt him” needs to be changed. Even though using antibiotics may not harm that individual animal at that time, we are now, and may continue, to suffer the consequences with the spread of resistant bacteria (both human and veterinary). Hopefully we can disrupt this disturbing trend with good stewardship of the use of antibiotics.

The diagnosis of ANY skin disease is based on obtaining a detailed history, evaluating clinical findings (identification of primary lesions, distribution of lesions), laboratory testing and therapeutic trials. For autoimmune skin diseases (AISD) the most beneficial diagnostic tool is histopathologic evaluation.

**Pemphigus**

In pemphigus, the immune system is directed to inappropriately attack the desmosomes. Desmosomes are spot like sites of intercellular contact and attachment between keratinocytes.

Pemphigus foliaceus (PF) is the most common form of pemphigus and is probably the most frequently diagnosed autoimmune skin disease (AISD) affecting cats and dogs. Other forms of pemphigus that will be seen in practice include pemphigus erythematosus (PE) and panepidermal pemphigus (PPP). In general, PF is a disease of young to middle aged animals with a mean age of onset of 4 years old. Sixty five percent of the dogs have the disease before 5 years of age. PF has been reported in numerous breeds with Chow Chows and Akitas being at increased risk in the author’s practice. There is no gender predilection in dogs.

In the author’s experience, even though there are three forms of PF reported in the literature- spontaneous pemphigus, drug-related (both drug induced (no genetic predisposition) and drug triggered (genetic predisposition) and a form that is associated with chronic skin disease, it is rare to have cases in which the dog has had chronic skin disease. In fact, there is no evidence to support that claim. Cases that occur spontaneously are by far the most common.

Historically, the owner may report that the lesions wax and wane or are progressive. The progression of the disease may be slow, especially cases with only facial involvement, or the dog may develop acute eruptions (most commonly associated with generalized disease). With the generalized form the dogs frequently will be febrile, may have limb edema and have constitutional signs. Pruritus with any form varies from non-existent to moderately intense.

There are 3 primary distribution patterns of PF -facial (most common) form which involves the bridge of the nose, nasal planum, periorbitally, pinnae (especially in cats); a footpad form (cats may present only with paronychia) and a generalized form where lesions usually begin on the face and then spread. Note that there is a subset of dogs that have generalized disease from the onset.

The lesions progress from an erythematous macule⇒pustule⇒collarettes⇒erosions⇒yellow brown crusts. Because there is involvement of the hair follicles, multi-focal to diffuse alopecia is frequently present. The primary lesions of PF are large nonfolicular pustules (there are also follicular pustules present) especially involving the bridge of the nose, footpads, nasal planum or pinnae (cats may have lesions around the nipples). This contrasts with pustules associated with a bacterial pyoderma in which the pustules are follicularly oriented. The pustules that are present in a bacterial pyoderma usually involve the ventral abdomen and/or trunk and are much smaller than those seen with PF. In cats and dogs with PF, secondary lesions are more commonly seen than the pustules. These lesions include epidermal collarettes (uncommon), yellow brown crusts and erosions. These animals may be systemically ill, have distal limb edema, fever, lethargy and/or lymphadenopathy.

Differential diagnosis would include any pustular, crusting and scaling disease such as: pemphigus erythematosus; zinc responsive dermatosis (especially with foot pad involvement); metabolic epidermal necrosis (especially with foot pad involvement); bacterial and fungal (dermatophytosis) infections; demodicosis, DLE (facial/nasal form); erythema multiformae; mycosis fungoides; Leishmaniasis; and sebaceous adenitis.

**Diagnosis**

A cytologic prep of a pustule or crust should be performed. Microscopic findings would include acantholytic keratinocytes, either individually or in clusters, surrounded by NON-degenerative neutrophils and/or eosinophils- bacteria should not be seen. Histopathology is the only definitive means to diagnose pemphigus. An intact pustule (or if none are present, a crusted lesion) should be biopsied. Infectious diseases that produce proteases, such as a bacterial pyoderma or a dermatophyte infection (Trichophyton mentagrophytes), can breakdown the intracellular glycoproteins (desmoglein) leading to acantholysis. Because these infectious
diseases mimic PF histologically, you should request special stains for both bacteria (gram stain) and fungi (GMS, PAS) anytime a there is a histopathologic diagnosis of PF.

**Prognosis**

PF may be drug related, either drug-induced or drug-triggered. The drug-induced form PF is caused by a drug and upon removal of the drug, sometimes with a short course of immunosuppressive treatment, the disease resolves. Drug-triggered PF occurs when a drug stimulates a genetically predisposed individual to develop PF. Typically, this form of PF must be managed long term, like idiopathic PF. Currently there is no way to identify which cases of drug related PF are drug induced and which ones are drug triggered. In fact, there is no test that can be used to predict how well a case of PF will respond to treatment.

A study at NCSU revealed that 6 of 51 dogs (11.7%) with PF were weaned off all medication and stayed in remission for >1 year. Recognizing that PF is a sunlight aggravated disease; it was interestingly the dogs in this study were from areas (NC or Sweden) with high UV light exposure. In this report the dogs took 1.5–5 months of therapy before the disease was in remission. The drug(s) were then slowly tapered and then all therapy was stopped. The total duration of immunosuppressive therapy varied between 3 and 22 months. These dogs stayed in remission for the entire follow up period (1.5–6 years after treatment). Supporting this finding is a study from the University of Pennsylvania that reported that 10% of their cases went into long-term remission after weaning off medication.

This study performed at the University of Pennsylvania suggests that dogs with PF survived longer when given antibiotics (usually cephalixin) in addition to their immunosuppressive regimen. This contrasts with the author’s clinical observation that if dogs with PF do develop a concurrent pyoderma it only occurs AFTER being placed on immunosuppressive therapy. Supporting the author’s observations is a study from CSU that reported that there was no difference in survival when antibiotics were part of the initial treatment.

In the study from University of Pennsylvania the survival rate was approximately 40% with 92% of the deaths occurring by 1 year. Other researchers have reported having a long-term survival rate of approximately 70%.

Cats may have a better prognosis than dogs with this disease. In the same report from the University of Pennsylvania, only 4/44 cats treated died (from their disease or therapy) during the study period. In the author’s practice, survival at 1 year also exceeds 90%. In addition, a significant number of the cats are eventually able to have all medications discontinued without suffering a subsequent relapse.

**Treatment**

Managing any AISD takes frequent rechecks and alertness to complications associated with immunosuppressive therapy such as demodicosis, dermatophytosis and bacterial pyoderma. Interestingly the author has rarely seen a dog with PF that had a secondary pyoderma at initial presentation. It is more common to develop after beginning immunosuppressive therapy. If a patient was controlled and then has a relapse or if the patient has been improving and suddenly worsens, there are 2 possibilities. The PF (which does wax/wane) is flaring up OR that the dog developed a secondary infection due to immunosuppression. If the new lesions are folliculocentric you must also rule the big 3 folliculocentric infections – bacteria, demodex and dermatophyte. Skin scrapings, Wood’s light examination (screening test) and impression smears are the minimum data based that should be performed when a dog is presented with these lesions. Whether you need to do a fungal culture now depends on the how frequently you see dermatophytosis in your practice and what is seen on cytology (acantholytic keratinocytes, cocci, demodex mites). If dermatophytosis is commonly seen in your practice, then a fungal culture should be performed. Otherwise a fungal culture and a repeat skin biopsy can be considered second tier tests to be performed if the case doesn’t respond to appropriate therapy (eg antibiotics).

In addition to the treatment options listed below, shampoo therapy should be included for symptomatic treatment of the crusting dermatitis. Pending biopsy results, if intracellular cocci are seen on cytology the author will dispense cephalixin (10-15 mg/# bid-tid),

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unless there is a suspicion that it is a case of cephalixin induced PF. If only extra cellular cocci are seen, then topical shampoo therapy with an antiseptic (eg chlorhexidine, benzoyl peroxide, etc)

Treatment must be individualized for each patient since there is no “best” treatment that works in all PF patients. This is why monitoring the progress of the disease closely by PHYSICALLY examine the dog or cat is critical for successful management of PF. It is especially important to recheck the patient prior to any adjustment in medication. When devising a treatment plan, be sure to consider the severity of the disease so that the treatment side effects are not worse than the disease itself.

There may be regional differences in how aggressively PF needs to be treated. Some of this may be due to the differences in the gene pools of the patients. But since PF is a sunlight aggravated disease, it also may be related to the differences in sun exposure. Regardless of the locale, sun avoidance should be part of the treatment for PF.

Because diet has been implicated as a cause of PF (endemic) in humans, the author will review the dietary history and consider dietary modification if the initial response to therapy is poor. The ingredients implicated in human endemic PF contain thiols (eg garlic, onion), isothiocynates (mustard, horseradish), phenols (food additives) and/or tannins (tea, bananas, and apples).

Vitamin E (400-800 IU bid) and essential fatty acids may be used as part of the treatment since these nutrients have anti-inflammatory properties and anti-oxidant activities.

For mild or localized disease, a tetracycline with niacinamide may be used. This is because tetracycline family and niacinamide (T/N) have a variety of anti-inflammatory & immunomodulating properties the combination has been used in treating a variety of immune mediated skin diseases, such as discoid lupus erythematosus, vesicular cutaneous lupus erythematosus (idiopathic ulcerative dermatosis of collies and Shelties), lupoid onychodystrophy, pemphigus erythematosus, German Shepherd Dog metatarsal fistulae, sterile paniculitis, sterile periadnexal granulomatous dermatitis (idiopathic sterile granuloma-pyogranuloma syndrome), vasculitis, dermatomyositis and cutaneous histiocytosis. The author may use this combination for any of the previous mentioned diseases if the disease is relatively mild. If any of these diseases fail to respond well to immunosuppressive therapy, T/N may also be added to the therapy in dogs.

Traditionally tetracycline was the drug used from the tetracycline family but when it became unavailable then doxycycline was used. But with the increasing occurrence of methicillin resistant staphylococcus infections, using antibiotics for immunomodulating properties has become a concern, this is especially true for doxycycline because it is considered a second line antibiotic in the treatment of bacterial pyoderma in the dog. Studies in humans have failed to show any evidence that sub-antimicrobial doxycycline treatment (20 mg bid for 9 months) exerted an effect on the composition, or doxycycline resistance level, of either the fecal or the oropharyngeal and intestinal microflora.

It appears as though this dosage may be suitable for long-term treatment of gelatinolytic inflammatory diseases.

Additional studies in humans using 40 mg for 16 weeks revealed that a subantimicrobial doxycycline dose (40 mg) had a minor ecological effect on the oropharyngeal and intestinal microflora. A third study in humans revealed that long-term oral administration of 40 mg of doxycycline once daily results in no antimicrobial resistance. A study was performed on beagle dogs that revealed that doxycycline at 2 mg/kg daily appeared to be an appropriate subantimicrobial regimen for dogs with periodontitis. It appears as though this dosage may be suitable for long-term treatment of gelatinolytic inflammatory diseases. Whether that is true for other inflammatory or immune mediated diseases has not been studied. Based on these 4 studies, the author’s concern about antimicrobial resistance and the lack of any evidence based studies supporting any other dose, the author now will use doxycycline at 2 mg/kg sid (or 1 mg/kg bid). On the limited number of cases that have been treated it appears this dose is effective.

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86 Olivry T. Canine pemphigus foliaceus: an update on pathogenesis and therapy In: Clinical Programme Proceedings of the Fifth World Congress 222-227
The dosage niacinamide in dogs <10 kg is 250 mg of each, q 8 hours. For dogs >10 kg - 500 mg of niacinamide q 8 hours is administered. If there is a clinical response, which may take a few months, the frequency of administration may be slowly decreased (q 6 months or so) Side effects are rare but they include vomiting, anorexia, lethargy, diarrhea and elevated liver enzymes.

Glucocorticoids (GC) are the mainstay of therapy for AISD. They may be applied topically or administered systemically depending on the severity of the disease and the amount of the body involved. Since some cats can't metabolize inactive prednisone to the active form, prednisolone, ONLY PREDNISOLONOE should be used in cats. In dogs either prednisone or prednisolone may be used. The author has seen cases of feline PF, which were well controlled on prednisolone, but when prednisone was dispensed relapsed, only to go back into remission once the cat was placed back on prednisolone-all at the exact SAME dosage and frequency.

The most potent topical GC (veterinary product) is a product containing fluocinolone acetonide (Synotic®). For localized disease, the author will apply this product bid until clinical remission (not to exceed 21 days) and then tapered slowly over the next few months. Be sure to have the owners wear gloves when applying this product. If this treatment is unsuccessful the one of the following systemic therapies will be instituted.

In dogs with more extensive disease or those that fail topical therapy, prednisone or prednisolone is administered at 1 mg/# bid for 4 days then ½ mg/# bid for another 10 days. If the dog is rechecked every 14 days. If the disease is in remission, the dose is decreased 25% at each recheck examination. The author defines “remission” as the absence of any active lesions (no pustules and any crusts that are present are easily removed with the underlying epidermis appearing pink rather than erosive). DON’T TAPER THE DOSE TOO QUICKLY. The goal is to maintain the dog on 0.25 mg/# or less every other day of prednisone/prednisolone. If this is not achievable, then azathioprine is added to the therapy (see below). Some dermatologist will use the combination therapy from the onset, but because at least 75% of the dogs in the author’s practice can be maintained on just GC and there are additional risks and costs associated with this drug the author considers this a second-tier therapy 85. Only if the dog fails to respond to GC, or can’t be managed with every other day administration, will the author add azathioprine to the therapy.

For cats, ONLY prednisolone is used and in fact only prednisolone is stocked in the author’s pharmacy- this is to avoid the inadvertent administration of prednisone to a cat. The dose for cats is 1 mg/# bid for 14 days. From that point forward the management of the cat with prednisolone is the same as the dog. If the disease is not controlled with prednisolone, then CHLORAMBUCIL (see below) is added to the therapy NOT AZATHIOPRINE!!!

If an animal fails to respond to prednisolone other immunosuppressive agents (see below) will be added to the therapy.

Animals on chronic GC, regardless of dose should have a CBC, serum chemistry profile, urinalysis every 6 months. The urinalysis is performed to identify proteinuria due to the steroid administration. There is no benefit to doing a urine culture if the dog or cat are asymptomatic since you will identify animals with asymptomatic bacteriuria which doesn’t need/should be treated. There is no evidence to the author’s knowledge that leaving a dog on cyclosporine or steroids with asymptomatic bacteriuria will lead to pyelonephritis. In a consensus statement, the ACVIM states that “treatment may not be necessary in animals that have no clinical signs of UTI and no evidence of UTI based on examination of urine sediment.”93 In humans they don’t treat asymptomatic bacteriuria except in pregnant women or type 1 diabetic patients or if undergoing a urologic procedure in which mucosal bleeding is anticipated94. In humans, no recommendation can be made for screening for or treatment of asymptomatic bacteria in renal transplant or other solid organ transplant recipients.

Azathioprine (AZA) is an antimetabolite that is a competitive inhibitor of purine. Purine is necessary for DNA formation, so in the presence of AZA, defective DNA is formed preventing cell replication. It has a lag phase of four to six weeks before it reaches its full effectiveness. The drug is administered concurrently with GC. The initial dose of azathioprine is 1.0 mg/# sid. Once remission is achieved, and the dog is either off GC, or the lowest dose of GC has been obtained, AZA is then tapered every 60-90 days. Usually the author will decrease the frequency, not the dose of azathioprine, first decreasing it to every other day and then if the disease is still in remission, to every 72 hours. A CBC, platelet count, serum chemistry profile is performed every 14 days for 2 months, then q 30 days for 2 months then q 3 months for if the dog is on azathioprine. Potential adverse effects include anemia, leukopenia, thrombocytopenia, hypersensitivity reactions (especially of the liver) and/or pancreatitis. AZA should not be used in cats- it may cause irreversible bone marrow suppression.

Chlorambucil (CAL) is used in cats and in dogs who failure to respond to azathioprine or can’t tolerate it. The protocol/precautions/monitoring for CAL is the same as w/AZA. The induction dose is 0.1-0.2 mg/KG/day. Note it too may have a 4-6-week lag effect.

Cyclosporine A (CSA), a calcineurin inhibitor, has been used orally at a dose of 5 mg/kg sid in cases of PF with poor results in dogs. Recently the author has used CSA at 5 mg/kg sid- bid with success either as monotherapy or as steroid sparing agent. There have been anecdotal reports of successful treatment of PF in cats (especially nail bed form) with CSA. Recently topical tacrolimus has been reported to be effective in the treatment of facial PF and PE. The author has limited experience with this product.

Sulfasalazine (SSZ) is a sulfa that has both anti-inflammatory and/or immunomodulatory properties due to its prostaglandin synthetase and leukotriene inhibition. In the past it has been used for the treatment of colitis but more recently it has been used for neutrophilic vasculitis. SSZ is metabolized by colonic bacteria to 5-aminosalicylic acid (5ASA) and sulfapyridine (SP). SP is well absorbed, metabolized in the liver, and excreted by the kidney while 5-ASA is much less well absorbed. Because SSZ is metabolized to aminosalicylic (“aspirin”) this drug should be used cautiously in cats. The biggest concern with this medication is the possibility of developing irreversible keratoconjunctivitis sicca. This appears to be an idiosyncratic reaction that occurs more in smaller dogs but may occur in any dog. It is essential that you warn the owner that if the eyes become red or they notice an ocular discharge or squinting to contact you immediately so that you can do tear testing. Other side-effects associated with this drug include anemia, KCS and hepatotoxicity so a CBC, serum chemistry profile and Schirmer tear test are performed every 14 days for 2 months, then q 30 days for 2 months then q 3 months for if the dog is on SZA. In cases of neutrophilic vasculitis that fail SZA treatment w/dapsone may be effective, however, dapsone appears to be more toxic than SZA. The dose is 20-50 mg/kg tid (maximum 1 gm/dose), usually beginning with 20-30 mg/kg tid. Once the disease is in remission, the dose is slowly tapered.

Specific treatment approach- for mild cases of facial PF (or cases of pemphigus erythematosus), a topical glucocorticoid is used and/or T/N. For generalized forms, or in cases with severe facial and/or footpad involvement, prednis(ol)one should be used as described above. If the disease is in remission at each recheck, the steroids are tapered as previously described. If the disease is not in remission at the first 14 day recheck or it can’t be kept in remission with steroids at a dose of <0.25 mg/# q 48 hrs, then either azathioprine (dogs) or chlorambucil (cats) is added to the treatment.

If the disease is not responding to the above treatment, CONFIRM that the diagnosis is correct (be sure to have ruled out dermatophytosis, demodicosis and bacterial pyoderma) then, changing to either dexamethasone or triamcinolone may be helpful. Use 0.05-0.1 mg/# bid of either drug, as the starting dose, and then taper as previously discussed.

As a “rescue” treatment for refractory cases of PF, high dose GC pulse therapy has been reported to be successful. Pulse therapy is followed by ½ mg/# bid of prednisolone and then taper as described previously. There are 2 protocols for pulse therapy:
1. 11 mg/kg of methylprednisolone sodium succinate (mixed w/250 ml of D5W) IV sid x 3-5 days
2. 10 mg/kg once daily for 3 days of prednisone ORALLY

Discoid lupus erythematosus (DLE)
The approach to diagnosing DLE is the same as PF- signalment, detailed history, physical findings, histopathology changes and response to therapy. In the dog, DLE is the 2nd most common autoimmune skin disease. The author has never recognized it in a cat. It has been suggested that there is no age predilection, but in the author’s experience it seems to be more common in young to middle aged-dog. Collies, Shelties, German shepherd dogs, Siberian huskies and Brittany spaniels are at risk breeds.

Clinical findings include depigmentation, erythema, erosions, crusts and alopecia. When the nasal planum is first affected there is loss of its normal cobblestone appearance and it develops a slate gray appearance. Depigmentation, erythema, erosions and crusts may occur over time. DLE usually begins on the nasal planum and may process to involve the bridge of the nose. It may also involve the lips, periorcular region, pinnae, and genitalia. Dogs affected with DLE are not clinically ill.

Differential diagnoses may include mucocutaneous pyoderma, pemphigus complex, cutaneous drug reaction, erythema multiformae, cutaneous lymphoma, uveodermatologic syndrome, SSC, solar dermatitis/collie nose and systemic fungal infections.

96 Griffies JD, Mendelsohn CL, Rosenkrantz WS et al Topical 0.1% tacrolimus for the treatment of discoid lupus erythematosus and pemphigus erythematosus in dogs. Veterinary Dermatology, 2002; 13: 211–229
100 Scott DW, Miller WH Jr, Griffin CE. Miscellaneous skin diseases. In: Scott DW, Miller WH Jr, Griffin CE, editors. Muller and Kirk’s Small Animal Dermatology, 6th
Mucocutaneous pyoderma (MCP) (the author feels a better name is “antibiotic responsive dermatitis” since bacteria are not seen histologically) is a crusting disease that may affect the lips, nasal planum (exclusively), the bridge of the nose, periocular region, genitals or anus. Clinically it is indistinguishable from DLE. There is no identifiable cause for this disease and the diagnosis is based on the signalment (adult dog, most commonly in German Shepard Dogs (or mixes)), clinical appearance and distribution of the lesions and most importantly response to antibiotic therapy. In the past MCP was differentiated from DLE based on histopathologic findings. DLE was diagnosed when a lichenoid lymphocytic to lymphoplasmacytic interface dermatitis with hydropic degeneration and/or individual necrotic keratinocyte involving the basal cell layer, pigmentary incontinence and a thickened basement membrane was present. Mucocutaneous pyoderma would be diagnosed histologically when a lichenoid plasmacytic to lymphoplasmacytic infiltration was present without an interface change and without basal cell damage. HOWEVER, this criterion has been called into question with a study that reported that histologically mucocutaneous pyoderma and DLE are indistinguishable! In that study, dogs were separated, based on histologic findings, into 3 groups, ones with lymphocytic lichenoid interface dermatitis with hydropic degeneration; ones with plasmacytic lichenoid dermatitis, and lastly ones with a mixture of the first 2 patterns- lymphoplasmacytic lichenoid, interface dermatitis with hydropic degeneration. The authors then evaluated whether the group responded to antibiotics or immunomodulating therapy. There was no statistical difference when histopathologic features were compared between the 2nd and 3rd groups! The author now believes that all cases of canine nasal dermatitis should have a 30-day course of cephalexin prior to immunomodulating therapy- in fact prior to biopsy a 3-4-week course of a cephalosporin is appropriate and may establish a diagnosis without needing to biopsy the lesion!

A better way to approach cases of nasal dermatitis that presents clinically as the “typical” DLE is to recognize that is a reaction pattern rather than a disease. This reaction pattern (lymphoplasmacytic lichenoid nasal dermatitis) may be antibiotic responsive (MCP) or may require immunomodulating therapy (DLE). Since the biopsy findings will be identical in both cases as will the physical findings, if “DLE” is a differential, a 30-day trial of a cephalosporin prior to biopsy should be administered.

**Diagnosis**

Dogs with DLE/MCP are clinically healthy and are normal hematologically and serologically (including a negative ANA). Historically the histopathologic changes consistent w/DLE/MCP included a lymphocytic to lymphoplasmacytic lichenoid interface dermatitis w/hydropic degeneration of basal keratinocytes. Scattered apoptotic keratinocytes may also be present. Failure to respond to a 30-day course of a cephalosporin is also required to differentiate the antibiotic responsive form vs the immune mediated responsive form.

**Treatment**

When treating dogs with DLE it is important to avoid aggressive therapy since it is primarily a cosmetic disease. Occasionally the lesions seem to bother the dog because of pruritus. It is therefore important to treat cases in proportion to the severity of the symptoms. Be sure that the therapy is not worse than the disease. The author treats this disease in a stepwise progression with each step added to the previous therapy except where noted. The steps are as follows: Cephalexin 10-15 mg/# bid- tid for 30 days (since DLE and MCP are indistinguishable); if the dog does not respond to the cephalexin, and a biopsy has not been performed, then a nasal biopsy needs to be done. If the diagnosis of LPLD is confirmed but the dog has not responded to cephalaxin, then the cephalexin is discontinued and the following treatment is begun, sun avoidance, sun screens and vitamin E and omega 3 fatty acids. Depending on the severity, either topical steroids (if localized and very mild) or oral niacinamide and doxycycline (mild to moderate disease) are begun as previously described. If after 60 days, of using topical therapy the dog doesn’t respond to this treatment and the next step is to try oral niacinamide and doxycycline. If after 60 days, there is no response, or from the onset the disease is moderate to severe, then stop the doxycycline and niacinamide (if receiving it) and begin systemic prednisolone (anti-inflammatory doses) that is slowly weaned over a period of months to achieve the lowest possible dose. If that is ineffective, first confirm the diagnosis before going to immunosuppressive therapy. If the diagnosis is confirmed and the previous therapies are ineffective, then begin treatment as you did for pemphigus.

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The mite Demodex spp., which belongs to the Class Arachnida, Order Acarina, lives in hair follicles of all mammals. Demodex canis is the dog follicular mite while in cats it is D. felis (in cats). In dogs Demodex injai is found within sebaceous glands and ducts. D. cornei and gatoi live in the stratum corneum of dogs and cats respectively. They complete their life cycle in about 30 days and the adults will survive for about 21 days. The life cycle of demodex is that an egg (fusiform shaped) develops into 6 legged larvae that then develops into an 8 legged nymph (differentiated from an adult by it lack of an “armor-like” breastplate). This nymph then matures into an adult.

Neonates are thought to acquire mites from their dam/queen via direct skin-to-skin contact during nursing. Direct transmission, other than from dam/queen to the pup/kitten, only occurs with D. gatoi in cats.

In a normal animal the mite does not cause any symptoms. However in some dogs it may cause either localized or generalized disease. There is no universally accepted definition of localized vs generalized disease but recently it has been suggested that with localized disease there are no more than four lesions with a maximum diameter of to 2.5 cm. Demodex is also categorized based on age of onset—those less than 12 months of age (18 months in large or giant breeds) are considered juvenile onset while older dogs are considered adult onset. The prognosis is excellent for the localized form either in puppies or adult dogs while the generalized form carries a more guarded prognosis.

Demodex causes disease when there is an overgrowth of the commensal mites either associated with a genetic defect (juvenile onset) or immune suppression (adult onset). In the adult dog, hyperadrenocorticism (iatrogenic or spontaneous), hypothyroidism, leishmaniasis, or chemotherapy are the most identifiable causes of adult onset generalized demodexis. Note that contrary to what was previously taught, “dogs with adult onset demodexis have cancer or some other very serious life threatening disease”, in the author’s experience, idiopathy is the rule not the exception. In a retrospective study, less than 50% of the adult onset generalized demodexis cases had an identifiable underlying cause.

The lesions associated with demodexis include non pruritic alopecia, scaling, follicular casts, follicular papules/pustules (if a secondary bacterial infection is present), comedones, crusts, erythema, hyperpigmentation, and lichenification. Pruritus is variable but is mild except in cases with a secondary bacterial folliculitis.

Lesions frequently involve the face and/or forelegs and may progress to affect other body sites. Since the lining of the external ear canal is epidermis, demodexis may cause a bilateral ceruminous otitis externa. As the disease progress dogs may develop a deep bacterial folliculitis and furunculosis and draining tracts. In these cases peripheral lymphadenopathy, lethargy and fever are commonly present. In some patients their presentation is exclusively pododemodexis. These cases a deep bacterial folliculitis and furunculosis is frequently present and the feet are swollen and painful leading to lameness.

In contrast to D.canis and cornea, D. injai tends to be associated with a greasy hair coat on the dorsum of the trunk. Many times alopecia is not present and only a low number of mites may be found on skin scrapings. It has been reported that terriers, especially wire haired fox terrier and West Highland white terrier, are at risk of developing this form of demodexis.

Since demodexis is a folliculocentric disease it will look identical to follicular lesions caused by a bacterial pyoderma and dermatophytosis. Because of the similarity in appearance these folliculitides, clinical appearance is not an acceptable method to rule-in or rule-out demodexis. Superficial (for D.cornea) and deep skin scrapings (for the other species of demodex) are the most reliable and cost effective method to diagnose demodexis. In medium or long haired dogs, clip a small “window” in the hair coat to get easier access to the skin and to prevent the loss of the scraped material into the surrounding hair. Skin scrapings are performed with a No. 10 scalpel blade after dulling the blade on the frosted end of the microscope slide.

To perform a deep skin scraping it is best to squeeze the skin prior to and during the scraping to push the mites out of the hair follicles. Scape the skin in the direction of hair growth until capillary bleeding occurs. When lesions are present on the face or paws the animal should either be sedated before scraping or a hair pluck/trichogram may be performed in an awake animal. Hair plucks are performed with mosquito hemostat forceps that grasp and pull out hairs. It is best to collect hairs from the leading edge of the lesion. To increase your yield, squeeze the skin as you are plucking the hairs and be sure to collect a large number of hairs (50–100). Take the collected hairs and lay them on a slide containing a drop of mineral oil and add a cover slip. Sample multiple sites in each patient. Trichograms, or in cases of pustular demodexis examination of the exudate, will detect Demodex mites in about 85% and 100% of dogs respectively with demodexis.

If the trichogram is negative but other sites are positive, sedation and skin scrapings of the feet should be performed since the mites may be present even if the feet appear alesional. It has been the author’s experience that pododemodicosis, if present, is usually the hardest component of generalized demodexis to resolve and so should be used as one of the monitoring sites.
Recently it has been reported that applying tape to a skin lesion and then squeezing the skin is as an effective way to identify demodex mites in dogs. A study was performed to confirm this observation. Specifically the study was to evaluate and compare the sensitivities of acetate tape impression deep skin scraping for the diagnosis of canine demodicosis. They concluded that squeezing the skin followed by acetate tape prep was found to be as sensitive as deep skin scraping for the diagnosis of canine demodicosis. Unfortunately the author has not had the same experience. So if you want to do it as a screening test, in difficult to handle dogs or sensitive locations on the dog, be sure to follow it with deep skin scrapings (with sedation if needed) if the tape prep is negative.

Be sure to collect samples from multiple sites and note the site that the sample is collected from since localized disease is treated differently than generalized disease. When examining the slides you need to evaluate for the approximate number of each stage that is present (eggs, larva, nymph and adults). Also note how many of the mites alive vs are dead. These results will be important to compare to future skin scrapings as you are monitoring the dog’s response to therapy. With effective treatment a decreasing number of immature mites and the disappearance of eggs should occur. The number of live mites should also decrease. In all cases of demodicosis be sure to perform an examination of an otic swab. Otodemodicosis is identified by collecting roll swabs from each ear using a cotton swab that has been dipped in mineral oil. The sample collected is place onto a glass slide that also has a drop of mineral oil on its surface. A cover slip is applied and then the sample is examined.

If samples are collected as described it would be extremely uncommon to miss the presence of demodex mites. Occasionally this may occur, even with properly performed skin scrapings and hair plucks, if the dog has scarring due to chronic disease or because of the thickness of their dermis (therefore the deeper depth of their hair follicle making expulsion of the mite more difficult) (i.e. Shar-Pei). If demodicosis is strongly suspected, but no mites are found on skin scrapings and hair plucks, skin biopsy is recommended to rule in or rule out their presence.

How to treat a dog with demodicosis depends on whether it is localized or generalized. In cases of localized demodicosis, less is best. In many cases, especially juvenile onset, the disease will spontaneously resolve within a couple months. Miticidal therapy is not required unless the disease becomes generalized. Since the progression of localized disease to more generalized form is not influenced by whether the localized form is treated or not, treatment of localized disease is not necessary. However, in the author’s practice “benign” topical treatment is prescribed. This is done so that if the disease does progress, the owner feels that something had been done to try to prevent for occurring. Topical therapy with benzoyl peroxide shampoo and/or gel can theoretically be helpful due to its antibacterial properties and follicular flushing activity. Due to its suppressive effect on the immune system you should avoid using any steroid containing product (topically or systemically) in patients with demodicosis (localized or generalized). Ensuring a proper diet and intestinal deworming program should also be part of the treatment of dogs with demodicosis. To evaluate the effectiveness of treatment, a follow up examination, including repeating skin scrapings, should be performed in 30 days.

Treating a dog with generalized demodicosis requires much more aggressive therapy than localized. Multimodal therapy, a common approach that is used to treat other diseases (eg arthritis, atopic dermatitis or congestive heart failure) will be necessary when treating generalized demodicosis. Acaricidal therapy and treating secondary bacterial infections if present is required for both adult and juvenile onset disease. In adult onset cases attempts should be made to identify and treat the underlying systemic disease.

Dogs with juvenile onset generalized demodicosis, in addition to the above mentioned treatment should be neutered. This is important not only to prevent the propagation of this genetic defect but also estrus may trigger recurrence of clinical disease.

As mentioned previously, in cases of adult onset generalized demodicosis attempts should be made to identify and treat the underlying disease. Evidence shows that successful treatment of an underlying cause increases the likelihood that adult onset demodicosis can be cured. In the author’s practice, diagnostics performed in cases of adult onset generalized demodicosis include a CBC, serum chemistry profile and a urinalysis. Depending on the age of onset, abdominal ultrasound and thoracic radiographs may be included in the minimum data base. Because of the influence that bacterial pyoderma or generalized demodicosis has on evaluating thyroid or adrenal gland disease, evaluation of these organs is delayed until any secondary bacterial infection has been resolved and the demodicosis has improved or is in remission.

Specific treatment of generalized demodicosis is outline in table 1. This table is the result of the most recent consensus guidelines written by an international group of dermatologists. The author has indicated in bold the approach used in his practice. However other therapies have come to the forefront since these guidelines have been published. Specifically the isoxazoline class of ectoparasiticides (fluralaner, afoxolaner and , sarolaner). These products have a broad spectrum of insecticidal and acaricidal activity. Besides their efficacy against fleas and ticks, there are limited studies reporting the effectiveness of these drugs against a variety of mites including demodex, sarcoptes and otodectes. Given their ease of use and safety many practitioners are using these products as a first line treatment for canine demodicosis. The following is the most current information concerning these products effectiveness against canine demodicosis.
Fluralaner (Bravecto)- a study was done in 2015 that compared the efficacy of oral Bravecto™ (fluralaner) with the efficacy of topically applied Advocate/Advantage multi® (imidacloprid/moxidectin) for the treatment of generalized demodicosis (GD) in dogs104. In this study 16 dogs, all over 12 months of age that had been diagnosed with generalized demodicosis, were randomly assigned to being treated with either 1 dose of fluralaner or 3 doses (q 28 days) of imidacloprid/moxidectin. Dogs were examined (and had skin scrapings) at the beginning of the study and then every 28 days for 12 weeks. The results revealed a 99.8% reduction in mite numbers on Day 28 and 100% on Days 56 and 84 after 1 dose of fluralaner. Mite numbers in the dogs treated topically on three occasions at 28-day intervals with imidacloprid/moxidectin were reduced by 98.0% on Day 28, by 96.5% on Day 56 and by 94.7% on Day 84. The biggest drawback in this study was that the dogs were only followed up for 12 weeks so that we don’t know the relapse rate. Since juvenile onset GD has a higher success rate than adult onset GD it would have been beneficial to stratify the dogs into 2 groups based on age of onset.

Also in 2015 another study evaluating the efficacy of fluralaner for the treatment of canine demodicosis was reported105. One hundred sixty three dogs of different breeds with GD. Animals were divided into two age groups based on age at presentation: group one, 2–18 months (62.6%) and group two, over 2 years of age (37.4%). Dogs were treated with fluralaner (25 mg/kg) orally, twice three months apart. Skin scraping and/or hair plucking were performed 1, 2 and 3 months after the first fluralaner administration. The overall response to therapy was 100%. The majority of dogs (87.1%) had negative skin scrapings at the 30 day exam (note that this was an abstact so they didn’t state if the negative skin scraping was only in reference to live mites or to any stage or fragment of a mite). Twenty-one individuals (12.9%) (all belonging to group two) needed two months after the initial fluralaner administration to achieve negative scrapings. As with the previous study, no long term follow up was performed so relapse rate is unknown. Note even though the drug insert states that the dog needs to be > 6 months old to administer fluralaner the label in Europe states it should not be used on puppies less than 8 weeks old and/or dogs weighing less than 2 kg. The FOI sheet states that it is not a safety issue, that the margin of safety in 8 week old puppies is adequate but substantial evidence to support a12-week duration of effectiveness in dogs less than 6 months of age has not been demonstrated.

Because fleas are a concern in the authors practice and many of these dogs need frequent bathing initially the author used a combination of ivermectin daily and 1 dose of Bravecto every 3 months. Note that it has been shown that the concurrent administration of fluralaner and ivermectin (0.3 mg/kg) does not alter the pharmacokinetics of either compound. Based on the plasma pharmacokinetic profile and the clinical observations, there is no evident interaction between fluralaner and ivermectin, and co-administration does not increase the risk of ivermectin associated neurotoxicity. The response to this combination was less than that of the studies previously discussed so this practice has been discontinued. It is theorized that because Bravecto is a selective inhibitor of arthropod γ-aminobutyric acid- and l-glutamate-gated chloride channels and ivermectin’s mechanism of action is that it enhances the effects of glutamate at the invertebrate-specific glutamate-gated chloride channel, with minor effects on gamma-aminobutyric acid receptors.

Afoxolaner has also been studied for treatment of generalized demodicosis. A 3 month study was performed in which 16 dogs with generalized demodicosis were divided into 2 treatment groups.106 In the first group the eight dogs were treated w/afoxolaner (NexGard® ) at the label dose but given every 14 days (days 0, 14 and 28) and then 1 monthly treatment (day 56). The other 8 dogs were treated with topical imidacloprid/moxidectin (Revolution/Advocate®, Bayer) at the same treatment interval. The mite reduction is listed below

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105 Karas-Tecza J, Dawidowicz J Efficacy of fluralaner for the treatment of canine demodicosis, Veterinary Dermatology,2015; 26, 307
There were statistically significantly fewer mites found on Days 28, 56, 84 in the afoxolaner group. The authors concluded that afoxolaner given orally every 2 weeks for 3 treatments, then monthly is highly effective against generalized demodicosis, within 2 months BUT a few things to point out= The study states that On Day 84, no live mites were recorded for any dog in the Nexgard- treated group- the key word is LIVE mites. This is apparent because later in the article it states that a significant portion of the afoxolaner treated dogs (7/8) had no mites in their skin scrapings at Day 84. So I interpret this to mean that at day 84 there were still mites present on the 1 dog treated with Nexgard, perhaps dead or maybe fragments of the mite. This impression is further supported by the statement that “in the present study, seven out of eight NexGard_-treated dogs had two successive negative skin scrapings at a one-month interval, indicating that treatments at appropriate intervals can provide remission of the disease”. So the key to this is that there needs to be 2 consecutive negative skin scrapes to declare the demodicosis in remission so not all dogs were in remission. Other issues are they didn’t say how old the dogs the dogs were other than > 6 months of age – it is important to stratify the groups into adult and juvenile onset since the later is in general more difficult to get in remission and in fact juvenile onset generalized demodicosis may self cure. They didn’t state if any of the dogs had previously been treated for demodex- only that they had not received an ectoparasiticide or macrocyclic lactone for at least 12 weeks prior to Day 0, as far as it could be reasonably established by verbal communication with the owners. Lastly they didn’t state how long the dogs had generalized demodicosis before entering into the study..

There was another study\(^\text{107}\) using afoxolaner for the treatment of generalized canine demodicosis that involved 4 dogs- ages 8 months to 10 years of age. All dogs had been affected for at least 2 months. These dogs were treated at label dose on day 1 then at 4 weeks and 8 weeks after the initial dose. There was a reduction of live mites by week 4. All 4 dogs were negative for live mites 8 and 12 wks after treatment. The problems with this study are the same as the previous study using afoxolaner other than it did state that the dogs were affected for at least 2 months with generalized demodicosis. The author reported that at the time of preparation of the paper, 6 months after the initiation of treatment with afoxolaner, all four4dogs remain clinically free from clinical signs of demodicosis. The problem is that it has been shown that clinical remission is not the same as parasitic remission – the later be required to state that the dog is in remission and can only be determined by skin scrapings.\(^\text{108}\) Note that the product insert contains a caution about use in dogs with a history of seizures.

Sarolaner was recently studied\(^\text{109}\) where it was compared to topical imidacloprid-moxidectin for the treatment of generalized demodicosis. Sixteen dogs over the age of 6 months were entered into the study. The dogs were divided into 2 groups- group 1 was treated w/sarolaner orally on days 0, 30 and 60 while the other group was treated with the topical imidacloprid-moxidectin weekly. Efficacy for sarolaner based upon live mite counts was 97.1% and 99.8% on Days 14 and 29, respectively and 100% on all subsequent days. For the topical therapy group efficacy based upon live mite counts was 84.4%, 95.6% and 99.7% on Days 14, 29, and 44, respectively and 100% on all subsequent days. Limitations of this study were they only followed up for 30 days after the last treatment so we don’t know the relapse rate. Since juvenile onset GD has a higher success rate than adult onset GD it would have been beneficial to stratify the dogs into 2 groups based on age of onset. They didn’t state if any of the dogs had previously been treated for demodex and lastly they didn’t state how long the dogs had generalized demodicosis before entering into the study..

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**Table 4. Mite count reduction in treated groups (based on geometric means).**

<table>
<thead>
<tr>
<th>Day</th>
<th>Group 1 - Imidacloprid/moxidectin</th>
<th>Group 2 - Afoxolaner</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>808.1</td>
<td>650.8</td>
</tr>
<tr>
<td>28</td>
<td>82.4*</td>
<td>5.3*</td>
</tr>
<tr>
<td>56</td>
<td>119.9*</td>
<td>6.0*</td>
</tr>
<tr>
<td>84</td>
<td>108.5*</td>
<td>0*</td>
</tr>
</tbody>
</table>

* Group 2 differed statistically significantly (\(p < 0.05\)) from group 1.

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Two studies have evaluated the efficacy of doramectin in the treatment of generalized demodicosis.\textsuperscript{110,111} Both studies used a dose of 0.6 mg/kg body weight given every 7 days; in the first study the drug was administered by subcutaneous injection to 23 dogs and in the second it was administered orally to 29 dogs. Administration subcutaneously appeared to be marginally more effective but the cohort size in each case was relatively small and thus caution needs to be exercised in the interpretation of results. It has been shown that subcutaneous injection of doramectin results in slower drug absorption and greater bioavailability of the drug compared to oral administration\textsuperscript{112} A study therefore was performed in which 400 dogs with generalized demodicosis were treated using weekly s.c. injections of doramectin at a dose rate of 0.6 mg/kg body weight.\textsuperscript{113} Results revealed that 66.7% of the dogs over the age of 4 achieved remission by having two consecutive negative skin scrapings while the overall remission rate based on intent to treat was 86.3%. This may be a good option for those dogs in which people are unable to give oral ivermectin due to the taste of the ivermectin.

It is interesting that when you look at any of the treatment protocols it seems none are 100% effective so the bottom line is to start with the easiest, safest and least expensive treatment and if that is ineffective to try another treatment.

Remember, regardless of the selected treatment, miticidal therapy should be followed up with skin scrapings since dogs may look normal clinically but still have active disease (as determined by the presence of mites on skin scrapings) treatment must be continued beyond clinical resolution. Parasitic cure is defined as multiple negative skin scrapings, including lack of dead or fragmented mites, on 3 consecutive monthly visits. Skin scrapings should be used to determine the therapeutic end-point. This end point is reached when the dog looks normal clinically and skin scrapings have been performed monthly ALL areas that have EVER been positive on skin scraping and have been negative for 3 consecutive visits. If during a visit the skin scraping is positive, it is important to compare the number of live and dead mites and the number of each stage of the mite life cycle to the previous visit. An indication of effective treatment is that during therapy the number of live mites found on skin scrapings and the number of immature mites should be reduced from the previous visit. If this doesn’t occur, therapy should be re-examined and possibly changed.

Diagnosis and treatment of demodicosis is an important concept that all small animal practitioners should feel comfortable with. By taking time to thoroughly examine and evaluate the dog, and spending time explaining the disease to the owner, the outcome will usually be successful.

Table 1- Summarized treatment of canine demodicosis * (Items in bold are the author’s preferences)

<table>
<thead>
<tr>
<th>Treatment of a dog with severe generalized disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Perform cytology and if there is evidence of a deep bacterial skin infection or the dog has been treated previously with antibiotics a bacterial culture and sensitivity. With inflammatory cells and bacteria present, appropriate oral antibiotic therapy is required.</td>
</tr>
<tr>
<td>2 Use topical therapy with chlorhexidene or benzoyl peroxide shampoo weekly to possibly twice weekly. (Unless amitraz is being applied)</td>
</tr>
<tr>
<td>3 There are several treatment options for the treatment of canine demodicosis. The best option will depend on the legalities pertaining to the use of veterinary pharmaceutical products in the country of residence, the finances of the owner and the clinical situation. However, independent of the treatment specifics the dog should be neutered because dogs in need of mite treatment should not be allowed to breed, and the disease may relapse in cycling bitches.</td>
</tr>
<tr>
<td>a. Ivermectin at an oral dose of 0.3–0.6 mg/kg (0.4 mg/kg) or moxidectin at 0.2–0.5 mg/kg p.o. daily are further options. Note- many herding breed dogs have a genetic predisposition to adverse drug reactions involving ivermectin due to a defective MDR-1 gene. This gene is responsible for pumping drugs out of the mammalian’s brain. When this gene is defective, drugs accumulate in the brain leading to adverse events. Gene testing for the defect can help eliminate at risk dogs but there are a number of dogs with adverse effects to ivermectin and an intact MDR-1 gene due to alternative mechanisms. Thus adverse events may still occur in dogs w/normal MDR-1 genes. Therefore with both drugs, a gradual increase from an initial dose of 0.05 mg/kg to the final dose (of 0.4 mg/kg) within a few days is recommended to identify dogs that cannot tolerate those drugs. Monitoring for neurological adverse effects should occur throughout the course of therapy. Ivermectin is the treatment of choice in the author’s practice.</td>
</tr>
<tr>
<td>b. Amitraz weekly or every 2 weeks in a concentration of (0.025–0.06%) can be used. Dogs with a medium to long hair coat need to be clipped, and skin should stay dry between rinses to avoid washing off the drug. Rinsing should be performed in well-ventilated areas. The author only uses this therapy</td>
</tr>
</tbody>
</table>


if the dog has failed to respond to ivermectin or is a herding breed. Please note that amitraz is EPA registered and doesn’t EVER allow any off label use (label states 1 bottle/2 gallons every 14 days)

c. Milbemycin oxime may be administered orally at a dose of 1–2 mg/kg/day. Moxidectin orally (see below) is in the milbemycin family, is much less expensive than milbemycin, and is used if the dog fails to respond to ivermectin (again a non herding breed)

d. Moxidectin as a spot-on in combination with imidacloprid may be used weekly. This spot-on formulation has a markedly higher success rate in dogs with milder disease or juvenile onset

e. Doramectin weekly at 0.6 mg/kg p.o. or SQ is a possible treatment. A gradual increase from an initial dose of 0.1 mg/kg to the final dose seems prudent to identify dogs that cannot tolerate the drug and will show neurological adverse effects.(SEE PREVIOUS DISCUSSION ABOVE ABOUT PO VS SQ)

So to summarize- this report states that “There is good evidence for the efficacy of weekly amitraz rinses and daily oral macrocyclic lactones such as milbemycin oxime, ivermectin and moxidectin for the treatment of canine demodicosis.”

Other recommendations are

- Dogs should be evaluated monthly, and treatment should be continued until 3 consecutive visits with multiple negative skin scrapings have been achieved.
- Treat secondary bacterial infections
- Factors predisposing to demodicosis, such as malnutrition, endoparasites, endocrine disease, neoplasia and chemotherapy, should be identified and corrected to maximize response to therapy.

STEP 1: Identify the primary cause of the otitis. Primary causes of otitis externa include parasitic diseases, hypersensitivity disorders, foreign bodies, disorders of keratinization, juvenile cellulitis, autoimmune diseases, neoplasia, and polyps. These are the conditions or disorders that initiate the inflammatory process within the ear canal. In a retrospective study evaluating 100 dogs with acute and chronic-recurrent otitis externa, the most common primary cause of the otitis was due to allergic dermatitis (n=43 dogs). On the other hand, in the cat, the most common causes of recurrent otitis externa are polyps, parasites (e.g. Otodectes cynotis) and allergies.

STEP 2: Identify the predisposing factors of otitis. Predisposing factors facilitate the inflammation by permitting the external ear canal microenvironment to be altered allowing pathogenic or opportunistic bacteria to become established. These factors may include variation in ear conformation (pendulous pinnae, hair in the ear canal, congenital stenosis of the ear canal), moisture in the ear, and inappropriate prior therapies. Question the owner about previous treatments, the use of cotton-tipped applicators, or hair removal from the ears. It is important to eliminate as many of these factors as possible, realizing that some of these, such as ear conformation, cannot be changed.

STEP 3: Identify the perpetuating factors of otitis. Perpetuating factors sustain and aggravate the inflammatory process and prevent resolution or worsen an already present otitis externa. Bacterial and yeast infections, otitis media, and progressive pathologic changes (e.g. hyperplasia) are perpetuating factors of otitis externa, which need to be identified and controlled.

STEP 4: Treat the present otic infection with topical ear cleaning and drying agents, topical antimicrobial agents and topical glucocorticoids (if needed). If the ears are stenotic and hyperplastic, systemic glucocorticoids are indicated as well. Treat any other concurrent skin conditions (e.g. superficial bacterial pyoderma, yeast dermatitis). Otic preparations that are ointment/suspension-based may not be as effective as those that are solution/emulsion-based, if the ears are stenotic or hyperplastic, as may be the case in those patients with chronic otitis externa, but may be utilized if the ears are not stenotic or hyperplastic.

STEP 5: Recheck the patient in three to four weeks to assess response to therapy, by performing an otic examination and otic cytology in addition to the general examination. This step is so critical to the management of otitis. If the patient is responding, initiate a food trial, if the otitis and pruritus (if present) is non-seasonal. In cases of seasonal otitis and pruritus, where other causes of the otitis and pruritus have been ruled out, a diagnosis of atopic dermatitis is made, and allergy testing or symptomatic therapy are initiated. If, however, the ears have not responded, go to Step 6: and schedule a deep ear flush, to clean the ears and evaluate the patient for concurrent otitis media. In dogs with recurrent ear infections of 6 months or longer, up to 82% of these dogs may have concurrent otitis media, with 70% having an intact but abnormal tympanic membrane.

STEP 6: A short course (two to three weeks) of glucocorticoids should be utilized prior to the deep ear flush to decrease inflammation and stenosis of the horizontal and vertical ear canals. The deep ear flushing procedure is best done under general anesthesia in order to completely clean the ear. Once the animal is under anesthesia, prior to the deep ear flush, radiographic imaging of the tympanic bulla is performed to stage the ear disease, remembering that normal radiographic imaging does not rule out otitis media. Next, the external ear canal is soaked for 10 minutes with a non-ototoxic ceruminolytic ear cleaner, like Cerumene. The ear is then flushed with warm sterile isotonic saline using a bulb syringe to remove large debris and exudate. This is followed by flushing with warm sterile isotonic saline using an 8 French polypropylene urinary catheter attached to a 12 cc syringe passed through an otoscopic cone. Once the ear is clean, the tympanic membrane is evaluated with an otoscope or video otoscope. If the tympanic membrane is not intact, cytology and bacterial C/S is performed from the middle ear cavity. This may be performed using the hand-held otoscope or the video otoscope. Using a hand-held otoscope, a sterile otoscopic cone is inserted into the horizontal ear canal and a sterile pediatric-size swab is passed into the middle ear cavity. The first swab is used for C/S. A second swab is passed into the middle ear for cytological analysis. If the video otoscope is used, an open-end 3 1/2 French Tom cat catheter or 5 Fr polypropylene urinary catheter attached to a 12 cc syringe is placed through the port of the otoendoscope. Warm sterile isotonic saline is flushed into the middle ear cavity and aspirated back, the first sample for cytology, and the second sample for culture. Then the middle ear is flushed repeatedly with warm sterile isotonic saline to flush the middle ear cavity. If the tympanic membrane is intact, appears abnormal, and otitis media is suspected, a myringotomy is needed to obtain samples for cytology and bacterial C/S, and to flush the middle ear cavity. In the dog, an intact tympanic membrane does not rule out the possibility of otitis media. Using a hand-held otoscope, a sterile otoscopic cone is inserted into the horizontal ear canal and the tympanic membrane is visualized. Using a sterile swab, an incision is made into the caudoventral quadrant of the tympanic membrane, specifically the pars tensa. The swab used for the myringotomy incision is submitted for bacterial C/S. A second swab is inserted into the original incision and the sample obtained is used for cytological analysis. If the video otoscope is used to perform the myringotomy, an open-end 3 1/2 French Tom cat catheter or 5 Fr polypropylene urinary catheter is placed through the port of the otoendoscope, and is used to make the incision. Saline is
flushed into the middle ear cavity and aspirated back using a 12 cc syringe attached to the catheter, and the first sample is for cytology, and the second sample is submitted for bacterial C/S. The normal tympanum heals in 21 to 35 days. Therefore, if the ear is kept free of infection after the myringotomy procedure, the tympanic membrane should heal. Possible complications of ear flushing and myringotomy are Horner’s syndrome, facial nerve paralysis, vestibular disturbances, and deafness. Owners should understand these complications and sign a consent form prior to the procedure. After the otic flush, it is important that the patient is sent home on empiric topical and systemic therapy based on cytology, and the oral antibiotic treatments (only needed if dog has infectious otitis media) may be modified once the cultures have been completed. Ointment or suspension-vehicle otic products should be avoided if a myringotomy was performed or if the tympanic membrane was torn or ruptured. As discussed in step 5, if the patient has non-seasonal otitis and pruritus, a food trial is commenced while in cases of seasonal otitis and pruritus, a diagnosis of atopic dermatitis is made, and allergy testing or symptomatic therapy are initiated.

**STEP 7:** Recheck the patient three to four weeks after the ear flush to monitor the response to otic treatments as well as to the food trial (if a food trial was performed). In most cases of chronic otitis externa, where continual inflammation and stenosis have occurred along with increased cerumen production, which may alter epidermal migration, some type of maintenance otic therapy is required, such as a cleaning and drying agent, to keep the ear canal free of wax build up.
Once an otic infection has been diagnosed, treatment may include topical as well as systemic therapy. At each recheck, the patient’s response to treatment should be monitored, cytology performed, and products changed accordingly. In the majority of the cases of infectious otitis externa, topical therapy alone is sufficient. In those ears with severe infections, or those that have long-standing chronic otitis externa, the addition of a systemic antimicrobial agent may be required to clear the infection that is present in the ear tissue as well as in the lumen of the ear canal. For those dogs with infectious otitis externa and otitis media, topical and systemic antimicrobial therapy is usually required.

Topical otic preparations usually contain various combinations of glucocorticoids, antibiotics, and/or antifungals in a vehicle base. Selection of the active ingredient needed in the product for topical use should be based on cytology. It is important to remember that C/S results indicate the plasma level of an antimicrobial agent. The advantage of topical therapy is that you can achieve 100 to 1000 times the plasma level of the antimicrobial agent by administering it topically. The patient’s progress while on these medications should be monitored cytologically at each re-evaluation and the topical therapy adjusted accordingly.

None of the commercially available otic topical treatments or the extra-label otic preparations are labeled for use with a non-intact tympanic membrane. However, most all of these products have been used to treat otic infections in dogs with otitis media. Always warn the owner of the possibility of neurological signs of ototoxicity while administering topical medications when the tympanic membrane is not intact. The otic topicals that I will not use in the ear with a non-intact tympanic membrane are those in an ointment or suspension base.

Glucocorticoids are antipruritic, anti-inflammatory, and antiproliferative. During the acute stage of otitis, the ear canal becomes edematous and erythematous. As the inflammation progresses, the dermis becomes infiltrated with a mixed population of cells. Apocrine glands dilate and become hyperplastic, which leads to excessive cerumen production. Therefore, glucocorticoids are beneficial in decreasing the pain, pruritus, stenosis, and edema associated with otitis. In addition, they are effective in decreasing sebaceous and apocrine secretions. They are usually in combination with other agents but may be beneficial when used alone in allergic cases of otitis and some ceruminous otitis cases. It is important to use the lowest potency glucocorticoid at the lowest frequency needed to control the otitis to prevent iatrogenic hyperadrenocorticism. Examples include Cort/Astrin Solution (Vedco) and Synotic (Zoetis).

Topical aminoglycosides such as neomycin and gentamicin have good activity against gram-positive and gram-negative otic pathogens for treatment of acute otitis externa. Gentamicin and neomycin are available in many combination products, some which contain an antifungal and glucocorticoid such as Tresaderm (Merial), Otomax Ointment (Merck), Mometamax Suspension (Merck), Panolog Ointment (Zoetis), easOtic Suspension (Virbac). Another aminoglycoside, tobramycin, is available as an ophthalmic solution and is very effective against *Pseudomonas* otitis infections, especially in those cases of chronic otitis externa: Tobramycin Ophthalmic Solution (generics)

Fluoroquinolones have a broad spectrum of antibacterial activity against gram-negative and gram-positive bacteria and are found in Baytril Otic Emulsion (Bayer) and Posatex (Merck).

Polymyxin has excellent *in vitro* activity against *Pseudomonas* with resistance rarely developing but is inactivated in purulent debris so the ear needs to be kept clean during treatment. Products containing polymyxin B are Neomycin, polymyxin B, and hydrocortisone (generics) and Surolan (ELANCO).

Florfenicol has been available for a number of years as a fast-acting, long-lasting injectable antibiotic for treatment of bovine respiratory disease. Recently, two new otic medications have been approved for the treatment of bacterial (*Staphylococcus pseudintermedius*) and yeast (due to the addition of terbinafine in the products). They also contain a topical glucocorticoid. These otic products are Claro (Bayer) and Osurnia (ELANCO).

Tris-EDTA is a topical product that enhances the activity of topical antibiotics against otic pathogens by decreasing stability and increasing the permeability of the cell wall of gram-negative bacteria. There are numerous products containing Tris-EDTA: Triz-EDTA (Dechra), TrizUltra (with ketoconazole) (Dechra), TrizChlor (with chlorhexidine) (Dechra), Mal-A-Ket Plus TrizEDTA Flush (with ketoconazole and chlorhexidine) (Dechra) and T8Keto (with ketoconazole) (Bayer).

Antifungal agents are used in cases of otitis caused by *Malassezia* or *Candida*. Ingredients that are active against yeast include nystatin (Panolog), thiabendazole (Tresaderm), miconazole (generics, Surolan, easOtic), ketoconazole (TrizUltra, T8keto), posaconazole (Posatex), clotrimazole (Otomax, Mometamax), and terbinafine (Claro, Osurnia).

An extra-label topical preparation containing enrofloxacin may be formulated using 1 part of the injectable enrofloxacin (22.7 mg/ml) added to 4 parts of an appropriate vehicle (Cort/Astrin for example) or 1 part injectable enrofloxacin to 1 part miconazole to 1 part polymyxin B to 1 part neomycin.
Topical dexamethasone (2 mg/kg). The results of cytological examination of otic exudate are the basis for the selection of the active ingredient. Otic preparations that are ointment/suspension-based may not be as effective as those that are solution/emulsion-based, if the ears are not stenotic or hyperplastic, or in an animal with acute otitis externa. First line topical otic medications should be selected for those cases of acute or occasional otitis externa, while second-line otic medications, such as those containing fluoroquinolones, should be reserved for cases of bacterial otitis due to *Pseudomonas* otitis externa or those chronic infections that have not responded to first-line topical otic antimicrobials. Systemic antimicrobial therapy for infectious otitis externa and otitis media is controversial. In dogs with end-stage otitis externa and concurrent otitis media, bacterial organisms may be isolated from the exudate in the lumen of the vertical ear canal and middle ear cavity as well as from the tissue from these sites. Therefore, most agree that systemic antibiotics (based on culture and susceptibility testing) are indicated in patients with otitis media, patients with severe proliferative chronic otitis externa, patients with ulcerative otitis externa, patients where inflammatory cells are seen cytologically (indicating deeper skin involvement) and in patients where owners cannot administer topical therapy. The selection of systemic antimicrobial agent must be made based on C/S from the external ear (for otitis externa) and middle ear (for otitis media). However, therapy may be initiated based on cytologic results while awaiting the results of the C/S.

Indications for systemic antifungal agents are similar to those above for bacterial infections and include patients with yeast otitis media, patients with severe yeast otitis externa, or in patients where owners cannot administer topical therapy. However, otic yeast infections require topical therapy in addition to systemic therapy for resolution. Both ketoconazole (5 mg/kg q24h) and itraconazole (Sporanox 5 mg/kg PO q24h or pulse-dosed 2 days on and 5 days off) have been used in dogs for treatment of yeast otitis. Systemic glucocorticoids are used to decrease stenosis, edema, and hyperplasia of the vertical and horizontal ear canal to allow a complete otic examination as well as allow proper cleaning of the ear. They are also indicated in cases of allergic otitis externa. In older patients, or those with concurrent diseases, it may be necessary to perform bloodwork prior to starting the patient on glucocorticoids. Initially, 0.5-1 mg/kg q24h orally may be needed, followed by a low-dose, alternate day dosing schedule. The lowest dose needed should be administered to prevent the occurrence of side effects with the end goal to discontinue the systemic glucocorticoid.
P-R-U-R-I-T-U-S, the unpleasant sensation of “itch”, is one of the most common presenting signs in dogs and is most often a hallmark feature of allergic skin disease. However, the differential list for pruritic skin disease is legion, and in fact, several pruritic diseases may be occurring simultaneously. Instead of throwing the medicine cabinet at the pruritic dog, a deliberate methodical approach is important in establishing a likely list of differential diagnoses for which further investigation can help confirm a definitive answer. So where does one begin?—the dermatological history (e.g., age of onset; duration of pruritic disease; rash that itches [often implying an infection] vs. itch that rashes [may imply allergic skin disease]; seasonality of itch; pruritic body distribution of itch [Table 1]; other individuals affected; adulticidal flea prevention used appropriately; foodstuffs offered; and response [sores and itch] to previous medication. A thorough history can provide tip-offs as to the cause of disease allowing the clinician to prioritize a list of possibilities that can be critiqued against the physical examination findings and validated with further testing, including in-hospital diagnostics (e.g., scrapes, cytology, culture, +/- biopsy) and at-home diagnostic/therapeutic treatment trials (e.g., response to therapy, elimination diet trial).

Table 1: Common pruritic distribution patterns in the dog

<table>
<thead>
<tr>
<th>Body location</th>
<th>Differentials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pinnae</td>
<td>Scabies, CAFR, CAD</td>
</tr>
<tr>
<td>Otic canal</td>
<td>Otodectosis, bacterial infection, Malassezia otitis, CAFR, CAD</td>
</tr>
<tr>
<td>Face</td>
<td>Demodicosis, MD, CAFR, CAD</td>
</tr>
<tr>
<td>Mucocutaneous junctions</td>
<td>Demodicosis, bacterial infection, MD, CAD</td>
</tr>
<tr>
<td>Elbows</td>
<td>Scabies, pressure point/callus pyodermia</td>
</tr>
<tr>
<td>Dorsal topline &amp;/or lumbosacral</td>
<td>Acute moist dermatitis, postgrooming furunculosis, pediculosis, cheyletiellosis, demodicosis (D. injai), FAD</td>
</tr>
<tr>
<td>Paws</td>
<td>Demodicosis, bacterial infection, Malassezia pododermatitis, CAFR, CAD</td>
</tr>
<tr>
<td>Claw folds</td>
<td>Bacterial infection, Malassezia paronychia</td>
</tr>
<tr>
<td>Body folds</td>
<td>Bacterial infection, MD, CAFR, CAD</td>
</tr>
</tbody>
</table>

CAFR: cutaneous adverse food reactions (food allergy); CAD: canine atopic dermatitis; MD: Malassezia dermatitis; FAD: flea allergy dermatitis

Almost all dermatological patients have a primary (underlying) disease complicated by secondary infections. Although these infections must be identified and eliminated, they will recur unless the primary reason for them is not addressed and managed. A helpful framework to improve overall success in dermatology is to answer the essential questions: “What are the [common] infections?” and “Why are they [infections] there?”. Notice this construct implies infections are likely and there is a reason behind them. The most common canine infections include folliculitis (e.g., superficial staphylococcal pyoderma, demodicosis, dermatophytosis), Malassezia (yeast) dermatitis, and otitis externa (bacterial and/or Malassezia infection). As to the reason for the infections, allergic skin disease, endocrinopathy, cornification disorders, and autoimmune dermatoses. The vast majority of pruritic young adult dogs experiencing recurring infections is allergic skin disease (hypersensitivities and their look-alikes), assuming demodicosis has been excluded with the use of deep skin scrapes or hair plucks. If these non-demodectic, pruritic, young adult dogs are receiving appropriate adulticidal flea prevention, in areas where fleas are endemic, then atopic dermatitis is often the primary disease. For the sake of this lecture series, varying weight will be given to aforementioned diseases, based on what the author sees daily as it relates to canine atopic dermatitis. A detailed description of demodicosis, sarcotic mange, otitis externa, cutaneous adverse food reactions (CAFR), and the pathophysiology of canine atopic dermatitis (CAD) is beyond the scope of these notes.

What are the infections?

Pyoderma

Pyoderma, a pyogenic cutaneous bacterial infection, is one of the most common skin diseases of the dog. Although Staphylococcus pseudintermedius is the most prevalent bacterium recovered from canine pyoderm, other staphylococcal species have been isolated including S. schleiferi, S. aureus, and S. lugdunensis. Pyoderma is almost always secondary to an underlying disease process, mainly demodicosis, allergic skin disease, and endocrinopathies. Consequently, if the underlying cause is not identified and corrected, pyoderma will recur.

Pyoderma tends to affect haired skin, repeatedly traumatized skin (e.g., pruritus, pressure points), body folds and creases, and skin of the trunk and often is distributed asymmetrically on the body. Pruritus may or may not be a feature of the clinical picture. Classification of disease is based on the depth of bacterial infection, which is associated with characteristic lesions and recognized clinical presentations. Recognition of the type of pyoderma (surface, superficial, or deep), along with cytological confirmation of the
The presence of coccoid bacteria, allows for a diagnosis and rudimentary treatment plan (Table 2). Empiric systemic antimicrobial therapy can then be prescribed, when needed, for the majority of first time episodes of pyoderma (Table 2). By far, superficial bacterial folliculitis (SBF) is the most common presentation of pyoderma in the dog.

Antimicrobial-resistant infections is an emerging problem in health care. Staphylococci that have acquired the mecA gene are classified as methicillin- (synonymous with meticillin and oxacillin) resistant, signifying resistance to penicillins, cephalosporins, and carbapenems. Often methicillin-resistant staphylococci (MRS) acquire resistance to other antimicrobials too. In veterinary medicine, MRS are becoming more common for reasons such as, repeated systemic antibiotic exposure (especially fluoroquinolones), subtherapeutic administration of systemic antibiotics (dose and/or duration), failure to identify and manage the underlying cause for repeated infection, and patient contact with human health care workers and/or facilities. Methicillin-resistant S. pseudointermedius (MRSP) is a potential zoonosis, but human infections appear to be rare in immunocompetent people. Transmission of methicillin-resistant S. aureus (MRSA) is mostly from human to pet (reverse zoonosis), but these animals may then be harboring a potential zoonosis. Given the potential for (reverse) zoonosis, veterinarians must practice good infection control practices with each case of pyoderma (e.g., washing hands, cleaning/disinfection), with these measures enhanced when MRS has been documented in the patient (e.g., gloves, protective outerwear, separation of MRS patient from rest of hospital patients).

Pyoderma caused by MRS are clinically indistinguishable from susceptible opportunistic staphylococci. Therefore, the patient’s history, in combination with clinical and cytological findings, provide the clues suggestive of an antimicrobial-resistant infection (Box 1). When evidence exists for one of these infections, bacterial culture (including bacterial speciation) and susceptibility testing is indicated, if systemic antibiotics are thought to be warranted due to the extent and severity of the pyoderma and inherent patient factors. Once MRS pyoderma is documented by culture, simple hygienic measures can be offered to the owner to help reduce fear and enhance patient care (see www.wormsandgermsblog.com, www.bsava.com, and www.thebellamossfoundation.com). At this time, routine screening for MRS in clinically healthy pets is not recommended.

Topical treatments for pyoderma include the use of antibacterial shampoos, mousses/foams, sprays, rinses, ointments, creams, gels, and wipes often with the active ingredients including, but not limited to, chlorhexidine 2-4% (also in combination with miconazole), benzoyl peroxide, ethyl lactate, bleach, and silver-containing products. In general, topical formulations that are not rinsed off will have a longer treatment effect. Given its routine use in human cases of MRSA, topical mupirocin ointment should be avoided in canine MRS pyoderma, unless other topical treatments fail and there are no other suitable treatment options based on culture and susceptibility testing. Though not antibacterial, per se, topical ceramide creams may help improve the barrier function of the skin, especially in atopic dogs, thereby limiting the chance for infection recurrence. When systemic antibiotic therapy is deemed necessary, then the correct antimicrobial and dosage is to be prescribed (Tables 2 and 3). Glucocorticoid use is discouraged during treatment of the pyoderma, as it will alter the clinical picture to the owner and veterinarian alike. If the dog is severely pruritic, oclacitinib should be considered (3-7 days) over glucocorticoids. Regardless of the presence of methicillin resistance, patients should be re-examined near the end of the treatment schedule to ensure clinical resolution of pyoderma or lack thereof. MRS superficial pyoderma can usually be effectively treated with the daily to every other day use of topical treatments, and when possible systemic antibiotics, but time to clinical resolution may take longer for these infections.

Table 2: Classification scheme for canine pyoderma

<table>
<thead>
<tr>
<th>Depth of infection/pyoderma</th>
<th>Characteristic lesions</th>
<th>General treatment plan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surface</td>
<td>Erythema, surface exudate, crusts, erosions, excoriations, slightly raised plaques</td>
<td>--Often only requires topical therapy to remove bacteria and excessive sebum until clinical resolution&lt;br&gt; --Topicals may be used as maintenance therapy once infection is controlled</td>
</tr>
<tr>
<td>Superficial</td>
<td>Erythematous macules, papules, pustules, crusted papules, scale, crusts, epidermal collarettes, erosions, excoriations, hyperpigmentation, lichenification, patchy alopecia</td>
<td>--May only need sole topical therapy for focal lesions&lt;br&gt; --If warranted, systemic antibiotics are prescribed for a minimum of 3 weeks and should be administered for 1 week beyond clinical resolution&lt;br&gt; --Adjunctive topical therapy hastens time to resolution&lt;br&gt; --Highly antimicrobial-resistant infections will need a combination of different topical treatments every 24-48 hours for 1 week beyond clinical resolution&lt;br&gt; --Identifying the underlying cause will reduce the frequency of recurring pyoderma in the future</td>
</tr>
<tr>
<td>Deep</td>
<td>Nodules, plaques, furuncles</td>
<td>--Systemic antibiotics should be selected based off...</td>
</tr>
</tbody>
</table>
Infection of the dermis, subcutis, and/or deeper soft tissue hemorrhagic bullae, fluctuant to firm soft tissue swelling, draining tracts, devitalized tissue, gelatinous skin, ulcers, necrosis, scarring alopecia, hyperpigmentation, skin thickening of a tissue culture antibiogram with treatment continuing for at least 2 weeks beyond clinical resolution (6-12+ weeks in total). --Adjunctive topical therapy hastens time to resolution

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### Table 3: Systemic antimicrobial therapy for canine bacterial skin infections

<table>
<thead>
<tr>
<th>First-Line Empiric Antimicrobial Therapy</th>
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<tbody>
<tr>
<td>Amoxicillin clavulanate</td>
<td>13-25 mg/kg PO q 8-12h</td>
</tr>
<tr>
<td>Cefadroxil</td>
<td>22 mg/kg PO q 8-12h</td>
</tr>
<tr>
<td>Cefpodoxime proxetil</td>
<td>5-10 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>30 mg/kg PO q 12h</td>
</tr>
<tr>
<td>Cefovecin</td>
<td>8.0 mg/kg SQ q 2wks</td>
</tr>
<tr>
<td>Lincomycin</td>
<td>15-25 mg/kg PO q 12h</td>
</tr>
<tr>
<td>Ormetoprim/ sulfadimethoxine</td>
<td>55 mg/kg PO q 24h on day 1 then 27.5 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Trimethoprim/ sulfadiazine or sulfamethoxazole</td>
<td>15-30 mg/kg PO q 12h</td>
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<table>
<thead>
<tr>
<th>Second-Line Culture-Determined Antimicrobial Therapy</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin</td>
<td>15-30 mg/kg SC, IM, or IV q 24h</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>40-50 mg/kg PO q 8h</td>
</tr>
<tr>
<td>Clindamycin¹</td>
<td>11 mg/kg PO q 12h</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>5-10 mg/kg PO q 12h</td>
</tr>
<tr>
<td>10 mg/kg PO q 24h</td>
<td></td>
</tr>
<tr>
<td>Enrofloxacin</td>
<td>10-20 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>9-14 mg/kg SC, IM, or IV q 24h</td>
</tr>
<tr>
<td>Marbofloxacin</td>
<td>2.75-5.5 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Minocycline</td>
<td>10 mg/kg PO q 12h</td>
</tr>
<tr>
<td>Orbenfloxacin</td>
<td>7.5 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Pradofloxacin</td>
<td>3 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Rifampin</td>
<td>5-10 mg/kg PO q 12-24h</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Last Resort Antimicrobial Therapy</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Linezolid</td>
<td></td>
</tr>
<tr>
<td>Teicoplanin</td>
<td></td>
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<tr>
<td>Vancomycin</td>
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</table>

¹ A D-test is required to determine clindamycin susceptibility in staphylococci that are resistant to erythromycin

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### Box 1: Indications for bacterial skin culture

- Failure to respond to appropriately prescribed systemic empiric therapy
- Deep lesions present (e.g., nodules and draining tracts)
- Systemic antibiotics have been administered to patient within the last 30 days
- Repeated courses of systemic antibiotics have been previously prescribed to patient
- Presence of recurring episodes of superficial pyoderma (e.g., papules, pustules, crusts, collarettes, and/or patchy alopecia)
- Resistant bacterial infection (e.g., methicillin-resistant staphylococcal infection) previously diagnosed in the patient
- An individual in the patient’s home has been diagnosed with a resistant bacterial infection
- Patient has direct or indirect contact with human health care workers and/or facilities
- Patient has been recently hospitalized
- Patient has recently received an indwelling surgical device
- Rod-shaped bacteria predominate on skin cytology from superficial or deep lesions

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### Malassezia dermatitis

If you are not diagnosing *Malassezia* dermatitis, you are missing a common pruritic dermatosis!

*Malassezia* dermatitis (a.k.a. malasseziasis) is an extremely common inflammatory skin disease of many warm-blooded vertebrates caused by the overgrowth of *Malassezia* spp. yeast. Although the lipophilic unipolar budding yeast *M. pachydermatis* (formerly *Pityrosporum canis*) is responsible for most canine cutaneous infections, six other accepted species exist including *M. farfur*, *M. globosa*, *M. obtusa*, *M. restricta*, *M. slooffiae*, and *M. sympodialis*. Interestingly, the yeast later to be named *M. pachydermatis* was first isolated from the skin of an Indian rhinoceros with exfoliative dermatitis. It should be no surprise then that a clinical feature of *M.*
**Pachyderma**

Infection is lichenification (thick pachyderm-like skin). Being part of the normal skin microbiota, *M. pachyderma* can be found on healthy dogs in gland-rich areas of the skin and mucosa such as the ear canals, nose, lips, axillae, interdigital web, vagina, and anal sacs. Adherence to keratinocytes is thought to be by way of trypsin-sensitive protein adhesion molecules. Of breeds studied, healthy Basset hounds normally have significantly higher yeast counts.

Yeast become opportunistic pathogens with changes in the cutaneous microclimate. Excessive sebum production, increased humidity, and altered host defense mechanisms, including disruption of the epidermal barrier, favor yeast proliferation. Consequently, many dermatoses resulting in inflammation (allergic skin disease, pyoderma, ectoparasitism) and altered cornification (keratinization disorders, endocrinopathies, metabolic diseases, nutritional disorders) can lead to *Malassezia* dermatitis (MD). Once the skin is colonized, yeast release enzymes such as protease, lipase, phosphatase, peroxidase, and urease which can directly or indirectly alter epidermal kinetics (hyperplasia, seborrhea) and the skin immune system (inflammation, pruritus). In some dogs, especially atopic ones, *Malassezia* yeast might also be capable of eliciting a type-I cutaneous hypersensitivity reaction. Regardless, *M. pachyderma* causes inflammation and pruritus which continues to foster a cutaneous niche for yeast organisms.

Dogs of any age, gender, or breed may be affected with MD. However, terriers, Shih Tzus, Dachshunds, Shar peis, spaniels, hounds, and shepherd dogs appear to be predisposed. Many of these same breeds are also commonly affected by aforementioned diseases favoring yeast overgrowth. Depending on the underlying primary dermatosis, dogs with MD may show signs seasonally (atopic dermatitis) or year-round. *Moderate to severe pruritus that is only partially responsive to glucocorticoids or antibiotics is typical of MD*. Dogs may have a “yeasty” odor in which the owner finds offensive. Affected animals may have localized disease restricted to the head (perioral, muzzle, and ears), ventral aspect of the trunk (neck, axillae, groin), perineal area (ventral tail, perianal region), and pedal surfaces (interdigital spaces, nail fold) or generalized dermatitis (involving several body regions). Skin lesions, often found in intertriginous (skin folds) areas, reflect existing pruritus and seborrhea and are therefore not specific to MD. The skin may be erythematous, hyperpigmented, scaly, greasy or dry, alopecic, lichenified, saliva-stained, and/or excoriated. Occasionally yellow/orange to gray seborrheic plaques are present. Hyperpigmented lichenification implies chronicity. Sometimes the only clinical sign may be a brown waxy discharge within the claw fold suggesting yeast paronychia and hence pedal pruritus. Again, as most dogs are affected by a primary predisposing disease and since no pathognomonic lesion exists, differential diagnoses can be lengthy for MD. However, allergic skin disease is by far the most common underlying reason and “elephant skin” is caused by *Malassezia* yeast until proven otherwise.

Although cytological, microbiological, and histopathological examination of affected skin can identify elevated *Malassezia* yeast counts, cytology is the diagnostic method of choice in a clinical setting since it is simple, quick, and cheap. Cotton swab smears (ears, body folds, moist areas), direct impressions (flat and greasy areas), dry scrapings (dry lesions), and acetate tape preparations (mostly anywhere) are routinely used to recover and identify *M. pachyderma*. Many dermatologists prefer the tape technique. The adhesive side of clear acetate tape is applied to affected skin and then stained with Diff-Quik® (omit the first and second steps), Giemsa, or methylene blue. The stained tape is fixed to a glass slide with the adhesive side down. Using the oil immersion lens of a light microscope, the slide is examined for unipolar budding yeast which have been described as “peanut-”, “bowling pin-”, “footprint-”, “bottle-“ or “exclamation point-” shaped organisms. The finding of any yeast from clinically affected areas is diagnostic as it is difficult to find commensal yeast cytologically on normal canine skin. Quantitative terminology (mild, moderate, severe) or a scale of 1+ to 4+ can be used to grade the severity of yeast overgrowth as this aids in assessing response to treatment. The main utility of skin biopsy is to exclude predisposing skin diseases since the histopathological abnormalities of MD is nonspecific and yeast are many times lost from the surface during tissue processing.

Treatment consists of improving seborrheic conditions, killing yeast, and controlling for underlying primary diseases. In doing so, the main goal of treatment, pruritic relief, is achieved. *It cannot be overestimated that the primary underlying disease(s) need be identified and controlled in order to effectively treat MD*. Topical therapy consisting of shampoos, creams, ointments, lotions, powders, sprays, impregnated wipes, and spot-on formulations can be prescribed for focal or widespread disease. Inclusion of antiseborrheic (phytosphingosine, salicyclic acid, selenium sulfide, sulfur, tar), degreasing (benzoyl peroxide, tar), acidifying (boric and acetic acid), and antifungal (clotrimazole, chlorhexidine, miconazole, climbazole, ketoconazole, silver sulfadiazine, terbinafine) ingredients in many available topicals hasten time to recovery. Sole topical therapy may benefit some dogs while others require concurrent systemic therapy. Indeed prophylactic use of sole topical agents may prevent recurrence once the active infection is resolved. Systemic therapy is prescribed for severe cases of MD or when topical therapy is unsuccessful (including owner incompliance). Azole derivatives such as ketoconazole, itraconazole, and fluconazole, as well as the synthetic allylamine terbinafine, are effective against *Malassezia* species but are not licensed for animal use in the USA (Table 4). Therefore a patient-client-veterinarian relationship must be established before their use. A review of the literature regarding interventions for MD in dogs has shown some salient points including no significant difference in ketoconazole efficacy between 5 and 10 mg kg⁻¹ per day for 3 weeks, no significant differences in efficacy between daily and pulse (2 consecutive days per week) administrations of itraconazole, and lastly no significant differences in any results between pulsed itraconazole and daily administered ketoconazole. Nonetheless, results must be cautiously interpreted as study designs were not globally standardized. Importantly, it is recognized that griseofulvin has no effect
against *Malassezia* species. Liver parameter monitoring is usually reserved for dogs receiving high dose or long-term therapy, experiencing adverse effects, or having other medical conditions that warrants testing.

Systemic antifungal therapy is usually prescribed in conjunction with topical treatments for a minimum of 3-4 weeks upon which the patient is reassessed to determine the need to continue pharmacologic intervention. Clinical response (reduced pruritus, improved cutaneous topography) and occasionally cytological comparison of yeast numbers from the previous visit help gauge the decision for or against continued treatment. Basically therapy is continued until clinical signs resolve and yeast are not recovered cytologically. Again, topical therapy can be continued in a prophylactic manner as needed.

The likely transmission of *M. pachydermatis* from the contaminated hands of dog-owning health care workers to children in an intense care nursery signifies the potential for zoonosis. Owners should be instructed to wash hands after treating affected dogs and to reduce contact between immunoincompetent or immunosuppressed people and dogs heavily infected with *Malassezia* yeast.

### Table 4: Systemic antifungal therapy for canine malasseziasis

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketoconazole</td>
<td>5-10 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>5-10 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>5-10 mg/kg PO q 24h</td>
</tr>
<tr>
<td>Terbinafine</td>
<td>30 mg/kg PO q 24h (may not be as effective as azoles)</td>
</tr>
</tbody>
</table>

### Why are they [infections] there?

The most common reason for recurring infections in the pruritic dog is allergic skin disease, especially when signs of disease started in early adulthood. The clinical signs and patterns of allergic skin disease can look very similar, if not identical. Consequently, a detailed history depicting the problem(s), in light of the clinical findings, is paramount (Tables 5-9). “Allergy” may affect the skin, ears (pinnae and/or canals), or a combination of the two. Concomitant secondary infections (bacteria and yeast) are commonly seen with these diseases and must be identified and eliminated. Similarly, the allergic look-alikes, canine demodicosis and sarcoptic mange, must be excluded, as they can cause pruritus and lesional skin in a distribution pattern mimicking allergic skin disease. The presence of *Demodex canis* can be confirmed with routine deep skin scrapes, hair plucks, or squeeze tape impressions of lesional alopecic skin. It is important this diagnostic step be performed before leapfrogging to “allergy” and before the initiation of many anti-inflammatory/anti-pruritic therapies (e.g. glucocorticoids, oclacitinib, and cyclosporine), which will complicate the clinical picture. If demodicosis is confirmed, the extra-label use of isoxazolines (e.g., sarolaner, fluralaner, and afoxolane), prescribed at recommended label doses for flea prevention, should be considered, as this class of drug is proving to be efficacious and safe regardless of the breed of dog. Ultimately, treatment response should be based on improvement in mite numbers. If demodicosis is not a feature of the pruritic dermatosis, and “allergy” is suspected, then pruritic relief may be provided through the administration of oclacitinib (0.4-0.6 mg/kg PO q 12-24h) for several days or weeks, while the underlying disease is being identified (diagnostic work-up period). Why think about oclacitinib over glucocorticoids?: 1) glucocorticoids may alter the clinical picture during the diagnostic process, 2) glucocorticoids may lengthen time to or prohibit infection resolution, and 3) glucocorticoid side effects (PU/PD/PP) are not really tolerated by many clients, despite what you think.

### Table 5: Canine sarcoptic mange (scabies)

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Depends on geographical region</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Any (because of direct contact)</td>
</tr>
<tr>
<td>Seasonality</td>
<td>No</td>
</tr>
<tr>
<td>Pruritic pattern</td>
<td>Pinnal margins, elbows, hocks, ventrum</td>
</tr>
<tr>
<td>Response to anti-inflammatory prednisone</td>
<td>Poor to fair (short-lived)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>History (Recent adoption/boarding? Other individuals affected?); clinical signs (alesional pruritus or pruritic papules); positive pinnal-pedal response; superficial skin scrapes; scabies antibody ELISA; response to scabicidal therapy</td>
</tr>
<tr>
<td>Treatment</td>
<td>Scabicidal therapy for all in-contact dogs: consider isoxazole therapy; antipruritic therapy: consider short course of oclacitinib or glucocorticoids (5-10+d)</td>
</tr>
</tbody>
</table>

### Table 6: Canine flea allergy dermatitis (FAD)

<table>
<thead>
<tr>
<th>Frequency</th>
<th>FAD&gt;CAD&gt;&gt;CAFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset (1)</td>
<td>Any</td>
</tr>
<tr>
<td>Seasonality</td>
<td>Depends on geographic region</td>
</tr>
<tr>
<td>Pruritic pattern</td>
<td>Caudal body, lumbosacral skin, umbilical fold</td>
</tr>
<tr>
<td>Response to anti-inflammatory prednisone</td>
<td>Poor to fair (short-lived)</td>
</tr>
</tbody>
</table>
Diagnosis History (Recent boarding? Outdoor exposure? Recurring hot spots?); clinical signs (self-induced trauma, fleas/flea dirt, tapeworms; response to adulticidal flea prevention

Treatment Adulticidal flea prevention for in-contact dogs and cats: consider isoxazolines for dogs receiving frequent baths or swimmers; antipruritic therapy: consider short course of oclacitinib or glucocorticoids (5-10d)

(1) Allergy tends to be skewed to youngsters and young adults, but sometimes can affect animals of any age.

Table 7: Canine atopic dermatitis (CAD)

<table>
<thead>
<tr>
<th>Frequency</th>
<th>FAD&gt;CAD&gt;&gt;CAFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset (1)</td>
<td>0.5-3 years old</td>
</tr>
<tr>
<td>Seasonality</td>
<td>Variable, but often becomes nonseasonal and chronic</td>
</tr>
<tr>
<td>Pruritic pattern</td>
<td>Allergic dog (2)</td>
</tr>
<tr>
<td>Response to anti-inflammatory prednisone</td>
<td>Excellent</td>
</tr>
<tr>
<td>Diagnosis History</td>
<td>History (Recurring Malassezia dermatitis? Recurring pyoderma? Recurring otitis externa?); clinical signs (allergic dog (2)); exclude other pruritic dermatoses (3); Favrot’s criteria /IDT or IgE serology (4)</td>
</tr>
<tr>
<td>Treatment</td>
<td>Allergen avoidance mostly through bathing; antibiotherapy; antipruritic therapy (5); allergen-specific immunotherapy (5)</td>
</tr>
</tbody>
</table>

1. Allergy tends to be skewed to youngsters and young adults, but sometimes can affect animals of any age.
2. Allergic dog: face, ears, paws, axillae, groin, flexure surfaces, and/or perineum. Additional breed-associated variations of body sites affected by CAD: Dalmatian (lips); French bulldog (eyelids, flexure surfaces); German shepherd dog (elbows, hindlimbs, thorax); Shar pei (thorax, flexure surfaces, dorsolumbar area); WHWT (dorsolumbar area, lips, flexure surfaces; Boxer (ears).
3. Exclude pruritic differentials: folliculitis (pyoderma, demodicosis, dermatophytosis), Malassezia dermatitis, fleas/FAD, scabies, CAFR
4. “Allergy testing” is used to select candidate allergens for immunotherapy once other pruritic etiologies have been excluded as other diseases (both internal and external) can cause increased (and potentially cross-reacting) IgE antibodies. Similarly, normal non-atopic dogs can have ‘positive’ test reactions. Consequently, these tests should be used after the clinical diagnosis of atopic dermatitis has been made, in dogs with clinical signs >6 months/year and/or in which are not tolerating/responding to sole symptomatic treatments.
5. Topical/oral glucocorticoids or oclacitinib (Apoquel) for episodes of acute pruritic flares. Cyclosporine (Atopica) and/or ASIT for long-term maintenance. Lokivetmab (Cytopoint) can be administered for acute atopic flares or chronic atopic dermatitis. Use chronic oclacitinib on a case-by-case basis. Antihistamines are usually not effective as sole treatments, but may have a dose-sparing effect on other symptomatic treatments.

Table 8: Canine cutaneous adverse food reactions (CAFR)

<table>
<thead>
<tr>
<th>Frequency</th>
<th>FAD&gt;CAD&gt;&gt;CAFR (author tends to see in German breeds of dogs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset (1)</td>
<td>Any</td>
</tr>
<tr>
<td>Seasonality</td>
<td>Nonseasonal</td>
</tr>
<tr>
<td>Pruritic pattern</td>
<td>Allergic dog (2)</td>
</tr>
<tr>
<td>Response to anti-inflammatory prednisone</td>
<td>Variable</td>
</tr>
<tr>
<td>Diagnosis History</td>
<td>History (Nonseasonal pruritus? Recurring urticaria? Large bowel signs?); clinical signs (very young or older allergic dog (2)); exclude other pruritic dermatoses (3); elimination diet trial + food challenge (4)</td>
</tr>
<tr>
<td>Treatment</td>
<td>Avoid offending foodstuffs</td>
</tr>
</tbody>
</table>

1. Allergy tends to be skewed to youngsters and young adults, but sometimes can affect animals of any age.
2. Allergic dog: face, ears, paws, axillae, groin, flexure surfaces, and/or perineum
3. Exclude pruritic differentials: folliculitis (pyoderma, demodicosis, dermatophytosis), Malassezia dermatitis, fleas/FAD, scabies, MCT
4. Consider an elimination diet trial for dogs failing to respond to symptomatic therapy for CAD. A “food trial” should be offered for a minimum of 8 weeks, attempting to use mainly protein foodstuffs for which the dog has not regularly consumed. Ideally, flavored medication should be withdrawn. Diet challenge, offering pre-diet trial foodstuffs, is used after an elimination diet trial to determine if rebound pruritus occurs (within 2 weeks), thus confirming the diagnosis of CAFR.
Table 9: Canine allergic contact dermatitis (not irritant contact dermatitis)

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Not very common compared to scabies, FAD, CAD, and CAFR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset (1)</td>
<td>Any</td>
</tr>
<tr>
<td>Seasonality</td>
<td>Depends on offending contactant/substance (2)</td>
</tr>
<tr>
<td>Pruritic pattern</td>
<td>Sparsely haired body regions (especially short-coated dogs)</td>
</tr>
<tr>
<td>Response to anti-</td>
<td>Fair to good</td>
</tr>
<tr>
<td>inflammatory prednisone</td>
<td></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>History; clinical signs (Papular dermatitis?); avoidance + re-exposure; patch test</td>
</tr>
<tr>
<td>Treatment</td>
<td>Avoidance offending contactant/substance</td>
</tr>
</tbody>
</table>

1. Allergy tends to be skewed to youngsters and young adults, but sometimes can affect animals of any age.
2. Reported plants to cause reaction: wandering Jew, spreading dayflower, doveweed, Asian jasmine, dandelion, cedar wood, Hippastrum

**Canine atopic dermatitis**

Canine atopic dermatitis (CAD) is a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features associated with IgE antibodies most commonly directed against environmental allergens (e.g., pollen, mites, and epithelia). Our understanding of this disease is evolving with advanced research techniques in immunology and molecular biology. Although allergen-specific IgE is involved with CAD, disease materialization and fulfillment is likely also dependent on a faulty epithelial barrier allowing allergens entry into the body, altered antimicrobial peptide function, cutaneous microbiome dysbiosis/impairment, bacterial microbiota, T-helper lymphocyte polarization (Th2>Th1) favoring allergic inflammation, cell-signaling (cytokine) imbalances/dysregulation, loss of tolerance to environmental allergens, enhanced cellular trafficking to the skin, increased degranulating capability of mast cells, and neuroimmune interactions in the skin, such as that through the “pruritogenic” cytokine, interleukin-31 (IL-31). This receptor for IL-31 is found on keratinocytes, macrophages, eosinophils, and neurons. Binding of IL-31 to its receptor activates the intracellular JAK/STAT pathway resulting in gene transcription and physiological responses, including increased pro-inflammatory cytokine and cytokine receptor production, elevated chemokine expression, lymphocyte proliferation, IgE synthesis, and itch. A detailed description on the pathophysiology of CAD can be found in *JAVMA* 241(2): 194-207, 2012. Though the prevalence has likely increased with improved awareness and diagnostic criteria, CAD is estimated to affect at least 10% of the canine population.

**CAD: diagnosis**

Although the reader is referred to the aforementioned, it cannot be overstated that CAD is a diagnosis made clinically through the history, physical examination, and the exclusion of other pruritic dermatoses. Specifically, “allergy tests” (intradermal and IgE serologic testing) do not diagnose CAD, as normal non-atopic dogs may have ‘false positive’ test results (perhaps sensitive, but not clinical). Indeed, clinical criteria have been established to help make a clinical diagnosis of CAD (e.g., Willemse, Prelaud, Favrot). Regardless, of the “list” of criteria used, it is well-established that if other pruritic dermatoses are excluded (ruled-out) before using the said clinical criterion, the sensitivity and specificity of the list increases. Currently, Favrot’s clinical criteria, which have been critically evaluated in peer-reviewed literature for CAD, appear to be generally favored at the moment in the dermatology community (Box 2).

**Box 2: Favrot’s clinical criteria for diagnosing canine atopic dermatitis**

1. Age of onset < 3 years old
2. Mostly indoors
3. Glucocorticoid-responsive pruritus
4. Chronic or recurrent yeast infections
5. Affected front paws
6. Affected ear pinnae
7. Non-affected ear margins
8. Non-affected dorsolumbar region

**CAD: management**

In order to achieve successful long-term CAD management, clients/owners must be appropriately educated on reasonable treatment expectations. CAD is a chronic relapsing and remitting disease, so pruritic flares (e.g., infections, worsening CAD during a particular time of the year, stress, etc.), possibly requiring a change to the therapeutic regimen, are to be expected. Itch attrition, not elimination *per se*, and reduction in the frequency of infections are goals to establish up front. Ultimately, improving the patient’s quality of life through a regimen of therapy tailored to the tolerance of the patient and willingness of the client to administer it, all while within an established budget, is the objective.

The author has established a set of unpublished guidelines, *not rules*, which he presents to clients as options based on the age of the patient, duration of disease during a year, severity of disease, client willingness, and cost of care. These guidelines are based on a survey of the literature and clinical experience. First and foremost, are the “absolutes”—regular administration of adulticidal flea
prevention, infection control, and periodic baths are required. From there, the duration of clinical disease during the calendar year is determined using a division of less than (seasonal) or greater than (becoming or is nonseasonal) 6 months out of the year. For dogs experiencing signs of CAD for less than 6 months out of the year, then lokivetmab (Cytopoint: label chart recommended dose SC q 4-8wks), oclacitinib (Apoquel: maintenance at 0.4-0.6mg/kg PO q 24h), or the lowest possible dose of glucocorticoid (topical or oral) administered as infrequently as possible (prednisone 0.5-1.0mg/kg PO q48-72h) are options, assuming there are no contraindications prohibiting their use. When clinical disease is experienced the majority of the year (> 6 months), then the patient’s age has some bearing on the recommended options (Table 10). It should be remembered that cyclosporine (Atopica: 5mg/kg PO q 24h) must be given daily for the first 4-6 weeks before attempting to taper it to alternate day administration. Indeed, dogs will have pruritic flares of CAD even when receiving some form of maintenance therapy. Assuming the pruritus is not the result of sole infection and/or parasites, the author prefers to gain control of CAD pruritus with a short course of oclacitinib (Apoquel; assuming not already receiving it for maintenance therapy) or glucocorticoids (topical or oral). Alternatively, if the flare is expected to last several weeks, then lokivetmab (Cytopoint) is a “rescue” option for dogs receiving it less frequently than every 4 weeks or for dogs receiving other symptomatic maintenance therapies for CAD.

Table 10: Patterson's guidelines in determining treatments for dogs afflicted with CAD

<table>
<thead>
<tr>
<th>Canine atopic dermatitis</th>
<th>Dog &lt; 7-8 years old</th>
<th>Dog &gt; 7-8 years old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical disease &lt; 6 months/year</td>
<td>Cytopoint</td>
<td>Apoquel</td>
</tr>
<tr>
<td></td>
<td>Glucocorticoids</td>
<td>Cytopoint</td>
</tr>
<tr>
<td></td>
<td>Apoquel</td>
<td>Glucocorticoids</td>
</tr>
<tr>
<td>Clinical disease &gt; 6 months/year</td>
<td>ASIT</td>
<td>Cytopoint</td>
</tr>
<tr>
<td></td>
<td>Cyto point</td>
<td>Cytopoint (lokivetmab)</td>
</tr>
</tbody>
</table>

Cytopoint (lokivetmab)

Cytopoint is a canine-specific (“caninized”) anti-canine-IL-31 monoclonal antibody approved for canine atopic dermatitis (CAD). Binding of this antibody to its extracellular target, IL-31, prevents activation of the intracellular JAK/STAT pathway, thereby limiting IL-31-mediated pro-inflammatory responses in atopic dogs. Since this monoclonal antibody is canine-specific, it can be repeatedly administered by subcutaneous injection every 4-8 weeks in-office to dogs with atopic dermatitis. This non-drug therapy is degraded like any other natural antibody in the dog, allowing it to be co-administered with many other medications administered for CAD or other comorbidities, since drug-drug interactions are not to be expected. Cytopoint should be considered as a safe and viable option for CAD when: an in-office injection is convenient to the dog owner; a dog is difficult to “pill”; the dog owner wants a non-drug therapy; other atopic treatments are ineffective or are not well-tolerated; an atopic flare is occurring in a dog receiving other atopic treatments; the atopic dog is receiving other medication for other comorbidities; other atopic treatments are not safe because of comorbidities and the treatments for them; and a clinically confirmed atopic dog less than 12 months of age.

References

Appendix 1: Diagnostic algorithm and treatment considerations for canine allergic skin disease

Itchy dog: What are the infections?

- Fleas/FAD
- Folliculitis (pyoderma/demodicosis/dematophytosis)
- Yeast dermatitis
- Scabies

Stop itch (demodiosis excluded)

- Antihistamine
  - May be effective for mild allergic itch
  - Combine with fatty acids
- Apoquel
  - Fast-acting providing immediate relief
  - Consider only prescribing for once daily administration
  - Can prescribe with diet trial, if needed
  - Not to be used with demodiosis
- Glucocorticoids
  - Topical or oral
  - Lowest effective dose
  - Can prescribe with diet trial, if needed
  - Not to be used with demodiosis
- Bathing
  - Anti-inflammatory
  - Soothing
  - Improved barrier function

Confirm canine atopic dermatitis

- Cytopoint
  - In-clinic mAb injection providing up to 4-8 weeks of relief for atopy
  - Can be used with comorbidities
  - Combine with Apoquel or steroids for first 3-5 days
- Apoquel
  - Daily home administration
  - Already established effectiveness
- Atopica
  - Daily home administration for 4-8 weeks then taper
  - Good anti-inflammatory effect
  - Combine with Apoquel or steroids for first 3 weeks, if needed
- Glucocorticoids
  - Not ideal for chronic use
  - Lowest effective oral dose q 2-3 days
- ASIT
  - Retrain immune system when signs > 8 months/year or dog does not tolerate or improve with symptomatic therapy
  - Sparring effect of other therapy
Another Ear Infection? 
Dealing with Chronic Otitis
Anthea Schick, DVM, DACVD 
Dermatology for Animals 
Tempe, AZ

Pathogenesis 
Canine otitis externa affects approximately 10-20% of canine population and is one of the most common diagnoses after dental disease in general veterinary practice. There is evidence that the prevalence of canine otitis externa is on the rise. A 2011 Banfield Pet Hospital’s State of Pet Health Report evaluated data from 770 hospitals and found that the diagnosis of canine otitis has increased by 9.4% since 2006. Since canine otitis externa can be one of the most frustrating diseases for pet owners, having a thorough and aggressive approach to its diagnosis and treatment is essential.

When confronted with a case of chronic or recalcitrant otitis in a dog, I first attempt to discuss three fundamental concepts with the pet owner. The first point they must understand is that otitis externa is really just a clinical sign, not a final diagnosis. The second is that normal dogs do not get otitis. The third point is that bacteria and yeast are not the cause of otitis but the result of otitis.

Why normal dogs do not get ear infections: Ears have a way of cleaning themselves; this process is largely due to epithelial migration (EM). EM in the ear begins at the tympanic membrane. As epithelial cells divide, they migrate up and out in a slight spiral pattern, carrying cerumen and trapped debris and organisms out of the ear. When there is damage to the tympanic membrane or the ear canal epithelium, EM is the process of repair. A recent study evaluated the speed of EM and found that the epithelial cells travel about 100 to 200 μm/day.

Why bacteria are not the cause of otitis: The ear is also normally colonized by a variety of organisms including Staphylococcus spp., Streptococcus spp., Corynebacterium and Malassezia spp. These organisms live in harmony with a normal ear canal. With inflammation, these resident organisms and other opportunistic bacteria like Pseudomonas spp. (a ubiquitous organism often found in water and able to colonize plumbing fixtures) can overgrow and cause infection.

Why otitis externa is not a diagnosis: A commonly used conceptual framework for understanding canine otitis externa uses the terms predisposing, primary and perpetuating factors. A combination of these factors is needed to produce clinical disease.

Predisposing factors
Predisposing factors are factors that increase the risk of development of otitis externa. For example, conformation (narrow ear canals like in the Chinese Shar-Pei), environment/lifestyle (humid climates, trauma from ear plucking, excessive swimming, over exuberant ear cleaning).

Primary factors
Primary factors begin the condition, For example, parasites (Otodectes, Demodex, ear ticks), foreign bodies (grass awns), tumors (benign-polyps and adenomas, neoplastic-adenocarcinoma, squamous cell carcinoma), hypersensitivity disorders (atopy, adverse food reactions, drug/contact reactions), keratinization disorders, glandular disorders (excessive cerumen/sebum accumulation, sebaceous adenitis), immune-related (hypothyroid) and auto-immune (pemphigus complex).

Perpetuating factors:
Perpetuating factors prevent the resolution of otitis. For example, organism overgrowth (bacteria, yeast), loss of epithelial migration, chronic changes in the ear (epidermal/glandular hyperplasia, stenosis, fibrosis, ossification, otitis media).

Failure to identify and address these factors likely will result in ongoing otitis and possible end-stage otitis. In end-stage otitis, inflammation and ear canal hyperplasia leads to stenosis and thickening of the ear canal wall, often with complete closure of the ear canal. Fibrosis of the canal and ossification of the auricular and annular cartilages is the final step in the end-stage ear. At this point, medical therapy cannot reverse these changes, and total ear canal ablation with lateral bull osteotomy (TECA-LBO) is often recommended.

Diagnostic approach
Here are some steps and tips to treat canine otitis externa and prevent the progression to end-stage otitis. At first exam, a thorough history should be taken. For example, we must know age of onset, unilateral vs. bilateral, seasonal patterns, additional dermatologic signs, current and previous diet, behavior and environment, general health, neurologic signs observed, other pets affected, and what treatments are currently being used and how often and with what success.

Next, a thorough clinical exam (both physical and focused dermatologic exam) should be performed. I begin by palpating the external ear canal to assess pliability and pain. I palpate both mandibular lymph nodes since they often give hints if there is otitis media or more severe inflammation is present in one ear. Check for neurologic deficits that might indicate otitis media. Lastly, I open the mouth widely to assess for discomfort at the temporomandibular joint, which also may indicate otitis media.
Ear anatomy

The external ear is made of two pieces of cartilage that fit together forming an irregular L-shaped bend in the canal. The annular cartilage makes up the horizontal canal and attaches to the skull. The auricular cartilage attaches to the annular cartilage and makes up the vertical part of the ear canal. It becomes funnel shaped as it travels distally and then finally expands to form the pinna. The vertical canal travels vertically and slightly rostrally before turning medially and forming the horizontal canal. At the junction of the vertical and horizontal canal, there is a prominent ridge (called either the dorsal ridge or Noxon’s ridge). When the ear is in normal position, this ridge blocks the passage of an otoscope.

The following quote from a classic anatomy text dryly sums up this significant anatomical problem: ‘Unfortunately, its external acoustic meatus is curved, making the passage of the straight otoscope for the examination of the proximal part of the meatus and eardrum difficult’ From Dyce, Textbook of Veterinary Anatomy.

There is a variable amount of hair in the ear canals of dogs. Some breeds, like poodles, will have abundant hair extending all the way into the horizontal canal. In the proximal horizontal canal, just at the level of the entrance of the cartilaginous external acoustic meatus are a small number of few fine hairs and often a small amount of cerumen. These hairs are a helpful landmark that indicates you are close to the tympanic membrane.

The tympanic membrane or ear drum is made of two parts: the pars tensa and the pars flaccida. The pars tensa is the taught, usually transparent part of the tympanum, which is the most obvious part of the ear drum to observe. The manubrium of the malleus attaches to the pars tensa and slightly pulls the tissue of the pars tensa proximally into the middle ear. The attachment of the malleus to the tympanum is called the stria mallearis, which has a slight C-shaped curve, which points toward the nose. From the stria mallearis, small tension lines or striations can be seen as well as blood vessels that supply the ear drum. At the tip of the stria mallearis is the umbo. The umbo is an important landmark, as it is a source of epithelial migration and healing for the tympanum. Care should be taken to avoid damaging the umbo when flushing ears or performing a video-otoscopy. The pars tensa should be clear enough to visualize a bony ridge in the middle ear, which looks like a dorso/ventral white line running. The pars flaccid, or soft part of the tympanum, is dorsal and caudal to the pars tensa and is sometimes blends into the epithelium of the horizontal canal. In other dogs, it is more obvious and looks like a pink, smooth, ‘puffy’ bulge. Occasionally, dogs will have such prominent pars flaccidas, they can be mistaken for masses or polyps.

Middle ear

The tympanic cavity consists of a small epitympanic recess and a large ventral bulla. The tympanic bulla proper is adjacent or behind to the tympanic membrane. In the dog, there is an incomplete bony septum or tympanic bulla ridge (also called Rosychuk’s Ridge). On the medial wall of the tympanic cavity, there is a bony promontory, which houses the cochlea. The cochlear (round) window is located on the caudolateral portion of the promontory. When flushing the middle ear, avoid this promontory or the round window, to avoid damaging the inner ear. The middle ear cavity of the cat is completely divided by a bony septum into two separate tympanic cavities. The auditory tube is a short canal that extends from the nasopharynx to the rostral portion of the tympanic cavity proper.

The three auditory ossicles, the malleus, incus and stapes, are the bones that transmit and amplify air vibrations from the tympanic membrane to the inner ear.

Otic exam

One of the challenges of assessing otitis externa is the difficulty of performing a good otic examination. In my opinion, few practitioners (including myself!) were taught to properly examine an ear while in veterinary school. In fact, most interns and even some visiting residents that I teach need help with their ear exams. There are two kinds of heads for hand-held otoscopes available in most clinics, the operating head and the diagnostic head. The diagnostic head has a large lens that can completely cover the otoscope head as well as a small port to attach a tube for pneumotympanoscopy. The operating head has a small lens that can be moved and instruments can be passed into the ear canal while still visualizing the ear. I recommend experimenting with both heads if they are available. In my opinion, the operating head is superior for most ear exams as the focal length of the lens is better suited for visualizing the tympanic membranes.

Once you have chosen your otoscope type, you are ready for your ear exam. Understanding the normal anatomy of the canine ear is essential in performing a good ear exam. The dog should be around chest height on a table with a technician holding the muzzle of the dog for restraint (not with their arm around the neck-since this impedes movement of the ear canal necessary for a good ear exam). The otoscope should be placed in the intertragic notch while the canal is then visualized. The most challenging part of the ear exam is passing the dorsal ridge, which lies before the junction of the vertical and horizontal canal. The dorsal ridge is what most people run into when dogs seem painful during examination. To avoid this, pull the pinna out (laterally) and slightly down (ventrally) while simultaneously ‘diving’ underneath the dorsal ridge while you advance into the horizontal canal. I find that performing a good ear exam requires more movement and body repositioning than one would think. A good way to practice your ear exam is on sedated or anesthetized patients. After 5-10 ears, you get the ‘pull and dive’ move down. Once in the canal, assess for evidence of ulceration, extent of hyperplasia or exudate/debris. Once in the horizontal canal, advance toward the tympanic membrane. In order to see all parts
of the tympanum (pars tensa, pars flaccida, and the stria mallearis) as well as the ‘corner pocket’ - a little recess rostral to the tympanum where foreign bodies sometimes hide - you must move your head quite a bit, much like trying to visualize a room through a keyhole.

**Otic cytology**
After your ear exam, take samples for cytology. Samples should be taken from the vertical and horizontal canal junction using a cotton-tipped applicator. To remember which sample came from which ear, I break the wooden part of the applicator with the left ear sample. I smear the sample from the left ear near the frosted part of a glass slide and the right at the other end. Heat fixing is not important. After a quick stain with Diff-Quik, examine the slide under oil immersion (x100). The number of organisms and/or inflammatory cells should be determined at each visit.

Record organisms by their size, shape and number. For bacteria, a scale of 1+ to 4+ is commonly used, with 1+ reflecting a few bacteria (easy to count per filed) and 4+ reflecting a large number (impossible to count, almost a uniform layer of organisms per field). As *Malassezia* is larger and easier to count, record an approximate average of yeast per high powered filed (hpf), for example, 1-5/hpf, 5-10/hpf, 10-20/hpf or TNTC (too numerous to count-TNTC). More than 4 *Malassezia* and 10 bacteria per oil immersion (x100) are abnormal in dogs. The presence of organisms is not synonymous does not mean infection. Rare bacteria or yeast noted within the cerumen or on epithelial cells, with no inflammatory cells indicates colonization. Inflammatory cells indicate more significant infection and any organisms engulfed by white blood cells are clear indication of infection.

**To culture or not to culture**
The results of bacterial culture and sensitivity and minimum inhibitory concentration (MIC) measurement may be used to determine the best systemic antibiotic choice. There is some evidence that systemic antibiotic therapy is not helpful in the treatment of otitis externa, and may contribute to colonization by resistant organisms. Since most cases of otitis externa will respond to topical therapy (as long as the underlying disease/problem is addressed), I rarely perform a bacterial culture. In my opinion, you will almost always choose the correct topical antimicrobial by making and empiric choice based on your cytologic findings. Even in a case of ‘resistant’ *Pseudomonas* spp., if you use high enough concentrations and large volumes of topical antibiotics (enrofloxacin for example), you will largely overcome resistance mechanisms. Remember that MICs on culture and sensitivity panels reflect the concentration of antibiotic achievable in the serum after systemic administration of the antibiotic. With topical therapy, the concentrations of the antimicrobial with far exceed drug concentration achievable in the blood. When you suspect otitis media, however, performing a culture and sensitivity is valuable in aiding systemic antibiotic therapy since the organisms present in otitis externa and those present in otitis media are often different. Obtaining an uncontaminated sample of the middle ear is best done through video-otoscopy.

**Diagnostic imaging**
Radiology: This is relatively easy diagnostic since can be done in-house. The most commonly uses views are used when assessing ear disease with radiographs: dorsoventral-allows comparison of bullae and petrous temporal bones between sides, rostocaudal open mouth- allows comparison of bullae and external ear canal between sides and lateral oblique-allows bulla evaluation without interference of other bony structures. Non-fancy radiographs can be taken easily and quickly to evaluate whether ossification of the external ear canal is present, which would indicate an ‘end-stage ear’. Radiographs are helpful if obvious middle ear disease or present but studies have shown that normal radiographs do not rule out middle ear disease.

Computed tomography (CT): CT allows more precise evaluation of bony structures in the middle and inner ear than MRI. The tympanic bulla and any bony osteolysis/proliferation can be readily seen with CT as can the ossicles. The sensitivity of CT for detection of otitis media has been reported to be around 83%.

Magnetic resonance imaging (MRI): allows for more precise evaluation of soft-tissue structures than CT and radiographs and should be used when there is concern of soft tissue changes/masses.

**Video otoscopy**
This procedure is both a diagnostic tool and therapeutic tool. It can be used for deep and precise cleaning of the ear canals and middle ear cavity as well as to evaluate the ear canal middle ear cavity. Foreign bodies can be found easily and removed while minimizing damage to the tympanic membrane. Polyps and masses can often be removed or at least debulked and submitted for histopathology. VO also allows collection of specimens for bacterial culture and sensitivity from the middle ear cavity and application of medications directly into the bulla. To maximize access to the ear canal and to decrease debris, I recommend treating the patient for at least 10-14 days with 1-2mg/kg prednisolone orally once daily. I will often prescribe tramadol to help with pain before the procedure and to make it easier for the owner to treat the ear. After several days of glucocorticoids and tramadol, I will have the owners begin flushing the ears (no more than twice weekly) and applying topical ear medications that contain glucocorticoids (twice daily for one week then daily until the procedure). At the two week recheck, if the ear canals are very stenotic and fail to open up with such aggressive oral and topical anti-inflammatory therapy, medical therapy is unlikely to fully resolve the otitis and surgery may be necessary.
VO Procedure: Obtain samples of the otic exudates from the junction of the vertical and horizontal canal for culture and sensitivity. These cultures can then be set aside for possible submission. I rarely submit culture from otic exudate in cases of otitis externa, but once you start flushing the ear, much exudate will be lost and culture results possibly altered by flushing agents. If there is not too much exudates or mass(es) present and you suspect otitis media, collect samples of exudates from the middle ear for culture and sensitivity. To collect samples from the middle ear cavity introduce a sterile 3.5 Fr x 5-1/2" Tom cat catheter with a 2ml syringe filled with 1-2ml of sterile saline through the working port of the video-otoscope into the middle ear cavity. Flush the fluid into the cavity and aspirate for culture and sensitivity. If the tympanic membrane is intact but middle ear disease is present or suspected, you can perform a myringotomy. I use a sharpened Tom cat catheter to push through the tympanic membrane at the caudoventral part of the pars tensa at 5 to 7 o’clock. It is difficult to rupture a normal tympanum but diseased tympanums will tear with little pressure.

I then take pictures of the canal at mid-vertical canal, just beyond the junction and at the level of the tympanum before placing any flushing agents into the canal. These pictures are helpful to show the owner ‘before and after’ pics. I use a cerulentic agent to fill the canal and then massage the canal for several minutes while pulling the pinna to take the L-shape out of the ear canal. This will allow material to more easily be removed. Warm saline is the used to flush the canal with a Tom cat catheter and 12 cc syringes. There are flushing/suction machines available as well but I like the force that the 12cc/Tom cat combination creates. If there is abundant debris or inspisated material, I will aspirate the saline out and repeat the cerulentic to try to ‘break up’ the debris. Repeat flushing until the canal is clean. I often find a biofilm of sorts on the pars tensa of the tympanum in cases of chronic otitis, which needs to be removed by close-up flushing. This material often peels off like old wallpaper being removed.

If there is a tear in the tympanum, flush the middle ear cavity multiple times with saline. After flushing, aspirate all saline and instill ½ cc each of large animal enrofloxacin (100mg/ml) and dexamethasone SP into the middle ear. Warn the owner that temporary or permanent (very rare) vestibular syndrome, facial nerve paralysis and Horner syndrome can occur post-myringotomy or even with an intact tympanum in a cat. After the procedure, I continue topical and systemic steroids after to reduce inflammation help the ear canal heal itself.

**Therapy**

**Ear cleaning**

Start all treatment(s) with a clean ear. Flushing the ear canal is necessary to remove cerumen and debris to allow topical products to reach the canal epithelium. Purulent exudate and inflammatory mediators can inactivate some medications. In cases of sever otitis, where erosions/ulcerations are present, I recommend 4 days of systemic glucocorticoids to reduce inflammation before having the owners clean or medicate the ears.

Over the years I have discovered (often the hard way) that most owners have no idea how to properly clean their dog’s ears. Even owners who have had to manage ear infections for years rarely clean ears correctly. I have ear models in each exam room, which I use to show owners the anatomy of the dog’s ear. Demonstrate ear cleaning in exam room with the owner. If I suspect that they are not cleaning correctly, I sometimes have the owners show me how they are flushing. I recommend that they hold on to the pinna and fill the ear canal with cleaner. While still holding onto the pinna, they should gently pull the ear outwards and feel the ear canal starting from the outer ear down to the base. Once they have felt where the ear attaches to the skull, they should massage the horizontal canal so that a ‘squish squish’ sound is made. While still holding the pinna and continuing to pull laterally, they should use cotton balls or gauze to wipe out the excess fluid and debris. I instruct them to repeat this process until the cotton balls come out clean (or if any blood is seen). Once they are done cleaning, they can let the pinna go and the dog will shake any excess fluid out (and sometimes more debris). Here is a link to a video for owners showing how to clean a dog’s ear:

http://www.youtube.com/watch?v=brCwQftfJ0o&feature=plcp

For more severe otitis or when otitis media is suspected, an ear flush under anesthesia is recommended. The most thorough and accurate was to perform a flush, which also allows precise myringotomy and mass removal/biopsy is a video otoscopic flush. After obtaining samples for cytology and/or culture, I instill a cerumenolytic agent and massage for 5 minutes. Warm saline is then used to flush the ear and curettes or graspers can be used to mechanically remove large chunks of debris. I will often spend 30 minutes per ear in order to adequately clean a chronically infected ear. Another benefit of a video-otoscope is that it allows forceful and directed yet precise flushing of the tympanic membrane. In my experience, chronic purulent otitis will leave a ‘biofilm’ of sorts adhered to the pars tensa of the tympanum. This film is somewhat transparent, making it difficult to assess with a regular otoscope. After flushing the majority of the exudate from the ear, I will place my flushing catheter directly against the pars tensa and flush, often observing peels of adherent exudate dislodging from the pars tensa. After flushing and aspirating the excess fluid, I instill 0.5 ml each of 100 mg/ml enrofloxacin and dexamethasone SP.

**Topical medications**

As mentioned above, choose topical antimicrobials based on cytology findings. When cocccoid bacteria are seen, you are most likely dealing with *Staphylococcus pseudintermedius* or *schleiferi* or possibly *Streptococcus* spp. When rod shaped bacteria are seen, *Pseudomonas aerugiosa* is the most likely culprit.
Many veterinary products are available for canine otitis externa containing combinations of antibiotics such as neomycin, gentamicin, polymixin B, and enrofloxacin with anti-fungal agents and/or corticosteroids. Common treatment protocols include twice-daily treatment for the first week, then once daily for the second week pending a re-evaluation. With any topical ear medication, make sure the patient is receiving enough volume to be effective. Anywhere from 0.5 ml to 1 ml (10 to 20 drops) is usually enough to treat most canine ears.

**Glucocorticoids**

Virtually all cases of canine otitis externa should have the benefit of glucocorticoids. Most organisms thrive in an inflamed environment, reducing the inflammation alone is sometimes enough to allow the ear canals to 'self-cure'. Glucocorticoids reduce cytokine production, resulting in decreased inflammation, pruritus and pain. Glucocorticoids also decrease the production of cerumen and sebum, as well as secretions from the mucoperiosteum lining the middle ear. There are many topical corticosteroids available, including fluocinolone, betamethasone and dexamethasone. Some dermatologists recommend using topical steroids alone (without additional medications like antimicrobials).

Moderate to severe cases of otitis externa usually benefit from systemic glucocorticoids at relatively high doses (prednisone/prednisolone/methylprednisolone orally at 1-2 mg/kg/day). Glucocorticoids have also been shown to aid in the elimination of resistant Pseudomonas strains through changes in the microclimate that no longer favor the growth of the bacteria.

**Antifungal therapy**

Many of the topical antimicrobial combination products containing an antifungal should be effective for *Malassezia* otitis. Since there is little concern about the development of resistance in *Malassezia*, I will sometimes use oral antifungals, such as ketoconazole 5 mg/kg q 24h, itraconazole 5 mg/kg q 24h, or fluconazole 5-10 mg q 24h for 2-4 weeks for recurrent *Malassezia* otitis.

**How I treat**

With a typical severe painful purulent otitis externa, I prescribe a gentle pH neutral ear flush and a concentrated enrofloxacin/dexamethasone combination. I prescribe tramadol for pain and recommend that the owner start systemic glucocorticoids at about 1-2 mg/kg per day for 10-14 days. After four days of systemic glucocorticoids and pain medication, the owners are allowed to clean and medicate the ears. I have the owners clean the ears no more than twice weekly and use the topical medication twice daily for the first week then once daily. I instruct the owners not to apply either ear wash or medications for 2 days before a recheck examination so that I can more easily assess the tympanum and deep ear canal. At recheck (in 10-14 days), most cases will be improved greatly allowing the systemic glucocorticoids to be tapered. I perform cytology at initial examination and at all re-evaluations. If the primary cause of the otitis has not been evaluated and treated, tapering the glucocorticoids will simply lead to an eventual flare in otitis. Therefore, the MOST important part to successful treatment of canine otitis externa is finding the cause. Depending on the details of each case, this can mean checking lab work +/- thyroid levels, performing elimination diet trials, intradermal allergy testing and immunotherapy, etc..

**References**


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Every spring, the American College of Veterinary Dermatology (ACVD) and the American Academy of Veterinary Dermatology (AAVD) host a North American Veterinary Dermatology Forum (NAVDF). This last NAVDF was held in Orlando, FL in April 2017. The NAVDF is open to everyone with an interest in veterinary dermatology, including both boarded and non-boarded veterinarians, technicians and veterinary students. The three-day conference presents the latest research in veterinary dermatology as well as clinical updates to aid veterinarians and specialists in diagnosing and treating dermatologic diseases. Many of the research abstracts and poster presentations presented at the NAVDF will not be published for months to years after the conference, so I have tried to summarize the more clinically relevant information. The notes for this lecture were due before the conference was held so I summarized some of the most timely and important topics that are being researched and discussed at the NAVDF and other large Veterinary Dermatology conferences.

Atopic dermatitis/pathogenesis
The most recent definition of canine atopic dermatitis (CAD) as proposed by the International Task Force on Canine Atopic Dermatitis is: “A genetically predisposed inflammatory and pruritic skin disease with characteristic clinical features associated with IgE antibodies most commonly to environmental allergens". Canine atopic dermatitis is a genetically predisposed and multifactorial disease involving immune dysregulation, allergic sensitization, skin barrier defects, microbial colonization and environmental factors. Approximately 10% of cases that present clinically as classical CAD have no measurable allergen-specific IgE; these cases are called Atopic-like Dermatitis (ALD).

Increased risk factors of developing AD include living in urban areas, exposure to dense human population areas, early adoption of bathing and regular bathing. Living in a rural environment, living with other animals and non-commercial diets appear to be protective. In the study of human AD a theory called the hygiene hypothesis has been proposed where certain macrobiotic diets, probiotics, endotoxins and micro-organisms reduce the incidence of human AD.

As in human AD, studies in dogs have shown that skin barrier function is important in canine AD. Removing part of the stratum corneum through tape-stripping enhances house dust mite allergen specific IgE levels, intradermal test reactions, and peripheral T-cells responses. Transepidermal water loss (TEWL) is also higher in atopic dogs than in healthy controls. The stratum corneum of atopic dogs is thinner with wider intercellular spaces. The lipid layers important in protection of the skin are shorter, thinner and irregular in atopic dogs compared to healthy dogs. Changes to the balance of lipids in the skin of atopic dogs has been shown with reduced levels of ceramides 1 and 9 and increased levels of cholesterol.

The development of a healthy barrier function is complicated and associated with keratinization. An important molecule in this process is filaggrin. In some people with AD, a genetic defect in filaggrin is present. In a Beagle model of AD, filaggrin immunostaining was significantly lower in atopic compared to healthy dogs. Another study found that 22% of atopic dogs appeared to have a mutation in their filaggrin.

New therapies for atopy: Immunotherapy
In allergen-specific immunotherapy (ASIT) extracts of allergens to which the patient is sensitive are given gradually to decrease the allergic response. Unlike drug therapies, ASIT is the one proven treatment for allergies that change the patient’s immune response rather than blocking inflammatory mediators. ASIT is appealing as it is associated with very few adverse effects and is effective in the majority of patients at some level. Challenges with ASIT include a prolonged time to effect and requires subcutaneous injections, which owners are sometimes hesitant to perform.

There are many theories on how ASIT works including the following: the blocking antibody theory, where IgG is produced, competing with IgE and the immune deviation theory, where T helper lymphocytes types are altered. ASIT has also been shown to down regulate mast cells and eosinophils and increase numbers of anti-inflammatory mediators like regulatory T cells, IL-10, and TGF-beta.

Sublingual Immunotherapy (SLIT): Sublingual immunotherapy (SLIT) involves administration of allergen extract into the mouth, to expose the allergen to the oral mucosa. SLIT has become a common form of immunotherapy in Europe for people with allergic rhinitis and asthma. Studies on the efficacy of SLIT in people show varying degrees of success, possible due to large variability in treatment protocols although overall, SLIT in people has shown to be effective and safe. There have been several small studies looking into SLIT in dogs with AD and several companies are now offering it (HESKA ALLERCEPT® Therapy Drops, SkinVet™)
Recent trials of SLIT in dogs have shown variable efficacy as in people, again possibly likely to differences in protocols. An initial pilot study by Dr. DeBoer in mite-sensitive dogs was used a protocol modified from human SLIT methods showed clinical improvement in the majority of dogs with reductions in mite allergen-specific IgE and increases in allergen-specific IgG. This same group has conducted a larger study with 174 atopic dogs evaluated. They concluded that SLIT is effective in approximately 60% of patients. There is some evidence that SLIT can be effective in a percentage of patients who have failed standard ASIT. A double-blinded, controlled study by another group evaluated 18 atopic dogs and found 66% had greater than 40% improvement in clinical signs after a year of SLIT. However, 50% of control dogs also had greater than 40% improvement. Although SLIT in dogs has shown some promise in treating AD, there is more evidence based medicine as well as clinical experience with standard ASIT based on allergy testing. We will likely see more research on the best protocols for performing SLIT in dogs as well as more well-designed clinical trials to better prove its efficacy in the coming years.

HESKA ALLERCEPT® Therapy Drops are currently only available through a veterinary dermatologist but will likely be released to veterinarians soon. Allergy testing is needed through either intradermal allergy testing or serology testing to identify specific allergens. Allergens can be ordered through HESKA with twice daily administration. SkinVet™ RESPIT™ Oromucosal spray is administered once daily and does not require allergy testing since it uses regionally important allergens. NelcoVet Allerpaws oral allergy treatment is once daily and can be formulated on results of allergy testing or can be formulated using requested allergens.

New therapies for atopy: Medications

Stem Cell therapy: The most commonly studied and utilized stem cell type in veterinary medicine are mesenchymal stem cells (MSC), which can be harvested relatively easily from bone marrow and adipose tissue. MSC have been used primarily in orthopedic applications but new research is looking into using them for both atopic dermatitis and auto-immune diseases like pemphigus. Studies are ongoing for allogenic stem cell products that can be mass produced for immediate and ‘off-the-shelf” access. Human trials have used IV stem cells to treat multiple sclerosis, rheumatoid arthritis and lupus. In vivo studies have shown stem cells to aid in wound repair. In the next few years, look for new studies on the use of MSC in veterinary dermatology.

Oclacitinib: Pfizer Animal Health is working on a new medication for canine AD called Oclacitinib. It development is based on research showing the importance of the cytokine IL-31, which is released by T lymphocytes and is implicated in human atopic dermatitis. Oclacitinib is a selective janus kinase (JAK) inhibitor that blocks the pruritogenic effects of IL-31. Several abstracts were presented at WCVD presenting clinical trial data. A multi-center placebo controlled study with 341 dogs (0.4 mg/kg q12h for 14 days) found significant reductions in pruritus and clinical scores with GI signs (vomiting and diarrhea) the most common side effect. Another study compared oclacitinib and prednisolone in two models or itchy dogs (those injected with IL-31 and those with flea allergy dermatitis) and found oclacitinib to have a faster onset and greater suppression of itch than prednisolone in both groups.

Cytopoint™ is a monoclonal antibody that targets and neutralizes IL-31. IL-31 is an important interleukin that has been shown to be important in contributing to itch in atopic dogs. Cytopoint™ is labeled for use in dogs (NOT IN CATS) given as a SQ injection to block itch. The injection lasts for most dogs for 4-8 weeks. We have also been part of the clinical trials for Cytopoint™. In our experience, it may last even longer but also much shorter, or not at all, in some dogs. We have found Cytopoint™ to be effective in some dogs where other modalities have failed and its safety makes it helpful for dogs that may need other medications to control their allergies.

New therapies: Barrier function aids

Shampoos, lotions and conditioners can be useful in increasing skin moisture, decreasing pruritus and removal of superficial pathogenic bacteria and yeast. In dogs, a double blinded randomized controlled trial showed that a weekly bath with a 10 min application of a shampoo containing ceramides, essential fatty acids, monosaccharides and alkyl polyglucosides (Allermyl®, Virbac) led to a 50% reducing in pruritus scores within 24 hours in 25% of treated. Another study using a weekly bath with Allermyl® shampoo (Virbac) in allergic dogs showed reductions in pruritus, clinical scores and TEWL. Another study looking at twice weekly bathing for 4 weeks with a medicated shampoo containing chlorhexidine, lactoferrin, piroctone olamine, chitosan and essential fatty acids showed significant improvement in pruritus. However, the same improvement was also seen in the control shampoo. This study and others indicate that shampooing in general can help in treatment of atopic dogs and that some shampoos may be better than others.

Topical fatty acid application: The use of topical compounds containing mixtures of skin lipids found in the stratum corneum (cholesterol, free or essential fatty acids, and ceramides) has become a popular therapy for AD. Several recent studies have shown encouraging results using these spot-on therapies with improvement in histological changes in atopic canine skin after 18 days of treatment with a compound containing ceramides, free fatty acids and cholesterol (Allerderm®, Virbac). A more recent study using a weekly spot-on formulation (Dermoscent Essential 6®, LDCA) containing essential oils and unsaturated fatty acids and a daily spray (Dermoscent Atop 7®, LDCA) containing similar ingredients to the spot-on for 8 weeks showed a significant decrease in clinical
scores and pruritus. A larger study using Dermoscent Essential 6® applied once weekly for 8 weeks showed improvement in clinical scores and pruritus compared to control (placebo) dogs. Since these products must incorporated into the stratum corneum, they may take several weeks before becoming effective.

**New therapies: Hydrolyzed diet**
Royal Canin has introduced a relatively new hydrolyzed dry diet called Ultamino where 100% of the novel protein source is under 1kDa in weight with 88% free amino acids and very low molecular weight oligopeptides. The parent protein is feather protein, hydrolysed using a method developed for human products where free amino acids are required. The carbohydrate source is purified maize starch, which contains no protein. Most of the proteins (95%) are less than 1 kDa, which is much smaller than other hydrolyzed diets on the market. A clinical study in 22 dogs with presumed food allergy showed all dogs showing improvement in their clinical and pruritus scores.

**Staphylococcal pyoderma**

Multi-resistant bacteria: Antimicrobial resistance is becoming a problem in veterinary medicine as it has become in human medicine. Methicillin-resistant *S. pseudintermedius* (MRSP) carries the *mecA* gene, which encodes for a mutant penicillin binding protein, which prevents binding of beta-lactam antibiotics. A study first presented in April 2011 looked at 165 dogs with pyoderma cultured and found that over 50% of cases were resistant strains of Staph. After treatment with appropriate antibiotics dogs with sensitive strains of Staph. These dogs were re-cultured and 31% were found to harbor a resistant strain, indicating that acquisition of MRSP during treatment appears to be common. These authors also found that persistence of MRSP on skin and carriage sites is common after the resolution of MRSP pyoderma.

The recently formed Working Group on Antimicrobial Guidelines by the International Society for Companion Animal Infectious Disease (ISCAID) has come up with some guidelines about bacterial culture and antimicrobial susceptibility testing. They recommend bacterial culture be performed in the following cases: if there is a poor response to two weeks of appropriate systemic antimicrobial therapy, if there is emergence of new lesions two weeks or more after the initiation of such therapy, if there are residual lesions after six weeks of therapy combined with cytology demonstrating infection with coccoid bacteria or when cytology demonstrates intracellular bacterial rods.

Samples for culture should be taken from pustules if possible or taken from beneath crusts, or from papules or epidermal collarettes. Laboratories should differentiate coagulase positive and coagulase negative staphylococci and should be able to distinguish *S. aureus*.

First line drugs which for Staph pyoderma include clindamycin, first generation cephalosporins, potentiated sulphonamides, erythromycin, lincomycin and doxycycline. Second line drugs can be used when first line drugs are not effective ( cefovecin and cefpodoxime, fluoroquinolones, chloramphenicol and rifampin). In my opinion, third tier drugs, including vancomycin and linezolid, should not be used in veterinary medicine and saved for human use.

**New therapies: Staphylococcus**

An abstract presented at the WCVD looked at efficacy and adverse effects of rifampicin in canine pyoderma. They studied 19 dogs treated with rifampicin (5-11 mg/kg twice daily for 10 weeks), and found good response to the pyoderma but also GI signs and elevated liver enzymes in several patients. Rifampicin will likely be used more now in the face of MRSP/A infections, but monitoring should be performed. A new fluoroquinolone, called pradofloxacin is available for use in Europe and scheduled to be released in the US. pradofloxacin has good antimicrobial activity against gram positive bacteria, including Staph. An abstract showed good in vitro efficacy of pradofloxacin when tested against 60 isolates from dogs with pyoderma.
Some of the most common skin masses that can usually diagnosed with and FNA and in-house cytology include: vaccine reaction, follicular cyst, lipoma, histiocytoma and mast cell tumor.

**Vaccine reaction**

Vaccine reactions can occur at vaccination sites weeks or months following administration. These masses are subcutaneous and can be soft to slightly firm. The skin over these areas is usually normal. Cytology shows mixed inflammatory infiltrate of mostly lymphocytes and macrophages with some plasma cells, neutrophils, and eosinophils. Macrophages become activated with increased cytoplasmic basophilia, foamy cytoplasm, binucleated forms, and multinucleated giant cells. The key diagnostic feature is macrophages containing phagocytized vaccine adjuvant (seen as a bright pink, purple, or blue globular or granular material). These masses usually resolve spontaneously but can be surgically removed if needed.

**Follicular cyst**

Follicular cysts (epidural inclusion cysts, epidermoid cysts) are nonneoplastic, noninflammatory, sac-like lesions lined by epithelium. Most canine and feline skin cysts are follicular cysts (arise from hair follicles) and include several histologic subtypes that are not cytologically distinguishable. These histologic subtypes have little clinical significance; they are benign and can be completely excised surgically.

FNA of follicular cysts will show abundant keratinocytes with possible cholesterol crystals, hair fragments, and activated macrophages. Within the preparation background, follicular cysts can also contain melanin granules that should be differentiated from bacteria. Cyst rupture and immunogenic keratin exposure to the dermis or subcutis can result in foreign body reaction with mild-to-marked infiltrates of neutrophils, macrophages, and multinucleated giant cells. Because, on cytology, a cyst cannot be differentiated from a cyst within a neoplasm (usually benign follicular neoplasm), histopathology is required to assess architecture; cytologic differentials include trichoepithelioma, infundibular keratinizing acanthoma, and pilomatrixoma.

**Lipoma**

Lipomas, benign neoplastic adipocyte growths, are typically soft, freely movable masses of varying sizes within the subcutis; however, infiltrative lipomas may be firmer and attached to underlying musculature. Before staining, lipoma preparations appear greasy and fail to air-dry. During staining, adipocytes may dissolve in the fixative to produce an acellular cytologic sample. If they do not completely dissolve in the fixative, adipocytes appear as large balloon-like cells arranged in aggregates held together by fine stroma. Individual cells are round to polygonal and contain a large, colorless intracytoplasmic vacuole that peripherally displaces a small, round, condensed nucleus. Complete surgical excision is curative.

**Histiocytoma**

Canine histiocytomas are benign, self-limiting dermal growths that usually occur on the ears, face, and distal extremities of young dogs. Histiocytomas can also occur in older dogs, but other tumors (other round cell tumors) should be considered. These lesions are frequently erythematous, alopecic, and dome-shaped with or without ulceration. On cytologic examination, many individualized, (irregularly) round histiocytes may display minimal or mild anisocytosis and anisokaryosis and contain some lightly basophilic cytoplasm that often stains paler than the platform background and is paler at the periphery as compared with that evident on perinuclear staining. A few punctate, colorless vacuoles may be noted. Nuclei are round to ovoid, variably placed in the cell, and display lacy chromatin with absent or occasional nucleoli. Varying numbers of small lymphocytes may be dispersed with increased lymphocyte numbers observed in regressing histiocytomas. Because histiocytomas cannot be cytologically differentiated from cutaneous or systemic histiocytosis, the latter conditions should be considered in dogs with multiple histiocytoma-like skin lesions.

**Mast cell tumor**

Canine mast cell tumors may be solitary or multicentric in the skin and can appear sequentially or simultaneously. They tend to be alopecic, erythematous, and varied in size. Ulceration may be present in larger masses, and the mass may change size as histamine is sporadically released from the neoplastic cells. On cytologic examination, these tumors frequently contain several mast cells and eosinophils with fewer neutrophils, fibroblasts, and collagen strands. Mast cells are round and individualized and contain lightly basophilic cytoplasm with few-to-numerous deeply basophilic intracytoplasmic granules. The degree of granularity depends on the granule’s staining characteristics, stain type, degree of cellular differentiation, and whether mast cells have recently degranulated in situ.

Often, the granules are so numerous that they physically obscure nuclear features or absorb so much stain that the nucleus stains pale. Diff-Quik may stain mast cells poorly or fail to stain the granules. If the granules do not stain, diagnosis can be achieved by
observing eosinophils mixed with round mast cells that display round, centrally placed nuclei and foamy or vacuolated cytoplasm. Any mast cell tumor should be considered potentially malignant, but greater nuclear pleomorphism (anisocytosis, binucleation) and observation of mitotic figures should increase concern for malignancy. Canine mast cell tumors should be surgically removed with wide margins and submitted for histopathologic evaluation and grading.

**Sebaceous masses**
Sebaceous masses (often adenomas) are “wart-like” masses and are often pedunculated and multilobulated, and they frequently develop on the head or dorsal trunk. FNA will show low cellularity, with rare sebaceous epithelial cells identified. Sebaceous hyperplasia and adenomas cannot be distinguished from each other by using cytologic examination alone; however, sebaceous adenomas are more likely to form a discrete mass.

**Squamous cell carcinomas**
These are very common tumors and the most common tumor of the head and neck of cats. Samples often contain a mixture of cell types because the neoplastic squamous cells are frequently accompanied by inflammatory cells (primarily neutrophils). Depending on the amount of inflammation, diagnosis can be challenging because squamous cells may become large and atypical as a result of the inflammatory process. The large epithelial cells are typically arranged in clusters or sheets. These cells may have a waxy, pale blue keratinized cytoplasm and a round nucleus with prominent nucleoli. Occasionally, cells may contain perinuclear vacuoles. Neutrophils are often observed coating the cytoplasm.
Feline Otitis
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Less common than otitis in dogs, otitis in cats is usually a multifactorial problem that can be just as challenging (or more so) to diagnose and treat. As in dogs, successful management of otitis requires an understanding of the ear anatomy, the various causes and perpetuating factors that contribute to otitis.

Pathogenesis
When confronted with a case of chronic or recalcitrant otitis in a cat, I first attempt to discuss three fundamental concepts with the pet owner. The first point they must understand is that otitis externa is really just a clinical sign, not a final diagnosis. The second is that normal cats (even Persians) do not get otitis. The third point is that bacteria and yeast are not the cause of otitis but the result of otitis.

Why normal cats do not get ear infections: Ears have a way of cleaning themselves; this process is largely due to epithelial migration (EM). EM in the ear begins at the tympanic membrane. As epithelial cells divide, they migrate up and out and in a slight spiral pattern, carrying cerumen and trapped debris and organisms out of the ear. When there is damage to the tympanic membrane or the ear canal epithelium, EM is the process of repair. A recent study evaluated the speed of EM and found that the epithelial cells travel about 100 to 200 μm/day.

Why bacteria are not the cause of otitis: The ear is also normally colonized by a variety of organisms including *Staphylococcus* spp., *Streptococcus* spp., Corynebacterium and *Malassezia* spp. These organisms live in harmony with a normal ear canal. With inflammation, these resident organisms and other opportunistic bacteria like *Pseudomonas* spp. (a ubiquitous organism often found in water and able to colonize plumbing fixtures) can overgrow and cause infection.

Why otitis externa is not a diagnosis: A commonly used conceptual framework for understanding canine otitis externa uses the terms predisposing, primary and perpetuating factors. A combination of these factors is needed to produce clinical disease.

Predisposing factors
Predisposing factors are factors that increase the risk of development of otitis externa. These factors alone do not cause otitis but increase the risk of development and persistence of chronic infection as they facilitate inflammation by altering the ear canal microenvironment and allowing pathogenic or opportunistic organisms to become established. These factors work in conjunction with primary causes and perpetuating factors. Examples include increased environmental temperature and humidity, excessive moisture, adverse reactions to treatments and systemic diseases (e.g. FeLV or FIV, diabetes mellitus, neoplasia).

Primary factors
Primary factors begin the condition. The most common primary causes in cats are ear mites (*Otodectes cynotis*), which is reported to be responsible for up to 50% of otitis externa causes in cats. Other primary factors include hypersensitivities (environmental allergies, cutaneous adverse food reaction, contact allergy, flea allergy dermatitis), other ectoparasites (Demodex, ticks), foreign bodies, keratinization disorders (idiopathic ceruminous otitis), masses (neoplasia, polyps and ceruminous cystomatosis), upper respiratory diseases, soft palate abnormalities, autoimmune diseases (pemphigus complex), drug eruptions, vasculitis.

Perpetuating factors
These factors are changes in the anatomy and physiology of the ear that occur in response to otitis externa even if the primary factor has been resolved and can prevent the resolution of otitis. These factors may prevent resolution of otitis when treatments are only directed at primary and secondary causes. They can accentuate or permit secondary causes such as infection, by providing environments and microscopic niches that favor their persistence. These factors include ceruminous debris and concretions (ceruminoliths), otitis media, over cleaning and topical therapy (excessive moisture, maceration, physical trauma) and less commonly in cats, proliferative changes including hyperplasia and stenosis.

Diagnostic approach
At first exam, a thorough history should be taken. For example, we must know age of onset, unilateral vs. bilateral, seasonal patterns, additional dermatologic signs, current and previous diet, behavior and environment, general health, neurologic signs observed, other pets affected, and what treatments are currently being used and how often and with what success.

Next, a thorough clinical exam (both physical and focused dermatologic exam) should be performed. Begin by palpating the external ear canal to assess pliability and pain. Palpate both mandibular lymph nodes since they often give hints if there is otitis media or more severe inflammation is present in one ear. Check for neurologic deficits that might indicate otitis media. Apply gentle pressure on the ventral aspect of the tympanic bulla using the index finger to apply pressure dorsally past the mandible. Lastly, open the mouth widely to assess for discomfort at the temporomandibular joint, which also may indicate otitis media.
Ear anatomy
The external ear is made of two pieces of cartilage that fit together forming an irregular L-shaped bend in the canal. The annular cartilage makes up the horizontal canal and attaches to the skull. The auricular cartilage attaches to the annular cartilage and makes up the vertical part of the ear canal. It becomes funnel shaped as it travels distally and then finally expands to form the pinna. The vertical canal travels vertically and slightly rostrally before turning medially and forming the horizontal canal. At the junction of the vertical and horizontal canal, there is a prominent ridge (called either the dorsal ridge or Noxon’s or Rosychuk’s ridge). When the ear is in normal position, this ridge blocks the passage of an otoscope.

The following quote from a classic anatomy text dryly sums up this significant anatomical problem: ‘Unfortunately, its external acoustic meatus is curved, making the passage of the straight otoscope for the examination of the proximal part of the meatus and eardrum difficult’ From Dyce, Textbook of Veterinary Anatomy.

The tympanic membrane or ear drum is made of two parts: the pars tensa and the pars flaccida. The pars tensa is the taught, usually transparent part of the tympanum, which is the most obvious part of the ear drum to observe. The manubrium of the malleus attaches to the pars tensa and slightly pulls the tissue of the pars tensa proximally into the middle ear. The attachment of the malleus to the tympanum is called the stria mallearis, which has a slight C-shaped curve, which points toward the nose. From the stria mallearis, small tension lines or striations can be seen as well as blood vessels that supply the ear drum. At the tip of the stria mallearis is the umbo. The umbo is an important landmark, as it is a source of epithelial migration and healing for the tympanum. Care should be taken to avoid damaging the umbo when flushing ears or performing a video-otoscopy. The pars tensa should be clear enough to visualize a bony ridge in the middle ear, which looks like a dorso/ventral white line running. The pars flaccid, or soft part of the tympanum, is dorsal and caudal to the pars tensa and is sometimes blends into the epithelium of the horizontal canal. In other cats, it is more obvious and looks like a pink, smooth, ‘puffy’ bulge. Occasionally, cats will have such prominent pars flaccidas, they can be mistaken for masses or polyps.

Middle ear
The tympanic cavity consists of a small epitympanic recess and a large ventral bulla. The tympanic bulla proper is adjacent or behind to the tympanic membrane. In the cat, the bulla is completely divided by a bony septum. On the medial wall of the tympanic cavity, there is a bony promontory, which houses the cochlea. The cochlear (round) window is located on the caudolateral portion of the promontory. When flushing the middle ear, avoid this promontory or the round window, to avoid damaging the inner ear. The auditory tube is a short canal that extends from the nasopharynx to the rostral portion of the tympanic cavity proper.

The three auditory ossicles, the malleus, incus and stapes, are the bones that transmit and amplify air vibrations from the tympanic membrane to the inner ear.

Otic exam
One of the challenges of assessing otitis externa is the difficulty of performing a good otic examination. In my opinion, few practitioners (including myself!) were taught to properly examine an ear while in veterinary school. In fact, most interns and even some visiting residents that I teach need help with their ear exams. There are two kinds of heads for hand-held otoscopes available in most clinics, the operating head and the diagnostic head. The diagnostic head has a large lens that can completely cover the otoscope head as well as a small port to attach a tube for pneumotympanoscopy. The operating head has a small lens that can be moved and instruments can be passed into the ear canal while still visualizing the ear. I recommend experimenting with both heads if they are available. In my opinion, the operating head is superior for most ear exams as the focal length of the lens is better suited for visualizing the tympanic membranes.

Once you have chosen your otoscope type, you are ready for your ear exam. Understanding the normal anatomy of the feline ear is essential in performing a good ear exam. The cat should be around chest height on a table with a technician holding the nose/rostral face of the cat for restraint (not with their arm around the neck-since this impedes movement of the ear canal necessary for a good ear exam). The otoscope should be placed in the intertragic notch while the canal is then visualized. The most challenging part of the ear exam is passing the dorsal ridge, which lies before the junction of the vertical and horizontal canal. The dorsal ridge is what most people run into when cats seem painful during examination. To avoid this, pull the pinna out (laterally) and slightly down (ventrally) while simultaneously ‘diving’ underneath the dorsal ridge while you advance into the horizontal canal. I find that performing a good ear exam requires more movement and body repositioning than one would think. A good way to practice your ear exam is on sedated or anesthetized patients. After 5-10 ears, you get the ‘pull and dive’ move down. Once in the canal, assess for evidence of ulceration, extent of hyperplasia or exudate/debris. Once in the horizontal canal, advance toward the tympanic membrane. In order to see all parts of the tympanum (pars tensa, pars flaccida, and the stria mallearis) as well as the ‘corner pocket’-a little recess rostral to the tympanum where foreign bodies sometimes hide-you must move your head quite a bit, much like trying to visualize a room through a keyhole.

Otic cytology
After your ear exam, take samples for cytology. Samples should be taken from the vertical and horizontal canal junction using a cotton-tipped applicator. To remember which sample came from which ear, I break the wooden part of the applicator with the left ear.
sample. I smear the sample from the left ear near the frosted part of a glass slide and the right at the other end. Heat fixing is not important. After a quick stain with Diff-Quik, examine the slide under oil immersion (x100). The number of organisms and/or inflammatory cells should be determined at each visit.

Record organisms by their size, shape and number. For bacteria, a scale of 1+ to 4+ is commonly used, with 1+ reflecting a few bacteria (easy to count per filed) and 4+ reflecting a large number (impossible to count, almost a uniform layer of organisms per field). As Malassezia is larger and easier to count, record an approximate average of yeast per high powered filed (hpf), for example, 1-5/hpf, 5-10/hpf, 10-20/hpf or TNTC (too numerous to count-TNTC). More than 4 Malassezia and 10 bacteria per oil immersion (x100) are abnormal in dogs. The presence of organisms is not synonymous does not mean infection. Rare bacteria or yeast noted within the cerumen or on epithelial cells, with no inflammatory cells indicates colonization. Inflammatory cells indicate more significant infection and any organisms engulfed by white blood cells are clear indication of infection.

Obtain a sample of material from the external canal for a mineral oil preparation to look for ear mites and Demodex mites.

To culture or not to culture: The results of bacterial culture and sensitivity and minimum inhibitory concentration (MIC) measurement may be used to determine the best systemic antibiotic choice. There is some evidence that systemic antibiotic therapy is not helpful in the treatment of otitis externa, and may contribute to colonization by resistant organisms. Since most cases of otitis externa will respond to topical therapy (as long as the underlying disease/problem is addressed), I rarely perform a bacterial culture. In my opinion, you will almost always choose the correct topical antimicrobial by making and empiric choice based on your cytologic findings. Even in a case of ‘resistant’ Pseudomonas spp., if you use high enough concentrations and large volumes of topical antibiotics (enrofloxacin for example), you will largely overcome resistance mechanisms. Remember that MICs on culture and sensitivity panels reflect the concentration of antibiotic achievable in the serum after systemic administration of the antibiotic. With topical therapy, the concentrations of the antimicrobial with far exceed drug concentration achievable in the blood. When you suspect otitis media, however, performing a culture and sensitivity is valuable in aiding systemic antibiotic therapy since the organisms present in otitis externa and those present in otitis media are often different. Obtaining an uncontaminated sample of the middle ear is best done through video-otoscopy.

Specific primary causes and treatments

**Otodectes cynotis**
The most common primary cause of otitis externa in cats. Ear mites are contagious and most commonly seen in young cats. Some individuals may carry large numbers of mites and have minimal signs. Others may harbor relatively small numbers of mites, but have significant otitis. Asymptomatic carriers can be a source of infestation. The exudate is usually dark-brown to black waxy, dry and granular. Secondary infections may occur. Diagnosis is usually by otoscopic examination and mineral oil preparation of otic exudate examination. Treatment options: Selamectin two doses every 3-4 weeks; Advantage Multi® spot-on for cats two doses every 4 weeks; fipronil one drop in each ear and rest on back, 2 treatments every 3-4 weeks; oral ivermectin at 0.3 mg/kg PO once weekly for 4 weeks or SQ every 10-14 days for two treatments; topical ivermectin (Acarexx®) or milbemycin (MilbeMite®) two treatments every 2-3 weeks. All "in contact" individuals should be treated.

**Demodex cati**
Some cats may only have easily identified D. cati from ear preparations and will have negative skin scrapes. Clinical signs may be mild and confined to ear canals or abundant brown waxy debris may be seen. D. cati may be seen more commonly in immunocompromised cats. Treatment options: Lime sulfur weekly for 6 treatments or feline flurolaner (Bravecto®) include

**Foreign bodies**
Although it usually presents as a unilateral problem, it may be bilateral. Most commonly, affected cats have acute onset of head shaking and ear pruritus. Secondary infections can occur. Diagnosis is through ear exam and removal of the foreign body under sedation is often curative but may require use of a video-otoscope.

**Hypersensitivity disorders**
Food and environmental allergies, and less commonly, flea allergy dermatitis are common causes of recurrent or chronic otitis externa. Concurrent clinical signs including generalized skin disease and pruritus may be present. Otitis externa may be the only sign of allergic disease. Secondary infections are common. Treatment for FAD, diet trials and allergy testing may be needed to identify underlying hypersensitivity.

**Otic or nasopharyngeal polyps**
Inflammatory polyps are the most common benign otic masses seen in cats. The etiology is unknown but may be due to historical inflammation from respiratory viral infections. Polyps can arise from the mucosal lining of the middle ear, auditory tube or nasopharynx. Polyps arising from the middle ear can extend through the tympanic membrane into the external ear canal and result in otitis externa. Polyps tend to occur in cats younger than 2 years of age but can be seen in older cats. Abyssinian cats may be overrepresented. Polyps may be acute or chronic and may go unrecognized. Polyps are usually unilateral, but can be bilateral. Clinical signs are variable and include pruritus, head shaking, abnormal exudate, inflammation, head tilt, ataxia, nystagmus, Horner's syndrome. Cats with nasopharyngeal polyps may show dysphagia, upper respiratory signs such as stertorous respiration, nasal
discharge, sneezing, voice change, or dyspnea. Secondary ear infections are common. Diagnosing otic polyps begins with signalment, history, and oral and otoscopic examinations and may require general anesthesia. Nasopharyngeal masses tend to be pink and pedunculated. External ear polyps are oval to elliptical, often red, pink, or white, and glisten due to a mucosal covering. Polyps that have not extended through the tympanic membrane may distort or discolor the tympanic membrane before perforation. Imaging under general anesthesia may be needed to help diagnose and identify extension of aural polyps. Skull radiographs may be helpful; however, CT and MRI allow better evaluation of the bulla. A definitive diagnosis requires histopathology. Traction removal with video otoscopy to attempt removal of any poly stalk that may be in the bulla followed by anti-inflammatory doses of steroids can be successful although some polyps will regrow. Removal through a ventral bulla osteotomy (VBO) has a high success rate but has potential surgical complications including temporary or permanent Horner's syndrome, vestibular disturbances, otitis media, hemorrhage, wound drainage, hypoglossal nerve damage, damage to auditory ossicles and vascular structures, and facial nerve paralysis.

Ceruminous cystomatosis
These benign apocrine cysts can occur in any age and are relatively common. Persian cats are predisposed. They are single to multiple dark brown-blush nodules present in the pinnae and ear canals. Diagnosis is based on the characteristic lesions and histopathology to rule out neoplasia. Unless they are occluding the canals, they are often tolerated and do not require therapy. Discomfort and pruritus, stenosis and secondary infections may occur and should be treated properly. Surgical excision or laser ablation can be curative.

Proliferative and necrotizing otitis externa
This is an uncommon cause of otitis most often occurs in young cats and has an unknown etiology. Lesions are usually bilateral with tightly adherent golden-brown hyperkeratotic crusts overlying erythematos plaques present in the medial pinnae and external ear canals. In some cases, lesions may be limited to the ear canals. Erosion, ulceration, pain, depression and anorexia may occur. Otoscopic exam shows digitally proliferative lesions, growing in the entire length of the ear canals, without middle ear involvement. Lesions may be seen on the face, periconically and periorally and generalized lesions have been reported. Histopathological changes are characteristic and confirm the diagnosis. Some cases may undergo spontaneous regression. Treatment options include topical tacrolimus 0.1%, betamethasone or hydrocortisone aceponate, and systemic prednisolone, ciclosporin, retinoids and famcyclovir have been reported to be of benefit.

Otitis media
This can occur without otitis externa in cats, in contrast to dogs, who most often have otitis media due to progression of otitis externa. Upper respiratory disease is a common cause of otitis media in cats. Diagnosis may be made during otoscopic exam as the tympanic membrane may be ruptured or bulging outward. A normal intact tympanum does not rule out otitis media. Head tilt and Horner’s syndrome may be seen. Advanced imaging (CT scan and MRI) is ideal to confirm the diagnosis. Treatment involves myringotomy and middle ear flushing. Ideally, cytology and C&S from the bulla material should be performed in order to select topical and systemic antibiotics. Several months of systemic antibiotics may be needed. Common associated infections include Staphylococcus, Streptococcus, Pasteurella and Pseudomonas.

Neoplasia
Neoplasia is an uncommon cause of unilateral otitis in cats. Most common types of otic cancer includes cerumen gland adenoma/adenocarcinoma, and, less commonly, squamous cell carcinoma. Lymphoma and fibrosarcoma involving the middle ear have been rarely reported in cats. Cats may present infection, discomfort, pruritus, bleeding and inflammation. Diagnosis is based on imaging (CT scan, MRI) and histopathology. Early diagnosis and proper therapy improves disease control and prolonged survival. Adenomas/adenocarcinomas are locally invasive but rarely metastasize.

Secondary ear infections
Otic infections affect cats less often than dogs but are more common in in allergic cats and those suffering from systemic diseases.

Medications for feline otitis
Cats appear to be more susceptible to ototoxicity than dogs, which may be caused by their otic anatomic differences. Cats also appear to develop more contact reactions and irritation from topical medications compared to dogs. In addition, ototoxicity of most topical medications in cats is not known. Extreme care should be taken when selecting otic medications for cats. Sterile physiologic saline is a safe ear cleaner for cats. Douxo Micellar® is also gentle and safe for cats, even with ruptured tympanums. Iodine and chlorhexidine should be avoided. Gentamycin and diocetyl sulfosuccinate should be avoided in cases of ruptured tympanum.

Ear cleaning
Start all treatment(s) with a clean ear. Flushing the ear canal is necessary to remove cerumen and debris to allow topical products to reach the canal epithelium. Purulent exudate and inflammatory mediators can inactivate some medications. In cases of severe otitis, where erosions/ulcerations are present, a short course of systemic glucocorticoids is helpful to reduce inflammation before having the owners clean or medicate the ears. For more severe otitis or when otitis media is suspected, an ear flush under anesthesia is recommended. The most thorough and accurate was to perform a flush, which also allows precise myringotomy and mass removal/biopsy is a video otoscopic flush.

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**Fun with Fungus: New Ideas on Managing Ringworm**

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**Ringworm basics**

Ringworm, or dermatophytosis, is a fungal infection caused by protein-eating fungi. There are many species of ringworm, but the most commonly cultured fungi are *Microsporum* spp. and *Trichophyton* spp. These fungi invade the hair shaft and sometimes surface of the skin (the keratin of the upper layers of the skin) and produce large amounts of infective microconidia. Ringworm is much more common in cats than dogs and most often become infected with *Microsporum canis*. Dogs are more likely to become infected with soil and small mammal-associated species like *Microsporum gypseum* and *Trichophyton mentagrophytes*. In our practice, the following dogs are more prone to dermatophytosis: Boston terriers, Yorkshire terriers and Jack Russell terriers.

**Diagnostics**

1. **Wood’s lamp**

A Wood’s lamp is a hand-held device that emits long-wave (between 320 and 400 nm) ultraviolet radiation through a nickel or cobalt glass filter. Electric (plug-in) Wood’s Lamps are generally more consistent than the battery powered ones and I prefer the brand Burton’s, which has two rows of light bulbs with magnifying lens in the center. The magnifying lens allows the clinician to see if individual hairs fluoresce near the base of each hair, which is important in differentiation between positive ringworm fluorescence and false positive from crusts or topical medications. *Microsporum canis* fluorescence is bright apple green and infected hairs glow from the bulb to the tip. Wood’s lamp is only a screening tool for *Microsporum canis* infections, because not all ringworm strains will fluoresce; negative fluorescence does not rule out dermatophytosis. Wood’s lamp examination is cheap and easy to perform and we find it helpful for examining known positive animals (*Microsporum canis*) or animals where *Microsporum canis* is likely. Fungal culture and/or fungal PCR is important to confirm infection. When examining an animal without lesions, focus carefully on the face, ears and paws, as these are commonly infected areas. Wood’s lamp can help identify which hairs to sample for culture/PCR or for direct examination of hairs under the microscope. Wood’s lamp examination can also help monitor response to treatment. With antifungal therapy, there should be fewer Wood’s lamp-positive hairs and the location of fluorescence should progress from the base of the hair to the tip as the hair grows out, moving the infected section of hair upwards.

2. **Directly examination of hairs**

Hairs plucked from near an affected area or ones that fluoresce under the Wood’s lamp can be placed in mineral oil, covered with a cover slip and examined under 10x. Infected hairs may have fungal hyphae within the hair shaft and small round microconidia or spores on the outside of the infected hair. You may see spores ‘exploding’ from the hair shaft and spores surrounding the hair.

A tape prep can be made of a suspicious area and stained with either lactophenol blue or the purple Diff-Quik stain. Ringworm spores will appear round or ovoid and look a bit like non-budding *Malassezia*. Spores often appear like they have a clear capsule around them.

3. **Fungal culture or dermatophyte culture**

There are many brands of dermatophyte cultures or DTMs (dermatophyte test medium) but the larger surface ares, bi-plate DTM called Derm-Duet II made by Hardy Diagnostics is ideal. The Derm-Duet II has two section, one section for DTM (Dermatophyte Test Media) and the other section for RSM (Rapid Sporulation Media). Each plate is individually wrapped, does not require refrigeration for storage and has a shelf life of a year. Cultures can be kept in a plastic storage bin to prevent desiccations and contamination. Ideally, incubation temperature should remain at 75ºF to 80ºF. To inoculate the plate, you can pluck individual hairs that have or use a new toothbrush or gauze to brush the entire body or a site. Individual hair sampling is best suited for Wood’s-lamp-positive hairs or hairs that are grossly abnormal. When plucking hairs, grasp the hair in the direction of growth and tug gently, trying to get the root bulb. Gently press the hair onto the growth medium. For toothbrush or gauze sampling, comb or wipe until hair is clearly visible on the surface or in the bristles or gauze. Be sure to sample near the eyes, in the ears, and between the toes. When inoculating a plate with a toothbrush or gauze, gently stab the bristles or press the gauze over the entire surface of the plate. Plates should be checked daily for fungal culture growth. Ringworm growth will appear white or buff-colored on the DTM (never darkly colored) and will develop a red color change around them as they grow. Most ringworm species will grow and sporulate within 7 to 10 days but *Trichophyton* cultures often take up to 21 days. Plates should be kept for at least 14 days and preferably for 21 days. Collect tape sample from suspicious colonies and stain lactophenol blue stain or the purple Diff-Quik stain then examine on 10x then 40x for macroconidia.

4. **Fungal/Ringworm PCR**

The Ringworm (Dermatophyte) RealPCR™ Panel from Idexx Laboratories is a newish diagnostic tool for diagnosing dermatophytosis in cats and dogs. The panel includes *Microsporum* spp., *Microsporum canis* and *Trichophyton* spp. real-time PCR tests and performs...
with greater than 95% sensitivity and 99% specificity. Results are available in 1–3 working days. You can submit samples in a few ways: Use a soft bristle toothbrush to comb the suspect lesion, then submit the toothbrush in a ziplock plastic bag. You can also pluck hair with follicles, lift or remove crusts and/or perform skin scrapings from the active border of suspect lesion, then place in a red-top tube. Nails with nail bed scrapings or clippings can be submitted in sealed fungal envelope or sterile container. As with most PCR tests, this test is very sensitive in detecting any dermatophyte DNA. The only downside I can see with this test is the risk of collecting spores from an animal that has picked up spores from the environment and is not actually infected, leading to a false positive. Using the Wood’s lamp to identify suspicious hairs samples can help decrease the possibility of a false positive. We have recently had several cases of ringworm where the fungal PCR was negative and the DTM was positive. This could be due to differences in sampling, but we are currently performing both ringworm PCR and fungal cultures.

**Treatment**

Treatment involves a multi-pronged approach: topical/and or systemic therapy, environmental management, and in the case of *Microsporum canis*, assessment for household canine and feline carriers. Recheck with repeat culture should be performed 1-3 weeks after initiation of therapy and every 1-3 weeks thereafter. Treatment should be continued until 2-3 negative cultures are obtained. Treatment duration is variable and may take from 14 days to 6 months. In healthy patients, spontaneous resolution may occur within three months.

**Itraconazole**

- **Dogs:** 5 mg/kg PO once a day for 7 days, stop for 7 days; repeat pattern 3 times
- **Cats:** 5 mg/kg PO once a day for 7 days, stop for 7 days; repeat pattern 3 times

Itraconazole, a fungistatic triazole, inhibits the cytochrome P450 enzyme lanosterol 14α-demethylase, which converts lanosterol to ergosterol. Decreases in ergosterol affect fungal cell membrane permeability. Avoid alkalinizing agents (eg, H2-blockers, antacids) with itraconazole. Alanine aminotransferase (ALT) and serum alkaline phosphatase (ALP) levels may rise without liver disease signs, though hepatotoxicity is rare. In cats, the oral solution is preferred to capsules. Generic and compounded itraconazole have not been shown to be bioequivalent to Sporanox®. Generic formulations have shown similar pharmacokinetic data; compounded itraconazole has produced low plasma concentrations in dogs and should be avoided. There is a new formulation of itraconazole recently approved for cats called Itrafungol™. This is less expensive than the Sporanox® formulation and we have been impressed with its efficacy and ease of administration.

**Terbinafine**

- **Dogs:** 30-35 mg/kg PO once a day
- **Cats:** 20 mg/kg PO once a day

Terbinafine, an allylamine antifungal agent that inhibits the enzyme squalene epoxidase with a net effect of decreasing ergosterol formation. Terbinafine does not inhibit mammalian CYP and therefore has fewer drug-drug interactions thanazole antifungals. Terbinafine concentrates well in the skin, is well tolerated, and may be used as an alternative when a toxic reaction develops after administration of other antifungal drugs. Some dogs may have heptotoxicity with terbinafine but cats seem to not have similar problems. To my knowledge, there have been no efficacy comparison studies between itraconazole and terbinafine.

**Fluconazole**

- **Dogs:** 5-10 mg/kg PO twice a day
- **Cats:** 50 mg/cat PO once a day

Fluconazole is a fungistatic bistriazole that inhibits cytochrome P450-mediated sterol synthesis affecting fungal cell wall function. Food and gastric pH do not alter bioavailability. It is well tolerated orally.

**Topical recommendations (once to twice a week)**

- Lime sulfur (1:16)
- Enilconazole (1:100)
- Accelerated hydrogen peroxide rinse (1:20)
- Climbazole mousse
- Ketoconazole (1%-2%) shampoo
- Miconazole (1%-2%) shampoo

**Environmental control**

If possible, positive animals should be isolated from negative animals, ideally in an easily-cleaned area. Owner should be instructed to clean all non-porous surfaces with 1:10 household bleach or accelerated hydrogen peroxide (Accel®) twice weekly. New studies show that other cleaning agents like Lysol wipes and 409 are effective in decreasing fungal spores. Daily vacuuming should be performed in areas where ringworm positive animals are kept. Swiffer wipes can be used to clean the floors and walls as their electrostatic nature helps attract spores. Any bedding or upholstered items that are difficult to clean should be thrown out-this includes cat tress as they are notoriously difficult to clean. With severe multi-pet infection situations, it might be necessary to clean the ducts and vents in the house.
and replace air filters. For patients with *T. mentagrophytes*, reduced exposure to heavily-populated rodent habitats or rodent control is recommended. If rodents are kept as household pets, they may be screened for ringworm carriage using the toothbrush technique. Owners should know that ringworm is zoonotic and told to wash hands their after handling all pets. In the clinic, dermatophyte-positive animals should be isolated from other patients and gloves should be worn during examination. Scrubs or lab coats should be changed prior to examining other patients or a disposable gown can be worn.

Exam rooms should be cleaned with 1:10 bleach or accelerated hydrogen peroxide (Accel®).

Guidelines for the diagnosis and antimicrobial therapy of canine superficial bacterial folliculitis
Managing Atopic Dermatitis

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Atopy or Atopic dermatitis continues to be one of the most common dermatological disorders afflicting both dogs and cats. At our referral dermatology specialty practice, 75% of our patients have atopic dermatitis as one of the final diagnosis. The problem is so common and severe that many drugs have been utilized in an attempt to offer relief to the suffering patient. The challenge for the clinician is to try and find the right balance between all of the therapy options, their cost, efficacy and safety. The disease continues to generate research, with new therapies being developed.

The International Task Force on Atopic Dermatitis developed guidelines in 2010 for the treatment of atopic dermatitis which involve a multifaceted approach including

- Treatment of acute flares
- Attempt to ID and avoid all triggers of flare
- Improve skin & coat hygiene
- Treat ongoing pruritus with drug therapy
- Allergen specific immunotherapy should be offered when feasible

The diagnosis of atopic dermatitis is not based on any laboratory or skin test but is based on a combination of signalment, history, clinical signs and the ruling out other causes of inflammatory skin. Obtaining a certain and complete diagnosis for the pruritic patient can be challenging, but is a necessity if efficient and effective care is to be delivered.

When attempting to effectively help a patient with atopic dermatitis it is necessary to understand the pathogenesis of the disease, and teach the client these basic concepts. In dogs, atopic dermatitis is known to be an inherited type 1 hypersensitivity reaction to percutaneously absorbed antigens. Epidermal barrier defects contribute to the pathogenesis. Bacterial and yeast infections provide additional antigens which may exacerbate pruritus.

We try and simplify options with clients and explain there are five groups of options for the treatment of atopic dermatitis. They include supportive therapy, corticosteroids, cyclosporine, oclacitinib (Apoquel®), Cytopoint™ and allergen specific immunotherapy. The point of this lecture is how to minimize the corticosteroids, oclacitinib and cyclosporine (Big Gun Drugs). Allergen specific immunotherapy is covered in more detail in a separate lecture. These options are frequently used in combination in order to obtain synergistic effects, which is an important concept to teach clients. In order to use less of the “big guns” clients must administer more intensive supportive therapy.

Supportive therapy is always a good place to start when treating a “mildly” affected atopic patient and includes antihistamines, essential fatty acids, bathing, restoration of the epidermal barrier, control of secondary infections, and potentially topical anti-inflammatory products. A number of antihistamines have been utilized to control pruritus in dogs. Good clinical trials with placebo controls show the benefits of reducing pruritus ranging from zero to 30%. Many dermatologists will utilize antihistamines as part of the ongoing maintenance control of atopic dermatitis, but recognize their limited value when treating an acute or intense flare. Antihistamines which we currently recommend at our practice include cetirizine, amitryptilline, clemastine, diphenhydramine, and chlorpheniramine. Most are available in generic formulation, and are over the counter, which helps keep the cost low. I usually try 2-3 different antihistamines, but expectations need to be realistic in understanding the value of these drugs may be in their steroid sparing effects. Remind owners to avoid formulas which contain decongestants and pain relief products. At the end of these lecture notes is an antihistamine handout we give to owners.

There are many published reports regarding efficacy of essential fatty acids (EFAs) for the treatment of atopic dermatitis. Unfortunately many of these studies failed to control, or account for the amount of EFAs in the diet, which makes interpretation and comparison of these studies difficult. Most dermatologists support the use of EFAs in the treatment of chronic atopic dermatitis. Despite claims to the contrary, currently it is the position of the Task for on Atopic Dermatitis that there is no evidence of superiority of any particular EFA combination, dosage, ratio or formulation (including enriched diets) to improve skin and coat quality. As with antihistamines, EFAs are not adequate as a single therapy for atopic dermatitis except in mildly affected patients.

Improvement of the epidermal barrier has recently been getting more investigation and implementation. Simply bathing the atopic patients has many benefits including physical removal of antigens, reduction of bacterial and yeast populations, repair of epidermal barrier defects and the anti-pruritic effects of cool water cooling hot inflamed skin. Despite the widespread belief that frequent baths will dry out the skin, most dermatologists believe that a client cannot over bathe an allergic dog. The biggest drawback of frequent baths is the concern of washing away some of the flea control products.

A plethora of OTC and prescription antipruritic shampoos are available with ingredients including oatmeal, corticosteroids, diphenhydramine, pramoxine, lidocaine and coal tar just to name a few. It is the feeling of this author that the higher cost and short-term benefit of these products usually do not justify their use. Instead, at our practice we utilize products with antiseptic and
epidermal restoration effects. Knowledge of any and all infections of the skin should influence the choice of antimicrobial shampoo. Chlorhexidine, triclosan with ethyl lactate, or benzoyl peroxide are chosen for most allergic patients prone to recurring pyoderma. If the skin is oily, or the infection is deeper than a superficial folliculitis, ethyl lactate or benzoyl peroxide is chosen since they are more potent “degreasers” and have follicle-flushing activity. Shampoos with miconazole or ketoconazole are chosen if the skin is infected only with Malassezia, otherwise a shampoo with multiple ingredients may be needed for a mixed infection of bacteria and yeast. Recently we have utilized a shampoo and spray containing Tris EDTA with a 4% chlorhexidine, particularly when dealing with methicillin resistant Staphylococcal infections of the skin.

Formulations which extend or prolong the antimicrobial effects of the product include “Leave on” lotions/sprays/conditioners/mousses. Also the active ingredient can be formulated into “Spherulites™” or “Liposomes” which adhere to the skin and hair with a slow prolonged release.

The final “goal” of shampoo therapy is to repair or restore the epidermal barrier. Products marketed for this function include L-Rhamnose and phytosphingosine, both of which also contain chlorhexidine. There are also a number of new topical “pour on” products available which attempt to mimic and replace the endogenous lipid barrier of the epidermis. They include ceramides with fatty acids (Virbac), phytosphingosine (Sogeval) and EFAs (Dermoscent). Clinical trials are ongoing, but these products make sense if they are in fact able to restore the epidermal barrier, reduce transepidermal water loss, and reduce percutaneous absorption of allergens.

Simple management techniques can be employed to reduce overall allergen load on the skin surface. In addition to frequent baths, the coat can be wiped down on a daily (or more often) basis in an attempt to wipe off allergens. Keeping the hair coat short can reduce the “dust mop” affect of a longer coat. Wearing T-shirts and boots or socks can act as a physical barrier to the allergens.

The advantages of the supportive care options outlined above include safety and benefits, which are seen relatively quickly, although EFA supplementation may require two months before a benefit is seen. Another benefit is that no specific diagnostic testing is required once the diagnosis of atopic dermatitis has been made. There is no cost for monitoring of blood work, or even examinations if OTC products are used. Drawbacks include rather lower efficacy, moderate (or more) cost and they are labor intensive.

The fourth recommendation of the International Task Force is to “treat ongoing pruritus with drug therapy.” We will generally have a lengthy and detailed conversation with the client and explain both short and long-term benefits and side effects of corticosteroids, cyclosporine, oclacitinib (Apoquel®), and Cytopoint™. The pros and cons of corticosteroids are well known to veterinarians and most clients. Cyclosporine can certainly help many pruritic allergic patients, but can be one of the more expensive therapies to maintain except in very small patients. It also commonly causes GI disturbance and cannot be tolerated. There are also many examples of abnormal infections, gingival hyperplasia, and perhaps increased incidence of neoplasia.

One of the newer and exciting medications to treat ongoing pruritus is oclacitinib (Apoquel®), which became commercially available in 2014. Our clinics have used the medication in clinical trial settings since 2009. The advantages of oclacitinib include its rapid onset of efficacy and low incidence of side effects. One of the frustrations of oclacitinib is that for some patients, the efficacy does not last for the full 24 hours after the once daily dosing regimen has been started. We frequently receive calls from both clients and veterinarians about resuming twice daily dosing. We do not recommend twice-daily administration long-term as the chances of immunosuppression are likely to increase. Concerns of increased infection and neoplasia would have to be discussed if twice daily dosing is going to be recommended by a veterinarian. As with all the other options for treating atopic dermatitis, oclacitinib does not adequately control pruritus in all our patients either, so the veterinarian and client are required to consider what additional therapies are needed in addition to oclacitinib to find the best “balance” between drugs, efficacy and safety.

Cytopoint™ is a monoclonal antibody that targets and neutralizes IL-31. IL-31 is an important interleukin that has been shown to be important in contributing to itch in atopic dogs. Cytopoint™ is labeled for use in dogs (NOT IN CATS) given as a SQ injection to block itch. The injection lasts for most dogs for 4-8 weeks. We have also been part of the clinical trials for Cytopoint™. In our experience, it may last even longer but also much shorter, or not at all, in some dogs. We have found Cytopoint™ to be effective in some dogs where other modalities have failed and its safety makes it helpful for dogs that may need other medications to control their allergies.

Even though the final recommendation of the Task Force is “allergy specific immunotherapy (ASIT) should be offered when available”, it seems that with the popularity of drugs such as cyclosporine and now oclacitinib, this option has been cast aside by many practitioners, or only considered if or when these drugs fail. It is the opinion of this author and of the International Task Force on Canine Atopic Dermatitis that this is a mistake. For many atopic patients ASIT can become one of the easier, safer, more cost effective therapies. It can be the only effective therapy that is not a drug, and the only therapy without negative effects on the immune system.

The utilization of sublingual immunotherapy instead of the more traditional injectable immunotherapy has also led to many atopic patients benefiting from immunotherapy more quickly, easily and safely. For ASIT in any form (sublingual or injectable) to be its most efficacious, the clinician will require skills relating to the formulation of allergens, prioritizing positive test results, and teaching the client how to monitor the process.
Pathogenesis
Canine otitis externa affects approximately 10-20% of canine population and is one of the most common diagnoses after dental disease in general veterinary practice. There is evidence that the prevalence of canine otitis externa is on the rise. A 2011 Banfield Pet Hospital’s State of Pet Health Report evaluated data from 770 hospitals and found that the diagnosis of canine otitis has increased by 9.4% since 2006. Since canine otitis externa can be one of the most frustrating diseases for pet owners, having a thorough and aggressive approach to its diagnosis and treatment is essential.

When confronted with a case of chronic or recalcitrant otitis in a dog, I first attempt to discuss three fundamental concepts with the pet owner. The first point they must understand is that otitis externa is really just a clinical sign, not a final diagnosis. The second is that normal dogs do not get otitis. The third point is that bacteria and yeast are not the cause of otitis but the result of otitis.

Why normal dogs do not get ear infections: Ears have a way of cleaning themselves; this process is largely due to epithelial migration (EM). EM in the ear begins at the tympanic membrane. As epithelial cells divide, they migrate up and out and in a slight spiral pattern, carrying cerumen and trapped debris and organisms out of the ear. When there is damage to the tympanic membrane or the ear canal epithelium, EM is the process of repair. A recent study evaluated the speed of EM and found that the epithelial cells travel about 100 to 200 μm/day.

Why bacteria are not the cause of otitis: The ear is also normally colonized by a variety of organisms including Staphylococcus spp., Streptococcus spp., Corynebacterium and Malassezia spp. These organisms live in harmony with a normal ear canal. With inflammation, these resident organisms and other opportunistic bacteria like Pseudomonas spp. (a ubiquitous organism often found in water and able to colonize plumbing fixtures) can overgrow and cause infection.

Why otitis externa is not a diagnosis: A commonly used conceptual framework for understanding canine otitis externa uses the terms predisposing, primary and perpetuating factors. A combination of these factors is needed to produce clinical disease.

Predisposing factors
Predisposing factors are factors that increase the risk of development of otitis externa. For example, conformation (narrow ear canals like in the Chinese Shar-Pei), environment/lifestyle (humid climates, trauma from ear plucking, excessive swimming, over exuberant ear cleaning).

Primary factors
Primary factors begin the condition, For example, parasites (Otodectes, Demodex, ear ticks), foreign bodies (grass awns), tumors (benign-polyps and adenomas, neoplastic-adenocarcinoma, squamous cell carcinoma), hypersensitivity disorders (atopy, adverse food reactions, drug/contact reactions), keratinization disorders, glandular disorders (excessive cerumen/sebum accumulation, sebaceous adenitis), immune-related (hypothyroid) and auto-immune (pemphigus complex).

Perpetuating factors
Perpetuating factors prevent the resolution of otitis. For example, organism overgrowth (bacteria, yeast), loss of epithelial migration, chronic changes in the ear (epidermal/glandular hyperplasia, stenosis, fibrosis, ossification, otitis media).

Failure to identify and address these factors likely will result in ongoing otitis and possible end-stage otitis. In end-stage otitis, inflammation and ear canal hyperplasia leads to stenosis and thickening of the ear canal wall, often with complete closure of the ear canal. Fibrosis of the canal and ossification of the auricular and annular cartilages is the final step in the end-stage ear. At this point, medical therapy cannot reverse these changes, and total ear canal ablation with lateral bull osteotomy (TECA-LBO) is often recommended.

Diagnostic approach
Here are some steps and tips to treat canine otitis externa and prevent the progression to end-stage otitis. At first exam, a thorough history should be taken. For example, we must know age of onset, unilateral vs. bilateral, seasonal patterns, additional dermatologic signs, current and previous diet, behavior and environment, general health, neurologic signs observed, other pets affected, and what treatments are currently being used and how often and with what success.

Next, a thorough clinical exam (both physical and focused dermatologic exam) should be performed. I begin by palpating the external ear canal to assess pliability and pain. I palpate both mandibular lymph nodes since they often give hints if there is otitis media or more severe inflammation is present in one ear. Check for neurologic deficits that might indicate otitis media. Lastly, I open the mouth widely to assess for discomfort at the temporomandibular joint, which also may indicate otitis media.
**Ear anatomy**

The external ear is made of two pieces of cartilage that fit together forming an irregular L-shaped bend in the canal. The annular cartilage makes up the horizontal canal and attaches to the skull. The auricular cartilage attaches to the annular cartilage and makes up the vertical part of the ear canal. It becomes funnel shaped as it travels distally and then finally expands to form the pinna. The vertical canal travels vertically and slightly rostrally before turning medially and forming the horizontal canal. At the junction of the vertical and horizontal canal, there is a prominent ridge (called either the dorsal ridge or Noxon’s ridge). When the ear is in normal position, this ridge blocks the passage of an otoscope.

The following quote from a classic anatomy text dryly sums up this significant anatomical problem: ‘Unfortunately, its external acoustic meatus is curved, making the passage of the straight otoscope for the examination of the proximal part of the meatus and eardrum difficult’ From Dyce, Textbook of Veterinary Anatomy.

There is a variable amount of hair in the ear canals of dogs. Some breeds, like poodles, will have abundant hair extending all the way into the horizontal canal. In the proximal horizontal canal, just at the level of the entrance of the cartilaginous external acoustic meatus are a small number of few fine hairs and often a small amount of cerumen. These hairs are a helpful landmark that indicates you are close to the tympanic membrane.

The tympanic membrane or ear drum is made of two parts: the pars tensa and the pars flaccida. The pars tensa is the taught, usually transparent part of the tympanum, which is the most obvious part of the ear drum to observe. The manubrium of the malleus attaches to the pars tensa and slightly pulls the tissue of the pars tensa proximally into the middle ear. The attachment of the malleus to the tympanum is called the stria mallearis, which has a slight C-shaped curve, which points toward the nose. From the stria mallearis, small tension lines or striations can be seen as well as blood vessels that supply the ear drum. At the tip of the stria mallearis is the umbo. The umbo is an important landmark, as it is a source of epithelial migration and healing for the tympanum. Care should be taken to avoid damaging the umbo when flushing ears or performing a video-otoscopy. The pars tensa should be clear enough to visualize a bony ridge in the middle ear, which looks like a dorso/ventral white line running. The pars flaccid, or soft part of the tympanum, is dorsal and caudal to the pars tensa and is sometimes blends into the epithelium of the horizontal canal. In other dogs, it is more obvious and looks like a pink, smooth, ‘puffy’ bulge. Occasionally, dogs will have such prominent pars flaccidas, they can be mistaken for masses or polyps.

**Middle ear**

The tympanic cavity consists of a small epitympanic recess and a large ventral bulla. The tympanic bulla proper is adjacent or behind to the tympanic membrane. In the dog, there is an incomplete bony septum or tympanic bulla ridge (also called Rosychuk’s Ridge). On the medial wall of the tympanic cavity, there is a bony promontory, which houses the cochlea. The cochlear (round) window is located on the caudolateral portion of the promontory. When flushing the middle ear, avoid this promontory or the round window, to avoid damaging the inner ear. The middle ear cavity of the cat is completely divided by a bony septum into two separate tympanic cavities. The auditory tube is a short canal that extends from the nasopharynx to the rostral portion of the tympanic cavity proper.

The three auditory ossicles, the malleus, incus and stapes, are the bones that transmit and amplify air vibrations from the tympanic membrane to the inner ear.

**Otic exam**

One of the challenges of assessing otitis externa is the difficulty of performing a good otic examination. In my opinion, few practitioners (including myself!) were taught to properly examine an ear while in veterinary school. In fact, most interns and even some visiting residents that I teach need help with their ear exams. There are two kinds of heads for hand-held otoscopes available in most clinics, the operating head and the diagnostic head. The diagnostic head has a large lens that can completely cover the otoscope head as well as a small port to attach a tube for pneumotympanoscopy. The operating head has a small lens that can be moved and instruments can be passed into the ear canal while still visualizing the ear. I recommend experimenting with both heads if they are available. In my opinion, the operating head is superior for most ear exams as the focal length of the lens is better suited for visualizing the tympanic membranes.

Once you have chosen your otoscope type, you are ready for your ear exam. Understanding the normal anatomy of the canine ear is essential in performing a good ear exam. The dog should be around chest height on a table with a technician holding the muzzle of the dog for restraint (not with their arm around the neck-since this impedes movement of the ear canal necessary for a good ear exam). The otoscope should be placed in the intertragic notch while the canal is then visualized. The most challenging part of the ear exam is passing the dorsal ridge, which lies before the junction of the vertical and horizontal canal. The dorsal ridge is what most people run into when dogs seem painful during examination. To avoid this, pull the pinna out (laterally) and slightly down (ventrally) while simultaneously ‘diving’ underneath the dorsal ridge while you advance into the horizontal canal. I find that performing a good ear exam requires more movement and body repositioning than one would think. A good way to practice your ear exam is on sedated or anesthetized patients. After 5-10 ears, you get the ‘pull and dive’ move down. Once in the canal, assess for evidence of ulceration,
extent of hyperplasia or exudate/debris. Once in the horizontal canal, advance toward the tympanic membrane. In order to see all parts of the tympanum (pars tensa, pars flaccida, and the stria mallearis) as well as the ‘corner pocket’-a little recess rostral to the tympanum where foreign bodies sometimes hide-you must move your head quite a bit, much like trying to visualize a room through a keyhole.

**Otic cytology**

After your ear exam, take samples for cytology. Samples should be taken from the vertical and horizontal canal junction using a cotton-tipped applicator. To remember which sample came from which ear, I break the wooden part of the applicator with the left ear sample. I smear the sample from the left ear near the frosted part of a glass slide and the right at the other end. Heat fixing is not important. After a quick stain with Diff-Quik, examine the slide under oil immersion (x100). The number of organisms and/or inflammatory cells should be determined at each visit.

Record organisms by their size, shape and number. For bacteria, a scale of 1+ to 4+ is commonly used, with 1+ reflecting a few bacteria (easy to count per filed) and 4+ reflecting a large number (impossible to count, almost a uniform layer of organisms per field). As *Malassezia* is larger and easier to count, record an approximate average of yeast per high powered filed (hpf), for example, 1-5/hpf, 5-10/hpf, 10-20/hpf or TNTC (too numerous to count-TNTC). More than 4 *Malassezia* and 10 bacteria per oil immersion (x100) are abnormal in dogs. The presence of organisms is not synonymous does not mean infection. Rare bacteria or yeast noted within the cerumen or on epithelial cells, with no inflammatory cells indicates colonization. Inflammatory cells indicate more significant infection and any organisms engulfed by white blood cells are clear indication of infection.

**To culture or not to culture**

The results of bacterial culture and sensitivity and minimum inhibitory concentration (MIC) measurement may be used to determine the best systemic antibiotic choice. There is some evidence that systemic antibiotic therapy is not helpful in the treatment of otitis externa, and may contribute to colonization by resistant organisms. Since most cases of otitis externa will respond to topical therapy (as long as the underlying disease/problem is addressed), I rarely perform a bacterial culture. In my opinion, you will almost always choose the correct topical antimicrobial by making and empiric choice based on your cytologic findings. Even in a case of ‘resistant’ *Pseudomonas* spp., if you use high enough concentrations and large volumes of topical antibiotics (enrofloxacin for example), you will largely overcome resistance mechanisms. Remember that MICs on culture and sensitivity panels reflect the concentration of antibiotic achievable in the serum after systemic administration of the antibiotic. With topical therapy, the concentrations of the antimicrobial with far exceed drug concentration achievable in the blood. When you suspect otitis media, however, performing a culture and sensitivity is valuable in aiding systemic antibiotic therapy since the organisms present in otitis externa and those present in otitis media are often different. Obtaining an uncontaminated sample of the middle ear is best done through video-otoscopy.

**Diagnostic imaging**

Radiology: This is relatively easy diagnostic since can be done in-house. The most commonly uses views are used when assessing ear disease with radiographs: dorsoventral-allows comparison of bullae and petrous temporal bones between sides, rostocaudal open mouth- allows comparison of bullae and external ear canal between sides and lateral oblique-allows bulla evaluation without interference of other bony structures. Non-fancy radiographs can be taken easily and quickly to evaluate whether ossification of the external ear canal is present, which would indicate an ‘end-stage ear’. Radiographs are helpful if obvious middle ear disease or present but studies have shown that normal radiographs do not rule out middle ear disease.

Computed tomography (CT): CT allows more precise evaluation of bony structures in the middle and inner ear than MRI. The tympanic bulla and any bony osteolysis/proliferation can be readily seen with CT as can the ossicles. The sensitivity of CT for detection of otitis media has been reported to be around 83%.

Magnetic resonance imaging (MRI): allows for more precise evaluation of soft-tissue structures than CT and radiographs and should be used when there is concern of soft tissue changes/masses.

**Video otoscopy**

This procedure is both a diagnostic tool and therapeutic tool. It can be used for deep and precise cleaning of the ear canals and middle ear cavity as well as to evaluate the ear canal middle ear cavity. Foreign bodies can be found easily and removed while minimizing damage to the tympanic membrane. Polyps and masses can often be removed or at least debulked and submitted for histopathology. VO also allows collection of specimens for bacterial culture and sensitivity from the middle ear cavity and application of medications directly into the bulla. To maximize access to the ear canal and to decrease debris, I recommend treating the patient for at least 10-14 days with 1-2mg/kg prednisolone orally once daily. I will often prescribe tramadol to help with pain before the procedure and to make it easier for the owner to treat the ear. After several days of glucocorticoids and tramadol, I will have the owners begin flushing the ears (no more than twice weekly) and applying topical ear medications that contain glucocorticoids (twice daily for one week then daily until the procedure). At the two week recheck, if the ear canals are very stenotic and fail to open up with such aggressive oral and topical anti-inflammatory therapy, medical therapy is unlikely to fully resolve the otitis and surgery may be necessary.
VO Procedure: Obtain samples of the otic exudates from the junction of the vertical and horizontal canal for culture and sensitivity. These cultures can then be set aside for possible submission. I rarely submit culture from otic exudate in cases of otitis externa, but once you start flushing the ear, much exudate will be lost and culture results possibly altered by flushing agents. If there is not too much exudates or mass(es) present and you suspect otitis media, collect samples of exudates from the middle ear for culture and sensitivity. To collect samples from the middle ear cavity introduce a sterile 3.5 Fr x 5-1/2" Tom cat catheter with a 2ml syringe filled with 1-2ml of sterile saline through the working port of the video-otoscope into the middle ear cavity. Flush the fluid into the cavity and aspirate for culture and sensitivity. If the tympanic membrane is intact but middle ear disease is present or suspected, you can perform a myringotomy. I use a sharpened Tom cat catheter to push through the tympanic membrane at the caudoventral part of the pars tensa at 5 to 7 o’clock. It is difficult to rupture a normal tympanum but diseased tympanums will tear with little pressure.

I then take pictures of the canal at mid-vertical canal, just beyond the junction and at the level of the tympanum before placing any flushing agents into the canal. These pictures are helpful to show the owner ‘before and after’ pics. I use a cerulytic agent to fill the canal and then massage the canal for several minutes while pulling the pinna to take the L-shape out of the ear canal. This will allow material to more easily be removed. Warm saline is the used to flush the canal with a Tom cat catheter and 12 cc syringes. There are flushing/suction machines available as well but I like the force that the 12cc/Tom cat combination creates. If there is abundant debris or inspisated material, I will aspirate the saline out and repeat the cerulytic to try to ‘break up’ the debris. Repeat flushing until the canal is clean. I often find a biofilm of sorts on the pars tensa of the tympanum in cases of chronic otitis, which needs to be removed by close-up flushing. This material often peels off like old wallpaper being removed.

If there is a tear in the tympanum, flush the middle ear cavity multiple times with saline. After flushing, aspirate all saline and instill ½ cc each of large animal enrofloxacin (100mg/ml) and dexamethasone SP into the middle ear. Warn the owner that temporary or permanent (very rare) vestibular syndrome, facial nerve paralysis and Horner syndrome can occur post-myringotomy or even with an intact tympanum in a cat. After the procedure, I continue topical and systemic steroids after to reduce inflammation help the ear canal heal itself.

Therapy

Ear cleaning
Start all treatment(s) with a clean ear. Flushing the ear canal is necessary to remove cerumen and debris to allow topical products to reach the canal epithelium. Purulent exudate and inflammatory mediators can inactivate some medications. In cases of severe otitis, where erosions/ulcerations are present, I recommend 4 days of systemic glucocorticoids to reduce inflammation before having the owners clean or medicate the ears.

Over the years I have discovered (often the hard way) that most owners have no idea how to properly clean their dog’s ears. Even owners who have had to manage ear infections for years rarely clean ears correctly. I have ear models in each exam room, which I use to show owners the anatomy of the dog’s ear. Demonstrate ear cleaning in exam room with the owner. If I suspect that they are not cleaning correctly, I sometimes have the owners show me how they are flushing. I recommend that they hold on to the pinna and fill the ear canal with cleaner. While still holding onto the pinna, they should gently pull the ear outwards and feel the ear canal starting from the outer ear down to the base. Once they have felt where the ear attaches to the skull, they should massage the horizontal canal so that a ‘squish squish’ sound is made. While still holding the pinna and continuing to pull laterally, they should use cotton balls or gauze to wipe out the excess fluid and debris. I instruct them to repeat this process until the cotton balls come out clean (or if any blood is seen). Once they are done cleaning, they can let the pinna go and the dog will shake any excess fluid out (and sometimes more debris). Here is a link to a video for owners showing how to clean a dog’s ear:
http://www.youtube.com/watch?v=brCwQftfJ0o&feature=plcp

For more severe otitis or when otitis media is suspected, an ear flush under anesthesia is recommended. The most thorough and accurate was to perform a flush, which also allows precise myringotomy and mass removal/biopsy is a video otoscopic flush. After obtaining samples for cytology and/or culture, I instill a cerumenolytic agent and massage for 5 minutes. Warm saline is then used to flush the ear and curettes or graspers can be used to mechanically remove large chunks of debris. I will often spend 30 minutes per ear in order to adequately clean a chronically infected ear. Another benefit of a video-otoscope is that it allows forceful and directed yet precise flushing of the tympanic membrane. In my experience, chronic purulent otitis will leave a ‘biofilm’ of sorts adhered to the pars tensa of the tympanum. This film is somewhat transparent, making it difficult to assess with a regular otoscope. After flushing the majority of the exudate from the ear, I will place my flushing catheter directly against the pars tensa and flush, often observing peels of adherent exudate dislodging from the pars tensa. After flushing and aspirating the excess fluid, I instill 0.5 ml each of 100 mg/ml enrofloxacin and dexamethasone SP.

Topical medications
As mentioned above, choose topical antimicrobials based on cytology findings. When coccoid bacteria are seen, you are most likely dealing with Staphylococcus pseudintermedius or schleiferi or possibly Streptococcus spp. When rod shaped bacteria are seen, Pseudomonas aeruginosa is the most likely culprit.
Many veterinary products are available for canine otitis externa containing combinations of antibiotics such as neomycin, gentamicin, polymixin B, and enrofloxacin with anti-fungal agents and/or corticosteroids. Common treatment protocols include twice-daily treatment for the first week, then once daily for the second week pending a re-evaluation. With any topical ear medication, make sure the patient is receiving enough volume to be effective. Anywhere from 0.5 ml to 1 ml (10 to 20 drops) is usually enough to treat most canine ears.

**Glucocorticoids**

Virtually all cases of canine otitis externa should have the benefit of glucocorticoids. Most organisms thrive in an inflamed environment, reducing the inflammation alone is sometimes enough to allow the ear canals to 'self-cure'. Glucocorticoids reduce cytokine production, resulting in decreased inflammation, pruritus and pain. Glucocorticoids also decrease the production of cerumen and sebum, as well as secretions from the mucoperiosteum lining the middle ear. There are many topical corticosteroids available, including fluocinolone, betamethasone and dexamethasone. Some dermatologists recommend using topical steroids alone (without additional medications like antimicrobials).

Moderate to severe cases of otitis externa usually benefit from systemic glucocorticoids at relatively high doses (prednisone/prednisolone/methylprednisolone orally at 1-2 mg/kg/day). Glucocorticoids have also been shown to aid in the elimination of resistant Pseudomonas strains through changes in the microclimate that no longer favor the growth of the bacteria. Antifungal therapy Many of the topical antimicrobial combination products containing an antifungal should be effective for *Malassezia* otitis. Since there is little concern about the development of resistance in *Malassezia*, I will sometimes use oral antifungals, such as ketoconazole 5 mg/kg q 24h,itraconazole 5 mg/kg q 24h, or fluconazole 5-10 mg q 24h for 2-4 weeks for recurrent *Malassezia* otitis.

**How I treat**

With a typical severe painful purulent otitis externa, I prescribe a gentle pH neutral ear flush and a concentrated enrofloxacin/dexamethasone combination. I prescribe tramadol for pain and recommend that the owner start systemic glucocorticoids at about 1-2 mg/kg per day for 10-14 days. After four days of systemic glucocorticoids and pain medication, the owners are allowed to clean and medicate the ears. I have the owners clean the ears no more than twice weekly and use the topical medication twice daily for the first week then once daily. I instruct the owners not to apply either ear wash or medications for 2 days before a re-check examination so that I can more easily assess the tympanum and deep ear canal. At re-check (in 10-14 days), most cases will be improved greatly allowing the systemic glucocorticoids to be tapered. I perform cytology at initial examination and at all re-evaluations. If the primary cause of the otitis has not been evaluated and treated, tapering the glucocorticoids will simply lead to an eventual flare in otitis. Therefore, the MOST important part to successful treatment of canine otitis externa is finding the cause. Depending on the details of each case, this can mean checking lab work +/- thyroid levels, performing elimination diet trials, intradermal allergy testing and immunotherapy, etc..

**References**

Primary Secretary Otitis Media
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Primary secretory otitis media (PSOM), also known as “glue ear” or “middle ear effusion” or “otitis media with effusion” (OME) is a disease that affects many Cavalier King Charles spaniels (CKCS). PSOM has also been reported in other breeds, including a Boxer, Dachshund and a Shih Tzu. Clinical signs suggestive of PSOM include deafness, neck scratching, abnormal yawning, otic pruritus, head shaking, head tilt, facial paralysis, or vestibular disturbances.

Pathogenesis
In children OME is more common when there are craniofacial abnormalities like cleft palate present: this is thought to be due to abnormal drainage from the Eustachian tube. The cause of PSOM in dogs is not known but several theories have been posed, including Eustachian tube dysfunction and abnormal quantity or thickness of secretion in the middle ear made by the lining of the tympanic bulla (the mucoperiosteum). To evaluate the possibility of Eustachian tube dysfunction, Hayes et. al. evaluated the relationship between nasopharyngeal conformation and otitis media with effusion (OME) in CKCS. They found an association between OME and the brachycephalic conformation. The CKCS with bilateral OME had a significantly greater thickness of the soft palate and reduced cross-sectional area of the nasopharynx compared to CKCS with unilateral OME and those without OME. The association of these changes in relation to the development of PSOM is not known but these anatomic changes in the nasopharynx may impair auditory tube drainage.

Diagnosis
A bulging pars flaccida (soft part of the ear drum) is often seen in dogs with PSOM, but not all dogs with PSOM have a bulging pars flaccida. A study performed by Lynnette Cole in 2012, compared three diagnostic tests (tymanometry—where an instrument changes the pressure in the ear and measures the eardrum responses to sound at different pressures, pneumotoscopy—where a puff of air is blown into the ear canal and the movement of the tympanum is observed and tympanic bulla ultrasonography) using computed tomography (CT) as the gold standard for the diagnosis of PSOM in CKCS. Sixty CKCS (31 females, 29 males) with clinical signs suggestive of PSOM (e.g. hearing loss, neck scratching, pruritic ears) were enrolled in the study. Forty-three (72%) CKCS had PSOM (30 bilateral, 13 unilateral). A large bulging pars flaccida was identified in only those CKCS with PSOM (specificity of 100%); however, only 21/73 ears with PSOM had a large bulging pars flaccida (sensitivity of 29%). Sensitivities and specificities for tympanometry, pneumotoscopy, and tympanic bulla ultrasonography were (84%, 47%), (75%, 79%), (67%, 47%), respectively. Based on the results of this study, a large bulging pars flaccida indicates the presence of PSOM, while a flat pars flaccida may be present in CKCS that have PSOM as well as those that do not. In CKCS with a flat pars flaccida, none of the above diagnostic tests can be recommended as a replacement for a CT scan in the diagnosis of PSOM.

PSOM and Chiari-like malformation (CLM) and syringomyelia
The typical signs of PSOM including: severe head or cervical pain, head tilt, nystagmus, and facial/cervical pruritus are similar to those seen in Chiari-like malformation (CLM) and syringomyelia. Since CKCS are prone to Chiari-like malformation and syringomyelia, Loughin completed a study which looked at the prevalence of PSOM in dogs with CLM. They evaluated sensitivity and specificity of computed tomography (CT) and magnetic resonance (MR) for these two diseases. MR and CT images from 310 dogs with CLM were evaluated for PSOM and compared with surgical findings (myringotomy). MR results were identical to those of surgical findings. 38.7% of dogs with CLM had PSOM. 32% of dogs were asymptomatic. Clinical signs in this study included: pruritus (31%), hyperpathia (28%) and hearing loss (2.5%). There was a significant difference between the prevalence of PSOM in CKCS (46%) and non CKCS (13%); p<0.0001. Compared with MR, CT had a 62% and 100% sensitivity and specificity for the right bulla and a 72.5% and 97.9% sensitivity and specificity for the left. Based on the findings of the Loughin study, PSOM is a common in dogs with CLM, with CKCS being overrepresented. Their findings suggest that MR is superior to CT for the diagnosis of PSOM in dogs.

Hearing loss evaluation
Brain-stem auditory evoked responses BAER testing in dogs measures hearing by measuring changes in the brain’s electrical activity as it responds to sensory stimuli. The changes are recorded and are referred to as evoked responses. Peripheral hearing loss is categorized as either sensorineural or conductive in origin. Sensorineural hearing loss may be due to injury to the cochlear hair cells in the inner ear (sensory) or to the auditory nerve (neural). The most common form of sensorineural hearing loss in dogs is old-age associated hair cell degeneration. Conductive hearing loss is due to abnormal transmission of sound through the external, middle, and inner ears.
A study by Harcourt-Brown et al., measured BAER responses in dogs with PSOM and found that the middle ear effusion in CKCS with PSOM was associated with a mean conductive hearing loss of between 10 to 33 decibels relative to normal hearing level (dB nHL). Interestingly, none of the owners reported any hearing loss in their CKCS. In the study by Stern-Bertholtz et al. impaired loss was reported in sign of PSOM in 13% of the cases 61 PSOM cases evaluated.

**Treatment**

Typical treatment involves performing a video-otoscopic guided myringotomy into the pars flaccida or the caudalventral quadrant of the pars tensa with subsequent flushing of the mucus out of the bulla. If possible, pre- and post-flush air- and bone-conducted BAER testing is recommended to determine the extent of the hearing loss and whether it is due to PSOM or possibly unrelated. Oral glucocorticoids like prednisone (0.5 to 1 mg/kg q 24h for 10-14 days) help with post video-otoscopic inflammation. If concerned, a broad spectrum systemic antibiotic can be prescribed. Clinical signs are usually improved after myringotomy but, eventually, the middle ear will refill with mucus and require another procedure. Some veterinarians have used oral over-the-counter N acetyl cysteine (600 mg q 24h), which may help to extend the symptom-free time. Unfortunately, no prospective studies have been performed to determine the efficacy of N acetyl cysteine in the management of PSOM.

In people with chronic OME, tympanostomy tubes have been used to provide drainage. Two veterinary studies have been published using tympanostomy tubes to treat CKCS with PSOM. Dogs in both studies had relief of clinical signs for a maximum of 8 months when tubes were placed. Some problems were plugging of the tubes and tubes migrating from their placement. Tympanostomy tubes could be an alternative to repeated myringotomies for treatment of PSOM, but requires specialized training (at the level of a human Ear, Nose and Throat surgeon) and special equipment like an operating microscope would likely be needed.
Allergy shots (immunotherapy or desensitization) have been one of the safest, non-drug methods to treat allergies in people and animals for many years. It also remains one of the more challenging aspects of dermatology to master. The success of immunotherapy depends on the accuracy of the test used to identify allergens, the formulation of the allergen and the experience of the practitioner.

Allergen specific immunotherapy is most definitely not a “one size fits all” program. If a veterinarian wants to become proficient at administering immunotherapy, she or he should first become familiar with the regional pollen producing plants, when they bloom, how long they bloom, and how prevalent the plant (and allergen) is in the area. An awareness of the prevalence of indoor, potentially year round allergens, such as house and storage mites, mold spores, animal and human dander and insect particles is also necessary. This requires advanced training beyond what can be learned in veterinary school. The first critical step in achieving success with allergy shots is determining accurately and completely what the patient is allergic to. Some veterinarians, clients, and drug companies spend a lot of time discussing the pros and cons of different types of allergy testing. We utilize intradermal skin testing almost exclusively pinpointing the allergic triggers. We find we get the most accurate results from intradermal testing. This also allows us to customize the list for which we are testing based on specific location, not just region. Our current skin test panel includes 70 different allergens and we are constantly modifying what we test for and strength of the testing allergen based on current research. We feel that the more we are proactive about what we test for and how we adjust our patient’s immunotherapy, the better our results are.

When blood (serology) testing is performed, the testing is performed by various regions. Does it really make sense to lump Southern Arizona in the same region as northern Montana when considering what allergens to test for? Intradermal allergy testing is expensive to set up and maintain, and requires practice and skill interpreting results and is therefore mostly performed only in a specialty setting. If intradermal testing is not available, then serology testing must be utilized. It should be emphasized that the only reason to perform any type of allergy (blood or skin) testing is to follow up with immunotherapy.

In the past the only way to desensitize a patient was to give the allergen by injection. Now we also have the option of sublingual. Once allergy test results are obtained, these results should always be critically analyzed to insure that the results are consistent with the patient’s itch history. This determination should include historical information regarding what seasons of the year are better or worse. If you have an allergic dog, cat or horse, we always want the client to pay close attention to these details since it can make a difference on our allergen selection. If allergy testing reveals positive reactions only to seasonal pollens in a patient who is pruritic year-round, then something is being missed. There is a saying in medicine that goes “treat the patient, not the lab results”. This certainly applies to desensitization and the selection of allergen but this is where knowledge of the regional allergens is necessary. For the outdoor working dog that is pruritic only in the summer and fall, then positive reactions to grasses and weeds should be present, and they need to be emphasized or prioritized when formulating the extract. For the indoor Chihuahua which sleeps under the covers at night and who is itchy year round, then indoor allergens such as dander, mold spores, house dust and house mites need a higher priority in the extract recipe.

The volume, concentration, and frequency of the allergen injections are additional variables which will affect the success of the immunotherapy program. We have utilized a “rush immunotherapy protocol” in over 6,000 patients over 25 years. With this schedule, patients receive beneficial levels of allergens within two weeks. We find patients respond more quickly to this program, which can be important for the suffering patient. Yet each patient will respond differently to immunotherapy so there is no “set in stone” protocol. Determining the most effective volume and frequency of injections requires close observations by the owners and the ability of the clinician to make proper adjustments of the protocol. If you have a patient on a desensitization program, we always like to know about any consistent patterns.

For many allergic patients, immunotherapy is one of the more safe, cost effective and medically effective options for managing their disease. In general it is easy for most owners to administer. It is an excellent choice in large and or young patients where the long term lower maintenance costs are best realized. It is also an excellent choice for the non-seasonal patient where treatment with corticosteroids or cyclosporin on a long-term basis would have medical or financial drawbacks. Consequently it is not as good a choice for the geriatric patient, or patient with short-term seasonal disease. Immunotherapy does not lend itself to starting and stopping (using as needed) unlike the other medical options.
Otitis-video otoscopy
This procedure is both a diagnostic tool and therapeutic tool. It can be used for deep and precise cleaning of the ear canals and middle ear cavity as well as to evaluate the ear canal middle ear cavity. Foreign bodies can be found easily and removed while minimizing damage to the tympanic membrane. Polyps and masses can often be removed or at least debulked and submitted for histopathology. VO also allows collection of specimens for bacterial culture and sensitivity from the middle ear cavity and application of medications directly into the bulla. To maximize access to the ear canal and to decrease debris, I recommend treating the patient for at least 10-14 days with 1-2mg/kg prednisolone orally once daily. I will often prescribe tramadol to help with pain before the procedure and to make it easier for the owner to treat the ear. After several days of glucocorticoids and tramadol, I will have the owners begin flushing the ears (no more than twice weekly) and applying topical ear medications that contain glucocorticoids (twice daily for one week then daily until the procedure). At the two week recheck, if the ear canals are very stenotic and fail to open up with such aggressive oral and topical anti-inflammatory therapy, medical therapy is unlikely to fully resolve the otitis and surgery may be necessary.

VO Procedure: Obtain samples of the otic exudates from the junction of the vertical and horizontal canal for culture and sensitivity. These cultures can then be set aside for possible submission. I rarely submit culture from otic exudate in cases of otitis externa, but once you start flushing the ear, much exudate will be lost and culture results possibly altered by flushing agents. If there is not too much exudates or mass(es) present and you suspect otitis media, collect samples of exudates from the middle ear for culture and sensitivity. To collect samples from the middle ear cavity introduce a sterile 3.5 Fr x 5-1/2" Tom cat catheter with a 2ml syringe filled with 1-2ml of sterile saline through the working port of the video otoscope into the middle ear cavity. Flush the fluid into the cavity and aspirate for culture and sensitivity. If the tympanic membrane is intact but middle ear disease is present or suspected, you can perform a myringotomy. I use a sharpened Tom cat catheter to push through the tympanic membrane at the caudoventral part of the pars tensa at 5 to 7 o' clock. It is difficult to rupture a normal tympanum but diseased tympanums will tear with little pressure.

I then take pictures of the canal at mid-vertical canal, just beyond the junction and at the level of the tympanum before placing any flushing agents into the canal. These pictures are helpful to show the owner ‘before and after’ pics. I use a cerulysatic agent to fill the canal and then massage the canal for several minutes while pulling the pinna to take the L-shape out of the ear canal. This will allow flushing/suction machines available as well but I like the force that the 12cc/Tom cat combination creates. If there is abundant debris or inspissated material, I will aspirate the saline out and repeat the cerulysic to try to ‘break up’ the debris. Repeat flushing until the canal is clean. I often find a biofilm of sorts on the pars tensa of the tympanum in cases of chronic otitis, which needs to be removed by close-up flushing. This material often peels off like old wallpaper being removed.

If there is a tear in the tympanum, flush the middle ear cavity multiple times with saline. After flushing, aspirate all saline and instill ½ cc each of large animal enrofloxacin (100mg/ml) and dexamethasone SP into the middle ear. Warn the owner that temporary or permanent (very rare) vestibular syndrome, facial nerve paralysis and Horner syndrome can occur post myringotomy or even with an intact tympanum in a cat. After the procedure, I continue topical and systemic steroids after to reduce inflammation help the ear canal heal itself.

Staphylococcal pyoderma
Antimicrobial resistance is becoming a problem in veterinary medicine as it has become in human medicine. Methicillin-resistant S. pseudintermedius (MRSP) carries the mecA gene, which encodes for a mutant penicillin binding protein, which prevents binding of beta-lactam antibiotics. The recently formed Working Group on Antimicrobial Guidelines by the International Society for Companion Animal Infectious Disease (ISCAID) has come up with some guidelines about bacterial culture and antimicrobial susceptibility testing. They recommend bacterial culture be performed in the following cases: if there is a poor response to two weeks of appropriate systemic antimicrobial therapy, if there is emergence of new lesions two weeks or more after the initiation of such therapy, if there are residual lesions after six weeks of therapy combined with cytology demonstrating infection with coccoid bacteria or when cytology demonstrates intracellular bacterial rods.

Samples for culture should be taken from pustules if possible or taken from beneath crusts, or from papules or epidermal collarettes. First line drugs which for Staph pyoderma include clindamycin, first generation cephalosporins, potentiated sulphonamides, erythromycin, lincomycin and doxycycline. Second line drugs can be used when first line drugs are not effective (cefovecin and cefpodoxime, fluoroquinolones, chloramphenicol and rifampin). In my opinion, third tier drugs, including vancomycin and linezolid, should not be used in veterinary medicine and saved for human use.

Erythema multiforme/Toxic epidermal necrolysis
Erythema multiforme (EM) is caused by a host specific cell mediated hypersensitivity reaction induced by various antigens. The most common cause of EM in people is herpes virus. In dogs EM can be triggered by drugs, chemicals, infections, neoplasia and food allergy. Toxic epidermal necrolysis (TEN) is usually caused by a drug reaction and occasionally linked to infection or neoplasia. In both EM and TEN, altered keratinocytes become targets of an aberrant immune response, resulting in keratinocyte apoptosis/cell death.
Clinical presentation EM: Erythematous macules to slightly raised papules which spread peripherally and clear centrally. Urticarial plaques, vesicles, bullae, ulcers can occur. Lesions most commonly affect ventrum, mucocutaneous junctions, pinnae and footpads. EM can sometimes cause generalized scaling/crusting, erythema and alopecia. Oral ulcerations may be present in some cases of EM. The affected dogs may be painful and rarely pruritic. Dogs with a severe case of EM may be febrile and systemically ill. Clinical presentation TEN: TEN is a life threatening, acute onset disease. Patients will present with pyrexia, anorexia, lethargy, depression. TEN skin lesions appear as multifocal to generalized erythematous macules or patches involving skin and multiple mucosal surfaces. There are often painful vesicles, bullae, necrosis and ulcers. The oral mucosa, mucocutaneous junctions and footpads are often affected. There may be a positive Nikolsky sign.

Treatment of EM: Identify and treat underlying cause. Discontinue all suspect drugs administered in 2-4 weeks before disease onset. Look for infections and neoplasia.

Mild cases may spontaneously resolve within 2-4 weeks. Immunosuppressive treatment may be needed for severe or refractory cases (prednisone 1-2 mg/kg po q 12-24, cyclosporine 5-10 mg/kg po q 24, azathioprine 50mg/m2). Pentoxifylline 10-30 mg/kg q 8-12 may be helpful.

Severe cases may require supportive therapy and some cases may need IVIG.

Treatment of TEN: Correct underlying cause. The sequelae similar to second degree burns. Provide symptomatic and supportive care. Glucocorticoid use is controversial. Plasmapharesis and immunoglobulin 0.5-1.5 mg/kg IVIG once or twice, 24 hours apart may help but there is still a guarded to poor prognosis for dogs with TEN.

Dorsal thermal necrosis

Most burns in veterinary patients are caused by heat from fires, hot liquids, heating pads, driers and hot metals like wood stoves. Of these, most of us are familiar with the dramatic and often extensive burns caused by heating pads, which can cause full thickness burns of large areas of the skin, often on the dorsum. Sustained exposure to sunlight combined with high ambient temperatures can cause burns that are very similar in appearance to heating pad burns. This type of burn has been reported in dogs and has been called ‘dorsal thermal necrosis’. As opposed to most other types of sun damage, which tend to affect light-colored animals or areas of sparse hair, dark haired dogs or those with patches of dark hair are affected. Black skin absorbs approximately 45% more solar radiation than white skin, so it is likely that these dogs are absorbing more damaging UV and visible radiation. Many cases of dorsal thermal necrosis occur in dogs that have been accidentally left outside at high temperatures or in dogs that are taken on long walks/hikes during the summer. It is probable that these dogs could feel the heat and likely pain during the sun exposure but were unable or not allowed to move away from the heat.

Clinical Features: There is often a delay of several days to a week or more from the time of injury to presentation. Some dogs are presented to veterinarians immediately after the heat exposure for heat related problems like fever, lethargy, and dehydration. They have no overt clinical cutaneous lesions and are often treated with supportive care. The owners of some dogs presented to our clinic for dorsal thermal necrosis report that their dogs seemed to resent petting of their backs soon after exposure with increasing discomfort with time. Cutaneous signs include well-demarcated erythema to erosion and ulceration with deeper burns. The affected area is irregular but affecting the dorsal midline although one of our cases was more affected on one side presumably due to the angle of the sun during a long summer hike. In full-thickness burns, eschar will form and the skin will slough. Secondary infection, with purulent exudate and crust formation is usually present, especially with full-thickness burns.

Treatment: Control of secondary infections, pain management and wound care are essential in cases of dorsal thermal necrosis. Surgical removal of affected skin (once the affected area has declared itself) can speed healing. In cases with large surface area affected, sometimes multiple surgeries are required involving skin grafts and stretching techniques. For smaller areas or for owners where surgery is not an option, wound management with frequent rechecks can lead to a good outcome.

Vasculitis

Vasculitis is inflammation of blood vessels resulting in compromise of blood supply to affected areas. The inflammation is due to overstimulation of the immune system by many possible causes including infections (bacterial, viral, fungal, or tick-borne diseases), drug or vaccine reactions, tumors, and autoimmune diseases (especially systemic lupus). In many cases, an underlying cause cannot be determined. Vasculitis is uncommon in dogs and rare in cats. Any age, breed, or gender can be affected, although some breeds may be over-represented such as Jack Russell Terriers and (in cases of vaccine-induced lesions) small silky coated breeds such as Poodles and Yorkies.

Clinical Features: Symptoms include bruising, localized areas of necrotic (dead) skin and skin ulcers especially in areas such as the ear pinnae, lips, mouth, paws, tail, and scrotum. In vasculitis caused by rabies vaccination, there is localized hair loss at the site of the vaccine which can occur 1-3 months after the vaccine. Some animals with vaccine reaction can later go on to develop more generalized lesions of vasculitis. Some animals with vasculitis can show other symptoms such as lethargy, decreased appetite, fever, muscle disease, joint inflammation, and swelling of extremities.

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Diagnosis: Diagnosis of vasculitis is made by clinical signs, diagnostics to identify underlying causes of the blood vessel inflammation (such as bloodwork and testing for infectious or autoimmune diseases), and skin biopsies. Skin biopsies may show inflammation of blood vessels with resultant damage to skin glands and hair follicles. Biopsies taken later in the course of disease may show more non-specific changes such as thinning or ulceration of the skin and loss of skin glands and hair follicles.

Treatment: Treatment of vasculitis involves identifying and treating underlying causes, if possible, and using medications to suppress blood vessel inflammation. Medications which may be effective include steroids, pentoxifylline, the combination of tetracycline and niacinamide, dapsone, sulfasalazine, cyclosporin, or azathioprine. In some cases medication may eventually be discontinued, however some animals will require lifelong medication for control.

Pemphigus foliaceus

Pemphigus foliaceus is an autoimmune disease whereby antibodies produced by an animal’s own immune system attack the bridges that hold skin cells together. It is the most common autoimmune disease diagnosed in dogs and cats.

Dogs and cats of any age or gender can be affected. In dogs, Akitas, Chow Chows, Doberman Pinschers, Dachshunds, and Newfoundlands may be predisposed. No breed predilections exist with cats. Three forms of Pemphigus foliaceus exist in the dog. The first and most common is the spontaneous form which develops in dogs with no history of skin disease or drug history. The second form of Pemphigus foliaceus is initiated via a drug reaction. The third form occurs in dogs with a history of chronic skin disease (e.g. allergies).

Clinical signs: The primary lesion of Pemphigus foliaceus is a pustule. These lesions typically begin along the nasal bridge, around the eyes, and ear pinnae. It is typical for the lesions to spread and occur along the trunk, feet, clawbeds, groin, and footpads. In cats, the nail beds and nipples can also be commonly affected. In most cases, the pustules form and rupture very quickly, so that all that is left to observe are areas of hair loss, yellow-brown dried crusts, redness and scale. Severely affected animals may become anorexic, depressed and have a fever. The disease itself often displays a waxing/waning course.

Diagnosis: The diagnosis of Pemphigus foliaceus is made by clinical signs, cytology, and biopsy. Other diseases that can appear similar to Pemphigus foliaceus include infection (bacterial, parasitic, fungal), seborrheic skin disease, and varying forms of lupus. Skin scrapes would be performed to rule out external parasites via microscopic analysis. A fungal culture would be done to rule out ringworm (a type of common fungus). Samples of debris from intact pustules or crusts can allow for a diagnosis of Pemphigus foliaceus. In some cases, multiple skin biopsies are required to confirm the diagnosis of Pemphigus foliaceus.

Treatment: Localized cases of Pemphigus foliaceus can be treated with varying strengths of topical steroids. The mainstay of therapy for more generalized cases in both dogs and cats are oral glucocorticoids (e.g. Prednisone). In order to minimize the potential side effects of glucocorticoids (e.g. weight gain, excessive drinking and urinating, liver enlargement), nonsteroidal immunosuppressive drugs are added to the regimen. In dogs, azathioprine and/or cyclosporine can be utilized, while in cats leukeran and/or cyclosporine are the most popular supportive drugs. Other nonsteroidal immunosuppressive drugs include gold salts (dogs and cats) and tetracycline/niacinamide (dogs). Affected animals are started at higher dosages initially until remission is achieved (4-12 weeks), and then are tapered to the lowest possible dosages that maintain remission.
It is a common statement, "animals hide their pain." But do they really? And is pain the same as suffering? Do our animals not want us to see their pain or are they simply genetically programmed to not emotionally respond to it the same way we are? All veterinary professionals know that different species (and even different breeds within species) react differently to stress and physical discomfort.

Understanding these differences and how to think about pain and death from a pet’s point of view helps us better communicate these nuances with clients. This results in a more comprehensive and understandable explanation to the owner of the patient’s perception of discomfort and the capacity for suffering during the end of life experience.

We all understand the broad concept of “pain” (i.e., nociception). Whether or not animals can feel pain is luckily not debated any more. (However, we don’t think insects feel pain since they do not ‘pain guard’ (Eisemann et al 1984), such as limping on a broken leg. The fruit fly is the only known exception to this, they have been shown to pain guard when injured (Tracey et al 2003)). So what is the difference between pain and suffering? After practicing emergency medicine followed by thousands of veterinary hospice cases, I have come to define suffering as the inability to both think about anything else AND the inability to physically do anything else other than address the pain (be it mental or physical) that an individual is experiencing. This would hold true for both the cat in congestive heart failure that is struggling to breathe and the dog with severe thunderstorm anxiety; they are both suffering.

Think about how your dog reacts when you step on his toe and you realize that he has no problem communicating his discomfort to you. But then think about the female Labrador that was spayed a few hours ago and now must be restrained from roughhousing; is she really hiding her pain? Why are these differences in these outward signs of discomfort when we know both of these examples are painful (although much different types of pain)? The first step is coming to an agreement on the term “hide.” When clients tell us they are worried about properly identifying pain in Fluffy because she hides it, does that mean Fluffy doesn’t want her owner to see her pain and therefore displays these outward signs of discomfort in private? Or does it mean that Fluffy is biologically programmed to not show pain at all in order to protect her standing in the pack or to avoid predation (like prey animals)? Or perhaps Fluffy simply doesn’t care about her pain (although fully feels it) in the same way humans do? Does the difference between these concepts really matter? The answer is probably somewhere in the middle but without the ability to speak “dog,” we will never fully know for sure. But any veterinary professional will tell you that even when an animal is alone, they will usually (not always though), act as if their injury or illness does not hurt them as much as we believe it would hurt a person (research is still torn on this subject, however).

So do animals simply experience pain differently than humans? Again, we know there are species differences, but are there major anatomical differences that would help us conceptualize this? As humans, we are considered “higher beings” due to the more developed frontal lobes in our brains. This is what allows us to make music and contemplate our own existence, to name a few. Since animals do not have as much grey matter as we do (and theoretically less “consciousness”), is their experience of pain different than ours? We know that different species can use different parts of the brain for different functions. The connection between the frontal lobes and pain (mainly chronic pain) has been studied for years (Lorenz et al 2003) and was the idea between the controversial and strange practice of leucotomies in the 1940’s and 1950’s. Before antipsychotic medications or therapists this was how society began dealing with the mentally impaired. Basically, the procedure involved cutting the connections to and from the prefrontal cortex, the anterior part of the frontal lobes of the brain (Acharya et al 2004). Besides some pretty awful side effects, there were interesting post-surgical developments in those patients experiencing life-limiting and debilitating pain before the procedure. Patients that were completely nonfunctional due to extreme physical suffering (probably akin to fibromyalgia in modern day) were up and about, playing card games and conversing just days later. They appeared to be better. One patient, after being asked how he was doing, responded, “the pains are the same, but I feel fine now, thank you” (Demasio 1994). There are numerous accounts describing this phenomenon. After the procedure, it is said that the patients stopped caring about their pain; Dr. Demasio noted that they “kept their pain but lost their suffering.” These patients still asked for painkillers but were satisfied with aspirin, no longer needing morphine. It’s clear that they still felt pain because when poked with a pin they shrieked; in fact, they shrieked louder than a normal person, probably due to lower impulse control from the disconnected frontal lobes. These patients were most likely feeling what normal humans consider mild pain; that which still exists and causes discomfort but can be ignored and does not ruin your life by consuming your mental thoughts. (This procedure was all but extinct by the 1970’s due to a myriad of undesirable side effects such as loss of initiative, inhibition, and decreased cognition to name a few.)

Perhaps animals lie somewhere in the middle of a leucotomy patient and a normal human being. To me, there are many similarities, although certainly not the same, between the leucotomy patients and dogs in how they emotionally react to pain. I am not inferring that animals don’t physically feel as much pain as normal humans do, simply that they don’t emotionally interpret, respond, and react to it the same way a normal human with intact frontal lobes would. For example, a few years ago my rat terrier jumped out
of a friend’s arms, completely fracturing her radius (complete mid-diaphyseal fracture). She did not whine, cry out, or even hide (although other dogs might have done these things). She simply jumped on the couch and sat there looking at me with her bright eyes, holding up her mangled leg. I knew she was hurting just from the look on her face, but she honestly reacted the same as if I had stepped on her foot. Of course I was a mess; I knew this meant my little girl would have to go to the veterinary hospital (which she hated), put under anesthesia, surgery, recovery, cage rest, and so on. I took on the emotional component while she experienced the pure and unadulterated physicality of pain, seemingly void of interpretation to what that pain meant. And yet the benefit of my understanding was that I knew her pain would eventually end. Animals, on the other hand, cannot perceive an ending to their state of pain, making our job of pain identification and treatment incredibly important.

If it were me that broke my arm, I would be anxious about the impending surgery, recovery, loss of time with my children, and so on. I would generate negative emotions that lead to amplification of my physical pain. The bright side, however, is that I know that with some medical attention I will be out of pain in the future. Animals may not experience this in the same exact way that I would, but watch a fearful dog walk into a veterinary clinic, or a thunderstorm completely debilitate an animal and you will see a pure form of suffering. Temple Grandin, PhD. says in her book Animals in Translation, “the single worst thing you can do to an animal emotionally is to make it feel afraid… fear is so bad for animals I think it’s worse than pain.” Herein lies the most important part of managing end of life cases in our hospice practice; address physical pain but most importantly address any stress, anxiety, and fear that our pets are experiencing as a result of either their physical or mental discomfort. Many of our arthritic or immobile pets appear more agitated by their inability to stand up rather than the pain that standing up elicits. These dogs may not understand why they cannot ambulate, leading to excessive panting, whining, crying, and additional physical pain through their attempts to move. Much of the time these symptoms are alleviated simply by the owner’s presence, but this is not always possible. Many times, the mental battle is bigger than the physical one with our patients.

These are concepts I discuss with families on a daily basis. Veterinary hospice care, by striving to maintain quality of life versus quantity of life, is centered on addressing pain AND any other mental stressors that may be present. To this extent, the owner becomes our greatest source of early identification of new developments with their pet’s condition. They generally feel that their bond is so strong that they can sense the discomfort, and with a little retraining and education on how an animals may react and perceive pain AND anxiety differently than we do, we can become partners in the journey of making the end of life period as pain-free, anxiety-free, and fear-free for both the pet… and yes, for the owner as well. There needs to be a clear understanding of the differences between discomfort (something we will all have when we’re 95 years old!), pain (something that should always be addressed), and suffering (a mental state that should be avoided at all costs). Euthanasia is not just about ending suffering that is occurring at that moment, but rather about preventing it from occurring in the first place. And with a better understanding of mental and physical pain and/or suffering, clients feel better equipped to make that important decision with the guidance of their veterinarian.

Helping families identify pain in their pets
As veterinarians, we have many resources available to assist in pain identification in animals. The International Veterinary Academy of Pain Management is a wealth of information. Below are some additional helpful tips that we have found particularly useful when talking with clients during the hospice period.

1. **Does your pet act overly concerned when you approach him?** Does he seem to shy away from your caresses? This may indicate anticipatory pain. Your pet may be anticipating discomfort that is elicited when being touched or moved. Humans in hospice care show a similar phenomenon of not wanting to be touched when the body is nearing the end (usually days to a few weeks before death).
2. **Is your pet appearing more hunched back or grumpy, especially after waking up?**
3. **Pay special attention to the way your dog lies down.** Hiding a certain paw can indicate even mild pain in a related part of the body. Taking a few pictures of him throughout the day may illustrate mild changes.
4. **Many dogs blink the moment they feel pain.** If you see it, try to replicate the movement again or note how and why the increased blinking occurred. Along those same lines, follow your dog's eyes. Avoidance of eye contact or looking away can indicate pain.

Resources
A Sure-Fire Approach to Cases of Canine Thyroid Disease
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Canine hypothyroidism, while a common endocrinopathy in the dog, may be over diagnosed due to confusion/inconsistencies in establishing a definitive diagnosis.

Etiology/pathophysiology
Hypothyroidism is due to decreased thyroidal production of the thyroid hormones thyroxine (T4) and triiodothyronine (T3). Greater than 90% of cases are primary and are due to acquired immune mediated destruction of the thyroid gland which is preceded by thyroiditis, idiopathic atrophy or less commonly neoplasia. Secondary forms of the disease include thyroid stimulating hormone (TSH) deficiency, pituitary neoplasia, and cystic Rathke’s pouch, are uncommon clinical entities. Tertiary hypothyroidism with thyrotropin releasing hormone (TRH) deficiency has not been documented in dogs. Congenital cases have been reported in both dogs and cats.

Signalment/history
Hypothyroidism most commonly occurs in young to middle aged dogs with an average age of 7 years. Dogs with autoimmune disease tend to develop hypothyroidism at a younger age. While thyroid values decrease within the reference range in senior dogs, hypothyroidism is very uncommon and other factors (see below) are likely responsible for the observed decreased thyroid concentrations in euthyroid older patients. Spayed females and neutered males are at an increased risk when compared to sexually intact animals. Breed predispositions have been reported for golden retrievers and Doberman pinschers. Thyroiditis is heritable in the beagle, Borzoi, golden retriever, great Dane, Irish setter, Doberman pinscher, and old English sheepdogs.

Risk factors
No known environmental factors have been identified. Breed predispositions as outlined above.

Historical findings
As thyroid hormone regulates the metabolic rate and influences the functions of many organs, clinical signs are often non-specific and insidious in onset. Many other diseases can have similar clinical signs to hypothyroidism, which may lead to an incorrect diagnosis. As such laboratory testing of thyroid function is often performed as part of the diagnostic work in animals with non-thyroidal illness.

Clinical features
Common clinical signs include lethargy, mental dullness, weight gain, exercise intolerance, alopecia, and obesity.

Differential diagnosis
Many metabolic, infectious, neoplastic, congenital, degenerative, and inflammatory diseases can cause similar clinical signs and biochemical abnormalities seen with hypothyroidism.

Diagnostics
Laboratory diagnosis
Thyroxine is the major secretory product of the thyroid while the majority of T3 is derived from extra-thyroidal sources. Both T4 and T3 are highly protein bound to serum carrier proteins such as thyroid binding globulin, transthyretin and albumin. Only unbound (free) hormone is able to penetrate cell membranes, bind to receptors and result in biologic activity. Protein bound hormone acts as a reservoir to maintain steady concentrations of free hormone in the plasma despite rapid alterations in release and metabolism of T3 and T4 and changes in the plasma protein concentrations.

Serum total T4
Serum T4 is a sensitive (>90-95%), but not specific test (70-75%) for the diagnosis of canine hypothyroidism. The vast majority of dogs with hypothyroidism have a serum T4 below normal, but some normal dogs and those with a variety of other problems may have a low serum T4. A diagnosis of hypothyroidism can be ruled out if the T4 is in the upper 50% of the reference range. Autoantibodies to T4 occur in about 15% of hypothyroid dogs, and these antibodies may falsely increase the serum T4 concentration from below normal into or above the normal range. In house testing of TT4 is not recommended.

Serum total T3
Serum T3 concentration is an unreliable test for evaluation of thyroid function.

Serum free T4 (fT4)
Thyroxine is highly (99.9%) protein bound in the circulation. Protein binding can be altered by many nonthyroidal illnesses and by certain drugs. Measurement of the unbound or free hormone can provide a more accurate assessment of thyroid function in these cases (sensitivity > 95%, specificity > 97%). The sensitivity of fT4 is equivalent to or slightly better than total T4 in diagnosing hypothyroidism in routine cases. More importantly, fT4 is more specific, particularly when non-thyroidal factors that can influence
total T4 are present. Free T4 is less affected by most non-thyroidal illness and drugs, but still can be altered in cases of moderate to severe illness. In addition, fT4 by equilibrium dialysis is not affected by the presence of T4 autoantibodies that will falsely elevate total T4. Measurement of fT4 by equilibrium dialysis should be performed when uncommon clinical signs of hypothyroidism are present, the dog is being treated with a drug that may affect thyroid function, when non-thyroidal illness is present, and if autoantibodies to T4 are detected.

**Serum TSH**
Primary hypothyroidism results in a decrease in T4 and thus decreased negative feedback on the pituitary gland. In response, the pituitary secretes more TSH and plasma TSH levels increase. In man, TSH is elevated prior to any decrease of T4 or fT4 outside the normal range. In the dog, TSH concentration is elevated in only 65-75% of cases of hypothyroidism, as such it lacks sensitivity for use as a screening test. The combination of decreased total T4 or fT4 with an elevated serum TSH is diagnostic of hypothyroidism (specificity > 95%). Therefore, a normal TSH does not rule out hypothyroidism, but an elevated TSH combined with a low T4 or fT4 provides a definitive diagnosis.

**Diagnosis of thyroiditis**
Antibodies against either T4 or T3 or both are sometimes present in dogs with thyroiditis with or without hypothyroidism. The presence of these antibodies does not indicate that the dog is hypothyroid, but suggests that autoimmune thyroid disease is present. These antibodies frequently cause false elevation of T4 or T3 concentrations that can result in marked elevation of the hormones. Autoantibodies to T4 are present in about 10-15% of hypothyroid dogs.

Dogs with autoimmune thyroiditis may have circulating antibodies to thyroglobulin, the primary protein in the colloid of the thyroid gland. This is not a test of thyroid function, but rather a marker for the presence of autoimmune thyroiditis. In one long-term study at Michigan State University, 20% of asymptomatic, antithyroglobulin positive dogs with normal thyroid function progressed to hypothyroidism in 1 year. The presence of these antibodies in a dog with borderline laboratory evidence of hypothyroidism and clinical signs supports a diagnosis of hypothyroidism.

**Additional considerations**

**Breeds**
Certain breeds have normal ranges of thyroid hormones that are different from most other breeds. Few have been evaluated, but greyhounds have serum total T4 and fT4 concentrations that are considerably lower than most other breeds. Scottish deerhounds, Saluki’s and whippets also have total T4 concentrations that are well below the mean concentration of dogs in general. Alaskan sled dogs have serum T4, T3, and fT4 concentrations that are below the reference range of most pet dogs, particularly during periods of intense training or racing.

**Time of day**
In one study 50% of normal dogs had a low serum T4 concentration at some time during the day.

**Medications**
The drugs that are known to commonly alter thyroid function tests are glucocorticoids, phenobarbital, sulfonamides, clomipramine, aspirin, and some other NSAIDs. Glucocorticoids suppress total T4 and sometimes fT4 as well. Phenobarbital causes decreased total T4 and mild increases in TSH. Sulfonamides can induce overt primary hypothyroidism with clinical signs and thyroid function tests that support the diagnosis. The changes may be reversible when the medication is discontinued. There are dozens of drugs that affect thyroid function and thyroid function tests in man, so many others likely affect the dog as well.

**Nonthyroidal illness**
Illness not involving the thyroid gland can alter thyroid function tests and has been labeled "non-thyroidal illness" or "euthyroid sick syndrome". Any illness can alter thyroid function tests, causing a fairly consistent decrease in total T4 and T3 concentrations in proportion to the severity of illness. Serum TSH concentration is increased in 8-10% of dogs with non-thyroidal illness. Serum fT4 measured by equilibrium dialysis is less likely to be affected, but can also be increased or decreased. However, in dogs with substantial non-thyroidal illness, the fT4 is likely to be decreased. It is recommended that testing of thyroid function be postponed until the non-thyroidal illness is resolved. If this is not possible, measurement of T4, TSH and fT4 are indicated.

**Ancillary testing**

**Thyroid gland ultrasound**
Although rarely necessary, ultrasound of the thyroid glands (by an experienced ultrasonographer) can be used to aid in differentiating dogs with primary hypothyroidism from those with non-thyroidal illness. Thyroid glands of hypothyroid dogs tend to be smaller, less homogeneous, and hypoechoic than those of euthyroid dogs. There is considerable overlap with the ultrasonographic appearance and size of the thyroid glands of euthyroid and hypothyroid dogs. Thyroid ultrasound can only be used to help support a diagnosis of hypothyroidism if the thyroid glands are quite small.
Therapeutics
Drugs
Levothyroxine is the only hormone that appears necessary for treatment of hypothyroidism. The frequency of levothyroxine dosing is controversial, and the only study to closely evaluate the response to treatment showed that once daily treatment is adequate. However, in clinical practice some dogs seem to respond better to twice-daily treatment.

The initial starting dose is 0.02 mg/kg PO q 24 h. In general you will never have to exceed exceed 0.8 mg as an initial daily dosage even in very large dogs. If the dog has significant cardiovascular disease, diabetes mellitus, or hypoadrenocorticism, treatment should be instituted at 25% of the standard dose, with the dosage increased by 25% every 2 weeks based on clinical response and post-pill testing. Most dogs show improvement within the first 1-2 weeks, with increased activity, improved attitude, and partial or complete resolution of neurologic signs. The cutaneous manifestations of hypothyroidism may take several weeks to months to resolve. Post treatment monitoring may be carried out but clinical response is the most important monitoring tool. Peak T4 concentrations generally occur 4-6 hours after administration of levothyroxine and should be in the high normal to slightly above normal range (40-70 nmol/L). However, the bioavailability of thyroxine ranges from 13 to 87% in the same dog from day to day bringing into the question the utility of random post pill monitoring of TT4. It is likely more meaningful (though more expensive) to measure TSH (especially if the TSH concentration was elevated pre-treatment) or fT4 concentrations after replacement therapy has been started, especially in animals that show a poor clinical response to therapy. Serum TSH concentrations should be in the normal range or undetectable and fT4 concentrations should be in the normal range. Serum concentrations of TSH and fT4 should not be performed until the patient has been on supplementation for at least 2 weeks. If the patient was initially started on twice daily therapy, treatment can be reduced to once daily treatment when a good clinical response has been obtained.

Hyperthyroidism is the most common complication of treatment with levothyroxine, but it is rare in dogs. Clinical signs are similar to those of hyperthyroidism in cats and the diagnosis is confirmed by documenting a substantial elevation of serum T4. Treatment consists of stopping levothyroxine treatment for 2-3 days, then instituting treatment at a lower dose.

Comments
Expected course and prognosis
Response to therapy should be observed in the first 4-8 weeks post treatment. Improvements in mentation and physical activity may be noted within the first week though some abnormalities, especially dermatologic signs, may take several months to resolve. An absent or incomplete response to therapy may be due to an incorrect diagnosis, poor owner compliance, inadequate dosing, or poor absorption.
How I Treat Diabetes in Cats
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Diabetes mellitus is a common endocrine disorder in dogs and cats. Recent data has shed light on the pathogenesis of the disorder in dogs and cats and has highlighted the role of diet, insulin and novel hypoglycemic therapies. In the majority of cases, the most appropriate therapy in both dog and cats includes the administration of insulin.

The key to successful management of the diabetic patient lies in close communication with the pet owner and prompt recognition and treatment of concurrent disorders.

Key facts
1. Insulin is still the mainstay of therapy in the majority of dogs and cats with diabetes mellitus.
2. Diet is an important part of diabetic management especially in obese patients and cats.
3. Auto-immune disease, pancreatitis and amyloidosis are the most common causes of diabetes in dogs and cats.

Successful management of the diabetic patient involves many factors. An understanding of dietary therapy, insulin preparations, oral and novel hypoglycemic agents and management of concurrent illness, are all required to optimize glycemic control. The goals of therapy are to control clinical signs, prevent or slow the progression of cataracts, avoid hypoglycemia and maintain ideal body weight. An additional goal in cats is to obtain remission. The challenge is to address these concerns while attempting to help the owners deal with what they may consider a time consuming, expensive and chronic medical condition.

Diabetes Mellitus in dogs and cats results from a decrease in insulin secretion from the beta cells of the pancreas and/or a decrease in insulin action. There are three classifications of diabetes:

**Type I** diabetes is comparable to insulin dependent diabetes mellitus (IDDM) in humans. It results in low basal insulin concentrations with impaired insulin secretion following a glucose load. Treatment requires insulin injections. It is the most common form of diabetes in dogs.

**Type II** diabetes is similar to non-insulin dependent diabetes (NIDDM) in humans and is managed with dietary therapy and oral hypoglycemics. It causes normal to increased basal insulin concentrations with decreased secretion following a glucose load. Insulin may or may not be required for animals with Type II diabetes.

**Type III** diabetes is seen most commonly in hormonally-induced diabetes in dogs and cats and is similar to impaired glucose tolerance (IGT) in humans. Diabetogenic hormones (epinephrine, cortisol, glucagon and growth hormone) or medications interfere with insulin action and cause glucose intolerance, which can lead to diabetes.

Etiology and signalement
Feline
The most common causes of diabetes in cats are obesity, pancreatitis and most commonly, amyloidosis of the pancreatic beta cells. There appears to be very little gender predisposition to this disease in cats, although it is slightly more common in males than females. As with dogs, the onset of diabetes in cats occurs most often in middle age.

Clinical signs
The clinical signs of diabetes include PU/PD (polyuria and polydipsia) from hyperglycemia, resulting in glycosuria and a resultant osmotic diuresis. Polyphagia and weight loss is common although many animals will still be obese upon presentation. In addition to the polyphagia, there may be variable degrees of dehydration especially in the cat. Cataract formation is very common in dogs with diabetes, but rare in cats. Cats often present with icterus as a result of concurrent hepatic lipidosis and/or pancreatitis. Icterus is not common in dogs unless they have pancreatitis. Cats may also exhibit a plantigrade stance (peripheral neuropathy) that is directly related to the severity and duration of hyperglycemia. Clinical neuropathies do occur in dogs, but are extremely rare.

Differential diagnoses include: hyperthyroidism (in cats), gastrointestinal lymphoma, hepatic disease, renal disease, pancreatitis, hyperadrenocorticism, and acromegaly.

Diagnosis
Diagnosis involves testing for persistent fasting hyperglycemia, with fasting blood glucose greater than 200mg/dl. Clinicians also will need to rule out transient hyperglycemia that may be due to: post-prandial hyperglycemia; diabetogenic hormones (endogenous or exogenous); and stress hyperglycemia. Stress hyperglycemia can be a problem in cats due to the release of epinephrine when stressed or handled.
Laboratory abnormalities include:
- Hemogram
  - non-specific
  - signs of dehydration
- Biochemistry profile
  - hyperglycemia
  - increases in SAP and ALT
  - increases in bilirubin (usually in cats)
    - hepatic lipidosis
    - pancreatitis
- Urinalysis
  - glycosuria
    - renal threshold for glucose
      - canine 180-220mg/dl
      - feline 240-300 mg/dl
  - ketonuria
  - up to 40% of patients will have positive urine cultures in the absence of an active urine sediment.

Treatment
The number one cause of death in diabetic dogs and cats is not the disease itself, rather, it is the owner's frustration with the disease. This is an extremely important point to remember when treating diabetic animals. Good communication with the pet owner is perhaps the most important component of managing the disease.

It is recommended that clinicians schedule a 30-minute appointment with the client at the time of discharge before sending the diabetic patient home for the first time. During this appointment, clinicians should thoroughly discuss the care required for the patient. Include the following instructions in that discussion: how to give the animal injections; how to store insulin, what types of food to feed and how often; how to recognize the signs of hypoglycemia and how to react to this condition. Also include information on what clinical signs to look for in terms of monitoring water intake and urine production. The client should be give written instructions for use as a reference once they are caring for the patient at home. It is essential that the clinician and veterinary staff strive to educate the caregiver and motivate them to get involved in the care of their diabetic pet.

The goals of treatment include elimination of the clinical signs of diabetes, prevention or slowing of cataract formation and resulting blindness, prevention of potentially dangerous hyperglycemia, and prevention and/or treatment of concurrent illness.

Therapy for diabetes centers on three main areas: Treatment of concurrent illness (i.e., urinary tract infections, pyoderma, etc.), insulin therapy, and dietary management.

Concurrent illness
Monitoring for concurrent illness is very important in effectively managing diabetic dogs and cats. Clinicians must effectively recognize and treat the other disorders because the concurrent illness will impact the diabetic regulation and many common diseases have similar clinical signs to diabetes mellitus. Even simple problems such as UTI's and pyoderma can result in activation of stress hormones and result in insulin resistance.

Insulin therapy
There has been a considerable amount of confusion over the various insulin preparations that are available. In general, animal origin insulins are being discontinued as the desire and ability to treat people with human derived insulin preparations has progressed.

There is concern that animals receiving human insulin will develop antibodies resulting in decreased insulin activity and/or effectiveness. Dogs receiving any insulin product that is not derived from pork may make antibodies. However, studies have shown that those antibodies do not interfere with the glucose control. In fact, dogs that made antibodies against insulin had a longer duration of insulin action, which actually enhanced the effect of the insulin rather than decreased its efficacy. A recent study in cats showed that 13% developed anti-insulin antibodies. None of the cats should signs of insulin resistance.

The options with human insulin include ultra short acting, short acting, intermediate acting, and long-acting insulins. The short acting insulins are primarily used for ketoacidosis, and therefore, are not covered in this article. The intermediate acting insulins are classified as either NPH or Lente. It is important to note however, that even though they are classified as intermediate, they do not behave the same way in the dog or cat. Lente is actually a mixture of two different insulin preparations, which results in a bimodal onset of actions. This is helpful in some patients because it helps block post-prandial hyperglycemia. Conversely, a lente insulin is not recommended for use in an animal that does not develop post prandial hyperglycemia. It is recommended that NPH be used in the
majority of dogs and cats with diabetes and it is also understood that most patients will require two injections a day to achieve glycemic control.

**Feline patients**

**Newly diagnosed patients**

1. **Insulin glargine (Lantus):** Glargine is a modified, recombinant, long acting insulin analog. A study presented at ACVIM in 2005 showed a very high rate of remission (8/8 in remission within 4 months with 6/7 still in remission at 1 year) in feline diabetics with the use of glargine and a low carbohydrate-high protein diet. The recommended starting dose is 0.5 units/kg BID if the fasting blood sugar is greater than 360 mg/dl and 0.25 units/kg BID if the initial fasting blood glucose is less than 360 mg/dl. For additional product information see: www.lantus.com. Glargine highlights:
   a. Should not be diluted or mixed as this will affect pH
   b. Should be kept refrigerated. Once open the vial has a shelf life of 4 weeks at room temperature. I would discard any remaining insulin after 8 weeks of refrigeration pending further clinical data.

2. **PZI:** As with dogs we only recommend the use of PZIR from BI.

3. **Humulin N and Novolin N:** Similar to PZI with remission rates of 40-50% when used with a low carbohydrate-high protein diet. Starting doses are generally 1-3 units/cat once a day.

4. **Vetsulin:** Again similar to PZI and Humulin N with remission rates of 40-50% when used with a low carbohydrate-high protein diet. Starting doses are generally 1-3 units/cat once a day.

**Transitioning feline patients**

If you have patients currently taking either Humulin L or Humulin U, I would switch them to either Vetsulin or Humulin N. The initial starting dose will remain the same with re-assessment of clinical signs and a serial blood glucose curve performed 1 week after changing insulin preparations. If you wish to transition them to glargine, I would follow the dosage recommendations as outlined above under newly diagnosed patients. It is important to note that remission rates will be much lower with glargine and a low carbohydrate-high protein diet in long standing diabetic patients (cats with diabetes for more than 6 months) than in newly diagnosed patients.

With the recent introduction of the AlphaTrak Blood Glucose Monitoring System (Abbott) we have the ability to very accurately measure blood glucose concentrations in both dogs and cats using very small quantities of blood. This will allow both veterinarians and pet owners to obtain very reliable results in both the hospital and home setting. This information can then be used to make informed decisions regarding the management of diabetic patients. These decisions impact the type and dose of insulin selected, the frequency of insulin administration, aid in the assessment of glycemic control, help in preventing hypoglycemic episodes and monitor for remission of diabetes especially in feline patients.

Glycemic control can be evaluated in a numbers of ways. Owner assessment of clinical signs (polyuria, polydipsia, weight gain or loss), progression of diabetic cataracts (dogs), presence of peripheral neuropathy (cats), and episodes of hypoglycemia are often the best indicators of glycemic control. Changes in insulin dosage or documenting remission of diabetes, is best determined by blood glucose measurement. Recognizing that the measurement of blood glucose concentrations can be problematic in the hospital setting (especially in cats as a result of stress induced hyperglycemia) recent work has evaluated the practicality and value of at home blood glucose monitoring in dogs and cats. At home blood glucose monitoring is essential in the management of human patients with diabetes given that a number of the complications associated with long term diabetes are directly related to persistent hyperglycemia. While diabetic retinopathy, nephropathy, painful neuropathies and cardiovascular disease are rare in our veterinary patients, adequate glycemic control is required to eliminate clinical signs and decrease morbidity and mortality in dogs and cats. Control of clinical signs does not require the restoration of euglycemia but rather involves keeping the blood glucose levels below renal threshold for the majority of the day. Renal threshold for glucose is 180 mg/dl in the dog and approximately 280 mg/dl in the cat. It is very important that we remember the owners of diabetic dogs and cats are being asked to do a great deal to help in the management of their pet’s chronic illness and we need to do whatever we can to make the clients job easier while at the same time taking steps to assure maximal diabetic control.

**Using the information derived using at home or in hospital glucose monitoring**

The data obtained with at home blood glucose monitoring in conjunction with clinical signs is used to adjust the dose of insulin and to monitor for remission of diabetes. We will look at scenarios for both cats and dogs. The recommendations for cats are based on our experience as well as the data generated by Dr Jacquie Rand at the University of Queensland.

**Cats**

1. **Cats on Glargine and PZI Insulins**
   a. If the preinsulin blood glucose concentration is > 360 mg/dl and/or the nadir blood glucose (PZI) or 4 hour (glargine) post blood glucose concentration is > 180 mg/dl the dose of insulin is increased by 0.5 to 1 unit BID.
b. If the preinsulin blood glucose concentration is 270 to 360 mg/dl and/or the nadir glucose (PZI) or 4 hour (glargine) post blood glucose blood glucose concentration is 90 - 180 mg/dl the dose of insulin is maintained.

c. If the preinsulin blood glucose concentration is 190 - 270 mg/dl and/or the nadir glucose (PZI) or 4 hour (glargine) post blood glucose blood glucose concentration is 54 - 90 mg/dl use the nadir, clinical signs and the next preinsulin glucose concentration to determine if the dose is decreased or maintained.

d. If the preinsulin blood glucose concentration is < 180 mg/dl and/or the nadir blood glucose (PZI) or 4 hour (glargine) post blood glucose glucose concentration is < 54 mg/dl the dose of insulin is decreased by 0.5 to 1 unit BID. If the total insulin dose is already 0.5 – 1 unit BID, stop the insulin and check for diabetic remission.

2. Cats on NPH, Lente or Ultralente Insulins
   a. If preinsulin blood glucose is < 210 mg/dl withhold insulin and check for diabetic remission.
   b. If preinsulin blood glucose is 234 - 288 mg/dl total insulin dose should not be higher than 1 unit BID.
   c. If nadir blood glucose is < 54 mg/dl insulin dose should be reduced by 50%.
   d. If nadir blood glucose is 54 - 90 mg/dl dose should be reduced by 1 unit BID.
   e. If nadir blood glucose is 91 - 162 mg/dl insulin dose should remain the same.
   f. If nadir blood glucose is > 180 mg/dl insulin dose should be increased by 1 unit BID.

**Diet**

There is a considerable amount of reliable research data showing that diets high in carbohydrates, low in fat and high in fiber are helpful in regulating diabetic dogs. These types of diets lower the average insulin dose, the average blood sugar, the amount of urine being produced and glycosolated hemoglobin and fructosamine levels.

The carbohydrates in these diets are complex carbohydrates. It is important to avoid diets high in simple sugars, which includes any commercial semi-moist food, primarily those packaged in foil packets. Diets high in simple sugars are absorbed very rapidly before the insulin has time to work. The goal with diet is to balance the absorption of sugar with the onset of action of the insulin. A high carbohydrate/low fat diets also decreases plasma free fatty acid and cholesterol concentrations, and increases the number and activity of insulin receptors.

High fiber diets reduce insulin resistance. The fiber acts to decrease post prandial hyperglycemia, primarily because it delays gastric emptying. A high fiber diet also decreases absorption of glucose and increases insulin action at the receptor.

It has recently been suggested that diabetic cats be fed a high protein/low carbohydrate diet. This can be accomplished with several commercially available canned diets (Hill’s M/D, IVD Development, Purina DM, many other canned kitten diets). These diets may result in remission of the diabetes and elimination of the need for exogenous insulin and/or oral hypoglycemic agents. High protein/low carbohydrate diets more closely resemble the diet of felines in the wild and may help reduce glucose intolerance, insulin resistance and obesity.

**Feeding**

Ideally, the feeding schedule should be coordinated with the onset of action of the insulin. With dogs, this is fairly easy to regulate, but with cats, it is nearly impossible due to their "grazing" style of eating. For cat owners who may not be able to follow a strict feeding schedule or those with multiple pet households, insulin therapy will have to be adjusted to meet the owner's needs. The most important component of the dietary plan is to stress consistency in the diet. The following feeding schedule can be used for dogs and some cats. With insulin given once a day, feed three meals a day (of equal calories) at six-hour internals. Give the first meal at the time of the insulin injection. For animals receiving insulin twice a day, feed four meals a day. Schedule them to coincide with the insulin injections and feed mid-afternoon and late evening.

If the owner is unable to follow this schedule, advise them to feed twice a day, at the time of injection and 8-10 hours later (for once a day insulin patients); or at the times of insulin injections (for twice a day insulin patients).

**Home management**

1. Instruct owner on proper injection techniques, injection locations, storage and handling of insulin.
2. Instruct owner on how to monitor clinical signs.
3. Continue feeding schedule and dietary therapy.
4. Instruct owners to initially monitor urine glucose/ketone levels daily, usually in the morning or evening prior to feeding. If persistent glycosuria or ketonuria is observed, ask owner to contact the veterinary hospital.
5. Advise owners of the signs of and treatment for hypoglycemia. Have owners keep a bottle of Karo syrup on hand if signs occur (i.e., weakness, ataxia, seizures) so they can rub syrup on the gums immediately. Instruct them to call the veterinary hospital.
6. Home monitoring of a diabetic cat is frequently based on observance of clinical signs only.
7. Serial sugars after the first week of home management.

**Re-check evaluations**

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1. Obtain owner assessment of clinical signs.
2. Serial blood sugars are helpful due to:
   a. Variability of insulin action in a given patient.
   b. Inaccuracy of random blood or urine sugars in monitoring the degree of glycemic control.
   c. Not particularly helpful as a routine procedure in animals that are well controlled clinically.
3. Body weight
4. Physical examination/ophthalmic exam
5. Discuss urine log book with owner
6. Laboratory work as clinically indicated
7. Role of glycosylated hemoglobin and fructosamine:
8. Fructosamine may be helpful in distinguishing stress-induced hyperglycemia from diabetes in cats. These tests can be used every 3 – 4 months as an indicator of long term (2-3 weeks fructosamine; 4-6 weeks glycosylated hemoglobin) glucose control. Rising values indicate the need for further evaluation.

**Problems with insulin therapy**

1. Insulin induced hyperglycemia (Somogyi phenomenon)
   a. Hypoglycemia (<65mg/dl) followed by hyperglycemia (>300mg/dl) within 24 hours of insulin injection.
   b. Suspect when insulin requirements exceed 2 U/kg and clinical signs persist.
   c. Suspect when animal has signs of hypoglycemia in afternoon.
   d. Diagnosis with serial sugars.
   e. Treat by decreasing insulin dose 25-50% and review insulin administration with the owner to rule out management problems.
   f. Re-check serial sugars in one week.
2. Rapid insulin metabolism
   a. Duration of insulin less than 18 hours.
   b. Signs return in the evening.
   c. Diagnosis is with serial sugars. Hyperglycemia (>250) within 18 hours of insulin injection without previous hypoglycemia.
   d. Treatment:
     e. -Review management with owner
     f. -Switch to twice daily insulin administration. Most dogs and cats require insulin twice a day to achieve adequate glycemic control. Consider switching to PZI in cats.
3. Insulin Resistance
   a. Hyperglycemia (>300) throughout the day, despite insulin dosages > 2 U/kg.
   b. Diagnosis based on serial sugars.
   c. Potential causes of insulin resistance:
   d. Management problems
   e. Hyperadrenocoticism
   f. Steroid or Ovaban administration
   g. Diestrus or pregnancy
   h. Acromegaly
   i. Concurrent illness, infection
   j. Anti-insulin antibodies
   k. Hypothyroidism (dogs), hyperthyroidism (cats)
   l. If insulin dose exceeds 2U/kg, the animal should be evaluated for one of these causes of resistance.
4. Hypoglycemia
   a. Insulin overdose
   b. Suspect if animal shows weakness, shaking, ataxia, seizures at time of insulin's peak effect.
   c. Therapy (instructions for owners)
   d. Mild signs - give food and call veterinarian
   e. Moderate signs - apply Karo syrup to the mouth, offer food when alert and then notify veterinarian.
   f. Comatose - apply Karo syrup to mouth and take animal to hospital.
   g. When hypoglycemia occurs, serial sugars should be performed to re-assess insulin dose
Insulin resistance is a condition in which a normal amount of insulin produces a suboptimal biologic response. Insulin resistance may result from problems occurring before the interaction of insulin with its receptor (e.g., insulin-binding antibodies), at the receptor (e.g., altered insulin receptor binding affinity or concentration), or at steps distal to the interaction of insulin and its receptor. Post-receptor problems are difficult to differentiate clinically from receptor problems, and both often coexist. In dogs and cats, receptor and post-receptor abnormalities are usually attributable to obesity, inflammation (such as occurs with pancreatitis or gingivitis), a disorder causing excessive secretion of a potentially insulin-antagonistic hormone (such as cortisol in dogs and cats or growth hormone and T4 in cats), or a disorder that causes a deficiency of hormone necessary for insulin action (such as thyroid hormone).

No insulin dose clearly defines insulin resistance. For most diabetic dogs and cats, control of glycemia can usually be attained using 1.0 U or less of NPH, lente insulin or glargine (cats) per kilogram of body weight given twice daily. Insulin resistance should be suspected if control of glycemia is poor despite an insulin dosage in excess of 1.5 U/kg, when excessive amounts of insulin (i.e., insulin dosage >1.5 U/kg) are necessary to maintain the blood glucose concentration below 300 mg/dL, or when control of glycemia is erratic and insulin requirements are constantly changing in an attempt to maintain control of glycemia. Failure of the blood glucose concentration to decrease below 300 mg/dL during a serial blood glucose curve is suggestive of but not definitive for the presence of insulin resistance. An insulin resistance-type blood glucose curve can also result from stress-induced hyperglycemia (cats), the Somogyi response, and other problems with insulin therapy, and a decrease in the blood glucose concentration below 300 mg/dL can occur with disorders causing relatively mild insulin resistance. Serum fructosamine concentrations are typically greater than 500 µmol/L in animals with insulin resistance and can exceed 700 µmol/L if resistance is severe.

Two diseases that have the potential to cause the most severe insulin resistance are hyperadrenocorticism and hypersomatotropism (acromegaly), although insulin resistance may also be mild or variable. Approximately 80% of cats with hyperadrenocorticism and nearly all cats with hypersomatotropism will develop diabetes mellitus. Hyperadrenocorticism is rare: 75% to 80% of cats have pituitary-dependent disease and 20% to 25% have cortisol secreting adrenocortical tumors. In rare circumstances, adrenocortical tumors secrete other steroid hormones (e.g., progesterone). However, clinical signs are identical to those of hypercortisolism, and diabetes mellitus may develop as well. In addition to PU/PD and weight loss, which are usually due to concurrent diabetes mellitus, typical clinical signs are abdominal enlargement, an unkempt seborrheic hair coat, thinning of the hair coat, failure of hair to regrow, or alopecia and muscle weakness. Severe cases may have thin, fragile skin that tears easily. Cats with large pituitary masses may have CNS disturbances. However, clinical signs may also be mild and hyperadrenocorticism is often not suspected until it becomes evident that the diabetes is difficult to regulate. The dexamethasone suppression test is the preferred screening test. Whether poorly regulated diabetics do indeed have hyperactivity of the hypothalamus-pituitary-adrenal gland axis that leads to abnormal test results is controversial. Based on recent studies, the dexamethasone test (0.1 mg/kg dexamethasone IV with a pre, 4 and 8 hour post) appears to be a suitable part of the diagnostic workup in diabetic cats suspected of having hyperadrenocorticism and should be carried out only after insulin therapy has been instituted for 6-8 weeks to mitigate the effects of poor glycemic control on the HPA axis.

Hypersomatotropism in cats is caused by a growth hormone (GH-)producing tumor (usually an adenoma) in the pars distalis of the pituitary gland. GH has catabolic and anabolic effects; the latter are in part mediated by insulin-likemrowth factor-1 (IGF-1). The catabolic effects are mainly due to insulin antagonism and are the reason for the diabetes mellitus. The anabolic effects include proliferation of bone, cartilage, soft tissue, and organs resulting in a large body size, broad head and large paws, weight gain, prognathia inferior, respiratory difficulties because of thickening of pharyngeal tissues, degenerative arthropathy, and organomegaly with potential organ dysfunction. Growth of the tumor may lead to signs of CNS disease. As previously mentioned for hyperadrenocorticism, clinical signs may also be very subtle or even absent. Acromegaly has long been considered a rare disorder. However, it was recently suggested that acromegaly occurs more frequently than previously thought and is most likely underdiagnosed. Because the availability of a validated GH assay for cats is inconsistent, diagnosis is usually based on the finding of high IGF-1 concentration. Two important points should be kept in mind. First, circulating IGF-1 is bound to proteins, which must be removed before measurement. Not all assay methods are equally effective, and intra assay inference of binding proteins may lead to false high IGF-1 levels. Therefore, only assays validated for the cat should be used. Second, IGF-1 concentrations are often low in newly diagnosed diabetic cats and increase markedly after initiating insulin therapy. Low IGF-1 levels have also been seen initially in untreated diabetic cats with acromegaly. This observation is explained by the fact that relatively high insulin concentrations are required in the portal vein for the expression and function of GH receptors on hepatocytes, and this mechanism is impaired in insulin-deficient states. IGF-1 is therefore measured 6 to 8 weeks after initiating insulin therapy.
Problems with insulin therapy

- Inactive insulin
- Improper insulin syringe
- Diluted insulin
- Improper administration technique
- Inadequate dose
- Somogyi response
- Inadequate frequency of insulin administration
- Impaired insulin absorption
- Anti-insulin antibody formation (rare)

Caused by concurrent disorder

- Diabetogenic drugs
- Hyperadrenocorticism
- Diestrus (intact female dogs)
- Infection, especially of skin, oral cavity and urinary tract
- Chronic inflammation, especially pancreatitis and oral cavity
- Severe obesity
- Hyperlipidemia
- Hypothyroidism
- Hyperthyroidism (cat)
- Acromegaly (cat)
- Renal insufficiency
- Liver insufficiency
- Cardiac insufficiency
- Pancreatic exocrine insufficiency
- Neoplasia
- Glucagonoma
- Pheochromocytoma

Many disorders can interfere with the effectiveness of insulin therapy. The most common disorders causing insulin resistance in dogs include severe obesity, use of diabetogenic drugs (glucocorticoids), hyperadrenocorticism, diestrus, chronic pancreatitis, renal insufficiency, oral and urinary tract infections, hyperlipidemia, and antiinsulin antibodies in dogs receiving beef source insulin. Obtaining a complete history and a thorough physical examination are the most important steps in identifying these concurrent disorders. Abnormalities identified on the physical examination may suggest a concurrent insulin-antagonistic disorder or infectious process, which will give the clinician direction in the diagnostic evaluation of the dog. If the history and physical examination are unremarkable, a CBC, serum biochemical analysis, serum progesterone concentration (intact female dog), abdominal ultrasound, and urinalysis with bacterial culture should be obtained to further screen for concurrent illness. Additional tests will be dependent on results of the initial screening tests.

Diagnostic tests to consider for the evaluation of insulin resistance in diabetic dogs and cats

- Complete blood count, serum chemistry profile, UA and UMIC
- cPLI (pancreatitis)
- TLI (if suspect EPI)
- Adrenal Function Testing
  - Dexamathson suppression test (cats)
- ACTH stimulation (likely less affected by concurrent diabetes in dogs)
  - Thyroid Function Testing
  - TT4
- fT4 (if TT4 is less than 1.5 ug/dl in a dog or between 2.5 – 4.0 ug/dl in a cat)
- Serum progesterone levels (diestrus in dogs)
- Serum IGF-1 concentrations (cats with suspected acromegaly)
- Fasting triglycerides and cholesterol
- Abdominal ultrasonography (pancreatitis, neoplasia, adrenal masses or enlargement)
- Thoracic radiographs (cardiopulmonary disease, neoplasia)
- MRI (if document PDH or acromegaly)
Hyperthyroidism is recognized as the most common endocrinopathy of older cats. Despite worldwide occurrence, the pathogenesis of feline hyperthyroidism remains unclear. Traditional methods of managing feline hyperthyroidism include thyroidectomy, anti-thyroid medications, and radioactive iodine. Recent studies document that another option now exists for hyperthyroid cats; feeding a limited-iodine food normalizes thyroid hormone concentrations and alleviates clinical signs of hyperthyroidism. Surgery and radioactive iodine are designed to provide permanent solutions, whereas, oral anti-thyroid drugs and nutritional management control hyperthyroidism and are needed daily to achieve/maintain their effect. All management options are effective and each has its pros and cons. It’s important to discuss all options with pet owners so the appropriate management can be selected for each hyperthyroid cat.

Diagnosis
Diagnosis most often is based on the presence of one or more typical clinical signs and increased serum total thyroxine (T4) concentration. However, up to 10% of all hyperthyroid cats and 40% of those with mild disease have serum T4 values within reference range. In these cases, serum free T4 (fT4), measured by equilibrium dialysis, may provide an alternative means of diagnosing hyperthyroidism in cats with normal serum total T4 values. Studies document that up to 20% of sick euthyroid cats can have increased fT4 concentration. Therefore, it is most appropriate and reliable to interpret the two values together. Mid-to-high reference range total T4 and increased fT4 concentrations are consistent with hyperthyroidism. In contrast, low total T4 and increased fT4 values are usually associated with non-thyroidal illness.

Management options
Once hyperthyroidism has been diagnosed, all management options (thyroidectomy, radioactive iodine, anti-thyroid drugs, nutritional management) should be discussed with pet owners. All options can be ≥90% effective for controlling hyperthyroidism when used appropriately. The selected management option will differ for each cat based on several considerations. Radioactive iodine therapy is considered the gold standard for treatment of hyperthyroidism; however, most pet owners currently opt for medical management. Until recently, this included oral or transdermal anti-thyroid drugs. Now nutritional management using a limited-iodine food is another option for cats with hyperthyroidism.

Radioactive iodine
Radioiodine treatment is often considered the best option for many hyperthyroid cats because:
1. It has the potential to eliminate a benign thyroid tumor or abnormal thyroid tissue with a single treatment
2. It treats extra-thyroidal thyroid tissue, which may occur in 10 to 20% of hyperthyroid cats
3. No general anesthesia is required
4. Reported side effects are minimal

Cats should be stable prior to radioiodine therapy; those with clinically significant cardiovascular, renal, gastrointestinal, or endocrine (e.g., diabetes mellitus) disease may not be very good candidates, especially because of the time necessary for boarding after treatment.

After administration, radioactive iodine is actively concentrated by the thyroid gland and has a half-life of 8 days. It emits both β-particles and γ-radiation; the β-particles are responsible for the majority of tissue destruction, but are only locally destructive, traveling a maximum of 2 mm. Therefore, no significant damage to adjacent parathyroid tissue, atrophic thyroid tissue, or other cervical structures is expected. The main limitation to widespread use of radioactive iodine is the requirement for special licensing and the isolation of the cat for variable periods after treatment. This can range from several days to several weeks depending on state or local radiation regulations and the dose administered.

The goal of treatment is to restore euthyroidism with the smallest possible single dose of radioactive iodine, while avoiding development of hypothyroidism. Controversy exists as to the best method of calculating the optimum dose for individual cats. Based on the majority of reported cases, post-treatment hypothyroidism is transient and generally uncommon (2 to 7% of cases); even fewer cats have clinical signs or appear to require thyroid hormone replacement. However, up to 30% (50 of 165 cats) were hypothyroid 3 months after radioiodine therapy in one study; of these, 56% (19 of 34 hypothyroid cats with available information) had clinical signs of hypothyroidism and 52% (23 of 44 cats) were given thyroid hormone supplementation. Thyroid hormone replacement may be needed in some cats, especially those with concurrent kidney disease, since hypothyroidism has been associated with azotemia and decreased survival time in previously hyperthyroid cats. Owners should be advised of this possibility, particularly if their motivation is to avoid long-term oral medication.
Anti-thyroid drugs

Anti-thyroid drugs (e.g., methimazole, carbimazole) are commonly used for treatment of hyperthyroidism in cats. If administered appropriately, they reliably inhibit the synthesis of thyroid hormones and thereby lower serum thyroid hormone concentrations. These drugs do not affect the thyroid gland’s ability to trap inorganic iodide or release preformed hormones. They are widely recommended to stabilize hyperthyroid cats prior to surgery and are the only drugs that can be used chronically for management of hyperthyroidism. Almost all cats are potential candidates unless thyroid carcinoma is suspected.

Anti-thyroid drugs used most often in cats include methimazole and carbimazole; both can be given orally or formulated for transdermal application. Custom formulation of transdermal products may increase expense of therapy and stability of the product is not guaranteed. Results of a recent prospective study conducted in New Zealand showed that once daily treatment for 12 weeks with transdermal methimazole in a novel lipophilic vehicle was as effective as twice-daily carbimazole administered orally.

While many cats have been successfully managed long-term with anti-thyroid drugs, it’s important to monitor for potential side effects that have been associated with their use. In the study with the largest number of cats, 18% had side effects associated with methimazole; a more recent study revealed that 44% of 39 cats had side effects. In 44 cats receiving carbimazole for 1 year, 44% had associated side effects with gastrointestinal signs (decreased appetite, vomiting, diarrhea) being most common. In another study, 13% of 39 cats treated with carbimazole experienced side effects. It’s difficult to determine what % of side effects are caused by the drug versus something else such as concurrent disease.

Most adverse reactions occur within the first few weeks to months after beginning therapy and include depression, inappetence, vomiting, and self-induced excoriation of the head and neck (facial pruritus). Gastrointestinal signs are less common with transdermal administration of methimazole. Mild to serious hematological complications, including agranulocytosis and thrombocytopenia either alone or concurrently, and more rarely immune-mediated hemolytic anemia may also occur. Hepatic toxicity with marked increases in bilirubin concentration and hepatic enzyme activities has been described in less than 2% of cats treated with methimazole. Cessation of therapy is required if either serious hematologic or hepatic reactions develop. Serum antinuclear antibodies develop in approximately 50% of cats treated with methimazole for longer than 6 months, usually in cats on high-dose therapy (> 15 mg/day). Although clinical signs of a lupus-like syndrome have not been reported, decreasing the daily dosage is recommended.

Nutritional management

Production of thyroid hormone requires uptake by the thyroid gland of sufficient amounts of iodine, which is provided by dietary intake. The only function for ingested iodine is for thyroid hormone synthesis. This observation led to the hypothesis that limiting dietary iodine intake could be used to control thyroid hormone production and potentially manage hyperthyroidism in cats. After more than a decade of research and development, a limited-iodine therapeutic food (Hill’s® Prescription Diet® y/d Feline) containing ≤ 0.3 ppm (mg/kg) iodine on a dry matter basis (DMB), is now available as an option for managing cats with hyperthyroidism.

Iodine content of commercial cat foods

Iodine occurs naturally in many ingredients typically used in the manufacture of commercial pet foods (particularly fish, shellfish and fresh meats) and unless steps are taken to strictly control the iodine content of ingredients, the final iodine concentration in pet foods varies widely. Commercial cat foods in New Zealand had iodine amounts ranging from 0.19 to 21.2 ppm in one study whereas in Germany a range of 0.22 to 6.4 ppm was reported. Evaluation of 28 canned cat foods in the US revealed an iodine content ranging from 1.09 to 52.3 ppm (mean = 7.83) and 14 dry cat foods contained iodine amounts ranging from 1.34 to 5.94 ppm (mean = 2.77). Based on these studies, the amount of iodine is much higher in many canned foods compared with dry foods and variability of iodine content is much greater in canned food.

Multiple feeding trials have been conducted in a research colony using over 100 cats with naturally occurring hyperthyroidism to determine the safety and effectiveness of limited dietary iodine in the management of the disease. The results of all studies support that a therapeutic food with dietary iodine ≤ 0.3 ppm iodine (dry matter basis) provides a safe and effective management option for cats with naturally occurring hyperthyroidism. Serum total thyroxine concentrations return to the normal range within 4 to 12 weeks of initiating nutritional management and 90% hyperthyroid cats maintained on the limited-iodine food as the sole source of nutrition become euthyroid.

Three studies were designed to determine the magnitude of iodine control necessary to return newly diagnosed cats to a euthyroid state, the maximum level of dietary iodine that maintains cats in a euthyroid state, and the effectiveness of a therapeutic food formulated based on the previous studies to control naturally occurring hyperthyroidism in cats. In summary, results of these studies demonstrated that a food with 0.17 or 0.32 ppm iodine (DMB) maintained normal thyroid hormone concentrations in hyperthyroid cats, helping to further define the range of iodine effective for managing hyperthyroidism.

We have treated 22 cats to date with feline y/d with follow-up data for at least 6 months. All of the cats found at least one form of the diet (dry or canned) to be palatable. Nineteen of 22 (86%) cats experienced clinical improvement with normalization of their TT4 concentrations. Of the three cats that failed to achieve remission, 2 cats were discovered to be eating foods other than y/d and when
the owners switched them to y/d exclusively remission of hyperthyroidism was achieved. One cat (5%) failed to respond to dietary therapy and was subsequently treated with 131-I.

We are currently conducting a prospective study evaluating the efficacy of feline y/d in managing feline hyperthyroidism to include monitoring of thyroid function (TT4, fT4ED, TSH), clinical signs, body weight, renal function and blood pressure pre and post-treatment. The study should be completed in 2015.

Newly diagnosed patients
After confirming the diagnosis and performing a thorough patient evaluation, nutritional management should be discussed along with other options for managing hyperthyroidism. If selected as the management option, gradual transition to the limited-iodine food (Hill’s® Prescription Diet® y/d Feline) over at least 7 days is recommended. It is very important to counsel owners so they understand that success of nutritional management depends on the limited-iodine food being the sole source of nutrition for their cat.

The first recheck evaluation should be done 4 weeks after completing the transition to y/d Feline (i.e., once the cat has eaten y/d exclusively for 4 weeks) and as a minimum should include physical examination and measurement of T4, BUN, serum creatinine, and urine specific gravity. All cats should have decreased T4 concentrations compared with baseline and many will have returned to normal by the 4-week evaluation. Clinical improvement including weight gain, improved hair coat and decreased tachycardia/cardiac murmur also may be noted by the first evaluation. Clinical signs should continue improving by the next re-evaluation at 8 weeks and most cats will be euthyroid. Some cats require slightly longer to become euthyroid; however, it’s expected that 90% will have normal T4 concentrations if the limited-iodine food is their sole source of nutrition.

If euthyroidism is not achieved within 4 to 12 weeks, a thorough history is indicated to confirm that only the limited-iodine food is being fed.

Managing hyperthyroid cats with concurrent kidney disease
Chronic kidney disease (CKD) and hyperthyroidism are more likely to be diagnosed in older cats so it’s not surprising that many hyperthyroid cats have CKD. Untreated hyperthyroidism complicates the diagnosis of CKD because it’s associated with increased glomerular filtration rate (GFR) and therefore often masks biochemical markers of CKD. Regardless of the therapeutic modality (methimazole, surgical thyroidectomy, or radioiodine), decreased GFR, increased serum urea and creatinine concentrations and development of overt clinical signs of kidney disease have been reported after successful treatment of hyperthyroidism. The presence of underlying CKD may affect the prognosis - one study documented a shorter survival time in hyperthyroid cats with azotemia. However, two recent studies comparing survival of cats that developed azotemia with those that did not after treatment of hyperthyroidism found no significant difference between the two groups if cats did not become hypothyroid post-treatment.

The reported occurrence of azotemia after treatment of hyperthyroidism ranges from 15 to 49%. Iatrogenic hypothyroidism has been reported to decrease GFR in human patients. Post-treatment iatrogenic hypothyroidism has been reported in cats after radioiodine therapy and bilateral thyroidectomy, which constituted the predominant therapeutic modalities in previous studies. In one recent study, cats with iatrogenic biochemical hypothyroidism were almost twice as likely to develop azotemia post-treatment as euthyroid cats. The hypothyroid cats with azotemia had shorter survival times than cats without azotemia, whereas, consistent with previous reports, there was no difference in survival times of euthyroid cats with or without azotemia.

It’s not possible to consistently predict which cats will develop overt CKD after treatment of hyperthyroidism or have progression of their kidney disease. This should be considered when deciding on treatment options, particularly those that are irreversible (thyroidectomy, radioactive iodine). Regardless of the option selected for managing hyperthyroidism, it’s important to remember that the only intervention shown to improve quality of life and prolong survival time in cats with naturally occurring CKD is feeding a therapeutic renal food. Until recent availability of limited-iodine food, nutritional recommendations have not generally been considered for hyperthyroid cats without azotemia. In cats with compromised renal function, but without azotemia (IRIS Stage 1), the decrease in GFR associated with normalizing serum T4 levels may be sufficient to prevent effective clearing of protein metabolic by-products (BUN and creatinine) when dietary intake of protein and phosphorus is high. This could contribute to the occurrence of post-therapy azotemia in hyperthyroid cats.

In our work with 22 cats with hyperthyroidism treated with feline y/d, 4/22 cats (18%) were azotemic (IRIS Stage 1 and 2 CKD) prior to starting the diet. All 4 cats experienced normalization of their BUN and creatinine within 30-150 days along with normalization of their TT4’s. One potential explanation is that the expected decrease in GFR associated with normalizing serum T4 may be offset by the nutrient profile of the limited-iodine food which is similar foods for mature adult cats or cats with early CKD. Additional study is needed to better understand the effects of using limited-iodine food on hyperthyroid cats with concurrent kidney disease.

Conclusions/summary
Hyperthyroidism is the most common endocrine disease of older cats worldwide. While the pathogenesis is unclear, several effective management options are available. All should be discussed with pet owners, including pros/cons, so that the best option can be
selected for individual patients and their owners. Feeding a limited-iodine food is now available as an option for effective management of hyperthyroid patients. When fed as the sole source of nutrition, approximately 90% of hyperthyroid cats become euthyroid within 4 to 12 weeks. To date, over 150 cats with naturally occurring hyperthyroidism have been managed successfully by feeding a limited-iodine food, most for 2-3 years and some cats for as long as 6 years.
Solving the Puzzles of Puddles: PU/PD
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Introduction
A. Polyuria and polydipsia (PU / PD) refer to excessive water consumption and urine production respectively. These are common clinical signs in both dogs and cats.

B. Water consumption exceeding 100 ml/kg or urine production exceeding 50 ml/kg body weight per day is considered abnormal and should be pursued. These numbers have been established in laboratory reared dogs and may not reflect "normal" water consumption in pets. They are to be used only as guidelines.

C. Water consumption can vary greatly from day to day so it is important to have owners subjectively assess water consumption in the home environment for several consecutive days in order to obtain an accurate picture before beginning unnecessary and expensive diagnostic tests. Actual quantification of water consumption can be very difficult and may not be practical for the majority of pet owners.

Normal water homeostasis
A. Extracellular fluid volume is maintained by regulation of fluid intake and urine production.

B. The thirst center is stimulated by an increase in plasma osmolality (sodium concentration) and/or a decrease in blood volume (hypovolemia) resulting in an increase in water consumption.

C. Increasing plasma osmolality and hypovolemia also stimulate osmoreceptors in the anterior hypothalamus and baroreceptors in the aortic arch resulting in the release of antidiuretic hormone (ADH) from the anterior pituitary.

D. ADH circulates and binds to receptors on the renal tubular cells of the distal tubules and collecting ducts resulting in the production of cAMP. This causes the opening of pores in the luminal membrane of the tubular cells and allows for reabsorption of water from the glomerular filtrate resulting in a concentrated urine. In order for water to be pulled out of the tubule it must move along a concentration gradient maintained by the hypertonic renal medullary interstitium. Loss of this gradient (medullary washout), will result in an inability to concentrate urine even in the face of normal ADH activity. Urea and sodium are largely responsible for maintaining the hypertonicity of the interstitium.

E. The sensation of thirst and secretion of ADH are suppressed when plasma osmolality and blood volume are returned to normal.

Differential diagnosis: Mechanisms of PU/PD
A. Renal disease:
   a. Chronic renal failure: A decrease in the number of functional nephrons causes an increase in tubular flow in the remaining nephrons and leads to a solute diuresis. A decrease in urine concentrating ability may be the only laboratory abnormality indicating renal disease (especially in feline patients) presented for PU/PD.
   b. Pyelonephritis: Bacterial induced tubular destruction and an increase in renal blood flow cause a decrease in medullary hypertonicity.
   c. Primary renal glycosuria (Fanconi's Syndrome): A proximal tubular defect results in renal glycosuria leading to an osmotic diuresis. The blood glucose is normal.
   d. Post-Obstructive diuresis: May be seen in previously blocked cats. Due to osmotic diuresis from loss of large amounts of sodium and urea into the urine following relief of urethral obstruction.

B. Diabetes mellitus:
   a. Hyperglycemia results in glycosuria and an osmotic diuresis. Threshold for renal glycosuria is a blood glucose of 180 – 220 mg/dl (dog) and 240 – 300 mg/dl (cat).

C. Liver disease:
   a. PU/PD may occur as the result of: (1) decreased production of urea which is a major component of the hypertonic medullary interstitium, (2) increased renin and cortisol levels due to a lack of hepatic degradation, (3) increased aldosterone concentration leading to increased sodium concentration, and (4) hypokalemia (see hypokalemic nephropathy).

D. Hyperthyroidism:
   a. Increased total renal blood flow reducing the toxicity of the medullary interstitium.
   b. Psychogenic polydipsia or primary polydipsia is reported in humans with hyperthyroidism.

E. Hypercalcemia:
a. Interference with cAMP activation by ADH, damage to ADH receptors, and mineralization of renal tubular cells.

F. Hyperadrenocorticism:
   a. Glucocorticoids interfere with the action of ADH at the renal tubule and decrease
   b. ADH secretion by reducing osmoreceptor sensitivity to rising plasma osmolality.

G. Hypoadrenocorticism:
   a. Renal sodium wasting leads to decreased medullary hypertonicity.

H. Pyometra:
   a. coli endotoxins interfere with sodium reabsorption and damage ADH receptors and may result in an immune-complex glomerulonephritis.

I. Hypokalemia:
   a. Degeneration of renal tubular cells, (2) decreased medullary hypertonicity, stimulation of thirst, and (4) stimulation of renin release.

J. Polycythemia:
   a. Mechanism unknown; may be related to sluggish blood flow in kidney or hypothalamus.

K. Medications:
   a. Exogenous steroids, diuretics, salt supplementation, primidone, phenobarbital, KBr and vitamin D.

L. Pituitary or central diabetes insipidus (CDI):
   a. Due to inadequate production, storage or release of ADH. May occur as a congenital defect or secondary to trauma, mass lesions, infection or infarction of the pituitary or hypothalamus.

M. Nephrogenic diabetes insipidus (NDI):
   a. Congenital structural or functional defects in ADH receptor. Rare in dogs and cats.

N. Primary polydipsia or psychogenic polydipsia:
   a. Underlying cause unknown (possible CNS lesion); results in increased renal blood flow and a decrease in medullary hypertonicity. Extremely uncommon in dogs and cats and is largely a diagnosis of exclusion.

**Diagnostic approach to PU / PD**

A. Document PU/PD actually exists. Recommend assessment of water consumption in the home environment. Hospitalized animals frequently do not drink as much as they would in their natural surroundings.

B. Quick evaluation of urine specific gravity and glucose is cheap, easy, and very helpful in evaluating animals for possible pathologic PU/PD. If the urine specific gravity of a non-glycosuric sample, obtained from a dog or cat without signs of dehydration, is greater than 1.030 (dog) or 1.035 (cat), the likelihood of pathologic PU/PD is small and further work-up may not be required.

C. Most causes of PU/PD will be identified following a good history, physical examination, and an initial data base consisting of a CBC, chemistry profile, and urinalysis with bacteriologic culture.

D. If a cause has not been discovered after step C, the most likely diagnoses are hyperadrenocorticism (dog only, cats with Cushings's are usually overtly diabetic), central and nephrogenic diabetes insipidus, and primary polydipsia. As hyperadrenocorticism is far more common than either of the other causes, an ACTH stimulation test, urine cortisol/creatinine ratio or low-dose dexamethasone suppression test should be performed before proceeding to the modified water deprivation test (See Canine Hyperadrenocorticism).

**Modified water deprivation test (MWDT)**

A. This test is designed to help differentiate CDI, NDI, and primary polydipsia. It is not very helpful unless other causes of PU/PD have been ruled out.

B. The test is designed to determine whether ADH is released in response to dehydration and whether the kidneys can respond to the circulating ADH.

C. VERY IMPORTANT !! THE TEST SHOULD NEVER BE PERFORMED ON AN ANIMAL WITH PRE-EXISTING AZOTEMIA OR OBVIOUS DEHYDRATION. DOING SO IN ANIMALS WITH RENAL INSUFFICIENCY MAY RESULT IN DECOMPENSATION AND THE DEVELOPMENT OF OligURIC RENAL FAILURE OR ANURIC RENAL FAILURE.

D. Severe dehydration can occur very rapidly (4-6 hours) especially in animals with diabetes insipidus. Leaving them unattended without water for several hours or overnight may result in severe hyperosmolality, coma, and death.

E. Gradual water restriction should be instituted at home for 2-3 days prior to performing the MWDT in order to help minimize medullary washout from long-standing PU/PD.
Phase one

1. Animal is weighed, bladder emptied and urine saved for specific gravity and osmolality (if available).
2. Blood is obtained for BUN and osmolality.
3. Water is withheld. BUN, plasma osmolality and body weight are obtained hourly. The bladder is emptied every hour and a sample is saved for specific gravity and osmolality.
4. Test concluded with either a 5% loss in body weight, azotemia (BUN > 30), or urine specific gravity > 1.030 (1.035 cats). The bladder is emptied and urine is saved for specific gravity and osmolality, and plasma is obtained for osmolality.

Phase two

1. Aqueous vasopressin (Pitressin) 2 - 3 units (dog) or 0.25 U/# (cat) is given SQ. Alternatively DDAVP may administered into the conjunctival sac (1 – 2 drops for dogs and 1 drop for cats).
2. Urine and plasma osmolality and urine specific gravity are obtained every 30 min for 90 minutes.
3. Bladder must be emptied at every 30 minute sampling period.
4. Water is withheld throughout the test.

Interpretation of the MWDT

A. Normal Animals: Following water deprivation will concentrate urine to > 1.030 (dog) or 1.035 (cat). Urine osmolality in excess of 1,200 mOsm/kg.
B. CDI: Unable to concentrate urine in excess of 1.008 (< 300 mOsm/kg). After ADH administration, urine specific gravity should increase to greater than 1.012 with a 50 - 500 % increase in urine osmolality.
C. NDI: Similar to CDI following water deprivation. No further response following ADH injection.
D. Partial CDI: Results depend on how much ADH is available. Following water deprivation urine specific gravity between 1.008-1.019 and urine osmolality between 300 to 1,000 mOsm/kg. Urine specific gravity and osmolality increase after ADH administration. Similar response seen with hyperadrenocorticism and a number of the other causes of PU/PD. This is why it is important to rule-out these processes prior to a MWDT.
E. Primary polydipsia: Depends on degree of medullary washout. With minimal washout results are similar to normal animals. More severe washout gives results similar to partial diabetes insipidus.

Treatment of polyuria and polydipsia

A. Treat the underlying disorder!
B. Treatment of CDI
   a. DDAVP (Desmopressin acetate) 1-2 drops into the conjunctival sac or 0.01 to 0.05 mls subcutaneously SID or BID. May also dose orally with 0.1 to 0.2 mg once or twice a day.
      i. 1 drop = 1.5 to 4.0 ug. Can use TB syringe to dose.
      ii. Duration 8 - 24 hours.
      iii. Redosed when polyuria returns.
      iv. Most commonly used treatment today.
      v. Use the intranasal preparation.
   b. Chlorpropamide (Diabenese)
      i. Oral hypoglycemic. Stimulates ADH release and potentiates ADH action. Hypoglycemia is the limiting factor.
      ii. 25 - 40 mg once or twice a day (cat). Limited experience.
C. Treatment of NDI
   a. Salt restriction
   b. Thiazide diuretics:
      i. Natriuresis results in a decrease in blood volume and increased sodium reabsorption in the proximal tubule.
      ii. Hydrochlorothiazide 12.5 - 25 mg once or twice a day (cat).
      iii. Chlorthiazide 20 - 40 mg/kg BID (dogs).
      iv. May also help with partial CDI.
D. Treatment of Primary Polydipsia
   a. Treatment to restore hypertonic renal medullary interstitium.
   b. Gradual water restriction over several days.
   c. Behavioral modification or referral to a behaviorist may be needed.
1. Introduction
   - Cushing's syndrome refers to all causes of hyperadrenocorticism with overproduction of cortisol.
     - ACTH-dependent
       - Cushing's disease: Pituitary hypersecretion of ACTH which results in bilateral adrenal hyperplasia (90% of cases)
       - Ectopic ACTH production: Non-pituitary tumors secreting ACTH resulting in bilateral adrenal hyperplasia. Has not been completely documented in dogs or cats.
     - ACTH independent
       - Adrenocortical adenoma or carcinoma: Hypersecretion of cortisol with atrophy of normal adrenal and suppressed ACTH concentrations (10% of cases).
     - Iatrogenic
       - Excessive or prolonged administration of glucocorticoids. Clinically indistinguishable from natural disease. Results in adrenal atrophy and suppressed ACTH levels.

2. Signalment
   - Poodles, Dachshunds, Schnauzers, Boston Terriers, Boxers.
   - Middle to old age. Average 12 years; range 6 months to 17 years.
   - No sex predilection.
   - Rare in cats. Usually seen with insulin resistant diabetes mellitus and/or cats with severe dermal atrophy/ulceration.

3. Clinical signs
   - PU / PD
   - Pendulous, "pot-bellied abdomen": Due to muscle catabolism by glucocorticoids and hepatomegaly.
   - Bilaterally symmetric alopecia: Head and extremities spared.
   - Thin skin
   - Muscle weakness and muscle atrophy; cruciate ruptures
   - Mineralization of skin (calcinosis cutis)
   - Hyperpigmentation: ACTH similar to MSH, co-existing hypothyroidism, chronic skin irritation.
   - Reproductive abnormalities
     - Anestrus
     - Clitoral hypertrophy
     - Testicular atrophy
     - Perianal adenomas in females and neutered males.
   - Respiratory signs
     - Panting: Pulmonary hypertension and decreased compliance, primary CNS disturbance, pulmonary mineralization.
     - Dyspnea: Rare; seen with pulmonary thromboembolism and concurrent congestive heart failure.
   - Central nervous system
     - Seen with large pituitary tumors (macroadenomas). Present at time of diagnosis or following therapy for Cushing's disease as microscopic pituitary tumors enlarge into macroadenomas.
     - Signs due to compression/invasion of pituitary and/or hypothalamus:
       - Seizures
       - Pacing
       - Lethargy
       - Inappetence
       - Behavior change
       - Head pressing
       - Circling
4. Diagnosis of hyperadrenocorticism

- History and clinical signs
  - R/O iatrogenic disease with questions concerning current or past medications. These medications can include oral, ophthalmic, otic, and topical medications. Make sure the owner tells you about everything and anything that went on or in their pet.

- Laboratory data
  - Hemogram
    - Polycythemia (PCV 45-55%)
    - Stress leukogram
      - Lymphopenia
      - Eosinopenia
      - Neutrophilia (mature)
  - Biochemistry profile
    - Elevations in:
      - Serum alkaline phosphatase (SAP)
      - Cholesterol
      - Serum alanine aminotransferase (ALT)
      - Fasting blood glucose: Diabetes in 5-10%.
  - Thyroid function tests
    - T3 and T4 basal levels are generally decreased.
    - Response to TSH parallels normal.
    - Secondary to negative feedback of cortisol on pituitary.
    - 80% have a normal fT4ED
    - Does not require thyroid supplementation.
  - Blood pressure: 50 – 80% are hypertensive, cause unknown.
    - Recent study demonstrated normal or decreased levels of atrial natriuretic factor (ANF) in dogs with hyperadrenocorticism. Argues against hypervolemia as the etiology of the hypertension.
  - Urinalysis
    - Decreased urine specific gravity.
    - Proteinuria

- Radiographic abnormalities
  - Thoracic films
    - Bronchial calcification
    - Metastases from adrenal adenocarcinoma
  - Abdominal films
    - Hepatomegaly
    - Osteopenia
    - 50% of adrenal tumors are visualized as soft tissue or calcified masses.
    - Subcutaneous calcification

- Adrenal function tests
  - Three tests used to diagnose hyperadrenocorticism. They do not differentiate between PDH or AT.
    - ACTH stimulation test
      - Look for exaggerated cortisol response in response to ACTH.
      - See protocols at the end of this discussion.
      - Diagnostic in 85% of pituitary-dependent cases (PDH)
      - Diagnostic in 70% of adrenal tumors (AT)
      - Overall accuracy 80-85%
      - A suppressed response to ACTH in animals with clinical signs of hyperadrenocorticism suggests iatrogenic disease.
    - Low-dose dexamethasone suppression test
      - Low doses of dexamethasone inhibit ACTH release from the pituitary via negative feedback and decrease plasma cortisol concentrations in normal dogs.
      - Dogs with Cushing’s are more resistant to steroid suppression. Therefore, lack of suppression following dexamethasone = hyperadrenocorticism.
- Diagnostic in 95% of PDH
- Diagnostic in 100% of AT
- Overall 90-95%
- May also be used to distinguish PDH from AT (see below)
- See protocols

### Urine cortisol/creatinine ratio
- Assessment of cortisol production and excretion rate.
  - Sensitivity of this test is greater than that of the LDDS (some animals with clinical signs of hyperadrenocorticism may have normal LDDS response tests but elevated urine cortisol to creatinine ratios). Used as a screening test.
  - Test is easy to perform.
  - As with all adrenal function tests, elevated results may occur in animals with non-adrenal disease.
  - Positive tests confirmed with a LDDS.
  - Must be performed on urine obtained at home, preferably in the AM

### Tests to differentiate PDH from AT (performed after confirming diagnosis of hyperadrenocorticism).
- High-dose dexamethasone suppression test
  - With PDH, a high dose of dexamethasone results in a decrease in ACTH release from the pituitary and a decrease in plasma cortisol.
  - With AT, the tumor secretes cortisol autonomously thereby suppressing ACTH production. With low ACTH concentrations already present, dexamethasone has no effect on plasma cortisol.
  - 70% of patients with PDH suppress plasma cortisol to less than 50% of the pre-treatment value.
  - 100% of patients with AT do not suppress.
  - Therefore: Suppression = PDH; Lack of suppression = Inconclusive
  - See protocol
- Endogenous ACTH concentration
  - PDH: Levels normal or high
  - AT: Levels low to undetectable
  - Contact lab regarding sample handling and collection. Use of the preservative (Aprotinin) allows for greater utilization of this test.
  - Excellent method to differentiate PDH from AT.

### Testing protocols
These are suggested protocols that are used in the evaluation of patients with hyperadrenocorticism. You must use the protocol and normal values from the laboratory to whom you are submitting samples to properly evaluate endocrine tests.

- **ACTH Stimulation Test**
  - Synthetic ACTH (Cortrosyn) 5 ug/kg IV or IM; collect serum at 0 and 1 hour, or
  - ACTH gel (Acthar) 2.2 U/kg IM; collect serum at 0 and 2 hours.
  - Hyperadrenocorticism if post-cortisol > 20 ug/dl (530 nmol/L)

- **Low-Dose Dexamethasone Suppression Test**
  - 8 A.m: Baseline serum cortisol. Administer 0.01 mg/kg dexamethasone sodium phosphate (0.015 mg/kg dexamethasone) IV.
  - 12 p.m: Collect 4 hour post-dexamethasone cortisol.
  - 4 p.m: Collect 8 hour post-dexamethasone cortisol.
  - In normal animals cortisol suppresses to less than 1.0 ug/dl (27.5 mmol/L) at 8 hours.
  - 50% or greater suppression at either 4 or 8 hours together with lack of suppression at 8 hours is diagnostic for PDH and additional tests are not necessary.

- **Urine Cortisol/Creatinine Ratio**
  - First morning urine sample is preferred. Sample should be obtained at home. Requires 1 – 2 mls.
  - Stable at room temperature or refrigerated for 3 days.
  - Normal range 2.8 - 4.8. A normal result effectively rules-out hyperadrenocorticism, an abnormal result should be confirmed with a LDDS or ACTH stimulation test.

- **Differentiating PDH From AT**
  - Low-Dose Dexamethasone Suppression Test
    - See above.
  - High-Dose Dexamethasone Suppression Test
8 a.m: Obtain serum cortisol. Administer 0.1 mg/kg dexamethasone sodium phosphate (0.15 mg/kg dexamethasone) IV.
4 p.m: Collect post-dexamethasone cortisol.
Suppression defined as greater than a 50% reduction of cortisol.
Suppression = PDH, non-suppression = Inconclusive

- Endogenous ACTH Concentration
  - Check with lab on sample collection and handling.
  - Normal: 20-100 pg/ml (4.4-22.0 pmol/L)
  - PDH: 40-500 pg/ml (8.8-110 pmol/L)
  - AT: < 20 pg/ml (<4.4 pmol/L)

Treatment options
A. Pituitary-dependent hyperadrenocorticism
  1. Surgical management
     a. Bilateral adrenalectomy
        i. Technically difficult
        ii. Poor surgical/anesthetic risk
        iii. Permanently hypoadrenal and require lifelong replacement therapy
     b. Hypophysectomy
        i. See discussion at the end of this section
        ii. Lifelong therapy with thyroid hormone and prednisone necessary.
  2. Medical therapy

Prognosis
Most dogs with PDH live normal lives (average 2.2 years, but remember most are geriatric to begin with.)

1. Complications
   a. Recurrence of disease.
   b. CNS signs.
   c. Pulmonary thromboembolism.
   d. Infections.
   e. Hypertension.
   f. Congestive heart failure.

2. Adrenal tumors:
   a. Adenomas: Good if no evidence of local invasion.
   b. Carcinomas: Guarded to grave with metastases.

Trilostane therapy of canine hyperadrenocorticism
The efficacy and safety of trilostane in the treatment of canine PDH were evaluated in a multicentre study at the Royal Veterinary College in London, the Veterinary Teaching Hospital in Dublin and Small Animal Hospital in Glasgow. Seventy-eight dogs with confirmed PDH were treated with trilostane for up to 3 years. The starting dose varied from 1.8 to 20 mg/kg (mean = 5.9 mg/kg).

Trilostane appeared to be well tolerated by almost all dogs with only 2 dogs developing signs and biochemical evidence of hypoadrenocorticism. One of these dogs recovered with appropriate therapy. The other died despite withdrawal of trilostane and administration of appropriate therapy. A further two dogs died within one week of starting trilostane but in neither case could a direct link with the trilostane therapy be established. The low prevalence of side effects compared favourably to those reported with mitotane.

Trilostane was found to be nearly as effective as mitotane in resolving the signs of hyperadrenocorticism. Polyuria, polydipsia and polyphagia had dissipated in 40 dogs within 3 weeks after starting trilostane. Within 2 months, a further 20 dogs showed decreases in their water and food consumption. These improvements were maintained as long as the dogs remained on adequate doses of trilostane. Skin changes resolved in 24 out of 39 (62%) of dogs that initially presented with dermatological signs. All of these improvements were maintained as long as the dogs remained on adequate doses of trilostane. Only 8 dogs that were treated with trilostane for more than 2 months showed poor control of clinical signs. In contrast, mitotane is effective in about 80% of cases of pituitary dependent hyperadrenocorticism (PDH).

Trilostane caused a significant (p<0.001) reduction in both the mean basal and post-ACTH stimulation cortisol concentrations after 10 days of treatment. The post ACTH cortisol concentration decreased to less than 250 nmol/l (9 µg/dl) in 81% of dogs within one
month and in another 15% at some time whilst on treatment. These improvements were also maintained in the study population for the duration of the trial.

Thirty-five dogs had at least one dose adjustment over the treatment period. The dose was increased in 23 dogs up to four times the starting dose. In one dog the dose was increased nine fold over a period of six months. The dose was decreased in nine dogs to as low as a quarter of the starting dose.

The mean survival of all trilostane treated dogs was 661 days. Direct comparison with mitotane was difficult as 65% of the dogs were still alive at the time of censor and therefore the mean survival may still increase. By comparison, the mean survival of mitotane treated dogs has been reported to be 810 to 900 days.

**Dosage and administration**

The current suggested initial starting dose range for dogs with PDH is 1-2 mg/kg once daily. This needs to be adjusted according to clinical signs and serum cortisol values (see below). Doses up to 40-50 mg/kg (divided twice daily) have been given with no unwanted side effects. In some dogs twice daily dosing may be necessary. The drug is given with food.

**Transsphenoidal hypophysectomy**

A variety of treatments are available for PDH. Medical treatment options include drugs that chemically destroy the adrenals (lysodren or op-DDD) inhibit enzymes in the adrenal leading to the synthesis of cortisol (ketoconazole, trilostane) or inhibit the release of ACTH from the pituitary gland (Anipryl or selegiline). While these treatments can improve the clinical signs in 40-80% of patients they need to be chronically administered, necessitate frequent monitoring and do not cure or address the primary cause of the disease (the pituitary tumor). In humans, surgery to remove the tumor is the most successful long-term therapy. The most common approach used is the transsphenoidal method, in which a passage way is made in the sphenoid sinus, an air space behind the back of the nose, which is just below the pituitary gland. Surgical cure rates for PDH are reported to be in the range of 65-85%, although more recent long-term follow up data suggest that the recurrence rate is as high as 25% within 5 years. When no discrete adenoma can be identified, remission of hypercortisolism is observed in only about 40%. Surgery has also been used to treat PDH in dogs. Several groups, most notably in the Netherlands have performed these surgeries with success rates paralleling those reported for humans. However, these surgeries have generally not been performed in the US. Veterinarians at VCAWLAAH, in collaboration with human neurosurgeons that regularly perform transsphenoidal surgery in humans have developed the methods to perform these surgeries in the US and are conducting a research study to determine how effectively these surgeries can be performed.
Feline acromegaly is a disease characterized by excessive growth hormone secretion. Growth hormone is produced in the pars distalis of the anterior pituitary, specifically by acidophilic cells called somatotrophs. The release of growth hormone is regulated by many factors, the most important being growth hormone releasing hormone (GHRH) produced in the hypothalamus. Recently, another hormone called ghrelin has been identified as also being a potent stimulator of the release of growth hormone. Ghrelin is released by the stomach after it has received a meal. Release of growth hormone is inhibited by the hypothalamic hormone somatostatin. In addition growth hormone itself and insulin like growth factor-1 (IGF-1) exhibit negative feedback on the release of growth hormone.

Growth hormone has 2 classes of actions. The first are the catabolic actions of growth hormone that includes insulin antagonism, lipolysis, and gluconeogenesis with the end effect of creating hyperglycemia. The second class of actions are the slow anabolic (or hypertrophic) effects. These effects are mediated by insulin like growth factors which are produced in many different tissues. The most important is insulin like growth factor-1 (IGF-1) which is produced in the liver. The net effects of the anabolic actions of growth hormone are responsible for the characteristic appearance of acromegalic people, dogs and cats.

Feline acromegaly is caused by a functional adenoma of the pituitary that releases growth hormone despite negative feedback, which leads to excessive growth hormone production and release. Growth hormone has 2 classes of actions. The first are the catabolic actions of growth hormone that includes insulin antagonism, lipolysis, and gluconeogenesis with the end effect of creating hyperglycemia. The second class of actions are the slow anabolic (or hypertrophic) effects. These effects are mediated by insulin like growth factors which are produced in many different tissues. The most important is insulin like growth factor-1 (IGF-1) which is produced in the liver. The net effects of the anabolic actions of growth hormone are responsible for the characteristic appearance of acromegalic people, dogs and cats.

Feline acromegaly is an uncommon disease although it may be under diagnosed. A recent study in the United Kingdom measured IGF-1 levels in variably controlled diabetic cats. Of the 184 cases, 59 (32%) had markedly increased IGF-1 concentrations. Eighteen of these 59 cats underwent pituitary imaging and confirming a diagnosis of acromegaly in 17/18 (94%).

**Signalment, history, clinical signs**

Feline acromegaly most commonly affects middle aged to older, male castrated cats. In the aforementioned study 15 of the 17 cats diagnosed with acromegaly were males with an average age of 10.1 years. This association may be biased, however, as most cats that are diagnosed with acromegaly present for insulin resistant diabetes mellitus which is also most common in older, male castrated cats. Based on available data there is no known breed association for acromegaly.

Most acromegalics present for insulin resistant diabetes mellitus (insulin doses greater than 1.5-2.2 units/kg BID) with concurrent weight gain rather than weight loss. Growth hormone theoretically affects all the tissues of the body and therefore the disease has a range of clinical signs. Physical characteristics of acromegaly include increased body weight, broadened face, enlarged feet, protrusion of the mandible (prognathia inferior), increased interdental spacing, stertorous breathing, organomegaly, and poor haircoat. Cardiovascular signs include the presence of a heart murmur, hypertension, arrhythmias (gallop), and is associated with hypertrophic cardiomyopathy. Neurologic disease associated with feline acromegaly is uncommon but can occur with a pituitary macroadenoma. Neurologic signs that have been observed with acromegaly include dullness, lethargy, abnormal behavior, circling, and blindness.

Neurologic signs that have been observed with acromegaly include dullness, lethargy, abnormal behavior, circling, and blindness. Glomerulopathy and secondary renal failure has also been associated with feline acromegaly. Histopathologic evaluation of the kidneys from acromegalic cats have revealed thickening of the glomerular basement membrane and Bowman’s capsule, periglomerular fibrosis, and degeneration of the renal tubules. Arthropathy and peripheral (diabetic) neuropathy have been shown to cause lameness in acromegalic cats.

**Diagnosis**

Diagnosis of feline acromegaly starts with clinical suspicion, using a thorough history, signalment and clinical signs. Minimum database abnormalities include erythrocytosis, hyperglycemia, increased liver enzymes (ALT, ALP), hypercholesterolemia, hyperphosphatemia, hyperglobulinemia, and azotemia. Common findings on urinalysis include glucosuria, ketonuria, proteinuria, and isosthenuria. Many of these abnormalities reflect concurrent diabetes mellitus.

Specific assays for feline growth hormone are not widely available. An ovine test for feline growth hormone has been validated for use, but is only available in Europe. However, growth hormone concentration may not be a reliable diagnostic on its own. Growth hormone production is cyclic and levels may vary throughout the day. A single low or high value may not necessarily be diagnostic for acromegaly. Additionally, it has been shown that growth hormone may be elevated in non-acromegalic diabetic cats. This is due to the fact that portal insulin is required for the liver to produce IGF-1. In diabetics that are being treated with insulin subcutaneously, portal insulin concentrations remain low resulting in decreased IGF-1 production and decreased inhibition of GH release. Growth hormone levels may also not be elevated early in the course of the disease, but later typically increase significantly.

IGF-1 is the most commonly used endocrine assay used to diagnose feline acromegaly and it is widely available through the Michigan State University Diagnostic Center for Population and Animal Health. Unlike growth hormone, IGF-1 concentrations are less likely to fluctuate over the course of the day as the majority of IGF-1 is protein bound giving it a longer serum half-life. Insulin like growth factor-1 increases in response to chronically elevated growth hormone concentrations and is thought to be a reflection of
growth hormone levels over the last 24 hours. However, just like growth hormone, elevations in IGF-1 concentration alone may not be diagnostic for acromegaly. One study found that IGF-1 levels in non-acromegalic cats on long-term insulin treatment (>14 months) had higher levels of IGF-1 than non-diabetics. The proposed mechanism for this was that insulin treatment allowed for beta cell regeneration and increased portal insulin leading to elevations in IGF-1. A subsequent study evaluating IGF-1 levels in confirmed acromegalic diabetics, non-acromegalic diabetics, and healthy cats found that acromegalic diabetics had significantly higher levels of IGF-1 than diabetics and non-diabetics. This study concluded that IGF-1 was 84% sensitive and 92% specific for diagnosing feline acromegaly. No correlation between long-term insulin use and elevations in IGF-1 concentrations were found in this study.

Diagnostic imaging
Radiographic findings associated with feline acromegaly are related to the hypertrophic effects of excessive growth hormone. Hyperostosis of the calvarium, spondylosis of the spine, and protrusion of the mandible are common findings. Periosteal reaction, osteophyte production, soft tissue swelling, and collapse of joint spaces are signs associated with the degenerative joint arthropathy linked to feline acromegaly. Thoracic radiographs may reveal cardiomegaly (hypertrophic cardiomyopathy) or congestive heart failure. Non-specific signs such as abdominal organomegaly (hepatic, renal, and adrenal) may be revealed by abdominal ultrasound.

Advanced imaging is needed to document the presence of a pituitary macroadenoma. Computed tomography (CT) and magnetic resonance imaging (MRI) are both useful for identifying pituitary masses. However one study found MRI to be the more sensitive imaging modality. The presence of a pituitary tumor alone is not diagnostic for feline acromegaly as there are other tumor types that can affect the pituitary. Conversely, the absence of a pituitary mass does not rule out acromegaly as there have been reported cases where a patient had a negative MRI but a pituitary mass was identified at necropsy and histopathology confirmed a growth hormone secreting adenoma.

Histopathology
Histopathology is needed for definitive diagnosis which makes ante-mortem diagnosis challenging. However, with advancements in surgical procedures such as transsphenoidal hypophysectomy, surgical excisional biopsy is possible. Histopathology of pituitary tumors in acromegalic cats reveals acidophil proliferation and adenoma formation.

Adrenocortical testing
There is no single test for the diagnosis of feline acromegaly. Clinical suspicion based on a thorough history and physical exam are essential. As earlier stated the most common presenting complaint for patients with acromegaly is insulin resistance. The 2 most common causes of insulin resistance in cats are hyperadrenocorticism and acromegaly. Both of these diseases can be associated with a pituitary mass and bilateral adrenomegaly. As such, all suspected acromegalics should undergo adrenocortical function testing via the ACTH stimulation test and/or low dose dexamethasone suppression test. Normal results on these tests would then be an indication to screen for acromegaly.

Medical management
Somatostatin is a hypothalamic hormone that acts on the pituitary to inhibit growth hormone release. Somatostatin analogs are commonly used in human medicine for the treatment of acromegaly and have efficacy rates approaching 90%. The somatostatin analog, octreotide, has been evaluated in a small number of feline acromegalics with limited success. One study in 4 cats, found no change in growth hormone concentrations following treatment. Another study measured the short term effects of octreotide in 5 feline acromegalics and found a decrease in growth hormone concentrations for up to 90 minutes. The results of the second study warrant further examination of somatostatin analogs especially newer long-acting formulations (name them here). Future studies are also required to identify the somatostatin receptor subtypes being expressed on feline pituitary adenomas and determine if these receptor subtypes are similar to the ones found in humans.

Growth hormone receptor antagonists and dopamine agonists are also used in human medicine. I would briefly mention the efficacy of these medications in humans. The use of growth hormone receptor antagonists has not been reported in cats. A single case study using a dopamine agonist (L-deprenyl) for the treatment of feline acromegaly showed no effect on reducing insulin requirements or clinical signs of disease.

Finally increasing the dosage of insulin to gain control of the clinical signs of the diabetes, is the most conservative choice for treating insulin resistant diabetics with acromegaly. However, there have been reports that some of these patients suddenly and inexplicably become sensitized to insulin resulting in hypoglycemic crises. In one study, several acromegalic cats were euthanized after experiencing severe episodes of hypoglycemia.

Surgical treatment
Surgical removal of the pituitary tumor (adenectomy) is the treatment of choice for acromegaly in human medicine. The procedure can be performed in cats and dogs as well usually employing complete removal of the entire pituitary (hypophysectomy). Availability
is limited and the only hospital that regularly performs the procedure in the United States is the VCA West Los Angeles Animal Hospital, though other institutions may soon be able to offer this option. In veterinary medicine a transsphenoidal approach is used involving only a small incision through the soft palate and then approaching the pituitary gland through the basisphenoid bone. Complications associated with the surgery include, hemorrhage, incision dehiscence and formation of an oronasal fistula. Additionally, after surgery the patient’s are at risk for hypopituitarism and may require life long supplementation with cortisone, l-thyroxine, and desmopressin making patient selection an important pre-requisite for surgery. The same surgical procedure is also used to treat pituitary dependant hyperadrenocorticism. A study in which 7 cats with pituitary dependant hyperadrenocorticism were treated with transsphenoidal hypophysectomy resulted in 5 cats showing complete resolution of the disease. Four of these cats had concurrent diabetes mellitus, 2 of which showed increased insulin responsiveness after surgery. A single case report exists for the treatment of feline acromegaly with transsphenoidal hypophysectomy. Prior to surgery the patient was also an insulin resistant diabetic that was still clinical despite receiving 25 U of insulin 4 times per day. Three weeks after surgery the patient no longer required any insulin therapy and up to a year later the patient’s IGF-1 and GH concentrations were within normal limits (no further follow up was available in the study).

We can put in our cat that underwent surgery here. Do you have the images of the MRI?

An alternative procedure, cryohypophysectomy, has been reported in 2 cats but the procedure has shown to be less effective and resulted in increased complications.

Radiation
Radiation therapy is another option for the treatment feline acromegaly especially if the tumor is inoperable, the patient is not a suitable candidate for anesthesia, or if surgical treatment is not available. In human medicine radiation therapy is regarded as a second line treatment due to undesired long-term effects of radiation on brain tissue. However, in veterinary medicine many of our patients, especially those that suffer from acromegaly, are not expected to live long enough to experience these long-term side effects. The majority of studies that have been performed in veterinary medicine focus on the treatment of pituitary tumors in general regardless of underlying etiology for the sake of sample size. There is no standard treatment protocol for pituitary masses in veterinary medicine and varying methods have been used including both single and multiple dose fractions administering total dosages ranging from 1,500 – 4,500 cGY. The majority of the cats included in these studies had insulin resistant diabetes (suspected acromegaly or Cushing’s disease) and/or neurologic signs. Response to treatment was good as most patients responded (Can you list % of cats having remission of clinical signs, remission of diabetes and overall survival for a few of these studies?) . Neurologic improvement was seen within weeks to months and an improved insulin response was seen within the first month, however, most still required insulin therapy. In cases where repeat imaging was available a decrease in tumor size was also noted. Disadvantages of radiation therapy are the early and delayed effects of radiation, repeated anesthesia, and expense. Early effects from radiation therapy include hair loss, skin pigmentation and otitis externa. Reported late side effects include brain tissue necrosis, tumor regrowth, and visual and hearing impairment.

Conclusion
Feline acromegaly is likely an under diagnosed disease in older male cats, especially in patients with insulin-resistant diabetes. There is no single diagnostic test for acromegaly. The diagnostician should use history, clinical signs, laboratory tests (GH and IGF-1), and advanced imaging to arrive at a diagnosis. There are several treatments options, however, clinical studies on long-term safety and efficacy are limited and often lack controls. Until more work is done evaluating medical treatments such as somatostatin analogues and growth hormone antagonists, most patients are best treated with either surgery or radiation therapy to control GH levels, improve glycemic control, and either remove or control the growth of the pituitary tumor.
Hyperadrenocorticism

Hyperadrenocorticism develops most commonly in middle-aged to older cats (mean age = 10.4 years; range 6 - 15 years). Of the reported cases of feline Cushing's syndrome (78%) have been females. This female sex predilection resembles the human syndrome and contrasts with canine hyperadrenocorticism, where no sex predilection occurs.

The most common historical findings and clinical signs associated with feline hyperadrenocorticism are polyuria, polydipsia, and polyphagia. These signs likely correspond to the high incidence of concurrent diabetes mellitus (76%) found in cats with hyperadrenocorticism, and are consistent with the lack of overt signs preceding marked glucose intolerance observed in experimentally-induced disease. The typical "Cushingoid" pot-bellied appearance with hepatomegaly, weight gain, and generalized muscle wasting is common in cats as in dogs. Dermatologic abnormalities frequently recognized include an unkempt hair coat with patchy alopecia, and very thin skin prone to traumatically induced tears and secondary infections.

Hyperglycemia is the most common laboratory abnormality found on serum biochemistries. Cats appear more sensitive to the diabetogenic effects of glucocorticoid excess than dogs. Cats with concurrent diabetes mellitus often exhibit cortisol-induced insulin resistance, requiring high daily doses of insulin to control their hyperglycemia and glucosuria. Hypercholesterolemia is also common, and may relate to insulin resistance and increased lipolysis. Cats lack the steroid-induced isoenzyme of alkaline phosphatase found in the canine, and the half-life of the enzyme appears to be significantly shorter in the cat. Elevation of serum alkaline phosphatase (SAP) is present in only approximately one-third of cats compared to nearly 90% of dogs with hyperadrenocorticism. Increases in SAP and the hepatocellular enzyme ALT appear to correspond with the regulation of the diabetic state, rather than representing direct resistance, requiring high daily doses of insulin to control their hyperglycemia and glucosuria. Hypercholesterolemia is also common, and may relate to insulin resistance and increased lipolysis. Cats lack the steroid-induced isoenzyme of alkaline phosphatase found in the canine, and the half-life of the enzyme appears to be significantly shorter in the cat. Elevation of serum alkaline phosphatase (SAP) is present in only approximately one-third of cats compared to nearly 90% of dogs with hyperadrenocorticism. Increases in SAP and the hepatocellular enzyme ALT appear to correspond with the regulation of the diabetic state, rather than representing direct indicators of glucocorticoid excess. These enzymes frequently normalize with adequate regulation of diabetes, even without therapy directed towards the hyperadrenocorticism. Hematologic findings associated with hypercortisolemia (lymphopenia, eosinopenia, and neutrophilic leukocytosis) occur inconsistently in feline hyperadrenocorticism. Despite clinical polyuria and polydipsia, cats appear to maintain urine specific gravities of greater than 1.020 more frequently than dogs, and only occasionally exhibit dilute urine and decreased blood urea nitrogen concentrations commonly seen in dogs with hyperadrenocorticism.

Endocrinologic evaluation of cats suspected of hyperadrenocorticism involves screening tests to confirm the diagnosis, and differentiating tests to distinguish pituitary-dependent disease (PDH) from adrenal tumors (AT). Adrenocorticotropicin (ACTH) stimulation testing in adrenocortical hyperfunction is not as definitive as for hypoadrenocorticism. Fifteen to 30% of cats with confirmed hyperadrenocorticism have had normal cortisol response to ACTH administration (false negatives). In addition, stressed cats and those with non-adrenal illnesses may show an exaggerated response to ACTH in the absence of hyperadrenocorticism (false positives). A normal urine cortisol-to-creatinine ratio (UCCR) can be used to exclude the diagnosis of hyperadrenocorticism in cats as described in dogs. The UCCR is attractive due to the ease of sampling compared to other endocrine function tests, but is non-specific and will be elevated in a variety on non-adrenal illnesses. An exaggerated ACTH stimulation test or an elevated UCCR should be pursued with suppression testing prior to initiating any therapy.

Normal cats are more variable than dogs with respect to the degree and duration of adrenocortical suppression following dexamethasone administration. Intravenous doses of dexamethasone that have been evaluated in the cat range from 0.005 to 1.0 milligrams per kilogram. A dosage of 0.01 mg/kg of dexamethasone, commonly used in low-dose dexamethasone suppression testing in dogs, led to a significant drop in serum cortisol levels in ten normal cats, but 2 of the cats showed a slight escape from suppression by 8 hours after injection. Intravenous dexamethasone sodium phosphate (DSP), 0.01 and 0.1 mg/kg, produced equivalent reductions of plasma cortisol levels, but suppression was sustained below baseline longer with the higher dosage. Cats with various non-adrenal illnesses have also shown inadequate cortisol suppression after a low-dose (0.01 mg/kg) of DSP. The 0.1 mg/kg dosage of dexamethasone seems to more reliably suppress cortisol levels in normal cats and cats with non-adrenal illnesses. Elevated cortisol levels eight hours post-dexamethasone injection, using the 0.1 mg/kg dosage, appears to be a sensitive a diagnostic test for feline hyperadrenocorticism (89%) similar to the low-dose (0.01 mg/kg) screening test in the dog.

The combined dexamethasone suppression/ACTH stimulation test has been used successfully to diagnose hyperadrenocorticism in the cat. Affected cats display inadequate suppression of cortisol 2-4 hours after an injection of 0.1 mg/kg of dexamethasone, and an exaggerated response 1-2 hours after ACTH stimulation. The ability of the combined test to discriminate PDH from AT is unclear. Several cats with confirmed pituitary disease failed to suppress 2-4 hours after dexamethasone. Extending the duration of post-dexamethasone monitoring, or using higher doses of DSP may improve the ability of the combined test to distinguish PDH from AT. Currently, the combined test does not appear to offer more clinical utility than either the ACTH stimulation or dexamethasone suppression test evaluated separately.
An ultra-high dose, 1.0 mg/kg, dexamethasone suppression test has been used to distinguish PDH from AT in the cat. Two cats with hyperadrenocorticism diagnosed by the combined high dose dexamethasone suppression/ACTH stimulation test had exaggerated responses to ACTH with no cortisol suppression 2-4 hours after 0.1 mg/kg DSP. These cats did suppress following the ultra-high dose of dexamethasone, and were later confirmed to have PDH. Cortisol levels should be monitored at several time points following dexamethasone administration to determine if any suppression (a 50% or greater reduction in pre-test values) is occurring. Cats with PDH may show suppression 2, 4, or 6 hours into the test only to escape from the suppressive effects of dexamethasone by 8 hours. One cat with an adrenal adenoma failed to suppress following dexamethasone doses ranging from 0.1 to 1.0 mg/kg. As is the case in dogs, suppression following high doses of dexamethasone is diagnostic for PDH, but failure to suppress requires further testing to distinguish pituitary from adrenal disease.

Determination of plasma ACTH concentrations is an effective way of diagnosing PDH. The normal range of plasma ACTH is lower in cats than in dogs, and many normal cats may have concentrations of ACTH below the lower limits of the sensitivity of the assay. Cats with PDH will have normal to elevated ACTH concentrations while cats with adrenocortical adenomas or carcinomas will have undetectable plasma ACTH levels. Plasma ACTH samples need to be collected and handled carefully. Veterinarians should consult their diagnostic laboratory for specific instructions prior to performing the test. Incorrect sample handling can falsely lower measured values. Normal to elevated plasma ACTH levels support a diagnosis of PDH, whereas low concentrations may require additional diagnostic testing. As in the differentiation of canine hyperadrenocorticism, ACTH levels should only be used to distinguish PDH from AT after hyperadrenocorticism has been confirmed by other screening diagnostics.

Pituitary-adrenal function tests need to be interpreted in conjunction with historical, clinical, and clinicopathologic findings before any conclusions can be drawn. No single diagnostic test is infallible. Equivocal results or discordant findings should be reevaluated. Hyperadrenocorticism is an uncommon disorder in cats. Consequently, false positive test results should be anticipated. Interpretation of endocrinologic testing should incorporate all available information before any therapeutic intervention is attempted.

Diagnostic imaging can facilitate differentiation of PDH from AT when screening tests and clinical findings suggest hyperadrenocorticism. Approximately half of canine adrenal tumors are mineralized and can be recognized radiographically. The frequency of mineralization in feline adrenocortical tumors is unknown, but up to 30% of normal cats may have calcification of their adrenal glands. Abdominal radiographic findings in cats with hyperadrenocorticism included hepatomegaly (69%) and obesity. Ultrasonographic evaluation of adrenal size and morphology has been described for dogs and cats. Nonfunctional adrenal tumors can be incidental findings in humans undergoing abdominal imaging. The incidence of "silent" adrenal masses in the cat is unknown. The presence of unilateral adenomegaly or distortion of adrenal architecture in a cat suspected of hyperadrenocorticism is strong evidence of AT. Abdominal computerized tomography (CT) and magnetic resonance imaging (MRI) offer improved resolution for the detection of adrenal tumors or hyperplasia. CT and MRI detection of pituitary masses is now feasible for small animal patients.

Adrenal tumors accounted for 22% of the reported cases of feline hyperadrenocorticism. Half of the adrenocortical tumors were found histologically to be adenomas and half carcinomas. The treatment of choice for adrenal tumors is surgical adrenalectomy. Two cats with adrenocortical adenomas responded well to unilateral adrenalectomy, with clinical signs resolving over 4 to 8 weeks. One cat with an adrenal adenoma removed surgically developed a recurrence of signs 12 months postoperatively. An adenoma of the contralateral adrenal gland was diagnosed. The cat survived a second adrenalectomy and was disease-free for over two years following the second procedure. Surgical therapy and long term follow-up for adrenocortical carcinomas in cats has not been reported.

Treatment options for pituitary dependent hyperadrenocorticism in the cat include both surgical and medical alternatives. Bilateral adrenalectomy followed by mineralocorticoid and glucocorticoid replacement therapy was performed in 11 cats. Nine cats responded well to surgery with cessation of polyuria and polydipsia, regrowth of hair coat, and marked improvement (4) or resolution (5) of diabetes mellitus. One cat developed acute signs of circling, wandering aimlessly, and apparent blindness 2 months post-operatively. An expanding pituitary tumor was suspected, but necropsy was performed. Two cats died within one week of surgery from sepsis. Survival times for 6 cats with adequate follow-up after bilateral adrenalectomy for PDH ranged from 1 to 12+ months (median 5 months). Two cats are still alive, one year post-operatively. These results suggest that surgical complications of bilateral adrenalectomy may be less frequent in cats than in dogs.

Surgical treatment can also include transphenoidal hypophysectomy which is performed at WLA for cats with pituitary masses extending above the sella (macroadenoma). Cats with functional tumors have similar success rates to those reported in dogs with PDH.

Four drugs (ketoconazole, mitotane, metyrapone and trilostane) have been investigated for the medical management of spontaneous feline hyperadrenocorticism. Ketoconazole, an antifungal imidazole derivative, has been shown to inhibit adrenal and gonadal steroidogenesis in humans and dogs. One month of ketoconazole (15mg/kg orally twice daily) administration in 4 cats did not significantly reduce baseline plasma cortisol or ACTH responsiveness at doses 3 times greater than those effective in dogs. Two of 4 cats treated with 10 - 20 mg/kg/day of ketoconazole had adequate control of hypercortisolemia. One of the 4 cats developed severe thrombocytopenia after only one week of therapy and had to discontinue the medication. A cat with adrenocortical adenocarcinoma...
treated with 30 mg/kg/day for 3½ months showed improved regulation of diabetes and reduction in pu/pd despite no improvement in hyperresponsiveness to ACTH. The cat ultimately was euthanatized subsequent to a non-healing skin laceration, chronic infections, and worsening insulin resistance. No evidence of hepatotoxicity or thrombocytopenia was seen at the 30 mg/kg/day dosage of ketoconazole, but the effectiveness and safety of this therapy remains questionable.

Mitotane, o,p'-DDD, is an adrenal cytotoxic agent and has been used successfully to treat dogs with PDH and AT. Use of mitotane in cats has been discouraged due to the feline sensitivity to chlorinated hydrocarbons. Three of 4 normal cats treated with o,p'-DDD at dosages ranging from 25 - 50 mg/kg, divided twice a day, tolerated the drug well, and remained clinically normal throughout treatment with mitotane. Only 2 of the 4 cats showed a decreased responsiveness to ACTH with mitotane. The cat with the largest reduction in post-ACTH cortisol levels developed vomiting, diarrhea, and partial anorexia lasting 2 weeks after a 50 mg/kg dosage of mitotane. Two cats with PDH treated with o,p'-DDD (25 mg/kg/day x 25 days, and 25 - 50 mg/kg/day x 59 days) tolerated the drug without apparent toxicity, but therapy was ineffective in controlling clinical signs in either cat. A cat with PDH treated with mitotane (50 mg/kg/day x 1 week, then 50 mg/kg/week) developed signs compatible with iatrogenic hypoadrenocorticism after 40 weeks of therapy with o,p'-DDD. At that time the cat was anorectic, lethargic, and exhibiting neurologic signs including mydriasis, pacing, and head pressing. Computerized tomography revealed a large pituitary mass extending above the sella turcica. Mitotane was discontinued, and the cat was treated with 90Co teletherapy. Subsequent CT examinations revealed shrinkage and then disappearance of the mass 10 months post-irradiation. The cat was euthanatized for continued diabetes mellitus and post-irradiation cataracts 2 years after the initial diagnosis of hyperadrenocorticism. We have had 3 other cases where a positive response to mitotane was observed clinically.

Metyrapone, an inhibitor of the 11-b-hydroxylase enzyme that converts 11-deoxycortisol to cortisol, has been used effectively in man to reduce the clinical signs of hypercortisolemia. A reciprocal rise in plasma ACTH levels occurs with falling cortisol concentrations and can eventually override the enzymatic block, allowing a return of clinical signs. In humans, metyrapone is utilized as an adjunctive therapy with pituitary irradiation or surgery. Dosages ranging from 195 - 250 mg/day have been used in cats with hyperadrenocorticism without observed toxicity. In a recent report, a diabetic cat with PDH and severe nonhealing skin wounds was treated with 65 mg of metyrapone orally 3 times a day. After 2 days of therapy the cat developed signs of glucocorticoid deficiency including depression, tremors, and ataxia. The cat improved rapidly following treatment with injectable steroids, and was discharged on twice daily metyrapone therapy. Cortisol response to exogenous ACTH was absent when evaluated on day 7. The cat was re-examined 24 days later after a hypoglycemic episode. The cats skin wounds had resolved and hair regrowth was evident. A follow-up ACTH stimulation test revealed a slightly exaggerated response. The cat underwent successful bilateral adrenalectomy and was euglycemic, with a normal haircoat, 4 months post-operatively. Two of 3 other cats reported in the literature also showed clinical improvement with metyrapone therapy, but follow-up periods were short (less than 6 months). Whether longterm therapy with metyrapone can control hypercortisolemia in cats, or whether rising ACTH levels eventually overwhelm enzymatic blockade has not been determined. Metyrapone appears to permit rapid correction of hyperadrenocorticism in some cats, and may be useful for presurgical stabilization prior to adrenalectomy.

We have recently evaluated the safety and efficacy of trilostane therapy (Vetoryl, Dechra Pharmaceuticals) in 15 cats with PDH. Clinical signs (13 of 15 cats) and ACTH stimulation testing results (13 of 15) improved with trilostane therapy. Diabetes mellitus was reported in 9/15 cases. Insulin requirements decreased by 36% within 2 months in 6/9 diabetic cats. Median survival time was 617 days for all cats (range 80-1,278 days). Complications included weight loss, urinary tract infections, chronic kidney disease, seizures, and recurrent pancreatitis. Hypocortisolemia was documented in 1 case. Cause of death occurred as a result of non-adrenal or non-diabetic illnesses (renal failure, seizures [caused by hypoglycemia or unknown]), or lymphoma. Trilostane ameliorates clinical signs of HAC in cats, is tolerated well in the long term, and can lead to improved regulation of diabetes. It should be considered first line therapy for cats undergoing medical management of PDH.

**Hypoadrenocorticism**

Primary hypoadrenocorticism has been described in cats. Addisonian cats are middle-aged, with a median age of 4 years (mean 5.8 +/- 3.7 years) and range in age from 1.5 to 14 years. No sex or breed predilection is seen. The most common historical problems include lethargy, anorexia, and weight loss. Unlike dogs with adrenal insufficiency, diabetes is not noted in Addisonian cats. Forty percent of cats have histories of episodic vomiting. Similar to hypoadrenocorticism in the canine, cats often have a waxing and waning clinical course, including temporary "remissions" associated with parenteral fluid and/or corticosteroid administration.

The most common findings on physical examination include depression, weakness, and mild to severe dehydration. Up to 40% present with in severe shock with weak pulses, slow capillary refill times, and extreme weakness or collapse. The duration of clinical signs preceding the diagnosis of hypoadrenocorticism ranges from 5 to 100 days, with a median of 14 days.

Clinicopathologic findings in cats with primary hypoadrenocorticism parallel the patterns seen in the dog. Serum electrolyte changes characteristic of mineralocorticoid deficiency are seen in most cats. Serum sodium:potassium ratios are less than 24 (range
17.9-23.7) with hyponatremia, hypochloremia, and hyperkalemia. All cats have had mild to severe azotemia (blood urea nitrogen 31-80 mg/dl, normal range 5-30 mg/dl; creatinine 1.6-6.0 mg/dl, normal range 0.5-1.5 mg/dl), and hyperphosphatemia (inorganic phosphorus 6.1-9.1 mg/dl; normal range 3.0-6.0 mg/dl). Hypercalcemia has been noted in one cat. Despite signs of dehydration and prerenal azotemia, urine specific gravity was greater than 1.030 in only 40% of cats. The loss of renal medullary solutes, particularly sodium, is believed to result in impaired renal concentrating ability. Distinguishing hypoadrenocorticism from acute or chronic renal failure is critical to establishing an appropriate prognosis for clients.

Long-term management of cats with primary hypoadrenocorticism requires lifetime mineralocorticoid and glucocorticoid supplementation. Oral fludrocortisone acetate (0.1 mg/day) or intramuscular injections of desoxycorticosterone pivalate (DOCP; 10 -12.5 mg/month) have been successful in maintaining Addisonian cats. The dose of mineralocorticoid is adjusted as needed based on follow-up serum electrolyte concentrations monitored every one to two weeks during the initial maintenance period. Normal electrolyte parameters 2 weeks following DOCP suggests adequate dosing, but does not provide information concerning the duration of action of each injection. Eighty percent of dogs require DOCP more frequently than every 30 days (5% need to receive DOCP every 3 weeks), so frequent sampling during the early management period is recommended. Prednisone, 1.25 mg orally once a day, or intramuscular methylprednisolone acetate, 10 mg once a month, can be used to provide adequate long term glucocorticoid supplementation. Cats surviving the initial adrenal crisis can be managed successfully for many years. 60% of cats diagnosed with primary hypoadrenocorticism are alive a median of 2.75 years after diagnosis. With appropriate glucocorticoid and mineralocorticoid supplementation, cats with adrenocortical insufficiency should have a normal life expectancy.

Primary hyperaldosteronism

Feline primary hyperaldosteronism is diagnosed based on clinical signs, serum biochemistry, plasma aldosterone concentration, adrenal imaging and histopathology of adrenal tissue. Cats may present with blindness caused by systemic hypertension. Many will also present with weakness resulting from hypokalaemic polymyopathy. Elevated concentrations of plasma aldosterone and adrenocortical neoplasia have been documented in all cases. Seven cases had adrenal adenomas (unilateral in five and bilateral in two) and six had unilateral adrenal carcinomas. Three cases underwent medical treatment only with amlodipine, spironolactone and potassium gluconate; two cases survived for 304 and 984 days until they were euthanized because of chronic renal failure, while the third case was euthanized at 50 days following failure of the owner to medicate the cat. Ten cases underwent surgical adrenalectomy following a successful stabilization period on medical management. Five cases remain alive at the time of writing with follow-up periods of between 240 and 1803 days. Three cases were euthanized during or immediately following surgery because of surgical-induced hemorrhage. One cat was euthanized 14 days after surgery because of generalized sepsis, whilst the remaining cat was euthanized 1045 days after surgery because of anorexia and the development of a cranial abdominal mass. It is recommended that primary hyperaldosteronism should be considered as a differential diagnosis in middle-aged and older cats with hypokalaemic polymyopathy and/or systemic hypertension and this disease should no longer be considered a rare condition.

In recent years, there has been renewed interest in primary hyperaldosteronism, particularly because of its possible role in the progression of kidney disease. While most studies have concerned humans and experimental animal models, a recent paper highlighted the occurrence of a spontaneous form of (non-tumorous) primary hyperaldosteronism in cats. At presentation, the main physical features of 11 elderly cats were hypokalemic paroxysmal flaccid paresis and loss of vision due to retinal detachment with hemorrhages. Primary hyperaldosteronism was diagnosed on the basis of plasma concentrations of aldosterone (PAC) and plasma rennin activity (PRA), and the calculation of the PAC:PRA ratio. In all animals, PACs were at the upper end or higher than the reference range. The PRAs were at the lower end of the reference range, and the PAC:PRA ratios exceeded the reference range. Diagnostic imaging by ultrasonography and computed tomography revealed no or only very minor changes in the adrenals compatible with nodular hyperplasia. Adrenal gland histopathology revealed extensive micronodular hyperplasia extending from zona glomerulosa into the zona fasciculata and reticularis. In three cats, plasma urea and creatinine concentrations were normal when hyperaldosteronism was diagnosed but thereafter increased to above the upper limit of the respective reference range. In the other eight cats, urea and creatinine concentrations were raised at first examination and gradually further increased. Even in end-stage renal insufficiency, there was a tendency to hypophosphatemia rather than to hyperphosphatemia. The histopathological changes in the kidneys mimicked those of humans with hyperaldosteronism: hyaline arteriolar sclerosis, glomerular sclerosis, tubular atrophy and interstitial fibrosis. The non-tumorous form of primary hyperaldosteronism in cats has many similarities with "idiopathic" primary hyperaldosteronism in humans. The condition is associated with progressive renal disease, which may in part be due to the often incompletely suppressed plasma renin activity.

References


Back Pain in the Horse
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Horses presenting for back pain will often present for owner complaints of poor or reduced performance manifested as loss of hindlimb propulsion, disunited canter, improper collection, etc. Back pain in the horse can be due to primary or secondary problems. Primary causes of back pain in the horse include: Impinging or over-riding dorsal spinous processes, osteoarthritis of thoracolumbar facets, stress fracture of thoracolumbar facets, lumbosacral intervertebral disc, lumbosacral muscle injury, supraspinatus ligament injury. Secondary causes of back pain include: concurrent lameness, incorrect training, poorly skilled/balanced rider, and poorly fit tack for horse and/or rider. The majority of horses with thoracolumbar pain (~70%) have concurrent lameness. Horses with thoracolumbar disease will have poor epaxial and hindlimb muscular development on physical examination. Limited flexibility of the thoracolumbar spine will be observed when horses are asked to perform dynamic mobilization exercises (i.e. carrot stretches). Palpation of the back during physical examination, should be performed consistently every time to establish an internal scale of the clinician’s ability to produce a response with similar digital pressure. If tension, pain or spasm is elicited during a consistent, repeatable digital exam of the back than thoracolumbar pain is further established. The horse should then be evaluated during ambulation. A horse with back pain will exhibit a stiff gait with poor hind limb propulsion. Concurrent lameness may be evaluated, with concurrent hindlimb lameness more common than forelimb. Complaint and/or observation of “saddle slip” can be seen in horses with concurrent lameness. If a concurrent lameness is noted, diagnostic analgesia should be performed to eliminate the lameness. After addressing any concurrent lameness, evaluation of the thoracolumbar spine should be performed. The thoracolumbar spine can be investigated with ultrasound to evaluate epaxial muscle asymmetry and the ultrasonographic margins of the thoracolumbar facets +/- the sacroiliac joint. Radiographs of the thoracolumbar spine can also be obtained. Both lateral and lateral oblique radiographic projections should be obtained. Typically thoracolumbar facet disease and impinging dorsal spinous processes occur in the caudal thoracic and cranial lumbar spine. These disease processes often occur concurrently. Nuclear scintigraphy can also be performed to evaluate the thoracolumbar spine. Impinging dorsal spinous processes can be treated medically with injection of corticosteroids under radiographic guidance, but reoccurrence of back pain is common. Therefore surgical management may be pursued, including desmotomy of the interspinous ligament and/or ostectomy of the dorsal spinous processes. Osteoarthritis of the thoracolumbar facets can be managed with ultrasound guided injection of the thoracolumbar facets. Other adjunctive treatments can assist in rehabilitation of the thoracolumbar spine.

References
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**Introduction to regenerative medicine**

Regenerative medicine is a rapidly developing field of translational biomedicine focused on stimulating or replacing the body’s own intrinsic capacity for repair and/or regeneration of damaged, malfunctioning tissues. Scientists and clinicians working within this field of medicine are focused on in vitro manipulation of cells, cellular signals (growth factors, cytokines, chemokines), and extracellular matrices for in vivo implantation and directed tissue regeneration with the goal of restored tissue function. These agents are typically derived from donor tissues or blood. Blood derived products, including autologous conditioned serum (Interleukin-1 receptor antagonist protein- IRAP), autologous protein solution, autologous conditioned plasma (also known as platelet rich plasma) and other platelet derived products (platelet lysate, platelet rich fibrin) produce a “soup” of cellular signals that are used to stimulate the healing processes of resident cells in the local wound environment. These products are available to the veterinary market using commercially available kits and centrifuges for point of care processing. However, these products are primarily used in the veterinary market for treatment of musculoskeletal conditions, but will not be the focus of these proceedings, but deserve mention. Tissue derived products include cells (progenitor and stem/stromal cells) and decellularized extracellular matrices for cellular engraftment. Cellular therapy, particularly stem cell therapy, initially gained attention for treatment of musculoskeletal disease, but recently has rapidly gained attention in the field of regenerative medicine for treatment of immune-mediated, inflammatory, and cancerous disease processes.

**Modulation of the wound environment**

The immune system plays a pivotal role in tissue repair and regeneration. After acute injury, microvascular damage results in increased vasopermability and hemostasis. Stressed or necrotic cells and diseased extracellular matrix along with pathogens within the wound release numerous danger signals (danger associated molecular patters-DAMPS and pathogen associated molecular patterns- PAMPS) that activate chemotaxis of cells of the innate immune system (neutrophils, monocytes, and macrophages) to the wound’s microenvironment. Neutrophil infiltration of the wound occurs first resulting in decontamination of the wound and recruitment of other immune and effector cells. Blood and tissue resident macrophages are recruited to the wound and differentiate into a pro-inflammatory phenotype in the early acute inflammatory phase to assist neutrophils with cleanup of the local microenvironment. During this clean up process, necrotic neutrophils accumulate and are phagocytosed by pro-inflammatory macrophages. This causes a phenotypic switch of the macrophages to a more anti-inflammatory phenotype in the late stage of the acute inflammatory response. The macrophage plays a critical role in the process of wound healing and can be both a blessing and a curse. Depletion of macrophages within the wound results in delayed wound healing and persistent, destructive neutrophilic infiltration. These anti-inflammatory macrophages secrete factors that stimulate cellular recruitment and proliferation of fibroblasts, myofibroblasts, and endothelial cells during the proliferative phase of wound healing. During this stage of wound healing fibroblasts lay down extracellular matrix while and endothelial cells stimulate angiogenesis. This physiologic fibrotic matrix is remodeled overtime through delicate cellular interactions and signals. MSCs play an integral role in regulating these intricate mechanisms of wound healing to ensure adequate tissue repair.

**Mesenchymal stem cells (MSCs)**

In large animal species, tissue derived cellular therapeutics are most commonly obtained from adipose or bone marrow. These tissues are harvested and processed to concentrate the mononuclear cell population, which can be used in a point of care manner as a cellular concentrate or expanded in culture. These cellular concentrates including, bone marrow aspirate concentrate (BMAC) and adipose derived stromal vascular fraction (ADSVF), contain a heterogenous population of hematopoietic stem, progenitor cells, endothelial cells, erythrocytes, fibroblasts, lymphocytes, monocytes/macrophages, and pericytes and limited mesenchymal stromal cells. Use of these cellular concentrates in large animals has primarily been limited to musculoskeletal related injury, but its use in modulating inflammatory conditions in lab animals has shown promise. Culture expansion of this mixed mononuclear cell population results in a less heterogenous population of plastic adherent, fibroblast-like cells capable of self-renewal deemed mesenchymal stem/stromal cells (MSCs). MSCs are derived from pericytes or the stroma of the perivascular niche of vascularized mesodermal tissues of fetal or adult origin. MSCs in large animals have been cultured from hematopoietic tissues, adipose, synovial tissues, skin, dental tissues, lung, genital tissues (endometrium), and gestational tissues (placenta and umbilical cord). Standardization of research and scientific laboratory MSC culture methodology is lacking and culture methodology should be tailored to the tissue from which the MSC was derived and the therapeutic need for the MSCs. For example, when clinicians are trying to restore denuded articular cartilage is it better to implant naive MSC or MSCs that have been pre-differentiated toward the chondrogenic lineage? Is it better to use MSCs obtained from synovial or articular tissues vs. non-joint
related tissues? Given the varied phenotypes among MSCs it is important to establish criteria for identifying the MSC phenotype that is being studied, so researchers are comparing apples to apples so to speak rather than apples to oranges. In 2006, the International Society of Cellular Therapy (ISCT) defined the following minimum criteria for definition of an MSC obtained from human bone marrow: Adherence to plastic, specific surface antigen expression (CD105\textsuperscript{hi}, CD73\textsuperscript{hi}, CD90\textsuperscript{lo}, CD45\textsuperscript{lo}, CD34\textsuperscript{lo}, CD14\textsuperscript{lo}, CD11b\textsuperscript{lo}, CD79a\textsuperscript{lo}, or CD19\textsuperscript{lo} and MHC II\textsuperscript{lo}), multipotent differentiation (In vitro differentiation into osteoblasts, chondroblasts, and adipocytes). Since this statement, the ICST and the International Federation of Adipose Therapeutics and Sciences has defined minimum criteria for adipose derived MSCs with differences in surface antigen expression (CD13\textsuperscript{hi}, CD73\textsuperscript{hi}, CD90\textsuperscript{lo}, CD36\textsuperscript{lo}, CD11b\textsuperscript{lo}, CD45\textsuperscript{lo}, and CD106\textsuperscript{lo}). Interestingly, neither position statement discusses the immunomodulatory functions of MSCs within their minimum criteria, though, ISCT has proposed several key areas of refinement for standardization for defining the immunomodulatory phenotype of MSCs. Full characterization of MSCs in the veterinary literature is difficult due to limited availability of species-specific antibodies and reagents. Surface antigen expression of Equine BMSCs has been studied and proposed phenotype signature would include MHCII\textsuperscript{lo}, CD44\textsuperscript{hi}, CD29\textsuperscript{hi}, CD90\textsuperscript{lo}, CD45RB\textsuperscript{hi}, CD11a/CD18\textsuperscript{lo} expression, however, consensus in the literature is lacking. For Equine ADMSCs the phenotype signature would include CD29\textsuperscript{hi}, CD105\textsuperscript{hi}, CD44\textsuperscript{hi}, CD90\textsuperscript{lo}, CD140b\textsuperscript{lo}, CD164\textsuperscript{hi}, CD34\textsuperscript{lo}, CD45\textsuperscript{lo}, CD73\textsuperscript{lo}, MHCII\textsuperscript{lo}. Various surface antigen markers have been reported and/or confirmed for bovine MSCs, but due to the limited number of studies, recommendations for surface antigen expression are not defined.

Regenerative and reparative qualities
MSCs were first investigated therapeutically for their “stem” properties, particularly their ability to differentiate into different tissues of mesodermal lineage under certain in vitro conditions. Evidence of naive MSC differentiation following implantation is limited. In fact, demonstration of MSC engraftment and retention at sites of implantation has been disappointing. Despite these findings, however, evidence for improved research and clinical outcomes following MSC therapy is mounting. This has led to the “hit and run” theory, in which scientists believe MSCs are introduced to the environmental niche, they react to this niche via cell-to-cell and paracrine signals, adjust their phenotype resulting in the release of niche appropriate cytokines, chemokines, extracellular vesicles and growth factors. The MSC directly and indirectly interacts temporarily with the environmental niche to establish an environment supportive of tissue repair and regeneration through modulation of site-specific immune and progenitor cells. Stimulation and modulation of progenitor cells is exerted via trophic factors with four main functions: inhibition of ischemia-related apoptosis, inhibition of fibrosis, promotion of angiogenesis and progenitor mitogen activation. These factors include transforming growth factor β (TGFβ), hepatocyte growth factor (HGF), epidermal growth factor (EGF), vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), platelet derived growth factor (PDGF), insulin-like growth factor (IGF-1), stromal derived factor-1 (SDF-1), and angiopoietin-1.

Adipose vs. Bone Marrow: Adipose is arguably the most popular adult tissue-derived source of MSCs. Adipose has a higher MSC yield within the stromal vascular fraction (up to 200x greater) compared to bone marrow. Certain aspects with fat collection include appearance of cosmetic blemish and/or inadequate exposure to the tailhead for peri-operative collection when horse is placed in dorsal recumbency can make bone marrow a more appealing source for MSC collection. There is evidence to support that there is enhanced tenogenesis and chondrogenesis with BMSCs. However, there are some that feel fetal derived MSCs and/or adipose derived MSCs have enhanced immunomodulatory abilities. However, there continues to be a strong debate for and against these tissues as to which is the “best” tissue for treatment of various musculoskeletal injuries. Therefore, it remains an owner and/or clinician preference decision.

Donor matters: There is evidence to suggest that for the most robust, optimal stem cell population a young, healthy donor should be used for MSC harvest. For example, MSCs obtained from donors with advanced degenerative joint disease have MSCs with reduced chondrogenic capacity compared to their younger, disease free counter parts. Another important patient-related factor to consider in equine medicine is the metabolic status of the horse. Horses diagnosed with Equine Metabolic Syndrome (EMS) also have impaired chondrogenesis due to deterioration of the mitochondrial network and are not suitable donors for MSC therapeutics. However, in equine medicine, horses that present for regenerative therapeutics of musculoskeletal injuries are typically middle-aged horses with chronic or acute on chronic injuries. For this reason as well as other reasons mentioned below, allogeneic use of MSCs (MSCs obtained from young, healthy horse A and placed in older, diseased horse B) has been evaluated in the horse. Allogeneic use would allow practitioners to rapidly obtain an appropriate “dose” of MSCs avoiding long culture expansion times. Additionally, these MSC lines would have undergone appropriate MSC characterization (haplotype identification, surface antigen expression analysis and tri-lineage differentiation confirming their “stem” properties) and function tests (measurement of their chondrogenic capacity, tenogenic capacity, and/or immunomodulatory functions). MSCs were also previously thought to be immunoprivileged due to their absence of MHC II and costimulatory molecule (CD 40, CD 80, CD 86) expression. In large animal species, allogeneic MSCs have been investigated from a safety and/or efficacy perspective following intravenous, intra-arterial, intradermal, intraleisional, intra-articular, and intra-ocular. The majority of these studies conclude that allogeneic administration of MSCs appears to be safe, however, most of these studies did not characterize the MHC haplotype of the donor and recipient making it difficult to know if these injections were truly allogeneic or syngeneic. This is important because Equine MHC II expression has been demonstrated and proven to be
dynamic and its expression variable between subject, cell harvests within subject, passage number, and culture conditions. In fact, humoral and cell-mediated immunity has been documented following allogeneic administration in humans and documentation in large animals (horses) is accumulating. Owens, et. al. retrospectively evaluated the serum of horses injected with allogeneic MSCs and found that 37% of horses had developed anti-MSC antibodies. In vitro, equine MHC-mismatched MSCs expressing MHCII activated T cells, causing significant increases in T cell proliferation compared to MHC-matched MHC class II negative and MHC matched MSCs provoking an immune response. Furthermore, this research group demonstrated cytotoxicity of MSCs when exposed to MHC II mismatched recipient sera. These findings have led to recommendations for haplotype matching prior to allogeneic administration. In vivo, MHC-mismatched allogeneic cells do not persist in the tissues as long as allogeneic MHC-matched or autologous MSCs, yet allogeneic MHC-mismatched MSCs do persist in tissues longer than other allogeneic cells. This would suggest that these cells are not immunoprivileged, but may be capable of immune evasion long enough to exert their immunomodulatory effects on the environmental niche. The clinical relevance of these alloantibodies in regards to their effect on MSC efficacy is unknown and warrants further investigation.

MSC administration: MSCs can be administered systemically (usually implant within the lung tissue), regionally (regional limb perfusion, intra-articular injection) or locally (intra-lesional injection in tendon or cartilage defect). When injecting MSCs via regional limb perfusion, the practitioner must keep in mind the cellular size of MSCs and potential for thrombus formation. It is recommended that the cellular dosage be kept low (< 20 million cells) and that an appropriate volume of infusion (30 mls distal limb, 60 mls upper limb) be used for cellular suspension. Perfusion should be administered slowly over 5-10 minutes to allow adequate cellular diffusion. Arterial administration has been shown to produce better MSC distribution, but potential for thrombus formation remains a concern. Therefore, for musculoskeletal injuries, MSCs are often administered locally. For intra-articular administration MSCs are administered at a dose of 10-20 million/joint. The cellular suspension used for intra-articular administration is important and can affect cellular viability. Suspension of MSCs in hyaluronic acid has shown to have little to no effect on MSC viability. However, suspension of MSCs in platelet rich plasma (PRP) can affect the immediate cellular viability of MSCs if the pH (acidic pH) of the cellular suspension is not buffered prior to clinical application. Antibiotics commonly used in intra-articular injections (Amikacin and Gentamicin) can severely affect MSC viability and should not be administered with MSCs. Additionally, co-injection of MSCs with commonly used corticosteroids (methylprednisolone acetate and triamcinolone) will severely affect MSC viability and capacity for osteogenic differentiation and therefore should be avoided. Local anesthetics may be administered regionally, far from collection and/or administration site of MSCs, but their use concurrently with MSCs should also be avoided. Systemic administration of sedatives will not impact MSC viability and therefore sedation of the patient for collection and/or administration appears safe. High doses of NSAIDs (flunixin and phenylbutazone) can affect MSC viability and capacity for osteogenic differentiation and therefore should be avoided during collection and administration. MSCs are administered by the author locally under standing sedation without NSAID administration for these reasons. If applicable, a hospital-style bandage is placed for 24 hours and the horse remains on stall rest with handwalking (5 minutes twice daily). The hospital bandage is removed after 24 hours and a stable bandage is placed by the owner, with directions to re-adjust/wrap the bandage every 12-24 hours for 7-10 days. If local inflammation (focal edema, swelling) is noted by the client, NSAIDs are administered at that time.

**Immunomodulation by MSCs**

MSCs have intense immunomodulatory functions, in fact they have been referred to as the “Guardians of Inflammation”. Much like a guardian is called to duty when alerted to danger, MSCs must be alerted to danger and activated through inflammatory cytokine priming or ligation of their toll like receptors (TLRs) with danger or pathogen associated molecular patterns (DAMPs and PAMPs). Following activation MSCs modulate the microenvironment through direct cell-cell contact and/or secretion of numerous anti-inflammatory cytokines, chemokines, and adhesion molecules, modulating the surrounding environmental niche toward a more regulatory, immune tolerant environment. Secreted immunomodulatory factors include prostaglandin E2 (PGE2), indoleamine 2, 3 dioxygenase (IDO), nitric oxide (NO), IL-6, TGFβ1, HGF, and tumor necrosis factor inducible gene 6 protein (TSG6). The mediators by which MSCs use to exert their immunomodulatory effects is different between species, tissue source, and targeted immune effector cells. For example, Equine MSCs derived from hematopoietic tissues will secrete NO to induce T cell suppression, whereas MSCs from solid tissue will not secrete NO.22 Monkey, pig, and human primarily useIDO for T cell suppression whereas rat, mice, rabbit, and hamster NO23 In large animals, horses primarily use PGE2 to modulate the inflammatory environment. Additionally, MSCs secrete extracellular vesicles that carry gene regulatory proteins (mRNA and microRNA) and mitochondria that can exert immunosuppressive effects on the target cells.

This activation of MSCs to induce immunoregulation is termed cytokine priming. In vitro, activation of MSCs seems to require IFNγ, but other pro-inflammatory cytokines such as TNFα and IL-1β act synergistically with IFNγ. MSCs are plastic and activation is a dynamic process. The inflammatory stimulus, concentration, and duration of exposure to this inflammatory stimulus is important in MSC activation. For example, equine MSCs exposed to 20% allogeneic inflammatory synovial fluid did not alter their immunomodulatory gene expression profile, but exposure to high concentrations (50 ng/ml) of IFNγ and TNFα resulted in significant
upregulation of immunomodulatory genes (COX-2, NO, IDO, and IL-6). However, MSC priming with these high concentrations of IFNγ and TNFα reduced MSC viability and impaired trilineage differentiation. When activated with lower concentrations of IFNγ and TNFα for a shorter period of time (12 hours) upregulation of COX-2, NO, IDO, IL-6 was maintained. Additionally, MSC viability and multipotent differentiation was unaffected. Cytokine priming results in a temporary change in the MSC immunomodulatory phenotype, with equine MSCs no longer showing upregulation of COX-2, NO, IDO, and IL-6 expression 7 days after cytokine priming.

It is important to note that under certain conditions, MSCs can actually become pro-inflammatory and act like antigen presenting cells. MSCs can also lose their ability to be activated and induce immunosuppression, this occurs in vitro in MSCs at high passage (long culture times), long cryopreservation times, and lack of post thaw equilibration.

MSC administration for immune-mediated diseases of the equine patient are currently under investigation. It is important that the practitioner understand their potential use, should therapeutic use of MSCs for diseases such as recurrent airway obstruction, semen mediated endometritis, etc. become more prevalent.

References are available upon request at lhb0021@auburn.edu
Bone marrow derived mesenchymal stem cells (BMSCs) and adipose derived mesenchymal stem cells (AdMSCs) have gained popularity as a biological reparative strategy as well as an anti-inflammatory/immunomodulatory therapy for common musculoskeletal injuries of the equid. Proper collection and shipment of bone marrow and/or adipose can affect the output of bone marrow and adipose culture expansion equating to proper BMSC and AdMSC isolation.

Prior to collection of bone marrow or adipose it is important that the client identify a commercially available laboratory for shipment and processing of cells for concentration or expansion. Commercial kits are available for stall side production of bone marrow aspirate concentrate (BMAC adipose cell concentrate (adipose derived stromal vascular fraction) and/or culture expanded MSCs a commercial laboratory will be needed. Most of these laboratories will have a kit that will be shipped to the practitioner with instructions for collection and shipment of bone marrow or adipose. Some of the kits will require additional samples be taken such as peripheral blood or more bone marrow for processing of autologous serum or bone marrow concentrate as a cellular suspension for cell delivery when the product is returned to the client. Cellular concentrate processing will take approximately 48 hours, but culture expansion will often take 3-4 weeks to obtain an appropriate cell dose (10-20 million cells). The practitioner needs to be aware of these time frames to keep the client informed of the process.

**Bone marrow aspiration from the sternum**

The sternum is the most commonly used aspiration site due to the age of the patients commonly treated, but sternebral aspiration is not without complications including iatrogenic cardiac puncture, induced cardiac dysrhythmias, or pneumopericardium. Therefore, it is important to be comfortable with sternebral anatomy in relation to the cardiac silhouette and to be cautious when performing sternebral aspiration.

The patient is sedated with an α2 agonist (detomidine hydrochloride 0.1mg/kg) with or without the use of an opiate (butorphanol tartrate 0.1 mg/kg) depending on the temperament of the patient. A 10 x 10 cm2 area over the sternum is clipped and aseptically prepared. Sites for aspiration can be identified using anatomical landmarks and/or ultrasound guidance. The safest sternebrae for aspiration has been identified as the 5th sternebrae. This sternebrae has the greatest dorsoventral depth of the caudal sternebrae and is located cranial to the cardiac apex. This sternebrae is found just caudal to the point of the elbow. The 4th and 6th sternebrae are also accessible for sternebral aspiration with the 4th sternebra located between the front limbs and the 6th sternebra located cranial to the cartilaginous xiphisternum partially fused to the 7th sternebrae. Using a 7.5-10 mHz ultrasound probe the “V” shaped defect in the bony contour of the sternum between the various intersternebral spaces (between the 4th, 5th, and 6th sternebrae) are identified and marked by either clipping the hair or marking the adjacent skin with a sterile marker pen or white out pen. A rough prep is applied to the sternum and two sites for aspiration are blocked using 5 mls of Lidocaine hydrochloride between the marks used to identify the intersternebral spaces. The first 5 mls of bone marrow is the most rich in stem cells and increasing the volume of bone marrow obtained from that sternebrae only dilutes the stem cell population, therefore, it is the preference of the author to aspirate from a second sternebrae to increase the stem cell yield during harvest. The clipped area is aseptically prepared. A stab incision is made with a #15 scalpel blade directly on midline through the site(s) anesthetized for aspiration. An 11 gauge jamshidi bone marrow biopsy needle is used for aspiration. The jamshidi needle is placed through the stab incision and advanced approximately 2 cm through the soft tissues covering the sternum until the cortical bony surface of the sternum is encountered. The needle is then twisted 180° from left to right while gently pushing the jamshidi proximally. The index finger is placed along the shaft of the jamshidi to guide depth of insertion and ensure that the jamshidi is not inserted more than 1-2 cm. When the cortex has been penetrated and the jamshidi enters the medullary cavity there will be a slight change in resistance, but if this change is resistance has not been appreciated it is recommend to remove the stylet and attach a syringe pre-loaded with anti-coagulant after the jamshidi has been advanced 1-2 cm. If bone marrow is not obtained and the aspiration site was identified using anatomical landmarks and palpation further advancement of the jamshidi should be performed with caution and should not exceed more than 1 cm more of proximal advancement. If bone marrow is still not obtained it is likely that the site of aspiration is within a cartilaginous intersternebral space and further advancement may result in thoracic and/or cardiac puncture. Therefore, the jamshidi needle should be removed and advanced cranially or caudally within the same stab incision approximately 1-2 cm. If proper insertion within the medulla of the 4th, 5th, and 6th sternebrae, bone, bone marrow is aspirated into two 35 ml leur-lock syringes pre-loaded with 2.5 mls Heparin sodium (250-500 mls Heparin sodium/ml of bone marrow) or containing the anti-coagulant and anti-coagulant volume of choice of the company that is used. When first aspirating bone marrow from the sternum horses will “tent” the thorax proximally. The aspirated bone marrow will appear foamy and/or oily. The syringes should be gently inverted to ensure proper mixing of the anti-coagulant with the bone marrow to prevent clotting. If using a commercial company, the practitioner...
should refer to the company’s shipping instructions to ensure adequate cellular viability. The aspiration sites are not closed, but triple antibiotic ointment is applied to the site of aspiration. It is common for the site to drip blood for 10-20 minutes following aspiration, much like that of an abdominocentesis site.

**Bone marrow aspiration of the ilium**

The ilium is preferred in young horses (<5 years of age) for which BMSCs. The ilium is not preferred in older horses because as the patient matures fat accumulates within the wing of the ilium making aspiration difficult. The ilium also has a thicker bony cortex making penetration and aspiration difficult. Additionally, ilial aspirates have been shown to have significantly lower yields of BMSCs/ml compared to sternebral aspirates in middle-aged horses, but similar BMSC yields have been obtained in younger (2-5 year old) horses.

For ilial aspiration, the patient should be sedated as described above. The tuber coxae of both ilia are shaved and a prepared roughly with antiseptics. The subcutaneous tissue is infiltrated with 3-5 mls lidocaine hydrochloride directly over the ventral 2/3 of the iliac wing. The ilium is aseptically prepared. An 11 gauge jamshidi needle is used for biopsy. A stab incision is made with a #15 scalpel blade in the center of the anesthetized aspiration site. The jamshidi needle is inserted 5-8 cm in a slight caudoventral direction toward the opposite coxofemoral joint perpendicular to the skin. The jamshidi needle is inserted in the same manner as described for sternebral aspiration. The stylet of the jamshidi is removed and a leur-lock syringe preloaded with anticoagulant as described above is attached and bone marrow is aspirated. Aspiration of bone marrow from the ilium requires greater negative pressure than sternebral aspiration. The aspiration site is not closed and triple antibiotic ointment is applied to the aspiration site.

Adipose Collection: In horses, the most readily accessible site for fat collection is adjacent to the tail head in horses. The patient is sedated as described above for bone marrow collection. A 15 x 15 cm2 site is clipped approximately 6-8 cm lateral to the tailhead, just proximal to the dorsal aspect of the tail head. The site is aseptically prepared. 20 mls of Lidocain Hydrochloride is injected subcutaneously in an upside down “U” pattern surrounding the proposed site for placement of the linear skin incision. The site is again prepared aseptically. The site is draped. A 6-8 cm linear skin incision is made in the middle of the upside down “U”. Blunt dissection is performed with curved mayo scissors to separate the skin and the fat for approximately 2 cm from the incised skin margin. Fat is then collected using mayo scissors and a combination of sharp and blunt techniques. Approximately 20 grams of fat should be collected and placed into the commercially provided vial. The incision is lavaged with 60 mls sterile saline and closed in two layers. Broad spectrum antimicrobials are not used perioperatively by the author, but is at the discretion of the practitioner. The sutures should be removed in 14 days.

Lipoaspiration can be used as a technique for fat collection. A stab incision is made just below the tail head. A commercially available lipoaspiration cannula is gently directed under the skin, subcutaneously and aspiration is applied to obtain lipoaspirate. The lipoaspirate is then processed according to the kit or commercially laboratory used.

References are available upon request from author at lhb0021@auburn.edu
Healing of tendons

Tendon healing is divided into three phases including the acute inflammatory phase, the proliferative phase, and the remodeling phase. The acute inflammatory phase consists of clean up (phagocytosis) of damaged tissue leading to declaration of the extent of the tendon/ligament fiber loss. The proliferative phase is when a provisional matrix is laid down on which fibroblast and tissue resident progenitor cells migrate and begin their production of an disorganized collagenous network. During the remodeling phase this disorganized collagen network is remodeled to establish at best <80% of the original tensile strength of the diseased tendon.

Conservative management of tendon injuries focus on reduction of inflammation during the acute inflammatory phase with administration of non-steroidal anti-inflammatory phases +/- cold therapy and compression bandaging. In addition the horse is rested to ensure that the biomechanical integrity of the remaining tendinous fibers are not further affected and to protect the biomechanically fragile reparative matrix. The horses activity is slowly increased to provide subtle biomechanical stimulation to the tissue resident progenitor cells that are laying down new tendon matrix to assist with the proliferative and remodeling phase. Adjunctive therapies have been evaluated to enhance tendon healing by increasing the cellular population within the wound, increasing the cellular communication within the wound (cytokine, chemokine, and growth factor therapy) and/or by enhancing the provisional tendon matrix that is established in the wound environment. Most of these therapies are meant to enhance the QUALITY of the repair tissue to reduce the re-injury rate and do little to speed up the recovery process.

Cellular therapy

Mesenchymal stromal cells (MSCs) are delivered at doses of 20-50 million cells/lesion and the volume of cellular suspension is dependent on clinical judgement. MSCs are typically re-suspended in autologous serum, autologous bone marrow supernatant, or platelet rich plasma for intra-lesional injection. In experimental models of tendonitis, injection of MSCs have been shown to improve the quality and strength of repair. When MSCs were used to treat naturally occurring tendinopathies, a reduction in the re-injury rate was shown when compared to historical controls. There is supportive evidence to show that cellular therapy in the horse can augment the repair process of tendon and/or ligament injury in the horse.

Cellular signaling therapy

Autologous conditioned serum: Autologous conditioned serum (ACS) is obtained after whole blood exposure and incubation to medical grade glass beads that stimulate cellular production of anti-inflammatory cytokines, including interleukin-1 receptor antagonist protein. This product also contains important growth factors, including insulin-like growth factor (IGF) and transforming growth factor β (TGFβ). These contents are meant to modulate the inflammatory events within the wound environment and promote cellular proliferation and cellular production of extracellular matrix. Most of the evidence for improvement following intra-lesional treatment of ACS is anecdotal. One study in which seventeen horses with naturally occurring SDF tendinopathies were treated with saline or autologous conditioned serum showed temporary improvement in ultrasound and lameness following treatment with ACS.

Platelet rich plasma: Platelet rich plasma (PRP) is derived from the patient’s own blood using commercially available products that concentrate platelets ~2-6 fold over the baseline platelet count via centrifugation or filtration techniques. PRP contains platelets, cells (white blood cells and red blood cells), and numerous growth factors important in cellular recruitment, cellular proliferation, and angiogenesis. Cell and growth factor concentration differs due to patient related factors and variations in the processing of commercially available products. A case-controlled study evaluating National Hunt horses with naturally occurring SDF tendinopathy treated with bar firing, tendon splitting, platelet rich plasma, or tendon splitting in combination with bar firing. No significant effect of treatment was found for return to racing when compared to controlled exercise alone. A placebo controlled clinical trial of 20 horses with naturally occurring SDF tendinopathy treated with PRP or saline showed an earlier reduction in lameness scores and improved...
repair tissue organization based on B mode ultrasonography. Again, it seems that PRP helps in augmentation of the healing process in tendinous injuries.

**Scaffolding agents**

Cross-linking agents: Novobrace™ (Iridoid genipin) is a commercially available cross-linking agent that is meant to reduce return to work times by 50%, but no controlled clinical trials have been performed to support this reported data. Iridoid genipin is a natural cross-linking protein extracted from the gardenia plant that forms covalent cross-links. A pilot study evaluating the safety of this product, injected 1 ml IG into normal SDFT of 6 horses. Horses had mild swelling and transient lameness following injection, increased linear collagen was present at 30 days within the tendon where the product was implanted in normal tendon. Anecdotal concerns have been raised concerning product interaction with other intra-lesional therapies and further research is warranted into the safety and efficacy of the product.

**Delivery**

During the acute stage of injury (heat, swelling, pain) the horse should be stall rested and administered NSAIDs. Two to three weeks later, an ultrasonographic examination is performed and injury location and extent of injury is documented. Treatment is instituted based on severity of the lesion and includes: conservative therapy (stall rest and controlled rehabilitation) with or without the use of external adjunctive treatments (extracorporeal shockwave or radial pressure wave therapy, therapeutic ultrasound, therapeutic laser) and/or intralesional therapeutics. Therapeutic agents for tendinous injuries are usually delivered intra-lesionally under ultrasound guidance, but some can be administered via regional limb perfusion. A recent paper, by Scharf et. al. called into question the accuracy of intra-lesional administration of MSCs under ultrasound guidance and leakage of cellular product from the site into peritendinous tissues. Following injection, a bandage is applied for 24 hours and then supportive bandaging is continued or discontinued at the clinicians discretion. NSAIDs are administered at the discretion of the clinician, there is some evidence to recommend that NSAID administration be limited due to its effects on various biological agents. NSAIDs are typically not administered by the author, unless local reaction is noted by the owner (heat, swelling, and lameness). Often times the intra-lesional therapy is dictated by the availability of the product to the practitioner and/or the owner’s finances. Horses are re-evaluated every 6-8 weeks for improvement in lameness and improvement in the ultrasound image.

There are numerous intra-lesional therapeutics available to aid the repair process in the horse, but the scientific rigor to which these products have been tested is limited.

**References**

Available upon request from Lindsey Boone at lhb0021@auburn.edu
Standing Enucleation in the Horse
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For standing enucleation, the horse should be restrained in a clean, dry environment that is in a low traffic area of the hospital. An area of the hospital that has been designated for standing surgical procedures is recommended if possible. Tetanus prophylaxis is administered if status cannot be appropriately confirmed. Pre-operative antibiotics (Procaine penicillin 22,000 IU/kg IM) and non-steroidal anti-inflammatories are administered (Flunixin Meglumine 1.1 mg/kg IV). The horse is sedated with detomidine hydrochloride (0.01 mg/kg IV) and butorphanol tartrate (0.03 mg/kg IV). Standing sedation is maintained with intermittent bolus administration of detomidine or a continuous rate infusion of detomidine hydrochloride. The periorbital region surrounding the diseased eye is clipped and aseptically prepared with 1% povidine-iodine solution. A retrobulbar block is performed by inserting a 22 gauge, 7 cm spinal needle just caudal to the dorsal rim of the orbital fossa advancing the needle perpendicular to the orbital fossa until the retrobulbar cone is penetrated. When this occurs the eye will move show slight dorsal movement. Ten -12 milliliters of mepivicaine is administered slowly. The eye will become exophthalmic with proper placement of local anesthetic. Proper placement of the retrobulbar block is imperative to assist with reduced patient motion during surgical removal. The frontal nerve and auriculopalpebral nerves are also blocked. Further local anesthetic is administered in a subcutaneous ring block surrounding palpebrae (This blocks the infratrochlear, lacrimal, and zygomatic nerves). The nasolacrimal duct and conjunctival sac are flushed with 1% Povidine-iodine solution. The eyelids are sutured closed in a simple continuous pattern, the tags are left long and tied together to form a handle to assist with gentle retraction of the orbital sac during dissection. The surgical site is aseptically prepared following eyelid closure. The author prefers to clamp sterile surgical hand towels along the nose and cheek portions of a nylon halter, but more extensive draping is not performed. A full thickness elliptical skin incision is made 3-5 mm from the eyelid margins. Dissection using blunt and sharp dissection with mayo scissors. The medial and lateral canthal ligaments are sharply transected and blunt dissection along the sclera and extra-ocular muscles is performed. Transection of the extraocular muscles is made close to the tendinous insertion on the globe. If the conjunctival sac is penetrated during dissection, the conjunctival sac at the site of penetration should be closed to prevent further contamination. After circumferential dissection of the orbital sac, a large, curved hemostatic forcep is placed across the optic nerve. Curved mayo scissors are used to transect the optic pedicle distal to the clamp. After removal of the eye within the conjunctival sac the socket is gently flushed with sterile saline solution. The clamp is kept in place during subcutaneous closure and is removed when the final subcutaneous tissue bites are taken, but not tied. The subcutaneous tissues are closed with an absorbable suture in a simple continuous pattern. The clamp is removed and the subcutaneous tissues are closed. The dermis is closed with an absorbable suture is a simple continuous intra-dermal suture pattern. Following closure, the incision is covered with a non-adhesive sterile gauze that is backed by several sterile gauze held in place by an adhesive bandage that is kept in place for 24-48 hours. Horses are treated with non-steroidal anti-inflammatories for 3-5 days following surgery.

Closure of the orbital rim with a non-absorbable, mesh-like implant in an attempt to prevent sinking of the soft tissues within the orbit has not been shown to improve cosmetic appearance and the author does not augment the closure in this manner. An implant is only placed by the author if the eye was removed for reasons other than infection or neoplasia and only when the enucleation is performed in a surgical suite under general anesthesia. Placement of an ocular implant increases the risk of surgical site infection post-enucleation, therefore, these should only be placed under strict aseptic conditions. In addition, performing standing enucleation also increases the risk of surgical site infection and again strict aseptic conditions must be maintained during the surgical procedure. Most horses (~85%) will return to work following enucleation despite acute or chronic loss of vision.

References
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Proximal suspensory desmitis is a common cause of reduced performance and lameness (unilateral or bilateral) in horses. Owners often complain of a loss of impulsion, stiffness, resistant, and/or evasive behavior.

Anatomy of the proximal suspensory ligament: The proximal suspensory ligament of the forelimb originates from the thick fascia present along the palmar aspect of the third carpal and metacarpal bones. The proximal portion is bilobed with the medial lobe being thinner and wider than the lateral lobe. The proximal portion contains muscle, fat, and ligamentous fibers, making diagnostic imaging of the proximal suspensory ligament difficult necessitating a multimodal diagnostic approach (i.e. blocking of the lameness and ultrasonographic abnormalities). Distribution of fat and muscle is not necessarily symmetric and can vary between limbs. The forelimb proximal suspensory ligament in innervated by the deep branch of the lateral palmar nerve.

The proximal suspensory ligament of the hindlimb is triangular in shape with the thickest portion of the ligament adjacent to the prominent fourth metatarsal bone. Distribution of fat and muscle is not necessarily symmetric and can vary between limbs as well. The hindlimb proximal suspensory ligament is innervated by the deep branch of the lateral plantar nerve originating from the tibial nerve.

Diagnosis: Diagnosis of proximal suspensory ligament desmitis requires a multimodal approach. Physical examination is performed by the practitioner, paying special attention to the horses conformation. A horse with straight hock (> 120° between antebrachium and metacarpus/tarsus) and or hyperextension of the metatarsal/carpophalangeal joint are more likely to be afflicted with abnormalities of the proximal suspensory ligament. The horse is watched in hand on straight lines and on the lunge in hard and soft ground. Ambulatory evaluation under saddle can augment the ambulatory exam in hand and is recommended. If a lameness is detected, the distal limb should be blocked (low-four point block) prior to blocking the proximal suspensory ligament. If a low-four point nerve block is not performed to rule out lameness of the distal limb, false positives will occur due to the close association of the lateral palmar/plantar nerves that innervate the distal limb.

Blocking the deep branch of the lateral palmar nerve: The block is performed with the limb weight bearing. The limb is aseptically prepared. A longitudinal groove along the medial aspect of the accessory carpal bone dorsal to insertion of the flexor retinaculum within the distal 1/3 of the accessory carpal bone is palpated. A 1.5 cm 25 gauge needle is inserted in a medial to lateral direction until contact with the accessory carpal bone is achieved and a low volume (<3 mls) of local anesthetic is deposited. The anesthetic agent should inject easily. The horse is evaluated every 5 minutes for a total of 30 minutes. Penetration of the carpal sheath can occur with this block. Therefore, it is the author’s preference to use sterile prep and sterile gloves when performing this block.

Blocking the deep branch of the lateral plantar nerve: The block is performed with the distal limb in flexion. The limb is aseptically prepared. 1.5 cm 25 gauge needle is directed 15 mm distal to the head of the fourth metatarsus along the axial face of the fourth metatarsus until the needle is buried. A love volume (< 3 mls) of local anesthetic are administered. The anesthetic should inject easily. The horse is evaluated every 5 minutes for a total of 30 minutes. Penetration of the tarsal sheath and/or tarsometatarsal joint can occur with this block. Therefore, it is the author’s preference to use sterile prep and sterile gloves when performing this block. Additionally, the possibility of these anatomically structures being inadvertently blocked must be considered if diagnostic imaging does not confirm abnormalities of the proximal suspensory ligament.

Radiography: Radiographs should be obtained to evaluate the travecular bone pattern of the proximal cannon bone for evidence of sclerosis and relationship of the MC/MT II and IV with the suspensory ligament.

Ultrasound: After proper preparation of the limb (clip, clean, cover with alcohol +/- ultrasound gel) the proximal suspensory ligament is evaluated using standard and “off angle” ultrasonographic techniques in both weight bearing and flexed positions. Off angle ultrasonographic techniques assist the practitioner in identifying ligament fibers vs. muscle and/or fat. When the incidence angle of the ultrasound probe is perpendicular to the ligamentous fibers, fibers are echogenic. If this angle is adjusted to “off angle” ligament fibers become echogenic, but muscle and fat remain echogenic. This technique provides good correlation of ultrasonographic images to MRI findings. Abnormalities in size, shape, margin, and echogenicity should be evaluated.

Magnetic Resonance Imaging: MRI evaluation is pursued to fully evaluate the suspensory and provide comprehensive diagnosis. MRI evaluation allows comprehensive evaluation of the periligamentous structures including abnormalities of the hock that could be “blocked” during lameness evaluation.

Treatment: Return to work following conservative management (stall rest and controlled rehabilitation) is considered good in the forelimb, but fair to poor in the hindlimb. Adjunctive therapies include the use of radial or extracorporeal shockwave therapy and/or injection of intra-ligamentous therapies such as bone marrow concentrate (BMAC). Horses with chronic proximal hindlimb suspensory desmitis that have undergone rest and rehabilitation, but fail to return to work may be candidates for fasciotomy and...
neurectomy of the deep branch of the lateral plantar nerve. This procedure has allowed horses with chronic proximal suspensory desmitis to return to work, but those horses with poor confirmation (straight hocks and hyperextended fetlocks) respond poorly to surgical management. Neurectomy does result in neurogenic atrophy of the muscle contained within the suspensory ligament. Long term outcomes > 5 years post-neurectomy have not been extensively reported.

The diagnosis of proximal suspensory ligament desmitis can be challenging and treatment must be tailored to the nature of the injury and the long-term goals of the owner for the horse’s athletic future.

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Coronavirus: Clinical impact

Coronaviral infection has recently been recognized as a cause of colic, lethargy, fever, and diarrhea in horses. Equine coronavirus (ECoV) is a β-coronavirus and has been reported to cause gastrointestinal disease in horses of all ages and is associated with outbreaks of gastrointestinal disease among populations of horses in the United States and other countries.

Following exposure to the virus, which is acquired primarily by environmental contamination and the fecal/oral routes, ECoV infects the small intestine and spreads to the colon, infecting the crypt cells and leading to a malabsorption/maldigestion gastrointestinal disease. The incubation period is reported to be 48-72 hours, and once infected, horses can shed virus for approximately 3-9 days. Reported clinical signs include diarrhea, which is more frequently observed in foals; anorexia; lethargy; and fever. In most cases, the course of the clinical signs is self-limiting; however, deaths have been associated with ECoV infection. There is high morbidity, up to 57% of a closed population affected, and low mortality associated with outbreaks. In several studies of outbreaks, the duration of the outbreak was approximately 3 weeks.

Definitive diagnosis of ECoV is by fecal PCR, serum titers, and/or immunohistochemistry. Clinical findings, as well as hematological changes (particularly leukopenia characterized by neutropenia), support the diagnosis.

There is no targeted therapy for treatment of ECoV. Supportive care including restoration and maintenance of hydration, anti-inflammatory or analgesic therapy (non-steroidal anti-inflammatory drugs), rest, and fecal transfaunation, may be the most beneficial.

Biosecurity: Guiding principles

Rapid recognition of transmissible/infectious diseases and prompt implementation of biosecurity protocols is vital to minimizing spread of disease. Rapid recognition encompasses knowledge of the clinical signs of transmissible infectious diseases, as well the differential diagnoses of infectious etiology for non-specific clinical signs. Additionally, incorporation of the principles of biosecurity in daily practice, whether in a field scenario or hospital, is critical for prevention of transmission of disease. Biosecurity should focus on patient factors, personnel factors, and environmental/fomite management.

Patient factors include vaccination, isolation, and therapeutic intervention. For some transmissible diseases, such as equine herpesvirus, equine influenza, and strangles, vaccination prior to transmission may be a strong preventative strategy for minimizing the outbreak of disease in a population. While vaccination is not a guarantee that horses won’t be infected, it can certainly help attenuate the disease process/viral shedding and may lessen the spread of the disease when other biosecurity measures, such as isolation, are implemented. Currently there are no vaccines available for the prevention of ECoV.

In an effort to prevent disease transmission, or stop the spread of disease in a population, isolation of horses is a necessary step. Frequently, the introduction of new or naïve horses into a population can be a source of disease. Respiratory infectious diseases (commonly equine herpesvirus, equine influenza, and strangles) are often implicated in herd outbreaks. For the viral respiratory diseases, isolation of new or naïve horses for 3-4 weeks (based on the frequency of viral shedding from the nasal secretions) prior to introducing them into the population may decrease the risk of disease transmission. Infectious gastrointestinal disease processes, such as coronaviral infection, salmonellosis, and clostridiosis may result in outbreak scenarios, and horses should be isolated for the duration of shedding of organisms. This is typically based on repeated testing to ensure that the infection is resolved. In cases in which diagnostic testing has not be finalized, or cannot be completed, isolation should be implemented when the differential diagnoses include possible transmissible diseases.

An additional consideration when evaluating patient-associated biosecurity measures is, of course, treatment of the infectious etiology. While this is appropriate with some infectious diseases, such as clostridial infection, it is difficult in the cases of viral disease, like ECoV, where supportive care is often the primary treatment. When treatment of the clinical condition is appropriate, it should be considered.

Management of personnel (veterinarians, owners, horse care professionals) and their role in prevention of disease transmission is primarily based on ensuring that the personnel minimizes contamination between the animals for which they are caring. This is best accomplished by ensuring sick horses are isolated from healthy or unexposed horses and that the personnel cares for the horses by not working frequently between groups (i.e. taking care of healthy horses prior to sick horses) and minimizing human traffic amongst isolated areas/horses. Additionally, strict hygiene protocols between treatment groups and individual horses is very important. Hygiene protocols typically include handwashing and use of disposable gloves, use of hand-sanitizers, footbaths, gowns or coveralls, dedicated stall cleaning equipment or disinfection of equipment between horses. For veterinarians in a field scenario when horses with transmissible diseases are examined, hygiene protocols, as above, are important for not transmitting the disease from farm to farm.
Field veterinarians should additionally ensure that equipment used on an affected farm is disinfected prior to returning the equipment to the practice vehicle for use on another farm, and that the practice vehicle is not a source of contamination.

Management of the environment in which the horse is residing is a major component of ensuring biosecurity. Ensuring that the horse has minimal exposure to routes of transmission (inhalation or fecal-oral) is critical. For inhalation transmitted diseases, preventing contact with respiratory secretions is necessary. This is often due to contact between horses, but can also be through fomites. Fecal-oral transmission similarly can be through horse to horse contact, but can definitely be transmitted through contact with fomites. Ensuring that personnel are not a source of transmission is described above, but additionally, fomites may include pets in the stable (cats, dogs, chickens, etc.) as well as rodents, birds, and wildlife. Minimizing movement of these animals can be difficult, but should not be overlooked. Strategies for pet and wildlife management should be discussed with the owners/farm personnel. Rigid hygiene protocols should be implemented as described above and should include disinfection of all common areas where diseased horses have been housed/examined; disinfections of trailers, water troughs, feed buckets, stall cleaning equipment, shared grooming equipment, etc.; and management of manure pastures. Affected pastures should have manure removed and preferably bagged and removed from the property; however, composting affected manure can be performed as long as the compost pile is not in an area where horses have access to it and it is away from run-off water sources.

Each infectious and transmissible disease is accompanied by a unique pathogenesis that impacts the biosecurity measures that may be implemented in cases of outbreaks within a herd or in a solitary horse. Implementing educated prevention strategies centered-on managing the disease process in the horse(s), preventing transmission through isolation and appropriate hygiene, and managing the environmental contamination should result in minimal transmission of infectious diseases. Ultimately, ensuring that there are no breaks in the biosecurity chain leads to success.

**Practice tips**

Be prepared to encounter infectious diseases during practice and have your practice vehicle stocked with barrier precautions (gloves, gowns, change of clothes) and disinfectant for your equipment or to dispense to clients.

Members of the American Association of Equine Practitioners have access to white papers on biosecurity measures that can be recommended for specific disease processes. These documents facilitate efficient implementation of biosecurity principles. Printed copies or access to the website links for owners, is an efficient way to provide valuable information to owners to which they can refer as they implement recommendations.

References available upon request.
The chronic lower airway inflammatory diseases of recurrent airway obstruction (RAO) and inflammatory airway disease (IAD) are grouped as equine asthma syndrome (EAS). Recurrent airway obstruction is a chronic respiratory condition commonly identified in middle-aged to older horses, while IAD is a respiratory condition commonly identified in young performance horses and does not show seasonal recurrence. Horses with RAO typically show clinical signs referable to the respiratory tract at work and at rest, while horses with IAD are usually normal at rest and may only show signs of exercise intolerance. Hallmarks of equine asthma syndrome are pulmonary inflammation with bronchoconstriction and airway mucus. Recognition of the difference between the two clinical conditions of RAO and IAD is valuable for assessing the prognosis and chance of recurrence, however, clinical management and treatment RAO and IAD is similar and centers-on attenuation of bronchoconstriction and inflammation through environmental management and pharmaceutical therapy.

Diagnosis of RAO or IAD should include a thorough physical examination of the horse, including rebreathing examination. On physical examination, horses with RAO generally have difficulty during expiration; hypertrophy of the abdominal musculature (heave line) if chronically affected; a cough; and flared nostrils. Exercise intolerance, mucoid or serous nasal discharge, and audible respiratory signs are noted by owners as well. Empirical diagnosis of RAO, based on signalment, history, and clinical signs, is common when evaluating a horse on the farm; however, it is important to be mindful of the differential diagnoses for lower respiratory tract disease before initiating treatment. Differential diagnoses include inflammatory airway disease, parasitic pneumonia, fungal pneumonia, bacterial pneumonia, neoplasia, exercise-induced pulmonary hemorrhage, or viral respiratory disease (equine herpesvirus 1, 4, or 5 or equine influenza). When diagnosing IAD, physical examination findings are generally normal with no definitive signs referable to the respiratory tract and a history of poor performance or exercise intolerance. Similar to empirical diagnosis of RAO, definitive diagnosis of IAD can be based on signalment, history, and physical examination findings and similar differential diagnoses should be considered.

Definitive diagnosis of the causes of EAS is best performed by sampling lower airway secretions and subsequent cytological analysis. Diagnosis of RAO is by the demonstration of neutrophilic (non-degenerative) inflammation (>20% neutrophils), decreased lymphocytes, decreased alveolar macrophages, Curschmann’s spirals (mucus strands from the lower airways), and the absence of bacteria on analysis of bronchoalveolar lavage (BAL) or transtracheal wash (TTW) fluid. Diagnosis of IAD is by the demonstration of mild, non-degenerative neutrophilia, lymphocytosis, monocytosis, increased mast cell concentration (>2%), and eosinophilia (>0.1%) on analysis of BAL or TTW fluid. Clinically, if a TTW is to be performed, it should be performed before a BAL to prevent contamination of the airway. A TTW allows for culture of the fluid and exclusion of bacterial infection as a cause of the clinical signs. While this may involve performing two diagnostic tests, it allows for increased confidence that bacterial infection is absent in the face of treatment with corticosteroids. Pulmonary function testing, currently available at select referral centers, is an alternative way to seek a definitive diagnosis.

Farm management practices for treatment of EAS are considered crucial for a successful clinical outcome, particularly in horses diagnosed with RAO. On-farm management practices are centered on minimizing the burden of airborne respirable particles, and with diseases like RAO, are based on the nature of the horse’s husbandry, i.e. if the horse is showing clinical signs more often when stabled, it should be turned-out, or when on pasture, it should be stabled. Stabling practices that can be changed to reduce the clinical signs of EAS include moistening of the feed (hay and grain), bedding, and barn aisle. Improving the ventilation of the barn or of the stall where the horse is kept is often beneficial. This often means moving the horse to the end of the barn aisle, or to a stall with more windows. Eliminating practices that increase the environmental dust will also be helpful. This includes hosing down barn aisles rather than blowing or sweeping them, moving hay and shavings while the horse is out of the barn, and not cleaning the stalls while the horse is stabled. Most importantly, if the horse’s signs are associated with being housed in the stable, then it is reasonable that the management of the horse should include spending the majority of the time out in a pasture. If the horse has clinical signs that are associated with the pasture, it is important that the horse spend more time off the pasture to decrease allergen exposure; however, additional management changes that can help to attenuate the signs associated with pasture associated RAO include close mowing of the pasture and moistening dusty areas (often near gates and feeders/water troughs). These management changes should be instituted for all horses with EAS before, or in conjunction with, pharmaceutical therapy. In recent years, additional management options have become available. In the past, moistening of hay and grain meant soaking the hay in water, and today, hay steaming machines are available and have shown that they significantly reduce allergens and dust in the hay while preserving the nutritional content of the hay. Additionally, in the past low allergen bedding that could be used for horses was difficult to find in the United States or was impractical and expensive. Low-allergen bedding options (particularly paper based products) for horses are now mass produced at a
cost that is reasonable. The supplementation of the diet with omega-3 polyunsaturated fatty acid has been shown to provide a beneficial clinical response in horses with RAO on a low-dust diet. Regardless of the type of products used, the principles of on-farm management remain the same…reduce the exposure to dust and allergens.

Pharmaceutical therapy for horses with EAS is centered-on decreasing pulmonary inflammation and bronchoconstriction. There are several ways to do this in horses. Determining orally-administered versus inhaled therapy is the first step in choosing the medications that will best suit the horse and the owner. Orally-administered therapy is generally inexpensive and effective, but has the potential to result in more adverse effects than inhaled therapy. Inhaled therapy is more expensive, effective in compliant patients, and results in few adverse effects. Inhaled therapy can be administered by direct actuation of a metered dose inhaler into the nostril (although this is not consistently effective), or by use of a specifically designed administration device for use in horses such as the AeroHippus® (Trudell Medical International; London, Ontario, Canada). In the last few years, the option for nebulization of corticosteroids and bronchodilator medication has become available for use on the farm with the development of the Flexineb® (Nortev; Galway, Ireland) nebulization mask. This portable mask provides another way to safely and effectively deliver medication directly to the respiratory tract. While many owners may choose either orally-administered or inhalation therapy (i.e. one or the other), a combination of each type of therapy (i.e. orally administered corticosteroids with inhalation delivered bronchodilators, or vice versa) can also be tailored to suit the horse and owner if needed to optimize treatment.

The most commonly administered corticosteroids used for orally delivered therapy include dexamethasone and prednisolone (keep in mind that prednisone is not well metabolized by horses and should be avoided). The most common bronchodilator for orally delivered therapy is clenbuterol. A common corticosteroid used for inhaled therapy is fluticasone delivered by metered dose inhaler. This is moderately expensive, but is quite effective and results in minimal systemic adverse effects of corticosteroid use if administered appropriately. A common inhaled bronchodilator is albuterol, a β-2 adrenergic agonist. This is inexpensive and effective, although short-acting. Long-term (hours) bronchodilator therapy can be achieved with the use of ipratropium bromide, an anti-muscarinic bronchodilating agent, or salmeterol, a β-2 adrenergic agonist. The long term bronchodilator therapy is not appropriate for treatment of acute bronchoconstriction.

The treatment duration and dosage of medications for horses with EAS is highly variable and can be influenced by the clinical experience of the prescribing veterinarian, the clinical condition of the horse, and the needs of the owner. Based on severity, level of exposure to allergens, recurrence, and owner compliance, a treatment and farm management regimen can be outlined to optimize the horse’s response to therapy. The corticosteroid and bronchodilator therapy is initially administered at a dosage to rapidly suppress the inflammatory response and induce bronchodilation. Upon improvement in clinical signs, the dosage is gradually decreased to the lowest possible dosage that will control clinical signs. The medications are subsequently discontinued and the horse is monitored closely for recurrence. This typically spans a 4-6 week period of time and occurs in conjunction with on-farm management changes that are appropriate for the horse and achievable by the owner.

Generally, this initial plan of on-farm management changes and inhaled or oral pharmaceutical therapy will be effective for the average horse with EAS. Often these horses exhibit improved breathing within days of initiation of therapy. Occasionally, additional therapy changes may also be warranted and may include rescue therapy with epinephrine. If a horse fails to respond to a therapeutic protocol (or worsens) within weeks of initiation of therapy, it is necessary to re-evaluate the horse and the diagnosis. Other differential diagnoses should be re-considered (such as interstitial or bacterial pneumonia), as well as the horse’s/owner’s compliance with the recommendations/therapeutic protocol. It is important that owners acknowledge that this disease frequently recurs and may require lifetime management changes tailored for the horse.

References available from the author
Field Diagnosis of Lower Respiratory Tract Disease
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Examination of the lower respiratory tract in horses begins with a distance observation of the horse. This should include an overall impression of the horse (is the horse in good or poor body condition, is the horse aware of its surroundings, etc.). Observe the horse’s respiratory effort and character (is the horse relaxed and breathing slowly, normally, or is the horse exhibiting labored breathing, distress, making noise, etc.). These general findings should begin to provide information to begin determining if there is a problem associated with the respiratory system.

As part of a thorough physical examination, observe the horse’s respiratory character and rate. The character of breathing should be slow, even, and without significant effort or noise. Changes in the rapidity, depth (shallow or deep), or effort (labored, distressed, or increased expiratory effort) may indicate a problem with the respiratory tract. The normal resting respiratory rate for an adult horse is 8-16 breaths per minute. This number will vary with the fitness of the horse, with healthy, fit horses often having a lower rate while older, unfit horses may have a higher rate. The respiratory rate may also vary with the level of stress the horse is under at the time of the examination (stressed, nervous horses may have a higher rate), ambient temperature (slightly higher rate in the summer months), and it may vary with disease states. It is important to note that other body system abnormalities can cause changes in respiratory rate or character (such as fever, various states of shock, neurological disorders, etc.). In general, lower respiratory tract (LRT) disease does not cause significant, audible without a stethoscope, respiratory noise, although one may hear tracheal noise or wheezing. These sounds (tracheal noise and wheezing) are quite different from sounds associated with obstruction of the upper respiratory tract (URT) which are classically audible without a stethoscope.

Following the distance examination and determination of rate and character, evaluate for the following (and also keep in mind if the signs could be associated with an infectious disease): 1) Are the nostrils flared? Flared nostrils can be associated with increased depth or effort of breathing, but it is also important to note that some horses just have flared nostrils and the owner might report this as normal for the horse. 2) Does the horse move air normally, freely in and out of both nostrils? Masses in the nasal passages can obstruct airflow and this can be easily determined by examination (i.e. one often doesn’t need advanced diagnostic tests to determine that the horse isn’t moving air from one nasal passage). 3) Is there abnormal noise associated with breathing without the assistance of a stethoscope? Fixed or variable obstructions of the airway can result in audible noise. The timing of the sounds should also be noted (i.e. inspiratory or expiratory). Fixed obstructions, such as a tumor in the nasal passage, often result in sounds that are similar on inspiration and expiration. Variable obstructions, such as laryngeal hemiplegia, will show a significant variation in sound from expiration to inspiration. 4) Does the horse have nasal discharge? Note the character (serous, serosanguinous, frank hemorrhage, mucoid, mucopurulent, feed material) as this can often help to include or exclude certain differential diagnoses. Note whether the discharge is unilateral or bilateral, and if it is consistently one or the other (unilateral discharge often originates rostral to the nasal septum, while bilateral discharge can be from any part of the respiratory tract). Note the odor of the discharge (foul smelling discharge is often associated with an anaerobic bacterial infection). 5) Does the horse cough when the larynx or trachea are palpated? An elicitable cough is an indicator of inflammation (or other abnormalities) and may help identify the respiratory tract as an area of concern. 6) Are the mandibular lymph nodes enlarged? Enlargement of the lymph nodes can be a non-specific sign associated with airway inflammation, but can also indicate an infectious disease such as strangles (Streptococcus equi subsp. equi). 7) Do the sinuses percuss normally? While this is associated with evaluation of the URT, it should be a component of a thorough respiratory examination.

Thoracic auscultation is the best tool for evaluation of the LRT. It is important to remember that while auscultation can provide valuable information as to the nature of the disease in the thoracic cavity, it is poorly correlated with specific diseases (i.e. just because ‘crackles’ are audible doesn’t mean the horse has pneumonia). Auscultation should be performed in a quiet area when possible to increase the ability to auscultate bronchovesicular sounds. The tidal volume for an adult horse is approximately 4-7 liters at rest. Additionally, a resting horse will only use 30-40% of its lung capacity, so there is a significant amount of dead space that is not moving air. This makes it difficult to hear bronchovesicular sounds in a resting horse. For this reason, auscultation with assistance of a rebreathing bag is often necessary to auscultate the lungs. In a healthy horse, auscultation before and after exercise can be useful as exercise will increase the rate and depth of breathing.

Rebreathing examination involves the placement of a bag over the nose of the horse to stimulate increased depth of breathing. As the horse inspires increasing amounts of CO₂, chemoreceptors are stimulated and the respiratory center in the brain increases the rate and depth of breathing in an attempt to decrease CO₂ and increase O₂ concentration in the blood. This increases the volume and movement of air allowing better auscultation of the lung sounds throughout the thoracic cavity. A rebreathing examination should be a component of a thorough physical examination on a horse (especially with PE findings referable to the respiratory system, pre-purchase examinations, or when having difficulty identifying the source of a horse’s clinical problems). An important thing to note
about rebreathing is that it should only be performed if it will not distress the horse. If the horse is visibly distressed, dyspneic, or coughing excessively, avoid this procedure as it will worsen all of these signs and could result in the horse collapsing. Another important thing to remember is that a large rebreathing bag should be used because as the horse’s respiratory rate and depth of breathing increases, the tidal volume increases (to up to 35-45 L), so a large enough bag to accommodate the tidal volume is needed. Autoclave or heavy weight cellophane bags work beautifully, but another substitute that works well is a Ziploc® Big Bag XL or XXL. Once the bag is in place over the horse’s nose, it will take several breaths, and sometimes up to a minute or two in normal horses, for the horse to begin to increase the rate and depth of breathing. Auscultate over all lung fields bilaterally, and over the trachea, to identify normal and abnormal sounds, and where those sounds are most intense or absent. As with all diagnostic procedures, it is best to get in the habit of performing the procedure in a systematic fashion. The bag should be left on the horse until the depth of breathing makes it easy to hear bronchovesicular sounds, but should be slackened or removed if the horse begins to cough excessively or becomes too distressed. Leaving the bag in place until the horse is breathing deeply and then opening the side of the bag slightly to let in some fresh air keeps the horse from getting too distressed, but continues to allow easy auscultation. A normal horse should recover quickly upon removal of the bag (within 3-5 breaths) and should not cough during the examination. If a horse takes too long to recover, or coughs during or after rebreathing, the respiratory tract may not be normal. Sounds identified on auscultation of the thorax are variable and the subtle changes can be consistent with certain disease conditions. Common findings on auscultation of the thorax include normal bronchovesicular sounds (BVS), harsh BVS, absent BVS, crackles (the sound of fluid ‘popping’ in the airways), wheezes (the musical sound associated with narrowed airways/bronchoconstriction), intestinal sounds, pleural friction rubs, radiating heart sounds, and muffled heart sounds.

Thoracic percussion is a valuable skill to develop. This technique helps identify areas of the lung that are consolidated, or more consistently when the thoracic cavity contains effusion or a soft tissue mass between the body wall and lung surface. While other diagnostic modalities, such as thoracic ultrasonography and radiography may provide more detailed information, percussion of the thorax remains a useful skill in situations when those modalities are not available. Percussion is performed by systematically percussing each intercostal space with the use of plexor/pleximeter, or fingers. It is performed by moving fingers down the intercostal space while tapping them to identify a change in the sound (typically a dampening or dulling of the sound) that would indicate consolidation, fluid, or soft tissue between the lung surface and the body wall.

Thoracic ultrasonography is an extremely valuable tool to evaluate the peripheral thoracic anatomy. Typically ultrasonography of the thorax is used for identifying/confirming the presence or absence of pleural effusion (including the character of the effusion); identifying pleural irregularities that might be consistent with thickening, fibrosis, or masses in the pulmonary parenchyma that are in contact with the visceral pleura; thoracic adhesions; and limited examination of the mediastinum for abnormalities. Diseases in which thoracic ultrasonography can be very valuable include identification of pneumonia/pleuropneumonia due to bacterial infection, fungal infection, or neoplasia, and hemothorax.

Transtracheal wash/aspiration (TTW) is one of the most important diagnostic tests for evaluation of the lower airway of horses. The technique is used to collect airway secretions for cytological analysis and bacterial culture. Ideally, the technique allows direct, sterile collection of fluid from the trachea. Fluid can be collected by percutaneous transtracheal aspiration (preferred for preservation of a sterile sample for culture) or by transendoscopic tracheal aspiration. A TTW is indicated in horses with lower airway disease in which infection is considered likely, or if infection needs to be excluded before initiating treatments (i.e. in cases of recurrent airway obstruction (RAO) that will need to be treated with steroids, it is ideal to have a negative TTW culture result before initiating treatment). Commonly, horses with pneumonia, pleuropneumonia, or RAO are commonly subjected to TTW to evaluate for bacterial infection; however, it can also be useful for any other focal or diffuse disease of the pulmonary parenchyma as abnormal cells, or populations of cells, can be shed into the respiratory secretions and will be identified in the fluid collected from the trachea. Keep in mind that because culture the fluid will be performed, this procedure should be done prior to any other invasive respiratory diagnostic tests to preserve the integrity of the fluid sample (i.e. to keep from contaminating the airway when doing other procedures). Contraindications for TTW include severe dyspnea/respiratory distress (i.e. sometimes the stress of the procedure is too much for a horse having difficulty breathing) or really small patient size (i.e. the horse/foal is too small for the size of the materials to be used for the procedure). The samples collected from TTW should be submitted for cytological analysis and bacterial culture (aerobic with sensitivity, and anaerobic). Additionally, fluid samples can be evaluated for very specific diseases (i.e. fungal disease, Rhodococcus equi infection, and Streptococcus equi subsp. equi infection).

Typically, TTW samples should be sterile (i.e. there are not intracellular bacteria on cytological analysis and bacterial culture is negative). The presence of intracellular bacteria is significant assuming that the sample is read fairly quickly following collection. In horses with respiratory disease, the following are commonly isolated bacteria: Streptococcus equi subsp. zooepidemicus is the most common bacteria isolated from the respiratory tract of horses with respiratory disease; common gram negative isolates include Klebsiella sp., E. coli, and Pasteurella sp.; Bacteroides sp. is the most common anaerobic bacteria isolated from the respiratory tract of horses with respiratory disease. It is important to keep in mind that the correlation between the cytological findings of TTW fluid with the actual pulmonary pathology can be quite variable; therefore, interpretation must be made with the clinical findings in mind.
Bronchoalveolar lavage (BAL) is a technique used to sample the fluid of the lower airways in horses with diffuse disease. The procedure facilitates the collection of secretions from only a small segment of airways, therefore focal lung disease may not be recognized using this procedure. The fluid sample is collected from the terminal airways and associated alveoli. The technique is used to collect airway secretions for cytological analysis. The samples are not typically submitted for bacterial culture due to contamination of the sample as the tube is passed through the nasopharynx into the trachea. A BAL can be performed through nasotracheal intubation via BAL tube (fast; inexpensive; performed blindly) or by a transendoscopic technique. Indications for BAL are typically associated with the need to evaluate the lower airway secretions of horses with diffuse pulmonary disease and include fungal pneumonia, RAO, exercise-induced pulmonary hemorrhage (EIPH), neoplasia, parasitic lung disease, and multi-nodular pulmonary fibrosis (EHV-5).

Thoracocentesis is both diagnostic and therapeutic. The procedure is diagnostic in that the pleural fluid (blood, pus, metastatic effusion, inflammatory effusion, etc.) can be evaluated for character, cytological analysis, and for bacterial culture (or other microorganisms like fungi). The procedure is therapeutic in that the removal of the pleural fluid or air (as seen with pneumothorax) will allow the horse to more easily expand the lungs within the thoracic cavity. Thoracocentesis is indicated in horses with pleural effusion or pneumothorax. On physical examination, one may note an absence of ventral bronchovesicular sounds, abnormally dull sounds on thoracic percussion, and diagnostically, fluid observed in the pleural space on ultrasonographic evaluation. Fluid samples are submitted for cytological analysis, and bacterial (aerobic & anaerobic) or fungal culture. One thing to keep in mind is that bacterial culture of effusion secondary to pulmonary infection will often not produce a positive culture result. This is for 2 reasons: 1). The origin of infection in most pneumonia/pleuropneumonia cases is in the pulmonary parenchyma and the effusion is secondary to inflammation rather than primary pleural infection (as is seen with pleuropneumonia secondary to a penetrating wound to the thorax). For this reason, TTW remains the most important diagnostic test to perform to determine bacterial population. 2). The fluid collected from the pleural space is ultimately an inhospitable environment for bacterial growth when the effusion is inflammatory (i.e. pus).

Thorough diagnostic evaluation of the lower respiratory tract is valuable for appropriate diagnosis and treatment of horses. In addition to a thorough physical examination, the diagnostic tests described can be performed on the farm. As one’s clinical experience with the procedures grows, they can be performed easily and quickly.

Other diagnostic procedures, such as lung biopsy, thoracoscopy, arterial blood gas, and pulmonary functional testing are not typically performed in the field, but may be valuable in cases where a field diagnosis cannot be made, or if the horse fails to respond to on-farm therapy.

References available from the author.
Pneumonia is a generalized term used to describe clinical disease in which the lungs (lower airways) are inflamed and most likely infected. Pneumonia is typically classified as to the underlying etiology (bacterial, viral, parasitic, fungal, interstitial, etc.). It can be further described as to the location of the disease (e.g. lobar pneumonia, that affecting a large portion of a lung lobe, or bronchopneumonia, that affecting the bronchiole walls and characterized by foci of consolidation of 1 or more lobules). Typically in horses, pulmonary infection is described as either pneumonia/bronchopneumonia (of any cause) or pleuropneumonia (affecting the lungs and pleural space).

Bacterial pneumonia in horses, whether affecting only the pulmonary parenchyma, or encompassing the pleural space, is a common clinical condition. Horses are typically at risk for development of bacterial pneumonia for a variety of reasons including aspiration secondary to esophageal obstruction, viral respiratory disease, stress, or any other condition that impairs, compromises, or breaches the normal pulmonary defense mechanisms. The normal pulmonary defense mechanisms include mucociliary transport, alveolar macrophages, secretory defenses (IgA, IgG, mucus, complement, interferon, and surfactant), aerodynamic filtration (airway size, hairs, turbinates, velocity), and cough/sneeze. Pathophysiologically, the development of pneumonia can be broken down in the simplest terms to colonization by opportunist bacteria, multiplication of bacteria, inflammation of the pulmonary parenchyma, and then destruction of pulmonary parenchyma/endothelium.

The chief complaint for evaluation of a horse with respiratory disease is variable. Frequently owners recognize non-specific clinical signs rather than signs referable to the respiratory system. The on-farm examination is often centered on first determining if the respiratory system is affected, followed by determining whether the lower airway vs. upper airway is affected.

Frequent non-specific clinical signs of bacterial pneumonia may include fever, anorexia, lethargy, ventral or peripheral edema, exercise intolerance, colic signs, and/or dehydration. Common clinical signs in horses with bacterial pneumonia that are referable to the respiratory system may include cough, nasal discharge (often bilateral), tachypnea, dyspnea, tracheal rattle, and/or pleurodynia. On-farm examination includes a thorough physical examination and subsequent thorough evaluation of the respiratory system with rebreathing examination when appropriate (i.e. if the horse can tolerate rebreathing without distress), thoracic percussion, and thoracic ultrasonography. Once lower airway disease is confirmed, either by abnormal auscultation/percussion or abnormal ultrasonography, it becomes necessary to determine if bacterial disease is the cause of the clinical signs, or if signs are due to inflammatory conditions such as inflammatory airway disease or recurrent airway obstruction. The differentiation between an inflammatory disease and an infectious disease is frequently encountered when evaluating a horse on the farm with respiratory signs. The best diagnostic tool is to perform a transtracheal wash/aspiration with submission of the samples for cytological analysis and bacterial culture (aerobic/anaerobic). On cytological analysis, the presence of degenerative neutrophils and intracellular bacteria from a fresh sample without pharyngeal contamination is a strong indication of bacterial infection.

Bacterial culture with antimicrobial susceptibility of fluid collected by transtracheal wash/aspiration is an ideal diagnostic test for horses with pneumonia. It not only helps with the initial treatment, but guides management of the case as the case progresses; however, it is not always an option due to financial constraints of the owner, or clinically in a horse in significant respiratory distress. In cases where a transtracheal wash/aspiration cannot be performed, choosing an antibiotic based on a rational approach is ideal. This includes choosing an antimicrobial(s) that targets the known common organisms identified in horses with pneumonia and ensuring that the antimicrobial is capable of targeting infection in the pulmonary system. Additionally, a thoracocentesis can be a useful tool both therapeutically and diagnostically and can be performed on the farm. While cytological evaluation and bacterial culture of fluid collected by thoracocentesis is not as reliable as that obtained by transtracheal wash/aspiration for diagnosis of infection, it is valuable therapeutically in cases of pleuropneumonia and may provide clinical information when a transtracheal wash/aspiration cannot be performed.

Additional diagnostic evaluation that can be pursued while in a field situation includes a CBC and serum biochemistry analysis. While the clinicopathological data cannot diagnose respiratory disease, it provides valuable information for guiding treatment (i.e. white blood cell, fibrinogen, and serum amyloid A concentrations can help guide response to treatment and duration of treatment; and evaluation of organ function may impact the choice of medications).

Following the initial evaluation of the horse, a decision must be made whether or not to treat the horse on the farm, or to refer the horse for further evaluation and treatment at a referral center. Common indications for referral include severity of the clinical signs; the owner’s ability to perform or comply with the prescribed treatment regimen; and financial constraints.

Therapeutic plans for treatment of bacterial pneumonia are centered on rational antimicrobial therapy. Rational antimicrobial choices are guided by understanding the most common bacteria to affect a particular body system. In horses with respiratory disease, *Streptococcus equi* subsp. *zoopneumonia* is the most common bacteria isolated. It is a gram positive, facultative anaerobic organism.
that is normal flora of the equine upper airway. It is considered an opportunistic organism (i.e. bacterial overgrowth occurs when the normal pulmonary defense mechanisms are compromised). As it is facultative anaerobic organism it can grow in an oxygenated environment in vivo and in vitro, and can be treated with antimicrobials that target aerobic organisms. There is not one specific gram negative organism that is predominantly isolated from horses with pneumonia; however, common gram negative bacterial isolates from the respiratory tract of horses with pneumonia include Klebsiella sp., E. coli, Actinobacillus sp., and Pasteurella species. These gram negative organisms are typically pharyngeal contaminants that proliferate in the compromised lung parenchyma. They are facultative anaerobic organisms similar to Streptococcus equi subsp. zooepidemicus and can be treated similarly with antimicrobials that target aerobic organisms. In addition to the common gram positive and gram negative isolates, Bacteroides sp. is the most common obligate anaerobic organism isolated from the respiratory tract of horses with respiratory disease. This organism is gram negative. This is an important consideration when designing a rational therapeutic antimicrobial plan as obligate anaerobic organisms must be treated with an antimicrobial that targets gram negative obligate anaerobes (e.g. metronidazole).

Based on the common isolates, a rational approach for treatment of a horse with respiratory disease may include antimicrobial medications that target gram positive and negative organisms, and gram negative anaerobic organisms, with adjustments made in the antimicrobial plan based on the horse’s response to treatment and results of the bacterial culture/susceptibility if performed. The choice of antimicrobial must then be based on the route needed to successfully treat horse (e.g. intravenous in horses with severe disease); the ability of the owner to give the medication; the likelihood of antimicrobial resistance; the volume of distribution and bioavailability of the medication (i.e. once the medication is administered, can the drug be absorbed and then reach the target area of infection); the adverse effects that may affect the horse; affordability for the owner; and the ability of the horse to tolerate the medication for the course of treatment.

In addition to systemic antimicrobial therapy, therapeutic management of horses with bacterial pneumonia includes treatment with systemic anti-inflammatory therapy, typically non-steroidal anti-inflammatory drugs (NSAIDs). Flunixin meglumine is commonly used for its anti-inflammatory, anti-endotoxic, and analgesic effects; however, treatment with other NSAIDs such as firocoxib, phenylbutazone, or ketoprofen, may provide effective control of inflammation. Additional supportive care includes monitoring of water intake to ensure adequate hydration and prevention of colonic or cecal impactions; monitoring of appetite; laminitis prevention and monitoring for early clinical signs; and rest.

Complications associated with bacterial pneumonia are common. These include development of laminitis, thrombophlebitis in horses with an IV catheter, pulmonary abscess formation, cecal impaction, pulmonary/pleural adhesions, pneumotheorax, pericaritis, etc. Frequent monitoring of the physical examination and thoracic ultrasound examination is important for recognizing the early signs of complications.

The prognosis for bacterial pneumonia is typically guarded to good in horses without severe disease. The prognosis decreases significantly in horses with anaerobic infection, excessive intra-pleural fibrin deposition, horses that develop complications, and horses with pulmonary thromboembolism, disseminated intravascular coagulopathy, or pulmonary ischemia.

Prevention of bacterial pneumonia cannot always be achieved; however, management practices by the owner can often decrease the risk. Travelling shorter distances, less frequently, and with the head untied, can decrease the risk of development of pneumonia. Wetting or steaming hay being offered while travelling may minimize dust inhalation. Educating owners to have horses vaccinated against viral respiratory disease (EHV 1 & 4 and influenza) may also decrease the risk for development of bacterial pneumonia. Lastly, empirical treatment for aspiration pneumonia in horses with esophageal obstruction may decrease the risk of pulmonary infection.

Treatment of bacterial pneumonia is often successful depending on severity. In horses without severe disease, the prognosis for bacterial pneumonia is typically guarded to good. In the author’s experience, early diagnosis and rapid initiation of treatment improves the outcome and how quickly the horse returns to normal.

References available from the author
This session highlights clinical cases of equine respiratory disease. Below are small summaries of cases that may be presented within the time allotted and an associated ‘take home message’.

Case 1: pleuropneumonia in a thoroughbred stallion
Pleuropneumonia is a common clinical condition of horses classically associated with young performance horses that are stressed or commingling with other horses, are in training, travel long distances, or have upper respiratory tract viral disease. In the presented case, the horse developed profound pleuropneumonia following an episode of esophageal obstruction. The case outlines the diagnostic evaluation for a horse with pleuropneumonia which includes physical examination, thoracic auscultation, thoracic ultrasonography, thoracic radiography, and thoracocentesis. These diagnostic modalities are indicated in horses with significant pleural disease. The ‘take home message’ is to consider the impact of aspiration of oropharyngeal contents during an episode of esophageal obstruction. Antimicrobial therapy should be considered based on the initial clinical evaluation of the horse when treated for esophageal obstruction, the duration of the esophageal obstruction, and the ease/difficulty of relief of the obstruction. In horses already showing clinical signs referable to the respiratory tract; horses with prolonged obstruction; and/or horses with complicated or prolonged correction of the obstruction, broad-spectrum antimicrobial therapy is warranted.

Case 2: mineral oil aspiration in a warmblood gelding
Aspiration of mineral oil, or inadvertent administration of mineral oil into the lungs, is often fatal. Mineral oil is an inert compound that cannot be absorbed across tissue. Oil in the lungs prevents oxygen exchange and the ensuing inflammation can be devastating. In the presented case, the horse aspirated a presumably small amount of oil following nasogastric intubation and treatment of a colic episode. The nasogastric tube was not fully cleared of oil at the time of removal of the tube and the horse leaped in the air as the end of the tube was in the pharynx. Aspiration was not recognized at the time. The horse was referred for treatment of colic and when the horse began showing clinical signs referable to the respiratory tract, a thoracic ultrasound was performed and identified cranioventral comet tail artifacts. Transtracheal aspiration confirmed the presence of oil droplets in the fluid. The horse was treated initially with broad-spectrum antimicrobial therapy pending bacterial culture results, anti-inflammatory therapy, and intranasal oxygen. He responded well and was transitioned to corticosteroids. The horse did very well and returned to full training. The ‘take home message’ is to continue to ensure appropriate intubation of the esophagus if delivering mineral oil and to fully clear the tube upon removal.

Case 3: hemothorax following vertebral body fracture
A 20 year old thoroughbred mare was presented for a chief complaint of colic which had failed to respond to medical therapy on the farm. On presentation, the horse showed overt signs of discomfort and grade 2 pelvic limb ataxia. Physical examination identified radiating heart sounds and muffled bronchovesicular sounds. The mare was tachypneic and dyspneic. Clinical evaluation for colic included transrectal abdominal palpation and abdominal ultrasonography; no abnormalities were identified. Transthoracic ultrasonography showed bilateral accumulation of fluid of mixed echogenicity. A thoracocentesis was performed and 4 liters of hemorrhagic effusion was collected. Following drainage of the thoracic cavity, thoracic radiographs were performed and identified a displaced fracture of the T-15 vertebral body. The hemorrhage in the pleural space was presumably due to laceration of an intervertebral vessel at this site. The horse was euthanized due to poor prognosis. The ‘take home message’ is to allow your physical examination to guide your evaluation and diagnostic work-up. This horse was presented for colic, and had a thorough physical examination not been performed, thereby failing to identify the pleural hemorrhage and vertebral fracture, this case may have been treated for abdominal discomfort with little success. The physical examination remains the most valuable component of patient evaluation.
Recurrent colic in adult horses is a common cause of primary and referral evaluations by veterinarians. Due to the long list of differential diagnoses, and relatively limited diagnostic capabilities as compared to small animals (i.e. unable to perform abdominal computed tomography or magnetic resonance imaging in adult horses), determining an etiology may pose a diagnostic challenge.

There is not a clear definition of chronic or recurrent colic in horses, as the definition is relative to the number of colic episodes per a specified period of time. For practical purposes, a loose definition of multiple colic episodes within weeks to months is appropriate. The clinical signs exhibited by horses are variable from low-grade signs such as changes in demeanor, decreased appetite, quiet recumbency or stretching, to overt signs of abdominal discomfort such as persistent or recurrent recumbency, rolling, collapse, pawing, etc. Often the clinical signs of abdominal pain may be alleviated with medical therapy with analgesic/anti-inflammatory medication such as flunixin meglumine and administration of enteral fluids or cathartic medications via a nasogastric tube; however, they routinely recur within days to weeks/months of the original episode. Initial physical examination and on-farm diagnostic testing is often unrewarding in many of these cases, which may ultimately result in referral for further diagnostic evaluation following several episodes of abdominal discomfort. The ultimate goal of pursuing further diagnostic tests is to alleviate the horse’s discomfort and the owner’s anxiety, achieve a diagnosis and treatment plan, and return the horse to health and performance.

It is important to recognize that many clinical signs of abdominal discomfort (colic) are non-specific and diseases of many body systems can mimic the classic clinical signs of colic. When considering the differential diagnoses for chronic or recurrent colic, it is appropriate to consider etiologies that are gastrointestinal in origin and those that are associated with extra-gastrointestinal conditions. Gastrointestinal etiologies are extensive, but common causes leading to recurring bouts of colic include cyathostomiasis, tapeworm infestation, enterolithiasis, neoplasia, inflammatory bowel disease, right dorsal colitis, colon displacement, sand impaction, intestinal abscessation, esophageal disease and gastric ulceration. Extra-gastrointestinal causes may include conditions associated with the hepatobiliary system; the urinary tract; intraabdominal adhesions, abscesses, neoplasia, or granulomas; conditions associated with the cardiopulmonary system or thoracic cavity; and musculoskeletal diseases. As gastrointestinal etiologies are the most common, it is often more rewarding to center the initial diagnostic evaluation on etiologies in the abdomen, as well as to begin with the least extensive and least invasive tests; however, physical examination findings should always guide the diagnostic efforts.

### Diagnostic approach to recurrent colic

Colic is such a common clinical complaint, each practitioner likely has a common practice for the physical examination and diagnostic evaluation. This typically includes a thorough physical examination followed by passage of a nasogastric tube which acts both a diagnostic and therapeutic procedure, transrectal palpation of the caudal abdominal cavity, and may include abdominal ultrasonography if available. The horses are usually treated with analgesic, anti-inflammatory, or anti-spasmodic medications, enteral fluids/electrolytes, and observed for clinical response. When the horse responds to therapy, the horses are typically returned to feed and exercise gradually. If the horse develops clinical signs again some number of days to weeks/months later, more extensive diagnostic evaluation is warranted. It is important to note that the more extensive diagnostic evaluation may be more likely to result in a diagnosis if the horse is examined during an episode of colic, however, that should not deter a client from pursuing diagnostic testing (i.e. it is not necessary to wait for a horse to be showing clinical signs before pursuing the more extensive examination/referral).

As mentioned above, the diagnostic approach to cases of recurrent colic should begin with a thorough physical examination. This includes recording the vital parameters and examining all body systems in a thorough and systematic fashion. Physical examination should also include a thorough oral examination with a mouth speculum in place as dental disease may lead to improper mastication and subsequent increased feed particle size that may result in gastrointestinal distension/impactions and colicky behavior. Similarly, visual examination of the manure may show evidence of improper grinding of food (e.g. whole oats/corn and long hay fibers in the feces). If needed, a dental flotation can be performed and the patient re-evaluated for improvement of signs at a later date. This is a common cause of recurrent colic and should be one of the primary diagnostic evaluations performed if a horse exhibits signs of abdominal discomfort recurrently. Auscultation of the thoracic cavity with the assistance of a rebreathing bag should also be a component of the physical examination in an effort to exclude pulmonary abnormalities as a source of pain that mimics colic. Rebreathing is important as it can provide valuable information as to the nature of the disease in the thoracic cavity. Auscultation should be performed in a quiet area when possible to increase the ability to auscultate bronchovesicular sounds. The tidal volume for an adult horse is approximately 4-7 liters at rest. Additionally, a resting horse will only use 30-40% of its lung capacity, so there is a significant amount of dead space that is not moving air. This makes it difficult to hear bronchovesicular sounds in a resting horse. For
This examination of all body systems, a diagnostic plan is made, guided by any historical or physical examination findings.

Another common cause of recurrent colic is parasite infestation and this is an easy differential diagnosis for which to evaluate and treat. Fecal evaluation for parasites should be a component of the initial evaluation for horses with episodes of abdominal discomfort. Larval cyathostomiasis and tapeworm infestation are frequently implicated as causes of recurrent colic. It is important to remember that larval cyathostomiasis does not always result in strongyle eggs in the feces; therefore, a resolution of clinical signs following empirical therapy with larvicidal doses of fenbendazole or moxidectin can be indicative of a diagnosis of cyathostomiasis. Additionally, infestation of tapeworms can result in colic signs due to attachment of the parasites at the ileocecal orifice and subsequent decrease in lumen size. Fecal examination for tapeworms requires flotation with saturated sucrose solution, but a positive response to empirical therapy with praziquantel or a double dose of pyrantel pamoate may be indicative of the diagnosis.

Additionally, when considering common causes of recurrent colic, a dietary trial may be an effective empirical diagnostic tool that is inexpensive. The author frequently recommends switching to a different feed source, often a complete pelleted diet for a period of time and then slowly adding in hay sources, if appropriate, that vary from the horse’s original diet. Diet modification may alleviate the horse’s clinical signs of abdominal discomfort and the owner’s anxiety.

When findings on the physical examination and empirical treatments do not result in resolution of the clinical signs of recurrent colic, additional diagnostic evaluation is warranted. The author frequently begins by acquiring clinicopathological date. Complete blood count and serum chemistry profile are inexpensive and non-invasive ways to evaluate for chronic inflammation, organ dysfunction, and possibly neoplasia. Complete blood count should include fibrinogen concentration, as it is an indicator of chronic inflammation. Changes in the leukogram and hemogram may be indicative of inflammation/infection, neoplastic conditions of the bone marrow, et cetera. While these changes may not directly lead to a diagnosis, they can support findings during the remainder of the diagnostic evaluation. Serum chemistry profile should include liver enzymes (GGT and SDH), bile acids, renal evaluation (creatinine, BUN and electrolytes) and albumin/total protein concentrations. Information gathered from laboratory data can be used to determine further diagnostic tests to be performed, or can be used in conjunction with other test results to support a diagnosis.

Additional laboratory data should include a complete urinalysis with cytology to fully characterize the health of the urinary tract, and may also include serology for *Streptococcus equi* (M-protein titer) or *Corynebacterium pseudotuberculosis* (SHI test) when internal abscessation is suspected.

Transrectal abdominal palpation is an excellent way to evaluate the internal structures of the caudal abdomen and is indicated in cases of chronic colic. Abnormalities that cause recurrent episodes of abdominal discomfort, such as internal abscesses, neoplasia, colon displacement, or thickened bowel may be palpable and support the diagnosis, as well as justify further diagnostic efforts. Rectal mucosal biopsy can also be performed if infiltrative or infectious processes are suspected. Biopsies samples should be submitted for histology and culture. Unfortunately, this is the only location in which biopsy of the gastrointestinal tract caudal to the duodenum can be performed without laparotomy, and often infiltrative diseases do not routinely affect the rectum, but the information provided can be helpful in making a diagnosis. Duodenal biopsy specimens can be collected by gastroscopy and examined by histology and culture for abnormalities that could result in recurrent episodes of colic.

Gastroscopy is one of the more valuable diagnostic modalities for horses with chronic colic, especially those horses that are in training, stabled, and/or receiving high concentrate diets. Horses that exhibit signs of colic after eating may have gastric ulceration, outflow obstruction, gastric impaction, or gastric neoplasia, indicating a need for gastroscopy. Additionally, a positive response to empirical therapy with omeprazole, ranitidine, and/or sucralfate may support the diagnosis of gastric ulceration as a cause for recurrent colic.

Transabdominal ultrasonography is currently the best way to visualize the structures of abdomen without having to perform exploratory surgery, and is indicated in most cases of chronic colic as it allows indirect visualization of the abdominal organs, abdominal fluid, and abdominal wall. The duodenum and jejunum can be evaluated for thickened segments that may be consistent with inflammatory bowel disease (IBD) or neoplasia. Additionally, dilated segments may be indicative of chronically strictured areas aboral to the dilated segment. Thickened segments of colon, particularly the right dorsal colon, may be indicative of IBD, chronic right dorsal colitis or neoplasia. The abdominal fluid can be evaluated for increased cellularity, as might be consistent with blood or inflammatory cells, or effusion. The liver, kidney, and spleen can be evaluated for organ abnormalities. Additionally, abnormalities in the intestinal lymph nodes may be visualized. Chronic left dorsal displacements may also be imaged with ultrasonography. Thickening of the greater curvature of the stomach can be visualized in some horses with gastric ulceration or gastric neoplasia.

Abdominocentesis is a simple and inexpensive way to sample the abdominal fluid for cytological evaluation and possible culture. It is indicated in most cases of chronic colic as it allows a direct evaluation of the abdominal microenvironment. Analysis of abdominal fluid for color, nucleated cell count, protein concentration, and cytologic evaluation may support a diagnosis of peritonitis, hemorrhage, effusion, or neoplasia. Additionally, the abdominal fluid can be evaluated for bacterial culture, or PCR testing for *Streptococcus equi* or *Rhodococcus equi* infection/abscessation.
Small intestinal absorption tests with oral glucose can be used to determine a patient’s ability to absorb nutrients. Patients with inflammatory bowel disease, parasite infestation, or intestinal neoplasia may have abnormalities in absorption and can support the diagnosis. These patients often have weight loss or diarrhea in conjunction with clinical signs of colic.

Abdominal radiography is uncommonly used in adult horses for evaluation of chronic colic due to it being inaccessible in the field; however, mineral opacities from enterolithiasis, large metallic foreign bodies, or sand ingestion can be visualized. If sand or enteroliths are suspected, abdominal radiographs are indicated. Sand is most easily identified in the sternal flexure and if present in significant amounts, is visualized without difficulty. Enteroliths are difficult to visualize, but depending on the size and location of the stone, it may be diagnosed on a radiograph.

Laparoscopy can be used to visualize the surfaces of the abdominal structures and is indicated in cases where visualization is desired, but manipulation of the organs is not necessary. Laparoscopy is most useful for diagnosis of abnormalities in the dorsal abdomen. Exploratory laparotomy, as an elective diagnostic tool, is generally reserved for horses that have persistent colic in the face of extensive testing and no diagnosis. It is generally the last diagnostic test performed as it is highly invasive and expensive. However, it is the best way to make a definitive diagnosis for most causes of chronic colic, as biopsy samples can be collected and organs can be directly evaluated and corrections can be made if possible.

Additional diagnostic tests for extra-gastrointestinal causes of chronic colic may include cystoscopy for urolithiasis, liver biopsy for hepatic disease, and thoracic evaluation by thoracic ultrasonography and radiography.

In summary, the diagnostic evaluation for horses with a chief complaint of recurrent episodes of colic is centered on a strong initial physical examination and possibly empirical treatment for known common differential diagnoses. If the clinical examination indicates clinical signs referable to specific body systems, or if the horse fails to show a positive response to empirical therapy, additional diagnostic evaluation is warranted.

References available upon request
Weight Loss:
Diagnostic Evaluation and Challenging Cases
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Weight loss in adult horses is one of the more common causes of primary and referral evaluations by veterinarians. While some of these cases can be simply attributed to inadequate feeding, poor dentition, or inappropriate preventative care, others can be diagnostically challenging.

When evaluating a horse for weight loss, it is important to first identify possible inherent factors associated with the horse that could be contributing to weight loss (i.e. pituitary pars intermedia dysfunction, age, breed, etc.); environmental factors (i.e. turn-out on sand, poor pasture quality, ambient temperature); and management practices (i.e. preventative health care, diet, turn-out with others, frequency of feeding, etc.). These simple historical questions may direct many therapy recommendations or the diagnostic evaluation. When the common causes of weight loss have been addressed, it is then necessary to examine the problem of weight loss as a complicated case and pursue further diagnostic evaluation. In the author’s opinion, the best way to approach this problem is to devise a diagnostic plan that includes thoroughly evaluating all body systems in a least invasive to most invasive manner, always accounting for guidance by test results and the owner’s financial commitment.

When considering the differential diagnoses for weight loss, it is often appropriate to consider etiologies that are primarily gastrointestinal in origin; however, non-gastrointestinal causes are also frequent. Gastrointestinal etiologies are extensive, but common causes leading to weight loss include cyathostomiasis, gastrointestinal neoplasia, inflammatory bowel disease, right dorsal colitis, chronic colon displacement, sand enterocolopathy, intestinal abscessation, esophageal disease, and gastric ulceration. Extraintestinal causes include, but are not limited to, cholelithiasis, urolithiasis, neoplasia, abdominal abscessation, and thoracic disease. As gastrointestinal etiologies are the most common, it is often more rewarding to center the initial diagnostic evaluation on etiologies in the abdomen, as well as to begin with the least expensive and least invasive. However, physical examination findings should always guide your diagnostic efforts. In the problem oriented approach to weight loss, the problem is routinely characterized as either ‘weight loss with appetite’ or ‘weight loss without appetite’. This characterization may be helpful in illuminating various differential diagnoses for either problem, but may provide little by way of dictating the diagnostic evaluation (i.e. while the differential diagnoses may be different, the diagnostic evaluation is the same). Examining the findings of the history, physical examination, and diagnostic evaluation will hopefully highlight the cause and guide develop of a treatment regimen.

Diagnosis: Approach to weight loss
Due to the long list of differential diagnoses, and relatively limited diagnostic capabilities as compared to small animals (i.e. unable to perform abdominal or thoracic computed tomography or magnetic resonance imaging in adult horses), determining an etiology poses a diagnostic challenge.

When considering first opinion cases, empirical diagnosis/treatment may be appropriate. The author typically objectively calculates and recommends that the owner implement a diet that will facilitate weight gain. Additionally, deworming with moxidectin/praziquantel, assuming it wasn’t performed recently, is recommended to ensure that parasite infestation is not a component of the weight loss, and an oral examination with dental flotation is performed to ensure adequate ability to prehend, masticate, and swallow food. Following a period of time after these changes have been implemented, if the horse is continuing to lose weight, or failing to gain weight, the diagnostic evaluation is targeted toward specific differential diagnoses.

Diagnostic tests
Complete blood count and serum chemistry profile are inexpensive and non-invasive ways to evaluate for chronic inflammation, organ dysfunction, and possibly neoplasia. Complete blood count should include fibrinogen concentration, as it is an indicator of chronic inflammation. Serum chemistry profile should include liver enzymes (GGT and SDH), bile acids, renal evaluation (creatinine and electrolytes) and albumin/total protein concentrations. Information gathered from laboratory data can be used to determine further diagnostic tests to be performed, or can be used in conjunction with other test results to support a diagnosis for the cause of weight loss. Additional laboratory data should include a complete urinalysis with cytology to evaluate for infection or neoplasia of the urinary tract as a cause of weight loss, and may include serology for Streptococcus equi (M-protein titer) or Corynebacterium pseudotuberculosis (SHI test) when internal abscession is suspected.

Fecal flotation for parasites is also a valuable diagnostic tool. It is important to remember that larval cyathostomiasis does not always result in strongyle eggs in the feces; therefore, a resolution of clinical signs following empirical therapy with larvicidal doses of fenbendazole or moxidectin can be indicative of a diagnosis of cyathostomiasis. Additionally, infestation of tapeworms can result in colic signs due to attachment of the parasites at the ileocecal orifice. Fecal examination for tapeworms requires flotation with
saturated sucrose solution, but a positive response to empirical therapy with praziquantel or a double dose of pyrantel pamoate may be indicative of the diagnosis.

Abdominocentesis is a simple and inexpensive way to sample the abdominal fluid for cytology and possible culture. It is indicated in most cases of weight loss as it allows a direct evaluation of the abdominal microenvironment. Analysis of abdominal fluid for color, nucleated cell count, protein concentration and cytologic evaluation may support a diagnosis of peritonitis, hemorrhage, effusion, or neoplasia. Additionally, the abdominal fluid can be evaluated for bacterial culture, or PCR testing for Streptococcus equi or Rhodococcus equi infection/abscessation.

Transrectal abdominal palpation is an excellent way to evaluate the internal structures of the caudal abdomen and is indicated in cases of weight loss. Abnormalities, such as internal abscesses, neoplasia, colon displacement, or thickened bowel may be palpable and support the diagnosis, as well as justify further diagnostic efforts. Rectal mucosal biopsy can also be performed if infiltrative or infectious processes are suspected. Biopsy samples should be submitted for histology and culture. Unfortunately, this is the only large intestinal biopsy that can be performed without laparotomy, and often infiltrative diseases do not routinely affect the rectum, but the information provided can be helpful in making a diagnosis. Small intestinal biopsy samples can often be obtained transendoscopically from the most proximal aspect of the duodenum.

Small intestinal absorption of orally administered glucose can be used to determine if a horse’s ability/ inability to absorb nutrients is contributing to weight loss. Horses with inflammatory bowel disease, parasite infestation, or neoplasia may show absorption of glucose, supporting a diagnosis of malabsorption as a cause of weight loss. These horses may have signs of chronic colic or diarrhea in addition to weight loss, and thickened segments of small intestine are often noted on transabdominal ultrasonography.

Gastroscopy is a valuable diagnostic modality for horses with weight loss as it allows direct visualization of the upper gastrointestinal tract. Additionally, mucosal biopsy samples can be collected for cytology, histopathology, and culture/susceptibility. Horses that exhibit signs of colic after eating, or have a poor appetite as a component of the weight loss, may have gastric ulceration, indicating a need for gastroscopy. Gastroscopy may identify gastric ulcerations most commonly, but may also show gastric neoplasia or outflow obstructions. Additionally, a positive response to empirical therapy for gastric ulcerations with omeprazole, ranitidine, and/or sucralfate may support the diagnosis.

Transabdominal ultrasonography is currently the best way to visualize the structures of abdomen without having to perform exploratory surgery, and is indicated in most cases of weight loss as it allows indirect visualization of the abdominal organs, abdominal fluid, and abdominal wall. The duodenum and jejunum can be evaluated for thickened segments that may be consistent with inflammatory bowel disease (IBD), or neoplasia. Additionally, dilated segments may be indicative of chronically stranded areas aboral to the dilated segment. Thickened segments of colon, particularly the right dorsal colon, may be indicative of IBD, chronic right dorsal colitis, or neoplasia. The abdominal fluid can be evaluated for increased cellularity, as might be consistent with blood or inflammatory cells, or effusion. The liver, kidney, and spleen can be evaluated for organ abnormalities. Additionally, abnormalities in the intestinal lymph nodes may be visualized. Chronic left dorsal displacements may also be imaged with ultrasonography.

Thickening of the greater curvature of the stomach can be visualized in some horses with gastric ulceration or gastric neoplasia.

Thoracic ultrasonography is routinely performed in most cases of weight loss to ensure that occult pleural disease is not a component of weight loss. It can evaluate the visceral and parietal pleural surfaces, thoracic body wall, and mediastinum. Neoplasia, pleuritis, and abnormalities of the body wall have been reported as causes of weight loss.

Abdominal radiography is uncommonly used in adult horses for evaluation of weight loss; however, mineral opacities from enterolithiasis, large metallic foreign bodies, or sand ingestion can be visualized. If sand or stones are suspected, abdominal radiographs are indicated. Sand is most easily identified in the sternal flexure and if present in significant amounts, is visualized without difficulty. Enteroliths are difficult to visualize, but depending on the size and location of the stone, it may be diagnosed on a radiograph.

Thoracic radiography is indicated if the horse shows clinical signs of respiratory disease on the physical examination (which should include rebreathing examination). Typically, thoracic radiography is valuable for identifying abnormalities such as pulmonary or thoracic neoplasia, or chronic lower respiratory disease (interstitial pneumonia); however, other differential diagnoses such as fungal pneumonia or equine multinodular pulmonary fibrosis may also be identified. The findings on thoracic radiography, combined with physical examination findings, may also direct the practitioner to pursue other respiratory diagnostic tests such as transtracheal wash or bronchoalveolar lavage.

Laparoscopy can be used to visualize the surfaces of the abdominal structures and is indicated in cases where visualization is desired, but manipulation of the organs is not necessary. Laparoscopy is most useful for diagnosis of abnormalities in the dorsal abdomen, and acts as an alternative to exploratory laparotomy.

Exploratory laparotomy, as an elective diagnostic tool, is generally reserved for horses that have persistent colic in the face of extensive testing, weight loss, and no diagnosis. It is generally the last diagnostic test performed as it is highly invasive and expensive; however, it can be valuable as biopsy samples can be collected and organs can be directly evaluated. Frequently, the
diagnostic evaluation may lead to exploratory laparotomy, particularly in the case of intestinal masses where surgical resection and anastomosis may be a viable treatment option.

Additional diagnostic tests for extra-gastrointestinal causes of weight loss include evaluation of the urinary tract by cystoscopy for urolithiasis/pyelonephritis, or liver biopsy for hepatic disease. Typically, these tests are pursued when history, physical examination, clinicopathological data and transabdominal ultrasonography findings direct the practitioner to these body systems.

In summary, typically the cause of mild to moderate weight loss in horses can be remedied with good preventative care and an objectively evaluated feeding of high quality food; however, in some cases, extensive diagnostic evaluation is needed and the diagnostic plan should be guided by the physical examination and historical findings and knowledge of the differential diagnoses.

References available upon request.
Having worked with horses in a variety of roles, as a layman since 1942, and as a veterinarian since 1956, I have seen many traditions come and go and many trends develop. Here are some examples:

1. Horseshoeing: A tradition for millennia, until a European colleague began a recent trend for barefoot trimming. As so often happens, two opposing populations evolved: The fanatical anti-shoers who are enamored by whatever is “natural”, and the equally fanatic pro-shoers (which includes many farriers).
   - The truth? Many, many horses do not need shoes.
   - Some horses must be shod.
   - It varies with conformation, function, frequency of uses, ground surface, and most importantly, the condition of the foot.

2. Nutrition: There have always been nutritional errors in equine nutrition in my experience, usually due to excess rather than deficiency. What has changed is the nature of the excess. Whereas half a century ago this was mostly excessive grain, today it is excessive supplementation, processed feeds, “treats”, and just too much feed.
   - I am reminded of the young woman who called me to see her mare which was progressively losing weight at an alarming degree. I asked what she fed. The answer was “free choice hay and carrots”. I did a laboratory workup on the mare but found nothing wrong. Physically, she seemed normal except for the excessive leanness.
   - Puzzled, I returned to see the mare again. It was feeding time. The mare was fed a big flake of hay and a full wash tub of carrots. This was done twice a day. No room for the hay!

3. As I explained earlier, a leading cause of lameness, often permanent, is too much work at a young age. Although horsemanship, in general, has improved hugely during the past half century, one thing has worsened. Colts are being started too young and they are being over-worked, before they are orthopedically mature enough to withstand the stress of training.

4. Overprotection: Unnecessary blanketing, stall heaters, confinement, etc.
With the exception of the racehorse, colts were not traditionally started in physically demanding training disciplines, in our society, until the late 20th century.

Now, in most disciplines (English, Western, Classical, Draft) horses are started as two year olds and even younger.

In addition, selective breeding for exceptional speed, agility, and strength has increased the stress of training. As a result, the orthopedic damage, often permanent, traditionally seen in race horses from excessive work imposed on immature horses, is now a leading cause of unsoundness in other breeds and disciplines. This is inhumane, costly, and often tragic.
Each species' primary survival behavior is related to its' anatomy. The horse's primary survival behavior is flight, and the following nine behaviors are all related to the flight instinct. 2. The horse is uniquely perceptive. It's visual, olfactory, autistic, and tactile senses are exceptional, necessary in a flight creature. 3. The horse has the fastest response time of any domestic animal, necessary for effective flight. 4. Horses can be desensitized (habituated) to frightening but harmless stimuli with exceptional speed, IF correctly presented. 5. The horse is the fastest learner of all domestic animals. 6. Horses have the most persistent memory of all domestic animals. 7. Horses are the most easily dominated (accept leadership) of all domestic animals, if appropriate methods are used. 8. Dominance is established, primarily, by control of movement in the horse. 9. Like other species, the horse has its unique body language. 10. The horse is a precocial species which, unlike altricial species, has fully functional senses, and optimum learning ability and its imprinting capacity during the immediate postpartum period.
Update on Imprint Training
Robert Miller, DVM
Thousand Oaks, CA

I discovered the benefits of teaching foals immediately post-partum in 1959. I have learned of other values in the procedure from
other people that I was unaware of, despite handling thousands of newborn foals. These include:

1. If the mare was imprint trained, she will convey reassurance to her newborn foal while IT is being handled, facilitating
   the procedure.
2. The procedure eliminates post-partum mare aggression towards humans, a not uncommon behavior problem.
3. The method must be done correctly. The foal will learn the wrong things as swiftly and as lastingly as the correct
   things, so it is imperative that the trainer be properly instructed.
4. The “Dummy Foal” syndrome is not a rare problem. I have never seen an imprint trained foal end up as a “Dummy”.
   So I suspect the forcibly keeping the foal on the ground during the training procedure serves to emulate uterine
   pressures which has been shown to prevent “Dummy Foals”.
When I was a veterinary student, an important textbook was *Restraint of Domestic Animals*.

The tranquilizing drugs we rely upon so extensively today had not yet been developed. So physical restraint was an important part of veterinary practice.

Throughout my career I did clinical trials for various pharmaceutical companies, most of them agents for immobilizing patients. Many of those drugs were eventually approved and marketed. Most are still in use today. I have noted several changes in how we practice due to the availability and efficacy of these drugs:

1. The need for physical restraint has greatly been diminished. This has lessened the fear of our patients, the pain and discomfort to them that physical restraint so often inflicts, the consequent disapproval of clients who so often sympathize with and are bonded with their animals, the fear so many of our patients develop for us and for our office facilities.

2. An excessive and/or unnecessary use of such drugs. This can cause more financial cost to the client, and sometimes a loss in communication skills between doctor and patient. For example, I was taught to routinely twitch horses prior to administering vermifuges via a nasogastric tube. “Tube Worming” throughout most of my career was essentially the “bread and butter” of equine practice. I often spent entire days worming horses at large stables, farms, or at club or residential clinics.

I learned that, with time and patience, most horses could be trained to accept “Tube Worming” without restraint or sedation. This may have been initially time consuming, but eventually it paid off. I remember in 1987, going to a warmblood stable and “Tube Worming” 90 head of horses I had done many times before, using no restraint or sedation. There was one horse, number 91, that had just arrived from another state and I did have to sedate that horse.

Practicing that way, minimizing physical restraint, pleased many clients, enhanced my reputation and gave me satisfaction. I like horses and I want them to like me.

I like to share this with my colleagues.
Update on Training Newborn Foals
Robert Miller, DVM
Thousand Oaks, CA

I originally conceived of training neonatal foals to assure that they would become cooperative patients. Eventually I learned that the procedure enhanced future performance, facilitated future training, diminished the necessity for harsh methods, and helped to create a better human/animal bond.

I benefitted, the horses did, the owners did, and my practice did.

For many decades, the opposition to what I called “Imprint Training” was largely rejected for a variety of reasons: It was not traditional.

Finally, after more than half a century, the method now had rare criticism and is in use internationally. And, it is being widely used in other precocial species such as ruminants, elephants, etc.

CVC Lecture #4. Safer Horsemanship. Protecting Both Humans and Horses from Injury

Having done zoo practice throughout my career, working extensively with predatory species such as lions, tigers, etc., and with many other species such as elephants, zebra, buffalo, and dolphins, whales, the Great Apes, and all kinds of livestock, and having trained countless horses and mules, I can confidentially say that the horse is the most dangerous of all veterinary patients.

Only once in a lifetime involvement with equines, not only as a veterinarian, but as a drover, a packer, a bronco breaker, a wrangler, a breeder, and a trainer, was I hospitalized by a horse-caused injury.

That was because I have always practiced what I call “Defensive Horsemanship”. That means to always handle horses in ways to minimize injury to me, to others, and to the horse. Although these methods are very effective in minimizing the hazards of horsemanship, they are admittedly not absolute. Illustrated on the video screen, the risks in working with equines can be greatly reduced, if appropriate precautions are taken. These methods must be seen in order to be understood. This requires demonstration on live horses, or video or motion picture presentation.
You Work in the Most Dangerous of All Professions!
How to Teach Your Clients to Teach their Horses to be Cooperative Patients
Robert Miller, DVM
Thousand Oaks, CA

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Lymphoma connotes a solid-tissue tumor composed of neoplastic lymphocytes in visceral organs, skin, or lymph nodes throughout the body (Antinoff). To date, lymphoma is the most common malignant neoplasia reported in the domestic ferret at 10-15% of all neoplastic presentations in the US and Europe, and lymphoma is the third most common neoplasia of ferrets, behind adrenocortical neoplasia and insulinoma. It is documented as a spontaneous neoplasia (Mayer, Quesenberry), however there have been reports of horizontal transmission via cell and cell-free inoculation (Erdman), which suggests that there may a viral etiology, however an agent has never been reported. There is one report of Helicobacter mustelidae-associated (MALT) gastric B-cell lymphoma (Erdman), and this syndrome appears to mimic gastric B cell lymphoma caused H.pylori in humans.

Ferret lymphoma can occur across a number of age groups and has no specific sex predilections. In the early literature describing the disease, ferret lymphoma was classified by age of onset and assigned distinct prognosis, i.e. the aggressive and quickly fatal juvenile onset lymphoma form and the adult chronic onset form. This generalized classification scheme has been since retracted due to new clinical reports that reveal there is no specific age and cell-type trend. Most resources characterize lymphoma by cell line, i.e. large cell, lymphoblastic lymphoma (T cell) or small cell, lymphocytic lymphoma (B cell). Finally, there are several studies that report disease based on location, which include but are not limited to multicentric lymphoma (Ferreira), cutaneous lymphoma (Xi, Rosenbaum), malignant B-cell lymphoma with Mott cell differentiation (Gupta), polyostotic lymphoma (Long), epitheliotropic gastrointestinal T-cell lymphoma (Sinclair), focal thoracolumbar spinal cord lymphoma (Ingrao), myelo-osteolytic plasmacytic lymphoma in the femur (Eshar), and gastrointestinal lymphoma (Lee).

Due to the substantial variation in lymphoma classification in ferrets, there has been a call for an adoption of the standardized classification system for ferret (Mayer). Currently, most clinicians develop diagnostic plans to (1) stage, (2) grade, and when possible, (3) phenotype lymphoma in clinical patients. Until a universal classification scheme can be established for ferret lymphoma, most pathologists and oncologists characterize lymphoma based on the World Health Organization (WHO) staging system. Staging identifies the anatomic location of the neoplasia and the measure of dissemination throughout the body. A 5 level staging scheme (Table 1) has been adapted from Antinoff and Mayer. Cell morphology characterization, or grading, is also imperative when classifying lymphoma type and qualifies prognosis (Table 2). In clinical practice, large cell versus small cell, round versus irregular, and nuclear size are used to classify cell type and tumor behavior from tissue aspirates.

<table>
<thead>
<tr>
<th>Staging</th>
<th>Characteristics</th>
</tr>
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<tbody>
<tr>
<td>Multicentric</td>
<td>Multiple Lmn, usually on both sides of diaphragm, may also involve liver, spleen, bone marrow, or other extranodal sites</td>
</tr>
<tr>
<td>Alimentary</td>
<td>Solitary mass within GI tract or mesenteric node, multiple masses with or without regional involvement of intra-abdominal node</td>
</tr>
<tr>
<td>Mediastinal</td>
<td>Mediastinal Lmn, not involving the thymus</td>
</tr>
<tr>
<td>Extranodal</td>
<td>Renal, CNS, Ocular, Cardiac</td>
</tr>
<tr>
<td>Cutaneous</td>
<td>Severe skin ulceration with or without nodular skin masses, usually seen along the ferret, sacral area, inguinal area, extremities</td>
</tr>
</tbody>
</table>
Table 2. Grading Cell Morphology for Ferret Lymphoma

<table>
<thead>
<tr>
<th>Nuclear Size (relative to [RBC] Size)</th>
<th>Small: ≤ 1 RBC</th>
<th>Medium: &gt;1 but &lt; 3 RBC</th>
<th>Large: ≥ 3 RBC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitotic Index</td>
<td>Low: &lt; 3</td>
<td>Intermediate: 3-8</td>
<td>High: &gt; 8</td>
</tr>
<tr>
<td>Other Descriptives</td>
<td>Nuclear morphology</td>
<td>Nucleoli</td>
<td>Round</td>
</tr>
</tbody>
</table>

Phenotyping provides vital prognostic information and a definitive diagnosis. If tissue can be acquired, immunohistochemical stains help define the cell line as B or T cell. CD3 is a T cell marker, and CD 79α is a B cell marker. As in feline and canine medicine, the prognosis is based on cell line characterization, which predicts the progression of the disease process and response to treatment. As flow cytometry becomes more widely available for ferrets (Music), this may also be used to help define cell lines based on blood sample acquisition alone.

### Clinical signs, physical examination and diagnostics

There is no universal clinical presentation for ferret lymphoma, and in some cases ferrets are asymptomatic. Clinical signs are nonspecific, but can include lethargy, anorexia, weight loss, ataxia, weakness, diarrhea, dyspnea, and respiratory signs (Suran, Antinoff, Mayer). Gastrointestinal signs are common, however this should not be confused with chronic gastrointestinal disease. Mesenteric and peripheral lymphadenopathies can be present with other disease processes, this clinical finding is not pathognomonic for lymphoma, however, intra-abdominal lymphadenopathy occurs with multicentric lymphoma in ferrets.

The diagnostic approach for ferrets with suspected lymphoma should include a minimum database (CBC, biochemistry screen, urinalysis), diagnostic imaging (radiographs, ultrasound, CT or MRI), and cytology obtained from aspirates, however biopsy tissue is preferred. The most consistent CBC finding in ferrets with lymphoma is a nonregenerative anemia. (Ammerbach). Lymphocytosis and thrombocytopenia are rare. If a lymphocytosis is present, with total white blood cell counts exceeding 30,000, a true lymphocytic or lymphoblastic leukemia may be present (Mayer). Hyperglobulinemia has rarely been reported in ferrets with multiple myeloma, however a serum or urine protein electrophoresis may help characterize if a monoclonal gammopathy is present (Eshar). It is important to note that the presence of a hyperglobulinemia is also a hallmark of ferrets that suffer from Aleutian’s disease, which is a viral condition that also causes a hyperglobulinemia (Hess). In some cases, biochemical analysis reveal elevation in liver and renal enzymes if disease is causing organ function compromise, but these findings are not specific to lymphoma. One report of a hypercalcemia as a paraneoplastic syndrome of lymphoma has been reported (Fisher), however it is rare.

Cytology from organ or lymph node aspirates, as seen in humans, show poor correlation to definitive histologic diagnosis based on WHO classification, however it can provide the bases for tissue sampling based on cytological features (Antinoff, Hehn). Cytologic hallmarks for lymphoma are a monomorphic population of lymphocytes and the absence of peripheral blood elements (Antinoff). Evaluation of these samples by experienced pathologists is imperative, as false positives due to misdiagnosis in ferrets with reactive lymph nodes can occur. Once study revealed that the lymph node cell distribution in normal ferrets includes 50-60 small lymphocytes, 2-3 lymphoblasts and promylocytes, and 0-1 macrophages, plasma cells and nondegenerate neutrophils per 200 x field (Paul-Murphy). Biopsies are strongly recommended for a definitive diagnosis. Gastric biopsies have been described. Scapular and popliteal lymph node biopsies can be easily obtained with little surgical complication in ferrets. Patients that present with bone lesions should undergo bone marrow aspiration or careful bone biopsy to further characterize aggressive ostotic disease.

Diagnostic imaging can help aid in staging and collecting samples for evaluation. In a recent study evaluating the image findings in ferrets with lymphoma, Suran et al. concluded that the most common imaging finding was intra-abdominal lymphadenopathy and mild peritoneal effusion in ferrets with multicentric lymphoma, which is the most commonly reported in ferrets greater than 3 years of age. Characterization of lymph nodes on ultrasound revealed hypoechogenicity as the single most consistent abnormality, as lymph node sizes were within reported ranges. Increased changes seen in cat and dogs, such as increased short to long axis length ratios rations, hyperechoic perinodal fat, nodal heterogenicity, irregular nodal contour and shapes, were not appreciated in ferret lymphoma cases (Suran). Splenic infiltration was noted but correlation to splenomegaly should not be assumed, as extramedullary hematopoiesis occurs
in ferrets and this can confound assessment for the cause of splenomegaly. Extranodal infiltration was then characterized in the liver, kidneys, lungs, and in aggressive bone lesions.

Management
There are several modified chemotherapeutic protocols, which should be chosen based on consultation with a knowledgeable oncologist. There are 3 common protocols that have been adapted for use in ferrets with lymphoma, and BSA calculations have been validated for the species, which is 9.94 × (body weight)²/₃ (Jones 2015) with weight and m² charts available. The Tufts protocol provides an intravenous-free 12 week chemotherapy protocol that employs L-asparaginase, cyclophosphamide, cytabarine, predniisolone, leukeran, and procarbazine, and methotrexate. The Gulf Coast chemotherapy protocol utilizes a 52 week protocol that employs L’asparaginase, prednisone, vincristine, and cyclophosphamide (Antinoff). Older protocols employ vincritsine, asparaginase, prednisone, doxorubicin, cyclophosphamide, and methotrexate. All physical protocols and dosages are available in the Ferret, Rabbit, and Rodents third edition chapter for Neoplasia in ferrets, (pp112-115). Copies available upon request. For localized cutaneous lymphoma, surgical excision of lesions may help improve quality of life, however recurrence is common without concurrent chemotherapy. Radiation therapy has been employed to reduce tumor size as rescue treatment for solitary mediastinal masses to relieve respiratory distress. While the tumors are very radio-sensitive, due to the ferret’s body conformation, limiting radiation exposure to other organs can be very challenging.

Adjunctive therapies include optimizing nutrition, screening for leukopenia (< 1,000) and neutropenia and providing systemic antibiotic therapy when indicated, and co-management of additional morbidities, which often include management of Helicobacter gastritis, and other neoplastic conditions (adrenocortical neoplasia, islet cell tumors). Avoid employing homeopathic therapies without consulting a knowledgeable specialist, as some treatments can and will cause harm. One such case has been proven in a clinical reports in dogs, receiving bloodroot (Sanguinaria canadensis) treatments, and the agent has been found to cause dermal necrosis (Childress).

Prognosis
Survival times strongly correlate with cell type and dissemination. Staging also heavily influences survival estimates, as disseminated T cell lymphoma may result in shorter estimates. In one study evaluating the phenotype, treatment and survival of 29 ferrets with lymphoma, Ammerbach et. al concluded that the mean survival of ferrets not immediately euthanized was 5.0 months (T-cell lymphoma) and 8.4 months (B-cell lymphoma). Ferrets treated with chemotherapy survived an average of 4.3 months (T-cell lymphoma, n = 9) or 8.8 months (B-cell lymphoma, n = 4). Ferrets in this study were diagnosed with peripheral T-cell lymphoma (n = 17), anaplastic large T-cell lymphoma (n = 5), anaplastic large B-cell lymphoma (n = 4), diffuse large B-cell lymphoma (n = 1), and Hodgkin-like lymphoma (n = 2).

Conclusion
Ferret lymphoma is one of the most common neoplasias recognized in practice. Clinical staging, grading, and phenotyping can help optimize treatment approach and qualify prognosis. Several chemotherapy protocols have been adapted for use in ferrets to help with ease of administration and improve compliance, improve quality of life, and reduce drug-induced morbidity.

References
According to the 2015-2016 American Pet Products Association Annual Survey (http://www.americanpetproducts.org/press_industrytrends.asp), there were a reported, 14.3 million birds, 9.3 million pet reptiles and 12.4 million pet small mammals in US households. The demand for advanced medical care for these species has grown, but the clinical comfort of practitioners also needs to grow to match this demand. The best way to know improve care for these patients is by first knowing their owners. Ultimately, to improve their care, you will need to shape the behaviors of the human that’s attached to the beloved pet. When you understand their attachments, their strengths, and their commitment, you gain the trust of those who need your services. Let’s take a peak into different pet owner personalities and outline ways to improve compliance for your patients.

Rabbit owners
In general, most rabbit owners share one common thread; they love their bunnies. Chinchilla owners can be very similar. There are 2 types of owners, the well-meaning and the super-informed. The type one owner loves their rabbit, likely has dated husbandry practices but has a huge heart and is open to change. The type 2 owner may almost seem obsessed with minute changes in the rabbits behavior, and you should grow to trust their observations. They know their bunnies and are keen observers. They can pick up on subtle changes and that’s worth its weight in gold. For both clients, give them homework. Provide House rabbit society homework for the Type 1 and “academic” level homework for the Type 2. I often direct Type 2s to anatomy and physiology papers to read- this qualifies that you have a thorough understanding of medical physiology and that guides your treatment practices. Incorporate the owners into the treatment plan. When they feel they are contributing to the solution and are working with you, their compliance can be phenomenal.

Ferret owners
Ferret owners are not too dissimilar from some rabbit owners, except that their long-term memory and attention spans can sometimes be faulty, just like a ferret! Type 1 owners are very well-meaning, but compliance can be poor. Type 2 owners will do WHATEVER is necessary to save their ferrets and are open to advanced surgical and medical treatment options. For both owners, I recommend talking to them directly WITHOUT the pet in the room. They can become very distracted and miss specifics of your clinical findings and recommendations. Make sure you have “Ferret” disease information sheets on hand, including “Diarrhea, Insulinoma, Adrenal Gland Disease Handouts” so that they have a tangible reference. When a ferret owner is distressed, all they care about is relief for their loved pets. Do not be surprised that you will have to repeat yourself. Also send them home with treatment charts to help them stay on track. Have reminder calls/cards go out frequently for these clients to help improve compliance.

Guinea pigs
Similar to Type 1 and Type 2 rabbit owners. There is a Type 3, the super-devoted and very attached adult owner. These owners are incredibly sympathetic to their pets needs and have C&C cages that occupy entire rooms for their pigs. Avoid making the mistake of assuming monetary value for human-animal bond value. These owners often seek advanced medical care for their pigs and have excellent husbandry and compliance. Have common disease hand-out sheets ready and give them homework!

Rats
Many cat and dog owners argue that their pet rats are the most intelligent and the superior companion of their pets. Usually owners that are seeking rat care are extremely invested in their pets’ well being. Many come in desperate for care. The common diseases that afflict rats can be fatal and their lifespans are so short. It’s always a bittersweet bond. Owners are often prepared for any salvage or supportive care measures to prolong life if possible. There are many advanced care and preventative care options you can afford these owners to prolong the life of these enriching pets. By providing a compassionate approach to their care, even compassionate euthanasia options, you will undoubtedly gain you a loyal rat owner clientele.

Bird owners
There are 3 major owner types, and there are mixes of some! The Type 1 owner is the well-meaning but misinformed. They perhaps adopted the bird from a bad situation or may be first time bird owners that impulse-buy, etc. It’s clear that they care but they need guidance to maximize the bird’s physical AND mental health. Type 2 owners tend to be informed and do not trust veterinarians. They have their reasons, some may have been mistreated, their pets not treated and they require a lot of reassurance. Kill them with kindness and be honest. They are like their pets, they will either choose to accept you or you can kindly offer the contact information
of colleagues who are avian practitioners. Type 3 owners I lovingly refer to as the “ornithologists”. They have a deep ethical commitment to their feathered companions and are keen to make sure you know that. This is fine. Work to reassure them with your honesty, not with irritation. Have websites, husbandry references, bird club information and medical hand-outs ready; this will reveal to the owner that you are well informed. Human-avian bonds have a range of medical, mental and emotional spectrums and consequences involved. Be prepared to have a trusted behaviorist on stand-by and be honest about what you are qualified to treat- the bird’s MEDICAL illness.

Reptile owners
If you thought gaining the trust of a reptile owner was difficult, you’re right, it can be. Historically, many practitioners have held common negative stereotypes about reptile owners. Poor husbandry means that the owners do not care about their pets. And worse, reptile owners often do not have the financial means to pay for veterinary care. You can kick all of these beliefs to the curb! Reptile owners have doubts about practitioners. They may not know who can provide clinical care and who has expertise to provide that care? The cards have changed. We practitioners have a diverse group of animals to serve and the owners expect more from us. The author has been each of these owners and hopes that practitioners can appreciate the plight that the owners that own these very unique pets.

First, we have the Herper Kid owner type. Young naturalists are usually very intelligent and motivated about their pets. Most importantly, you can influence them to get compliance from their parents. Make sure you have handouts and homework in hand. Talk to the child about their pets, get excited and you both can form a team to get mom and dad on board with getting what’s needed for the pet. Often, they are more attentive than adults to the very subtle changes reptiles display when ill.

The “Owner” by Default may also walk into your office. First time guilt-driven reptile purchases, adoptions, or even the parent of a child heading off to college! These owners have an attachment to the animal and an investment in their well-being. They may not know all there is to know about husbandry. Provide resources and a community for these owners, turn their fear into pride about their new acquisitions! The Executive Herper is the owner whose pet is a reflection of status. They take pride in ownership. They expect top of the line and expect you to offer it. Surprised them with how much you know and celebrate their love for their pet. The HardCore Herper or “herpetologist” is the owner whose been neglected by veterinarians for years and has taken most measures into their own hands for medical care. In this case, you will have to abandon your vet ego. They DO know more about the animal’s natural history than you do. They have been caring for herps without help for years. Befriend them, learn from them and instill trust in them. If they own a business, that trust will set you up to care for collections.

Zoo keepers and curators
These are the ultimate patient advocates. The relationships can be rewarding but are ultimately met with challenges. Keepers can view themselves as the animal’s owner and decline or refuse your treatment recommendations. The reasons for this can be variable, but usually involve changing their trust and interaction dynamic with the patient, which can be dangerous for the keeper and the animal. Curators have an extremely difficult job, they sometimes have to make a decision based on prognosis and animal value, and their decisions affect all animals in the zoo. Be patient and offer unique and collaborative ways to manage patient care. This means you will also have to become friends with compounding pharmacists who have experience with unique species.

Mistakes to avoid
It’s important to remember assigning your value or a monetary value to the animal does not reflect the human animal bond values. Owners form strong bonds- no matter the species, be prepared to offer them all options. It may also be wise to set up “husbandry consultation” appointments that are separate from “recheck/consult” or “wellness exam” appointments. Many exotic pet owners are excited to meet vets that love their animals as much as they do, and this can lead to lengthy appointment sessions. Provide the owners with many options but carefully outline them with “specific time allotments” so that they do not feel dismissed because of your clinic schedule.
Managing Head Trauma in Exotic Pets
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Traumatic brain injury results from some compressive and blunt force to the skull. Often the trauma has affected other parts of the body as well. When triaging an exotics patient, the initial goals are to stabilize the extracranial disease, then proceed to stabilize intracranial injury while critically monitoring neurological status. In animals that have sustained trauma, extracranial trauma may include chest injuries, abdominal injuries, airway obstruction, compromise of oxygenation via ventilation or primary lung injury, and intravascular volume compromise, secondary to hemorrhage. When the extra-cranial conditions have been stabilized, the next steps to triage intracranial injury include (1) optimizing oxygen delivery to the brain, (2) maintaining cerebral perfusion pressure or CPP, (3) treating increased intracranial pressure or ICP, and (4) consistent neurologic monitoring.

The pathophysiological consequences of head trauma are often broken into two categories. First, primary injury results from concussive forces to the skull physically disrupting intracranial structures, causing a brief loss of consciousness that results from parenchymal hemorrhage and edema. Contusions can result in losses of consciousness for longer periods of time. The most severe concussive forces to the skull physically disrupting intracranial structures, causing a brief loss of consciousness that results from compression. These primary injuries can cause cerebral edema, increased CSF volume due to obstruction of flow by edema or clot formation, compromise of the blood-brain barrier, vasospasm, infection, and seizures, which can all cause neuronal death.

Secondary injuries result from a cascade of inflammatory processes and biochemical events that signal neuronal cell death. These biochemical events may result from systemic insults (hypotension, electrolyte imbalances, acid-base imbalances, hypoxemia, hyper and hypocapnia, hyper and hypoglycemia, hyperthermia) and/or intracranial insults. After impact, there is a substantial release of excitatory neurotransmitters that cause an influx of calcium and sodium into the neurons. The mechanisms of removal are overwhelmed, and this leads to intracellular damage and ATP depletion. Reactive oxygen species, nitrous oxide, and catecholamines also contribute to a continued biochemical insult to injured neurons.

The uninjured brain can tolerate a variance in mean arterial blood pressure (50-150 mmHg), yet maintain cerebral perfusion pressure through auto-regulatory means. When the auto-regulatory mechanism is lost due to brain injury, hyperperfusion and local tissue acidosis can occur. Cerebral blood flow correlates linearly to systemic blood pressure when the CPP goes above or below the aforementioned range. Cerebral perfusion pressure (CPP) is the difference between MAP and ICP. Therefore, slight increases or decreases in MAP, coupled with expected increases in ICP, significantly affect CPP. The CNS ischemic response, or Cushing’s reflex, may be an initial response to decreases in MAP, whereby there is a reflex increase in MAP and a decrease in heart rate. This is seen with life-threatening increases in ICP are present and merit aggressive treatment to reduce ICP immediately.

Triage
On presentation, vital signs should be assessed and when possible, completing a modified Glasgow Coma Score and Animal Triage Trauma Score can help qualify the animal’s prognosis during initial assessment. The MGCS can be found at http://bvns.net/wp-content/uploads/2016/09/Neurotransmitter-2.0-MGCS-final.pdf. The animal triage trauma score can be found at http://www.k9tecc.org/assets/Animal_Trauma_Triage_Score.pdf.

For exotics patients, including reptiles, the patients should initially receive oxygen support during the visual and physical examination. During triage, the attending clinician should aim to qualify the animal’s mentation, access HR, RR, perform a thorough evaluation of the skull and cranial nerve exam, obtain an estimate of systemic blood pressure, and obtain an estimate of hydration status. In addition to attending to any additional extra-cranial injuries that have occurred, obtaining a weight or weight estimation will be key. The patient should then be placed on heat and oxygen support as needed in preparation for stabilization procedures and diagnostics. When safe to collect, a biochemistry screen and acid-base evaluation will be imperative. The author often monitors urine chemistries for changes in USG, pH, ketones, blood and glucose, in lieu of repeated venipuncture attempts to follow trends and assess the patient’s metabolic and hydration status. Radiographs, CT, and MRI can be pursued when the patient is stable. More often than not, the authors require 24-48 hours of stabilization minimal before attempting imaging diagnostics. When permissible, CT is preferred for speed of image acquisition, reduction in handling (patient is put in a small induction chamber with oxygen and no anesthetic), and the image detail acquired across all species orders (patients as small as 100 grams).

Mentation
For exotic animal patients, a qualification of their mentation may be difficult, as several prey species are adept at hiding signs of illness. Owner report will be important in assessing their status, however if the patient is indifferent to your presence, has acute vision deficits, does not respond to handling, and does not engage in exploratory behaviors, you can begin to qualify their status as altered, obtunded, and in severe cases, moribund for unresponsive patients.
Skull exam & cranial nerves
Evaluation of the skull will be key in pre-emptive diagnosis of potential skull fractures. In avian and reptile species, the eyes often take the brunt of rostral concussive injuries. Structurally, the presence of scleral ossicles further supports their large orbit and the skull. Any ocular trauma in these species merits a good evaluation of the skull. In avian species, the presence of green bruising may be appreciated, and on the dorsal aspect of the head, the skull sutures can be distinctly seen through the skin, which can help rule out the presence of displaced frontal bone fractures. In certain lizard species, auricular hemorrhage can be noted upon visualization of the tympanum. Small herbivores often sustain ocular injury secondary to head trauma, however head position abnormalities appear to be a common finding. Seizure activity may also alter the mentation in the post-ictal phase. Owner report of the animal’s immediate mentation after injury and any report of seizure activity will be important to acquire prior to assessment. The cranial nerve examination for small mammals, avian, and reptile patients is relatively comparable with that of cats and dogs with few exceptions.

Evaluation of the pupil position and size will help the clinician qualify prognosis. Small mammal prognostic indications for lesion localization have been used for avian and reptile species with success. In birds and reptiles, a consensual PLR is not appreciated due to differences in optic nerve desiccation and the response to light is present and controlled, but not voluntary. These is due to the presence of skeletal muscle in the ciliary bodies, as compared to smooth muscle in mammals. In all cases, abnormal PLRs, the absence of PLRs, and abnormal pupil position and size all follow the similar prognostic indicators. It is important to note that the normal PLR response of small herbivores and prey species will likely appear slow in comparison to that of cats and dogs. In the author’s experience, anisocoria can occur several days after head trauma insults in birds, which may be less likely in small mammal medicine, provided additional injuries are not occurring after the primary insult.

*Pupil size, reactivity, and prognostic

<table>
<thead>
<tr>
<th>Pupil Position</th>
<th>Response to Light</th>
<th>Level of Lesion</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midposition</td>
<td>Normal</td>
<td>---</td>
<td>Good</td>
</tr>
<tr>
<td>Bilateral Miosis</td>
<td>Poor to none</td>
<td>Cannot localize</td>
<td>Variable</td>
</tr>
<tr>
<td>Unilateral Mydriasis with ventromedial strabismus</td>
<td>Poor to none</td>
<td>Midbrain</td>
<td>Guarded to poor</td>
</tr>
<tr>
<td>Midposition</td>
<td>None</td>
<td>Pons, medulla</td>
<td>Poor to grave</td>
</tr>
<tr>
<td>Bilateral Mydriasis</td>
<td>Poor to none</td>
<td>---</td>
<td>Poor to grave</td>
</tr>
</tbody>
</table>

*Silverstein, Small animal critical care medicine. Elsevier Health Sciences; 2014

Hydration & blood pressure
In avian species, a quick evaluation of the ulnar wing vein distension will help qualify hydration status and perfusion. Healthy avian patients can sustain losses of 30% total blood volume before showing signs of shock. Avian patients regenerate quickly, and PCVs can normalize within 24-48 hours. In reptiles, assessment of the periocular fat pad resilience, mucous membranes, and skin fold assessment may help qualify hydration status. In snakes, distension of the palatine vessel can be appreciated, but should be avoided to prevent additional head injuries. In small mammals, auricular and/or femoral pulse evaluation, and conjunctival color evaluation can help qualify hydration and estimated blood pressure. In rabbits, a compensatory tachycardia may not be appreciated in response to severe hypotension, as vagal nerve innervation is found near the carotid bodies. In many cases, subjective assessments will be required as objective monitoring equipment may be limited due to the animal’s size and/or lack of validation for the species. Monitoring for subjective trends, in conjunction with HR, will be labor intensive yet pivotal in enabling the clinician to respond to the patient’s status.

Treatment
After qualifying the severity of neurologic compromise, the treatment regimen mirrors those steps taken in small animal medicine. Extra-cranial therapy will include oxygen support, maintaining airway patency, heat support, reduction of environmental stressors (loud noises, bright lights, predators), and temporary stabilization of other wounds. Airway patency in small herbivores will be contingent upon reduction of compromise to the nasal passages, as many are obligate nasal breathers. In avian and snake patients, air sac cannulation may be warranted if there is obstructive upper airway disease. In small mammals, pleural effusions may need to be addressed to help reduce hypoventilation and thoracocentesis has been described. When possible, during initial stabilization, the animals head should be elevated to help reduce cerebral blood volume, increasing venous drainage and safely decreasing ICP. This can be achieved with using towels arranged in a cage that would mirror a slant board for cats and dogs, which are used to provide a 30-degree slant. When the patient’s head is positioned, it is important to make sure that there is no compression or kinking of the neck or jugular vessels, as this could prevent drainage from the head and increase ICP.

Fluid therapy, electrolyte monitoring, oncotic support, and intravascular therapies aimed to reduce increased ICP will be essential for intracranial therapy. Exotic animal practitioners are faced with the challenge of providing these options without causing injury of exacerbating existent disease. In most all exotic species, vascular access will be necessary to provide emergency access for fluid
therapy, oncotic support, transfusion therapy, and treatment for increased ICP. Reviews for intravenous and intraosseous catheter placement are available.

Species specific crystalloid and colloid fluid rates are variable, however some general guidelines for dosages are listed below. Colloid support is used as that same rate across species. Hetastarch is provided at 5 ml/kg slow bolus over 15 minutes, not to exceed 20 mls/kg/day.

- **Avian Patients:**
  - Crystalloids: 50 ml/kg/day SC, IV, IO,
- **Reptiles:**
  - Normosol, Plasmalyte: 15-35 ml/kg/day depending on species water requirements
- **Small Mammals Crystalloid fluids:**
  - Rabbits, chinchillas, guinea pigs: 100 ml/kg/day
  - Ferrets, rats, mice, sugar gliders, hamsters, hedgehogs: 60 mg/kg/day
  - Degus and gerbils: 50 ml/kg/day

The use of hypertonic saline or mannitol may be indicated in cases of apparent ICP. The author has used the following doses with guidance from a criticalist, in birds, reptiles and small mammals with success. Mannitol can be given, to help reduce ICP and improve CBP and increase oxygen delivery to the brain via osmotic reduction of brain edema. The author has had success with 25% mannitol, administered 0.5 g/kg over 15 minutes in mammals and 0.25-0.5 g/kg IV/IO over 15 minutes in birds. In cats and dogs, the range is 0.5 -1.5g/kg IV over 15 minutes. At high dosages, mannitol can cause a severe hypotension and has induced acute renal failure in humans. Additionally, it has been shown to leak into the brain parenchyma during prolonged use, and this can exacerbate edema. Mannitol must be followed with crystalloid and/or colloid therapy to maintain intravascular volume. Hypertonic saline is often used as a safer alternative to mannitol. As sodium does not freely cross the blood-brain barrier, it provides the same osmotic effect to reduce brain edema and provides intravascular volume without causing hypotension. The author has had success with 7.5% NaCl at a dose of 4 ml/kg or 3% NaCl at a dose of 5.4 ml/kg at 15-20 min IV or IO in birds and mammals.

In addition to fluid therapy and treatment of ICP, maintaining adequate glucose support will be necessary for exotics patients, as most have high metabolic demands. Dextrose supplementation (2.5-5%) in the crystalloid fluids may be necessary until the patient can recover enough to regain hunger. The use of anti-inflammatories may be considered provided the patient’s bloodwork support’s safe usage. The author uses doxycycline as an alternative to NSAIDs in compromised patients. Steroids are contraindicated and have been shown to be associated with worse clinical outcomes in clinical trials. Furosemide is reserved for cases were pulmonary edema and/or oligoanuric renal failure are present. Furosemide is not used to reduce cerebral edema as a sole agent because it can cause a decrease in intravascular volume and subsequently decrease CPP. Seizure management will become vital in reducing continued hypoxic threat during recovery. The use of phenobarbital as primary method may be species dependent and should be reserved for cases that are refractory to other anti-epileptics. Midazolam and keppra are commonly used to treat status and keppra is used as a first-line preventative agent. Doses for small mammals have been extrapolated from small animal medicine, which includes 60 mg/kg to treat status and 20 mg/kg q8 hrs for maintenance. Studies in birds reveal that therapeutic doses are achieved at 100 mg/kg q12h.

Pain management can be variable for different species. However, the authors often provide tramadol for avian (15-30 mg/kg q8) and reptile (5-10 mg/kg q24-48) patients. Gabapentin is also provided small mammal (5 mg/kg q8), and avian patients (15 mg/kg q8). The addition of gabapentin may also add in seizure management. In efforts to reduce nausea, Cerenia 1 mg/kg IV/SQ q 24 has been used in small mammal, avian, and reptile patients. Nutritional support is always provided; however syringe feeding is often implemented if need when the patients clinical signs are improving, which may take 24-48 hours.

**Long term consequences of traumatic brain injury**

If the patient shows improvements in blood pressure stabilization, hydration status, mentation, and oxygenation, the prognosis is good. If declines in neurologic status, systemic blood pressure, or additional injuries occur, this is associated with a poor to grave prognosis. On average, the author asks the owner to commit to a minimum 3-4 days of hospitalization, and more often it requires 7-10 days before the patient is released. Vision defects and/or persistent changes in mentation may occur after initial injury. Certain changes may be permanent. Seizure management may need to be maintained, as seizures may occur and/or recur weeks to months after initial injury. Young animals appear to have a better prognosis in recovering from severe traumatic brain injuries.

**References**


In herptile species, clinical signs of nutritional disease are like those seen in mammals. Calcium and phosphorus imbalances, diseases associated with hypercholesterolemia and obesity, vitamin D3 deficiencies, and vitamin A deficiencies remain common nutritional disorders in captive reptiles and amphibians. Many herptile caregivers have minimal information about the basic nutrition content of invertebrates or how to optimize it prior to feeding herptiles. This review will cover basic herptile nutrition, invertebrate diet preparation, and common nutritional diseases. To help make nutrition content easier to present to caregivers, invertebrate nutritional information is compared to common human foods. Additional information about insect composition and nutritive value can be found in the attached article (Updates on Amphibian Nutrition and Insect Nutritive Value).

**What we know**

Invertebrate nutrient composition has been formally studied for more than 50 years. Research on invertebrates for reptile and amphibian (herptile) nutrition is most extensive for the following invertebrates:

- Domestic cricket (*Acheta domesticus*)
- Earthworm (*Lumbricus terrestris*)
- Silkworm (*Bombyx mori*)
- Mealworm (*Tenebrio molitor*)
- Soldier fly larvae (*Hermetia illucens*)
- Supercrumb (*Zoophobus morio*)
- Madagascar hissing cockroach (*Gromphadorhina portentosa*)
- Butterworm or tebo worm, (*Chilecomadia moorei*)
- Turkistan or red rusty cockroach (*Blatta Lateralis*)
- Fruit fly (*Drosophila melanogaster*)

Although not formerly studied, enthusiasts also use the following as invertebrate feeders:

- Dubia cockroach, (*Blaptica dubia*),
- Hornworm (*Manduca quinquemaculata*),
- Pill bugs (*Armadillidium vulgare*)
- Springtails (*Collembola sp.*)

**What we don’t know**

The National Research Council (NRC) provides recommended minimum nutrient requirements for domestic and farmed animals based on comprehensive reviews of nutrition studies. No such database exists for herptile species and nutritionists often utilize the minimum requirements of laboratory rats or carnivores for insectivorous herptiles. The following is a review of major nutrient components and compares invertebrate composition to recommendations for rats, consistent with much of the herptile insectivore literature. This summary is based on the Nutrient Requirements of Laboratory Rats (http://www.nap.edu/openbook.php?record_id=4758&page=R1), unless otherwise stated.

**Energy needs, fat, protein**

Metabolizable energy is defined as the amount of net energy gained from food less the energy for digestion and absorption of the meal. This is influenced by species, age, activity, and environmental temperature. ME is measured as kilocalories per kilogram (kcal/kg). Invertebrate feeders typically range from 0.7 - 2.7 kcal ME/g, below rat minimal maintenance energy of 114 kcal ME/BWkg0.75 per day or 3.6 kcal ME/g.

The diet protein content reflects the energy concentration based on amino acid composition and availability. For rats fed a natural-ingredient diet, a minimum of 50 g/kg protein content is required. [The crude protein amount of larval insects may exceed the minimum requirement for rats by 2 - 4 times. The digestibility and bioavailability of insect protein is poorly researched, even in well-studied species. In one study, phoenix worms were passed undigested by mountain chicken frogs (*Leptodactylus fallax*), as the exoskeleton prevented digestion. When the larvae were macerated, the bioavailability improved. In addition, chitin in the exoskeleton and cuticle is not bioavailable to herptiles and amino acids in these structures vary in bioavailability.]

Fat (lipid content) provides a concentrated energy source, aids in fat-soluble vitamin absorption, provides essential fatty acids, and usually increases diet accessibility or palatability. The optimal lipid requirement for growing rats is 5% or 50 g/kg of diet fed, which also assures adequate vitamin A absorption. The total fat content of many larval insects is 3 - 6 times the optimal lipid requirement for rats.
Vitamins & minerals

Reptilian and amphibian vitamin and mineral requirements and absorption physiology may be considerably different than mammals.

Active forms of vitamin A include retinol, β-carotene, and retinyl esters. The absorption of Vitamin A, as a fat-soluble vitamin, corresponds with an adequate lipid content in the diet. Hypovitaminosis A is a common clinical problem in herptiles and can result in vision loss, epithelial hyperplasia, squamous metaplasia and keratinization of mucosal epithelium, growth failure, dermal ulcerations, and bone defects. In captive anurans, an inability to use the tongue effectively for prey apprehension, called “short tongue syndrome” is recognized. The minimum retinol requirement for rats is 2,300 IU/kg. Most invertebrate species contain less than 300 ug/kg or 1,000 IU/kg of retinol. Silkworms have the highest content at 1580 IU/kg.

Dietary supplementation has been reported by administering 0.1 mg liver from frozen food rodents (20 ug liver = 66 IU vitamin A) PO once a week to treat peri-orbital squamous metaplasia secondary to hypovitaminosis A in a Tiger salamander (Ambystoma tigrinum). Topical absorption studies have been performed in yellow and blue poison arrow frog, (Dendrobates tinctorius), New Guinea tree frog (Litoria infrafrenata), African foam nesting frog (Chiromantis xerampelina), and Puerto Rican crested toad (Peltophryne lemur). Collective results suggest that topical application of water miscible vitamin A palmitate (Aquasol A® parenteral) at 50 - 100 USP for frogs less than 20 gram and 100 - 150 USP for frogs greater than 20 grams resulted in resolution of short tongue syndrome and dermal ulcerations and, in the New Guinea tree frogs, significant increases in serum vitamin A levels. Parental supplementation must be provided with care, as iatrogenic hypervitaminosis A can occur and has been reported with the use of 10,000 IU/kg intramuscularly.

Vitamin E is often described as the body’s antioxidant vitamin. In rats, 42 umol/kg of α tocopherol is required in diets containing less than 10% fat. This is approximately 18 mg/kg (27 IU/kg diet). Surprisingly, some invertebrate feeders meet this requirement; adult houseflies (29.7 mg/kg), adult crickets (19.7 mg/kg), waxworms (13.3 mg/kg), and butterworms (13.0 mg/kg).

Calcium absorption relies on several factors including UVB supplementation, dietary vitamin D3 levels, oral calcium availability, and health status of the gastrointestinal tract, kidneys, integument, and musculoskeletal system. Endocrine regulation of calcium in herptile species shares similarities with mammalian species. However, the nutrient sensitivity profiles differ based on the mode of vitamin D3 acquisition. The photobiosynthesis of provitamin D3 (7-dehydrocholesterol) to previtamin D3 requires UVB supplementation in diurnal reptiles. The herbivorous green iguana has historically been recognized as the poster-child for metabolic bone disorders secondary to UVB deficiency. However, some insectivorous species are at risk as well.

While some species absolutely require UVB supplementation for vitamin D3 production, others may not. In crocodilians and snakes, many authors suggest that the “nutritional completeness” of the vertebrate, whole prey diet has altered or decreased the photobiosynthetic demand for vitamin D3. Interestingly, increases in serum cholecalciferol levels did occur when diurnal corn snakes (Elaphe guttata) were exposed to UVB light (Acierno 2008). However, the study did not determine if UVB was required for adequate calcium absorption in the species. Nocturnal species are often described as not needing UVB exposure. However, one study of the dusk compared to diurnal species. A similar study has shown comparable results in a shade-tolerant species of Jamaican anole (Anolis). Calcium deficiencies can lead to a host of disorders (e.g., growth retardation, pathological fractures, osteopenia, tetany, post-ovulatory stasis, seizures). In many invertebrates, levels under 25 IU/kg are reported. There are few studies of dietary and/or photobiosynthetic vitamin D3 needs of insectivores. Providing UVB exposure that is comparable to natural environment levels is often recommended, as dietary supplementation alone can be problematic. Dietary supplementation is typically provided via dusting feeder insects with a vitamin D3 powder. This is imprecise and the actual amount of supplement ingested is difficult to quantitate. For example, insects may groom off powder within minutes and the amounts of vitamin D3 in marketed reptile products can vary widely from reported product amounts. Vitamin D3 toxicity is reported and over-supplementation also needs to be avoided.

Calcium absorption relies on several factors including vitamin D3 levels, oral calcium availability, and is dependent on the health status of the gastrointestinal tract, kidneys, integument and musculoskeletal system. Calcium requirements vary with age and life-stage. For instance, reproductive activity and egg development in female reptiles will increase calcium utilization. The largest calcium reserve is the skeleton and nutritional secondary hyperparathyroidism is a major concern if nutritional deficiencies exist. Some herptile species, like Rhacodactylid geckos and some amphibians, store calcium in specialized lymphatic sacs as well.

The minimum calcium requirements for several herptile species have been extrapolated based on the clinical onset of diseases associated with hypocalcemia. In rats, 3.5 - 5 g/kg calcium is required for maintenance. Most insects do not have calcium contents that come close to this requirement. Two insect species are exceptions to this rule. The phoenix worm naturally contains 9.3 g/kg without dietary supplementation and the wood louse (Porcellio scaber) contains 14% calcium on a dry matter basis and has an 11.79 Ca:P
ratio. Rat diets easily meet minimum phosphorus requirement of 3g/kg diet. In most invertebrates, this minimal requirement is met or exceeded and values range from 1.5 - 3.7 g/kg. Many prey items have inverse calcium to phosphorus ratios and are unable to meet herptile calcium needs without calcium supplementation.

**How to prepare your invert feeder for consumption**

Optimizing the herptile’s nutrition begins first with appropriate invertebrate prey choice and by optimizing the prey’s nutrition. Most invertebrates are shipped, stored at low temperatures to prevent molting, and sold without water. Travel substrates are generally not designed for optimal nutrition, apart from silkworms that will only eat mulberry leaves and come in an appropriate substrate. Generally, invertebrate prey should be given at least a few days to eat and rehydrated after shipment or purchase from a pet store. In some cases, establishing breeding colonies of prey items may be warranted. Feeding healthy prey items that have been fed appropriate diets, will help ensure appropriate nutrition for insectivorous herptile species.

Altering the nutritive quality through external supplements (“dusting”), can be difficult to reliably achieve. Powders with calcium and vitamin D3 are commonly used as a dusting agent prior to consumption. Some supplements come with other vitamins or micronutrients as well. Although helpful, it is difficult to accurately measure the dosing effects, as mineral attachment can vary based on insect grooming behavior and particle size. It is important that the prey item is consumed quickly to maximize supplement ingestion.

Altering the insect’s internal nutritive quality is a more reliable way to improve the prey’s nutritional content. Gut-loading refers to act of providing invertebrate prey with a nutrient (e.g., calcium) dense diet for a specific time interval before the prey is fed to the intended insectivore. Several studies have shown a linear correlation between the intestinal contents of certain invertebrate prey and the feeding substrate provided. If the prey item is not ingested in a timely manner, the ingesta will pass from the insect and reduce the effectiveness of this supplementation method.

For most larval insects, you can feed them a diet that contains at least 9% calcium for 24 hours prior to feeding the insect to the reptile. Levels exceeding 40.7% calcium have been associated with dietary avoidance in crickets. The author has examined the calcium and phosphorus content of mealworm and superworm larvae given wheat-millings, avian starter diet, high calcium cricket feed, and organic avian seed mash. The calcium content of mealworm and superworm larvae rose to calcium to phosphorus ratios that were 1.31:1 and 1.47:1 respectively; these nearly meet the NRC recommendation for growing rats (1.66:1).

**Sources for insectivore diet guidelines**

Herpetological journals, nutritional journals, comparative endocrinology and physiology journals, and *Zoo Biology* offer frequent articles that overview species-specific husbandry and diet information for captive insectivores. Online forums (e.g., www.herpconbio.org hosted by the Herpetological Conservation and Biology Society, HerpDigest) can also provide enthusiasts with free access to articles. Consulting with local herpetologists and reptile collection curators may provide access to diet guidelines for unique species. The Nutrition Advisory Group (NAG), a group of veterinary nutritionists who specialize in zoo nutrition, provides free access to conference proceedings and information on the nutrient composition of several prey items at http://www.nagonline.net/. NAG serves as the scientific nutrition advisory group for Association of Zoos and Aquariums (AZA).

**Comparative nutrient content of common invertebrate feeders**

Most commercially reared insects are larval worms of beetles or moths that are high and fat in protein, low in calcium, and high in phosphorus. In efforts to improve their nutritive profile, we recommend gut-loading the larval species with a diet high in calcium and Vitamin A prior to serving it to insectivorous reptiles. Earthworms, silkworms, pill bugs, and katydids have a good nutritional profile and can be used as staple bug options for carnivorous species. See the table below for fun comparisons.

**Table 1.**

<table>
<thead>
<tr>
<th>Insect is known For</th>
<th>Human Food Comparison</th>
<th>Staple or Treat?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Domestic Cricket</td>
<td>Low protein, high fat (22.8%)</td>
<td>French Fries</td>
</tr>
<tr>
<td>Roaches</td>
<td>Lean protein with variable fat (14 - 50%)</td>
<td>Hamburger to Steaks</td>
</tr>
<tr>
<td>Silkworms</td>
<td>Some vitamin A, lean protein source</td>
<td>Turkey</td>
</tr>
<tr>
<td>Earthworms</td>
<td>Protein, good micronutrient content</td>
<td>Big Mac® + multi-vitamin</td>
</tr>
<tr>
<td>Phoenix Worms</td>
<td>Calcium content high</td>
<td>Tums® tablet</td>
</tr>
<tr>
<td>Wood Louse</td>
<td>Calcium (14.38%)</td>
<td>Tofu</td>
</tr>
</tbody>
</table>

300
<table>
<thead>
<tr>
<th>False Katydid</th>
<th>Vitamin A (retinal esters) &amp; lean protein</th>
<th>Salmon</th>
<th>Staple</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butterworms</td>
<td>High ME and fat content (29% DM)</td>
<td>2 Hotcakes with sausage (25%)</td>
<td>Treat, recovery diet</td>
</tr>
<tr>
<td>Mealworm larvae</td>
<td>Fat (31%)</td>
<td>2 McDouble Hamburgers®</td>
<td>Treats</td>
</tr>
<tr>
<td>Mealworm beetles</td>
<td>Fat (17.7%)</td>
<td>Staple</td>
<td></td>
</tr>
<tr>
<td>Superworm Larvae</td>
<td>Fat (40.8%)</td>
<td>Two Sausage Egg &amp; Cheese Biscuits (46%)</td>
<td>Treats</td>
</tr>
<tr>
<td>Superworm beetles</td>
<td>Fat (14.3%), Protein (68.1%)</td>
<td>Staple</td>
<td></td>
</tr>
<tr>
<td>Waxworm larvae</td>
<td>More fat (51.4%), can try to gut load with calcium</td>
<td>Butter (53% fat)</td>
<td>Treat, recovery diet</td>
</tr>
<tr>
<td>Freeze-Dried or Canned</td>
<td>Variable moisture and high in fat</td>
<td>Doritos®/Slim Jim®</td>
<td>Treat, use sparingly</td>
</tr>
</tbody>
</table>

**Take home points**

1. Larval insects are usually HIGH in fat, poor in vitamin and minerals, and have variable bioavailable protein content.
2. Adult beetles, earthworms, and roaches tend to have good protein levels and are low in fat.
3. Most insects have poor calcium levels; calcium content of the insect can be increased by “gut-loading” on a high calcium diet at least 24 hrs prior to feeding.
4. Calcium-rich insects include phoenix worms and the wood lice. To increase digestibility, these insects can be macerated (cuticle broken) prior to feeding insectivores.
5. For commercial diets, check the first ingredient to ensure that an appropriate animal protein product is listed.
   “Guaranteed” analysis minimums and maximums do not provide exact percentages.
6. Ensure that prey items are well-hydrated prior to consumption, as water is a major nutrient and many insectivores depend on prey moisture content to maintain hydration.

**References**


Finke MD. Complete nutrient composition of commercially raised invertebrates used as food for insectivores. Zoo Biology. 2002 Jan 1;21(3):269-85.


Most of the reptile physical examination begins with a thorough evaluation of the husbandry and history, even before a visual examination should be tackled.

Husbandry sheets should be prepared for dispersal to your clients. This means doing homework for the species that are most likely to walk through your door. Which might they be? Head to a pet store nearby and take note of what is being sold. Where is a good place to start? Red-eared sliders, anoles, ball pythons, cornsnakes, bearded dragons, iguanas, and Russian tortoises. Visit reptilemagazine.com, chat with a few friends that own these animals and make your own husbandry sheets. Making the sheets will make you remember. This gives them a reference for proper reptile care. This also reassures your owners that you can offer compassionate veterinary care for their reptile. This is very important! Get a Detailed History: Diet, cage type, cage design, lighting and heating, bedding, cage location, behavior, prior medical history, other pets in household. This information prepares you for what to specifically evaluate on physical, and what to expect.

The exam room should have supplemental heating source, a pen light, Doppler, cotton tip applicators, warm ambient environment, and as few on-lookers as possible.

The following systems can be evaluated WITHOUT disturbing the animal: Ocular, aural, hydration status, proper occlusion, overall body condition score, obvious musculoskeletal abnormalities, RR, CNS/Mentation, and integument.

The rest of the “physical” physical should take about 10 minutes. This is important because handling will stress your patient out. Save oral exam for last, this will be the most stressful part of the physical exam.

Oropharyngeal
Start by palpating the mandible. If you can feel “bumps” along the maxilla or mandible, you may be palpating abscesses. Is the maxilla and mandible properly occluded? Do you see exposed gingiva? Does the rostral skull bend when you apply mild pressure to the snout? Fibrous osteodystrophy secondary to metabolic bone disease? Use a lubricated guitar pic or cotton tip applicator and a light source to visualize the entire dental arcade, gingival, tongue, glottis, assess mucus membranes and hydration status. Dry and/or ropey oral secretions indicates dehydration. Snakes have lovely palatine vessels, assess their distension and provide evidence for adequate or lack of perfusion.

Ocular
Do the eyes appear dull or appear sunken, this can indicate severe dehydration. They may dazzle, but will not menace. Tortoises, crocodilians and lizards control their PLRs, skeletal muscle control of the iris. This means atropine will not be affect in dilating pupils to evaluate the back of the eye. Check the medial aspects of lids in testudines and lizards, spectacles in snakes. Retained sheds? Check conjunctiva & 3rd eyelid mucus membrane color. Any ocular Discharge? Periocular swelling? Photophobia? Characterize their problems the SAME way you would a mammal. Chemosis? Corneal Defects? Anterior Uveitis?

Aural
Testudines have a tympanum, evaluate for swelling, which may indicate auricular abscesses. For lizards, evaluate their tympanums. Check for evidence of fluid or blood behind the window.

Respiratory
Get a respiratory rate BEFORE you handle the reptile. Remember that defensive postures and displayes can affect rates. RR 12-36 bpm at rest is normal. For aquatic testudines: abnormal buoyancy is HIGHLY suggestive of pneumonia. Open mouth breathing in ANY REPTILE is an emergency.

Cardiovascular
Turtles, all lizards EXCEPT varanids: can be located in the thoracic window. For turtles, place Doppler on the Jugular vein OR behind the arm in the axillary pit. For lizards, place the Doppler probe in between the arms on the ventrum of the reptile. For our lovely monitor lizards, the varanids, place the Doppler in on the ventrum on the caudal thorax. For snakes, the heart is located on the ventrum 1/3rd the length of the body caudal to the head. For large snakes, the Doppler can be placed on the spectacle. The heart rate is heat and PAIN dependent. HR≤ 60 bpm can be normal. Elevations can be attributed to increased activity and/or most PAIN. Check for arrhythmias and murmurs. Unsure? Use an ultrasound to evaluate contractility and chamber size. It’s a beautiful pump, they can shunt in extreme circumstances, but the heart functions just like a mammals when the environmental conditions are right.
Gastrointestinal
The best way to evaluate the GI is to take a look at what came out of it. Fecal evaluations and a history of stool production are important. The GI, when palpated, is not as easy to distinguish when compared to mammals. They all have a stomach, small intestines, liver, gallbladder, and their colons vary in size based on diet. Our herbivores have large ones, the carnivores have short ones. You may be able to palpate ingesta and/or fecal matter. In turtles, you can palpate the femoral fossa to rule out obstipation. As many reptiles have unique coloration of their oral mucus membranes, it is important to evaluate the color of the cloacal membranes.

Urogenital
In turtles, the kidneys are located caudodorsally along the carapace. You cannot palpate them. Femoral fossa palpation may allow you to feel urinary stones. Male turtles have a phallus, do not extrude it but evaluate it IF it is displayed or traumatized. In iguanas, the kidneys are located in the pelvic canal. You can perform a cloacal exam to rule out renomegaly. In other lizards, you can visually locate hemipene sacs. Unlike in pet parrots, we have many species that are sexually dimorphic! Agamids, geckos, iguanas, males have large femoral pores. In bearded dragons, do not mistake uterine horns for coelomic fat pads in agamids. In snakes, you should not be able to palpate kidneys unless there are severe gross abnormalities. In boids, there have remnant pelvic "legs" that are actual short bones that articulate with the rib cage. They are called "spurs", and can be flexed and extended during courtship. Some use pelvic spur size as a tool for sexing snakes, males having larger spurs. For sexing non-boids, you can use a small, lubricated red rubber French catheter to probe hemipene sacs. Avoid probing needles. Males have a deeper hemipene sac length. Mark how far the probe was placed in the sac. Then count the number of scales that accounts for the marked length distal to the anal scale. Scale depths greater than 7-9 scales indicate the sex is male. Females usually have a hemipene sac scale depth of less than 5 scales. This can vary dependant on the species.

Integument
Assess the skin turgor. If there is prolonged tenting and lack of elasticity, the reptile is dehydrated. What is their shed cycle? Aquatic Turtles shed scutes commonly. Lizards shed skin in a piecemeal fashion. Snakes shed in one sock-like piece INCLUDING the spectacles. Snakes undergoing an active shed should be handled carefully, as they are physiologically dehydrated and cannot see well. Dysecdysis is a very significant finding. It signals severe deficits in hydration status. Note any scars, vesicular dermatitis, or lacerations and characterize them.

Musculoskeletal
Palpate long bones, spine, and hips and examine the normal posture and their ability to ambulate on padded exam floor. The body condition score in all reptiles can be assessed by a visual and physical evaluation of masseter and temporal musculature, normal or atrophied. In iguanids and geckos, evaluate the tail as they have bands of fat stored in between the coccygeal muscle bands. You should NOT be able to feel lateral processes of the coccygeal vertebrae in a well-conditioned lizard. Coelomic fat pads can be palpated in longitudinal bands in bearded dragons and spiny tailed lizards.

Nervous
Start by carefully evaluating their mentation. What does the owner say, is this normal? Are they observing your actions? Trying to get away? Are they threat displaying, are they looking around? Are they trying to escape? Are they trying to move away? Are they flicking their tongues? For the Cranial Nerves exam, you can perform a quick assessment. Assess dazzle, PLRs, eye position, facial symmetry, hearing. Most reptiles can appreciate noises that have a low decibel level. Tongue and gag reflex is checked during the oral exam. Palpate the spine, check panniculus, check CP by placing and avoid hemi-walking and checking peripheral reflexes unless indicated.

With practice, you will start to have fun learning about the unique differences and celebrating them with a contagious enthusiasm that their owners have! Treating reptiles mean you get the opportunity to learn something new each time- have a great time!
GI Stasis: *Oryctolagus cuniculi*, the domestic rabbit, has a very specialized gastrointestinal system. These hindgut fermenters rely heavily on the cecum and colon to ferment long stem grass hays and plant material. The rest of the GI system and the other biological systems of the rabbit are at the mercy of the functionality of these organs. When GI motility slows down or stops completely, it is a medical emergency for the rabbit or any small herbivore. Simple treatments can be implemented to quickly resolve the condition and afford a practitioner the time needed to identify the underlying cause- which could be pain or diseases that are not specifically related to the GI tract.

It is important to first stage the severity of GI stasis. Is the rabbit still passing small feces or has clinical signs persisting for more than 12 hours? We classify GI stasis cases as mild, moderate and severe.

- **Mild**: Potential dietary indiscretion, stress incident, unrelated mild illness. Patient is well hydrated, still eating, and fecal production is slowly decreasing, but has not stopped.
- **Moderate**: Patient has not produced feces in > 8 hour period, stopped eating, presents dehydrated and painful.
- **Severe**: Patient is severely dehydrated, painful, hypotensive, hypothermic and requires emergency fluid therapy and stabilization.

GI stasis treatment approach for herbivores

The treatment approach should include fluid therapy, analgesics, syringe feedings and tests to outline the underlying cause. Fluid therapy requirements range from 100-150 mls/kg/day. Give 50mls/kg SQ every 12 hrs if mild to moderate and if there are no indications of cardiac disease or hypoproteinemia. For severe cases, warm IV therapy and crystalloid and/or colloid shock boluses may be indicated. Fluid boluses can be 10mls/kg over 20 minutes, check SAP and modulate therapy as needed If there is a response. Colloid boluses of 5mls/kg over 20 minutes may be indicated if the patient is bleeding or hypoproteinemic. Buprenorphine 0.01-0.03 mg/kg SQ/IV q8-12hrs should be given as needed for moderate to severe cases. Tramadol 5mg/kg PO q8-12 can be used in recovering animals or in mild cases that require continued supportive care at home. Syringe feeding is the mainstay of GI stasis therapy.

Cecal motility and colonic motility respond to 0.5mm fiber particles that stimulate their mechanoreceptors. Without this, the process halts. Getting the fiber BACK into the gut is the goal of treatment to re-stimulate normal motility. Critical care feedings can be arduous but are a vital part of the treatment process. Patients require 20mls/kg per feeding, with a feeding occurring every 6-8 hours, until the animal begins eating on its own. When patients are recovering, stimulate them to explore their environment provided their underlying ailment does not prevent physical motility. Lastly, identify the underlying cause of the stasis. Perform a thorough exam is key- trust your hands and your observations! If possible, perform a CBC, PCV, TS, and chemistry screen in your bunnies. Radiographs may be indicated. In most cases, metoclopramide and cisapride, which can induce side effects themselves in herbivores and in people, are not warranted.

Diagnosing dental disease

It’s important to know that you can diagnose dental disease in small herbivores via visual and physical exam before attempting an oral exam. Molar disease is the usual culprit that gets missed on many exams. Palpate the ventral aspect of the mandible, it should be smooth! If you feel bumps, these may represent reserve crown extensions or abscessation. The same can be performed on maxillary palpation, particularly on the medial aspect of the zygomatic arch. The incisors should be properly occluded, if they aren’t, there is a high likelihood that the molars are mal-occluded. “Incisor-disease only” can occur but represents a small portion of rabbits with dental disease, therefore incisor adjustments alone do not always treat disease. Radiographs are commonly indicated to help qualify dental disease. There is an excellent reference for classifying radiographic evidence of disease in herbivores (E. Boehmer; D. Crossley: Objective interpretation of dental disease in small mammals. 2009), please email for a copy. Treatment options for dental disease vary, and may require clinical crown adjustments, diet modification (reducing pellets, increasing long stem grass hay) and in severe cases with abscessation, antibiotic therapy (Duo-Pen 50,000 IU/kg SQ q 7 days to cover infections-anaerobic), and surgery. Pain medications can include Meloxicam 0.5mg/kg PO q12hrs and Tramadol 5-10mg/kg PO q8-12 hrs.

The head tilt: What is the cause?

The young or old rabbit that presents with vestibular signs should be evaluated the same way one would evaluate a cat or dog. Distinguishing if an animal has peripheral or central signs is imperative. Despite what the old references tell us, *E.cuniculi* (ECUN)- a microsporidian parasite that can infect rabbits, dogs, and humans, does NOT cause peripheral vestibular signs. Unfortunately, central vestibular signs can be subtle and missed. More often than not, ECUN can cause cerebral signs, which may include only slight mentation changes. In severe cases, cerebral disease can cause cortical blindness and seizures and this is easier to diagnose.
Central vestibular signs, which include axial rolling, tight circling, abrupt hemiparesis, CP deficits, severe ataxia, truncal swaying, horizontal, rotary or vertical nystagmus, are easily diagnosed but not always reflective of ECUN infections. In a case review of 230 post mortem exams performed on rabbits at PennVet from 1985-2012, *E. cuniculi* spores that caused active CNS inflammation (secondary to neuronal cell rupture) were identified in 13% of the cases, with only one rabbit that had severe inflammation in the brainstem that may have correlated with central vestibular signs. Studies now confirm that the degree of inflammation on post does not correlate with clinical signs (Csokai 2009). Commonly, rabbits > 4 years of age can present with acute thromboembolic events that may resolve in as little as 48 hours or as long as 1-2 weeks with supportive care. Supportive care includes protective caging, assist feeding, and anti-inflammatories (meloxicam and/or doxycycline). Ruling out otitis, ototoxicity, and trauma becomes essential to hasten treatment for patients that have severe peripheral signs. True *E. cuniculi* cases can present with acute disease in newly infected or chronically affected rabbits. Renal failure secondary to repeat infections and damaged caused by spore migration can occur. CNS involvement occurs in chronically infected rabbits and can be difficult to diagnose ante mortem. A review of the disease and treatment options can be found in this free access summary article. (Latney et al, Encephalitozoon cuniculi in pet rabbits: diagnosis and optimal management. 2014). Fenbendazole does not cure chronically infected animals with CNS inflammation and case reports reveal that its use causes severe liver disease and failure in rabbits (Graham, Benzimidazole Toxicosis in Rabbits: 13 Cases from 2003 to 2011, 2014).

**Chinchillas**

**GI stasis prognosis**

Unlike our lagomorph patients, chinchillas afford owners an even narrower window to respond. Clinically, we appreciate that of the small herbivores, chinchillas develop severe metabolic disturbances secondary GI disruption. Ketosis occurs rapidly. It is not uncommon to see chinchillas present with severe metabolic derangements secondary to GI stasis. It is imperative to get a urine sample to assess for the presence of ketonuria to qualify prognosis. If a blood sample can be obtained, assessing for the presence of lipemia is also imperative. If the patient was previously overweight (>500 grams) and has hepatic lipoidosis, diabetic ketoacidosis is highly likely to occur within as little as 8 hours from not eating. The patient has a grave prognosis if ketonuria, glucosuria, and lipemia are noted and, even with aggressive care. If the patient presents muscle wasted/emaciated, a ketoacidosis may be present secondary to prolonged period of anorexia and muscle catabolism. If a ketonuria is noted, in the absence of lipemia, glucosuria, and/or hyperglycemia, the patient may recover with aggressive support care.

**Revisiting the murmur**

In the past, clinicians thought murmurs in chinchillas were a “normal” finding on physical exam. In a multi-institutional study evaluating cardiac disease in 260 chinchillas, murmur prevalence was found to be 23% (Pignon 2012). Chinchillas with a grade 3 murmur or greater 30 times higher chance of having echocardiograph disease than chinchillas without murmurs, and those with a grade 1 or 2 were 10 times more likely to have echocardiographic abnormalities. Mitral valve insufficiency, right ventricular outflow obstruction, tricuspid valve regurgitation, and left ventricular hypertrophy were among the specific cardiac diseases identified in 8 of 15 chinchillas that received full echocardiographs. Subsequently, we recommend monitoring chinchillas closely for murmur intensity and urge owners to pursue an echo if a Grade 3 is noted OR if the intensity of the murmur increases.

**Guinea pigs**

**Revisiting respiratory distress in the guinea pig**

Respiratory distress in the guinea pig comes with a long differential diagnoses list. Apart from *Streptococcus pneumoniae* and *Streptococcus zooepidemicus*, viral infections, like guinea pig adenovirus, can cause severe pneumonia. *Bordetella bronchiseptica*, commonly found as in rabbits and dogs, can cause acute respiratory distress and death in guinea pigs. Antibiotics for *S. pneumoniae* and *Bordetella* can be managed with enrofloxacin and chloramphenicol. *S. zooepidemicus* can cause an acute respiratory syndrome, which can be difficult to cure OR it can cause localized abscessation of the cervical lymph nodes. Medical management is the goal, as *S. zooepidemicus* is a normal flora in GPs. Although penicillins is an effective treatment for *S. zooepidemicus*, it will cause a dysbiosis in the guinea pig, even if given SQ. Perform cultures to rule out other infectious causes. Consider starting with enrofloxacin or chloramphenicol. Oral fluoroquinolones may cause mild GI upset and pigmenturia in some patients.

In some cases dental disease may be the cause of severe respiratory distress. Evaluation of the oral cavity is very important in this case, as treatment requires dental emergency adjustments. Their mandibular molar teeth can become overgrown, causing enamel point bridging that can entrap the tongue. Their normal occlusal surface is a steep 30 degrees, causing the lingual points to angle down toward the tongue. They have a palatial ostium that connects the oropharynx to the trachea, so they can literally occlude their airways with struggling to unentrap the tongue. In addition to dental disease, guinea pigs can present in severe respiratory distress secondary to cardiac disease, primary pulmonary neoplasia, mass effects from severe gastric dilatation or ovarian cyst enlargement. For these reasons, a thorough, safely staged examination should be performed carefully in the respiratory pig, as the cause of presentation can be quite variable.
Updates on antibiotic and analgesia doses for herbivores (off-label references)

- Tramadol: 5-10 mg/kg PO q8-12h for pain
- Buprenorphine: 0.01-0.05mg/kg q8-12h for pain
- Tonaxuril: 2.5 mg/kg PO once for Eimeria infections in rabbits
- DuoPen: 50,000 IU/kg SQ every 7 days for anaerobic dental disease, skin disease, chronic respiratory disease, Treponema infections in rabbits
  - Do NOT use in guinea pigs, some report use in chinchillas
- Enrofloxacin: 15-20mg/kg q24h- this is a dose dependent drug, smaller doses given q12 potentiates resistance patterns and reduces drug efficacy
- Metacam: 0.5 mg/kg PO/SQ q12h
- Maropitant: 1 mg/kg SQ/IV q 24h to reduce nausea and in severe GI stasis cases
- Cyanocobalamin: 20 mcg/kg PO/SQ q24h for liver and regenerative anemia support
- Keppra (Levetiracetam): 60 mg/kg to treat status epilepticus, 20 mg/kg SQ/IV/PO q8h
- Thiamine: 20mg/kg PO/SQ q24h for chronic liver disease

Ferrets
Diarrhea – Why we treat aggressively

As the experimental model, ferrets provided us with the scientific proof that *Helicobacter* can cause gastric ulceration and primary disease of the gut. In practice, it is common to see ferrets develop diarrhea secondary to any underlying disease. Immunosuppression studies and include the following: Amoxicillin PO (20mg/kg q12h), or ampicillin IV (20-22mg/kg q8h), Metronidazole PO or IV (20 mg/kg PO/IV q12h), Famotidine or Omeprazole IV or SC (0.5mg/kg IV/PO q12h), Carafate PO (20 mg/kg PO q6, 20 minutes prior to feeding). Begin syringe feeding them watered a/d every 4 hours BY hand. Resolution of complete clinical signs of a Helicobacter flare or ECE may take up to 2 weeks. Perform diagnostics to rule out additional causes of disease. Lawsonia intracellularis infections require PCR confirmation or silver stains performed on tissue samples, and chloramphenicol (50mg/kg q12h for 10-14d) is indicated for treatment.

Rats
Revisiting pneumonia

Rats suffer from an arsenal of respiratory pathogens and there are factors that can hasten and exacerbate disease in their delicate lungs. The 5 recognized pathogens are: *Mycoplasma pulmonis, S. pneumoniae, Cornebacterium kutcheri, Sendai virus* and *cilia-associated virus*. Rats may live their life of 2–2.5 years with signs of chronic respiratory tract infections. Once established in the lower airways, *Mycoplasma pulmonis causes a* chronic bronchitis, progressing to bronchiectasis and bronchiolectasis, and pulmonary abscessation. Acute disease may occur with signs of nasal discharge, ocular discharge and respiratory distress. Radiographs may reveal consolidation of lung lobes with abscessation. When approaching the dyspneic rat, qualify severity first. If they are open-mouth breathing, have a RR >100 bpm, have blue/cyanotic foot pads, and/or a severely increased respiratory rate, place them in an oxygen cage and assess for responsiveness. Warm saline nebulization may be necessary and may help during recovery. Airway support may be given with the use of a bronchodilator (Albuterol inhalation, aminophylline, or Terbutaline 0.1mg/kg SQ). If the patient is responsive, organize your treatments to administer quickly. Antibiotic therapy with enrofloxacin @ 15 mg/kg PO or SC q 24 hr (if giving SC need to dilute out in 5-10 ml sterile saline) and Doxycycline @ 5 mg/kg PO q 12 hr. Do not use the clear doxycycline hyclate for injection- this causes severe tissue necrosis. The depot formulation (Vibravensos) of doxycycline can be given carefully, SQ, at a dose of 75-100 mg/kg, and will last for 7 days until the patient can tolerate oral medications. Adjunctive therapy may also
include Azithromycin 15-30 mg/kg PO q 24 h. Provide fluid therapy and nutritional support during recovery, as these patients can become severely dehydrated secondary to respiratory disease and severely hypoglycemic.

Sincere efforts should be made to reduce the potentiation of clinical signs and this can be modulated by the pet owner. Rats require adequate ventilation and low dust, non-aromatic bedding. Encourage owners to maintain room temperatures from 65 to 80º F, humidity between 40 to 70%, and clean the cage frequently to minimize ammonia fumes. A reduction in obesity may also help reduce the clinical onset of disease.

**Neurologic presentations**

Pituitary adenomas are not uncommon in geriatric pet rats (Mayer 2011). It is seen more commonly in female rats, however males can present with signs as well. Affected rats present with CNS signs such as blindness, circling, head pressing, “spacey” appearance or severe depression. They are reluctant to move from the cage wall and owners may report that they have fallen or developed balancing issues at home. Pituitary adenomas are usually prolactin secreting and mammary development can be seen. This disease can be painful and very distressing. Stabilizing treatment includes fluid therapy and steroid medications (Dexamethasone SP 0.25mg/kg-0.5mg/kg once or oral prednisolone 0.5 mg/kg q24h) pending the acquisition of cabergoline. Cabergoline, a dopamine receptor agonist, has been reported to decrease tumor size in per rats at 0.6mg/kg PO q72h and has been used to manage disease for 8.5 months in a select case (Mayer 2011). Fluid therapy is warranted, as affected rats are display hypodipsic hypernatremia syndrome due to the hypothalamic space-occupying mass. Nutritional support is also necessary during stabilization, as patients are usually very painful when eating solid foods.

**Mammary fibroadenoma and pituitary tumor prevention**

Although females develop mammary fibroadenomas, both sexes develop an increased risk as they age (> 1 year old). The incidence is clinically high in females, however 2-16% has been reported experimentally in males (Mayer 2013). Mammary tumors and pituitary tumors frequency is significantly lower in 18- to 24-month-old ovariectomized rats (4%) than in sexually intact rats (mammary tumors, 49%; pituitary tumors, 59%). Decreased frequency of mammary tumor development could be related to the decreased frequency of prolactin-secreting pituitary tumors (Hotchkiss 1995). Based on these findings, it is recommended that rats undergo ovariectomy or neuter prior to 90 days of age to reduce the risk of mammary and pituitary tumor development. For patients that cannot undergo surgery, off-label use of deslorelin acetate (4.7mg SQ implant), a GnRH agonist, has been used to delay estrus for 1 year in rats (Alkis 2011). This may be helpful in modulating prolactin-secretion and reduce disease occurrence. Leuprolide acetate has been experimentally used to suppress gonadotropin and testosterone levels for 5 weeks in rats.
Cases of Cavitary Effusion:  
What’s Filling that Space??

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Pleural or peritoneal fluid analysis will be classified by evaluating the results of routine fluid analysis and virtual microscopy of digital slides. A flow chart that can be used to help establish the type of effusion is present on the first page of the previous topic (Cases of cavitary effusions: What's filling this space?).

7-1 Cavitary effusion CVC: Pleural fluid, cytocentrifuge prep.

- Case: 079234 (647)

(7-2 Peritoneal fluid -- next slide, same animal)

- Dog, Labrador retriever, male (neutered), 8-yr-old

The dog was referred because of an acute onset of a distended abdomen and a hypoproteinemia (TP = 4.2 g/dL, Alb = 2.2 g/dL). Physical examination revealed a distended abdomen due to a peritoneal effusion and muffled heart sounds. Pleural and peritoneal fluid samples were collected and submitted for analysis.

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<td>Color, postcentrifugation</td>
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<td>Hct</td>
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<td>Other</td>
<td>%</td>
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7-2 Cavitary effusion CVC: Peritoneal fluid, cytocentrifuge prep.

- Case: 079234 (651)
- Dog, Labrador retriever, male (neutered), 8-yr-old
- See CVC 2-1 information

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</table>

7-3 Cavitary effusion CVC: Peritoneal fluid, cytocentrifuge prep.

- Case: 079660 (655)
- Horse, quarter horse, male (neutered), 20-yr-old

The horse was referred because of an acute colic that now is of 24-hours duration. Physical examination revealed pawing and kicking of abdomen, tachycardia, and very few gut sounds.

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7-4 Cavitary effusion CVC: Peritoneal fluid, direct smear

- Case: 507605 (667)
- Horse, Thoroughbred cross, male (castrated), 14-yr-old

The horse was presented because of colic of 12-hr duration. The referring veterinarian reported that the horse passed a small amount of mucoid feces yesterday, rectal palpation revealed gas-distended loops of intestine, and gut sounds were absent. A small amount of peritoneal fluid was collected and submitted for analysis.
Patient | Color, precentrifugation | Clarity, precentrifugation | Color, postcentrifugation | Clarity, postcentrifugation | Total protein (ref) | Hct | TNCC |
---|---|---|---|---|---|---|---|
| Patient | yellow | cloudy | yellow | clear | 5.5 g/dL | < 3 % | 202,000/µL |

7-5 Cavitary effusion CVC: Peritoneal fluid, direct smear
- Case: 051233 (613)
- Dog, Labrador retriever, male (neutered), 6-yr-old

Owner first noticed abdominal distension about one week ago; the dog’s appetite and activity has not changed. Physical examination revealed a fluid-filled, distended abdomen and possibly a peripheral lymphadenopathy.

Patient | Color, precentrifugation | Clarity, precentrifugation | Color, postcentrifugation | Clarity, postcentrifugation | Total protein (ref) | Hct | TNCC |
---|---|---|---|---|---|---|---|
| Patient | Red | Opaque | Pink | Hazy | 5.0 g/dL | 30 % | 7,500/µL |

7-6 Cavitary effusion CVC: Peritoneal fluid, cytocentrifuge prep.
- Case: 079781 (658)
- Dog, Anatolian shepherd, male (neutered), 8-yr-old

The dog had intermittent episodes of diarrhea for about 2 months. About 2 weeks ago, it was dribbling urine and the referring veterinarian treated for a urinary tract infection. Urine dribbling continued up to yesterday; no urine passed in last 24 hours. Physical examination revealed a depressed dog with a distended and painful abdomen.

Initial laboratory data included a mild inflammatory leukocytosis, mild hyperproteinemia, almost an erythrocytosis, azotemia (UN 105 mg/dL, Crt 3.6 mg/dL), mild hyperphosphatemia, mild hyponatremia, almost hyperkalemia, and metabolic acidosis (HCO₃⁻ 14 mmol/L)

Patient | Color, precentrifugation | Clarity, precentrifugation | Color, postcentrifugation | Clarity, postcentrifugation | Total protein (ref) | Hct | TNCC |
---|---|---|---|---|---|---|---|
| Patient | Blood-tinged | Cloudy | Pink | Clear | 1.3 g/dL | < 3 % | 2,700/µL |

7-7 Cavitary effusion CVC: Peritoneal fluid, line prep.
- Case: 08-69874 (616)
- Dog, mixed breed, female, 1-yr-old

A veterinarian in NE Kansas submitted pleural and peritoneal fluid from a dog. Historical or physical examination findings were not provided.

Patient | Color, precentrifugation | Clarity, precentrifugation | Color, postcentrifugation | Clarity, postcentrifugation | Total protein (ref) | Hct | TNCC |
---|---|---|---|---|---|---|---|
| Patient | Blood-tinged | Cloudy | Colorless | Clear | 4.7 g/dL | < 3 % | 10,200/µL |

Other microscopic findings:

Note: A line preparation concentrates cells in the line, but also makes that area thick.
Note: The analysis of pleural fluid yielded essentially the same results except the TNCC was 5,000/µL.

7-8 Cavitary effusion CVC: Pleural effusion, cytocentrifuge preparation
- Case: 047859 (604)
• Cat, Birman, male (neutered), 16-yr-old
The cat was presented because of dyspnea. The owner reported intermittent inappetence during past week. Physical examination revealed muffled heart sounds. Radiographs revealed a pleural effusion – fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Pink</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Hazy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Light yellow</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>2.6 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>

7-9 Cavitary effusion CVC: Peritoneal fluid, cytocentrifuge prep.
• Case: 080190 (663)
The dog was referred because of abdominal ascites that might be due to heart failure. Physical examination revealed a grade 2-3, left-sided systolic murmur and a fluid-filled abdomen.

Preliminary serum laboratory data found UN of 16 mg/dL, Crt 3.6 mg/dL, hypoproteinemia (TP 2.5 g/dL, albumin 1.2 g/dL), hypocalcemia (tCa²⁺ 5.7 mg/dL), mild hyponatremia (144 mmol/L), normochloremia, decreased anion gap, and urine with a specific gravity of 1.009, and negative chemistry results.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>colorless</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>clear</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>colorless</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>0.1 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>
Pleural or peritoneal fluid analysis will be classified by evaluating the results of routine fluid analysis and virtual microscopy of digital slides.

**Guidelines: variations occur**

**Effusion**

- **[TP] < 2.0 g/dL**
  - Protein-poor transudate
  - Uroperitoneum (early)

- **[TP] ≥ 2.0 g/dL**
  - TNCC < 5,000/µL
    - Hemorrhagic (reddish)
      - Early: mimics blood
        - Hct near blood
      - Later: erythrocytes
    - Macrophages
    - Protein-poor transudate
  - TNCC > 5,000/µL
    - Most exudates
      - > 80 % neutrophils
      - > 80 % neutrophils & macrophages

**Hypocellular exudates**

- > 80 % neutrophils
- > 80 % neutrophils & macrophages
- Common in FIP

**Protein-rich transudate**

- > 80 % neutrophils & macrophages
- Heart failure
- Post-sinusoidal congestion

**Chylous**

- Creamy to white
- Mostly lymphocytes (early)
- Lymph, mac’s, neuts later

**Neoplastic lymphoid effusion**

- Neoplastic lymphocytes

**Other neoplastic effusions**

- Inflammatory cells
- Large atypical cells

6-1 Cavitary effusion CVC: Peritoneal fluid; direct smear

- Case: 315135 (630)
- Cat, DSH, female (spayed), 8-yr-old

The cat was presented because of a sudden onset of lethargy, anorexia, and more recently, vomiting. Physical examination revealed an increased rectal temperature, mild dehydration, depression, abdominal tenderness, and abdominal distension. Radiographs revealed a peritoneal effusion – fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>TNCC</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Tan</td>
<td>TNCC 115,000/µL</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Cloudy</td>
<td>Neutrophils</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Colorless</td>
<td>Monocytes/macrophages</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Clear</td>
<td>Lymphocytes</td>
</tr>
</tbody>
</table>

311
6-2 Cavitary effusion CVC: Peritoneal fluid, direct smear
- Case: 028595 (622)
- Cat, DSH, male (neutered), 9-mo-old

The cat was presented because of a progressive lethargy and inappetence during the past week. Physical examination revealed an increased rectal temperature, mild dehydration, and abdominal distension; the abdomen did not appear tender or painful. Radiographs revealed a peritoneal effusion – fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Yellow</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Hazy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Yellow</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Almost clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>5.1 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>

** The viscosity of the fluid prevents accurate pipetting and thus a total nucleated cell concentration cannot be determined accurately.

6-3 Cavitary effusion CVC: direct smear (3a) and cytocentrifuge (3b)
- Case: 040896 (607, 610)
- Cat, DSH, female (neutered), 8-yr-old

The cat was presented because it was having a hard time breathing. Physical examination revealed muffled heart sounds. Radiographs revealed a pleural effusion – fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>White</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Opaque</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>White</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Opaque</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>5.3 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>

6-4 Cavitary effusion CVC: Pleural fluid, sediment smear.
- Case: 175950 (637)
- Dog, Irish setter, male (neutered), 4-yr-old

The dog was presented because of difficult breathing. The owner reported intermittent inappetence for the past two weeks; also, the dog did seemed to tire easily. Physical examination revealed a lethargic dog with muffled heart sounds. Radiographs revealed a pleural effusion – fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Pale yellow</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Cloudy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>None</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>3.8 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>

6-5 Cavitary effusion CVC: Peritoneal fluid
- Case: 028757 (640)
- Dog, Yorkshire terrier, female (spayed), 9-yr-old

The dog was presented because of an acute onset of vomiting. Physical examination revealed icteric mucous membranes and intense abdominal pain. Peritoneal fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Icteric</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Cloudy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Icteric</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Nearly clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>4.3 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
<tr>
<td>--------------</td>
<td>-------</td>
</tr>
</tbody>
</table>

6-6 Cavitary effusion CVC: Peritoneal fluid
- Case: 12-115895 (1727)
- Dog, German shepherd, female, 8-yr-old

The dog was presented because of an acute onset of vomiting. Physical examination revealed intense abdominal pain. Peritoneal fluid was collected for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Dark yellow</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Cloudy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Dark yellow</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Nearly clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>4.0 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>

6-7 Cavitary effusion CVC: Peritoneal fluid, cytocentrifuge prep
- Case: 075542 (620)
- Dog, Cairn terrier, female (spayed), 3-yr-old

The dog was presented because icterus and difficult breathing. Physical examination revealed a distended abdomen due to a peritoneal effusion. Peritoneal fluid was collected and submitted for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Blood-tinged</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Hazy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Light yellow</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>0.5 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>

6-8 Cavitary effusion CVC: Peritoneal fluid direct smear
- Case: ASVCP 10-9 (885)
- Dog, miniature Australian shepherd, female (spayed), 8-mo-old

One week after intestinal resection, the dog was presented because of anorexia. Physical examination revealed a distended abdomen due to a peritoneal effusion. A direct smear of peritoneal fluid was prepared and submitted for evaluation (fluid was not available for analysis).

6-9 Cavitary effusion CVC: Peritoneal fluid, cytocentrifuge prep
- Case: ASVCP 08-9 (1166)
- Dog, Nova Scotia Duck-tolling retriever, male (neutered), 5-yr-old

The dog was presented because hematemesis and melena. Physical examination revealed pale mucous membranes. Abdominal ultrasound demonstrated multiple enlarge abdominal lymph nodes and a peritoneal effusion. Peritoneal fluid was collected and submitted for analysis.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Patient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color, precentrifugation</td>
<td>Light yellow</td>
</tr>
<tr>
<td>Clarity, precentrifugation</td>
<td>Hazy</td>
</tr>
<tr>
<td>Color, postcentrifugation</td>
<td>Light yellow</td>
</tr>
<tr>
<td>Clarity, postcentrifugation</td>
<td>Clear</td>
</tr>
<tr>
<td>Total protein (ref)</td>
<td>3.2 g/dL</td>
</tr>
<tr>
<td>Hct</td>
<td>&lt; 3 %</td>
</tr>
</tbody>
</table>
Is this Lymphoma- or Just a Reactive Lymph Node?

Steven Stockham, DVM, MS, DACVP (Clinical Pathology)
Kansas State University
Manhattan, KS

The major reason for a cytologic biopsy of lymph node aspirates is looking for the reason for an enlarged lymph node. Lymph nodes become enlarged from many diseases and typically are classified into one of the following groups.

Hyperplastic lymph node
Lymph node hyperplasia is characterized by increased numbers of lymphocytes: B-lymphocytes, T-lymphocytes, or both. The proportions of different types of lymphocytes may appear normal, in which case hyperplasia is suggested by normal cell populations in association with lymphadenomegaly. There may be increases in large lymphocytes and/or plasma cells, in which case the terms reactive or reactive hyperplasia are often used in place of hyperplasia, though the nodes are enlarged because of hyperplasia. A variety of infectious and noninfectious diseases, including bacterial, viral, fungal, and neoplastic disorders, can lead to the stimulation and proliferation of lymphocytes. If there is generalized lymph node hyperplasia, a systemic illness should be considered. If only one node is hyperplastic, a disease within the drainage field of that node should be considered.

Reactive lymph node
A node classified as reactive typically has increased numbers of plasma cells and/or large lymphocytes. The percentage of large lymphocytes is expected to be less than 50 % in a reactive node and is usually less than 10 %. An increase in plasma cells indicates B-lymphocyte stimulation.

The causes of a reactive lymph node are essentially the same as those for lymph node hyperplasia.

Lymphadenitis
Lymphadenitis is characterized by an increased number of nonlymphoid inflammatory cells in a lymph node. One inflammatory cell type might dominate (e.g., neutrophils), or there can be a mixture of inflammatory cells (e.g., neutrophils, macrophages, and eosinophils). The cause of the inflammatory state may be within the lymph node or, more commonly, in the node’s drainage field. For example, an allergic dermatitis may result in an eosinophilic lymphadenitis, or a lymph node draining a necrotic hemorrhagic lesion may have many macrophages containing cell debris and Fe pigments. Lymphadenitis is often associated with reactive (proplastic) changes, and the term reactive lymphadenitis is sometimes used to reflect both changes.

Lymphoma
Cytologically, lymphoma can be diagnosed when there is nearly a single population of atypical lymphocytes rather than the heterogeneous mixture of typical cell types present in normal, reactive, or inflamed lymph nodes. However, depending on the appearance of the cells, lymphoma can be an easy or difficult diagnosis cytologically.

When cytologic preparations consist of single populations of large lymphocytes with prominent nucleoli, the diagnosis of lymphoma is clear.

It is more difficult when the cells are of small to intermediate size or when substantial numbers of non-neoplastic cells are intermixed with neoplastic cells because of a nondiffuse form or a recent onset. In these cases, histologic examination may be necessary for a diagnosis.

Metastatic neoplasm
Lymph nodes can be enlarged because of the growth of non-lymphoid neoplastic cells in the node. Metastatic cells can also be found during biopsies of lymph nodes that do not appear enlarged. Many neoplasms have the potential to spread to regional lymph nodes. Those seen more frequently in the peripheral lymph nodes included squamous cell carcinoma, mammary carcinoma or adenocarcinoma, melanoma, mast cell neoplasia, and some hemic neoplasms.

Cell populations in lymphadenopathies other than lymphoma
Typical lymph nodes include popliteal, inguinal, and prescapular lymph nodes. Percentages are provided to illustrate the differences between the pathologic states. They are not firm decision limits; a true differential count is rarely completed.

<table>
<thead>
<tr>
<th>Lymphoid</th>
<th>Normal*</th>
<th>Hyperplasia #1**</th>
<th>Hyperplasia #2</th>
<th>Hyperplasia (reactive)</th>
<th>Lymphadenitis***</th>
<th>Metastatic neoplasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small</td>
<td>&gt; 80 %</td>
<td>&gt; 80 %</td>
<td>&gt; 60 %</td>
<td>&gt; 60 %</td>
<td>? &gt; 60 %</td>
<td>Varies; depends on how much of the LN has been replaced by</td>
</tr>
<tr>
<td>Intermediate</td>
<td>&lt; 10 %</td>
<td>&lt; 10 %</td>
<td>&lt; 30 %</td>
<td>&lt; 30 %</td>
<td>? &lt; 30 %</td>
<td></td>
</tr>
<tr>
<td>Large</td>
<td>&lt; 5 %</td>
<td>&lt; 5 %</td>
<td>&lt; 10 %</td>
<td>&lt; 10 %</td>
<td>? &lt; 10 %</td>
<td></td>
</tr>
</tbody>
</table>

*Percentages are provided to illustrate the differences between the pathologic states. They are not firm decision limits; a true differential count is rarely completed.
Plasma cells  < 2 %  < 2 %  < 2 %  > 2 %  ? < 2 %  neoplastic cells
Neutrophils  < 2 %  < 2 %  < 2 %  < 2 %  ? > 2 %
Macrophages  < 2 %  < 2 %  < 2 %  < 2 %  ? > 2 %
Mast cells  < 1 %  < 1 %  < 1 %  < 1 %  ? > 1 %
Organisms  ---  ---  ---  ---  Maybe  Yes

* Mandibular lymph nodes and mesenteric lymph nodes frequently have higher percentages of neutrophils, macrophages, or plasma cells
** The cell populations in this hyperplastic lymph node look like normal lymph node cells, but they came from an enlarged lymph node.
*** The distribution of the cell populations vary with the severity of the inflammatory process. The aspirate may look like a normal LN with only a minor increase in neutrophil percentage. Or, the aspirate may contain very few lymphoid cells as nearly all of the cells are inflammatory cells.

Cell populations in most lymphomas*

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Lymphoma (intermediate cell)</th>
<th>Lymphoma (large cell)</th>
<th>Lymphoma** (small cell)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphoid</td>
<td>&gt; 90 %</td>
<td>&gt; 90 %</td>
<td>&gt; 90 %</td>
</tr>
<tr>
<td>Small</td>
<td>&lt; 50 %</td>
<td>&gt; 10 %</td>
<td>&gt; 80 %</td>
</tr>
<tr>
<td>Intermediate</td>
<td>&gt; 20 %</td>
<td>&gt; 30 %</td>
<td>&lt; 10 %</td>
</tr>
<tr>
<td>Large</td>
<td>&lt; 10 %</td>
<td>&gt; 30 %</td>
<td>&lt; 5 %</td>
</tr>
<tr>
<td>Plasma cell</td>
<td>&lt; 2 %</td>
<td>&lt; 2 %</td>
<td>&lt; 2 %</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>&lt; 5 %</td>
<td>&lt; 2 %</td>
<td>&lt; 2 %</td>
</tr>
<tr>
<td>Macrophages</td>
<td>&lt; 5 %</td>
<td>&lt; 2 %</td>
<td>&lt; 2 %</td>
</tr>
<tr>
<td>Mast cells</td>
<td>&lt; 1 %</td>
<td>&lt; 1 %</td>
<td>&lt; 1 %</td>
</tr>
</tbody>
</table>

* Lymphoma classification based on the diameters of most of the neoplastic lymphoid cells in the sample: small cell = nuclei < 10 µm; intermediate (medium) cell = nuclei 10–15 µm; large cell = nuclei > 15 µm
** The small-cell lymphoma is difficult to recognize with certainty in an aspirate; the cell populations are similar to those of a normal lymph node or a hyperplastic lymph node. Histopathologic examination of an incised or excised lymph node is typically needed to establish the diagnosis.

8-1 Lymph node CVC: Mandibular LN aspirate
- Case: 053394 (1346)
- Dog, Labrador retriever, 4-yr-old

Healthy dog

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Lymphoid</th>
<th>Neutrophils</th>
<th>Neutrophils</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphoid</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Macrophages</td>
<td>%</td>
</tr>
<tr>
<td>Intermediate lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
</tr>
<tr>
<td>Large lymphocytes</td>
<td>%</td>
<td>Organisms</td>
<td>%</td>
</tr>
<tr>
<td>Plasma cells</td>
<td>%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

8-2 lymph node CVC: Mandibular LN aspirate
- Case: 021111 (2407)
- Dog, German shepherd, 3-yr-old, female (spayed)
The dog was presented because inappetence and lethargy. Physical examination revealed several mildly enlarged peripheral lymph nodes. An aspirate from the right mandibular lymph node was submitted for analysis.

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Lymphoid</th>
<th>Neutrophils</th>
<th>Neutrophils</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphoid</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Macrophages</td>
<td>%</td>
</tr>
<tr>
<td>Intermediate lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
</tr>
<tr>
<td>Large lymphocytes</td>
<td>%</td>
<td>Organisms</td>
<td>%</td>
</tr>
<tr>
<td>Plasma cell</td>
<td>%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

8-3 Lymph Node CVC: Axillary LN aspirate
- Case: 079237 (548)
- Dog, German shepherd, 3-yr-old, female (spayed)
The dog was presented because of right, foreleg lameness. Radiographs revealed a small lytic bone lesion in the humerus. An aspirate from an enlarged axillary lymph node was submitted for analysis.

315
8-4 Lymph node CVC: Prescapular LN aspirate
- Case: 053708 (554)
- Dog, Golden retriever, 5-yr-old, female (spayed)
The dog was presented because of anorexia and lethargy. Several peripheral lymph nodes were enlarged. An aspirate from an enlarged prescapular lymph node as submitted for analysis.

<table>
<thead>
<tr>
<th>Lymphoid</th>
<th>%</th>
<th>Neutrophils</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Macrophages</td>
<td>%</td>
</tr>
<tr>
<td>Intermediate lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
</tr>
<tr>
<td>Large lymphocytes</td>
<td>%</td>
<td>Organisms</td>
<td></td>
</tr>
</tbody>
</table>

8-5 Lymph node CVC: Popliteal LN aspirate
- Case: 032086 (453)
- Dog, Basset hound, 6-yr-old, male (neutered)
The dog was presented because of polyuria and polydipsia. Initial laboratory data revealed a hypercalcemia. A slightly enlarged popliteal lymph node was aspirated and the sample was submitted for analysis.

<table>
<thead>
<tr>
<th>Lymphoid</th>
<th>%</th>
<th>Neutrophils</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Macrophages</td>
<td>%</td>
</tr>
<tr>
<td>Intermediate lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
</tr>
<tr>
<td>Large lymphocytes</td>
<td>%</td>
<td>Organisms</td>
<td></td>
</tr>
</tbody>
</table>

8-6 Lymph node CVC: Inguinal LN aspirate
- Case: 028587 (539)
- Cat, Tabbi, female (spayed), 8 years old
The cat was presented because of weight loss and poor appetite. Physical examination revealed enlarged peripheral lymph node. One lymph node was aspirated and cytologic preparations were submitted for examination.

<table>
<thead>
<tr>
<th>Lymphoid</th>
<th>%</th>
<th>Neutrophils</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Macrophages</td>
<td>%</td>
</tr>
<tr>
<td>Intermediate lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
</tr>
<tr>
<td>Large lymphocytes</td>
<td>%</td>
<td>Organisms</td>
<td></td>
</tr>
</tbody>
</table>

8-7 Lymph node CVC: Popliteal LN aspirate
- Case: 028260 (542)
- Dog, Boxer, 7-yr-old, male (neutered)
A cutaneous mass on the left hind leg had been removed 10 days ago. The excised mass was not submitted for histopathologic examination. When the dog was returned for suture removal, an enlarged popliteal lymph node was found. An aspirate of the lymph node was submitted for analysis.

<table>
<thead>
<tr>
<th>Lymphoid</th>
<th>%</th>
<th>Neutrophils</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Macrophages</td>
<td>%</td>
</tr>
</tbody>
</table>
The dog was presented because it was constantly scratching ears and neck. Physical examination revealed numerous fleas, red inflamed skin, and enlarged mandibular and prescapular lymph nodes. An aspirate of the lymph node was submitted for analysis.

<table>
<thead>
<tr>
<th>Lymphoid</th>
<th>Neutrophils</th>
<th>Neutrophils</th>
<th>Neutrophils</th>
<th>Neutrophils</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
<td>Mast cells</td>
</tr>
<tr>
<td>Intermediate lymphocytes</td>
<td></td>
<td>Organisms</td>
<td>%</td>
<td>Organisms</td>
</tr>
<tr>
<td>Large lymphocytes</td>
<td>%</td>
<td>Mast cells</td>
<td>%</td>
<td>Mast cells</td>
</tr>
<tr>
<td>Plasma cells</td>
<td>%</td>
<td>Organisms</td>
<td>%</td>
<td>Organisms</td>
</tr>
</tbody>
</table>

8-8 Lymph node CVC: Prescapular LN aspirate
- Case: 028445 (537)
- Dog, Cairn terrier, 2-yr-old, female
A thorough examination of erythrocytes (RBCs) in a blood film can provide valuable information about an animal’s anemia. For those who wish to develop their microscopy skills, the following should be considered essential.

- Develop techniques to make an excellent blood film that has an even distribution of cells and a good “counting window.”
- Have a quality hematologic stain that can provide reproducible results; quick stains can be acceptable
- Have a quality microscope that has excellent 40x- or 50x-oil and 100-x oil objectives (these objectives might cost $3000 to $5000 each)
- Have excellent textbooks and atlases for the species of interest (see list below)
- Have knowledge of the types of anemias that can be found and the many variations of each disorder

Erythrocytes in a stained blood film are Red Cell Cadavers – they are cells that died of dehydration (air-dried) and transformed into 2-dimensional shapes that can provide clues to what they were as living cells. Every erythrocyte in a blood film is an artifact – the cell did not have that shape or appearance when it was circulating in blood. A key aspect in blood film evaluations is recognizing artifacts that tell us something about the animal versus artifacts that are distractions.

During this session, we will use case information to provide a framework for the evaluation of Red Cell Cadavers. After examining pertinent microscopic fields, an audience response system will be used to assess your knowledge. The reasons for the observed poikilocytosis, anisocytosis, or other erythrocyte abnormalities will be explained.

The following tables are located at the end of the document.

- Table 3.6. Erythrocyte inclusions other than organisms: identifying features, clinical significance, and associated pathogenic processes
- Table 3.7. Poikilocytes: identifying features, clinical significance, and pathogeneses in domestic mammals

There are many books that provide images of blood cells and/or explain the significance of abnormal cells. The first listed book (by John Harvey) provides the most comprehensive set of images. The other books can also be valuable.

7. Duncan & Prasse’s Veterinary Laboratory Medicine; Clinical Pathology; 5th ed., KS Latimer, 2011
8. Atlas of Veterinary Hematology: Blood and Bone Marrow of Domestic Animals, JW Harvey, 2001

1-1 RBC CVC: Blood film from anemic cat

- Case: 194644 (98, 435)
- Cat, domestic long hair, male(c), 6-yr-old
- History: Listless, depressed, weak, and anorectic for 3 days
- PE: temp 102.3 °F, heart rate 128/min, resp. rate 80/min, dehydrated (5 %), pale mucous membranes, depressed, weak, and underweight
- CBC (plasma mild icterus)

<table>
<thead>
<tr>
<th>pTP</th>
<th>7.4</th>
<th>g/dL</th>
<th>6.0-8.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hct (s)</td>
<td>22 %</td>
<td>30-45</td>
<td></td>
</tr>
<tr>
<td>Hct (c)</td>
<td>20 %</td>
<td>30-45</td>
<td></td>
</tr>
<tr>
<td>Hgb</td>
<td>6.1</td>
<td>g/dL</td>
<td>9.0-15.0</td>
</tr>
<tr>
<td>RBC</td>
<td>3.4 x 10^6/µL</td>
<td>5.5-10.0</td>
<td></td>
</tr>
<tr>
<td>MCV</td>
<td>59 fl</td>
<td>39-55</td>
<td></td>
</tr>
<tr>
<td>MCHC</td>
<td>30 g/dL</td>
<td>30-36</td>
<td></td>
</tr>
<tr>
<td>MCH</td>
<td>18 pg</td>
<td>13-17</td>
<td></td>
</tr>
<tr>
<td>nRBC</td>
<td>41 /100 WBC</td>
<td>0-1</td>
<td></td>
</tr>
<tr>
<td>WBC (corrected)</td>
<td>7.8 x 10^3/µL</td>
<td>5.5-19.5</td>
<td></td>
</tr>
<tr>
<td>Seg. neut.</td>
<td>40 %</td>
<td>3.1 x 10^3/µL</td>
<td>2.5-12.5</td>
</tr>
<tr>
<td>Band neut.</td>
<td>0 %</td>
<td>0.0 x 10^3/µL</td>
<td>0.0-0.3</td>
</tr>
<tr>
<td>Lymph.</td>
<td>56 %</td>
<td>4.4 x 10^3/µL</td>
<td>1.5-7.0</td>
</tr>
<tr>
<td>Mono.</td>
<td>4 %</td>
<td>0.3 x 10^3/µL</td>
<td>0.0-0.8</td>
</tr>
<tr>
<td>Eos.</td>
<td>0 %</td>
<td>0.0 x 10^3/µL</td>
<td>0.0-0.8</td>
</tr>
<tr>
<td>Baso.</td>
<td>0 %</td>
<td>0.0 x 10^3/µL</td>
<td>0.0-0.1</td>
</tr>
<tr>
<td>Platelet</td>
<td>clumped</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
1-2 RBC CVC: Blood film from anemic cat

- Case: 305729 (357, 441)
- Cat, domestic long hair, female, 5-mo-old
- History: Cat was presented because it was not growing well; it was considerably smaller than its littermate; no other problems were noted by the owner. To increase eating, the owner has been feeding the cat a diet consistent of chicken and vegetables (carrots, celery, & onions).
- PE: temp heart rate, and resp. rate OK, not dehydrated, pale mucous membranes, underweight (2.6 lb)
- CBC (plasma mild hemolysis)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>6.4 g/dL</td>
<td>6.0-8.0</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>23 %</td>
<td>30-45</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>24 %</td>
<td>30-45</td>
</tr>
<tr>
<td>Hgb</td>
<td>9.5 g/dL</td>
<td>9.0-15.0</td>
</tr>
<tr>
<td>RBC</td>
<td>5.4 x 10^6/μL</td>
<td>5.5-10.0</td>
</tr>
<tr>
<td>MCHC</td>
<td>40 g/dL</td>
<td>30-36</td>
</tr>
<tr>
<td>MCH</td>
<td>18 pg</td>
<td>13-17</td>
</tr>
<tr>
<td>nRBC</td>
<td>4 /100 WBC</td>
<td>0-1</td>
</tr>
<tr>
<td>WBC (corrected)</td>
<td>56.4 x 10^3/μL</td>
<td>5.5-19.5</td>
</tr>
<tr>
<td>Seg. neut.</td>
<td>77 %</td>
<td>2.5-12.5</td>
</tr>
<tr>
<td>Band neut.</td>
<td>2 %</td>
<td>0.0-0.3</td>
</tr>
<tr>
<td>Lymph.</td>
<td>10 %</td>
<td>1.5-7.0</td>
</tr>
<tr>
<td>Mono.</td>
<td>5 %</td>
<td>0.0-0.8</td>
</tr>
<tr>
<td>Baso.</td>
<td>1 %</td>
<td>0.0-0.1</td>
</tr>
</tbody>
</table>

1-3 RBC CVC: Blood film from anemic dog

- Case: 074802 (216)
- Dog, mixed breed, female (spayed), 9-yr-old
- The dog was presented with a complaint of progressive lethargy and inappetence. Physical examination revealed a nonfebrile dog that had icteric mucous membranes and an enlarged spleen.
- CBC (plasma icteric)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>7.6 g/dL</td>
<td>6.0-8.0</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>30 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>31 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>9.3 g/dL</td>
<td>12.0-18.0</td>
</tr>
<tr>
<td>RBC</td>
<td>3.7 x 10^6/μL</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>85 fl</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>30 g/dL</td>
<td>33-37</td>
</tr>
<tr>
<td>MCH</td>
<td>26 pg</td>
<td>21-26</td>
</tr>
<tr>
<td>nRBC</td>
<td>0 /100 WBC</td>
<td>0-1</td>
</tr>
<tr>
<td>WBC</td>
<td>39.7 x 10^3/μL</td>
<td>6.0-17.0</td>
</tr>
<tr>
<td>Seg. neut.</td>
<td>78 %</td>
<td>3.0-11.5</td>
</tr>
<tr>
<td>Band neut.</td>
<td>11 %</td>
<td>0.0-0.3</td>
</tr>
<tr>
<td>Lymph.</td>
<td>2 %</td>
<td>1.0-4.8</td>
</tr>
<tr>
<td>Mono.</td>
<td>8 %</td>
<td>0.2-1.4</td>
</tr>
<tr>
<td>Baso.</td>
<td>0 %</td>
<td>0.0-0.1</td>
</tr>
<tr>
<td>Platelets</td>
<td></td>
<td>200-500</td>
</tr>
</tbody>
</table>

Chemistry profile (serum icteric)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea nitrogen</td>
<td>29 mg/dL</td>
<td>9-33</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.7 mg/dL</td>
<td>0.5-1.5</td>
</tr>
<tr>
<td>Glucose</td>
<td>150 mg/dL</td>
<td>73-113</td>
</tr>
<tr>
<td>Total protein</td>
<td>6.0 g/dL</td>
<td>5.4-7.5</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.5 g/dL</td>
<td>3.4-4.2</td>
</tr>
<tr>
<td>Globulin</td>
<td>2.5 g/dL</td>
<td>1.3-3.2</td>
</tr>
<tr>
<td>Ca²⁺, total</td>
<td>9.9 mg/dL</td>
<td>9.7-12.1</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>2.5 mg/dL</td>
<td>2.4-6.4</td>
</tr>
<tr>
<td>Bilirubin, total</td>
<td>3.1 mg/dL</td>
<td>0.1-0.3</td>
</tr>
<tr>
<td>Na⁺</td>
<td>145 mmol/L</td>
<td>147–154</td>
</tr>
<tr>
<td>K⁺</td>
<td>3.6 mmol/L</td>
<td>3.6-5.3</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>120 mmol/L</td>
<td>108–118</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>12 mmol/L</td>
<td>18–29</td>
</tr>
<tr>
<td>Anion gap</td>
<td>18 mmol/L</td>
<td>16-26</td>
</tr>
<tr>
<td>ALT</td>
<td>683 U/L</td>
<td>28-171</td>
</tr>
<tr>
<td>ALP</td>
<td>881 U/L</td>
<td>1-142</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>390 U/L</td>
<td>128-328</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>328 mg/dL</td>
<td>133-394</td>
</tr>
</tbody>
</table>

Urinalysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collection</td>
<td>Voided</td>
<td>pH 6.5</td>
</tr>
<tr>
<td>Color</td>
<td>None</td>
<td>WBC 0-2 / hpf</td>
</tr>
<tr>
<td>Transp</td>
<td>Clear</td>
<td>RBC None / hpf</td>
</tr>
<tr>
<td>Glucose</td>
<td>Trace</td>
<td>Epithelial cells</td>
</tr>
<tr>
<td>USGref</td>
<td>1.035</td>
<td>Occ. Squamous / lpf</td>
</tr>
<tr>
<td>Heme</td>
<td>Neg</td>
<td>Casts None / lpf</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>+3</td>
<td>Bacteria None / hpf</td>
</tr>
<tr>
<td>Urobilinogen</td>
<td>0.2</td>
<td></td>
</tr>
</tbody>
</table>

1-4 RBC CVC: Blood film from anemic dog

- Case: 270417 (1467)
- Dog, mixed breed, female, 1-yr-old
The dog was presented with a complaint of progressive weakness of 4 days duration and passing dark tarry stools 3 to 4 times per day the last 2 days. Physical examination revealed a nonfebrile dog that had pale mucous membranes, tachycardia, and mild cardiac murmur.

**CBC (plasma clear)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>4.2 g/dL</td>
<td>6.0-8.0</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>7%</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>6%</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>1.8 g/dL</td>
<td>12.0-18.0</td>
</tr>
<tr>
<td>RBC</td>
<td>1.4 x 10^12/\mu L</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>43 fl</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>30 g/dL</td>
<td>33-37</td>
</tr>
<tr>
<td>MCH</td>
<td>13 pg</td>
<td>21-26</td>
</tr>
<tr>
<td>nRBC</td>
<td>0/100 WBC</td>
<td>0-1</td>
</tr>
</tbody>
</table>

**1-5 RBC CVC: Blood film from anemic dog**
- Case: 299404 (518)
- Dog, golden retriever, Fe(s), 7-yr-old
- Two months prior to this presentation, the dog had a splenectomy for partial management of disseminated mast cell neoplasia involving spleen, lymph nodes, and skin. This presentation was part of a planned appointment to assess progress. The owner reported the dog had been eating and feeling well. Physical examination revealed a swollen left foreleg and edema in the left axillary region.

**CBC (plasma clear)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>5.2 g/dL</td>
<td>6.0-8.0</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>18%</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>17%</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>5.7 g/dL</td>
<td>12.0-18.0</td>
</tr>
<tr>
<td>RBC</td>
<td>2.2 x 10^12/\mu L</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>81 fl</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>33 g/dL</td>
<td>33-37</td>
</tr>
<tr>
<td>MCH</td>
<td>26 pg</td>
<td>21-26</td>
</tr>
<tr>
<td>nRBC</td>
<td>0/100 WBC</td>
<td>0-1</td>
</tr>
</tbody>
</table>

**1-6 RBC CVC: Blood film from dog**
- Case: 275480 (326)
- Dog, mixed breed, male, 10-mo-old
- The dog was referred because it had intermittent diarrhea for 2 months; there was no sustained response to symptomatic treatments. Last night, the dog got into a praying position (head down between front legs, hind legs up) which suggests anterior abdominal pain. Physical examination revealed mild tachycardia, 5% dehydration, pink mucous membranes, many fleas, and abdominal tenderness.

**CBC (plasma clear)**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Ranges</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>6.0 g/dL</td>
<td>6.0-8.0</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>38%</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>37%</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>13 g/dL</td>
<td>12.0-18.0</td>
</tr>
<tr>
<td>RBC</td>
<td>5.9 x 10^12/\mu L</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>63 fl</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>34 g/dL</td>
<td>33-37</td>
</tr>
<tr>
<td>MCH</td>
<td>21 pg</td>
<td>21-26</td>
</tr>
<tr>
<td>nRBC</td>
<td>140/100 WBC</td>
<td>0-1</td>
</tr>
</tbody>
</table>

Tables 3.6 and 3.7 from *Fundamentals of Veterinary Clinical Pathology, 2nd edition* (S.L. Stockham & M.A. Scott)

**Table 3.6. Erythrocyte inclusions other than organisms: identifying features, clinical significance, and associated pathogenic processes**

<table>
<thead>
<tr>
<th>Inclusions</th>
<th>Identifying features</th>
<th>Clinical significance</th>
<th>Associated pathogenic processes</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Basophilic stippling</em></td>
<td>Fine to coarse, blue to dark purple dots</td>
<td>Regenerative anemia (especially Young cells)—persistence of</td>
<td></td>
</tr>
</tbody>
</table>

320
321

or specks that represent aggregated ribosomes dispersed in an erythrocyte’s cytoplasm (Plate 4G)

Heinz body
Slightly pale, rounded, protruding structure that creates a membrane defect; may occur as free body; stains blue with NMB stain (Plate 4H and I)

Exposure to oxidants
Oxidants overwhelm reductive capacity of erythrocyte; hemoglobin precipitates and may bind with erythrocyte membrane

Hemoglobin crystals
Intensely stained, crystallized hemoglobin that forms a pencil, parallelogram, cube, or other polyhedron within erythrocytes (Plate 4J)

None in domestic mammals; most frequent in cats (and camelids)

*Howell-Jolly body
Usually a homogeneous, dark purple–staining, round structure in erythrocytes; not associated with membrane; can be ring forms (especially in cats)

Increased erythropoiesis, decreased splenic function
Nuclear remnant that remained free in the cytoplasm after mitosis; persists in erythrocyte if the spleen does not pit it

Siderotic granules
Loose aggregate of fine granular basophilic inclusions; stain blue with Fe stains (Prussian blue) (Plate 5B)

Excess Fe in body; plumbism in dogs; myeloproliferative disease, usually unknown

Fe accumulates in damaged mitochondria or in autophagocytic vacuoles

* A relatively common inclusion (Note: Basophilic stippling is more common in cattle than in dogs and cats, and it is not expected in horses.)

*appearance as seen on a Wright-stained blood film unless stated otherwise

Table 3.7. Poikilocytes: identifying features, clinical significance, and pathogenesis in domestic mammals

<table>
<thead>
<tr>
<th>Poikilocyte</th>
<th>Other name</th>
<th>Identifying features</th>
<th>Clinical significance</th>
<th>Pathogenesis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acanthocyte</td>
<td>Spur cell, burr cell</td>
<td>Spherical cell with 1–20 irregularly spaced, membrane projections of variable lengths; projections may blunt spurs or clubs (Plate 5G and 6J)</td>
<td>Hemangiosarcoma; occasionally splenic, hepatic, and renal disorders</td>
<td>Unknown in domestic mammals; can form from changes in membrane lipids; possibly fragmentation</td>
</tr>
<tr>
<td>*Codocyte (codo = “hat”)</td>
<td>Target cell, Mexican hat cell</td>
<td>Central focus of Hgb that is surrounded by a ring of pallor that separates it from peripheral Hgb; one form of leptocyte (Plate 5H)</td>
<td>Typical with regenerative anemias; also seen with hepatic, renal, and lipid disorders</td>
<td>Excess membrane relative to Hgb content; may occur with membrane lipid changes</td>
</tr>
<tr>
<td>Dacryocyte</td>
<td>—</td>
<td>Teardrop shaped (Plate 5I and J)</td>
<td>Marrow diseases such as myelofibrosis and neoplasia; also may be an artifact</td>
<td>Unknown except artifacts caused by stretching during film preparation</td>
</tr>
<tr>
<td>*Echinocyte (echino = “spiny”)</td>
<td>Burr cell</td>
<td>Vary from irregularly shaped cells (type I), to regularly spaced blunt projections (type II), to regularly spaced pointed projections (type III) (Plate 5L)</td>
<td>Hyponatremic dehydration, doxorubicin toxicity</td>
<td>Multiple causes (see the text)</td>
</tr>
<tr>
<td>Crenated erythrocyte</td>
<td>—</td>
<td>Crenated cells are artifacts Prolonged exposure to alkaline glass while drying</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Elliptocyte</td>
<td>(See ovalocyte)</td>
<td>Notched, flattened margin between two membrane projections (horns); variant has one horn (Plate 6A)</td>
<td>Vascularity, intravascular coagulation, hemangiosarcoma, caval syndrome, endocarditis Fe deficiency</td>
<td>Unclear: trauma, oxidative injury, and vesiculation have all been proposed</td>
</tr>
<tr>
<td>*Keratocyte (kerato = “horn”)</td>
<td>Helmet cell</td>
<td>Notched, flattened margin between two membrane projections (horns); variant has one horn (Plate 6A)</td>
<td>Vascularity, intravascular coagulation, hemangiosarcoma, caval syndrome, endocarditis Fe deficiency</td>
<td>Unclear: trauma, oxidative injury, and vesiculation have all been proposed</td>
</tr>
<tr>
<td>Leptocyte</td>
<td>—</td>
<td>Thin cell that appears as a hypochromic cell with increased central pallor (Plate 6B)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Poikilocyte</td>
<td>Other name</td>
<td>Identifying features</td>
<td>Clinical significance</td>
<td>Pathogenesis</td>
</tr>
<tr>
<td>----------------</td>
<td>---------------------</td>
<td>--------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Ovalocyte</td>
<td>Elliptocyte</td>
<td>Elliptical or oval cell (Plate 6C)</td>
<td>Protein band 4.1 deficiency in dogs, mutant proteins in hereditary spectrin in a dog,</td>
<td>Abnormal membrane deficiency in dogs, mutant proteins in hereditary spectrin in a dog, form, otherwise unknown</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>idiopathic myelofibrosis, idiopathic in cats, iron deficiency</td>
<td></td>
</tr>
<tr>
<td>Pincered cell</td>
<td>—</td>
<td>Button or knob joined to rest of cell by a pinched area (Plate 6D)</td>
<td>PK deficiency,</td>
<td>Unknown</td>
</tr>
<tr>
<td>Pyknocyte</td>
<td>Irregularly contracted cell</td>
<td>Spheroid erythrocyte with condensed or contracted Hgb and perhaps small tags of fragmented membrane (Plate 6E and F)</td>
<td>Overwhelming exposure, Unclear; may form from to oxidants; also rare cases eccentrocytes</td>
<td>G6PD or FAD deficiencies</td>
</tr>
<tr>
<td>*Schizocyte</td>
<td>RBC fragment, schistocyte</td>
<td>Triangular, comma-shaped, small round, or irregularly shaped piece of an erythrocyte (Plate 6G)</td>
<td>Intravascular coagulation, Same as keratocyte, hemangiosarcoma, caval syndrome, endocarditis</td>
<td></td>
</tr>
<tr>
<td>Selenocyte</td>
<td>—</td>
<td>A damaged erythrocyte that is crescent-shaped and has a large clear space</td>
<td>Associated with hemolytic Artifact (See the text) anemias, fragile erythrocytes</td>
<td></td>
</tr>
<tr>
<td>*Spherocyte</td>
<td>—</td>
<td>Decreased central pallor, decreased cell diameter, increased Hgb staining intensity, and smooth margins (Plate 6I and J)</td>
<td>Immune hemolysis, fragmentation hemolysis, action of macrophages or envenomations, clostridial trauma or abnormal infections, hereditary band cytoskeleton 3 deficiency</td>
<td>Membrane loss due to the action of macrophages or trauma or abnormal infections, hereditary band cytoskeleton 3 deficiency</td>
</tr>
<tr>
<td>Stomatocyte</td>
<td>—</td>
<td>Elongated (slitlike or mouthlike) area of cyttoplasmic pallor (Plate 6K)</td>
<td>Young erythrocytes or hereditary stomatocytosis membrane of dogs</td>
<td>Folding of excess membrane of dogs</td>
</tr>
<tr>
<td>Torocyte</td>
<td>—</td>
<td>Punched-out, central clear space that creates a donut-shaped cell (Plate 6L)</td>
<td>None; do not confuse with Artifact hypochromia</td>
<td></td>
</tr>
</tbody>
</table>

* A relatively common poikilocyte

* Classifying cells as burr cells is not recommended because the name is used for acanthocytes and echinocytes.
2-1 RBC CVC: Blood film from anemic dog

- Case: ASVCP 16-7 (2866)
- Dog, mixed-breed, 5-yr-old, female
- The patient presented with lethargy and hind-limb bruising. On Day 1, the dog’s TPR values were OK and it was laterally recumbent, quiet, responsive, and dehydrated and marked edema and bruising on her left hind limb with two adjacent puncture marks that oozed blood. The dog received supportive therapy for dehydration, pain control, and wound management. On Day 3 of hospitalization, the dog became progressively anemic, and clinical pathology testing was repeated (blood film from Day 3).

CBC (note: ↑ & ↓ arrows not shown for results; abnormal results in bold)

<table>
<thead>
<tr>
<th></th>
<th>D-1</th>
<th>D-3</th>
<th>WBC (not corrected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma</td>
<td>Pink</td>
<td>Pink</td>
<td>15.7 10.8 x 10^3/μL</td>
</tr>
<tr>
<td>pTP</td>
<td>4.0</td>
<td>4.3</td>
<td>5.9-8.2</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>20%</td>
<td>20%</td>
<td>43-62</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>20%</td>
<td>20%</td>
<td>43-62</td>
</tr>
<tr>
<td>Hgb</td>
<td>7.5 g/dL</td>
<td>7.5 g/dL</td>
<td>14.8-21.1</td>
</tr>
<tr>
<td>RBC</td>
<td>5.5 x 10^6/μL</td>
<td>5.9-8.6</td>
<td>68</td>
</tr>
<tr>
<td>MCV</td>
<td>72.68 g/dL</td>
<td>66-77</td>
<td>336</td>
</tr>
<tr>
<td>MCHC</td>
<td>34.37 g/dL</td>
<td>33-36</td>
<td>21-26</td>
</tr>
<tr>
<td>MCH</td>
<td>25.25 pg</td>
<td>21-26</td>
<td>0-1</td>
</tr>
<tr>
<td>nRBC</td>
<td>0.0 /100 WBC</td>
<td>0-1</td>
<td>Platelets 175 68 x 10^3/μL</td>
</tr>
</tbody>
</table>

Chemistry profile (serum mild hemolysis) (note: ↑ & ↓ arrows not shown; abnormal data in bold)

<table>
<thead>
<tr>
<th></th>
<th>D-1</th>
<th>D-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea nitrogen</td>
<td>17.8 mg/dL</td>
<td>9-30</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.7</td>
<td>0.7-1.3</td>
</tr>
<tr>
<td>Glucose</td>
<td>160</td>
<td>88-121</td>
</tr>
<tr>
<td>Total protein</td>
<td>4.3</td>
<td>5.3-7.0</td>
</tr>
<tr>
<td>Albumin</td>
<td>2.4</td>
<td>2.8-3.7</td>
</tr>
<tr>
<td>Globulin</td>
<td>1.9</td>
<td>2.1-3.8</td>
</tr>
<tr>
<td>Ca^2+ total</td>
<td>9.0</td>
<td>9.4-10.7</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>3.7</td>
<td>1.9-4.4</td>
</tr>
<tr>
<td>Bilirubin, total</td>
<td>0.2</td>
<td>0.2-0.4</td>
</tr>
</tbody>
</table>

2-2 RBC CVC: Blood film from anemic dog

- Case: 006320 (513)
- Dog, Gordon setter, 9-yr-old
- About a month ago and during diagnostic testing for inappropriate urinations, ultrasonography revealed several splenic masses. At this time, CBC results and a chemical profile results were within reference intervals. A urinalysis revealed a proteinuria. Exploratory abdominal surgery found several elevated splenic masses and the spleen was removed. The dog recovered well from the surgery. However, the current problem is weakness and icteric mucous membranes.

CBC (plasma icteric)

<table>
<thead>
<tr>
<th></th>
<th>D-1</th>
<th>D-3</th>
<th>WBC (not corrected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>6.9 g/dL</td>
<td>6.0-8.0</td>
<td>7.7 x 10^3/μL</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>14%</td>
<td>37-55</td>
<td>6.6 x 10^3/μL</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>15%</td>
<td>37-55</td>
<td>2.5 x 10^3/μL</td>
</tr>
<tr>
<td>Hgb</td>
<td>5.1 g/dL</td>
<td>12.0-18.0</td>
<td>0%</td>
</tr>
<tr>
<td>RBC</td>
<td>2.2 x 10^6/μL</td>
<td>5.5-8.5</td>
<td>Band neut. 0%</td>
</tr>
<tr>
<td>MCV</td>
<td>68 fL</td>
<td>62-76</td>
<td>Lymph. 38% 2.5 x 10^3/μL</td>
</tr>
<tr>
<td>MCHC</td>
<td>34 g/dL</td>
<td>33-37</td>
<td>Eos. 4% 0.2 x 10^3/μL</td>
</tr>
<tr>
<td>MCH</td>
<td>23 pg</td>
<td>21-26</td>
<td>Baso. 0% 0.0 x 10^3/μL</td>
</tr>
<tr>
<td>nRBC</td>
<td>16 /100 WBC</td>
<td>0-1</td>
<td>Platelets 280 x 10^3/μL</td>
</tr>
</tbody>
</table>
### CBC (plasma slightly icteric)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>5.0 g/dL</td>
<td>5.7-7.2 g/dL</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>32 %</td>
<td>36-54 %</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>31 %</td>
<td>36-54 %</td>
</tr>
<tr>
<td>Hgb</td>
<td>10.4 g/dL</td>
<td>11.9-18.4 g/dL</td>
</tr>
<tr>
<td>RBC</td>
<td>4.4 x 10^6/μL</td>
<td>4.9-8.2 x 10^6/μL</td>
</tr>
<tr>
<td>MCV</td>
<td>72 fL</td>
<td>64-75 fL</td>
</tr>
<tr>
<td>MCHC</td>
<td>33.1 g/dL</td>
<td>33-36 g/dL</td>
</tr>
<tr>
<td>MCH</td>
<td>24 pg</td>
<td>20-25 pg</td>
</tr>
<tr>
<td>nRBC</td>
<td>0 /100 WBC</td>
<td>0-1 /100 WBC</td>
</tr>
<tr>
<td>WBC</td>
<td>14.0 x 10^9/μL</td>
<td>3.0-10.4 x 10^9/μL</td>
</tr>
<tr>
<td>Seg. neut.</td>
<td>87 %</td>
<td>80-100 %</td>
</tr>
<tr>
<td>Band neut.</td>
<td>5 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Lymph.</td>
<td>4 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Mono.</td>
<td>4 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Eos.</td>
<td>0 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Baso.</td>
<td>0 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Platelets</td>
<td>105 x 10^9/μL</td>
<td>100-400 x 10^9/μL</td>
</tr>
</tbody>
</table>

### Chemistry profile (serum clear)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea nitrogen</td>
<td>28 mg/dL</td>
<td>5-20 mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>2.9 mg/dL</td>
<td>0.6-1.6 mg/dL</td>
</tr>
<tr>
<td>Glucose</td>
<td>--- mg/dL</td>
<td>75-120 mg/dL</td>
</tr>
<tr>
<td>Total protein</td>
<td>2.1 g/dL</td>
<td>5.1-7.1 g/dL</td>
</tr>
<tr>
<td>Albumin</td>
<td>1.0 g/dL</td>
<td>2.9-4.2 g/dL</td>
</tr>
<tr>
<td>Globulin</td>
<td>1.1 g/dL</td>
<td>2.2-2.9 g/dL</td>
</tr>
<tr>
<td>Ca²⁺, total</td>
<td>7.9 mg/dL</td>
<td>9.3-11.6 mg/dL</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>8.1 mg/dL</td>
<td>3.2-8.1 mg/dL</td>
</tr>
<tr>
<td>Bilirubin, total</td>
<td>2.3 mg/dL</td>
<td>0.1-0.4 mg/dL</td>
</tr>
<tr>
<td>WBC</td>
<td>21.9 x 10^9/μL</td>
<td>19.0 x 10^9/μL</td>
</tr>
<tr>
<td>Seg. neut.</td>
<td>87 %</td>
<td>80-100 %</td>
</tr>
<tr>
<td>Band neut.</td>
<td>5 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Lymph.</td>
<td>4 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Mono.</td>
<td>4 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Eos.</td>
<td>0 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Baso.</td>
<td>0 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Platelets</td>
<td>80 x 10^9/μL</td>
<td>100-400 x 10^9/μL</td>
</tr>
</tbody>
</table>

### CBC (plasma icteric)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>6.5 g/dL</td>
<td>6.0-8.0 g/dL</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>16 %</td>
<td>37-55 %</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>16 %</td>
<td>37-55 %</td>
</tr>
<tr>
<td>Hgb</td>
<td>5.2 g/dL</td>
<td>12.0-18.0 g/dL</td>
</tr>
<tr>
<td>RBC</td>
<td>2.2 x 10^6/μL</td>
<td>5.5-8.5 x 10^6/μL</td>
</tr>
<tr>
<td>MCV</td>
<td>71 fL</td>
<td>62-76 fL</td>
</tr>
<tr>
<td>MCHC</td>
<td>33 g/dL</td>
<td>33-37 g/dL</td>
</tr>
<tr>
<td>MCH</td>
<td>24 pg</td>
<td>21-26 pg</td>
</tr>
<tr>
<td>nRBC</td>
<td>0 /100 WBC</td>
<td>0-1 /100 WBC</td>
</tr>
<tr>
<td>WBC</td>
<td>21.9 x 10^9/μL</td>
<td>19.0 x 10^9/μL</td>
</tr>
<tr>
<td>Seg. neut.</td>
<td>87 %</td>
<td>80-100 %</td>
</tr>
<tr>
<td>Band neut.</td>
<td>5 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Lymph.</td>
<td>4 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Mono.</td>
<td>4 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Eos.</td>
<td>0 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Baso.</td>
<td>0 %</td>
<td>0-10 %</td>
</tr>
<tr>
<td>Platelets</td>
<td>80 x 10^9/μL</td>
<td>100-400 x 10^9/μL</td>
</tr>
</tbody>
</table>

### Chemistry profile (serum icteric)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea nitrogen</td>
<td>33 mg/dL</td>
<td>8-30 mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.8 mg/dL</td>
<td>0.5-1.5 mg/dL</td>
</tr>
<tr>
<td>Glucose</td>
<td>69 mg/dL</td>
<td>60-120 mg/dL</td>
</tr>
<tr>
<td>Total protein</td>
<td>2.5 g/dL</td>
<td>3.0-4.5 g/dL</td>
</tr>
<tr>
<td>Albumin</td>
<td>3.5 g/dL</td>
<td>1.8-4.2 g/dL</td>
</tr>
<tr>
<td>Globulin</td>
<td>9.1 mg/dL</td>
<td>8.2-12.8 mg/dL</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>6.5 mg/dL</td>
<td>2.3-6.5 mg/dL</td>
</tr>
<tr>
<td>Bilirubin, total</td>
<td>1.4 mg/dL</td>
<td>0.1-0.4 mg/dL</td>
</tr>
</tbody>
</table>

---

2-3 CBC CVC: Blood film from anemic dog  
Case: ASVCP 10-11 (2869)

- Dog, Airedale terrier, 3-yr-old, male(c)
- The patient presented to an emergency clinic because of smoke inhalation that occurred during a house fire. Adequate oxygenation was not obtained after emergency intubation and a mechanical ventilator. Continued supportive therapy included mechanical ventilation and constant rate infusions of propofol, fentanyl, ketamine, and diazepam. The laboratory data and blood film are from the 4th day of hospitalization and supportive therapy.

2-4 CBC CVC: Blood film from anemic dog  
Case: 037788 (498)

- Dog mixed breed, female, 13-yr-old
- The dog was referred because of anemia and an enlarged abdomen. The owner thought the dog’s abdomen had been enlarged for about a month but the dog did not appear ill. Recent problems included 2 days of vomiting and anorexia. Physical examination revealed pale mucous membranes, a distended abdomen (probably fluid-filled, and possibly an abdominal mass).
Urinalysis

<table>
<thead>
<tr>
<th>Collection</th>
<th>Voided</th>
<th>pH</th>
<th>6.5</th>
<th>WBC</th>
<th>0-3 / hpf</th>
</tr>
</thead>
<tbody>
<tr>
<td>Color</td>
<td>None</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transp.</td>
<td>Hazy</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>USGref</td>
<td>1.048</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ Bilirubin</td>
<td>+2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ Urobilinogen</td>
<td>0.2</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Protein</th>
<th>Trace</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>Neg</td>
</tr>
<tr>
<td>Ketone</td>
<td>Neg</td>
</tr>
<tr>
<td>Heme</td>
<td>Neg</td>
</tr>
<tr>
<td>↑ Epithelial cells</td>
<td>squamous / lpf</td>
</tr>
<tr>
<td>casts</td>
<td>None / lpf</td>
</tr>
<tr>
<td>crystals</td>
<td>None / lpf</td>
</tr>
<tr>
<td>↑ Bacteria</td>
<td>None / hpf</td>
</tr>
<tr>
<td>Transp.</td>
<td>Hazy</td>
</tr>
<tr>
<td>USGref</td>
<td>1.048</td>
</tr>
</tbody>
</table>

2-5 RBC CVC: Blood film from anemic dog
- Case: 831139 (1791)
- Dog, mixed-breed Labrador, female, 3-yr-old
- The dog was presented after a sudden onset of weakness and lethargy; the owner became greatly concerned when red urine was passed. Physical examination revealed a nonfebrile dog that had pale mucous membranes.

CBC (plasma hemolyzed)

<table>
<thead>
<tr>
<th>pTP</th>
<th>7.7 g/dL</th>
<th>6.0-8.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hct (s)</td>
<td>13 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>14 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>4.8 g/dL</td>
<td>12.0-18.0</td>
</tr>
<tr>
<td>RBC</td>
<td>1.9 x 10^9/μL</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>74 fL</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>34 g/dL</td>
<td>33-37</td>
</tr>
<tr>
<td>MCH</td>
<td>25 pg</td>
<td>21-26</td>
</tr>
<tr>
<td>nRBC</td>
<td>0 /100 WBC</td>
<td>0-1</td>
</tr>
<tr>
<td>↑ WBC (not corrected)</td>
<td>37.6 x 10^3/μL</td>
<td>6.0-17.0</td>
</tr>
<tr>
<td>↑ WBC (corrected)</td>
<td>28.7 x 10^3/μL</td>
<td>6.0-17.0</td>
</tr>
<tr>
<td>↑ Seg. neut.</td>
<td>59 %</td>
<td>16.9 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Band neut.</td>
<td>13 %</td>
<td>3.7 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Lymph.</td>
<td>10 %</td>
<td>2.9 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Mono.</td>
<td>15 %</td>
<td>4.3 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Eos.</td>
<td>3 %</td>
<td>0.9 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Baso.</td>
<td>0 %</td>
<td>0.0 x 10^3/μL</td>
</tr>
<tr>
<td>Platelets</td>
<td>decreased</td>
<td>x 10^3/μL</td>
</tr>
</tbody>
</table>

2-6 RBC CVC: Blood film from anemic dog
- Case: 154520 (2320)
- Dog, German shepherd, male(c), 6-yr-old
- The dog was presented because of lethargy and an enlarged abdomen. Physical examination revealed a nonfebrile dog that had an abdominal effusion and petechiae in oral mucous membranes.

CBC (plasma clear)

<table>
<thead>
<tr>
<th>pTP</th>
<th>6.2 g/dL</th>
<th>6.0-8.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hct (s)</td>
<td>34 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>33 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>11.8 g/dL</td>
<td>12.0-18.0</td>
</tr>
<tr>
<td>RBC</td>
<td>4.9 x 10^6/μL</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>66 fL</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>36 g/dL</td>
<td>33-37</td>
</tr>
<tr>
<td>MCH</td>
<td>24 pg</td>
<td>21-26</td>
</tr>
<tr>
<td>↑ nRBC</td>
<td>2 /100 WBC</td>
<td>0-1</td>
</tr>
<tr>
<td>↑ WBC</td>
<td>23.2 x 10^3/μL</td>
<td>6.0-17.0</td>
</tr>
<tr>
<td>↑ Seg. neut.</td>
<td>83 %</td>
<td>19.3 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Band neut.</td>
<td>4 %</td>
<td>0.9 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Lymph.</td>
<td>7 %</td>
<td>1.6 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Mono.</td>
<td>6 %</td>
<td>1.4 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Eos.</td>
<td>0 %</td>
<td>0.0 x 10^3/μL</td>
</tr>
<tr>
<td>↑ Baso.</td>
<td>0 %</td>
<td>0.0 x 10^3/μL</td>
</tr>
<tr>
<td>Platelets</td>
<td>110 x 10^3/μL</td>
<td>200-500</td>
</tr>
</tbody>
</table>
A cytologic biopsy (aka, fine needle biopsy or fine needle aspiration biopsy or “cytology”) of cutaneous and subcutaneous lesions (lumps and bumps) can result in a specific diagnosis or perhaps can better characterize a lesion. For nearly all lesions, the cytologic biopsy will not be as definitive as an incisional or excisional biopsy with a histopathological examination; but will be less expensive and yield results quicker.

Please see previous proceeding document for an introduction to the goals and approach of a cytologic biopsy.

5-1 Cytologic biopsy CVC: fine-needle aspirate of vulvar mass
- Case: 026163 (758)
- Dog, mixed breed, female, 5-yr-old

A 1x1 pink mass was protruding slightly from the vulvar mucosa. The owner first noticed the mass yesterday. The mass protruded into the vaginal vault; it might be extending into the submucosa. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.

5-2 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
- Case: 02-7821 (754)
- Dog, boxer, 1-yr-old

A 1x1x1 cm, pink mass was located in the lateral skin of the right shoulder. The owner first noticed the mass a few days ago. The mass seemed to involve the dermis and epidermis and did not extend into the subcutaneous tissues. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.

5-3 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
- Case: 028857 (727)
- Dog, Golden retriever, male (neutered), 5-yr-old

The dog was presented because of a 2x2x1 cm mass located in the lateral thoracic skin. The preparation is a smear of the sample aspirated from the mass.

5-4 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
- Case: 039973 (725)
- Cat, domestic short hair

The preparation is a smear of a sample aspirated from one of several small (< 1 cm) cutaneous masses.

5-5 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
- Case: 56533-98 (742)
- Dog, mixed breed

A smear of serosanguinous to purulent fluid was submitted; the fluid was collected from a subcutaneous swelling that had a draining tract.

5-6 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
- Case: 028729 (775)
- Dog, terrier-mix, female (spayed), 14-year-old

A 5x4x23 cm mass was found in the dorsal thoracic skin. The dog has several other similar masses in its thoracic and abdominal skin. The mass extends above the skin surface, the surface is ulcerated, and appears to involve dermal and possibly subcutaneous tissues. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.

5-7 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
- Case: 029402 (781)
- Cat, Persian, female (spayed), 19-year-old

The cat was presented because of a large (about 8 cm), broad-based mass located in the area of the 3rd to 4th left mammary gland. Physical examination revealed was covered with haired skin and appeared to involve the dermis and subcutaneous tissues. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.
Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass

- Case: 028874 (785)
- Dog, shar pei, male (neutered), 9-year-old

The dog was presented because of a mass in its skin. Physical examination revealed a dermal or subcutaneous mass of the right thorax. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.
A cytologic biopsy (aka, fine needle biopsy or fine needle aspiration biopsy or “cytology”) of cutaneous and subcutaneous lesions (lumps and bumps) can result in a specific diagnosis or perhaps can better characterize a lesion. For nearly all lesions, the cytologic biopsy will not be as definitive as an incisional or excisional biopsy with a histopathological examination; but will be less expensive and yield results quicker.

For some lesions (e.g., lipoma), it takes minimal expertise and diagnostic methods to arrive at a correct diagnosis; but other lesions require extensive knowledge gained through experience and excellent equipment. For those who wish to develop their cytologic biopsy skills, the following should be considered essential.

- Develop techniques to obtain cytologic preparations that have monolayers of cells
- Have a quality cytologic stain that can provide reproducible results; quick stains can be acceptable
- Have a quality microscope that has excellent 40x- or 50x-oil and 100x oil objectives (these objectives might cost $3000 to $5000 each)
- Have excellent textbooks and atlases for the species of interest
- Have knowledge of the types of lesions that can be found and the many variations of each disorder

During the microscopic examination of aspirates, scrapes, imprints, or other cytologic preparations, general goals are to arrive at one of these conclusions or opinions:

- Definitive diagnosis: can be achieved with a few neoplasms and some inflammatory lesions
- Consistent with _______: cells populations are seen in this condition but the findings are not unique to one diagnosis; additional diagnostic efforts are needed to confirm
- Suspicious of _________: findings are suggestive stated diagnoses but definitive evidence is not seen; additional diagnostic efforts are needed
- Not consistent with ______: A preliminary diagnosis had been made; the findings in this sample are not likely to be found in that disorder; or, the findings do not support the preliminary diagnosis

The following flowchart provides a basic guideline for the evaluation of a cytologic preparation. The concepts of the flow chart will be used during the virtual microscopy of several lesions involving the skin and subcutaneous tissues of dogs and cats.
4-1 Cytologic biopsy CVC: Smear of fluid from subcutaneous lesion
   - Case: 176517 (729)
   - Dog, mixed breed, 3-yr-old, female (spayed)
A smear of serosanguineous to purulent fluid was submitted; the fluid was collected from a subcutaneous swelling that had a draining tract.

4-2 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
   - Case: 02-1975 (723)
   - Dog, Labrador retriever, 4-yr-old
A 2x4x3 cm mass was located in the lateral skin of the left hind thigh or hip. The owner first noticed the mass a few weeks ago and it has been getting larger. The mass protruded slightly and felt like it extended into the subcutaneous tissue. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.

4-3 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
   - Case: 002885 (772)
   - Dog, Golden retriever, male, 12-yr-old
The dog was presented because of a mass located on the dorsal aspect of the tail head. Physical examination revealed 2-cm, soft mass in the dermis and was covered with haired skin. A fine-needle aspirate of the rear leg mass was collected and a smear was prepared for examination.

4-4 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
   - Case: 030056 (783)
   - Dog, basset hound, male (neutered), 7-yr-old
The dog was presented because of perianal masses. Physical examination revealed a small perianal mass and possibly enlarged regional lymph node. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.

4-5 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
   - Case: 02-2357 (557)
   - Dog; breed, age, and gender not provided
A smear of an aspirate obtained from a mass in the skin of a foot was submitted for evaluation.

4-6 Cytologic biopsy CVC: fine-needle aspirate of cutaneous mass
   - Case: 024854 (777)
   - Dog, schipperke, male (neutered), 15-yr-old
The dog had been coughing for 2-3 weeks. During a physical exam, a mass was found in the subcutaneous tissues of the left lateral thoracic; it appeared to be firmly attached to underlying tissues. A fine-needle aspirate of the mass was collected and a smear was prepared for examination.

4-7 Cytologic biopsy L&B CVC: Imprint of moist cutaneous lesion
   - Case: 256285 (737)
   - Dog, mixed breed, male (neutered), 4-yr-old
The dog was presented because of a swelling of the left flank that broke open yesterday and yellowish red material oozed out. The preparation is an imprint of the ulcerated area after superficial debris and hair were removed.

4-8 Cytologic biopsy CVC: Imprints of cutaneous mass
   - Case: ASVCP 1988-11 (748)
   - Cat, domestic short hair
The cat was presented because of skin lesions. Physical examination revealed several, pea-size, cutaneous masses. One mass was excised and imprints of the mass were submitted for evaluation.
Many of today’s hematologic analyzers evaluate leukocytes via flow cytometric methods and provide an automated differential count and calculated concentration of leukocyte populations. When properly calibrated for a given species (e.g., dog or cat), then the generated results are typically accurate for blood cells with relative normal leukocytes. Why, the instruments have software programs that are designed to recognize normal cells. When abnormal leukocyte are present (e.g., toxic neutrophils, reactive lymphocytes, neoplastic cells), the automated differential count will not be accurate.

A thorough examination of leukocytes (WBCs) in a blood film can provide valuable information about an animal’s leukocytosis. For those who wish to develop their microscopy skills, the following should be considered essential.

- Develop techniques to make an excellent blood film that has an even distribution of cells and a good “counting window.”
- Have a quality hematologic stain that can provide reproducible results; quick stains can be acceptable
- Have a quality microscope that has excellent 40x- or 50x-oil and 100-x oil objectives (these objectives might cost $3000 to $5000 each)
- Have excellent textbooks and atlases for the species of interest (see list below)
- Have knowledge of abnormal leukocytes or associated organisms that can be found and the many variations of each disorders,

Leukocytes in a stained blood film are White Cell Cadavers – they are cells that died of dehydration (air-dried) and transformed into 2-dimensional shapes that can provide clues to what they were as living cells. Every leukocyte in a blood film is an artifact – the cell did not have that shape or appearance when it was circulating in blood. A key aspect in blood film evaluations is recognizing artifacts that tell us something about the animal versus artifacts that are distractions.

During this session, we will use case information to provide a framework for the evaluation of White Cell Cadavers. After examining pertinent microscopic fields, an audience response system will be used to assess your knowledge. The reasons for the observed leukocytosis and leukocyte abnormalities will be explained.

There are many books that provide images of blood cells and/or explain the significance of abnormal cells. The first listed book (by John Harvey) provides the most comprehensive set of images. The other books can also be valuable.

7. Duncan & Prasse’s Veterinary Laboratory Medicine; Clinical Pathology; 5th ed., KS Latimer, 2011
8. Atlas of Veterinary Hematology; Blood and Bone Marrow of Domestic Animals, JW Harvey, 2001

3-1 WBC CVC: Blood film from a dog

- Case: 256917 (2420)
- Dog, coon hound, male, 4-yr-old

The dog was presented because of lethargy, weight loss, anorexia, and dyspnea of 2 months duration. The referring veterinarian had treated the dog for pneumonia (specifics of treatment not known) Physical examination revealed labored breathing, muffled heart sounds, and distended abdomen.

<table>
<thead>
<tr>
<th>pTP</th>
<th>6.7 g/dL</th>
<th>6.0-7.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hct (s)</td>
<td>23 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>22 %</td>
<td>37-55</td>
</tr>
<tr>
<td>Hgb</td>
<td>7.8 g/dL</td>
<td>12-18</td>
</tr>
<tr>
<td>RBC</td>
<td>3.2 x 10^6/µL</td>
<td>5.5-8.5</td>
</tr>
<tr>
<td>MCV</td>
<td>69 fL</td>
<td>62-76</td>
</tr>
<tr>
<td>MCHC</td>
<td>35 g/dL</td>
<td>32-36</td>
</tr>
<tr>
<td>MCH</td>
<td>24 pg</td>
<td>19-25</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>WBC</th>
<th>24.0 x 10^3/µL</th>
<th>6.0-17.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seg. neut.</td>
<td>56 %</td>
<td>13.4 x 10^3/µL</td>
</tr>
<tr>
<td>Band neut.</td>
<td>16 %</td>
<td>3.8 x 10^3/µL</td>
</tr>
<tr>
<td>Lymph.</td>
<td>18 %</td>
<td>4.3 x 10^3/µL</td>
</tr>
<tr>
<td>Mono.</td>
<td>8 %</td>
<td>2.0 x 10^3/µL</td>
</tr>
<tr>
<td>Eos.</td>
<td>2 %</td>
<td>0.5 x 10^3/µL</td>
</tr>
<tr>
<td>Baso.</td>
<td>0 %</td>
<td>0.0 x 10^3/µL</td>
</tr>
</tbody>
</table>
### 3-2 WBC CVC: Blood film from a dog
- Case: 289557 (347)
- Dog, mixed breed, 8-yr-old, female(s)

The dog was presented because she had not eaten for several days and seemed lethargic. Physical examination revealed no major abnormalities except mild enlargement of mandibular, prescapular, and popliteal lymph nodes.

<table>
<thead>
<tr>
<th>CBC (plasma clear)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
</tr>
<tr>
<td>Hct (s)</td>
</tr>
<tr>
<td>Hct (c)</td>
</tr>
<tr>
<td>Hgb</td>
</tr>
<tr>
<td>RBC</td>
</tr>
<tr>
<td>MCV</td>
</tr>
<tr>
<td>MCH</td>
</tr>
<tr>
<td>MCH</td>
</tr>
<tr>
<td>nRBC</td>
</tr>
</tbody>
</table>

|Platelets clumped| × 10^3/µL|
|------------------|
|3-2 WBC CVC: Blood film from a dog
- Case: 38501 (2319)
- Dog, Irish setter, 4-yr-old, female(s)

The dog was presented with a complaint of weight loss and persistent coughing. Physical examination revealed a nonfebrile, mildly dehydrated dog that had tachypnea and coarse crackles. Partial CBC results are provided.

<table>
<thead>
<tr>
<th>CBC (plasma clear)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
</tr>
<tr>
<td>Hct (s)</td>
</tr>
<tr>
<td>Hct (c)</td>
</tr>
<tr>
<td>Hgb</td>
</tr>
<tr>
<td>RBC</td>
</tr>
<tr>
<td>MCV</td>
</tr>
<tr>
<td>MCH</td>
</tr>
<tr>
<td>MCH</td>
</tr>
<tr>
<td>nRBC</td>
</tr>
</tbody>
</table>

|Platelets clumped| × 10^3/µL|

### 3-4 WBC CVC: Blood film from a dog
- Case: ASVCP 2002-16 (422)
- Dog, mixed breed, female (spayed), 10-yr-old

Dog was presented with the history of chronic progressive rear limb weakness, anorexia, and weight loss. Physical examination revealed depression, severe muscle atrophy of hind limbs, pain elicited with deep palpation of both femurs, and an enlarged right mandibular lymph node.

<table>
<thead>
<tr>
<th>CBC (plasma slight hemolysis)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
</tr>
<tr>
<td>Hct (s)</td>
</tr>
<tr>
<td>Hct (c)</td>
</tr>
<tr>
<td>Hgb</td>
</tr>
<tr>
<td>RBC</td>
</tr>
<tr>
<td>MCV</td>
</tr>
<tr>
<td>MCH</td>
</tr>
<tr>
<td>MCH</td>
</tr>
<tr>
<td>nRBC</td>
</tr>
</tbody>
</table>

|Platelets clumped| × 10^3/µL|

### 3-5 WBC CVC: Blood film from a dog
- Case: ASVCP 2006-4 (1613)
- Dog, Basset hound, M(c), 4-yr-old

The dog was presented after a week of inappetence. Physical examination revealed a nonfebrile, depressed, lethargic dog with pale mucous membranes and tachycardia. CBC results for blood sample collected after 2 days of IV fluids and prednisone.
Dog was presented for the excision of a small dermal mass. The presurgical CBC results were basically within reference intervals except for microscopic findings.

### CBC (plasma clear)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Reference Range</th>
<th>Value</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pTP</td>
<td>5.5 g/dL</td>
<td>3.7-7.2 g/dL</td>
<td>15.0 x 10^3/μL</td>
<td>6.0-17.0 x 10^3/μL</td>
</tr>
<tr>
<td>Hct (s)</td>
<td>18 %</td>
<td>36-54 %</td>
<td>14 %</td>
<td>3.0-10.4 x 10^3/μL</td>
</tr>
<tr>
<td>Hct (c)</td>
<td>19 %</td>
<td>36-54 %</td>
<td>4 %</td>
<td>0.0-0.1 L/L</td>
</tr>
<tr>
<td>Hgb</td>
<td>6.6 g/dL</td>
<td>11.9-18.4 g/dL</td>
<td>78 %</td>
<td>0.0-0.8 x 10^3/μL</td>
</tr>
<tr>
<td>RBC</td>
<td>2.7 x 10^6/μL</td>
<td>4.9-8.2 x 10^6/μL</td>
<td>3.0-11.5 x 10^6/μL</td>
<td></td>
</tr>
<tr>
<td>MCV</td>
<td>71 fL</td>
<td>64-71 fL</td>
<td>0 %</td>
<td>0.0-0.3 x 10^3/μL</td>
</tr>
<tr>
<td>MCHC</td>
<td>34.5 g/dL</td>
<td>32.9-35.2 g/dL</td>
<td>0 %</td>
<td>0.0-0.1 x 10^3/μL</td>
</tr>
<tr>
<td>MCH</td>
<td>24 pg</td>
<td>21-26 pg</td>
<td>0 %</td>
<td>0.0-0.1 x 10^3/μL</td>
</tr>
<tr>
<td>nRBC</td>
<td>0 /100 WBC</td>
<td>0-1 /100 WBC</td>
<td>0-1</td>
<td>0-1 x 10^3/μL</td>
</tr>
<tr>
<td>Platelets</td>
<td>131 x 10^3/μL</td>
<td>106-424 x 10^3/μL</td>
<td>0-1</td>
<td>0-1 x 10^3/μL</td>
</tr>
</tbody>
</table>
The old adage that it’s better to prevent problems that fix them is true -- especially when it comes to providing Fear Free veterinary visits. Think of a veterinary visit for a pet like a four-legged stick of dynamite with a short or long fuse, that gets lit or not, and burns fast or slow. If the visit is managed correctly, we can have a fuse, wetted with moist treats like EZ Cheese or Kong Stuffing that never gets lit. In contrast, if a pet’s fear, anxiety, and stress (FAS) fuse burns all the way, you can have a full-blown explosion that results in pets biting, clawing, taking flight, freezing, or going into what’s known as collapsing immobility.

Here are the top ten things you need to do to avoid the fast burn of the FAS fuse and the ensuing detonation

**Not cleaning the outside of the practice**

If a stressed dog defecates on the hospital grounds and other canines smell it, it’s like you replaced your practice signage with flashing warning signs and sirens with a message of “DANGER…DANGER…EVACUATE!” Likewise, most nervous dogs have nervous bladders and make it a point of leaving a message on a vertical surface outside the practice. This is not “happy pee pee” like when a dog’s on a walk by the lake, but “FAS pee pee,” which serves as a warning to others to “NOT ENTER!” The solution to the above FAS fuse-burners is to clean up feces and clean the most used urination marking spots up to every hour.

**Making your front door an entrance to the Dungeon of Horrors**

As a member of the veterinary healthcare team, have you ever tried bringing one of your own terrified pets through those double doors and into reception? What a hellish way to start the visit. If possible, help owners enter calmly with their pets. Take concrete steps to make the reception experience more “spa” and less like a bad “shelter.” Steps must be taken to dramatically reduce the amount of ambient noise (door chimes, phones ringing, loud voices, intercom use, etc.) and malodors (bleach, harsh cleaners, fear pheromones, scented candles, perfume).

**Always booking the next available appointment**

Most receptionists automatically try and book a pet owner into the next available opening in the schedule with a time the pet owner can make it. Look, we’re not Jiffy Lube booking a lube, oil, and filter change. Each pet and pet owner are unique and must be accommodated as such. Many successful Fear Free practices have special hours weekly or biweekly just for the most emotionally sensitive pets, and/or felines. During these hours, the kennels are not being serviced (decreased noise), the clippers are quiet, exuberant pets are not being seen, and the practice is more in the “sanctuary mode” and less in the “game day” one.

**Not willing to make special accommodations**

Besides polling the pet owner online or on the phone about what known FAS triggers their pet has, the receptionist needs to ask upon client check-in, “What can we do today to make Sparky and your experience here a better one?” After the pet owner processes this, smiles and nods, you’ll get answers like, “Sparky made a little mess in the car, that I could use help cleaning up,” or “Can I have a towel to put over the carrier?” or “Could I have a little water for Sparky?”

**Not setting up the exam room like a s-paw!**

In the bad old days, the exam table was more autopsy slab than spa table. The fear-inducing table was cold, slippery, and elevated. The room was decorated with people-pleasing colors and objects, illuminated with harsh fluorescent lights, with the doors swinging frequently to fetch forgotten items. In Fear Free, the room is painted in pet pleasing pastel colors, the exam room table has a soft, warm, non-skid surface that is free from pictures and objects depicting animals, and has pheromones wafting through the air and soft lighting.

**Being a Whirling Dervish in the exam room**

I used to come rushing into the exam room like a first-time cast member in a Broadway musical rushing the stage. My mentor taught me to show visible, audible, palpable enthusiasm. My old fear-inducing display included a raised voice, hearty hand shake, direct eye contact of both pet and person (what better way to show interest, I thought) and an immediate thrust to make contact/friends with the pet. Looking back, what a “hot mess of FAS” I was creating! Now I open the door softly, speak softly, and carry a big stick of Pupperoni. I still use a warm, engaging voice and make appropriate eye contact with the owner, but I avoid prolonged eye contact with the pet. Instead, I might glance at the pet and say hi, but then let my hands do the talking as I toss a tasty tidbit on the floor or table, then another, until the pet obviously wants contact with me.

**Using the same old harsh chemical cleaners**

You know what you need to do with bleach? Stop buying it to clean anything in your hospital. One whiff of bleach and many other harsh chemicals can make a pet go instantly nose-blind for seven days. They will still be seen sniffing, but these chemicals have destroyed their olfactory neurons and they won’t regenerate for a full week. So pets, who rely so much on smell for cues, will now be nose-blind – a huge source of FAS. You need to switch to the same cleaners that three-quarters of human dentists and over half of human hospitals use to eliminate chemical and pheromonal pollution. Start cleaning with accelerated hydrogen peroxide, which goes
under the trade name Rescue in the veterinary channel. This amazing product is a killing machine – 99.9 percent kill of pathogens including parvo in 15 seconds of contact time – plus it breaks down into water and oxygen.

**Letting your basic diagnostic tools be like pet torture devices**

At one veterinary lecture I asked the audience of vets and veterinary nurses, “In the absence of an infectious disease, how many of you clean your stethoscope less than once a month?” 80 percent of the audience members’ hands went up. One veterinarian left his hand up and said, “I haven’t cleaned my stethoscope for 37 years. That’s the year I bought it after graduation from veterinary school.” While we should clean our stethoscope more often for infectious diseases, what I’m even more worried about is the buildup of fear pheromones on the stethoscope, otoscope head (not cone) and accompanying battery pack, as well as tourniquets, nail trimmers, etc. After every usage, these basic tools-of-the-trade should be cleaned with Rescue wipes, and then wiped down with species-specific pheromone wipes (Adaptil for dogs and Feliway for cats).

**Consider tasty treats an unnecessary expense**

I lectured on “Why Fear Free is Better Medicine” in a room that held 1500 people at the world’s largest veterinary conference. Before the first person entered the room for an all-day session, I took about $20 worth of change in quarters, dimes, nickels, and pennies and scattered them like lawn seed around the room, in the back, front, aisles, and in the seats. Near the end of the day I asked people to look around and see if they saw any loose change on the floor. I told them if they held it up with their fingers, I’d give away prizes. The huge crowd’s eyes and hands went to the floor in a big kerfuffle. About 40 people found coins; 38 of the 40 were pennies, with one dime and one nickel to round out the treasure hunt. Where was the other almost $18.00 I’d scattered? In people’s pockets, probably starting before the first lecture started that day. So what’s the point here? Treats are like currency in the dog and cat world. If you’re giving low value treats with a pet-assigned value of one penny, maybe it’s not worth picking up off the floor/table. But if it’s something tasty, like a piece of turkey hotdog or Braunschweiger sausage, well that’s a dime in dog dollars and worth licking up. Liverwurst paste, Easy Cheese Cheddar & Bacon, or Kong Squeezable Peanut Butter, now these are worth 25-50 cents. Think of Fear Free like gambling for pets. The treat payoffs don’t have to be huge all the time, but big enough in texture, flavor, and quantity to keep them calm and happy during the current visit, and wanting to come back another time.

**Having the treatment area resemble a UFC ring**

Every hospital I’ve owned, and the vast majority I’ve been in, have had basically the same treatment area layout with 1-2 treatment tubs, treatment tables, pharmacy, lab, counters, and a bank of cages to file pets who need procedures. This design is guaranteed to create FAS! Why? Simple. Just think if this were a human hospital ER department filled with human cages next to the treatment bed/gurney/table. You sit there with a bird’s eye view, wide-eyed and gape-mouthed, watching someone struggling as his infected wound is being cleaned, listening to a baby screaming in pain from an injection, with the smell of fear in the air. Would you be calm and happy, resting comfortably before your turn to be seen? Oh, “hi” no! We need to either give pets in treatment some privacy and other FAS-busting gifts such as calming music, pheromones, privacy barriers for cages, or house them in something that could be considered “emotional isolation.”
Moses was given the “Ten Commandments,” not the “Ten Suggestions.” In a funny way, it’s the same for Fear Free. We were long on passion and short on proven fear-anxiety-and-stress-busting procedures when we started in 2009. Now with eight years of work by almost 200 experts on the Fear Free Advisory Group, we know what works and what steps must be done with each and every pet to prevent or relieve fear, anxiety, and stress (FAS).

Before we start taking those 10 steps, let’s do some important review

What causes fear?

Fear is caused by something painful and/or disturbing. When a pet has a painful skin, dental, or musculoskeletal problem, she is naturally fearful about those traumatized areas being handled. Or when you bring out the nail trimmers in front of a pet who’s been quicked, or an otoscope in front of a dog who has memories of many painful procedures with that instrument, he can blow up with fear.

Target audience

What percentage of pets who come into veterinary practices have fear? What percentage are in pain? How many are disturbed or in distress? I think we’d agree it’s the vast majority of pets. And that means we have to recognize the problem and work hard to provide solutions that will reduce or remove FAS triggers for each pet (some will be unique to a pet), or alleviate them if they flare.

One of the basic tenets of medical bioethics known throughout the world is the Hippocratic Oath. There are many variations of this oath, each changed and redesigned over the centuries to meet realistic demands of modern day medicine. The original concept, however, remains the same: Whenever possible, healthcare providers must “First, Do No Harm.”

What if you became aware that fear, anxiety, and stress were largely unrecognized, and often untreated? What if this not only affected a pet’s emotional well-being but her physical well-being? Ignoring these points likely causes repeated, serious damage to the very pets we treat. It would be a bitter pill for the public and our professional colleagues to swallow, knowing that their local veterinary practices were not embracing every tool at their disposal to help the pets under their care live happy, healthy, full lives.

What exactly do we mean by “happy, healthy, full lives”? The vast majority of practices have got the healthy part down pat. We already fully embrace our role as “true pet health experts.” We try through complete physical exams to look past obvious problems to potentially more serious ones, to prevent health problems through wellness programs, and to treat and manage accidents and illnesses while staying current on the latest equipment, products, and procedures. We now recognize the value of specialists, who can provide more sophisticated and specialized medical care for patients we refer to them.

But happy lives? Until very recently, we not only didn’t see this as our responsibility (or opportunity, as we’ll see later), we didn’t even recognize the signs of poor emotional health or the symptoms of FAS. We certainly didn’t think of behavior as part of the medical services we could provide for our patients. All too often, behavior was considered more a means to an end, usually relegated to training of house pets.

So let’s hit this nail directly and powerfully on the head right now, driving home the fact that since behavior produces a physiologic response, it is very much an integral part of today’s modern medicine.

Ultimately, Fear Free veterinary visits, where we work to reduce FAS and increase a sense of calm and happiness in our patients, is better medicine. Better for the pet, the owner and the veterinarian.

Not out of ignorance, but out of a lack of understanding of how important behavioral characteristics are to pet owners and the pets themselves, most veterinarians are probably not fully familiar with what it means for a pet to live a “full life.” It’s not nebulous, but a recognition that education, training, and enrichment activities are as important to our pets as they are for our children, grandchildren, and ourselves. Pets and people benefit from education, training, exercise (mind and body), as well as appropriate challenges.

Fear Free veterinary visits started out as just the right thing to do. After all, nobody gets into veterinary medicine to make life worse for animals -- in fact, the polar opposite, as we hear every day from young veterinary trainees and graduates. It certainly isn’t the income they anticipate earning that brings them into our profession!

We work with competence, confidence, and compassion to optimize health. And that’s why we are so excited to harness the power of Fear Free, which not only helps keep pets healthy but keeps them coming into the veterinary hospital regularly. It is a powerful adjunct tool to provide even better medicine. In many cases, much better medicine. How’s that for a good reason to see this added to our armamentarium of things we can do to improve on how we practice every day?

Is this a panacea for every animal? No, it is not, but it certainly will enhance the lives of the majority of the pets we are given the privilege of caring for every day.
Here are the top ten things to do to provide true Fear Free visits

Begin with the end in mind
We anticipate the pet and pet owner’s visit whenever possible. (Obviously, this can’t be done with emergency cases or unexpected walk-ins.) We competently, confidently, and compassionately set out to give the pet the gift of a Fear Free visit. Using proven products and protocols, we know what steps must be taken to reduce or remove FAS triggers, or alleviate them.

Pet owner as partner
We can’t have the pet owner deliver a wild-cat or mad-dog at the reception desk and expect us to waive a magic wand of a turkey-drumstick and make all of the FAS go away. FAS for each pet must be thought of as a fuse. At every step of the visit, the more of the fuse that gets burned up, the less reserve/time you have before the pet blows. The pet owner’s responsibilities include preparing for planned veterinary visits about a week out, starting a magic carpet ride of pheromones from carrier to car to clinic, preheating or precooling the vehicle, covering the carrier with a light towel, and not baby-talking the pet on the way to the hospital.

Know the FAS trap doors
Each pet and pet owner are different in terms of their history of veterinary visits, previous episodes of FAS, current medical and emotional health, and entering medical conditions as well as chronic ones. You must use a combination of online and personal checklists to find out, for each pet/pet owner, what are the known FAS triggers. Is it waiting with other pets/pet owners, having their weight taken, being placed on the exam table, having their temperature taken, etc.? Once you know what the triggers are, you either postpone (take the weight only if you’re going to have to sedate or prescribe meds) or don’t do them (exam the pet on a yoga mat on the floor).

Consider sedation a gift
Fear Free has three mantras: 1) Think of sedation as a first option, not a last resort; 2) Sedate early and often; 3) If you can’t abate (FAS), you must medicate (sedate). For far too long in far too many pets (the vast majority, in fact), we’ve only used sedation when a) pile-o’-techs restraint didn’t work, or b) somebody was going to get hurt. We have this “bass-akwards.” We either use sedation proactively to prevent a pet from going into full-blown FAS, or we deploy it at the first signs of a blowup.

Tiny or no waiting room in the practice
The ideal practice has enough exam rooms (and the technology) to be able to have pet owners walk into the practice with their pet and right into an exam room. The pet/pet owner are both checked in and checked out in the exam room. For most practices, this isn’t practical yet. The best alternative is to have the pet owner go into the practice upon arrival (without the pet) and then go back out to wait in their vehicle with their pet (the new waiting room), until it’s their turn to enter the spa room (formerly the exam room). NOTE: Have pet owners bring two sets of keys so they can lock the pet in the car with the heat or AC on for the few moments they need to check in.

Fear Free starts in reception
Have the receptionist give dog owners pheromone-impregnated bandannas for dogs, pheromone-spritzed towels to put over the carriers of small dogs and cats (or for larger dogs to lie on), or a cotton ball wetted with pheromones to toss into the cat carrier. Also, start the conveyor belt of treats with a goal of giving the pet up to 60 treats in a 15-minute exam (this is the target number at the CVM at OSU).

Top-shelf treats
I still remember the bad old days when we offered dogs a PetTab as a treat. That was about as popular with dogs as kids getting a new toothbrush and mini-tube of toothpaste at the dentist. You have to have a wide variety of soft chewable (like freeze dried liver), moist chewable (pieces of turkey hotdogs), and moist and lick-able (EZ Cheese or peanut butter) treats. Their palatability can be amped up by heating in a microwave (most treats, but not EZ Cheese or other products in a can).

Have species-specific exam rooms
While you can get by with using your exam rooms for seeing all pets, you’re going to providing much better service by trying as hard as possible to have separate exam rooms for dogs and cats. For each room, try to create an exam room disguised as a spa with species specific pheromones, music, treats, tools (like Zoom Groom for dogs and cats) examination areas (like a plastic cat tree or shelves for cats), and other accommodations such as litter boxes for cats.

Dress for unstressed success
In the bad old days of practice (bad for pets), I would wear a white lab coat with a stethoscope around my neck. The lab coat didn’t get laundered until a) hydrogen peroxide couldn’t clean the stains, or b) it got really dirty. My stethoscope? It hung on a hook in the treatment area and never got cleaned unless we’d had an infectious disease case in the practice (like distemper or parvo). This was a perfect storm of FAS for the pet. First of all, white is about the worst color you can have for pets. Secondly, the smells that get on our clothes (especially our scrubs or smocks) can contain fear pheromones that can instantly trigger FAS in a new pet. For example, you have one pet that expresses his anal glands during an exam or procedure; there’s a particular fear pheromone that’s secreted in the anal glands, and now when you go into another exam room and see another dog, say for vaccinations, the dog gets one whiff of your clothes and bam, instant “fight or flight” mode kicks in.
Work as a team
You can’t have the receptionist allow a terrified cat to wait next to an exuberant dog, or drag a reluctant dog onto the scale, and expect things to go well in the exam room. Likewise, the veterinary nurse can’t do a wonderful job keeping FAS in check, only to have the veterinarian burst into the room like the start of a vaudeville act. The entire veterinary healthcare team must be using the same FF playbook, tracking results, knowing the score (where the pet is on the FAS scale), fixing failures, and celebrating success. The team must have a goal of continual self-improvement.
Keeping pets calm and Fear Free begins with preparing the pet early and working in partnership with the human client to keep the experience as relaxing and calm as possible. Then, once in veterinary care it’s ideal to readily adjust care according to the pet’s preference, body language and in accordance with what works best to keep the entire human team safe and calm as well.

Calming aids for both the home, car and hospital include using calming music that’s species specific or classical. Audiobooks have shown some calming effect as well. Additionally, using pheromones, lavender and chamomile scents may help pets. The animal’s regular bedding and toys can provide comfort in the hospital as well as their familiar scents go with them during their care and potential stay. Food puzzle toys are one option to keep canines and cats busy both prior to and during care. Have the pets come in hungry and have the pet owner supply the pet’s favorite treats and toys.

In many cases pets will have certain sensitive areas where care isn’t as easily delivered without causing stress. In such cases techniques like distraction and changing the pet’s association with the handling may be beneficial. Additionally, consider teaching the pet a ‘predictor cue’ like ‘ears’ or ‘paw’ to communicate what’s happening once the experience has been turned into a positive for the animal.

Training can be done in the hospital, in conjunction with other partnerships, including relationships with referrals like a veterinary behaviorist. Working in partnership with reward based trainers is another essential way to provide the training guidance pets need. In training it’s essential to stay away from force and punishment based tactics and lean towards reward based training instead. Punishment and force has numerous fallouts, including the potential risk for increased aggression and stress.

Pay attention to cues the pet owner gives on what their pet finds calming and rewarding. Additionally watch the pet’s reaction to different elements of the interaction and care. In some cases dogs or cats may do better with the veterinary team only, but in the majority of cases having the pet owner present is essential for the pet’s wellbeing as their ‘safe’ person stays with them. Giving both the pet owner and pet time to settle in is extremely helpful on starting off care with the right paw forward. Additionally, consider changing where the exam or care is done, especially if a pet has a prior negative experience in the area.

Distractions are an important part of maintaining a calmer reaction. Minimizing the ‘freak out’ cue at the sight of needles, the approach of staff and handling can also be done with handling tips- such as examining from the place of the pet’s preference, disguising an exam into a petting session, and encouraging the animal to willingly move to places like the scale by their own choice. Distractions in the form of interesting sights, tantalizing treats and other intriguing events can also minimize the pet’s focus on the situation at hand.

Consider the use of muzzle training for dogs as well as conditioning to tools like like towels. There are also ways to minimize the stress of muzzling or handling a pet when it’s necessary to do a procedure immediately without the ability to condition to the equipment in a slower fashion. Pharmaceutical intervention is also imperative and important to communicate with throughout the visit as well as with the pet owner before the visit itself as the prepare to bring the pet in for care.

There are ways to increase cooperation with care by also conditioning the pet to associate handling and care with positives the pet enjoys. The pet can also be encouraged to willingly participate in their care and stress minimized by using communication cues in handling to minimize the surprise to the pet. In the future and immediately in exam, consider utilizing cues the pet already knows or build cooperative behaviors to increase willing participation and lessen the struggle.
Help the Owner, Help the Pet!
How to Set a Fear Free Foundation at Home
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Setting a Fear Free Foundation starts at home with the pet owner. By adequately preparing the pet owner to bring their pet in less stressed when they arrive for the visit, the pet’s more likely to be receptive to care and calmer. In addition, it’s important to guide the pet owner in strategies to ease the stress their pet experiences during care and handling; both for home care and future veterinary visits. Strategies to help the owner are possible both in preparation protocols for the visit, help offered during the visit and resources, classes and individual instruction to ready pet and owner for care given in the home and later veterinary care.

The pet is affected by their pet owner’s guidance and intervention before they even step a paw into the hospital. For this reason, training and communicating with the pet owner on how to bring the pet in less stressed is essential and necessary. Talking with the pet owner prior to the visit, offering educational materials and seminars, offering workshops and sessions for fun visits and Victory Visits, guiding the pet owner during the consult and offering ongoing behavioral help are all potential ways to keep in communication.

When a pet arrives at the hospital in a stressed state, calming that pet down becomes far more challenging. In comparison, if the pet arrives calmer, the pet’s likely to stay more relaxed throughout their entire visit and are more likely to be receptive to positives of care (e.g. treats). For this reason, it’s important to prepare the pet owner with strategies they can take to make the visit less threatening. Common anxiety triggers for pets include the crate, the car or even certain words used. Instead, preparation can begin early by conditioning the pet to travel and containment. In addition, the pet owner can employ ‘Victory Visits’ or structured fun visits to reduce the stress when actually undergoing care. Pets can be helped through creature comforts used in crate and car as well as through conditioning to these experiences and pairing with positives.

Part of the communication with the pet owner is teaching them to read body language and signs of stress in their pet. Rather than forcing their way through the fear, teaching them sensitivity and providing gentle guidance on how to handle stress is important. Creating an open pathway of communication with the client and veterinary team also helps on adjusting as necessary, including discussing the potential of pre-visit medications, supplements, changes to the exam or procedure being done and pre-visit preparation of accustoming the pet to aspects of care they’re upset and uncomfortable with.

Another factor to address is adding awareness of the pet owner as to their own behavior that either stresses or calms their pet down. Pets react to their owner’s stress levels with owners unaware of how their own actions influence their pet. The emotional tone, words used, facial expressions and movement of the pet owner are likely to be assessed and reacted to by the pet. Certain cues, like ‘vet’ or ‘it’s okay, it’s okay’ type of reassurance can actually stress out the pet more rather than calm, especially if they’re associated with past care indeed not being okay. Hovering, physically blocking or tensing up and other body language also are likely to be reacted towards. In some cases when the pet owner wants their pet to behave at the vet the tendency may be to force compliance for a sit or down. In each of these cases it’s important to guide the pet owner in a manner where they’re given direction on things they can do to keep their pet calm.

Practicing proper breathing techniques, posture and improving aspects of the environment are all strategies to help keep people calm as well as the pet. Aspects like lighting, colors, sound and communication signs in the hospital set the mood for the visit; from the waiting room to the exam room. In addition, communicating calm, encouraging relaxed behavior through reinforcement, and changing the subject or focus can also help.

The car ride is one such place where pet owners can be assisted in helping their pet. For pets who are upset with car rides, getting the pet equipped in a protective harness, carrier or crate can help by minimizing driver distraction and increasing safety for pet and person. Teaching the pet to relax with their equipment prior to car visits is also essential. Pairing the car, equipment and ride with positive and enjoyable consequences boosts the pet’s enjoyment of the situation and helps decrease the potential for problems. Dogs benefit from training like ‘go to a mat’ that’s placed inside of their designated area for car rides. In addition, pets benefit from learning to ‘target’ both into and out of the car and to use ramps when these tools are helpful for the given situation. Breaking these into small components to accustom the animal pays off with big results longterm.

Cats also need training to become accustomed to their crate area. Training the feline to walk into their own crate or carrier space and turning it into ‘purr-niture’ decreases the freak-out factor many felines experience when they see the crate that’s more of a torture device than a place of safety. Regular use and increasing the comforts of the crate increases use and makes the space more comfortable for the cat when it’s needed. Felines can be trained to go inside and also guiding in with coaxers like feeding meals beside the crate, dropping treats inside, supplying comfortable bedding inside and encouraging use with treats, toys and petting while inside. Placing the crate in easy to access places also encourages use. Some felines may need the components of the crate broken down into easier to handle steps, such as having the top or door off to begin with or eating treats near the outside edge of the carrier.
If the pet owner has trained their dog or cat a prior behavior of going to their mat—this is extremely useful for helping during veterinary visits. The mat or bed space may be placed on areas like the scale or exam area for care and transition with the pet throughout the clinic. Teaching a ‘touch’ to a hand or target is another helpful way to get a willing approach from the pet, redirect their focus and get willing movement from the animal towards or away from a certain space in the hospital. Training a pet to ‘rest’ and settle for needed care allows for a two way communication for both the pet and the person on ascertaining the pet’s comfort being provided and turning it into a situation where the animal is voluntarily cooperating during care with less stress and fear.

The wait in the waiting room is another stressful situation for pet and pet owner. During the wait stress levels can escalate. To reduce the stress of such a wait, consider the environment where the pet and their owner are asked to wait. In addition, consider other strategies to teach the pet owner on keeping their pet focused and productive with positive distractions or by reducing the ‘scary’ factors around the pet.

During the vet visit it’s often helpful to keep the pet owner near the pet as many times pets are calmer when kept near their owner than when they’re taken into a different area. Employ the pet owner’s help for distractions, such as petting, during the exam. In addition, communicate with the pet owner during care for what you’re looking for in the pet and adjustments you’re making to keep the pet calm. Such information can help the pet owner for ensuring their pet stays comfortable with care at home.

It’s important to communicate the why of what you’re doing during care as well as prep the owner on how to maintain proper care at home. Ear cleanings, medications, nail trims, ear drops and diabetic care are such situations where prepping the pet and pet owner can increase the chance of successfully getting needed care done at home. Additionally, communicating and teaching the way to do such care when necessary can help prevent the pet from escalating in fear, aggression and struggle that may otherwise occur. Ensuring the owner feels confident in what they’re asked to do—from getting their cat in the carrier to dog on the harness, to riding in the car, to walking into the hospital, to waiting for the exam, to leaving their pet with the veterinary team to pickup or leaving and going back home with the prescribed and designated care makes a major difference in the success of person, pet and ultimately the hospital.

Strategies to teach the pet owner for taking their pet home includes distractions, desensitization, counter conditioning, adding in communication cues and training cooperative behaviors where appropriate. There are a variety of ways to teach these to the pet owner—with possibilities varying from in the visit to group classes or individual work. In addition, it’s also helpful to coordinate with the pet owner on where their pet receives other care. If the groomer for instance uses forced nail trims, the Fear Free visit can be impaired from other negative experiences. As such, education and training in better methods ultimately helps the pet in other areas of their life as well as during veterinary care.

Ideally, aim to teach puppies and kittens to remain calm with handling from the start to prevent an escalation to fear and struggle that may otherwise occur without ongoing monitoring and preventive work. Puppy classes are an important way to accustom dogs from an early age to enjoy aspects of the hospital, other people outside of the family and to learn important dog communication and play behaviors. During puppyhood teaching the puppy to remain calm with handling and care, including mock veterinary care, is an important way to decrease the potential for problems later on. Kittens are also in need of proper help when young, including guidance on making handling positive and non-stressful. Guidance for the pet owner and pet can be extremely protective throughout the animal’s life.

For cat guardians the preparation may be in the form of helping them setup the proper space in their home. Another potential option for pet owners includes kitten classes that are a potential addition and resource for the community. Victory Visit and fun visits are helpful ways to prep people for the care their pet receives in the hospital. In addition, more advanced classes and workshops may be held in the hospital to help pets and their people both through potential challenges and in continuing to further their bond and communication. Hospitals with limitations for classes have other options for providing preventive care and addressing issues including the potential for scheduling in appointments with a technician or trainer, setting up open times for positive visits and partnering with others who will work on aspects of care the pet would benefit from, such as puppy classes that works on preventive handling and conditioning to a muzzle.

Educating owners on what to expect and providing the proper resources like food puzzles, exercise and mental challenge can decrease problem behavior and increase behavior that’s more compatible for life with humans. Teaching animals the proper way to play and inviting the animal to play with correct toys and engage in exploratory activity in the right outlets is also protective for the pet and the home.
The physical examination
The physical examination is second only to the history when evaluating an animal with any type of medical complaint or even in the well pet examination. Often we are limited by the inability of the pet to identify signs of disease. Symptoms are not identified in veterinary medicine as they are in human medicine because symptoms are facts related to what the patient experiences and since we do not verbally communicate effectively with our patients we do not have an opportunity to hear about feelings. Thus we are limited to what the client or caretaker can tell us about the pet’s problems, signs which may or may not be interpreted correctly. Since signs are related to what we see, we are dependent upon the animal’s presenter (should there be one) to identify what it is that he or she sees and describes to us.

State of the art and state of the heart
A cute introduction to the use of fear free medicine during the basic evaluation of the patient is very real when we recognize that the less frightened the animal is during the exam the happier the client is with us. While we often identify problems based on the story that the owner or caretaker present to us, most of our evaluation and tentative diagnosis is based more on what we find during the examination of the animal. This includes not only the heart but also every organ and body system we examine.

Beginning with the introduction of the animal to the veterinarian and the examination room it often is best to enter the room and briefly exchange pleasantries with the owner. Making the owner feel comfortable in the room and expressing some information that allows for communication between the doctor and the caregiver usually provides some level of relaxation and a less fearful introduction. Often the client is so involved with the pet’s problem or so worried with the seriousness of the problem that he or she will move quickly to an unrelated or insignificant detail to avoid discussing the more acute problem. Other times, the discussion moves directly to the pet along with information that may or may not be relevant to the history and present condition. The veterinarian can usually redirect this problem or identify the need to break the story down into several parts so that each may be looked at independently and evaluated as a whole once the entire animal has been “vetted”.

One objective in this situation is to realize that the client is relating a story but the patient is somewhere in the examination room, either very relaxed or more likely (especially if it is a cat) in a state of worry. Both dogs and cats are highly sensitive to smells and abnormal odors. Both produce pheromones that identify previous visitors to the room and the presence of scents may be frightening to the pet.

Too, the dog or cat may be familiar with the white coat, stethoscope and other veterinary tools that we use. This alone can be enough to frighten the animal. In my case I do not wear special coats or identifying clothing that sets me apart from the owner or those around us. Nevertheless, there is always the scent of the prior animal examined by me or having been in the examination room. Caution is highly suggested relating to discharges and materials from previous pets that are not removed from the room prior to a new pet being placed there. Sounds, noises and such are also enough to engage the pet and heighten its sense of anxiety. There are those who do believe that in addition to the use of pheromones and holistic solutions to calm the pet (“rescue remedy”), that music provides some relief to the pet. A recent BBC study suggested that dogs like some music and are particularly intrigued or soothed by reggae music followed in order by light rock. Personally heavy rock and rap are irritating to me and perhaps music that calms the examining doctor helps to also give off a lighter sensation to the client as well, thus transferring confidence to the dog or cat. Loud music or that which annoys the client or the pet does not make for FEAR FREE evaluations.

In the case of most cats presented for examination I have strong feelings about the use of different types of carrying cases that are used and how they are managed. Cats brought in with only a blanket are the most likely to be frightened and to steal free of the enclosure (light as it may be). While most cats prefer to sit on the owner’s lap or at least be in touch with them, others prefer their carrying case, sitting in a sink, trash can or any other place that provides them with a fairly tight enclosure. One excellent DVM I am familiar with uses a wicker basket to examine his feline patients and feels he gets to do a far more thorough examination that way. These are all important factors relating to the examination of the heart because listening to the heart and lungs in a cat can be difficult to do.

Often it is necessary to ask the client to remain quiet while the thorax is being examined so that the person auscultating the thorax can more definitively hear the sounds being produced. This is particularly true in an obese patient where the sounds are muffled or in one that has fluid or masses in the chest. While not a problem with the dog, cat purring can significantly interfere with hearing the heart and lung sounds. A number of good techniques for this include gently holding the fingers over the nares, turning on a water faucet (and the cat will stop purring) or gently massaging the laryngeal region. Dogs that are overweight or brachycephalic are often difficult to auscultate because the noises from the oropharyngeal region are so loud that they obscure any other sounds one is trying to
These animals do not stop their noisy breathing as easily and it is important to ask the owner and any children in the room to temporarily be quiet so the examining DVM can attempt to limit sounds and differ them from abnormal cardiac or lung sounds. Likewise with patients presenting with severe respiratory distress and potential laryngeal paralysis auscultation of the thorax is more difficult.

Clearing the lung sounds allows swallowing and digestive sounds to be the next step in the examination. Sometimes providing a small amount of food for the dog will demonstrate swallowing or chewing difficulties. Examination of the oral cavity can be relatively simple or very difficult based on the temperament of the pet and any pain or disease involving that area exacerbates the problem. In cats, wrapping the torso in a blanket sometimes permits a more thorough examination without the cat squirming and pulling away preventing a good examination. It may be necessary to recommend sedation using medications safe for that pet which may not be identified until specialized films and routine blood tests have been completed. None of these techniques are considered state of the heart but rather learned exercises that the DVM develops over time while practicing medicine. Regardless a good rapport between owner and DVM is important to facilitate such examinations and the ability to make a diagnosis.

Having worked in a number of practices including a rehabilitation group, I find that the heart rate is often relative to the state of the animal’s anxiety. In the rehabilitation clinic where dogs are massaged, allowed to swim and in general seem to enjoy their veterinary experience, their resting heart rate is lower then that in the veterinary hospital on a stainless steel cold table. Methods of reducing this feature will be discussed, utilizing some rather easy and inexpensive methods that can also be utilized at home, in situations where the animal needs to be sure footed and comfortable. Walking on a carpeted floor or one with yoga mats provides the dog or cat with a better level of stability and less likelihood of slipping or falling. Animals presented with skeletal abnormalities are often better examined on mats than on floors likely to be slippery or wet. Likewise when the time comes to test neurological reflexes or palpation of the abdomen the same can be said regarding the advantage of non-slippery surfaces. Rectal palpation, examination, cleaning the anal sacs and placing urinary catheters in the awake patient are similarly more favorable when done on a non-slippery surface. Each of these procedures are seen by the client and when the pet struggles this is perceived by the client as unpleasant and perhaps as painful, whereas the same situation done with a sure footed pet looks far better to them and less anxiety provoking. Often if the dog or cat begins to squirm so does the client and then the client begins to express anxiety that is quickly transferred to the pet. Considering techniques that reduce anxiety all around are techniques one should learn and incorporate into practice thus making it more of a continuum of FEAR FREE.

I have often encountered difficult dogs and cats that growl, snarl or snap on my entry into the examination room. Many times the owner will comment about the pet being a biter but the use of appropriate muzzles (basket type are often preferred) calms the dog down so that the examination may be easy and without difficulty. Often the clients comment on how easy it was to work that way and to suggest that in the future the same approach is used. Throughout the examination and later if additional studies need to be done it is important to incorporate these methods. We rarely find it necessary to muzzle dogs or cats for ultrasound examination if the pet is handled calmly and comfortably. When necessary however they should be placed before the pet becomes too excited. In those situations the use of sedative medications that are safe and appropriate are in order. When we deal with frightened dogs or cats that always express anxiety before coming to the hospital these may be times when pre-sedation with GABA or other relaxing agents are useful. More often, gentle care, appropriate handling and caging are effective in smaller pets under such circumstances.

State of the art physical examinations are too often neglected in the modern world of machinery and advanced medical equipment. Regardless of the complexity and uniqueness of such advanced medical equipment, most excellent veterinarians and physicians will always recognize that before testing is indicated, a thorough history and physical examination is completed. It provides the doctor not only with useful directed information but also permits evaluation of other organ systems that will provide helpful information as to the pet’s health status. Clients expect the DVM to provide their pet with a thorough examination and often comment negatively when one is not done and the outcome of the case is less than desirable. More importantly, the physical examination continues to provide information that is always going to be useful in determining the overall health and/or problems the pet may be experiencing.
The Best Medicine: Why Fear Free is a Practice Imperative
Stephen Ettinger, DVM, DACVIM
Los Angeles, CA

Relevance to our profession in 2017
Veterinary medicine has gone through many changes over the past few decades. Initially a para-health profession designed in the late 1800’s and early 1900’s as one of need for transportation and public health. The considerations for its importance laid more with transmissible diseases, diseases of horses and cattle and our food animals as well as the need for caring for horses or those animals that were important to the transport of food, equipment and supplies to the general public. At that time, the profession was largely located in the rural areas of the country and its application to human medicine and the needs of society were very different than they are today.

Times have changed and with it the needs of our profession. From the period of the division of medicine into essentially large and small animal studies, there have been many modifications to the profession. Veterinary schools were essentially part of the agricultural schools and their importance was associated with the work done in those areas. Little contact between physician and veterinarian occurred. Yes, there were a few relative small practices in urban areas but that was neither the focus of the profession nor where monies were allocated to as the profession grew.

Continued growth of the profession remained at the veterinary school rural level and funding was delivered to those schools where agricultural concerns were of primary importance. Research was directed in the same view, often where agricultural concerns outweighed medical and scientific advances. This was at the time good for the profession and the public but then there was a shift in the relevance of the profession beginning in the middle of the 20th century that has continued its progression to its current status.

Today, many of the veterinary schools have strong collaborative research programs with nearby human medical centers and much of the basic research in science is done both at medical and veterinary medical institutions. Much of this research is high tech science designed to investigate ultimately the nature and back ground of diseases in general and ultimately in particular to the human being that the hospitals serve. With this came a growth or spurt in knowledge of veterinary science and the diseases and problems associated with species other than the human. Many of the research veterinary institutions today have significant high funded federal grant monies for extensive basic science studies.

With the increase in scientific understanding of both human medical and veterinary medical problems came a change in the population density of the country as well, with a significant proportion of the population becoming urban dwellers (or suburban). Other species were incorporated into the households of many of the people living in the cities. Dogs and cats were no longer relegated to the “barns” or outside living conditions but slowly and progressively were invited into the homes of their providers. With this came a new interest in the pet as a member of the family and a tight bonding experience grew to the point that today, most “pets” are considered more a part of the family than they are as pets. Those taking these animals into their homes provide for their care in a most unique way and provide, food, exercise, living conditions and “free play” much as they do for their children. Care of the animals is a primary focus of concern and veterinary medical visits to the family veterinarian are common place experiences in the homes of the pet caregivers.

As pets became more and more a part of the family environment and circle, the practice of veterinary medicine became more important with clients searching not only for quality medicine but for veterinarians who showed compassion and concern towards these animals. New tools and methods for caring for the pets followed the lines of human medicine and no longer was the pet a laborer in the barn but rather a child in the house requiring and deserving the same quality care that the people in the house received.

Veterinarians have by now learned that they serve a unique function in that they run (or are employed) by a business and at the same time, need to have nice facilities that are comfortable for their clients, up to date and knowledge of new advances in medicine such as anesthesia, pain free methods of care, sterile surgical environments and very sophisticated medical equipment meant to provide rapid, accurate and quality medicine much as they would expect for themselves under similar circumstances. Learning to be a good scientist was one of the first things veterinarians were taught but today they are also learning good business skills, up to date state of the art medical and diagnostic skills as well as learning how to be both good listeners and communicators to the client. Those who do this well find that their practices are happy, fun places to work in, provide the veterinarian and clients with good medical and social experiences and provide the animals under their care excellence in medicine and emotional support.

This support includes learning how to talk effectively and in terms that the clients can understand. Often it is not as much what the veterinarian says to the client but how she or he gets that across to the client that separates a successful DVM from one unable to maintain a good clientele that remains loyal over the lifetime of the pet. Included today are many newer facets of medicine that the client has come to expect of their local veterinarian. Aside from the facility, the client also expects up to date knowledge in medical therapies, immunotherapy, chemotherapy, potentially isotope treatment, alternative medicines, physical medicine and FEAR FREE medical care.
Fear free is one of the newer areas of medicine designed to keep the owner and pet less stressed and more at ease when visiting the veterinary hospital. Pets recognize when the visit to the hospital is anticipated and many are frightened and anxious long in advance of the visit itself. Preparing the pet and the client for the visit is one of the important factors that go into FEAR FREE medicine. It is a goal of course to keep the pet free of pain or discomfort but that is not always possible with the newer standards of care and medical approaches that we take to help the patient. What has changed however it's the concern for each of these factors. Reducing anxiety about visiting the doctor is important to the client and the pet. Many clients avoid bringing their pets to the hospital because of excessive angst on the part of the pet when he or she arrives. Behavioral training and good medical options are thus the first part of the delivery of fear free medicine. Discussing some of the options to help alleviate this anxiety is an important part of the discussion at hand. Following up on this and keeping that anxiety level lowered or at least more comfortable is equally as important.

Going to the hospital and fear free
It has been shown that many owners acknowledge the trip of going to the veterinary facility as very stressful and thus avoid it (loss of income, good care and reputation) as long as possible. Thus stressing ways that the DVM can reduce this stress is very important. The choice of adjusting the animal to regular car rides that are not necessarily to the hospital is important. Prior training are to the pet teaches the animal that each ride is not a frightful one. Various techniques can be discussed and left open to group discussion in this regard. Use of anxiolytics n the form of supplements, treats or even medications can be utilized. How the transport is carried out is also relevant in this regard. These topics will be discussed in greater detail with time left for a general discussion on options others have used to reduce these stresses.

Visiting the hospital and introduction to the facility
Again, a new (or even worse the introduction of the pet to an environment that smells bad (to the pet), looks fearful and simply is filled with other animals scents that give reason for angst is a reason for a pet to have a negative reaction to the experience. Similarly the appearance of the waiting room, the examination room and the processes that are completed even before seeing the DVM provide many pets with significant anxiety. These animals do not know why they are being taken to the strange place and react accordingly.

Techniques for meeting the vet and the work that follows
In walks the new veterinarian gownned to the hilt with white coat (how many of you like them?) instruments previously just used on other animals with different scents and a strong need to move quickly since he or she is behind in the appointment schedule. This is enough to frighten anyone from the owner or caregiver to the house staff. Once again, many techniques some old and others new present the opportunity to give the pet a sense of comfort and reduce anxiety. It is at this point that the client and the pet make real decisions about the value of the office examination, vaccinations and general medical care. The veterinarian must be both comfortable, at ease and willing to provide both the time and concern that will allow the pet to remain comfortable and at ease while the owner is gaining confidence in the work that is being completed. During this time a discussion of finances may also take place adding additional anxiety to the client’s already mix bag of concern.

Recognizing fear in an animal is an essential part of this early part of the meeting. In dogs, stress is often associated with growling and showing the teeth but lower levels of fear or stress are shown by lowering the body, moving the tail down so that it is lower than the back and often held straight-out. Looking at the tense facial muscles, dilated pupils and wrinkles between the eyes are other commonly noted features that can identify stress and that tell the DVM that time is needed to reduce or lower that fear or that the examination should be completed in another more comfortable area.

In the cat, stress is identified with a crouched body, an arched back, holding the tail against the body often with the tip of the tail moving slowly back and forth (not a happy wagging of the canine tail example). Often the legs are tucked underneath the body, while the ears are back and casual observation prevents the ears from being observed as they normally would. Of course hissing and open mouth anger is the more obvious and recognizable feature that one would hope is not present.

Any of these signs can be those of stress and it is the job of the DVM to recognize these and make attempts to mollify the situation. Making the pet feel more comfortable will also allow the owner to be more relaxed and the DVM must determine whether the animal should be examined with or without the owner present. If the owner is present and can observe a calm pet, it helps them to recognize the situation as being comfortable and more fear free than they may have expected.

Other discussion about fear free
The above material covers only the beginning of the Fear Free experience. During this hour and the next to follow, techniques for limiting anxiety will be introduced and discussed. No examination or visit to any hospital can be entirely with out some anxiety but the goal of keeping the pet, the owner and the doctors more at ease helps to reduce angst, maintain a better relationship with the owner and most importantly to reduce stress and help the pet through what otherwise can be a very unpleasant experience while turn it into one that is more amenable to continuing the goal of quality of life and health care.
This presentation will examine different areas of the hospital and the roles of team members with the implementation of Fear Free™ concepts in those areas. It does not take a complete renovation of your hospital to cultivate a relaxed and inviting hospital “spaw” for the patient and client.

Pre-visit preparation

The client care representative (CCR) will be the main communicator regarding pre-visit preparation. However, the veterinarian and the veterinary technicians/assistants may also be involved with planning pre-visit recommendations with the client for future visits. The CCR should review the emotional record of the patient prior to contacting the client to confirm the scheduled appointment. Any special accommodations that have been noted in the record should be reviewed with the client. For example, if the veterinarian prescribed pre-visit pharmaceuticals or suggested the client call the front desk from the parking lot to notify the hospital of their arrival, the CCR will remind the client of the recommendations and ask them to contact the hospital if they have any questions. Other general pre-visit suggestions that can help to reduce fear, anxiety, and stress associated with travel to the veterinary hospital can be posted on the hospital website or sent in an email reminder prior to an appointment. Some general recommendations include:

- Acclimate and train the pet for transport (carrier, crate, seatbelt)- a qualified professional trainer may need to assist the client with this training.
- Provide non-slip surfaces during transport to prevent sliding.
- Utilize aroma and acoustic therapy to create a calm environment.
- If medically warranted, bring a hungry pet and the pet’s favorite treats.
- Bring familiar objects from home, such as a bed or toy. Something with the smells of home, brings familiarity to a novel situation.

General hospital recommendations

Some general concepts to incorporate throughout the entire hospital to create an environment that is calming and inviting include: providing non-slip surfaces for patients to have good footing, using aroma and acoustic therapy, providing all team members, who are versed on proper treat use, with treat bags for easy access to reinforcers for patients, and integrating a considerate approach when interacting with patients. To avoid overwhelming our patients, it is best to allow them to approach you. Turn sideways and avoid direct eye contact. This makes you appear more approachable. Talk slowly and softly to facilitate a relaxing environment. Move smoothly and calmly and avoid aversive scents and instead use calming ones.

Reception area

The CCR will be the primary Fear Free™ representative and play a large role in creating a pleasant experience with the patient and client’s arrival to the veterinary hospital. The CCR can play the role of the concierge and be ready to assist the client with movement into the hospital.

Remember to use a considerate approach and let the pet decide if he/she wants to interact with you. Pending the reason for the visit and the emotional record of the patient, the CCR should be ready with the patients preferred treats. Evaluation of the patient’s emotional state should be made and documented/communicated prior to handing off to the next team member. Although the CCR’s are the primary face of the reception area, all veterinary healthcare team members, veterinarians, technicians, management, and other team members, that are in the area should assist with creating a Fear Free™ environment. Management will have the responsibility of orchestrating certain cosmetic changes that can create a more relaxed and inviting reception area, such as using natural or incandescent lighting, avoiding seating near entrances and exits, utilizing barriers to block sight lines.

The scale

The scale bears special mention, as the pet owners or team members attempts to obtain a weight on the pet, often results in fear, anxiety, and stress if special considerations are not made. Ideally, the scale should be stable, with a non-slip surface, and in an open area to avoid the dog having to walk into a corner to get on it. Rather than pulling or forcing the dog to get on the scale, use a treat trail and/or motion to encourage the dog to make the choice to sit on the scale. Often times the more we try to force the issue, the more fearful the dog becomes and the more he/she wants to get away. In the end taking a few seconds and a few treats will save you a lot of time and stress.
Examination room
The veterinary technician/assistants will play a pivotal role in creating the Fear Free℠ environment in the examination room. The technician should allow the pet to explore the room, while he/she reviews and obtains the medical and behavioral history with the client. The technician's observations of the pet’s emotional state should be recorded and communicated to the veterinarian.

Ideally, once the pet has acclimated to the room, it is best to bring the procedures to the pet whenever possible. This allows the pet to stay in an area that is safe and comfortable and with their owner. If an owner is uncomfortable being in the room during procedures, you may ask them to step out into the reception area for a moment.

The use of gentle control and considerate approach should be utilized while performing procedures (examinations and other diagnostic tests or treatments) and they will be discussed in more detail in the next presentation.

The veterinarian, CCR, and management will play accessory roles. Each exam room should be stocked with a variety of treats and toys. A water bowl should be provided for each patient. Travel to the hospital and treatments can make for a thirsty pet. Cats should be provided with access to a litterbox to allow them an opportunity for relief. Cats especially need hiding spots to feel safe. Provide them with accessible hiding spots, such as their carrier with the top removed, a towel, or a cat tree with elevated perches. If possible, try designating exam rooms as feline only or canine only.

Treatment room
If it is necessary to take the patient to the treatment area, first reconnaissance your route without the pet. Inform other team members of your intent to bring the patient to the treatment area and make accommodations to avoid or minimize potentially frightening stimuli. Have all supplies prepared for the treatment prior to bringing the patient into the area. This will minimize the amount of time the pet has to be in the unfamiliar area. Utilize barriers as the pet is transported to the treatment area and while in the treatment area to decrease exposure to potentially frightening stimuli.

The veterinarian, technicians, assistants, and other team members, will be the primary Fear Free℠ advocates for the treatment room. Continue to assess the patient’s emotional well-being and make notes in the emotional record as warranted.

Housing area
The housing area is often the area in which other team members, such as the pet care specialists or kennel attendants, will be responsible for being the Fear Free℠ advocate for the pets. Technicians/assistants and the veterinarian will also play an accessory role in this area. The kennel team will be responsible for recognizing fear, anxiety, and stress in patients and recording and communication this information to others. Barriers can be used such as cage covers, hiding boxes, or screens to decrease stress inducing stimuli. Appropriate placement of patients should also be considered. Cats generally prefer to be up high. The dog that gets overly aroused when people or dogs walk past, should be placed in an area of lower traffic. Whenever possible, use a considerate approach when removing the pet from a cage or kennel and close kennel doors quietly. Conversations should be quiet and soft voices should be utilized to maintain a calm and relaxing environment. Use your “spa” tone.

Summary
You can create a veterinary hospital “spaw” without building a new hospital. Adapting our approach to patients and integrating small environmental changes, can create a relaxing oasis for our canine and feline patients. With Fear Free℠ techniques it is best to be proactive and try to prevent fear from developing in the first place. The goal of each visit is for the pet and client to have a positive and fun experience, rather than a neutral or negative experience. Each member of the veterinary team plays a vital role in creating a Fear Free℠ visit for the patient and client.

Resources
For more information on the Fear Free℠ certification program and resources visit: www.fearfreepets.com
What will you implement?
Make a list of Fear Free℠ concepts you will implement at your hospital/facility in the next 1-4 weeks.

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Fear Free:
Learning to Listen to Our Patients
Debbie Martin, LVT, VTS (Behavior)
Veterinary Behavior Consultations
Spicewood, TX

“The goal of Fear Free℠ is to improve patient health, welfare, and well-being as well as enhance the client and team experience.” In order to accomplish this goal, we need to be able to understand how our patients might perceive the veterinary hospital, recognize behavioral indicators of relaxation as well as fear, anxiety, and stress in our patients, and realize how pleasant and unpleasant associations can be formed.

Sensory perception
Stimuli in the veterinary hospital can create fear, anxiety, and stress in our canine and feline patients. Understanding how our patients perceive the veterinary hospital, allows us to not only empathize, but also develop solutions to make their experience more pleasant. In order to understand a dog or cat’s perception of our hospital, we need to have an understanding of their senses. We will explore how dogs and cats’ sensory perception compares to human perception.

Vision
Compared to humans, dogs and cats have poor visual acuity, a wider field of vision, and a smaller area of binocular vision. Cat see about 20/100-200, have a field of vision of 200° and a binocular vision overlap of 90-100°.1 The visual acuity of the dog is about 20/75 with a field of vision of 245° binocular overlap of only 30-60° pending facial morphology.2 In comparison, a human generally sees 20/20, has a field of vision of 180° with a binocular field of vision overlap of 140°. Although dogs and cats do not have very good visual acuity, they are very good at motion detection. Compared to humans, both cats and dogs can see better in dim light because of the increased number of rods in the retina and the tapetum lucidum, a reflective layer located behind the retina. Color vision is less developed in dogs and cats. Cats most likely have dichromatic vision with sensitivity to greenish-yellow and blue. Dogs are considered red-green color blind.

According to Heather E Lewis, AIA, “The ability to see the UVB spectrum is interesting because it means that some materials appear to fluoresce to dogs [and cats], including organic material like urine that contains phosphorous as well as bright white, manmade materials such as paper, plastic and white fabrics, Lewis says. Because these white items are more visually jarring to dogs [and cats], their use should be avoided.”

Hearing
Cats and dogs hear a wider range of frequencies than humans. The range of frequency for the cat is 20 Hz up to 85,000-100,000 Hz with the useful range probably up to 60,000 Hz.1-4 The range for dogs is 15 Hz up to 65,000 Hz with hearing best at around 4,000 Hz.2 The range for humans is 20 Hz up to 19,000.2 Because dogs and cats have moveable pinnae they are better able to locate the source of sounds.

Smell
Dogs and cats have more epithelium dedicated to smell than humans; Dogs 20-200 sq. cm, cats 20 sq. cm, and humans 2-4 sq. cm.2 Smells are an important form of communication for dogs and cats. The vomeronasal organ is located in the roof of mouth. In dogs it does not open into the nasal cavity as it does in cats. The vomeronasal organ is important for detecting pheromones and in social communication.2

Taste
The dog’s perception of taste is similar to humans. They are sensitive to sweets and prefer novel/fatty foods. Palatability is affected by texture, smell, temperature, and flavor. The typical adult cat responds to salty, sour, and bitter tastes. The cat’s response to sugars is inconclusive.4

Touch
Touch is important for maintaining social relationships. Touch receptors are located at base of every hair and the vibrissa are especially sensitive. Skin receptors sense proprioception, pain, temperature, chemical stimulation, and pressure. Touch can be calming, arousing, or aversive, depending on

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the type of touch, the circumstances, and the individual.

**Sensory perception chart**
Make a list of stimuli that the pet or client will see, hear, smell, taste, or feel in your hospital. Visually transport yourself from the parking lot to the housing/kennel area. Group the stimuli in categories of potentially pleasant and potentially unpleasant.

**Communication**
Being able to recognize signs of a relaxed or stressed patient is critical to creating a Fear Free℠ environment. Dogs and cats communicate primarily through visual (body language), auditory (vocal), and olfactory (pheromone and scent) communication.

**Vocalization**
Often high pitch sounds are considered distance decreasing, meaning coming closer. Examples of distance decreasing sounds in the cat and/or dog are meowing, purring, whines, whimperers, or high pitched barks. Low pitch sounds are generally considered distance increasing, meaning go away. Examples of distance increasing sounds in the cat and/or dog include the hiss, yowl, shriek, deep or guttural growl and/or bark.

**Olfactory**
Olfactory communication is extremely important to the cat. Each cat has his/her own signature scent. When one cat in the house visits the veterinary hospital, he/she will return home smelling differently. This can result in the resident cat being unable to recognize his housemate.

Dogs and cats release pheromones that can be detected by other members of their species. These pheromones can communicate pleasant and unpleasant information. A stressed cat may leave chemical messages behind that will act to create fear and anxiety in other feline patients throughout the day.

**Body language**
Being able to interpret body language in dogs and cats involves not only analyzing the entire pet (facial expression, tail carriage, and body posture) but also assessing the context of the interaction.

**Human communication with dogs and cats**
Threatening gestures include prolonged eye contact, approaching directly, and distance increasing vocalizations (deep, guttural sounds). To provide our patients with a considerate approach, we should avoid direct eye contact and a direct approach, turn sideways to look smaller and less threatening, move smoothly and calmly, talk slowly and softly, allow the pet to approach you, offer treats if medically appropriate to do so, avoid aversive scents and use calming ones instead.

**Associations**
Associations are being made all the time. Because we tend to be systematic in our approach to veterinary medicine, animals quickly learn the order of things to come. For example, when placed on a table and the tail is touched, the thermometer will follow. When the technician gets the nail trimmers out of the drawer, nail trimming is about to occur. We can create pleasant associations rather than neutral or negative ones with stimuli in the hospital by pairing pleasant stimuli with a neutral or possibly unpleasant one. For example, nail trimmers can become associated with getting canned dog/cat food. Consequently, the dog or cat becomes excited when he/she sees the trimmers.

**Conclusion**
Our patients are often more sensitive to environmental stimuli than we are. By taking into consideration how cats and dogs perceive the environment, we can create pleasant experiences and minimize unpleasant ones for them. Through early recognition of behavioral signs of fear, anxiety, and/or stress and intervention on our end, we can prevent the escalation of fear in our patients. Thus, we can facilitate pleasant associations with the veterinary hospital and the procedures we want to perform.

**References**

**Resources**
For more information on the Fear Free℠ certification program and resources visit: [www.fearfreepets.com](http://www.fearfreepets.com)
As a general rule, the closer one comes to the oral route of food intake and digestion, the more efficient is the assimilation and digestion of nutrients and the greater the flexibility in formula composition. Conversely, the further aboral one gets, the less efficient is the assimilation and digestion of nutrients and greater care must be taken when choosing formula composition. Route of administration also dictates feeding tube diameter; tube diameter in turn dictates usable feeding formulas due to varying formula viscosity and particulate matter size. The most common routes of administration for enteral hyperalimentation include oral, nasoenteral, enteric feeding, gastrostomy, and jejunostomy. Techniques for placement of an esophagostomy feeding tube will be presented.

**Esophagostomy**

**Indications**

Esophagostomy tube feeding is indicated in anorexic patients with disorders of the oral cavity or pharynx, or anorexic patients with a functional gastrointestinal tract distal to the esophagus.

**Contraindications**

Esophagostomy tube placement is contraindicated in patients with a primary or secondary esophageal disorder (e.g., esophageal stricture, after esophageal foreign body removal or esophageal surgery, esophagitis, megaesophagus) and patients with a history of vomiting.

**Advantages**

Advantages of esophagostomy tube feeding include ease of tube placement, tubes are well tolerated by the patient, large bore feeding tubes can be used allowing use of blenderized diets, tube care and feeding is easily performed by the client, patients can eat and drink around the tube, and tube removal can be performed anytime after placement. Esophageal tube placement eliminates local pharyngeal irritation, coughing, laryngospasm, or aspiration occasionally associated with pharyngostomy tubes.

**Disadvantage**

The major disadvantage of esophagostomy tube is the need for general anesthesia during placement.

**Placement technique**

Provide general anesthesia. Place the patient in right lateral recumbency with the left side uppermost. The tube can be placed on either the right or left side of the midcervical region, however the esophagus lies slightly left of midline making left sided placement more desirable. Aseptically prepare the lateral midcervical area from the angle of the mandible to the thoracic inlet. Slightly extend the neck and hold the mouth open with a mouth speculum.

Pre-measure and mark a 20 to 24 French feeding tube for dogs and a 16 – 18 French feeding tube for cats from the level of the mid-cervical region (i.e., exit point of feeding tube) to the level of the seventh or eighth intercostal space; ensuring mid- to caudal esophageal placement. Make certain the tube does not cross the lower esophageal sphincter (LES) as this may cause sphincteric incompetence, gastric reflux of acid, esophagitis and subsequent vomiting or regurgitation. Prior to tube placement, enlarge the two lateral openings of the feeding tube to encourage smoother flow of blended diets.

**Eld esophagostomy tube placement technique**

The following technique requires the use of an Eld feeding tube placement device and is illustrated in the esophagostomy video labeled E-tube. Place the oblique tip of the instrument shaft through the oral cavity and into the esophagus to the level of the midcervical region (i.e., equal distance between the angle of the mandible and thoracic inlet) and palpate the tip as it bulges the cervical skin. Make a small skin incision over the device tip. Activate the spring loaded instrument blade until it penetrates esophageal wall, cervical musculature, subcutaneous tissue and is visible through the skin incision. Carefully enlarge the incision in the subcutaneous tissue, cervical musculature and esophageal wall with the tip of a #15 scalpel blade to allow penetration of the instrument shaft. Place a 2-0 Nylon suture through the side holes of the feeding tube and through the hole in the instrument blade. Tighten the suture until the tip of the instrument blade and feeding tube tip are in close apposition. retract the instrument blade into the instrument shaft so the feeding tube tip just enters the instrument shaft (i.e., deactivating the instrument blade. Place sterile water-soluble lubricant on the
tube and instrument shaft. Retract the instrument and pull the feeding tube into the oral cavity to its predetermined measurement. Remove the 2-0 Nylon suture to free the feeding tube from the instrument. Place a stylet through one of the side holes of the feeding tube and against its tip (do NOT use a stylet when placing an E-tube in cats). Lubricate the feeding tube and advance it into the esophagus until the entire oral portion of the tube disappears. Gently retract the stylet from the oral cavity being careful to ensure its release from the feeding tube. If you encounter resistance and cannot pass the feeding tube into the esophagus you may have engaged the endotracheal tube. If this happens remove the feeding tube and replace it under direct visualization. Secure the tube to the cervical skin with a Chinese finger-trap suture of #1 Novafil.

Curved Carmalt hemostat technique

Instead of the Eld device a curved Carmalt hemostat can be used to place an esophagostomy feeding tube. Patient and feeding tube preparation is identical to that stated above for the Eld technique.

The curved Carmalt forceps is placed into the cat's oral cavity with the curve of the hemostat directed toward the cervical region. The Carmalt is directed to a point equidistant between the ramus of the mandible and point of the shoulder midway between the dorsal and ventral aspect of the neck. The hemostat is pushed laterally so as to make a 'bulge' in the cervical region at the desired exit point described above. A scalpel blade is used to incise over the tip of the Carmalt until the tip protrudes through the skin. The tip of the feeding tube is then grasped with the Carmalt hemostat and the tube is exited out through the oral cavity. The tube is pulled out until the flanged end of the tube just comes in contact with the cervical skin. The tip of the tube is then turned back on itself, grasped with the Carmalt forceps, and redirected into the oral cavity of the cat. The tube should remain in the jaws of the Carmalt hemostat until the tip of the tube is beyond the cervical exit point of the tube. The feeding tube is then released from the Carmalt and pushed into the esophagus until the tube is in the mid-esophagus (i.e., 7 or 8th intercostals space). The tube is secured using a Chinese finger-trap friction suture.

Regardless of technique used, the exit point of the tube can be left exposed or bandaged. A column of water is placed in the tube and the exposed end capped with a 3 cc syringe; this prevents intake of air, reflux of esophageal contents, and occlusion of the tube by diet. Most patients tolerate the tube without the need of an Elizabethan collar.

Esophagostomy tubes can be removed immediately after placement or left in place for several weeks to months. Care of the tube exit site may require periodic cleansing with an antiseptic solution. Tube removal is performed by cutting the finger-trap suture and gently pulling the tube. No further exit wound care is necessary; the hole seals in one or two days and heals by 7 - 10 days.

Complications

Complications associated with esophagostomy tube placement include early removal by the patient or vomiting the tube No significant long-term complications have been reported (e.g., esophagitis, esophageal stricture, esophageal diverticulum, or subcutaneous cervical cellulitis). Reflux esophagitis can occur from improper tube placement (i.e., through the lower esophageal sphincter) or esophageal irritation from the tube itself. Mid-esophageal placement of silicone rubber tubes greatly reduces the incidence of esophageal injury and eliminates reflux esophagitis.
Feline Linear Foreign Bodies
Howard Seim III, DVM, DACVS
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Key points
• Don’t let the sun set on a GI obstruction
• Always look under the tongue in suspected linear FB
• Multiple enterotomies may be needed
• Surgery prior to mesenteric perforation improves prognosis dramatically

If you would like a copy of the illustrated version of these notes on CD and a video of this surgical procedure on DVD, go to www.videovet.org and click VideoVet or contact videovet@me.com.

Linear foreign bodies
Clinical presentation
Linear foreign bodies (e.g., string, plastic bags, tinsel, tape deck tape, yarn, thread) occur in the dog and cat. The classic presentation is a patient four years of age or less with persistent vomiting, anorexia, and depression. These signs are common with many gastrointestinal disturbances and linear foreign body should be included in your differential diagnosis. Occasionally, patients are presented late in the course of the disease and may have a history of intermittent vomiting with anorexia, depression, and weight loss as the major presenting signs.

Diagnosis
A thorough physical examination should be performed with emphasis on oral examination and abdominal palpation. Oral examination often reveals the linear foreign body around the base of the tongue in cats. The foreign body itself may be seen or an area of inflammation may be present at the junction of the base of the tongue and frenulum. Abdominal palpation may reveal "bunched-up" small intestine due to the plication. When this finding is made, the clinician should be very gentle with further abdominal manipulations so as not to encourage bowel perforation.

Radiography
Definitive diagnosis is based on characteristic findings on survey and contrast radiography. Survey radiographs may reveal plicated bowel bunched up in one quadrant of the abdomen. Due to its plicated nature, air accumulation in the bowel lumen forms a characteristic "tapered enteric gas bubble”. Three or more tapered gas bubbles are diagnostic for linear foreign body. Evidence of peritonitis (i.e., ground glass appearance), free gas in the abdominal cavity, ileus, or the presence of a needle are findings that may be present on survey radiographs. Patients with subtle changes or questionable findings should have an upper gastrointestinal contrast study. The typical plicated appearance of the bowel is diagnostic for linear foreign body.

Presurgical treatment
Surgery for the removal of linear foreign bodies should be accomplished as soon as possible. Presurgical preparation of patients diagnosed early and in good health include an intravenous catheter, maintenance fluids (22 ml/kg TID), replacement of fluid loss from vomiting and dehydration, and antibiotics prior to abdominal exploratory. Patients that present in septic shock (i.e., perforation, peritonitis, severe dehydration) should be treated with a graduated replacement of fluids (as needed up to 90 cc/kg IV) and antibiotics (cefotixin, ampicillin and enrofloxicin, or gentamicin and ampicillin). Electrolytes (chloride, potassium, sodium) and acid-base evaluation are helpful in presurgical management. When fluid losses have been replaced and shock therapy instituted the patient is anesthetized for abdominal exploratory.

Surgical treatment
After ceiliotomy, the plicated bowel is gently exteriorized from the abdominal cavity. In order for a linear foreign body to result in intestinal obstruction and clinical signs, it must be lodged somewhere in the proximal gastrointestinal tract. Common areas include: base of the tongue (i.e., string is often looped around the base of the tongue), stomach or pylorus (i.e., a ball of string is often lodged at the pylorus), or duodenum (i.e., the string becomes impacted in the descending or ascending duodenum). The surgeons’ first task is to locate the area in which the foreign body is lodged and release it. If it is lodged under the tongue it should be cut at the time of exploratory laparotomy; if it is lodged in the stomach or pylorus, it is released via a gastrotomy; if it is lodged in the duodenum, it is removed via enterotomy.

Once the proximal end is released, the extent of the linear foreign body is evaluated, and 2-3 subsequent jejunal enterotomies are performed to remove the remainder of the foreign body.

Care is taken to remove the linear foreign body in segments short enough that further cutting of the mesenteric border of the intestine does not occur during removal, yet long enough to perform a minimum number of enterotomies. These numbers and distances vary with the type and length of linear foreign body involved. The mesenteric border is examined carefully for evidence of
perforation. All linear foreign bodies should be removed to the level of the ascending colon. Colotomies are not necessary, as once the linear foreign body is in the colon it can be passed with little danger of causing obstruction.

An alternate technique for removal of a linear foreign body is to identify and release the obstructed proximal aspect of the foreign body and attach the released end of the linear foreign body to the flanged end of a 12 - 18 French red rubber catheter/feeding tube. Pass the blunted end of the catheter into the gastrotomy or enterotomy and pass it aborally through the entire length of the intestinal tract and out through the anus. As the catheter is passed, it pulls the linear foreign body out of the GI tract and releases the bowel from its plication. This technique eliminates the need for multiple enterotomies to remove the foreign body. Difficulty can arise when attempting to pass the catheter through the small intestine. Care should be taken not to encourage further trauma to the mesenteric border while passing the catheter.

After the foreign body has been completely removed, a close examination of the mesenteric border is made for evidence of perforation. Any perforation should be debrided and sutured. If multiple perforations occur, a resection and anastomosis may be necessary. Serosal patching may be considered to protect an anastomosis or enterotomy site in a compromised patient. Serosal patching is not recommended to patch mesenteric perforations as suturing the patch may result in vascular compromise to the affected intestinal segment.

Patients with multiple mesenteric perforations that cannot be sutured without severely compromising bowel viability should undergo massive bowel resection. Remember, you can successfully resect 60 - 70% of the small intestine and have a nutritionally acceptable animal. If the client is willing to treat their dog or cat with an acid blocking agent, this resection can be expanded to a 75 - 80% small intestinal resection.

The abdominal cavity is lavaged with copious quantities (e.g., 200-300 ml/kg) of sterile physiologic saline solution prior to closure. Placement of a enterostomy feeding tube should be considered in severely debilitated patients. Postoperative management (i.e., fluids, antibiotics, feeding) is as previously discussed.

**Prognosis**

Prognosis for patients with linear foreign body is directly related to the presence or absence of bowel perforation at the time of surgery. Patients without preoperative perforation have an 85% chance of survival while those with preoperative perforation have only a 50% chance of survival. This survival rate further reinforces the importance of early diagnosis and surgical treat References Head

References
Feline Perineal Urethrostomy: A Novel Approach
Howard Seim III, DVM, DACVS
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Key points
- Patients with cystic and urethral calculi present with stranguria
- Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
- Aggressive lavage of the urethra and bladder should be performed during cystotomy
- Permanent urethrostomy is an acceptable method of managing chronic stone formers

Definition
Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

Diagnosis
Clinical presentation
Signalment
There is no age, sex or breed predisposition.

History
Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, blood tinged urine in the litter pan, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.

Clinical signs
The most frequently reported clinical signs in patients with cystic and urethral calculi include unproductive straining to urinate, blood tinged urine seen in the litter pan, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbant, shocky).

Physical examination
Abdominal palpation may reveal a full urinary bladder; occasionally, calculi within the bladder may be palpable. Patients with severe clinical signs (i.e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression. Abdominal palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.

Laboratory findings
Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and/or crystalluria. Patients presenting after several days of complete obstruction may have significant changes in their biochemical profile including increased BUN, increased creatinine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystalluria.

Radiography
Survey radiographs may show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. Careful radiographic evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

Ultrasoundographic examination
of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

Differential diagnosis
Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.
Medical management
Immediate care
In animals with complete obstruction long enough to cause azotemia, temporary urinary diversion is provided by performing a prepubic cystostomy (see technique described below) or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.

Urethral catheterization of a female cat
• Female urethral catheterization is easier than male
• Use a closed ended tom cat catheter
• Ventral recumbancy is recommended
• Pass the catheter with no evidence of resistance

Retrograde hydropulsion of lodged urethral calculi
Calculus removal
Retrograde hydropulsion: This technique should result in an 80-85% success rate for retropulsing urethral calculi into the urinary bladder!

Thoroughly mix 20 cc of sterile saline and 5 cc of Surgilube or K-Y Jelly in a 35 cc syringe and attach the syringe to a 3.5 - 5.0 French soft rubber catheter/feeding tube.

Anesthetize the patient, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.

Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure.

a) During injection, the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

b) This technique is attempted, and generally successful, regardless of how many stones are in the urethra and no matter where they are lodged.

If the above technique fails, use a stiffer catheter (i.e., open or closed ended tomcat catheter) and repeat the above maneuvers. Use care when manipulating these stiffer catheters against the calculus.

Surgical treatment
The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit from a permanent urethrostomy to allow continual passage of small urethral calculi.

Preoperative management
Patients that present acutely can be anesthetized immediately and retropulsion attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients’ electrocardiogram should be monitored if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via multiple cystocentesis (i.e., tid or qid), or placement of an antepubic cystostomy tube (described in detail below).

Anesthesia
Routine general anesthesia is performed in patients that present acutely without signs of azotemia. Azotemic, shocky patients with moderate to severe biochemical abnormalities should be treated as described above until these abnormalities return to normal.

Surgical anatomy
The male feline penile urethra consists of urethral mucosa (i.e., urothelium) surrounded by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the blood filled corpus cavernosum urethra and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

The urinary bladder consists of the following layers; serosa, muscular, submucosa and mucosa. The bladder is lined with transitional epithelium.

Positioning
Patients are positioned in dorsal recumbancy for retropulsion, cystostomy tube placement and routine cystotomy.

Urethrostomy
Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening that is large enough to accommodate passage of most urethral calculi, crystals and mucoid debris.
**Perineal urethrostomy; perineal approach**

The perineal urethra is the location of choice for urethrostomy in cats. It is a convenient location for surgical manipulation, the urethral diameter will accommodate passage of most urethral calculi and there is less urine scald postoperatively.

Prior to surgery a urethral catheter is passed, if possible. After a routine castration, an elliptical incision is made around the scrotum and penis. Then the subcutaneous tissues are dissected to expose penile urethra. The penile urethra is dissected free from surrounding connective tissue. The ventral attachment of the pelvic urethral to the pubis (i.e., ishiocavernosus m.) is identified and transected. The penile urethra is freed from its connective tissue attachments to the pelvic floor using blunt digital dissection. The retractor penis muscle is identified on the dorsal aspect of the penis and is dissected from its attachment on the penis. The dissected retractor penis muscle is then used to develop the dorsal plane of dissection to separate the pelvic urethra from its dorsal connective tissue attachments. Once the urethra is dissected enough to visualize the dorsolaterally located bulbourethral glands penile dissection can stop. The penis is catheterized and the urethral orifice identified. An incision is made from the penile urethra to the pelvic urethral to the level of the bulbourethral glands using a Stevens tenotomy scissor or Iris scissor. The urethral orifice at the level of the bulbourethral glands is generally of large enough diameter to accept the flange of a tomcat catheter.

After incision of the urethra, the glistening urethral mucosa is identified. 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author. The first urethrostomy suture is placed at the dorsal aspect of the urethropotomy incision on the right or left side at a 45° angle to include urethral mucosa and skin (suture split thickness of skin). The suture is tied and cut leaving the ends 3-4 cm long to act as a stay suture. A mosquito hemostat is placed on this suture to provide traction and countertraction to enhance visualization of the urethral mucosa. The second suture is placed opposite the first suture and tied as described for the first. A stay suture is also placed here. A third urethrostomy suture is placed directly on the dorsal midline to hold the dorsal margin of urethral mucosa to the dorsal margin of the skin incision. Alternating sutures from dorsal to ventral are placed until approximately one half of the penile urethra has been sutured to skin. The remainder of the penis is amputated and the subcutaneous tissue and skin are closed routinely. Fine ophthalmic instruments make tissue handling and suturing easier. Use of a 2X magnifying loupe and headlamp light source enhances visualization of the urethral mucosa and facilitates accurate suturing. It is critical for the surgeon to recognize the glistening urethral mucosa and carefully suture it to skin. This will decrease (or eliminate) the chance of urethral stricture.

**Perineal urethrostomy; dorsal approach**

Perineal urethrostomy can be performed with the patient placed in dorsal recumbancy. This positioning is more ergonomic for the surgeon and allows easy access of the urinary bladder for concurrent cystotomy. When positioning the cat tie the hind limbs cranially until the pelvis is slightly elevated off the surgery table. Place a folded towel under the pelvis to support this slightly elevated position. The surgical technique is as described above for the perineal urethrostomy performed using a perineal approach.

**Postoperative care and assessment**

Perineal Urethrostomy: An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals. An indwelling urinary catheter placed routinely postoperatively is NOT necessary following an uncomplicated urethrostomy.

**Prognosis**

The prognosis for surgical management of urethral and cystic calculi is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that have successful retropulsion of urethral calculi and do not require urethrostomy have an excellent prognosis. If careful attention is paid during cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique), the prognosis for cure is excellent. Long term prognosis is dependant on evaluation of calculus composition, dietary management, management of urinary tract infection, and attention to urine pH.

Patients that have an elective perineal urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is too large to pass through the urethrostomy stoma.
Key Points

- Intestinal sutures should engage at least 3 - 4 mm beyond the cut edge of serosa and placed
  - No further apart than 2 mm
- Always handle bowel wall with atraumatic technique
- Examine the integrity of your anastomosis visually
- 50 - 60% of the small intestine of cats can be resected

Enterotomy

An enterotomy incision may be necessary for removal of intraluminal intestinal foreign bodies (e.g., balls, rocks, toys, linear foreign bodies), intestinal biopsy and exploration of the bile duct papilla or intestinal lumen. The segment of bowel to be incised should be removed from the abdominal cavity and packed off with moistened laparotomy pads. An incision parallel to the long axis of the bowel (i.e., longitudinal) or perpendicular to the long axis of the bowel (i.e., transverse) may be made on the antimesenteric border, preferably in healthy bowel (i.e., the aboral side of the foreign body). Closure is performed using appositional techniques (i.e., simple continuous or simple interrupted). Omentum can be placed over the enterotomy, but need not be sutured.

Transverse closure

If a large full thickness piece of intestine must be excised (i.e., mural mass, full thickness biopsy, etc) longitudinal closure may result in stenosis. To prevent this, transverse closure of the linear incision is recommended. This ensures adequate lumen diameter without the need for intestinal anastomosis. However, this technique is only recommended if a piece of intestine must be removed.

Intestinal anastomosis

Intestinal anastomosis is indicated for resection of nonreducible intussusception, necrotic bowel wall secondary to complete intestinal obstruction, intestinal volvulus, stricture secondary to trauma, linear foreign body with multiple perforations, and intestinal neoplasia (e.g., leiomyoma, leiomyosarcoma, adenocarcinoma).

After a complete abdominal exploration, the affected length of bowel is delivered from the peritoneal cavity and isolated with the use of moistened laparotomy pads and crib towels. If possible, the intestinal anastomosis should be performed on a water resistant surface (e.g., plastic drape, crib towel) to prevent ‘strike’ through contamination.

Once the level of resection has been determined, the appropriate mesenteric vessels are identified and ligated, and the portion of intestine to be resected is isolated by clamping the bowel at a 60° angle away from the mesenteric border. This angle ensures adequate blood supply to the antimesenteric border.

Everted mucosa

Occasionally when the segment of intestine to be removed is amputated mucosa ‘everts’ from the cut edge of the intestinal wall making it difficult to visualize the cut edge of the serosa. If this occurs it is ‘highly’ recommended to excise the everted mucosa to enable the surgeon to easily visualize the cut edge of the intestinal serosa. It is vital that the surgeon engage at least 3 – 4 mm of intestinal wall (measured from the cut edge of serosa) with each suture to guarantee adequate bites in the collagen laden submucosa.

Bowel lumen diameters

In cases where the oral end of the bowel is dilated and the aboral end is normal size, several options exist to create intestinal lumens of equal diameter:

1. Increase the angle of resection on the smaller diameter segment of bowel (i.e., aboral segment). This will increase the orifice size by 5-10 mm depending upon bowel diameter (e.g., dog vs cat).
2. In larger lumen size discrepancies the antimesenteric border of the smaller diameter stoma can be incised longitudinally to enlarge the lumen diameter.
3. An end-to-side anastomosis can be performed by closing the larger diameter stoma of the intestinal resection with a single layer continuous apposing suture pattern then anastomosing the smaller diameter segment of bowel to an appropriate size enterotomy made in the antimesenteric border of the larger diameter segment of bowel.
4. The larger diameter segment of bowel can be made smaller in diameter by suturing its cut edge until its lumen is equal in size to the smaller diameter intestine.

Intestinal anastomosis technique

See the DVD for a detailed video description of this technique (www.videovet.org). When suturing an anastomosis, atraumatic handling of bowel wall and perfect anatomic apposition of incised margins is important. It is recommended to begin suturing at the mesenteric border as this allows adequate visualization of mesenteric vessels and helps
prevent encircling these vessels when placing each suture. Any of the appositional suture patterns previously described (i.e., simple continuous or interrupted) will result in a high success rate, both in the short-term (i.e., leakage, breakdown) and long-term (i.e., stricture, stenosis).

The following tips may prove helpful when performing an intestinal anastomosis (see the anastomosis video clip for detailed description of tips below):

1. First, place a stay suture to hold the mesenteric border of each segment of bowel in apposition. Tie this suture, leave the ends long, and place a hemostat on the suture end end without the needle.
2. Place a second stay suture to hold the antimesenteric border of each segment of bowel and bring the ends of the intestinal segments into apposition. Place a hemostat on the ends of this suture.
3. Place gentle traction on the mesenteric and antimesenteric stay sutures to bring the two intestinal segments into apposition.
4. Using the needle segment of suture from the mesenteric stay suture, begin a simple continuous appositional anastomosis being careful to get a 3 mm bite in the submucosa and placing each suture no more than 2 - 3 mm apart (2 mm apart in cats). When the anastomosis is complete, tie the suture to the mesenteric stay suture.
5. If a simple interrupted apposing suture pattern is used, be careful to get a 3 mm bite in the submucosa and place each suture no more than 2 - 3 mm apart.

The author’s preference for evaluating the integrity of anastomotic closure is to visually examine each suture to be certain that suture placement is no more than 2 - 3 mm apart and that each suture has a 3 to 4 mm bite.

Postoperative care
Intravenous fluids to maintain hydration and ensure renal function are continued postoperatively, until the patient begins to eat and drink. Intravenous fluids should then be tapered over a 24 to 48 hour period. Systemic antibiotics are continued postoperatively for 5-7 days; 10 - 14 days in cases with peritonitis and/or sepsis.

Feeding
Early return to enteral feeding is best for the overall health of the intesting. Feeding the postoperative gastrointestinal surgical patient is generally based on the following criteria:

a. preoperative condition of the patient
b. the condition of the bowel at the time of surgery
c. surgical procedure performed (i.e., enterotomy, anastomosis, pyloroplasty)
d. presence or absence of peritonitis
e. postoperative condition of the patient.

The earlier patients can be returned to oral alimentation the better.

Complications
The most common postoperative complication of small intestinal surgery is leakage; leak is either associated with breakdown of the anastomosis or improper surgical technique (i.e., improper suture placement, inappropriate suture material, knot failure, sutures too far apart, inappropriate bite in the collagen laden submucosal layer, suturing nonviable bowel).

A presumptive diagnosis may be accomplished by the following:

1. Body temperature (may be up if acute or down if moribund).
2. Abdominal palpation: periodic, gentle abdominal palpation for pain (gas or fluid?).
3. General attitude (depression-anorexia).
4. Incision: examination of the patients incision for drainage (look at cytology if drainage is present)
5. CBC: leukocytosis followed by leukopenia (sepsis), or a degenerative left shift may imply breakdown.
6. Glucose: low glucose generally implies sepsis (this occurs early in sepsis and may be used as a screening test).
7. Abdominal radiographs: generally not helpful, they are difficult to critically assess due to the presence of postoperative air and lavage fluid. It can take 1 - 3 weeks for peritoneal air to diffuse from the abdominal cavity after routine abdominal surgery. Time variation is dependant upon the amount of air remaining in the abdominal cavity postoperatively (i.e., large deep chested animal vs a small obese animal).
8. Abdominal tap (paracentesis): a four quadrant abdominal tap is accomplished by aspirating fluid using a 5cc syringe and 20 gauge needle or placing a plastic IV catheter into the peritoneal cavity and allowing fluid to drip onto a slide.
9. Peritoneal lavage (if paracentesis is not productive): infuse 10-20cc/kg of sterile physiologic saline solution into the abdominal cavity, then gently palpate the abdomen and repeat the four quadrant paracentesis. This technique increases the sensitivity of paracentesis to 90%.

Once fluid has been obtained, a smear should be stained and evaluated microscopically. Depending upon the cell types seen, a determination of the presence of leakage can be made. Below are examples of expected cytology in patients with and without leak.
1. Healthy PMNs with few degenerate PMNs and a moderate number of red blood cells: This cytology may be expected in any postoperative abdominal procedure (e.g., OHE, abdominal exploratory, cystotomy). Your index of suspicion for anastomotic breakdown should be low. However, if clinical signs continue to deteriorate, repeat paracentesis (2 - 3 times daily, if necessary) to determine the “trend” of the abdominal fluid cytology is recommended.

2. Healthy polymorphonuclear leukocytes with bacteria located intra or extracellularly, degenerate PMNs with intracellular bacteria, free bacteria, or food particles--imply breakdown. Exploratory laparotomy is indicated.

In a recent morbidity/mortality study of patients undergoing intestinal surgery it was found that animals requiring a second abdominal surgery to treat intestinal disorders were less likely to survive than patients requiring only one laparotomy. Also, the longer it took to determine whether or not intestinal leakage had occurred the less likely the patient would survive reoperation. The take home message is: pay attention to detail during the first surgery and if you suspect a leak, early diagnosis will result in a better outcome.

**Prognosis**

The overall prognosis for uncomplicated GI surgery is excellent. The surgeon must pay attention to detail when suturing any hollow viscus organ.
Surgical Management of Cystic and Urethral Calculi in Cats
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Key points
• Patients with cystic and urethral calculi present with stranguria
• Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
• Aggressive lavage of the urethra and bladder should be performed during cystotomy
• Permanent urethrostomy is an acceptable method of managing chronic stone formers

If you would like a video of this surgical procedure on DVD go to www.videovet.org or contact videovet@me.com. You may click on the ‘Seminar Price’ for any DVD you would like to purchase.

Definition
Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

Diagnosis
Clinical presentation
Signalment
There is no age, sex or breed predisposition.

History
Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, blood tinged urine in the litter pan, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.

Clinical signs
The most frequently reported clinical signs in patients with cystic and urethral calculi include unproductive straining to urinate, blood tinged urine seen in the litter pan, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbent, shocky).

Physical examination
Abdominal palpation may reveal a full urinary bladder; occasionally, calculi within the bladder may be palpable. Patients with severe clinical signs (i.e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression.
Abdominal palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.

Laboratory findings
Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and and/or crystalluria. Patients presenting after several days of complete obstruction may have significant changes in their biochemical profile including increased BUN, increased creatinine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystalluria.

Radiography
Survey radiographs may show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. Careful radiographic evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

Ultrasonographic examination
of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

Differential diagnosis
Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.

Medical management
Immediate care
In animals with complete obstruction long enough to cause azotemia, temporary urinary diversion is provided by performing a prepubic cystostomy (see technique described below) or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.
Urethral catheterization of a female cat

- Female urethral catheterization is easier than male
- Use a closed ended tom cat catheter
- Ventral recumbancy is recommended
- Pass the catheter with no evidence of resistance

Urethral catheterization – Female

**Indications**

Urethral catheterization is indicated in patients with urethral calculi (aids in retropulsion), measuring urinary output, chronic decompression of the urinary bladder, performing contrast cystography and preoperative placement to prevent cystic calculi from lodging in the urethra during cystotomy.

**Applied anatomy**

The urethra leaves the bladder at the neck and courses caudally. The female urethra is short, straight, and wide, passing directly to the vestibule. Urinary catheterization of female cats is relatively easy because of the anatomic characteristics mentioned above.

**Anesthesia**

Heavy sedation or preferably, general anesthesia, is recommended for predictably successful catheterization of the female urethra. Occasionally, unsedated, unanesthetized cats will tolerate the procedure if they are slightly depressed.

**Technique**

**Positioning**

The cat is placed in either lateral recumbency or ventral recumbency with the hindquarters elevated on a rolled fleece. Regardless of position chosen, it is important to maintain positional symmetry during the procedure. This author prefers ventral recumbency. The patient is placed on the rolled fleece with the hind legs hanging over the fleece, abducted slightly, and the tail held or tied directly over the back.

**Patient preparation**

The long hairs around the vulva can be clipped to enhance visualization of the vulvar lips. Alcohol preparation of the vulvar lips is performed prior to catheterization. The vaginal vault can be lavaged with a 1:50 dilution of 1% betadine solution and saline.

**Catheters**

A closed ended polyethylene tomcat catheter or a 3-1/2 French diameter feeding tube is recommended for urethral catheterization of female cats. Open-ended tomcat catheters may be used but may be more traumatic to the urethra during placement.

**Catheter placement**

The catheter is removed from the sterile packaging taking special care to maintain sterility during placement. Sterile K-Y jelly lubricant is generously placed on the tip and shaft of the catheter. Closed ended polyethylene tomcat catheters have a gentle curve when they are removed from their original sterile package. This curve is used to help ‘aim’ the catheter into the urethral papilla during placement.

With the catheter in the right hand, use the left index and middle finger to gently spread the vulvar lips. With the curve of the catheter pointing toward the floor, pass the tip of the catheter along the ventral midline of the vaginal vault and vestibule, taking care not to allow the catheter tip to enter the clitorin fossa. Gently pass the catheter in a cranial direction until the catheter can be felt to ‘fall’ into the urethral papilla. If any resistance is met during attempted placement, pull the catheter caudally into the vaginal vault, re-direct the catheter to the ventral midline of the vagina and re-insert the catheter. Once the catheter is felt to ‘fall’ into the urethra, pass the catheter into the urinary bladder until urine begins to drip from the catheter, ensuring proper placement.

**Securing the catheter**

If the catheter is to be maintained for an extended period of time select a soft 3.5 French diameter catheter and secure it to the vulva using a Chinese finger-trap friction suture technique. Attach the catheter to a closed collection device to maintain asepsis.

**Catheter removal**

Cut the Chinese finger-trap friction suture and gently pull the catheter. Hematuria may be seen for 12 – 24 hours after catheter removal.

Retrograde hydropulsion of lodged urethral calculi

**Calculus removal**

Retrograde hydropulsion: This technique should result in an 80-85% success rate for retropulsing urethral calculi into the urinary bladder!

- Thoroughly mix 20 cc of sterile saline and 5 cc of Surgilube or K-Y Jelly in a 35 cc syringe and attach the syringe to a 3.5 - 5.0 French soft rubber catheter/feeding tube.
- Anesthetize the patient, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.
Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure.

a) During injection, the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

b) This technique is attempted, and generally successful, regardless of how many stones are in the urethra and no matter where they are lodged.

If the above technique fails, use a stiffer catheter (i.e., open or closed ended tomcat catheter) and repeat the above maneuvers. Use care when manipulating these stiffer catheters against the calculus.

**Surgical treatment**

The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit from a permanent urethrostomy to allow continual passage of small urethral calculi.

**Preoperative management**

Patients that present acutely can be anesthetized immediately and retropulsion attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients’ electrocardiogram should be monitored if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via multiple cystocentesis (i.e., tid or qid), or placement of an antepubic cystostomy tube (described in detail below).

**Anesthesia**

Routine general anesthesia is performed in patients that present acutely without signs of azotemia. Azotemic, shocky patients with moderate to severe biochemical abnormalities should be treated as described above until these abnormalities return to normal.

**Surgical anatomy**

The male feline penile urethra consists of urethral mucosa (i.e., urothelium) surrounded by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the blood filled corpus cavernosum urethra and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

The urinary bladder consists of the following layers; serosa, muscular, submucosa and mucosa. The bladder is lined with transitional epithelium.

**Positioning**

Patients are positioned in dorsal recumbancy for retropulsion, cystostomy tube placement and routine cystotomy.

**Urethrostomy**

Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening that is large enough to accommodate passage of most urethral calculi, crystals and mucoid debris.

**Perineal urethrostomy; perineal approach**

The perineal urethra is the location of choice for urethrostomy in cats. It is a convenient location for surgical manipulation, the urethral diameter will accommodate passage of most urethral calculi and there is less urine scald postoperatively.

Prior to surgery a urethral catheter is passed, if possible. After a routine castration, an elliptical incision is made around the scrotum and penis. Then the subcutaneous tissues are dissected to expose penile urethra. The penile urethra is dissected free from surrounding connective tissue. The ventral attachment of the pelvic urethral to the pubis (i.e., ishiocavernosus m.) is identified and transected. The penile urethra is freed from its connective tissue attachments to the pelvic floor using blunt digital dissection. The retractor penis muscle is identified on the dorsal aspect of the penis and is dissected from its attachment on the penis. The dissected retractor penis muscle is then used to develop the dorsal plane of dissection to separate the pelvic urethra from its dorsal connective tissue attachments. Once the urethra is dissected enough to visualize the dorsolaterally located bulbourethral glands penile dissection can stop. The penis is catheterized and the urethral orifice identified. An incision is made from the penile urethra to the pelvic urethral to the level of the bulbourethral glands using a Stevens tenotomy scissor or Iris scissor. The urethral orifice at the level of the bulbourethral glands is generally of large enough diameter to accept the flange of a tomcat catheter.

After incision of the urethra, the glistening urethral mucosa is identified. 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author. The first urethrostomy suture is placed at the dorsal aspect of the urethrotomy incision on the right or left side at a 45o angle to include urethral mucosa and skin (suture split thickness of skin). The suture is tied and cut leaving the ends 3-4 cm long to act as a stay suture. A mosquito hemostat is placed on this suture to provide traction and countertraction to enhance visualization of the urethral mucosa. The second suture is placed opposite the first suture and tied as described for the first. A stay suture is also placed here. A third urethrostomy suture is placed directly on the dorsal midline to hold the dorsal margin of urethral mucosa to the dorsal margin of the skin incision. Alternating sutures from dorsal to ventral are
placed until approximately one half of the penile urethra has been sutured to skin. The remainder of the penis is amputated and the subcutaneous tissue and skin are closed routinely. Fine ophthalmic instruments make tissue handling and suturing easier. Use of a 2X magnifying loupe and headlamp light source enhances visualization of the urethral mucosa and facilitates accurate suturing. It is critical for the surgeon to recognize the glistening urethral mucosa and carefully suture it to skin. This will decrease (or eliminate) the chance of urethral stricture.

**Perineal urethrostomy; dorsal approach**

Perineal urethrostomy can be performed with the patient placed in dorsal recumbancy. This positioning is more ergonomic for the surgeon and allows easy access of the urinary bladder for concurrent cystotomy. When positioning the cat tie the hind limbs cranially until the pelvis is slightly elevated off the surgery table. Place a folded towel under the pelvis to support this slightly elevated position. The surgical technique is as described above for

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**Cystotomy**

See the DVD for a detailed video description of this procedure. After successful retropulsion of urethral calculi into the bladder the catheter used to retropulse calculi is passed into the urethra and bladder and left in place. Leaving a catheter indwelled in the urethra ensures that remaining cystic calculi will not roll back into the urethra during patient transfer to the surgery suite and during patient prep. The patient is placed in dorsal recumbancy with the hind legs tied gently cranially to slightly elevate the pelvis. A folded towel is placed under the pelvis to help support it in this position. This positioning will greatly facilitate exteriorizing the penis during surgery.

Just prior to aseptic preparation of the abdomen a soft, 5-8 French red rubber catheter or feeding tube is placed into the prepuce and a prepuceal lavage is performed using 20 cc of a 1:50 dilution of 1% betadine solution and sterile saline. This aseptically prepares the penis and prepuce so they can remain in the surgical field throughout the cystotomy procedure.

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A caudal midline incision is made from umbilicus to pubis. The bladder is exteriorized and examined. Stay sutures of 3-0 suture are placed in the apex and neck of the bladder. A scalpel blade is used to penetrate the ventral aspect of the bladder and enter the lumen. The ventral cystotomy incision is extended with Metzenbaum scissors. The bladder should be opened from apex to neck to allow proper visualization of bladder mucosa and easy retrieval of all calculi. Stay sutures are placed on each side of the incision at its midpoint to facilitate visualization of the bladder interior. Large hemostats are placed on the stay sutures to help retract the bladder margins. A cystotomy spoon is used to scoop the bladder neck for calculi. This is performed several times. When no more calculi can be removed with the spoon, digital palpation of the bladder neck is performed to identify presence of further calculi. If calculi are palpated further attempts are made to retrieve them. Once no more calculi can be spooned or palpated the indwelling urethral catheter placed after retropropulsion is removed.

Next, a 3.5 - 5 French urethral catheter is placed in the penile urethra (i.e., retrograde). A dry sponge is used to grasp the extruded penis to create a water tight seal around the catheter. A 35cc syringe filled with sterile saline is injected through the catheter under moderate pressure. The stay sutures on the bladder incision are retracted to enable visualization of the bladder lumen during lavage. Suction or intermittent spooning is performed during lavage in an attempt to identify and remove any remaining stones. After several high pressure lavages and negative results in obtaining stones, the catheter is placed from the bladder lumen into the bladder neck and pelvic urethra (i.e., normograde). Lavage is once again performed in an attempt to identify and remove any remaining stones. After several lavages and negative results, the catheter is advanced until it can be seen coming out of the penile urethra. The catheter is run back and forth in the urethra several times ('urogenital floss') to ensure there are no remaining calculi (i.e., gritty feeling while passing the catheter).

Finally, a piece of bladder mucosa is excised from the cystotomy incision for culture and susceptibility testing. The interior of the bladder is examined for urachal diverticulum, masses, etc. and biopsied as necessary. The bladder wall is closed with 3-0 or 4-0 absorbable monofilament suture material using a swaged on taper or taper-cut needle in a simple continuous or simple interrupted appositional suture pattern. Only one layer closure is necessary. Abdominal closure is routine.

**Suture material/special instruments**

Urinary catheters of various sizes, Foley catheter, head lamp light source, 2X loupes, ophthalmic instruments, 4-0 or 5-0 monofilament nonabsorbable suture material.

**Postoperative care and assessment**

Postoperative care varies depending upon procedure performed:

- Percutaneous cystostomy tube: It is important to keep the percutaneous cystostomy tube attached to a closed collection device. The tube can be connected to a sterile collection bag via a sterile intravenous catheter connection set. An elizabethan collar may be necessary in some patients to prevent iatrogenic removal of the cystostomy catheter. Careful management is important to control catheter related urinary tract infection.

- Cystotomy: An indwelling urethral catheter is not recommended after an uncomplicated cystotomy for removal of cystic calculi. An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals.

- Perineal Urethrostomy: An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals. An indwelling urinary catheter placed routinely postoperatively is NOT necessary following an uncomplicated urethrostomy.

**Prognosis**

The prognosis for surgical management of urethral and cystic calculi is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that have successful retropropulsion of urethral calculi and do not require urethrostomy have an excellent prognosis. If careful attention is paid during cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique), the prognosis for cure is excellent. Long term prognosis is dependant on evalaution of calculus composition, dietary management, management of urinary tract infection, and attention to urine pH.

Patients that have an elective perineal urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is too large to pass through the urethrostomy stoma.
Patients that sustain a traumatic injury severe enough to cause an abdominal hernia or patients that sustain penetrating abdominal wounds (i.e., gunshot, bite wounds) should have an exploratory laparotomy. After a review of general principles and techniques of abdominal surgery in cats, including the surgical techniques for ventral midline celiotomy, abdominal exploration, abdominal wall closure, and management of complications such as wound dehiscence, these proceedings outline the surgical management of traumatic abdominal wall hernias and peritonitis. Step by step descriptions of the surgical technique for repair of abdominal hernias are provided. For peritonitis, we will cover the indications and techniques for abdominocentesis, exploratory surgery, diagnostic abdominal lavage, and open abdominal drainage. In the lecture, case examples admitted to the author’s critical care service will be used to illustrate the surgeons' decision making techniques. Video of clinical case material will be used to illustrate all techniques.

If you would like a copy of the video of this surgical procedure on DVD go to www.videovet.org.

Key facts

- The abdomen generally is explored by means of a ventral midline incision from xyphoid to pubis. In most animals the entire abdomen, including the inguinal areas and the caudal thorax, should be prepared for aseptic surgery to allow extension of the incision into the thoracic or pelvic cavities if necessary.
- Various techniques can be used to systematically explore the entire abdomen; every surgeon should develop a consistent pattern to ensure that the entire abdominal cavity and all structures are visualized and/or palpated in each animal.
- Complications of abdominal surgery, including dehiscence (incisional hernias), may occur if improper surgical technique is used. The most common cause of wound dehiscence in the early postoperative period results from the surgeon’s inability to recognize the rectus sheath or not getting adequate bites in the collagen dense rectus sheath.
- Patients that sustain a traumatic injury severe enough to cause an abdominal hernia or patients that sustain penetrating abdominal wounds (i.e., gunshot, bite wounds) should have a xyphoid to pubis abdominal exploratory laparotomy.
- For most abdominal hernias, perform a ventral midline abdominal incision to allow the entire abdomen to be explored. Assess the extent of visceral herniation. Reduce the herniated contents and amputate or excise necrotic or devitalized tissue around the hernia. Close the muscle layers of the hernia with simple interrupted or simple continuous sutures.
- Abdominocentesis—the percutaneous removal of fluid from the abdominal cavity—usually is done for diagnostic purposes although it may occasionally be therapeutic. Indications include shock without apparent cause, undiagnosed disease with signs involving the abdominal cavity, suspicion of postoperative gastrointestinal dehiscence, blunt or penetrating abdominal injuries (i.e., gunshot wounds, dog bites, vehicular injury), and undiagnosed abdominal pain.
- Exploratory surgery is indicated when the cause of peritonitis cannot be determined or when bowel rupture, intestinal obstruction (e.g., bowel incarceration, neoplasia), or mesenteric avulsion is suspected.
- Although the practice of routinely lavaging the abdominal cavity of animals is controversial, lavage is always indicated with diffuse peritonitis. Lavage should be done with care in animals with localized peritonitis to avoid dissemination of infection.

General principles and techniques

Definitions

Celiotomy is a surgical incision into the abdominal cavity. The term laparotomy often is used synonymously, although it technically refers to a flank incision. A sudden onset of clinical signs referable to the abdominal cavity (e.g., abdominal distention, pain, vomiting) is called an acute abdomen.

Surgical techniques

The abdomen generally is explored by means of a ventral midline incision from xyphoid to pubis. In most animals the entire abdomen, including the inguinal areas, and the caudal thorax should be prepared for aseptic surgery to allow extension of the incision into the thoracic or pelvic cavities if necessary.

Ventral midline celiotomy in cats

With the patient in dorsal recumbency, make a ventral midline skin incision beginning near the xiphoid process and extending caudally to the pubis. Sharply incise the subcutaneous tissues until the external fascia of the rectus abdominis muscle is exposed. Ligature or cauterize small subcutaneous bleeders and identify the linea alba. Tent the abdominal wall and make a sharp incision into the linea alba with a scalpel blade. Palpate the interior surface of the linea for adhesions. Use scissors to extend the incision cranially or caudally (or both) to near the extent of the skin incision. Digitally break down the attachments of one side of the falciform ligament to
the body wall or excise it and remove it entirely if it interferes with visualization of cranial abdominal structures. Clamp the cranial end of the falciform ligament and ligate or cauterize bleeders before removing it.

**Abdominal exploration**

Systematically explore the entire abdomen. Various techniques may be used; however, every surgeon should develop a consistent pattern to ensure that the entire abdominal cavity and all structures are visualized and/or palpated in each animal.

Use moistened laparotomy sponges to protect tissues from drying during the procedure. If generalized infection is present or if diffuse intraoperative contamination has occurred, flush the abdomen with copious amounts of warmed, sterile saline solution with no additives (i.e., antiseptics or antibiotics). Remove the lavage fluid and blood and inspect the abdominal cavity before closure to ensure that all foreign material and surgical equipment have been removed. Perform a sponge count and compare it with the preoperative count to ensure that surgical sponges have not been left in the abdominal cavity.

**Abdominal wall closure**

The linea alba may be closed with a simple continuous (author’s preference) or a simple interrupted suture pattern. The simple continuous technique does not increase the risk of dehiscence when properly performed (i.e., secure knots, appropriate suture material, adequate bites in the rectus sheath), and it allows for a rapid and more secure closure. Synthetic monofilament absorbable suture (Maxon, PDS) should be used for continuous suture patterns, and six to eight knots should be placed at each end of the incision line.

On each side of the incision, engage a 5 to 7 mm bite of white rectus sheath with each suture. Place sutures no further apart than 3 to 4 mm, depending on the animal’s size. Tighten sutures sufficiently to appose but not enough to strangulate tissue, because sutures that strangulate tissue negatively affect wound healing. Incorporate full thickness bites of the abdominal wall in the sutures if the incision is midline (i.e., through the linea alba). If the incision is lateral to the linea alba and muscular tissue is exposed (i.e., paramedian incision), close the external rectus sheath without including muscle or peritoneum in the sutures. Close subcutaneous tissues with a simple continuous pattern of absorbable suture material and reappose the preputialis muscle fibers in the male dog. Use nonabsorbable sutures (simple interrupted or continuous appositional pattern) or stainless steel staples to close skin. Place skin sutures without tension.

**Complications**

Dehiscence (incisional hernias) may occur if improper surgical technique is used (see the above discussion). The most common cause of wound dehiscence in the early postoperative period results from the surgeon’s inability to recognize the rectus sheath or not getting adequate bites in the rectus sheath. Bites should engage at least 5 to 7 mm or more depending upon patient size.

**Traumatic abdominal wall hernias**

**Definitions**

External abdominal hernias are defects in the external wall of the abdomen that allow protrusion of abdominal contents; internal abdominal hernias are those that occur through a ring of tissue confined within the abdomen or thorax (i.e., diaphragmatic hernia, hiatal hernia). External abdominal hernias may involve the abdominal wall anywhere other than the umbilicus, inguinal ring, femoral canal, or scrotum.

**Surgical treatment**

Patients that sustain a traumatic injury severe enough to cause an abdominal hernia or patients that sustain penetrating abdominal wounds (i.e., gunshot, bite wounds) should have a xiphoid to pubis abdominal exploratory laparotomy. All visceral structures should be carefully examined to signs of trauma (e.g., mesenteric rents, ruptured hollow viscous organs, avulsed kidney, ureteral damage). In addition, abdominal celiotomy approach facilitates abdominal hernia closure. Most abdominal hernias can be repaired by suturing torn muscle edges or apposing the disrupted abdominal wall edge to the pubis, ribs, or adjacent fascia. In rare cases synthetic mesh must be used to repair the defect. Some hernias (i.e., intestinal strangulation, urinary obstruction, concurrent organ trauma) require emergency surgical correction. The extent of devitalized muscle may not be apparent initially, however, for patients in stable condition, delaying surgery until muscle damage can be accurately assessed facilitates surgical correction. The most common complications of surgery are hernia recurrence and wound infection. Abdominal hernias that occur secondary to bite wounds usually are contaminated; wound infection and dehiscence of the skin or hernial repair (or both) may occur. Mesh should not be placed in these hernias, hernial closure is performed during exploratory laparotomy, and the skin wounds should be left open to drain. Treatment of infected wounds includes cultures, drainage, antibiotics, and/or flushing.

**Positioning**

For ventral hernias the animal is placed in dorsal recumbency and the area around the hernia is prepared for aseptic surgery. Repair of ruptures of the cranial pubic ligament may be facilitated by placing the animal in dorsal recumbency with the rear limbs flexed and pulled cranially.
Surgical techniques

Abdominal hernias

For most abdominal hernias, perform a ventral midline abdominal incision to allow the entire abdomen to be explored. Assess the extent of visceral herniation. Reduce the herniated contents and amputate or excise necrotic or devitalized tissue around the hernia. Close the muscle layers of the hernia with simple interrupted or simple continuous sutures.

Cranial pubic ligament hernias

Make a ventral midline skin incision and identify the ruptured tendon and its pubic insertion. Evaluate the inguinal rings and vascular lacuna; these hernias may extend into the femoral region as a result of rupture of the inguinal ligament. Reattach the free edge of the abdominal wall to the cranial pubic ligament with simple interrupted sutures. As an alternative, suture the tendon remnant to the muscle fascia and peristemium covering the pubis or anchor it to the pubis by drilling holes in the pubic bone through which sutures can be placed. If the hernia extends into the femoral region, it may be necessary to suture the body wall to the medial fascia of the adductor muscles. When doing so, take care to avoid damaging the femoral vessels or nerves.

Prognosis

The prognosis generally is good, and recurrence is uncommon. When recurrence occurs, it generally is noted within a few days of surgery. Most animals have excellent long-term results when appropriate techniques are used.

Peritonitis

Definition

Primary generalized peritonitis refers to spontaneous inflammation of the peritoneum without any pre-existing intra-abdominal pathologic condition. Secondary generalized peritonitis occurs in conjunction with an intra-abdominal pathologic condition and may be further classified as infectious or noninfectious.

Surgical treatment

Abdominocentesis (see below) is the percutaneous removal of fluid from the abdominal cavity, usually for diagnostic purposes, although it may occasionally be therapeutic. Indications include shock without apparent cause, undiagnosed disease with signs involving the abdominal cavity, suspicion of postoperative gastrointestinal dehiscence, blunt or penetrating abdominal injuries (i.e., gunshot wounds, dog bites, vehicular injury), and undiagnosed abdominal pain. A multifenestrated catheter should be used to enhance fluid collection. Physical and radiographic examinations should precede abdominocentesis to rule out instances in which it may not be safe and to guide needle placement. Four-quadrant paracentesis may be performed if simple abdominocentesis is not successful in retrieving fluid. It is similar to simple abdominocentesis except that multiple abdominal sites are assessed by dividing the abdomen into four quadrants through the umbilicus and tapping each of these four areas. Diagnostic peritoneal lavage should be performed in animals suspected of having peritonitis if the above methods are unsuccessful in obtaining fluid for analysis.

Exploratory surgery is indicated when the cause of peritonitis cannot be determined or when bowel rupture, intestinal obstruction (e.g., bowel incarceration, neoplasia), or mesenteric avulsion is suspected. Serosal patching and plication reduce the incidence of jejunal leakage, dehiscence, or repeated intussusception. Animals that require surgery and that have peritonitis secondary to intestinal trauma (disruption of mesenteric blood supply, bowel perforation, chronic intussusception, foreign body) often are hypoproteinemic. The role that protein levels play in healing intestinal incisions is not well understood. However, most surgeons are concerned that hypoproteinemic patients may not heal as quickly as patients with normal protein levels, despite one study that showed similar complication rates among animals with normal protein levels and those that were hypoproteinemic and undergoing intestinal surgery. Most experimental evidence has shown that retardation of wound healing is not seen with moderate protein depletion but only with severe deficiencies (<1.5 to 2 g/dL).

Although the practice of lavaging the abdominal cavity of animals with peritonitis is controversial, lavage generally is indicated with diffuse peritonitis. Lavage should be done with care in animals with localized peritonitis to avoid dissemination of infection. When lavage is performed, as much of the fluid as possible should be removed because fluid inhibits the body’s ability to fight off infection, probably by inhibiting neutrophil function. Historically, many different agents have been added to lavage fluids, especially antiseptics and antibiotics. Povidone-iodine is the most widely added antiseptic; however, its use may be contraindicated with established peritonitis. Furthermore, no beneficial effect of this agent has been shown in repeated experimental and clinical trials in animals. Although a great many antibiotics have been added to lavage fluids over the years, there is no substantial evidence that their addition is of any benefit to patients who are being treated with appropriate systemic antibiotics. Warmed sterile physiologic saline is the most appropriate lavage fluid.

Positioning

For abdominocentesis and diagnostic lavage, the abdomen should be clipped and prepared aseptically. These procedures may be performed with the animal in lateral recumbency or standing.

Abdominocentesis

Insert an 20- or 22-gauge, 1-inch plastic over-the-needle catheter (with added side holes) into the abdominal cavity at the most dependent part of the abdomen. Do not attach a syringe; instead allow the fluid to drip from the needle and collect in a sterile tube. If
sufficient fluid is obtained, place it in a clot tube and an ethylenediamine tetraacetic acid (EDTA) tube, submit samples for aerobic and anaerobic culture, and make four to six smears for analysis. If fluid is not obtained, apply gentle suction using a 3-mL syringe.

It is difficult to puncture bowel by this method because mobile loops of bowel move away from the tip of the needle as it strikes them. Perforations created by a needle this size usually heal without complications. The major disadvantage of needle paracentesis is that it is insensitive to the presence of the small volumes of intraperitoneal fluid and thus a negative result can be meaningless. At least 5 to 6 mL of fluid per kilogram of body weight must be present in the abdominal cavity of dogs to obtain positive results in most cases using this technique.

**Diagnostic peritoneal lavage**

Make a 2-cm skin incision just caudal to the umbilicus and ligate any bleeders to avoid false-positive results. Spread loose subcutaneous tissues and make a small incision in the linea alba. Hold the edges of the incision with forceps while the peritoneal lavage catheter (Stylocath) without the trocar is inserted into the abdominal cavity. Direct the catheter caudally into the pelvis. With the catheter in place, apply gentle suction. If blood or fluid cannot be aspirated, connect the catheter to a bottle of warm sterile saline and infuse 20 mL/kg of fluid into the abdominal cavity. When the calculated volume of fluid has been delivered, roll the patient gently from side to side, place the bottle on the floor, vent it, and collect the fluid by gravity drainage. Do not be surprised if you do not retrieve all of the fluid, particularly in dehydrated animals.

**Exploratory laparotomy**

Perform a ventral midline incision from the xiphoid process to the pubis. Obtain a sample of fluid for culture and analysis. Explore and inspect the entire abdomen. Find the source of infection and correct it. Break down adhesions that may hinder drainage. Lavage the abdomen with copious amounts of warm, sterile saline if the infection is generalized. Remove as much necrotic debris and fluid as possible. Close the abdomen routinely, place an abdominal drain, or perform open abdominal drainage.

**Prognosis**

The prognosis for animals with generalized peritonitis is guarded; however, with proper and aggressive therapy, many survive. Some authors have suggested that the mortality rate approaches 50%. The mortality rates reported in animals with generalized peritonitis treated with open abdominal drainage have varied from 20% to 48%.
Ruptured bladder
Trauma to the urinary bladder is relatively common in veterinary patients. It often results in uroperitoneum (uroabdomen) that is associated with severe metabolic and multisystemic disturbance which can be fatal if not treated urgently but appropriately.

Blunt abdominal trauma (vehicular is the most common cause) and direct injury from pelvic fractures are the most common reasons for injury to the bladder in dogs. In cats, blunt abdominal trauma, injury during catheterization and rupture during bladder palpation are the most common causes of urethral and bladder rupture. Other reasons include urethral obstruction, erosive neoplastic lesions, or penetrating abdominal wounds. The most common site of urinary tract trauma is the bladder. The apex is most often the site of rupture although any part of the bladder can be affected especially when pelvic fractures are the cause.

Patients with a ruptured bladder often do not show clinical signs immediately after injury. However over the subsequent 24-48 hours patients often become dehydrated and begin to develop metabolic and electrolyte disturbances that can become severe and life threatening. As urine accumulates in the peritoneal space a chemical peritonitis ensues which, if sterile, is not immediately life-threatening but causes abdominal pain and ileus. Urine is hyperosmolar to the extracellular fluid. This results in a net flux of fluid across the peritoneal membrane. Third-spacing of fluid in the peritoneal cavity along with decreased intake and often increased losses due to vomiting leads to severe dehydration. Hyperkalemia and azotemia develop as peritoneal potassium and urea equilibrate rapidly with extracellular fluid. Metabolic acidosis often develops due to decreased excretion of hydrogen ions in urine and progressively worsening hypovolemic shock.

Rapid diagnosis of urinary tract injury is vital. Aggressive emergency management of associated metabolic abnormalities to stabilize the patient should be performed prior to definitive surgical repair.

Diagnostic criteria

History
Gender predisposition: In cats there is no sex predisposition. Male dogs are predisposed to traumatic bladder rupture because the longer, less distensible male urethra is more able to withstand elevated intravesicular pressure. There are no known age or breed predispositions.

Physical examination findings
Lethargy, anorexia, dehydration, abdominal pain, ascites, hypothermia, other signs of traumatic injury

Laboratory findings
Azotemia, hyperkalemia, metabolic acidosis, hyperalbuminemia, increased hematocrit, neutrophilia

Fluid analysis: Samples of abdominal fluid can be obtained by abdominocentesis or diagnostic peritoneal lavage.

Collection of Peritoneal Fluid Sample:
Abdominocentesis – this technique is successful in the majority or cases.
The patient is positioned in lateral recumbency
An area is clipped and aseptically scrubbed along the ventral midline approximately 10x10cm
Insert a 20 or 22 gauge 1½ inch needle on a 3 or 6ml syringe 1cm caudal to the umbilicus and just off the midline (avoiding the falciform ligament)
Aspirate gently and collect samples for fluid analysis, cytology and microbial culture and sensitivity testing

Multiple quadrant abdominal taps can be performed if the above is unsuccessful
If one site yields a fluid sample the other taps are abandoned.

The fluid recovered can be a transudate, modified transudate or exudate depending on the chronicity and whether concurrent sepsis is present. Comparison of creatinine and potassium concentrations in peritoneal fluid and serum are the most reliable tests for confirming uroabdomen in cats. Because of the large molecular size of creatinine it diffuses only slowly across the peritoneum into the extracellular fluid. A significant gradient is established between abdominal fluid and serum, detection of which is a sensitive and specific test for uroabdomen. A similar gradient exists with potassium. Patients with creatinine and potassium levels in their abdominal fluid that are slightly to markedly higher than in their serum are very likely to have a uroabdomen. Reported ratios of abdominal fluid to serum creatinine concentrations in cats is a mean of 2:1. The same ratio for potassium in cats is a mean of 1.9:1

Plain abdominal radiography: The bladder may or may not be visible in patients with bladder rupture as small leaks will still allow distension of the bladder to some degree. Loss of abdominal detail will occur due to fluid accumulation which will worsen with time. Evidence of an underlying cause may be obvious such as pelvic fractures or cystic or urethral calculi.

Contrast radiography: Care should be taken administering contrast agents to dehydrated or azotemic patients as renal insult can result. Patients should be fully stabilized before undergoing these studies. Positive contrast retrograde urethrocystography is the
contrast study of choice in patients with bladder rupture and should be the first radiographic study performed. It is easy to perform and allows confirmation of the diagnosis and location of the site of leakage in the lower urinary tract in most cases. Fluoroscopic visualization during contrast injection is helpful as it aids in early detection of the site of leakage. If not available plain radiography can confirm leakage but dispersal of contrast material may obscure the origin of the leakage somewhat. If sufficient intravesicular pressure is not achieved during contrast injection false negative results may be seen especially with small tears and in unusual cases where the lesion has self sealed. As leakage of urine from the upper urinary tract cannot be detected with a retrograde cystourethrogram an intravenous urogram should be performed especially if no lesions were found during the first study.

Abdominal ultrasonography: Ultrasound examination should detect fluid accumulation in the peritoneal space and can be used to guide abdominocentesis. It may also help identification of underlying bladder pathology, calculi and other possible causes of uroabdomen such as renal, ureteral and urethral lesions.

Differential diagnoses
Leakage or urine from a location other than the bladder can usually be ruled out with contrast studies of the upper and lower urinary tract such as a retrograde urethrocystogram and an intravenous urogram.

Other causes of acute abdomen can usually be ruled out by abdominocentesis, radiographic and/or ultrasonographic examination.

Treatment recommendations
The aim of initial treatment is pre-surgical patient stabilization. Principal areas of concern are azotemia, electrolyte and acid-base disorders, and cardiac arrhythmias that result from severe hyperkalemia.

Drainage of urine from the abdomen is the next most important step. This can be achieved with a percutaneous placement of a peritoneal lavage catheter (feeding tube, Jackson Pratt drain, etc). Use of an indwelling urethral catheter or tube cystostomy is helpful in decreasing the amount of urine entering the peritoneal space from the bladder.

Surgical management is ultimately required in most cases. However, uroabdomen is a medical and not a surgical emergency. Patients operated on prior to adequate stabilization are likely to experience life threatening intraoperative and post operative complications.

Initial treatment
Intravenous fluid therapy: Fluids should be administered upon admission. An isotonic saline solution (0.9% NaCl) is the fluid of choice. Volume of fluid given is judged by degree of hypovolemia and is made on a case by case basis.

Treatment of hyperkalemia: Mild hyperkalemia will often resolve with fluid diuresis alone. More severe hyperkalemia (> 7mEq/L) may be associated with cardiotoxicity and specific treatment should be considered.

Cardiac monitoring: Continuous ECG monitoring is recommended. Most cardiac abnormalities are related to hyperkalemia and will resolve once normokalemia is re-established. Typical changes are absent or flattened P waves, prolongation of the P-R interval, widened QRS complexes, spiked T waves and bradyarrhythmias. ECG abnormalities however are not consistent and it should not be assumed that hyperkalemia is absent if the ECG is normal or vice versa.

Analgiesia: Pain relief should be instituted early as chemical peritonitis is very painful. Opioid analgesics are most commonly used such as morphine, hydromorphone, or buprenorphine.

Antibiotics: Intravenous antibiosis should be instituted. A first generation cephalosporin such as Cefazolin is an appropriate empirical choice.

Surgical management
Exploratory Laparotomy: A xyphoid to pubis exploratory laparotomy is performed. Patients that present with a ruptured bladder have sustained enough trauma to result in concurrent visceral organ injury thus a complete abdominal exploratory is recommended. Any rents in the bladder are identified and the area debrided of necrotic or damaged tissue. The bladder is sutured with one layer simple continuous or simple interrupted suture pattern using a synthetic absorbable suture (such as 3-0 or 4-0, Capryson, Monocryl, Biosyn Dexon, Vicryl, Polysorb) in an appositional pattern. No attempt is made to invert the incision. Copious lavage of the peritoneal cavity is performed with body temperature sterile physiologic saline solution followed by routine abdominal closure. If bladder wall integrity is of concern post-operatively an indwelling urethral catheter can be left in place for 24-48 hours to allow decompression but is not mandatory.

Supportive treatment
Intravenous fluid therapy with an isotonic saline solution should be administered post-operatively depending on the patient’s hydration status. This should be maintained until the patient is drinking.

Antibiotic therapy: If the abdominal effusion was sterile it is not necessary to continue antibiotic therapy beyond the intraoperative period. However if septic effusion was detected antibiotic therapy based on the results of culture and sensitivity should be continued for at least two weeks.

Analgiesia: Appropriate opioid analgesia should be continued post-operatively for at least 48 hours.

Gastric protectants: Uremic gastritis may cause vomiting and ulceration. Treatment with an H2 receptor antagonist or proton pump blocker should be considered.

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Patient monitoring
Repeat serum biochemical analysis should be performed post-operatively to demonstrate resolution of azotemia, electrolyte imbalances and acid-base abnormalities.

Urinary leakage post-operatively is a potential complication and should be diagnosed promptly if present. Failure to recover from surgery uneventfully, persistence of azotemia or hyperkalemia and recurrence of abdominal distension should alert the clinician to a possible problem.

Continuous ECG monitoring should be performed until complete resolution of all cardiac abnormalities.

Prognosis
In general prognosis after bladder rupture is excellent if diagnosis and treatment are prompt.

Favorable criteria
Simple rents in the apex of the bladder are easy to close. In cases where bladder wall damage is extensive or in ruptures secondary to avascular necrosis of the bladder wall prognosis is less favorable.

Patients systemically stable prior to surgery that can be taken promptly to surgery are likely to make an excellent recovery.

Unfavorable criteria
- Surgical intervention prior to reversal of the patients metabolic derangement.
- Presence of septic peritoneal effusion.
- Significant delay in time to diagnosis and treatment may adversely affect outcome.
- Severe concurrent traumatic injuries.
Surgical Repair of Diaphragmatic Hernia
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Key points
- Most diaphragmatic hernias are not acutely life threatening
- Suture the hernial rent from dorsal to ventral
- Use a one layer simple continuous appositional suture pattern for closure
- Evacuate all thoracic air prior to closure

General considerations and indications
Three classifications of diaphragmatic hernia may be diagnosed; acute traumatic, chronic traumatic and congenital diaphragmatic hernia.

Acute traumatic
This is the most common type of diaphragmatic hernia in dogs and cats. It is generally caused by vehicular trauma but can be caused by any form of trauma.

Chronic traumatic
This classification of diaphragmatic hernia is seen when a patient has an acute traumatic hernia that was undiagnosed at the time of occurrence. Later (months to years) the hernia is diagnosed due to sudden or chronic onset of respiratory difficulty.

Congenital
The most common congenital hernia involving the diaphragm is a peritoneal-pericardial diaphragmatic hernia. Whenever this defect is suspected, a thorough examination (i.e., physical, radiographic, cardiovascular) for evidence of further midline congenital defects (i.e., umbilical hernia, atrial and ventricular septal defects, cleft palate) should be performed.

Applied anatomy
The diaphragm projects into the thoracic cavity like a dome; it attaches to the lumbar vertebrae, costal arch, and sternum. Fibers arise on these skeletal parts and radiate towards the tendinous center. The diaphragm is composed of only one layer of muscle and two layers of tendon and therefore is weaker than the multilayered abdominal wall. The central tendon of the diaphragm of the cat is relatively small. In its tendinous portion, transverse fibers course from one side to the other as a reinforcing apparatus.

The muscular part is divided into the pars lumbalis, a pars costalis on each side, and the pars sternalis, all of which with the exception of the lumbar portion, have a uniform thickness of 2-3 mm in cats. The pars lumbalis of the diaphragmatic musculature is formed by the right and left diaphragmatic crura, the right crus being considerably larger than the left. Seen from the abdominal cavity each crus of the diaphragm is a triangular muscular plate whose borders give rise to the tendinous portions. The pars costalis on each side consists of fibers radiating from the costal wall to the tendinous center. The pars sternalis is an unpaired medial part unseparated from the bilateral costal portions.

The diaphragm domes far into the thoracic cavity, and its costal part lies on the medial surface of the last few ribs and costal arch (when tears occur here, the costal arch can be used in the repair). The stomach and liver attach by ligaments to the concave peritoneal surface of the diaphragm.

Diagnosis
Diaphragmatic hernia is generally diagnosed via thoracic and abdominal radiographs. Classic findings on thoracic radiographs is loss of the diaphragmatic line, air filled visceral structures in the thoracic cavity, loss of lung fields. Abdominal radiographs may reveal a lack of abdominal viscera. Classic thoracic radiographs of a patient with a peritoneo-pericardial diaphragmatic hernia shows a large, round pericardial sac. Occasionally, air filled viscera can be identified in the pericardial sac. Patients that present with an acute traumatic diaphragmatic hernia (e.g., hit by a car) may have a massive hernia with abdominal contents replacing most of the patients respiratory capacity.

Preoperative considerations
Immediate surgical intervention for the repair of a diaphragmatic hernia is rarely indicated. Emergency surgery should not be undertaken unless the surgeon and anesthesiologist are prepared to handle any complications and are confident they can maintain the animal's essential requirements while the animal is anesthetized. However, prompt surgical repair is indicated in acutely injured animals with severe dyspnea, cyanosis, and respiratory distress who demonstrate massive herniation, and in patients that present with an air filled stomach in the thoracic cavity (these patients can develop life threatening dyspnea if enough swallowed air enters the stomach).

The most commonly encountered patient with diaphragmatic hernia will fall between the two categories mentioned above and should be handled in a systematic manner that will not further compromise the patients already reduced breathing ability. Surgery is
not considered an emergency in mildly symptomatic or asymptomatic animals with congenital hernias or traumatic hernias of at least several days' duration. Remember that any stressed, dyspneic cat should be handled very carefully as further stress can produce catastrophic results.

**Anesthesia**
Patient stress must be kept to a minimum during the anesthetic induction phase as any exertion by the animal can be disastrous.

**Surgical approaches**
A midline abdominal celiotomy (xiphoid to pubis) is the easiest and most versatile approach. Positioning the patient's head toward the top of the table and tilting the table at a 30° to 40° angle will facilitate gravitation of abdominal viscera out of the thorax. Rarely is it necessary to extend the incision into the thorax via a median sternotomy however the animal should be prepared in case this becomes necessary.

**Surgical procedure**
See the DVD for a detailed video description of this technique. When an extra pair of hands is unavailable for retraction, a Balfour self retaining retractor is a helpful piece of equipment; large Gelpi retractors work well in cats. Using the abdominal approach, an incision is made from xiphoid to pubis. Once the peritoneal cavity is opened, the diaphragm is exposed and the situation evaluated. Some hernias, especially in the area of the dorsal attachments of the crura and the aortic hiatus are not easily visualized; therefore, this area should be carefully inspected even when another laceration is present. The herniated contents are replaced in their proper position and inspected for damage.

Using large sponges or laparotomy pads moistened with warm saline, the liver and bowel are retracted caudally. Visualization of the cranial quadrant of the abdomen can be facilitated by removing the viscera from the abdominal cavity and placing it on a moistened laparotomy sponge. The diaphragmatic tear is now more easily visualized so that a careful examination of the thorax can be done both visually and manually. All thoracic fluid should be aspirated.

In acute traumatic diaphragmatic hernia, the lungs should be expanded to remove atelectasis and to inspect for pulmonary tears and persistent areas of collapse.

In chronic traumatic hernias care is taken not to inflate the lungs. When lung parenchyma is atelelectatic for such a long period of time the alveoli collapse. If they are suddenly expanded with air the tight junctions of the normal alveoli are damaged and the inflated alveolus fills with fluid. This is referred to as re-expansion pulmonary edema. This is a life threatening disorder and should be avoided.

It is recommended to suture the hernia from dorsal to ventral thus making it much easier to visualize the dorsal structures (vena cava, aorta, esophagus) when suturing. The hernia is closed with a single layer, simple continuous suture pattern using synthetic absorbable suture material (Dexon, Vicryl, Biosyn PDS, Maxon) or monofilament nonabsorbable suture material (Nylon, Prolene, Novafil). Suture size recommended in cats is 3-0. It might be necessary to preplace the most dorsal sutures for better visualization of the tear during suturing. It is also helpful to reconstruct the tear with several simple interrupted sutures to facilitate visualization of the rent. When tears near the caval hiatus are sutured, care is taken to avoid constriction of the vena cava by placing sutures to close to the cava. The same principle applies to the aortic and esophageal hiati.

Air can be evacuated from the chest using several techniques.

1. Prior to tying the last knot of the hernial closure, a carmalt forceps is placed in the hernial rent between two sutures and gently spread open to allow access to the thoracic cavity. The lungs are inflated so as to fill the thoracic cavity. The carmals are removed and the last suture tied to provide an air tight and water tight seal.
2. After hernial rent closure a needle or plastic intravenous catheter is placed through the diaphragm and into the thoracic cavity. Thoracic cavity air is evacuated using a syringe.
3. Needle thoracentesis is performed after the procedure is complete.
4. A 12 - 14 French feeding tube is brought into the peritoneal cavity through a paramedian stab incision in the cranioventral body wall. The tube is passed through the diaphragmatic rent between to sutures just prior to its final closure. Make certain that all fenestrations in the tube are beyond the diaphragm. The diaphragmatic rent closure is then completed around the tube. With the use of a 3-way stop cock and 60 cc syringe, air is evacuated from the thorax until a gentle negative pressure is obtained. The celiotomy incision is closed in a routine fashion. When the celiotomy closure is complete, the tube is again aspirated. The patient should then be placed through a series of positional changes (ventral recumbency, right lateral recumbency, left lateral recumbency, and dorsal recumbency) while attempting to aspirate air. When negative pressure is obtained in all positions, the tube is gently pulled from the chest and abdominal incision.
5. A 12 -14 French diameter thoracostomy tube can be placed at the level of the 10th or 11th intercostal space, tunneled to the level of the 7th or 8th intercostal space and placed through the intercostal muscle and into the thoracic cavity. The
patient is then placed through a series of positional changes (ventral recumbency, right lateral recumbency, left lateral recumbency, and dorsal recumbency) while attempting to aspirate air. The tube is removed when the patient has had a negative pressure for 12 - 24 hours.

All patients are monitored carefully for the next six to eight hours. If signs of respiratory abnormalities arise (dyspnea, tachypnea, etc), the right and left hemithorax should be tapped with a needle and syringe.

**Postoperative care**

Post-surgical care includes systemic antibiotics and careful monitoring of the patient's breathing, temperature, and color. Cats should be kept on a warming device for at least 24 hours. Analgesics may be used to relieve patient discomfort, however care should be taken to monitor the effects of various analgesic drugs on respiratory effort. Thoracic radiographs may be taken to evaluate the chest drain and pleural space.

**Summary**

Successful repair of a diaphragmatic hernia depends on careful preoperative and postoperative care of the patient. During the surgical repair, the surgeon must work quickly and effectively to complete the procedure as efficiently as possible.
The feline species may generally be considered to be a solitary creature where social interactions are concerned, but domestication has allowed the adaptation to living in social communities with both humans and other cats. Domestic cats are well known to be able to develop bonds with humans and other cats. The ability to socialize with other cats and humans will vary between individual cats as a function of their genetics, prenatal environment, early socialization and life experiences. Kittens that are handled by and interact with humans in their first few weeks of life are friendlier and less fearful of people. In contrast to this, some studies have suggested that hand-reared kittens tend to show more aggression. When available, understanding the cat’s socialization history may be beneficial in rooting out some of the underlying issues.

The causes of feline aggression towards humans can be multifold. These include play, territorial, fear and redirected aggression. Play aggression is a common concern amongst cat owners, and many times the predisposing factors can be identified through careful behavioral questioning at kitten examination visits. Hand play is probably one of the most common concerns the author identifies when discussing play aggression with clients. Many kittens and cats showing play aggression towards their owners actively engage in hand play, with encouragement from the owner. Use of toys, not hands and feet, and the avoidance of any hand ‘wrestling’ will help to some play aggression concerns. During every kitten visit, appropriate play should be discussed, with a firm recommendation to avoid any hand/feet play. Kittens and cats are not able to distinguish degrees of acceptable behavior. If the client makes hand play acceptable, then to the kitten, all body parts are ‘fair game’. Environmental enrichment and structured play times are beneficial in reducing play aggression.

Territorial aggression may be directed at new humans in the household, existing humans posing a threat to resources (this is rare) or may lead to redirected aggression where the human has become a new target during a territorial dispute. For example, cats may be anxious and feel territorial towards visible outdoor cats and turn this aggression inadvertently onto a human in the household.

Fear is a common reason for aggression towards humans. The cat may be fearful of the owner because of an incident that frightened the cat that was associated with the human in question. For example, a large item falling and crashing down causing a loud noise may have frightened the cat. The cat then associated the incident with the human that was nearby when the incident occurred. Unfortunately cats may experience abuse or aggressive treatment in their lifetime, either at the hands of a previous human or by the current owner. These experiences may leave the cat with a persistent fear against humans in general, the specific human causing the abuse, or humans with similar characteristics to the abuser (ex. men).

Redirected aggression is a complex problem that can occur for many reasons. The notable example is that described above under territorial aggression. In cases of redirected aggression, it can be doubly hard to identify the inciting cause or incident. These triggers must be identified, as avoiding them will be the mainstay to avoiding further episodes and increasing the chance of treatment success.

Behavioral questions should be a normal part of every feline visit from preventive care visits to sick cat visits to behavioral consultations. Clients will not always reveal their concerns unless asked. Even in cases where there are no concerns, the clinician might be able to identify potential triggers that may lead to future concerns (ex. hand play with kittens). When a client approaches the veterinary team for help with a behavioral problem, immediate and full support should be offered. The first step is to assess the cat or cats in question and ensure that no medical concerns exist. A full behavioral history should be obtained. The behavioral consultation can be conducted in clinic, or at the client household. The latter is a more favorable option, as it allows the clinician to directly observe the layout of the home and where incident(s) occur(red). It also allows the clinician to assess the environmental enrichment and determine the availability of appropriate resources.

Goals for the behavioral interview and development of a treatment plan

1. Identify underlying motivation for the behavior
2. Identify triggers and formulate a plan on how to avoid these
4. Develop a treatment plan

Questions to ask during the behavioral interview

Basic information should be gathered about the patient signalment, household members (people and animals), patient medical history and how/where the pet was acquired. The environment should be reviewed, including resource management and availability, litter box care, and household enrichment. The patient’s daily activities should be reviewed and in cases of house calls, traced throughout the house layout. Relationships with other pets in the house and with humans in the household should be reviewed. The stability of the human population should be assessed. For example, humans that work shift work hours may be home inconsistently, causing the
patient’s environment to be inconsistent. Family children that attend college or university may come and go every 4 months, creating an unstable social circle.

The incident considered the initiating incident, as well as further incidents should be reviewed in detail. There should be an examination of the frequency, intensity and severity of the behavior(s) in question. The behavior of both the cat and the response by the human should be determined. Any attempts at treatment or punishment should be reviewed as these may negatively impact the prognosis.

**Points to consider with regards to the potential success of treatment**

Cases of aggression towards humans can be devastating to the human animal bond. A frank, open discussion is necessary with the client(s) in order to determine the prognosis for the patient. It is important to determine how weakened or broken the human-animal bond has become as a result of the behavioral issues. The clinician will need a frank admission by the client about how willing they are to implement the outlined treatment plan. The clients will need to be open about what options they are considering. Options may include following the prescribed treatment plan, drugs, relinquishment and/or euthanasia. Further, the clients’ expectations must be known. What are the clients’ goals and timeline? The clients may have unrealistic expectations, desiring complete resolution of the problem.

**Prognosis**

The ability to resolve a human-directed aggression is going to depend on many factors. First and foremost, it is going to depend on the points noted above, as to what client expectations are and whether they are willing to follow prescribed treatment plans. If this is not the case, then the chances of success diminish greatly. Secondly, the duration of the problem, and third, it’s severity, will impact the chance to implement change. Cats showing aggression towards a specific human may never achieve complete resolution of their issues, and a life long alteration of the household dynamics, as well as lifelong medications may become a necessity. Setting realistic expectations and realistic time frames for achieving goals early on in the consultation process is more likely to set the stage for success or at least partial resolution. Clients and clinicians should understand the limitations of the problem and what is reasonable to expect from treatment.

**Sample questionnaire for a home behavior consultation**

Patient ID:

Patient Signalment:

**Medical conditions**

1. 
2. 
3. 

**Current medications**

1. 
2. 
3. 

**What amount of day does cat spend in the following activities?**

Sleeping
Resting
Eating
Grooming
Hiding
Playing Alone
Playing with human
How often does cat use litter box for BM? U?

**Other pets?**

If Yes, details:

**Environment**

Layout- rough drawings attached

**Litter boxes**

Number
Location
Characteristics

**Feeding stations**

Number
Location
Characteristics
Feeding schedule
Food
Amount
Timing

Private locations/Hide spots
Number
Location
Used?

Elevated locations/3D space availability
Number
Location
Used?

Windows
Location
Look out onto:

Other environmental enrichment

Humans in household
Number
Stability of residence
Stability of employment hours:
Relationship with Cat:
Other regular visitors:

Potential external stressors noted outdoors
Unique considerations (ex. flooring type, smoking, cleanliness, clutter, noise levels

Specific incidents reported
Known Triggers of Aggression:
Aggression directed towards:
Client/Individual reaction to aggressive behavior:
Any punishments used?
Yelling, swatting, other
Veterinarian Interactions with Cat during visit:

References
Feline Chronic Kidney Disease: A Practical Approach to IRIS Staging
Kelly St. Denis, DVM, DABVP
Charlton Cross Cat Clinic
Brantford, ON

Chronic renal disease & IRIS staging
Decline in kidney function can result from a variety of causes including pyelonephritis, amyloidosis, polycystic kidney disease, neoplasia, nephrotoxicosis, hydronephrosis and chronic glomerulonephritis (Scherk, 2011). Although acute insult can lead to chronic kidney disease (CKD), age seems to be the only major, consistent risk factor associated with chronic renal insufficiency (White, 2011).

Mature cat visits ideally include a complete physical examination/consultation as well as data collection in the form of a minimum database (MDB) every 4 to 6 months. A minimum database for mature cats includes a full clinical chemistry, a total thyroid test (TT4), a complete blood count, a urinalysis and a blood pressure (BP) series. Blood urea nitrogen (BUN) and creatinine have traditionally been the go-to serum values for diagnosis of kidney disease. Early diagnosis can be challenging utilizing only these values, as azotemia does not develop until there is 75% loss of nephron function. The BUN can be influenced by factors other than renal disease, including dehydration, dietary protein content, gastrointestinal bleeding and hepatic insufficiency. Creatinine is a more reliable indicator of glomerular filtration rate (GFR). However, creatinine can be influenced by muscle wasting and by dehydration. Routine screening of these values can assist the clinician in documenting upward trends in these values.

Symmetrical dimethylarginine (SDMA) measures the methylated form of the amino acid arginine. This is a by-product of protein degradation excreted by the kidneys. SDMA increases with about 40% loss of kidney function. It can be impacted by dehydration. Symmetrical dimethylarginine is not a stand-alone test and should always be interpreted in light of patient status as well as other laboratory findings. Elevated SDMA in the absence of any other evidence of renal disease should be re-evaluated.

Table 1 Urine specific gravity varies with age & diet (Scherk, 2011)

<table>
<thead>
<tr>
<th>Age or condition</th>
<th>Expected USG</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-8 weeks of age</td>
<td>1.020-1.038</td>
<td></td>
</tr>
<tr>
<td>8+ weeks of age</td>
<td>Up to 1.080</td>
<td>Denotes age at which full concentrating ability is reached</td>
</tr>
<tr>
<td>Dehydrates/normal renal function</td>
<td>&gt;1.040</td>
<td>Diet dependent (wet vs dry)</td>
</tr>
<tr>
<td>Canned food only</td>
<td>&gt;1.025</td>
<td></td>
</tr>
<tr>
<td>Dry food only</td>
<td>&gt;1.035</td>
<td></td>
</tr>
<tr>
<td>Inability to concentrate urine</td>
<td>1.008-1.012</td>
<td>Nephrons no longer able to modify glomerular filtrate</td>
</tr>
<tr>
<td>Dehydrated/unknown renal function</td>
<td>1.007-1.039</td>
<td>Suggestive of renal insufficiency with or without azotemia</td>
</tr>
</tbody>
</table>

It is recommended that urine samples be collected by cystocentesis and tested immediately in the clinic laboratory. Urine testing should include chemistry testing using testing strips, measurement of urine specific gravity (USG) by refractometer and sediment analysis. Urine specific gravity can be impacted by age, diet and hydration status. Urine specific gravity varies throughout the day, such that a single low USG is not reliable evidence of a loss of concentrating ability (Scherk, 2011). Samples with a low urine specific gravity (USG; less than 1.035) should be submitted for culture.

International renal interest society (IRIS)
For cats that are diagnosed with CKD, it is critical for practitioners to develop and promote a relationship with clients that will allow continued monitoring of the disease, including disease staging. The application of human IRIS staging guidelines to the study of feline renal disease has dramatically advanced our ability to tailor our patient therapy, thereby improving quantity and quality of life. In addition to the MDB as discussed above, imaging is likely to be beneficial.

Table 2. IRIS staging guidelines

<table>
<thead>
<tr>
<th>Stage</th>
<th>Renal Azotemia</th>
<th>Creatinine</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Non-azotemic</td>
<td>&lt;140 µmol/L</td>
<td>Absent</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>140-249 µmol/L</td>
<td>Mild or absent</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>250-439 µmol/L</td>
<td>Moderate</td>
</tr>
<tr>
<td>4</td>
<td>Severe</td>
<td>&gt;440 µmol/L</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Adapted from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis
Table 3. Subclassifications of IRIS staging: Proteinuria

<table>
<thead>
<tr>
<th>Urine Protein:Creatinine Ratio (UPC)</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.2</td>
<td>Non-proteinuric (NP)</td>
</tr>
<tr>
<td>0.2-0.4</td>
<td>Borderline proteinuric (BP)</td>
</tr>
<tr>
<td>&gt;0.4</td>
<td>Proteinuric (P)</td>
</tr>
</tbody>
</table>

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis

Table 4. Subclassifications of IRIS staging: Blood pressure

<table>
<thead>
<tr>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150</td>
<td>&lt;95</td>
<td>Minimal risk (N)</td>
</tr>
<tr>
<td>150-159</td>
<td>95-99</td>
<td>Low Risk (L)</td>
</tr>
<tr>
<td>160-179</td>
<td>100-119</td>
<td>Moderate Risk (M)</td>
</tr>
<tr>
<td>&gt;180</td>
<td>&gt;120</td>
<td>High Risk (H)</td>
</tr>
</tbody>
</table>

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis

True proteinuria in cats is a known marker of poor prognosis in renal disease (Syme, H.M. et al, 2006; Syme, H.M., 2009). If proteinuria is established on the chemistry stick in the absence of active sediment, the sample will need to be submitted for a urine protein creatinine ratio (UPCR). The result should be used to direct therapy with medications to reduce the loss of protein into the urine. Ratios over 0.4 are significant and therapy is needed. If there is active sediment in the presence of proteinuria on the chemistry stick, and the UPCR is very high (>0.5), then the value may be significant and therapy may be indicated.

Blood pressure changes can be impacted by and/or impact the renal state of health (Brown, 2011). Sixty-five to 100% of cats with hypertension have evidence of reduced renal function (Jepson, 2011). The gold standard for blood pressure assessment in any species is central venous catheter assessment. Blood pressures can be measured non-invasively either by Doppler or oscillometric methods. Patient stress can be a limiting factor. Proper use of pain management in advance, as well as following cat friendly practice and handling guidelines will significantly reduce stress.

References


With the utilization of IRIS staging, the clinician gains significant ground in combatting chronic renal disease in cats. The data collected for the purpose of IRIS staging allows a tailored, individual approach to patient therapy. Although the clinician will have a wide range of therapeutics available to improve quality and quantity of life in the CKD patient, the client and patient relationship must always be considered. In particular, the clinician should consider the client’s ability and willingness to medicate the patient with multiple drugs multiple times a day. Available and necessary therapeutics may need to be prioritized in order to maintain client quality of life and the client-patient relationship.

Table 5: Survival time by IRIS stage

<table>
<thead>
<tr>
<th>IRIS Stage</th>
<th>2b*</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Survival (days)</td>
<td>115</td>
<td>778</td>
<td>103</td>
</tr>
<tr>
<td>Range (days)</td>
<td>2-3107</td>
<td>22-2100</td>
<td>1-1920</td>
</tr>
</tbody>
</table>

*2b Creatinine of 203-249 µmol/L

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM  *Chronic Kidney Disease: Making the most of early diagnosis*

Treatment for pain is essential. Medications such as gabapentin should be prescribed at a dosage of 15-20 mg/kg PO q12h. In debilitated cats, a dosage of 5-10 mg/kg POq12h is the initial chosen dosage. This medication is safe for use in all diseased states and the only initial side effect is sedation. After 1-2 weeks, any sedation will wear off and the cat will continue to be more comfortable. The dosage will need to be titrated up and down in order to meet the particular patient’s needs. Injectable products such as Cartrophen™ or Adequan™ for degenerative joint disease (DJD) are also beneficial. Additional pain medications such as buprenorphine, amantadine and non-steroidal anti-inflammatory drugs (NSAIDs) may also require consideration. Elevated stages of CKD may preclude safe use of NSAIDs.

Dietary changes recommended for cats with renal disease should be considered. Many renal specific diets are formulated with reduced phosphorus; ideal, highly digestible protein sources; increased energy content; vitamins such as B12 and a range of other beneficial ingredients. The decision to commence renal diets and at what IRIS stage will vary from patient to patient. Increasing water intake may be a key factor in improving renal function and overall patient hydration status. Indirectly this can reduce pain from dehydration and constipation.

Identification of BP values over 160-180, with or without retinal changes indicate the need for BP-controlling drugs. Calcium channel blockers such as amlodipine are the most effective at controlling blood pressure in the feline species. Some patients will have partially or uncontrollable hypertension with amlodipine and may require additional medications. Benazepril (Fortekor) is not effective in the control of hypertension. The newly available drug telmisartan (Semintra) may be effective at controlling hypertension at higher doses, but has yet to be evaluated for further benefit to hypertension cats.

The indiscriminate use of antibiotics in the absence of evidence of urinary tract infection is not recommended. Antibiotics should be selected based on urine culture and sensitivity patterns. A repeat urine culture 7 days following cessation of therapy is critical. In cases where urine culture is negative, but a low USG exists in the face of renal disease, ultrasound is recommended.

Patients who exhibit even mild decreases in potassium levels in their serum require supplementation with potassium gluconate. The majority of body potassium is held in the intracellular or interstitial space. The serum potassium represents only 2% of body potassium. Therefore any decrease noted in the serum is significant of a major decrease in the overall body stores.

Elevated UPCR indicates abnormalities with the renin angiotensin aldosterone system (RAAS). These changes alter intraglomerular pressures and result in protein loss into the filtrate/urine. The use of benazepril has been recommended in the past. However, this drug is not targeted to the particular pathway of RAAS that is affected and impacting the glomerulus. Over time, the RAAS can escape the control that benazepril exerts, resulting in resumed proteinuria. Telmisartan is a newer alternative that is more targeted and not likely to lead to escape mechanisms over time.

Improving hydration status in renal patients is generally considered to be beneficial to renal function and overall patient health. Addition of 20-40 mEq/L of potassium chloride to the fluids should be considered in the case of hypokalemia and/or where regular subcutaneous fluids will be administered.

Patients identified with elevated total calcium, elevated ionized calcium and/or elevated phosphorus may require phosphorus-binding agents to reduce phosphorus levels. The use of agents such as aluminum hydroxide can be challenging, as palatability is less than optimal. The use of phosphorus binding agents containing calcium should be minimized unless serial monitoring of ionized calcium can be pursued.
Calcitriol is a drug that is recommended frequently in renal patients. It’s primary indication for use is following diagnosis of renal secondary hyperparathyroidism. In these cases, the use of calcitriol, with regulated serum phosphorus levels, may benefit the patient in the short and long term. Low-dose calcitriol supplementation is recommended by some experts as a means of improving quality of life, however, more detailed studies on the benefits and risk of this approach are warranted (Sparkes et al, 2016).

Chronic kidney disease can lead to a reduced production of erythropoietin. The result is a reduced production of new red blood cells from the bone marrow. Some patients will also have iron deficiencies reducing production of new RBC. Evaluation of iron levels with consideration for supplementation is needed. These patients may also require injectable erythropoietin or darbopoietin to stimulate bone marrow production of RBC.

References

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Feline Diabetes Mellitus: Is Remission a Reasonable, Achievable Goal?
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Charing Cross Cat Clinic
Brantford, ON

Diabetes Mellitus (DM) is a common feline endocrinopathy. Most cases are primary and similar to type II diabetes in humans, which results from abnormal secretion of insulin from the pancreatic B cells and peripheral insulin resistance. The diagnosis of DM is made based on characteristic clinical signs of diabetes mellitus (polyuria, polydipsia, polyphagia, and weight loss), and documentation of hyperglycemia and glucosuria. In cats it may be complicated by the occurrence of stress hyperglycemia and sometimes stress glucosuria. When making a diagnosis of DM in cats, it is important not only to document persistent hyperglycemia and glucosuria, but also to rule out other diseases that may cause similar clinical signs. Measurement of fructosamine concentrations or urine glucose of samples collected in the home environment may allow the clinician to distinguish between stress induced hyperglycemia (and resultant glucosuria) and persistent hyperglycemia due to diabetes mellitus. Therapy for diabetes should be instituted as soon as possible after diagnosis.

The main goal of therapy is to achieve blood glucose levels within the normal range. The secondary goal is to achieve persistent normoglycemia with no further requirement for exogenous insulin. This latter goal is commonly termed diabetic remission. Diabetic remission is usually defined as the ability to maintain normal blood glucose without insulin treatment for 4 weeks without the reappearance of clinical signs. Clinicians need to accept that not all cats will achieve remission and in these patients the goal is to minimize the clinical signs without causing hypoglycaemia while avoiding excessive fluctuations of blood sugar above the normal range. The duration of remission is highly variable and unfortunately, at least 25% of cats that achieve remission subsequently become overtly diabetic and must receive insulin again. Tight glycemic control is required to achieve remission and as a result, there is an increased risk of at least one hypoglycaemic episode. This risk associated with tight glycemic control need to be discussed with the client. If this is not an approach the client is ready, willing or able to take, it may not be the ideal choice. Successful management of cats with DM includes minimizing clinical signs, improving quality of life, preventing complications such as DKA and preventing diabetic neuropathies and nephropathies. If the goal of diabetic remission is not an achievable target for the client, the clinician should continue to help them focus on the overall goals of DM management.

Administration of insulin and dietary modification are the principal therapies used for management of diabetic cats. A recent study showed that cats with newly diagnosed DM have a fair to good prognosis, with 46% living longer than 2 years. However, since 30% of cats affected with DM are euthanized within their first year of treatment due to the emotional and financial burden of insulin treatment and the required veterinary care, achieving diabetic remission is the ideal goal for every feline patient faced with this disease. Intensive glycemic control after diagnosis has been shown in humans with DM type1 to improve long – term remission rates. It appears that the same holds true for our feline patients. Cats receiving treatment for diabetes within 6 months of diagnosis with twice daily insulin treatment aimed at euglycemia in conjunction with the cats been fed an ultra-low carbohydrate diet have the best chance of remission.

Which cat will go into remission?? Studies are suggestive that DM remission in the cat is likely to occur through reversal of glucose toxicity. As in humans, cats that have experienced more prolonged hyperglycemia will have experienced a greater deterioration of beta-cell function resulting in a lower chance of remission. There is no factor that consistently predicts diabetic remission in the cat but the shorter the duration of DM, the faster glycemic control is achieved and those patients with less severe hyperglycaemia when starting appear to be factors that are favourable. A retrospective cohort study showed that cats without hypercholesterolemia were more likely to achieve remission. In one study, diabetes as a potential result of recent corticosteroid treatment was associated with nearly 50 percent remission. A lack of diabetic neuropathy has also been associated with future remission, but neuropathy is a result of prolonged hyperglycaemia so this should not be a surprise. Early client recognition, early diagnosis, intensive treatment with twice daily insulin and ultra-low carbohydrate diet are key.

One of the challenges we face as veterinarians is the opportunity to diagnose this disease in the early stages. Cats are “masters of disguise” They also do not receive regular veterinary care. Often by the time we see the patient and diagnose the disease, the cat already has lost weight and muscle mass, has a poor hair coat, glucose toxicity of the beta cells, diabetic neuropathy and possibly DKA. Using every opportunity, a veterinary team has to teach cat owners the importance of early disease diagnosis through regular veterinary care. Teaching the subtle signs of sickness is critical. The author recommends using Cat Healthy as a resource to educate every client that comes through our doors. In addition, once diagnosed with diabetes, the Cat Healthy website http://www.cathealthy.ca has a series of educational videos about diagnosis, treatment and outcome for the newly diagnosed diabetic cat family. The Cat Healthy Protocols contain a compliance section listing other useful resources for the family as they start the journey of insulin treatment and blood glucose monitoring for their cat. The earlier we diagnose and treat the disease, the better chance we have of remission.
The use of an ultra-low carbohydrate (CHO) diet is an important part of DM therapy in the cat. Low carbohydrate diets reduce post-prandial hyperglycaemia in people. It seems a low carbohydrate diet in the cat is equally important. A study giving twice daily insulin showed a 12-week remission rate of 17% in cats fed diets with variable carbohydrate content and a 12-week remission rate of 40% in diabetic cats fed an ultra-low to low carbohydrate diet. The Bennett study reported a greater chance of remission in diabetic cats fed a low CHO diet than those fed a high fibre diet. Obesity is common in DM cats. If present, it should be addressed with a therapeutic weight-loss diet and an energy-restriction plan. Metabolic energy or resting energy requirements (MERs or RERs) should be calculated for each individual cat thus allowing determination of the actual food intake permitted for weight maintenance or weight loss (or in some cases, weight gain).

The clinician’s choice of insulin will vary depending on experience, training and current studies. Cats can theoretically achieve remission with the use of any insulin type. The type of insulin used for the best chance at achieving remission may be less important than factors such as the presence of concurrent diseases, initiating the treatment as soon as possible and the plan for close monitoring. Diseases such as Acromegaly and Cushings disease can be causes of a lack of response to insulin. Co-existing pancreatitis can also have an effect on the blood glucose levels and requirement for insulin. Clinicians should be familiar with at least two types of insulins that are appropriate for treating cats, as it is difficult to predict in advance which insulin is best for an individual cat. Glargine (Lantus™) has been proposed as the optimum insulin for diabetic cats based on the relatively high remission rate reported in some studies using this insulin, but this may be because it is the most frequently studied insulin. In a study assessing the influence of low CHO diets on remission rates, the insulin PZI (Prozinc™) achieved similar remission to a study examining twice daily glargine. Further studies are required to compare if there are different rates of remissions between the different insulins.

### Table 2. Comparison of insulin products for treatment of feline diabetes mellitus

<table>
<thead>
<tr>
<th>Insulin</th>
<th>Licensed in cats</th>
<th>Manufacturer</th>
<th>Formulation</th>
<th>Action</th>
<th>Dose*</th>
</tr>
</thead>
<tbody>
<tr>
<td>ProZinc</td>
<td>Yes</td>
<td>Boehringer Ingelheim</td>
<td>U40 recombinant PZI</td>
<td>Nadir 5–7 hours Duration 8–9 hours</td>
<td>Start 0.25–0.5 U/kg, BID Median maintenance dose 0.6 U/kg, BID</td>
</tr>
<tr>
<td>Vetsulin, Caninsulin</td>
<td>Yes</td>
<td>Merck</td>
<td>U40 Porcine zinc</td>
<td>Nadir 4 hours Duration 8–12 hours</td>
<td>Start 0.25–0.5 U/kg, BID Median maintenance dose 0.5 U/kg, BID</td>
</tr>
<tr>
<td>Lantus</td>
<td>No</td>
<td>Sanofi Aventis</td>
<td>U100 Insulin glargine (recombinant human analog)</td>
<td>Nadir and duration not determined in diabetic cats</td>
<td>Start 0.25–0.50 U/kg, BID Median maintenance dose 2.5 U/cat, BID</td>
</tr>
<tr>
<td>Levemir</td>
<td>No</td>
<td>Novo Nordisk</td>
<td>U100 Insulin detemir (recombinant human analog)</td>
<td>Nadir and duration not determined in diabetic cats</td>
<td>Start 0.25–0.50 U/kg, BID Median maintenance dose 1.75 U/cat, BID</td>
</tr>
</tbody>
</table>

*Based on lean body weight

Teaching our clients to be comfortable to take blood glucose levels at home is critical for remission. “In clinic” blood glucose curves are inaccurate and a diagnostic method of the past. Having a few team members on staff that can guide the clients through the early stages of diabetic monitoring and treatment is critical and will greatly improve the chance of remission. Commonly used protocols are to “Spot Check”, do home blood glucose curves or multiple daily monitoring. What protocol is needed will be determined by the client’s schedule and lifestyle and the individual patient’s needs. It appears that remission is likely only achieved in those cats that received long term glucose monitoring.

The earlier we diagnose DM in our feline patients and initiate treatment with twice daily insulin in conjunction with an ultra-low carbohydrate diet, the better chance we have of diabetic remission. Teaching clients to monitor blood glucose levels at home is a critical part of the plan as well. Remission in the diabetic cat is possible!

### References


These lecture notes and associated presentation were modified from a previous presentation by Dr. St. Dens and Dr. O'Brien at the Ontario Veterinary Medical Association 2017 conference & Trade Show.
Identifying the underlying cause of house soiling can be problematic. House soiling often occurs as a result of multiple issues, which may include medical, environmental and/or anxiety-related issues. The main medical differential diagnoses for feline house soiling include feline idiopathic/interstitial cystitis (FIC), urolithiasis, crystalluria, infection, constipation, dehydration and/or neoplasia. Environmental concerns may include insufficient availability of resources such as litter boxes, feeding stations and sleeping locations (to name a few). Poor litter box management such as covered boxes, infrequently cleaned or inconveniently located boxes, may also contribute. Anxiety may arise from a wide variety of problems including illness, inconsistent provision of daily care (feeding, play etc), insufficient resources, inter cat aggression, as well as territorial anxiety. Intact males or males neutered after puberty may be more prone to house soiling in the form of territorial urine marking.

When a cat is presented with house soiling, a detailed history including information about the cat’s age, home environment, census of other pets in the home, behaviour, diet (including treats), water intake and other concerns are critical. History is followed up with a thorough physical examination with attention to the palpation of the abdomen. Is their pain? Does palpation of the bladder illicit urination? Does the bladder feel soft, thickened, firm? Is there evidence of firm, small stool and/or evidence of constipation? Does the cat have a urinary obstruction which requires immediate resolution? All cats with house soiling require a basic urinalysis which includes visual assessment, specific gravity, dipstick analysis, and sediment microscopy. This includes cats with feline house soiling, as some of these patients may have painful urination, leading them to defecate away from the pain-associated litter box. Blood work should be considered as an important part of the minimum data base required to rule out systemic disease. Depending on the findings, further investigations should then include radiographs of the abdomen and if needed ultrasound imaging of the bladder to identify evidence of constipation, urolithiasis or neoplasia. Urine culture and sensitivity should be carried out for any cat with an active sediment (WBC, bacteria), low urine specific gravity and/or glucosuria. Urine cultures should be obtained only by cystocentesis to prevent false positive results from contamination during a free-flow sample.

Feline idiopathic cystitis (FIC)
Feline idiopathic cystitis (FIC) is a complex disease process in cats that is not fully understood. The condition is often a diagnoses of exclusion, after all other potential medical causes have been ruled out. It is important to realize that FIC can occur as a result of contributing factors such as environmental mismanagement and anxiety. It is therefore difficult to tease out many factors related to FIC from factors associated with house soiling. The clinical signs of FIC may include pollakiuria, periuria, dysuria, hematuria and/or stanguria. Vocalization when urinating and hair loss on the ventral caudal abdomen may also occur. The severity of signs and the frequency with which they recur is variable. FIC can be obstructive or non-obstructive in its presentation. It is the most common cause of non-obstructive feline lower urinary disease. This disease is generally seen in younger and middle aged cats and is uncommonly diagnosed in cats greater than 10 years of age. In reported studies, excessive body weight, decreased activity, multiple cat households and indoor housing have been associated with increased of FIC. Affected cats can suffer recurrent episodes, which generally resolve without treatment over the course of 3–7 days. FIC can present as an acute episode or develop into a chronic re-occurring condition. While the condition of FIC currently remains, by definition, an idiopathic disorder, recent developments in the understanding of the neuro-hormonal abnormalities that exist in affected cats suggest that the signs develop from an inability to cope with chronic stress. This may manifest in a number of ways, including the development of bladder inflammation and pain. No cure is currently available for FIC, and treatment options are aimed at keeping the cat's clinical signs to a minimum, and increasing the disease-free interval. Clinical signs of acute FIC resolve spontaneously in as many as 85% of cats within 2-3 days, with or without treatment. Assessing the efficacy of any medical treatment for FIC is made difficult by the self-limiting nature of this disease. When a cat is diagnosed with FIC, analgesic therapy should be initiated for the acute management of the disease. These cats are painful and the pain needs to be treated in a multi-modal fashion with opioids, non-steroidal inflammatory drugs and other analgesics such as gabapentin. Prazosin hydrochloride may be helpful to relieve urethral spasm. It is important that the client appreciates that all current treatments for FIC are merely palliative and that without application of multi-modal environmental modification (MEMO) and measures to increase water intake, the FIC episodes will recur and will require continued management. A primary objective in managing FIC is to encourage the production of large volumes of dilute urine (SG < 1.035). Any measures which will increase the cat's water intake are likely to be helpful. Feeding canned food is particularly effective, as is offering the cat palatable fluids to drink (chicken or fish stock, water from tinned fish, etc.). Adding extra water to canned or dry foods works well. Monitoring of the success of the owner's attempts to increase water intake can be done via regular analysis of the urine samples collected at home or in the clinic. Aim is to keep the urine SG below 1.035.
Environmental management
Environmental modification is a key factor in the management of house soiling, since stress clearly plays an important part in the problem. Meeting the environmental needs of the cat and understanding the cat as a species is critical. Cats are not inherently social and in the wild are solitary hunters. They tend to be solitary and are territorial and although they are hunters, they are also prey. These traits make it challenging for cats to live in close proximity to other cats. 'Silent bullying' often goes unnoticed, but it is a major cause of chronic stress to the less dominant cat. When a cat is presented for house soiling, a questionnaire should be completed by the client to establish a thorough environmental history, followed by the recommendations for MEMO. For suggestions on developing a questionnaire, as well as a good client resource, the reader is referred to the following websites: http://www.indoorcat.org/ and http://www.cathealthy.ca.

Identifying and addressing environmental management issues in the affected households is a critical aspect in the reduction of house soiling. This applies to all causes of house soiling, but is of particular importance in cats with FIC. In one study, multi-modal environmental modification (MEMO) was evaluated in client-owned cats with FIC. Implementing MEMO as the sole management strategy with FIC was found to be successful in the majority of cats followed over a one year period of time.3

Dietary adjustments
Diets such as Royal Canin Calm® or Royal Canin Urinary/Calm® and Hill’s Multicare C/D Stress® can be helpful in the long-term management of cats with house soiling issues, especially those with FIC. These type of diets reduce the frequency and intensity of recurring episodes of lower urinary tract signs. To achieve this aim, they need to be fed as the cat’s sole source of nutrition and used consistently in the long term. Calming nutraceuticals such as Zylkene® or Anxitane® may be helpful.

As cats that develop litter box issues, degenerative joint disease, and in particular FIC tend to be overweight, a weight loss program with a strict calorie counted amount of food fed per day is likely critical.

Resource management
To meet the needs of each cat within the house, each individual cat must have free access to its own key resources, ideally positioned out of sight of the other cats. Key resources include food and water bowls that are sited apart from each other; clean uncovered litter trays (one box per cat, plus one) in various locations around the home; resting places at different vertical heights with some that only fit one individual cat; and multiple scratching posts and scratching resources. Cats need mental and physical activity several times a day and cat families need to make time in their day to play with their cat as they would their dog. Putting the hunt back in meal-time using feeding toys is a good form of entertainment for the “predator” in the cat.

Feline pheromones
Although a statistically significant difference was not found when Feliway® was used in a home compared to placebo in cats with FIC, cats that had Feliway® used in the environment had a trend for fewer bouts of FIC and reduced negative behavioral traits.

Pharmacologic interventions
A variety of drugs have been tried in cats with house soiling concerns, but their efficacy will vary with each cat in each separate situation. All medical, dietary, environmental and resource concerns must be addressed prior to or in conjunction with the use of pharmacologic agents in feline house soiling. Expectation that a drug may resolve house soiling on it’s own without these concerns being addressed is unrealistic and more likely to lead to treatment failure. Selection of drugs will be based on the identified areas of concern, whether these are anxiety based, or secondary to aggression or timidity in a multicat household.

Urinary tract infection
Bacterial infections are rare and most likely seen in older female cats with a low urine specific gravity or cats with glucosuria. Cats presenting with house soiling and/or evidence of FIC should not be prescribed empirical antimicrobials. As the condition of FIC is waxing and waning, it may appear that the cat has responded to antimicrobials which will lead to a false diagnosis of infection. Rarely do cats have a bacterial infection, even when they have a urolith. The need for antibiotics should be based on a positive urine culture and treatment selected according to the sensitivity results.

References

These lecture notes and associated presentation were modified from a previous presentation by Dr. St. Dens and Dr. O’Brien at the Ontario Veterinary Medical Association 2017 conference & Trade Show.
The feline species may generally be considered to be a solitary creature where social interactions are concerned, but domestication has allowed the adaptation to living in social communities with both humans and other cats. Domestic cats are well known to be able develop bonds with humans and other cats. The ability to socialize with other cats and humans will vary between individual cats as a function of their genetics, prenatal environment, early socialization and life experiences. The acceptability of other cats within the household will in part depend on the history of the cats in question, including how and when introductions were made. One of the most common causes of inter-cat aggression issues is introduction of a new cat into a pre-existing social environment of a household with 1 cat or 1 or more pets. Other causes of inter-cat aggression include play aggression, territorial aggression, fear aggression and redirected aggression.

Cats that cohabitate are in many cases able to develop a social bond, or at least co-exist in the same environment. Cats that are bonded and not experiencing inter-cat anxiety will allogroom, allo-rub, allo-play, and sleep in close or direct physical proximity to one another. This is called affiliative behavior. Allogrooming and other cat-cat behaviors such as play should not end in physical or vocal violence. For example, cats that allogroom for a short duration followed by swatting and/or hissing and growling are not likely to be living in complete harmony with one another. Cats that cohabitate may not exhibit covert signs of aggression. Agonistic behavior can be subtle, and clients may not recognize the signs. Cats can exhibit signs of aggression simply through certain facial expressions and body positioning. Clients may not be aware of the subtle facial and body language that indicates an aggressor or defensor. Clients may not be aware of the moderately subtle signs such as physical blocking of access to resources, or passive blocking of access to resources (ex. staring). Clients may consider chasing to be a form of play when it is in fact the act of an aggressor towards a defensive cat. Normal play between cats should involved reciprocal chasing with minimal to no vocalization. Clients are more likely to be aware of agonistic behavior that is obvious, including biting and scratching and full physical fights.

When a client approaches the veterinary team for assistance with an inter-cat aggression problem, it is critical for the clinician to delve into the history of the cats in question. Affiliative and agnostic behaviors may be in their history, but the client may have always assumed the cats were affiliative. Teasing out a history of inter-cat anxiety is important to developing a treatment plan for the current aggression problem. As with aggression towards humans (see Feline Aggression Towards Humans- Prevention and management), the clinician will need to determine the forms of aggression that are being exhibited. This includes sorting out a history of play aggression, determining if there are territorial issues, whether there is fear and whether a redirected aggression episode occurred. The situation can be multifactorial. For example, two cohabiting cats may have always had territorial and fear aggression that was subtle. This subtle issue may suddenly become a major issue when a new cat is brought into the house or an outdoor stray presents itself at the window. In the case of a new household pet, the group dynamics must shift and in the case of outdoor cats, redirected aggression may worsen the situation.

Goals for the behavioral interview and development of a treatment plan

1. Identify underlying motivation for the behavior
2. Identify pre-existing inter-cat issues (perhaps client was never aware)
3. Correctly identify the aggressor(s) and the defensor(s)
4. Identify triggers and formulate a plan on how to avoid these
5. Review enrichment and resource management. Correct any deficiencies.
6. Develop a treatment plan

Questions to ask during the behavioral interview

Basic information should be gathered about the patient signalment, household members (people and animals), patient medical history and how/where the pet was acquired. The environment should be reviewed, including resource management and availability, litter box care, and household enrichment. The patient’s daily activities should be reviewed and in cases of house calls, traced throughout the house layout. Relationships with other pets in the house and with humans in the household should be reviewed. The stability of the human population should be assessed. For example, humans that work shift work hours may be home inconsistently, causing the patient’s environment to be inconsistent.

The incident considered the initiating incident, as well as further incidents should be reviewed in detail. There should be an examination of the frequency, intensity and severity of the behavior(s) in question. The behavior of all of the affected cats and subsequent responses should be determined. Videotaping of these incidents can be very helpful to the clinician, allowing direct
visualization of body language rather than relying on the client’s memory. Any attempts at treatment or punishment should be reviewed as these may negatively impact the prognosis.

House calls are often the best approach to pursuing a behavior consultation, as the clinician can see first hand the environment that the cats live in, what resources are available and where incidents occurred. PLEASE NOTE: Where inter-cat aggression has become so severe that the cats cannot see each other without erupting into vocal and physical violence, they should be separated until such time as a treatment plan can be developed. During the home behavioral consultation, the cats should NOT be reintroduced to allow the veterinarian to see ‘what happens’. This can be potentially dangerous to cats and humans. The veterinarian must rely on the information gleaned during the interview as well as an video the client has.

**Points to consider with regards to the potential success of treatment**

Inter-cat aggression can disrupt animal bonds for life. The ability of two or more cats to live together again after the aggression has become severe may be limited. Reconciliation may be impossible. As with cat-human aggression, a frank, open discussion is necessary with the client(s) in order to determine the prognosis for the patient. It is important to determine how bad the aggression has become and whether this has also affected the human-animal bond. The clinician will need a frank admission by the client about how willing they are to implement the outlined treatment plan. The clients will need to be open about what options they are considering. Options may include following the prescribed treatment plan, drugs, relinquishment and/or euthanasia. Further, the clients’ expectations must be known. What are the clients’ goals and timeline? The clients may have unrealistic expectations, desiring complete resolution of the problem.

**Resource management: Critical in multi-pet environments**

Resource management in a multi-pet environment is critical to reducing territorial anxiety in cats. While litter box resources are often considered during house soiling consultations, these represent only one facet of household resources that are important to indoor cats:

- Litter boxes
- Sleeping and resting areas
- Food bowls
- Water bowls
- Toys
- Perches
- Scratch posts
- Scratching surfaces

Litter boxes should be provided at a ratio of one litter box per cat, plus one additional box. The boxes should not be located in the same room, and not all on the same level of the house. Suitable box size, unscented clumping litter substrate and coverless boxes should be used. Regular, daily or twice daily scooping of the boxes is necessary. Sleeping and resting locations should be ample to accommodate all cats in a variety of locations. Most cats do not wish to sleep close to other cats, which means that sleeping and resting locations should be distributed widely throughout the household. Some of the scratch posts and perches should be located near windows, to allow the cat to visualize outdoor activities such as birds and squirrels, which is mentally stimulating for the cat. In cases where outdoor cats or animals are causing territorial anxiety, the yard view may need to be blocked temporarily. Other scratch surfaces should be located near sleeping spots, so that the cat who wishes to scratch and stretch after a nap has immediate access to an acceptable scratching surface. The client may choose to place scratching surfaces in both busy and quiet areas of the household, so that the cat has multiple locations to scratch.

For most cats, catnip and catnip spray help to encourage use of these articles. It is important to note that kittens under four months of age will not respond to catnip and some rare adult cats are actually non-responders.

Food can be a major source of anxiety in multi-cat households. Ideally, cats should be fed three-four meals a day, in separate rooms. Cats fed within visual, olfactory and/or auditory distance of each other may experience anxiety as they eat. This may not be obvious to the client, as signs can be subtle. Some cats may eat their food rapidly, others may move from bowl to bowl, sometimes pushing the other cat away. Some cats may eat and then move to another area of the household to mark territory in an expression of their anxiety. The client should ensure that one cat is not bullying the other cat away from its food. It is usually necessary to confine cats to separate rooms for feeding. Water bowls need to be distributed throughout the household.

Toys should be ample in number, with types of toys being rotated every week if at all possible.

**Dietary adjustments**

Diets such as Royal Canin Calm® or Royal Canin Urinary/Calm® and Hill’s Multicare C/D Stress® can be helpful in the long-term management of intercat anxieties. To achieve effect, they need to be fed as the cat's sole source of nutrition and used consistently in the long term. Calming nutraceuticals such as Zylkene® or Anxitane® may be helpful.
Medications
Determining aggressor and defensor is a critical point in development of an appropriate treatment plan. This is most true when selecting potential behavior modifying drugs for each cat. For example, selection of a drug that will improve the confidence of a defensor, when the aggressor has been misidentified as the defensor can have disastrous consequences. The author refers the reader to the AAFP Behavior Guidelines booklet, which contains a thorough review of the available pharmaceuticals and the targeted individual for each of these drugs. Some of these drugs will be discussed during the lecture.

Prognosis
The ability to resolve inter cat aggression is going to depend on many factors. First and foremost, it is going to depend on the points noted above, as to what client expectations are and whether they are willing to follow prescribed treatment plans. If this is not the case, then the chances of success diminish greatly. Secondly, the duration of the problem, and third, it’s severity, will impact the chance to implement change. Setting realistic expectations and realistic time frames for achieving goals early on in the consultation process is more likely to set the stage for success or at least partial resolution. Clients and clinicians should understand the limitations of the problem and what is reasonable to expect from treatment.

Sample questionnaire for a home behavior consultation
Patient ID:
Patient Signalment:
Medical Conditions:
1.
2.
3.
Current Medications:
1.
2.
3.
What amount of day does cat spend in the following activities?
Sleeping
Resting
Eating
Grooming
Hiding
Playing Alone
Playing with human
Playing with other cat
How often does cat use litter box for BM? U?
Other Pets
1.
2.
3.
Environment:
Layout- rough drawings attached- Y N

Environmental resources
Litter boxes
Number
Location
Characteristics
Feeding Stations
Number
Location
Characteristics
Feeding Schedule
Food
Amount
Timing
Private Locations/Hidey spots
Number
Location
Used?
Elevated Locations/3D space Availability
Number
Location
Used?
Windows
Location
Look out onto:
Other environmental enrichment:
Toys:
Directive play with client:
Potential external stressors noted outdoors:
Unique considerations (ex. flooring type, smoking, cleanliness, clutter, noise levels)
Social Interactions
Humans
Number
Stability of residence
Stability of employment hours:
Relationship with Cat:
Other regular visitors:
Dogs
Cats
How long have cats lived together?
Do they groom each other?
How often?
Do they rest/sleep together?
Do they stay in the same room together?
Does one ever avoid the other?
Does one cat ever block access to the food or resources?
Do they play together?
Describe the play
Before the current incident have there ever been any incidents of concern?
Has either cat ever shown any aggression towards people or other pets?

Specific incidents reported
Known Triggers of Aggression:
Aggression directed towards:
Client/Individual reaction to aggressive behavior:
Any punishments used?
Yelling, swatting, other
Veterinarian Interactions with Cat during visit:

References
Understanding the Vomiting Cat

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Clients and veterinarians often consider that vomiting in cats is a regular occurrence that is not significant of 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). The majority of ingested hair passes through the cat’s digestive tract into the feces with no negative side effects (Panaman et al, 1981). Cats that vomit occasionally may not be considered to have any specific underlying gastrointestinal disease (GID). However, cats that are vomiting more often than every 2 weeks are significantly more likely to have some baseline underlying GID (Norsworthy et al, 2015).

During routine preventive care examinations, detailed questioning about diet, diet changes, vomiting and hairballs is essential. When clients are uncertain about vomiting and/or hairball frequency, a calendar recording system should be recommended. In addition to regular vomiting, the patient may be showing signs of nausea that are not obvious to the client. These signs might include a finicky appetite, occasional loss of appetite or periods of anorexia, licking of the lips, gagging, and/or ingestion of grass to stimulate vomiting.

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. The veterinarian should also carefully question the client to identify evidence of constipation. Conditions such as inflammatory bowel disease (IBD) can exist as a problem within the small intestine, combined small intestine/large intestine or solely the large intestine. Vomiting, diarrhea and/or constipation may manifest as a result.

A thorough physical examination of the vomiting cat will help elucidate signs of nausea. The patient should be observed for signs of lip licking and frequent swallowing. A thorough oral health 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. This change can be a hallmark of mild to significant GID.

The abdomen should be examined in quadrants and the patient carefully observed for evidence of nausea or pain during palpation of each quadrant. Evidence of pain during abdominal palpation may include very subtle changes. The patient’s face should be monitored closely for evidence of lip licking, 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, abdominal fluid, masses and/or enlarged lymph nodes.

Making a diagnosis

The list of differential diagnoses in the adult and senior feline patient with chronic vomiting is long and complex. In all cases, a minimum database (MDB) plus a gastrointestinal (GI) profile is ideal for diagnostic testing. The GI profile should include cobalamin (B12), folate, feline specific pancreatic lipase (sfPL) & in many cases, trypsin-like immunoreactivity (TLI).

The patient’s feline leukemia virus (FeLV) and feline immunodeficiency virus (FIV) status should be determined. Feline leukemia virus is a known cause of lymphoma in the feline patient. However, with the introduction of vaccination against FeLV, there has been a shift in the types of intestinal lymphoma in cats (Cotter et al, 2011; Louwerens et al, 2005). This shift does not change the value of knowing the patient’s retroviral status, as disease management will be impacted by retrovirus infection.

Radiography is beneficial in elimination of some differential diagnoses in the vomiting cat. 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 changes. Evaluation of skeletal structures may indicate the presence of painful spondylosis, osteoarthritis and/or degenerative joint disease.

Ultrasoundographic imaging is beneficial in identifying GI organ abnormalities (liver, gall bladder, spleen, pancreas) as well as the urinary tract. The intestines can be evaluated for abnormal gut motility, obstruction, or other intestinal accidents (ex. intussusception). The abdomen can be evaluated for a discrete mass or masses, including evidence of lymph node enlargement. Evaluation of intestinal wall thickness, as well as thickness and integrity of the four intestinal wall layers may help identify the presence of intramural disease such as IBD and lymphoma. Ultrasound changes associated with pancreatitis may be evident (Forman et al, 2004). The sensitivity of ultrasound in the diagnosis of pancreatitis is low (Cosford et al, 2010; Forman et al, 2004; Gerhardt et al, 2001).

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. Exploratory surgery permits full visual assessment of all intra abdominal organs, biopsy of extra-intestinal tissues (liver, pancreas, lymph nodes etc) and full thickness intestinal biopsy (Kleinschmidt et al, 2010).

**Symptomatic, targeted and empirical therapies**

Dietary changes may be beneficial to the patient with GID. Changing dietary format, such as dry to canned food, may improve digestion. The use of veterinary formulations that are easy to digest such as Royal Canin Gastro, Hill’s i/d or PVD EN may reduce or in some cases eliminate active GID signs. The role of dietary allergens in IBD and other GID is difficult to confirm. Food-responsive enteropathy is characterized by signs similar to other GID, although large bowel signs are more often observed and cutaneous disease may also be present. (Jergens et al, 2012).

Anti-emetics such as maropitant (Cerenia™) may be beneficial to the vomiting patient. Drugs with pro-kinetic effects should be used with caution in case of obstruction. Gastric acid blockers such as ranitidine and omeprazole are less likely to play a beneficial role in feline patients with GID.

Appetite stimulants for loss of appetite or anorexia may be beneficial in improving intake, but in the presence of nausea and GI inflammation, these drugs are likely to be of little utility until underlying disease is addressed. Mirtazapine provides both appetite stimulant and anti-nausea activity, making it a beneficial option in certain cases of feline GID.

Patients with GID may be experiencing pain as a result of or concurrent to their GID. 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 and non-steroidal anti-inflammatory medications are all beneficial in pain management. Multimodal analgesic protocols are most effective over single drug therapy.

It has been recommended that all cats with signs of GID 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 once weekly for 6 weeks followed by 250 micrograms one month later. Thirty days following this injection, a repeat measurement of B12 is recommended [http://vetmed.tamu.edu/gilab/research/cobalamin-information](http://vetmed.tamu.edu/gilab/research/cobalamin-information).

The empirical use of steroids is generally not recommended in any situation in feline medicine, however, this is a frequently used therapeutic in feline GID patients. Limitations of finances and client willingness to pursue diagnostic biopsy may impact the treatment selection process. Empirical steroid usage precludes or limits usefulness of ultrasound or biopsy, as the drugs will change the local inflammatory pattern, thus confounding diagnosis. Where steroids are to be employed, urine culture should be considered prior to drug initiation, in order to rule out occult UTI. Prednisolone or dexamethasone are the steroids of choice in cases of IBD or GI lymphoma. The author does not recommend the use of depot steroids such as methylprednisolone acetate. The usefulness of budesonide is questionable, although it may offer benefits as an adjunct therapy. Empirical use of cyclosporine or chlorambucil is not recommended.

**References**


Cats do not see or experience the world the way we do. Understanding natural cat instincts can help us improve environmental enrichment at home, as well as improving veterinary visits for cats. In a natural environment, cats are predators AND prey. We frequently think of cats as hunters but forget that they are also hunted. Cats must be on the alert even in their own homes, and particularly at the veterinary clinic. This is the baseline instinct that can lead to negative behaviors both at home and in the clinic.

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 affiliate communication like rubbing, head bunting, nose touching, kneading, treading and allo-grooming. 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.

Home environment
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 as well as by additions to the family (humans and other pets alike). Provision of appropriate resources will also go a long way to maintaining health and normal behavior. Resource management in a multi-pet environment is critical for cats, particularly those in a multi-pet household, or where young children reside. While litter box resources are often considered during house soiling consultations, these represent only one facet of household resources that are important to indoor cats:

- Litter boxes
- Sleeping and resting areas
- Food bowls
- Water bowls
- Toys
- Perches
- Scratch posts
- Scratching surfaces

Litter boxes should be provided at a ratio of one litter box per cat, plus one additional box. The boxes should not be located in the same room, and not all on the same level of the house. Suitable box size, unscented clumping litter substrate and coverless boxes should be used. Regular, daily or twice daily scooping of the boxes is necessary. Sleeping and resting locations should be ample to accommodate all cats in a variety of locations. Most cats do not wish to sleep close to other cats, which means that sleeping and resting locations should be distributed widely throughout the household. Some of the scratch posts and perches should be located near windows, to allow the cat to visualize outdoor activities such as birds and squirrels, which is mentally stimulating for the cat. In cases where outdoor cats or animals are causing territorial anxiety, the yard view may need to be blocked temporarily. Other scratch surfaces should be located near sleeping spots, so that the cat who wishes to scratch and stretch after a nap has immediate access to an
acceptable scratching surface. The client may choose to place scratching surfaces in both busy and quiet areas of the household, so that the cat has multiple locations to scratch.

More information can be found in the following brochure:

http://www.catvets.com/public/PDFs/ClientBrochures/Environmental%20GuidelinesEViewFinal.pdf and on the following website:

https://indoorpet.osu.edu/cats/basic-indoor-cat-needs

Food can be a major source of anxiety in multi-cat households. Ideally, cats should be fed three-four meals a day, in separate rooms. Cats fed within visual, olfactory and/or auditory distance of each other or other pets such as dogs, may experience anxiety as they eat. This may not be obvious to the client, as signs can be subtle. Some cats may eat their food rapidly, others may move from bowl to bowl, sometimes pushing the other cat away. Some cats may eat and then move to another area of the household to mark territory in an expression of their anxiety. Water bowls need to be distributed throughout the household. Toys should be ample in number, with types of toys being rotated every week if at all possible.

The veterinary visit

Step 1: It starts at home

Feline friendly handling starts when clients are scheduling the cat’s appointment. In advance of potential issues, asking the owner about their access to a good carrier, their ability to get the cat into the carrier and what experiences they have had in the past are important to addressing problems before they occur. A telephone script for all staff to use may be beneficial. Any previous issues should have been documented in the patient file for easy reference.

The provision of carrier-friendly resource materials and support is key. Many clinics provide information on the selection of the ideal carrier for cat transportation. In situations where clients may not own a carrier, or do not have a sufficiently secure or ideal carrier, clinics should offer a carrier on loan.

There are several pamphlets available that provide tips on travelling to the clinic with a cat. The American Association of Feline Practitioners (AAFP) pamphlet entitled ‘Getting your cat to the Veterinarian’ (Figure 1; http://www.catvets.com/cat-owners/brochures) is an excellent resource for clients. This type of literature should be provided well in advance of the veterinary visit.

Feline facial pheromones (Feliway) are frequently useful in the reduction of stress. In advance of travel, clients may wish to spray Feliway onto a cloth and place it in the carrier with the cat. Alternatively, clinics can provide the client with Feliway-infused cloth pieces, sealed into zip-lock bags. For some cats, a Feliway infused cloth will reduce agitation, vocalization and soiling during transport.

Step 2: Feline friendly waiting

Calming the feline patient and the client is critical from the moment they enter the clinic. Minimizing loud noises and reducing visual and auditory exposure to dogs is ideal. The existence of a feline friendly waiting area or a separate waiting room can significantly reduce the stress for both patient and client. If a waiting room is too chaotic, the patient and client should be moved immediately to their examination room. Reduced or non-existent wait-times assist in avoiding a buildup of tension in the waiting feline patient. Some clinics offer feline exclusive appointment hours. Clinics may also choose to have feline exclusive examination rooms.

A cat friendly advocate should be appointed in each practice. This individual should be instrumental in helping the practice achieve an optimal feline friendly environment. The appointed staff member should mark a goal of achieving AAFP feline friendly practice status (http://www.catvets.com/cfp/veterinary-professionals).

Step 3: A feline friendly outpatient visit

During the patient’s visit, the veterinary team must continue to strive to reduce stress. In advance of significant handling, the patient should be assessed for pain and treated appropriately. Sedatives should be employed where necessary. Physical restraint should be avoided.

The ambience in the consultation room is of critical consideration. Clinical settings can be harsh to the feline senses. Clinical settings often have strong odors of cleaning solutions, medicinal smells and the scents of other animals. The lighting may be bright and harsh. Examination surfaces are often hard and unforgiving. Stainless steel surfaces may give off disturbing reflections. Thin gauge stainless steel surfaces are known to shift, and as a result may make unsettling noises. Brushed stainless steel surfaces give off less reflection, which can reduce stress.

Soften it up

Provision of soft, fuzzy blankets and table top cushioning are valuable for all cat age groups, but in particular for the old and sick. Infusion of blankets with Feliway can further improve relaxation.

Warm it up

Cats prefer warmer ambient temperatures than humans. The relative warmth of the room air should be addressed. Warming blankets in a spa or medical towel warmer in advance of use can be very beneficial. Alternatively, heated oat bags can be used in a designated drawer to warm patient blankets.
**Darkest it up**

Bright, overhead fluorescent lighting has an important role in the medical examination. However, it is not a necessity during the entire visit. Use of softer incandescent lighting for the majority of the appointment will be more appealing to the feline patient.

**Keep it quiet**

The location of the feline examination room relative to other parts of the clinic may not be alterable. Still, consideration should be given to external noises that may frighten the feline patient. This includes barking dogs, delivery of supplies and loud veterinary equipment such as dental and laundry machines. If the examination room location cannot be altered, then the timing of feline examinations should be carefully planned to avoid these noises. Feline exclusive consultation hours can be beneficial in these scenarios.

**Make it calming with feline facial pheromones**

A Feliway diffuser should be plugged into an outlet in the feline examination room more than 30 minutes prior to commencement of appointment hours (Figure 3).

**Keep it scent free**

While the selection of cleaners for the examination is critical to reduce transmission of disease, it is best to select those with reduced odour. Cleaners should be used as far in advance as possible of the next appointment, in order to ensure that the majority of cleaner scent has dissipated. Soft towels used on surfaces can be removed for washing and will reduce the amount of surface cleaning required.

Soiling or spraying by other patients should be addressed well in advance of the next appointment. The affected areas should be cleaned. In some cases, Feliway spray at the location of soiling will reduce further anxiety in the next patient. Garbage cans containing either urine soaked towels or fecal matter should be emptied and cleaned. In extreme situations, it may be ideal to shift to an alternate examination room until offensive scents can be eliminated.

**Let the cat own the room**

Prior to the physical examination, the consultation should begin with conversation between owner and veterinary team member. The cat carrier should be placed on the floor in the examination room and the patient allowed to enter or exit the carrier at will. The patient should not be forced from the carrier. While the conversation continues, the patient should be permitted to explore the consultation room. Familiarization with the room, including being permitted into and onto objects as well as facial marking, will serve to reduce the patient’s stress levels. When it is time to examine the patient, the lid should be removed from the carrier and reluctant participants gently lifted out.

**The benefits of pain management**

Cats are masters at hiding illness. Concealing pain is no exception. For some painful cats, a defensive or offensive response to handling is a matter of apparent self-preservation. Predicting pain and understanding pain is critical to a feline friendly approach to handling.

Individual practices should establish or adopt a pain index within the veterinary practice. Colorado State University has developed a pain scale system that can be utilized in veterinary practices. [http://www.csuanimalcancercenter.org/assets/files/csu_acute_pain_scale_feline.pdf](http://www.csuanimalcancercenter.org/assets/files/csu_acute_pain_scale_feline.pdf). All staff should be trained to use a pain index consistently. This allows for a standardized approach to the assessment of feline pain. Every patient should be evaluated prior to any other handling, including the physical examination. The mildest handling of a painful patient may provoke a negative response. Identification of the painful cat allows the clinician to employ pain management techniques prior to further handling.

**Step 4: Inpatient care**

The stress of illness can reduce a cat’s tolerance for handling. Dehydration and the tissue trauma associated with illness can cause pain. Sources of stress and pain must be addressed in the intensive care unit (ICU) patient in order to improve recovery.

Patient housing should be at a distance from the noise and smell of canine patients, if at all possible. Other nearby noises should be minimized. Conversations should be kept quiet. Dimmed lighting should be considered.

Within the ICU, patients should be provided with a thick, warm layer of blankets (Figure 4). Soft or fuzzy surfaced blankets are generally preferred. The use of newsprint paper provides no comfort, is cold and unyielding. Warmed oat bags placed under the blankets will improve the ambient temperature of the ICU cage. Provision of boxes or make-shift tents allow the patient to conceal themselves within the ICU cage. A Feliway™ infused cloth or warmed Feliway™-infused blankets can be placed within the ICU cage. The litter box provided to the ICU patient should be wide, low-walled with soft textured, unscented litter.
As cats reach their senior and geriatric years, our focus on their health needs to intensify. Cats age rapidly. The domestic cat reaches its prime by 3 to 6 years of age. At the age of 7 cats are experiencing biological changes related to senescence. Cats aged 7-10 are ‘mature’, cats aged 11-14 are ‘senior’ and after 15 years of age, cats are considered to be ‘geriatric’ (1). For the purpose of simplicity, this lecture will refer to all three age groups as ‘senior’.

By the time a cat reaches it’s senior years, it is hopeful that we have established a good working relationship with our client over the years of the patient’s life. Clients recognize that their pet is getting old. However they do not always understand exactly what changes are going to occur and which of those changes are normal and which are a sign of disease or pain.

Clients usually have a good awareness of their cat’s normal behaviors and activities. As cats age, clients should consider starting some form of 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.

A key behavior that is often excused as a normal part of aging is sleep. Cats living in confinement (indoors) sleep up to 19 hours per day (2). Cats do not normally sleep more merely as a consequence of aging. Sleep patterns include hours spent sleeping or resting, choice of location, and timing of sleep in a 24-hour period. Any changes noted in the normal sleeping patterns should act as an alert to the caregiver that something is not normal. Changes in normal sleep patterns may occur as a result of pain, nutrition imbalances, disease or cognitive dysfunction. Knowledge of all of the cat’s normal behavior patterns is a basic foundation for knowing when changes occur.

The subtle signs of sickness
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.

The 10 subtle signs of sickness
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

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 disease, osteoarthritis (OA) and/or degenerative joint disease (DJD). Dental disease and dental 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.

Pain
If cats are masters at hiding illness, they are geniuses at hiding pain. Caregivers frequently expect to see obvious, outward displays that would indicate pain in their cat, thus leaving them unable to perceive subtle changes suggestive of pain. Some clients may excuse away any changes, citing age as a factor. Monitoring normal patterns of behavior will help detect changes that may be occurring as a result of pain. The caregiver should monitor the cat’s mobility pattern and willingness to jump up or down. Lameness or signs of stiffness after rest should be noted. Changes in litter box usage and/or elimination patterns may be observed in painful cats. For example, cats with painful DJD may no longer consider it necessary to travel to a litter box located in the basement. They may instead elect to use an inappropriate area on the main floor of the house. Increased sleeping hours can be a big indicator of pain, as the cat becomes reluctant to move. Some cats may howl and meow abnormally at odd times during the day, particularly at night. Any or all of these signs can be due to pain, but may also overlap with diseased conditions as well as cognitive dysfunction. Clients should be encouraged to actually make notes about their cat’s activities and behaviors, especially as they age. This way, subtle and gradual changes will not be missed. In many cases, the best way to ascertain whether a cat is truly experiencing pain is to administer a 2-4 week analgesic trial.
Body & muscle condition changes
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, body condition scoring (BCS) as well as muscle condition scoring (MCS) at every single veterinary visit is necessary to detect subtle changes early. 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 and often more rapid change cachexia. Cachexia can indicate the presence of disease, insufficient dietary needs and in particular, insufficient dietary protein. In addition to senior biannual examinations with the veterinarian, regular weigh-ins and BCS/MCS assessments with a registered veterinary technician will assist in early detection of changes in any of these parameters. The client can be taught how to assess these parameters at home as well, making them more aware of changes should they occur. [https://www.wsava.org/sites/default/files/Body%20condition%20score%20chart%20cats.pdf](https://www.wsava.org/sites/default/files/Body%20condition%20score%20chart%20cats.pdf) [https://www.wsava.org/sites/default/files/Muscle%20condition%20score%20chart-Cats.pdf](https://www.wsava.org/sites/default/files/Muscle%20condition%20score%20chart-Cats.pdf)

Dietary needs
As cats age, appetite will often diminish. Changes that may impact the aging cat’s appetite include diminished taste, smell and vision, dental disease, painful arthritis and cognitive changes. Treatable conditions should be addressed and adjustments made to improve intake in the home setting. Dental surgery should not be avoided on the basis of age. Safe general anesthesia of senior patients is possible, with the right care and attention.

As cats age, their caloric and nutritional needs change (1). 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 and geriatric feline patients can be susceptible to weight loss. Dietary palatability is a major concern in this age group, including ensuring that the patient is consuming sufficient calories to meet their metabolic energy requirements (MERs).

Daily dietary intake needs to be quantified by the client in detail. Metabolic energy requirements (MERs) need to be calculated often by the clinician or veterinary technician. Quantifying daily intake for cats is a critical piece of knowledge for clients with senior and geriatric cats, particularly when unexplained weight loss has been detected.

Intake can be improved in the home setting by offering smaller, more frequent meals of highly palatable, age-appropriate food. Slight warming of the food either with warm water or briefly in the microwave, will improve the smell and taste of the food. The client should ensure that the cat has easy access to the food bowls and that competition from other household cats or dogs is completely eliminated. Cats may be experiencing some cervical pain as a result of arthritis, or have neck weakness associated with disease. Feeding platforms raised to a comfortable height will be beneficial for these cats. Age-appropriate diets from a reputable pet food company are best chosen during the senior and geriatric years. Many generic and over the counter foods do not contain age-appropriate content with regard to calories, protein, phosphorus and other nutrients. In these cases, it is best to rely on companies that have a good nutrition research program with an excellent track record of developing diets based on high-level evidence-based medicine.

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 arthritis, 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. Analgesic trials lasting 2-4 weeks will help identify pain-related behaviors. Cats that are over 6 years of age have evidence of DJD in at least one joint (3), and as a result many should be on daily analgesics and OA/DJD therapeutics.

In some cases, cognitive dysfunction (CD) may be the primary source behind the behavior changes noted (4). 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 (4). Vocalization may also occur. Once other conditions have been treated and/or ruled out, CD becomes a more likely diagnosis. There are no specific medications that have been appropriately tested and shown to benefit cats with CD (1, 4), but adjustment for behavior and addressing environmental needs can go a long way to improving the patient. Mental stimulation is a key component in the aging cat and in particular the cat with CD (4). Regular play, feeding puzzles and other games will keep the mind active and engaged, reducing random CD behaviors.
Unique environmental needs
Senior and geriatric cats have unique environmental needs. This mainly stems from their likely reduced mobility secondary to arthritis, as well as weakness due to sarcopenia and/or disease. Particular consideration should be given to resource management. Litter boxes should be placed throughout the house, on more than one level, to reduce the travel time required for a senior cat to get to the box. Multiple litter boxes will also reduce competition issues in multi-cat households. 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. Food and water bowls should be placed strategically throughout the house. Competition for food intake and potential safety threats at mealtime should be eliminated. This can be accomplished by feeding the senior cat in a confined environment away from other cats, dogs and young children. Food bowl preferences should be considered. Some cats will prefer bowls, but others may prefer flat, open edged plates. Raised feeding surfaces may reduce discomfort in those cats with cervical arthritis pain or weakness from disease. Assistance with access to higher furniture such as beds and windowsills can be accomplished with steps or platforms to reduce necessary jumping heights. Aging cats still need to play. Diminished play activity should be viewed as a potential sign of pain requiring an analgesic trial of 2-4 weeks duration. Favorite toys and play activities may vary from day to day. Caregivers should offer options as needed. Toys and feeding games that stimulate mental activity are beneficial. Electronic games on tablets such as Friskies App for cats offer alternatives for mental stimulation. Rather than placing it in a bowl, simply hiding kibble throughout the house at mealtime can provide a unique hunting experience for any age cat.

End of life decision making
It is the clinician and veterinary team’s role to help the caregiver understand what is normal for their cat and how this normal changes with advancing age. Encouraging the caregiver to record their cat’s daily behaviors will improve the ability to identify diminishing quality of life. 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. Regular contact with the caregiver and patient through 2-3 health checkups per year as well as frequent weigh ins can improve the bond and trust between the client and the veterinary team. This type of regular care opens channels of communication necessary as quality of life diminishes and euthanasia decisions need to be made. Quality of life discussions are difficult at best, but they will be made easier by open relationships based on trust and mutual respect.

References
Approach to Gastrointestinal Bleeding

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Gastrointestinal (GI) hemorrhage is an important cause of blood loss that is more commonly encountered in dogs than cats. Sometimes GI bleeding is easy to detect as there are obvious clinical signs, such as hematemesis, melena or hematemesis (overt GI bleeding). Other times animals with GI blood loss do not have overt signs and may present because of anemia; these cases are more challenging to diagnose. If the cause of bleeding can be determined with conventional endoscopy these cases are said to have obscure GI bleeding. If the cause cannot be found during endoscopy these cases are said to have occult GI bleeding. This lecture mainly focuses on how to approach a case with GI bleeding but also discusses treatment for GI ulceration, which is its most common cause.

Causes of GI bleeding

In basic terms, there are three things that can cause GI bleeding (from most to least common): GI ulceration, abnormalities of the hemostatic system, and vascular disorders. Common causes of GI bleeding are listed below:

**Drugs**
- Non-steroidal anti-inflammatory drugs (especially ibuprofen)
- Glucocorticoids (especially dexamethasone)
- Anti-thrombotic drugs (aspirin, clopidogrel)
- Heparin

**Systemic/metabolic disease**
- Hepatic disease (portal hypertension, intrahepatic congenital portosystemic shunts)
- Uremia
- Pancreatitis
- Hypoadrenocorticism

**Neoplasia**
- Adenocarcinoma
- Lymphoma
- Mast cell tumor
- Gastrinoma

**Infectious**
- Parasitic e.g. hook worms
- Bacterial e.g. *Clostridium difficile*
- Viral e.g. parvovirus
- Fungal e.g. *Histoplasma sp*
- Algal e.g. *Prototheca sp*

**Hemostatic disorders**
- Coagulopathies e.g. coumarone toxicity
- Thrombocytopenia
- Platelet function disorders

**Other**
- Trauma e.g. foreign bodies
- Poor perfusion
- Stress
- Caustic agents
- Vascular ectasia

Approach to GI bleeding

**History and physical examination**

The client should carefully be questioned about potential access of the pet to NSAIDs and coumarone rodenticides. As previously mentioned the absence of hematemesis, melena, and hematochezia does not rule out GI bleeding. Dogs and cats with obscure/occult GI blood loss may present for non-specific signs such as anorexia, lethargy, behavioral changes, signs consistent with anemia, or they may not have any signs.

Note that hematemesis should be differentiated from hemoptysis (see below). Dogs and cats can swallow blood from epistaxis or hemoptysis and so respiratory and nasopharyngeal disease are possible differential diagnoses. Fresh blood in the feces tends to
indicate large bowel disease whereas melena indicates gastric or small intestinal bleeding. However, it is possible to have fresh blood in the feces in an animal bleeding from its small intestine if a large amount of blood passes quickly through the GI tract.

**Hemoptysis**

- Bright red blood
- Sputum present
- Frothy or clotted appearance
- pH>7
- No nausea or vomiting
- Respiratory difficulty

**Hematemesis**

- Dark red/brown blood
- astic contents present
- Coffee ground appearance
- pH<7
- Nausea and vomiting
- No respiratory difficulty

The patient’s vital parameters should be assessed first as occasionally patients with GI bleeding can present with hypovolemic shock, which may necessitate immediate intervention. A rectal examination should be carefully performed to look for blood (fresh or altered) and feel for masses and foreign bodies. The patient should be carefully examined for evidence of a generalized hemostatic disorder. Abdominal palpation is also essential. As mast cell tumors can causes GI bleeding the patient should be examined for cutaneous/subcutaneous masses, which if present should be aspirated.

**Laboratory testing**

Animals with GI bleeding are often anemic. However, with peracute hemorrhage their hematocrit and total solids may actually initially be normal. Obscure/occult GI bleeding may only be suspected after a CBC is performed. During the preregenerative stage, the anemia is normocytic normochromic with few reticulocytes. With time the bone marrow will mount a regenerative response but prolonged GI bleeding can lead to iron deficiency anemia. This is typically microcytic hypochromic with a variable reticulocyte count. Gastrointestinal bleeding may be accompanied by thrombocytopenia due to consumption or thrombocytosis, which is secondary to iron deficiency. Severe thrombocytopenia (<30,000 /μL) is associated with spontaneous bleeding but automated counts must be confirmed with microscopic evaluation of a blood smear.

Hypoproteinemia and an increased BUN/creatinine ratio are characteristic of GI blood but loss. However, the absence of these changes does not rule out GI blood loss and neither is specific for this problem.

Fecal occult blood tests are often used in human medicine and can detect blood loss at levels of only 2-5% of those needed to result in melena. The problem in veterinary medicine is that false positive results due to various dietary constituents. I very rarely perform fecal occult blood tests because of this limitation. If an immunochemical test can be developed that is specific for canine hemoglobin it would allow the development of a more specific and useful assay.

Other laboratory tests can be useful when working up a case with known/suspected GI bleeding. Coagulation testing is helpful to rule out coagulopathies as a cause of bleeding. A fecal float and direct smear exam should be performed to rule out parasites. Depending on the index of suspicion further infectious disease testing may also be indicated. Hypoadrenocorticism should be considered and in the absence of a stress leukogram testing of this endocrinopathy should be performed. Gastrinomas are an uncommon tumor but measurement of serum gastrin concentrations is available form a number of laboratories and should be performed prior to starting antacid therapy.

**Diagnostic imaging**

Abdominal radiographs rarely lead to a definitive diagnosis in cases with GI bleeding but could lead of the detection free fluid in the abdomen, pneumoperitoneum, foreign bodies and masses. Barium swallow studies have largely been superseded by other techniques and are seldom performed where I practice.

Abdominal ultrasound can allow the diagnosis of GI ulceration in some cases but false negative studies are definitely possible. GI ulcers are characterized by thickening around their periphery, possible loss of wall layering, and a defect or crater. Ultrasound is useful for detecting abdominal masses and lesions of other organs and so is a valuable tool in the investigation of GI bleeding.

Conventional endoscopy is the definitive way to diagnose gastroduodenal ulceration and also allows assessment of the colon (and distal ileum). It is possible to biopsy any lesions that are detected, although the center of ulcers should not be biopsied due to the risk of perforation. However, it is not possible to reach the entire intestinal tract using this technique and so it is possible to miss important lesions.
Recently a capsule endoscopy system marketed for use in dogs >6 kg in weight (ALICAM, Infiniti Medical) has become available. Capsule endoscopy is indicated where endoscopy does not localize the source of GI bleeding or perhaps in practices where endoscopy is not available. Capsule endoscopy obviously does not allow biopsy.

**Surgery**

Sometimes exploratory laparotomy is employed to look for or address the cause of GI bleeding. However, it can be difficult to detect the site of hemorrhage as surgical exploration does not allow assessment of the mucosal surface of the GI tract. One technique that can occasionally be useful is to combine an exploratory laparotomy with endoscopy. The surgeon can then help guide the endoscope further into the intestinal tract by pulling intestine over it, thus allowing the entire GI tract to be visualized.

**Treatment of GI ulceration**

**Histamine-2 receptor antagonists**

Histamine-2 receptor antagonists (H2RAs) reversibly inhibit H2-receptors thereby reducing gastric acid secretion. These drugs are generally well tolerated and cause few adverse reactions. The kidneys excrete them and so it may be wise to decrease the dose at which they are given in patients with renal failure. They can be ranked by their potency (highest to lowest): famotidine, ranitidine, nitazidine, cimetidine. Cimetidine is not routinely used in dogs and cats as it needs to be dosed every 6 hours and has limited efficacy while the dose of nitazidine is not well established in these species. Although ranitidine and famotidine are frequently administered to dogs and cats, recent studies have called their efficacy into question. In one study assessing the suppression of gastric acid production in dogs, ranitidine was not shown to be superior to a saline placebo. Famotidine is more effective than ranitidine but its ability to increase the gastric pH of dogs did not meet targets established in humans i.e. intragastric pH >3 for at least 75% of the day. Therefore, the use of famotidine and other H2RAs is not optimal in dogs and cats with known gastroduodenal ulceration. However, some patients do appear to respond to famotidine or even ranitidine, possibly because they have milder disease.

**Proton pump inhibitors**

Proton pump inhibitors (PPIs) irreversibly bind to active proton pumps, inhibiting gastric acid production. Omeprazole and pantoprazole are the most commonly used PPIs in small animal practice. Several studies demonstrated that they provide more profound suppression of gastric acid production than H2RAs. Therefore, they are recommended for the treatment of dogs and cats with known/suspected gastroduodenal ulceration or those at high risk of developing ulcers. Omeprazole is more effective when given at a dose of 1 mg/kg PO q12 hours rather q 24 hours. Pantoprazole can be given IV to dogs and cats that cannot tolerate oral medications. These drugs ideally should be given one hour before meals and take several days to reach maximal efficacy. Despite this, combined treatment with famotidine does not appear to be advantageous. Omeprazole capsules and tablets are enteric coated and therefore should not be split or crushed. It is possible to compound an oral suspension for smaller patients. Proton pump inhibitors appear to be well tolerated in dogs and cats, although diarrhea is occasionally reported as a side effect. Preliminary data suggests that cats treated with omeprazole for 60 days may show mild decreases in bone mineral density and that rebound hyperacidity can occur when this drug is stopped. Because of the potential for the development of rebound hyperacidity it is advisable to taper the dose of PPIs prior to stopping them. The optimal way in which to do this is not known but a two-week taper after long-term treatment seems reasonable. This drug has also been shown to alter the oropharyngeal and gastrointestinal microbiota of dogs. This in theory could predispose at risk patients to aspiration pneumonia. Therefore, the indiscriminate use of PPIs (and other antacids) is not advised. Proton pump inhibitors are metabolized by hepatic cytochrome P450 enzyme and so can potentially interfere with the metabolism of other drugs including benzodiazepines, clopidogrel, cyclosporine, azole antifungal drugs, and rifampin.

**Sucralfate**

Sucralfate is an ionic sulfated disaccharide part of which binds proteins in the stomach exposed by mucosal damage. This forms a physical barrier that protects the damaged mucosa from gastric acid, pepsin, and bile. Additionally, sucralfate stimulates the production of mucus, bicarbonate, epidermal growth factor, and prostaglandin E, all of which can have an additional cytoprotective effect. Sucralfate is indicated to patients with gastroesophageal reflux or gastroduodenal ulceration. The author usually uses sucralfate in conjunction with a PPI. Sucralfate is well tolerated, as it is not systemically absorbed. However, it can interfere with the absorption of concurrently administered medications and therefore other oral drugs should be given at least two hours before sucralfate.

**Disclosure**

Dr. Lidbury has acted as a paid speaker for and has received funding in support of research from Infiniti Medical the manufacturer of the ALICAM capsule endoscopy system.

**References/further reading**


Dogs are often presented to veterinarians for chronic (>3 weeks’ duration) gastrointestinal (GI) signs such as vomiting or diarrhea. While acute GI signs are often self-limiting and may not require extensive diagnostic evaluation, dogs with chronic GI signs require further assessment. There are many causes of chronic GI signs in dogs and therefore it is essential to have a logical approach to these cases. This session outlines an approach to dogs with chronic vomiting/diarrhea mainly focusing on diet responsive, antibiotic responsive, and immunosuppressive responsive enteropathy (sometimes called steroid responsive enteropathy). Protein losing enteropathy will be discussed in a separate session.

**Terminology**

The terminology used to describe chronic GI disease is important to understand but can be confusing:

**Inflammatory bowel disease**

This commonly used term has been borrowed from human medicine where its two main forms are Crohn’s disease and ulcerative colitis. The WSAVA GI Standardizations Group defined inflammatory bowel disease in dogs (and cats) as a group of idiopathic, chronic gastrointestinal disorders characterized by mucosal inflammation. The are some important differences between IBD in humans and dogs, e.g. IBD is mainly a colonic disease in people whereas it mainly effects the small intestine of dogs. In actual fact IBD may not be a helpful term for veterinarians to use.

**Chronic enteropathy**

Chronic enteropathy (CE) is defined as chronic GI signs e.g. vomiting, diarrhea, borborygmus, hyporexia, abdominal pains, nausea, and or weight loss) where extraintestinal, infectious, and intestinal disease of other etiology e.g. foreign bodies, intussusception, and tumors have been ruled out. This term is more helpful for veterinarians to use than IBD as it reflects how we work these cases up and the differences between IBD in humans and small animals. Dandrieux (2016) divided CE into diet responsive enteropathy (DRE), food responsive enteropathy (FRE), immunosuppressant responsive enteropathy (IRE), and non-responsive enteropathy (NRE).

**Initial workup**

The first priority is to rule out extraintestinal disease, infectious disease, foreign bodies, intussusception, and tumors. When initially presented with dogs with chronic GI signs often my initial action is to have a fecal sample sent for floatation and a direct smear exam to rule out parasitism, I will often treat with fenbendazole for 3-5 days regardless of the result in case of a false negative result. Often at this stage I will try the dog on an “intestinal” type diet (discussed below). If the dog fails to respond to these interventions my next step is to rule out metabolic disease by running a CBC, serum chemistry profile, and urinalysis. Remember that atypical hypoadrenocorticism can cause GI signs without electrolyte abnormalities. If the patient does not have a stress leukogram I will therefore measure their baseline serum cortisol concentration or perform an ACTH stimulation test. At this stage I will often do additional infectious disease such as antigen testing for Giardia. Abdominal imaging especially ultrasound is helpful to rule out obstructions or masses. In many cases the results of ultrasound do not directly contribute to making a diagnosis but this imaging modality appears to most useful in patients that are vomiting, have weight loss, or have a palpable abdominal mass. Pancreatitis and exocrine pancreatic insufficiency can be tested for using serum cPLI and cTLI tests, respectively. Measurement of serum cobalamin and folate concentrations may give an indication of small intestinal absorption. If the serum cobalamin concentration is low it may be beneficial to supplement it by the subcutaneous (or oral) route.

For stable dogs the next step is to perform dietary/therapeutic trials. These need to be performed in a logical way in order to get the best information from them. Ideally only one intervention at a time is made. If more than one change is made it can be difficult to determine which intervention was responsible. Of course, this is more of a guideline than a rule. For example, in a very sick patient it may be necessary to be aggressive and try several things at once. It is also important to perform the trial in a way in which they are likely to help the patient and to for a duration long enough for them to have an effect.

**Diet responsive enteropathies**

This is the most common form of CE in dogs, accounting for about two thirds of patients in some studies. Therefore, it is advisable to perform a diet trial before trying antibiotics or intestinal biopsy in most cases. These dogs tend to have a good long-term outcome compared to those with other kinds of CE. Dogs that respond to a diet trial don’t necessarily have a dietary allergy as dietary intolerance and intoxication are other potential types of adverse food reactions. Dogs with DRE more often have large bowel signs than those with other forms of CE and may also have dermatological signs. Additionally, properties of some of the diets used in diet trials themselves can have a beneficial effect on GI. The options for types of diet to feed are reviewed below:
“Intestinal” type diets are typically highly digestible, with moderate fat restriction (this varies). These diets may also be supplemented with fermentable fibers, which can help modulate the GI microbiota and omega 3 fatty acids that may reduce inflammation. I use them as first step early in the work up of chronic GI signs but novel protein and hydrolyzed antigen diets may be more effective.

Novel protein diets are commercially available and can also be home cooked. The idea is to provide a novel protein source e.g. venison, rabbit, duck, kangaroo. It is essential to take a thorough dietary history to feed a truly novel protein and the closer the taxonomic relationship between meat sources, the higher the risk of cross-reactivity e.g. if a dog has previously been exposed to chicken a turkey based diet would be less likely to be helpful.

Hydrolyzed antigen diets contain small peptides that are less likely to be immunogenic than proteins. In one study dogs with CE fed a hydrolyzed antigen diet had a more favorable response than those fed an easily digestible diet. It is not known whether novel protein or hydrolyzed antigen diets are more effective.

Supplementation of fermentable fiber is a reasonable option in dogs with large bowel signs. This can be added to another diet e.g. as Metamucil or canned pumpkin or there are gastrointestinal diets with added fiber (e.g. Royal Canin Gastrointestinal Fiber Response).

When a feeding trial is important that the trial diet is fed exclusively. This requires an informed and compliant client. Usually for GI disease a response can be seen within 2 weeks (animals can continue to improve beyond this point). If there is a response to fully document the presence of an adverse food reaction the original diet should ideally be reintroduced (I rarely do this). Interestingly, it has been reported that when dogs with FRE were fed an elimination diet for 12 weeks and then were switched back to their previous diet many of them remained in remission. If a dog fails to respond to a trial with one diet it does not mean that it will also fail to respond to others.

Antibiotic responsive enteropathy

There are various reasons why a dog may respond to antibiotics: specific enteric infections e.g. Campylobacter or Salmonella; granulomatous colitis (of Boxers); or because of dysbiosis. This session will not focus of specific enteric infections. Intestinal dysbiosis, an imbalance of the GI microbiota often occurs secondary to other GI diseases, but may also occur as primary entity, or could also contribute to the development of IRE or IBD. Therefore, if a dog fails to respond to a diet trial it is reasonable to perform an antibiotic trial. Common choices include tylosin or metronidazole. My preference is tylosin (25 mg kg PO q12 hours) because it has few side effects, a wide therapeutic range, and is rarely used to treat other infections. Dogs with tylosin responsive diarrhea tend to be younger than those with other forms of CE and are often larger breeds, especially German Shepherds. Frequently, these dogs have large bowel diarrhea. Usually if there is a response to tylosin it happens quickly, often within the first few days of treatment. Therapy is usually continued for 6 to 8 weeks. Some dogs will remain in remission when the tylosin is discontinued but many others relapse and require intermittent or long-term treatment. For long term treatment, a dose reduction is often possible (taper gradually to 5-10 mg/kg PO q24 hours). Interestingly, when given to healthy dogs, tylosin appears to cause a dysbiosis which in some individuals does not resolve after several months of follow-up. This is another reason why a diet trials should be performed before antibiotics are started.

Prebiotics, probiotics, symbiotics, or even fecal microbial transplant also have the potential to help address primary or secondary dysbiosis and to modulate the immune system in dogs with CE. However, there is not enough evidence available to make definitive recommendations on when and how to use them and so they are currently used on an empirical basis. I tend to reserve these options for refractors case of CE but it may also be reasonable to try them at an earlier juncture.

Immunosuppressive responsive enteropathy

If stable patients if a diet trial(s) and antibiotics trial are unsuccessful ideally the next step is to perform intestinal biopsy. This allows a histomorphological diagnosis and sometimes a causative diagnosis to be made. In most dogs with CE, histological assessment allows the confirmation, classification, grading of enteritis. Additionally, neoplasia and intestinal lymphangiectasia can be ruled in or out. It is important to remember that lymphoplasmacytic enteritis can be caused by a number of different things and is not a useful clinical diagnosis. Once other causes of clinical signs have been ruled out, trial treatment with an immunosuppressive medication can begin. Prednisone/prednisolone (2 mg/kg PO per day) are the most frequently used initial drugs. In most studies, initial response rates are >60%, although long-term outcomes are less favorable. The addition of metronidazole in the IRE dogs treated with prednisone did not appear to be beneficial. Budesonide has fewer systemic side effects then other glucocorticoids as it undergoes extensive first pass metabolism. A study suggested that it is associated with a similar response rate to prednisolone. In animals that fail to respond to these medications second line treatments are indicated. These include cyclosporine, chlorambucil, azathioprine, and mycophenolate. My preference is cyclosporine (5 mg/kg PO q24 hours) as results of initial studies using this agent were encouraging. Chlorambucil (2-4 mg/m² PO q24 hours) was reported to be more effective than azathioprine when each drug was given with prednisolone for treatment of PLE. However, further studies are needs before definitive recommendations can be made.
Reasons for a more aggressive course of action

This step-wise system of trial treatments is very helpful for stable case but is not appropriate for very sick patients. For these dogs, being more aggressive and collecting intestinal biopsies at an earlier stage is indicated. Often multiple interventions are made simultaneously e.g. in a dog with severe enteritis starting prednisolone, a diet trial, and tylosin all together. If the patient responds the prednisolone could be tapered and possibly discontinued. After that it may be possible for the tylosin to be discontinued and then the dog might be maintained on a therapeutic diet alone. Possible indications for a more aggressive approach are listed below:

- Protein losing enteropathy (see other session)
- Anorexia
- Severe weight loss/other clinical signs
- An intestinal mass or diffuse thickening

Scoring systems

Assessing the dog’s response to various interventions is an essential part of working up CE. In order to make this a more subjective process two clinical scoring scheme have been developed; the canine inflammatory bowel disease activity index (CIBDAI) and the canine chronic enteropathy activity index (CCEACI). The former is based on clinical findings alone that later is based on clinical findings and serum albumin concentration. I recommend the use of these scoring systems in dogs with CE.

References/further reading


Canine Chronic Pancreatitis: A Diagnostic and Therapeutic Challenge
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The differentiation between acute pancreatitis (AP) and chronic pancreatitis (CP) is ideally made based on histological criteria. Acute pancreatitis is a completely reversible condition with no histological evidence of fibrosis or exocrine atrophy. Acute pancreatitis is histologically characterized by necrosis, edema, and a neutrophilic infiltrate. Chronic pancreatitis is defined as a continuing inflammatory disease of the pancreas with irreversible morphological changes. The typical histological changes of CP are fibrosis, atrophy, and a mononuclear cell infiltrate. These in time may lead to “pancreatic failure” with exocrine pancreatic insufficiency and/or diabetes mellitus. In reality, the antemortem distinction between AP and CP is rarely made based on histological findings as pancreatic biopsy is seldom performed. Unfortunately it is not possible to reliably make a differentiation between AP and CP based on the duration of signs alone because acute flare-ups of CP can occur, AP can develop into CP, AP can be recurrent, and CP can present with acute clinical signs.

In studies of dogs undergoing necropsy for a variety of reasons, histological changes of the pancreas were very common, in fact only 8% of pancreata examined did not have any histological changes. Lymphocytic inflammation and fibrosis suggestive of CP were present in about half of the pancreata examined. Neutrophilic inflammation consistent with AP was present in about a third of the pancreata. This is intriguing as it suggests that pancreatitis, especially CP, is more common than we previously thought and may often go undiagnosed. The importance of pancreatic lesions in dogs that have no clinical signs is not known.

Chronic pancreatitis is more challenging to diagnose than acute pancreatitis and evidence-based treatment protocols have not been established. This session discusses the diagnosis and management of this challenging disease.

Etiology and risk factors
The causes of CP in dogs are not well defined but progression from AP probably accounts for at least some cases. Known/suspected risk factors for AP in dogs that may also be relevant for CP are listed below. Cavalier King Charles Spaniels, English Cocker Spaniels, and Collies from the United Kingdom were shown to be at increased risk. English Cocker Spaniels have been reported to have a form of CP, characterized by perilobular fibrosis, duct destruction, and a T-cell-dominated lymphocytic infiltrate. Some affected dogs also had keratoconjunctivitis sicca and glomerulonephritis. There are similarities between CP in this breed and type 1 (IgG4+) autoimmune pancreatitis in humans leading to speculation that this is a cause of CP in English Cocker Spaniels. It is not known if dogs get autoimmune pancreatitis but lymphoplasmacytic infiltrates consistent with this etiology are not uncommonly observed on histological sections from dogs of various breeds. In some humans, genetic variations play a role in the development of pancreatitis and it is possible that this is the case in dogs. For example, some humans have a genotype that results in translation of non-functional PSTI peptide predisposing them to pancreatitis. Recently, variations of the SPINK1 gene that encodes the PSTI protein in Miniature Schnauzers were shown to be associated with pancreatitis in this breed. However, a subsequent study did not reproduce this result and further work needs to be done to determine if these variations result in synthesis of non-functional PSTI. Other breeds of dogs have not been evaluated for these variations. Pancreatic duct obstruction is a common cause of CP in humans but is only occasionally diagnosed in dogs.

Known/suspected risk factors for AP that are relevant for CP
- Hypertriglyceridemia
- Diet (ingestion of an unusual food item, getting into garbage, and possibly high fat diets)
- Endocrinopathies (possibly hypothyroidism, hyperadrenocorticism, and diabetes mellitus)
- Drugs (possibly azathioprine, L-asparaginase, potassium bromide, phenobarbital, antimonial drugs, and organophosphates)
- Obesity

Clinical signs
In a recent retrospective study of dogs with CP the most common clinical signs recorded were lethargy (80%), decreased appetite (70%), vomiting (63%), diarrhea (36%), abdominal pain (27%), and pyrexia (24%). From this data, it is evident that some dogs with CP will present with classic signs of pancreatitis but others have a vaguer presentation. For this reason, Watson (2012) recommended that CP should be considered as a diagnosis in dogs with low grade waxing and waning gastrointestinal (GI) signs, intermittent anorexia and apparent postprandial discomfort, recurrent acute signs of pancreatitis, dogs with diabetes, in older dogs that develop signs of exocrine pancreatic insufficiency (EPI), and dogs with extrahepatic bile duct obstruction.
Diagnostic testing
Chronic pancreatitis is more challenging to diagnose than AP for a number of reasons. Firstly, as previously mentioned the clinical signs of CP can be vague so this condition may not be considered to be a possible diagnosis in the first place. Chronic pancreatitis is harder to diagnose with abdominal ultrasound than AP. Furthermore, based on studies of dogs undergoing necropsy it is probable that the Spec cPL and SNAP cPL tests are not as sensitive for diagnosing CP as they are for AP. Having said this, I usually diagnose CP based on compatible clinical signs, ruling out other causes of these, abdominal ultrasound findings, and the results of a Spec cPL assay. I seldom perform pancreatic biopsy on dogs I suspect of having CP. However, I would consider pancreatic biopsy if a dog is undergoing laparotomy for another reason and I suspected CP. It can also be difficult to determine if a dog has CP or AP.

Diagnostic imaging
Abdominal radiographs are not adequately sensitive for the diagnosis of canine pancreatitis. However, this imaging modality does play an important role in ruling out other causes of vomiting in dogs and therefore is relevant in the work-up of dogs suspected to have pancreatitis.

Abdominal ultrasonography is the most commonly used imaging modality to diagnose pancreatitis in dogs. Findings consistent with AP include peripancreatic fluid, hyperechoic peripancreatic fat, and an enlarged hypoechoic pancreas. The diagnostic performance of this test for AP has not adequately been evaluated but one study reported a sensitivity of 68%. The changes associated with CP such as fibrosis and atrophy are harder to detect with the imaging modality resulting in a lower sensitivity. If stringent criteria for diagnosing pancreatitis are used ultrasound is probably quite specific for diagnosing AP but false positives results can occur (for CP this has not been reported). Furthermore, the accuracy of this technique is very operator-dependent. Abdominal ultrasound is useful for ruling out other causes of the patient’s clinical signs and for detecting complications of pancreatitis such as pancreatic fluid accumulations, extrahepatic bile duct obstruction, or pancreatic masses.

In humans, contrast enhanced computed tomography is often used to diagnose pancreatitis. Results of initial studies in dogs were not promising but a subsequent a study suggested that this technique is feasible for the diagnosis of AP. The utility of contrast enhanced ultrasound for diagnosing CP also warrants further evaluation.

Pancreatic biopsy
Histological examination of a pancreatic biopsy specimen is theoretically the gold-standard test for diagnosing pancreatitis in dogs. Pancreatic biopsy can carefully be performed without complication in healthy dogs and so it is technically possible. However, pancreatic biopsy is invasive and requires general anesthesia. Additionally, sampling error is a diagnostic limitation of this technique as the lesions associated with CP may be focal.

Treatment
Supportive care
Fluid therapy is essential for acute episodes of pancreatitis but will not be reviewed here.

Anti-emetics are also an important part of treatment as they reduce fluid losses, improve patient comfort, and help allow early enteral nutrition to be provided. The NK-1 receptor antagonist maropitant is my first choice for dogs with pancreatitis. This drug is very effective and has both peripheral and central effects. Another advantage is that this drug inhibits the effect of substance P and therefore may have an analgesic effect. Ondansetron and dolasetron are serotonin receptor (5-HT3) antagonists that are also effective antiemetic agents and in severe cases of pancreatitis I often use one of these in conjunction with maropitant. Metoclopramide is a dopamine receptor antagonist that has antiemetic and prokinetic effects. It is not as effective as the agents discussed above but sometimes I will add it to the treatment regimen of dogs that are unresponsive to other antiemetics or those that have gastric stasis. I find it most effective when given IV as a constant rate infusion and so it has less relevance in a chronic setting.

Abdominal pain is likely to be under-recognized in dogs and so I often give patients with CP the benefit of the doubt by giving them an analgesic and seeing if they improve. There are several options for providing analgesia in the hospital setting but options for providing analgesia at home are more limited i.e. tramadol or butorphanol given orally. Butorphanol is not likely to be effective analgesic given this way and I don’t send patients home with fentanyl patches for a variety of reasons. Therefore, currently I use
tramadol, realizing that it may not be that efficacious. I don’t use NSAIDs in dogs with CP due to concerns about GI and renal side effects. There is a rationale being using gabapentin or pregabalin in these dogs but I have not personally had to do this. As previously mentioned, maropitant may have analgesic effects.

**Nutrition**

Nutritional considerations are very important in the management of dogs with pancreatitis. It is now recommended to provide nutrition to dogs early in the course of pancreatitis. The question of which is the optimal diet to feed dogs with pancreatitis has not been rigorously answered, but given the role of obesity and hypertriglyceridemia in causing pancreatitis, feeding an ultra-low-fat diet is recommended. Because patients with pancreatitis are in a catabolic state feeding high-fiber weight loss diets does not make sense. Because of this I advise feeding an ultra-low-fat highly digestible diet.

**Eliminate risk factors**

In dogs with CP it is important to look for any risk factors such as hyperlipidemia or drugs known to be associated with pancreatitis. If possible these risk factors should be addressed. For example, in persistently hyperlipidemic dogs, in addition to feeding an ultra-low-fat diet supplementing omega-3 fatty acids and/or initiating treatment with gemfibrozil (150-300 mg per dog PO q12 hours) may be helpful.

**Other treatments**

It is important to have an index of suspicion for complications of CP such as EPI and diabetes mellitus and these should be tested for and treated if necessary. The use of pancreatic enzyme replacement products to decrease post-prandial pain has been described in dogs and humans but there is little evidence to support this practice. As oxidative damage can play a role in the pathogenesis of pancreatitis there is a rationale for using antioxidants such as S-adenosylmethionine but there are currently no studies in dogs with pancreatitis to support this. Some dogs with CP have lymphoplasmacytic pancreatic infiltrates and this may indicate an autoimmune etiology. Therefore, in a dog with CP that is not responding to symptomatic therapy and dietary changes, a trial treatment with prednisone may be warranted. Again, there are no clinical trials to support this. There is however an ongoing clinical trial that is being coordinated by the Gastrointestinal Laboratory at Texas A&M University to evaluate the efficacy of cyclosporine for treating dogs with CP and diabetes.
Defining the cat’s problems
The first step in working up a cat with chronic diarrhea is to make an accurate list of the cat’s problems. Trying to determine if the diarrhea is small bowel, large bowel, or mixed in nature seems very obvious but clinicians often skip this step. Determining this helps when it comes to formulating a list of differential diagnoses and making a diagnostic plan. Having said this, sometimes it is oversimplistic to say the diarrhea is from either the large or small bowel as occasionally both are affected. Aside from helping to localize the site of disease the character of the cat’s stool can give other important diagnostic clues. For example, steatorrhea is consistent with exocrine pancreatic insufficiency, and especially foul smelling large bowel diarrhea with *Tritrichomonas foetus* infection. It is important to take note of non-gastrointestinal clinical signs as they can indicate metabolic causes of diarrhea. For example, polyuria/polydipsia would be consistent with hyperthyroidism, hypercalcemia, or renal disease.

Formulating a differential diagnosis list
The next step when working up a cat with chronic diarrhea is to formulate a list of relevant differential diagnoses. Again this may seem obvious but it can be very tempting to skip this step and try to reach a diagnosis by pattern recognition alone, or just to think of a list of the diagnostic tests that you will perform. The problem-oriented approach is especially useful for more complicated or atypical cases and for less experienced clinicians. It is important to recognize which of these diseases are more or less likely based on the patient’s signalment. For example, in cats less than two years old infectious and dietary responsive causes of diarrhea are common whereas in older cats inflammatory bowel disease and intestinal lymphoma are more likely. This helps the clinician go from a list of all the possible causes of the clinical signs to a list of the probable causes for that patient.

Causes of feline chronic diarrhea

**Extra-intestinal disease**
- Hyperthyroidism*, hepatobiliary disease, pancreatitis, exocrine pancreatic insufficiency, hypercalcemia, renal disease, peritonitis, toxemia/septicemia

**Infectious agents**
- Helminths, protozoa (*Tritrichomonas foetus* *, Giardia** **, *Cryptosporidium*), viral (Feline corona virus/feline infectious peritonitis, FeLV, FIV), bacterial (*Salmonella, Campylobacter, Clostridium*), fungal (*Histoplasma*)

**Inflammatory disease**
- Inflammatory bowel disease (IBD)* **
- Dietary responsive enteropathy

**Dietary allergy** **, dietary intolerance** **

**Neoplasia**
- Intestinal lymphoma*, mast cell tumor, carcinoma, gastrinoma (rare)

**Drugs**
- Non-steroidal anti-inflammatory drugs, antimicrobials, cancer chemotherapeutic agents

**Other**
- Dysbiosis (it is controversial if dysbiosis is a primary cause of diarrhea in cats)
  - *- a common cause of chronic small intestinal diarrhea
  - **- a common cause of chronic large intestinal diarrhea

Staged diagnostic approach
Probably the most important thing when approaching a cat (or dog) with chronic diarrhea is to have a logical staged approach to performing diagnostic testing. Initially, cheaper less invasive tests are performed in order to rule out metabolic and infectious diseases. As there are no reliable diagnostic tests for dietary allergy/intolerance or dysbiosis, diagnostic trials are an important part of this process. Ideally therapeutic trials are performed sequentially rather than in parallel, so if there is a positive response the clinician knows what it was due to. If there is no response to these therapeutic trials and no diagnosis is made after performing the initial diagnostic tests, more expensive and invasive tests are performed. In some cases intestinal biopsy is indicated later in the diagnostic process. Every case is different but general guidelines for this staged approach are detailed below:

**Stage 1: initial evaluation**
- Perform a fecal direct smear and floatation to rule out helminth infection
- Consider administering a broad spectrum anthelmintic, such as fenbendazole at a dose of 50 mg/kg by mouth once daily for three days, regardless of the fecal analysis results
• Perform a diet trial with a highly digestible intestinal diet

Stage 2: non-invasive testing
• Rule out metabolic/systemic disease by performing a complete blood count, serum chemistry panel, urinalysis, and thyroid function testing. Evaluation of a serum chemistry panel can also be helpful to determine if there are systemic complications of gastrointestinal disease or if there are any concurrent diseases.
• Perform further infectious disease testing
  o *Giardia/Crytosporidium* antigen testing should be sent to a commercial laboratory
  o *Trichomonas fetus* fecal PCR. This parasite is an important cause of diarrhea in cats, especially in cats <12 months old that have come from a shelter or a pedigree breeding colony
  o FeLV/FIV testing
  o If indicated evaluate the cat for FIP
• Perform non-invasive tests for gastrointestinal disease in cats with small intestinal diarrhea or cats with large intestinal diarrhea in which concurrent small intestinal disease is suspected
  o Serum pancreatic lipase immunoreactivity (pPLI) to screen for pancreatitis
  o Serum trypsin-like immunoreactivity (tTLI) to diagnose exocrine pancreatic insufficiency
  o Serum cobalamin and folate to screen for small intestinal malabsorption. It is possibly for a cat with small intestinal disease to have serum cobalamin and folate concentrations within the reference interval.
  o Abdominal ultrasound examination seldom leads to a definitive diagnosis but can be helpful in directing further diagnostic testing. If intestinal thickening is present this is consistent with intestinal lymphoma (especially if the muscularis layer is thickened) or inflammatory bowel disease. This may prompt the decision to recommend intestinal biopsy. Ultrasound examination may also help determine if there is concurrent pancreatitis and/or hepatobiliary disease.

Stage 3: therapeutic trials
• The cat should be supplement with parenteral cobalamin if indicated. I supplement cats with serum cobalamin concentrations less than 400 ng/L.
• The only reliable way to diagnose dietary intolerance/allergy is to perform a dietary trial. If the cat has failed to respond to a trial with an “intestinal” diet the next step is to feed a novel protein or hydrolyzed antigen diet. If the cat will eat one of these diets they should be fed exclusively. Gastrointestinal disease usually responds more quickly to a successful dietary trial than dermatological disease but the trial should last for a minimum of three weeks. Another option that can be helpful in some cats with diarrhea is to feed a higher protein lower carbohydrate diet.
• Consider a therapeutic trial with tylosin or metronidazole for dysbiosis. Cats with chronic enteropathies have been shown to have a different intestinal microbiota than healthy cats. However, it is controversial if dysbiosis in cats is a primary disease or if it occurs secondary to another disease process. I routinely perform a tylosin trial in dogs with chronic diarrhea but I do not do so with cats. However, I have seen some cats with chronic diarrhea that respond to tylosin but not to other medications including prednisolone. Other cats have responded well to probiotics but not to other medications.

Stage 4: intestinal biopsy
If a diagnosis has not been reached or the cat has not responded to any intervention, the next step is to recommend intestinal biopsy. This can be performed endoscopically or surgically during a laparotomy. Each technique has advantages and disadvantages. Obviously endoscopy is less invasive and the mucosal surface of the gastrointestinal tract can be evaluated. Where I practice, endoscopy is considerably cheaper and faster than laparotomy. The disadvantages of endoscopy are the need for specialized equipment and training to use it optimally, the relatively small size of the biopsy specimens that are collected, and the inability to reach the middle section of the small intestine using conventional techniques. If endoscopic biopsy is selected it is very important to collect multiple biopsies from the stomach, duodenum, ileum, and colon. Where possible both the duodenum and the ileum should be intubated during biopsy collection rather than collecting biopsies blindly. Good technique helps ensure that the full thickness of the mucosa is biopsied. It is also imperative that the specimens are examined at the time of collection to make sure they are adequate, that they are handled correctly by spreading them out on damp sponge mucosal side up.

The advantages of surgical biopsy collection are that the biopsies are of full thickness and that any part of the small intestine can be evaluated. Additionally, some cats with intestinal disease have concurrent hepatobiliary and/or pancreatic disease and laparotomy allows samples of these organs to also be collected. Surgical biopsy is more invasive than endoscopic biopsy and dehiscence of the enterotomy site is a serious potential complication. The suspected site of disease must be considered when choosing a biopsy technique. If the cat is suspected to have large intestinal disease without small intestinal involvement endoscopic biopsy is preferred as surgical colonic biopsies are rarely performed. Whereas surgical biopsy would be a better choice than endoscopy if abdominal
ultrasound examination demonstrates segmental thickening of the jejunum. Laparoscopic biopsy collection when available may offer the best of both worlds.

Stage 5: further treatment based on histological findings
Further treatments are selected based on the histomorphological diagnosis. The most common inflammatory infiltrates are lymphocytes and plasma cells. Lymphoplasmacytic infiltrates (or other types of infiltrate) are not diagnostic for IBD as they can occur due to other conditions such as dietary intolerance or allergies. Therefore, IBD should only be diagnosed when other causes of inflammation have been ruled. Mild lymphoplasmacytic enteritis can be seen in healthy animals and even when standardized criteria are used there is considerable disagreement between pathologists evaluating gastrointestinal biopsy specimens. This can make it difficult for clinicians to make treatment decisions based on histology reports. Additionally, it can be difficult to differentiate between severe lymphoplasmacytic inflammation in cats with IBD and small cell intestinal lymphoma, especially when evaluating endoscopically collected biopsy specimens. The use of immunophenotyping to determine if the cells comprising the infiltrate are T-cells, B-cells, or a mixture and PCR for antigen receptor rearrangements to determine lymphocyte clonality can be helpful in making this distinction. These tests should be used in conjunction with histological evaluation of biopsy specimens.

Some clients will decline intestinal biopsy procedures for financial or other reasons and some cats may not be stable enough to tolerate anesthesia and biopsy. If this is the case, once parasitism, other infectious diseases, extra-intestinal disease, and diet responsive diseases have been ruled out, the two most common differential diagnoses that remain are IBD and small cell lymphoma. Therefore, it is reasonable to perform a prednisolone trial treatment. Cats with IBD or small cell lymphoma often have a positive response to this medication so it is important to counsel clients about both these two possible diagnoses.

When to be more aggressive
Obviously this staged approach is time consuming and for some cats it best to pursue diagnostic testing more aggressively. This often means performing gastrointestinal biopsy before doing therapeutic trials. Indications for this more aggressive approach include but are not limited to: severe weight loss, anorexia, the presence of an intestinal mass, melena, and protein losing enteropathy.

Concurrent pancreatic and/or hepatobiliary disease?
It is important to remember that many cats with diarrhea will have pancreatic and or hepatobiliary disease in addition to intestinal disease. When cats have concurrent IBD and pancreatitis, the pancreatitis should be treated symptomatically and prednisolone should be used to treat the IBD. Often the pancreatitis resolves when the IBD is treated and the prednisolone does not seem to make the pancreatitis worse. The exception to this is when the cat also has concurrent acute neutrophilic cholangitis with a positive bacterial culture (bile or liver). If this is the case the cat should be treated with antimicrobials and possibly ursodeoxycholic acid before treating with prednisolone.

Nutritional support
Some cats with chronic diarrhea are hyporexic or anorexic. These cats may also have reduced absorption of nutrients from the food that they do consume and increased metabolic requirements. This can predispose them to malnutrition and possibly even to hepatic lipidosis. Therefore, it is essential to provide adequate nutrition to these patients. Placement of a feeding tube is therefore often indicated. Feeding tubes can also make it easier for owners to medicate sick cats. Esophageal feeding tubes are easy to place, rarely have serious complications, and can be removed when they are no longer needed. The need for a feeding tube should be considered in cats undergoing anesthesia for intestinal biopsy. Appetite stimulants such as mirtazapine can also used as a short-term measure to get cats to eat. Forced syringe feeding of cats should be avoided as it can lead to food aversion.

It is important to treat cobalamin deficient cats with cobalamin as well as treating their underlying disease process. Cobalamin deficient cats provided supplementation might be less likely to respond to treatment for their underlying disease. Cyanocobalamin is given at a dose of 250 μg per cat SQ once weekly for 6 weeks, then once monthly. Cobalamin also seems to act as an appetite stimulant and may also be administered for this reason.

Treatment tips
In cats with idiopathic inflammatory bowel disease, if the response to prednisolone alone is not optimal, chlorambucil can be used as an adjunctive treatment. There are several dosing protocols that have been reported. For cats with that are relatively easy to pill a dose of 2 mg per cat PO three times a week can be used. In smaller cats or cats that don’t tolerate chlorambucil well the dose frequency can be reduced to twice weekly. For cats that are harder to pill, pulse dosing can be used. This entails giving a dose of 20 mg/m² once every 2 weeks. Generally this drug is very well tolerated in cats but it may cause myelosuppression so complete blood counts should be monitored periodically. Some cats with IBD or lymphoma also have comorbid conditions, such as diabetes, that mean the use of prednisolone is contraindicated. Budesonide is a corticosteroid, which is extensively metabolized on its first pass through the liver. This means it causes fewer systemic side effects than prednisolone. In these cases budesonide can be used instead of prednisolone. However, there may still be some systemic side effects associated with this drug. A dose of 1 mg per cat PO q24 hours has been recommended.
References/further reading
Getting the Most Out of Liver Biopsies
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Although laboratory testing and diagnostic imaging are valuable during the diagnostic investigation of dogs and cats with hepatobiliary disease, liver biopsy is often required to make a definitive diagnosis. Microscopic evaluation of liver tissues allows a histomorphological and sometimes an etiological diagnosis to be made. It can also provide important prognostic information and help guide therapy. Furthermore, liver biopsy allows collection of samples for copper quantification and bacterial culture. In order to get the most out of this process, it is important to pay attention to detail at every step of the process; patient preparation, biopsy, samples handling, and histological assessment.

Indications for liver biopsy
An approach to dogs with increased liver enzyme activities is outlined in my previous lecture. While it is not possible to make a definitive set of criteria for when to perform liver biopsy because every case is different, I can offer the following suggested indications for liver biopsy:

- Persistently increased liver enzyme activities (ALT in dogs, ALT and ALP in cats) where extrahepatic disease has been ruled out
- Icterus/hyperbilirubinemia where hemolysis and extrahepatic bile duct obstruction have been ruled out
- Hepatic masses (avoid incisional, laparoscopic, and needle biopsy with suspected hemangiosarcoma)
- Patients with congenital portosystemic shunts or acquired portosystemic shunts (when pre-hepatic portal hypertension has been ruled out)

Suggested contraindications for liver biopsy

- Platelet count <80,000 /µL
- Prolonged buccal mucosal bleeding time
- Prolonged prothrombin time or activated partial thromboplastin time (>2 times the upper limit of the reference interval)
- Plasma fibrinogen concentration <50% of the lower limit of the reference interval
- Infectious disease that could be disseminated by biopsy
- Suspected hemangiosarcoma (excisional biopsy may be possible)
- Ascites (try to treat first)

Risks of liver biopsy
Liver biopsy is considered to be a relatively low risk procedure but it occasionally leads to morbidity and even mortality. The most common adverse event is excess hemorrhage. In a study of dogs and cats undergoing percutaneous needle biopsy (renal and hepatic), minor bleeding was found to occur in 22% of cases and major bleeding, requiring fluid therapy or a transfusion, occurred in 6% of cases. However, this study did not evaluate renal and hepatic biopsy separately and renal biopsy is generally more prone to excess hemorrhage. Other studies have shown the laparoscopic liver biopsy in dogs was associated with a low complication rate. One complication that is very important to mention is that in a fatal severe shock developed in 5 of 26 cats that underwent liver biopsy with an automated Tru-Cut biopsy device, possibly due to intense vagotonia. For this reason, automated Tru-Cut needles should never be used for liver biopsy in cats.

Limitations of liver biopsy
Liver biopsy has some important diagnostic limitations. No matter which biopsy technique is employed a relatively small amount of the liver a whole is sampled. Because many liver diseases are heterogeneously distributed throughout the hepatic parenchyma, liver biopsy is susceptible to sampling error. This can mean the histological findings reported do not represent the rest of the liver. Bigger specimens of liver are generally more representative and are easier for the pathologist to interpret. In a recent study, the mean number of portal triads obtained were 2.9 for needle samples (14 gauge), 3.4 for 5-mm laparoscopic cup forceps samples, 12 for 8 mm punch samples, and 30.7 for wedge samples. Diagnoses were in agreement with those from large wedge samples in 66% of needle samples, 60% of cup samples, and 69% of punch samples, and these proportions were not significantly different from each other. The investigators concluded that the histopathologic interpretation of a liver biopsy specimen in the dog is unlikely to vary if it contains at least 3 to 12 portal triads. These values should be seen as a minimum and it has been recommended that pathologists should be presented with specimens containing at least 11 portal triads. In another study, the morphologic diagnosis assigned to 18 gauge needle biopsy specimens by individual examiners agreed with the morphologic diagnosis assigned to larger wedge biopsy specimens for 56%
and 67% of the specimens. Even when biopsies of adequate size are collected substantial variation in lesions between liver lobes has been reported. Therefore, it is essential to sample several liver lobes.

Sampling error is also important when it comes to quantification of hepatic copper (and other metals). For example, if copper is quantified from tissue collected from a regenerative nodule it is expected to be lower than that of adjacent liver. Therefore, it is important to both quantify copper and to histologically assess it.

Another limitation of histological assessment of liver tissue is the lack of inter-observer agreement. In one study 3 examiners agreed on the morphologic diagnosis assigned to needle and wedge biopsy specimens for 44 and 65% of the specimens, respectively. In another study, there was fair inter-observer agreement when 6 pathologists assigned fibrosis scores and poor agreement when they assigned necroinflammatory activity scores to sections of liver.

**Patient preparation**

Prior to performing liver biopsy, the patient’s general health should be established. This usually involves performing physical examination, CBC, platelet counts, serum chemistry panel, and urinalysis. The hemostatic system is also usually evaluated by measuring PT and PTT, where possible fibrinogen concentrations, and buccal mucosal bleeding time. However, the ability of PT and PTT to predict bleeding during liver biopsy is controversial. Supplementation of vitamin K (0.5 to 1.5 mg/kg q 12 hours for 3 doses) should be considered in patients with intra or extra-hepatic cholestasis and patients with overt bleeding may benefit from plasma or other blood products.

**Biopsy techniques**

Three liver biopsies techniques are used in small animal medicine; percutaneous needle biopsy, laparoscopic biopsy, and surgical biopsy. Each has its own set of advantages and disadvantages. Laparotomy allows collection of relatively large wedge biopsies, with direct visualization. This technique does not require specialized equipment or training, and excessive bleeding can be readily identified. However, laparotomy requires general anesthesia and is the most invasive biopsy technique. Percutaneous needle biopsy techniques have been described. These techniques may be possible under heavy sedation and are the least invasive method for collecting liver biopsies. Ultrasound guidance is often used, allowing biopsy of focal lesions. It is also possible to biopsy tissue that is deeper within the hepatic parenchyma than is possible with other techniques. However, the specimens that are collected are relatively small and may be inadequate for accurate assessment in some patients. Therefore, if needle biopsy is performed a 14 gauge rather than a smaller needle should be used, except for small dogs and cats where a 16 gauge needle should be used. Excessive hemorrhage after biopsy may not be identified immediately. Laparoscopy allows collection of biopsies using forceps with laparoscopic guidance. This technique requires general anesthesia, but is less invasive than laparotomy. The biopsies collected are larger than needle biopsies and excessive bleeding can be visualized. However, laparoscopy requires specialized equipment and training. Regardless of the technique used, a tiny proportion of the organ is sampled and, because liver disease can affect the hepatic parenchyma in a heterogeneous manner, sampling error is possible. To reduce the effect of sampling error, several biopsies from different areas of the liver should be collected and focal lesions should be specifically biopsied.

**Sample handling**

It is very important to collect an additional specimen of liver that is stored (placed in a plastic container then frozen) or submitted for copper quantification. The pathologist who evaluates the specimen should be provided with a detailed but concise patient history including results of laboratory testing and diagnostic imaging findings. There are several pathology services that offer evaluation by pathologists who have expertise in histological assessment of the liver, which can be very valuable, especially for unusual cases. I request that sections are stained for copper, iron, connective tissue, and lipofuscin in addition to routine staining with H&E. Liver should be submitted in an appropriate container for aerobic and anaerobic bacterial culture. As bile cultures are more frequently positive than liver culture, bile should be collected whenever possible.

**Interpretation of the histology report**

It is important to read the descriptive section of the report as well as the diagnosis and comments. The reason for this is that is often possible to obtain useful information about what the pathologist is seeing from this section. It is often helpful to talk directly to the pathologist especially for unusual or challenging cases.

It is beneficial for clinicians to have a basic understanding of the terminology used to describe hepatic histology. It is particular important for clinicians to understand the basic structure of the hepatic lobule an acinus because the localization of lesions can provide vital information. Some commonly used histological terms are defined below:

- Periportal- lesions centered on the portal triads of the hepatic lobule
- Centrilobular- lesions centered around the central vein of the hepatic lobule
- Zone 1 hepatocytes- hepatocytes closest to the hepatic artery and portal (inflow)
- Zone 2 hepatocytes- transitional zone between zones 1 and 3
• Zone 3 hepatocytes - hepatocytes nearest the hepatic venule (outflow)
• Bridging fibrosis - fibrosis connects that portal triads to each other (portal-portal bridging fibrosis) or to central veins (portal-central bridging fibrosis). This is an important step in the progression of chronic hepatitis
• Cirrhosis - Formation of regenerative nodules surrounded by fibrous septa accompanied by vascular disorders (some pathologists do not use this term in dogs or cats)

References/further reading
Etiopathogenesis

Copper is an essential trace element that amongst other roles acts as a cofactor for cytochrome c oxidase, an enzyme involved in ATP generation (oxidative phosphorylation). It can exist in a variety of oxidative states the most important of which are Cu²⁺ and Cu³⁺.

Hepatic copper accumulation is identified as the underlying cause in approximately one third of dogs with CH. The liver is the principal recipient of copper that is absorbed from the gastrointestinal tract. Copper in hepatocytes is immediately bound to proteins such as glutathione or metallothionein. When the capacity of the hepatocyte copper binding proteins is saturated, free copper ions are released. These are toxic and can lead to the formation of hydroxyl radicals which can subsequently cause oxidative damage to the liver. This results hepatocyte necrosis and a resultant infiltration of inflammatory cells which can be mononuclear or mixed. The inflammatory cells contribute to further tissue injury and with chronicity fibrosis occurs.

Copper can accumulate in the liver due to defects in copper metabolism, cholestasis, possibly increased dietary copper intake, or a combination of these factors. The following breeds of dogs are proven or suspected to be predisposed to primary copper accumulation: Bedlington terrier, West Highland white terrier, Scottish terrier, Skye terrier, Labrador retriever, Dalmatian, and Doberman pincher. It is also possible to see copper associated CH in other dog breeds and in mixed breed dogs.

In the Bedlington terrier, a mutation of the COMMD1 gene that encodes the cellular copper exporter ABCA12 has been identified. This condition is recessive and a genetic test has been developed. The incidence of copper associated CH in Bedlington terriers has been dramatically reduced by testing and selective breeding programs.

A genetic basis for copper associated CH has also been suspected in Labrador retrievers and a genome-wide association study showed an association between increased hepatic copper concentrations and a mutation of the Wilson’s disease gene (ATP7B), which encodes a copper transporter protein. A mutation of another copper transporting protein ATP7A, seemed to attenuate copper accumulation. These mutations only explained about 12% of the heritability of this disease so other genes and environmental factors are also likely to play a role. A genetic basis or contribution is also possible for the other predisposed breeds but further studies are needed to determine whether or not this is the case.

Excess dietary copper intake may also contribute to the development of hepatic copper accumulation. For most laboratories, the reference interval for the hepatic copper concentration of healthy dogs is 120 to 400 ppm dry weight. Nine healthy research dogs fed a standard commercial diet were shown to have a hepatic copper concentrations ranging from 199 to 997 ppm while feral dogs presumed to eat a diet of foraged food had concentrations ranging from 69 to 372 ppm. This might be because commercial dog food contains more copper than the foraged food.

Fanconi syndrome has been reported in dogs with copper associated CH. These dogs had glycosuria in the absence of hyperglycemia, low urine specific gravity, and proteinuria caused by proximal renal tubule injury.

Primary vs. secondary copper accumulation

It is important to try to differentiate between primary and secondary hepatic copper accumulation. Primary copper accumulation tends to occur in the centrilobular zones of the liver. In such cases the hepatic copper content is typically >1,000 ppm and many require chelation therapy. In advanced disease, lobular collapse can make it difficult or impossible to identify the different zones of the hepatic lobules. The breeds previously mentioned that are predisposed to copper associated CH, typically have this pattern of copper deposition. When copper accumulates secondary to cholestasis it tends be found in the perportal zones of the liver and the hepatic copper concentrations are usually <1,000 ppm. Chelation therapy is generally not needed and therapy is aimed at the underlying cause of cholestasis.

Diagnosis

The diagnosis of copper associated CH is made based upon a combination of histological evaluation of liver biopsy specimens and hepatic copper quantification. Copper may not be apparent on standard H&E stained histological sections and is detected most easily using rubeanic acid or rhodamine stains. I therefore request that sections of liver stained for copper are made in every case to make sure copper accumulation it is not overlooked. It is also helpful if the pathologist provides a semiquantitative score for copper.

Although it is possible to measure copper by deparaffinizing a histological block it is much better to collect an extra liver biopsy specimen and save it for copper quantification (freeze sample in a plain container). In all dogs where chronic hepatitis is even a possible differential I submit liver tissue for copper quantification. About 20-40 mg of liver is needed for this and copper is ideally quantified on a dry weight basis. When submitting liver tissue for copper quantification it is important not to sample regenerative nodules as they usually contain less copper than other areas of the parenchyma. Indeed, as copper can be heterogeneously distributed
Throughout the hepatic parenchyma and because copper quantification is typically performed on only one specimen results can occasionally be discordant with histological assessment. Recently, computer-assisted image analysis has been used digitally estimate the hepatic copper content. Although this technique does not provide a direct measurement of copper it allows multiple pieces of liver to be evaluated without loss of paraffin embedded blocks.

**When do I chelate?**

Deciding which dogs with CH to start chelation therapy for can be difficult and is the subject of some controversy. Hepatic copper concentrations between 120 and 400 ppm are considered normal and concentrations >1,500 ppm are considered diagnostic for hepatic copper retention. This cutoff of 1,500 ppm was established from early studies of Bedlington terriers and is probably too conservative for other breeds. I consider treating dogs with concentrations >750 ppm with copper chelating agents if there is centrilobular copper accumulation, especially in breeds thought to be predisposed to copper associated CH. If the hepatic copper content is >1,500 ppm I will chelate regardless of the distribution. My reasoning is that regardless of whether the copper accumulation is primary or secondary to cholestasis, at this high level it is likely to be detrimental to the liver. If I get a hepatic copper concentration >400 ppm but <750 ppm with a histology report that reads very much like copper-associated CH, I will consider chelation or at least a copper restricted diet.

**Chelation**

The idea of treatment with chelating agent is to remove copper from the liver by making it soluble so that it can be excreted in the urine. D-penicillamine is my first choice of chelating agent for dogs (10−15 mg/kg PO q12 hours before feeding). This drug commonly causes gastrointestinal side effects, such as vomiting, diarrhea, and anorexia (about a third of dogs will develop these signs!). If this does happen it may be necessary to give the drug with food and/or reduce the dose. Glomerulonephropathy and dermatological lesions are other reported side effects of this drug that are probably due to idiosyncratic immune reactions. If either of these complications develops, therapy should be promptly discontinued. In humans D-penicillamine can also cause depletion of vitamin B6 and although this has never been reported to occur in dogs, some clinicians supplement this vitamin during treatment. Trientine (10−15 mg/kg PO q12 hours on an empty stomach) may be used if penicillamine is not tolerated. However, currently it is prohibitively expensive. Dogs with copper-associated CH should also be started on a copper restricted diet, such as one of the commercial liver support diets (these are also supplemented with zinc).

As the optimal duration of therapy has not been determined and is likely to vary between patients it is also challenging to decide how long to chelate for. Prolonged therapy can result in copper deficiency. This can be manifested by microcytic hypochromic anemia, anorexia, vomiting, and weight loss. There is currently no non-invasive method to assess hepatic copper content but monitoring the dog’s clinical signs and liver enzyme activities can provide some indirect information regarding the efficacy of treatment. A decreasing ALT activity during treatment is encouraging. Usually it takes several months for this to return to normal. A study in Labradors suggested that 6−10 months of chelation should be adequate for most dogs. Ideally at this point a second hepatic biopsy procedure and copper quantification is performed.

At this time, if hepatic copper concentrations, when measured, have returned to normal or are <500 ppm, treatment with zinc acetate at a dose of 5−10 mg/kg PO q12 hours of can be initiated. Zinc decreases the absorption of copper from the gastrointestinal tract. Plasma zinc concentrations should be measured during treatment to ensure that toxic concentrations are not reached. Normal plasma zinc concentrations for dogs are 70–200 μg/dL. Concentrations around 200 μg/dL seem to effectively reduce copper absorption but concentrations exceeding 800–1,000 μg/dL may cause hemolysis. The dog should also be continued on a copper restricted diet.

Some dogs require repeated intermittent therapy with D-penicillamine and Bedlington terriers often need lifelong therapy. The decision to restart therapy is usually made based on an increasing ALT activity or ideally repeat biopsy.

**Supportive care**

The most important aspect of treating copper-associated CH is to treat the underlying cause. However, many veterinarians also start other supportive treatment. Corticosteroids and other anti-inflammatory medications are not advised. As copper hepatic accumulation causes oxidative injury there is a therapeutic rationale for treating with antioxidants such as S-adenosylmethionine or vitamin E. However, it is important to state that neither has been proven to be beneficial in these patients. Therapy for complications of CH such as HE and ascites are discussed in the next session on idiopathic CH.

**References/further reading**


Etiology
A variety of factors can lead to liver injury and inflammation in dogs, including drugs (e.g. phenobarbital), toxins (e.g. cycads, aflatoxins, Amanita phalloides, and blue-green algae), infectious agents (e.g. Leptospira spp, canine adenovirus-1, Heterobilharzia americana, and Bartonella sp.), hepatic copper accumulation, and possibly autoimmune disease. However, some of these factors, such as Amanita phalloides intoxication and most drugs (with the exception of phenobarbital) are more likely to cause acute liver injury than CH. Copper accumulation is the cause of CH in about a third of dogs but in many (about 60%) by the time CH is diagnosed it is not possible to identify an underlying cause. For these dogs, the term idiopathic CH is used.

In a study of dogs from the UK, the following breeds were shown to be at increased risk of developing CH: American Cocker Spaniel, Cairn terrier, Dalmatian, Doberman pincher, English cocker spaniel, English springer spaniel, Great Dane, Labrador retriever, and Samoyed. However, this study did not differentiate between copper-associated CH and idiopathic CH and some of these breeds are also thought to be at increased risk of developing copper-associated CH. The median age of dogs in this study was 7 years with a range of 7 months to 16 years. Some breeds such as Doberman pinchers, Labrador retrievers and West Highland white terriers can develop either copper associated and idiopathic CH.

There are several other theories to explain the development of CH in dogs. An autoimmune etiology has been postulated but not confirmed in some breeds such as Doberman pinchers, English springer spaniels, and Cocker spaniels. Chronic hepatitis seems to be more common in female Doberman pinchers than males and studies from Scandinavia showed a strong association with dog leukocyte antigen class II alleles and haplotypes. Such an association has also been demonstrated in English springer spaniels. However, this does not necessarily imply an autoimmune etiology. Additionally, studies have demonstrated autoantibodies against various proteins found in the liver in dogs with CH. However, this does not determine whether these antibodies are a cause of CH or the result of tissue injury. It does not appear that the recently canine hepacivirus is a cause of CH and although enteric bacteria are a known cause of cholangitis and cholangiohepatitis in dogs they have not been proven to cause CH.

Pathogenesis
Regardless of the underlying cause CH is a syndrome that is histologically characterized by hepatocellular necrosis or apoptosis, a mononuclear or mixed inflammatory infiltrate, regeneration, and fibrosis. Chronic inflammation of the liver can lead to activation of myofibroblasts, including hepatic stellate cells and portal fibroblasts. This results in hepatic fibrosis, which can diminish liver function as hepatocytes are replaced by collagen and can contribute to the development of portal hypertension. As the liver has a large functional reserve capacity, loss of liver function is detected relatively late in the course of CH. Portal hypertension and decreased hepatic synthesis of albumin contribute to the development of ascites, which is a poor prognostic indicator in these dogs. This is probably because ascites occurs late in the course of CH and usually signifies that irreversible changes to the portal circulation have occurred. Hepatic portal hypertension can also lead to the development of acquired portosystemic collateral blood vessels. These allow ammonia rich blood from the splanchnic circulation to bypass the liver, which in turn may lead to hepatic encephalopathy (HE).

Clinical findings
Although certain breeds of dog seem to be predisposed to CH, dogs of any breed can develop this syndrome. It is important to remember that early in the course of CH, dogs may not show any clinical signs. Later in the course of disease, clinical signs may become apparent. The most common are anorexia, lethargy, vomiting, polyuria, and weight loss, which are not specific for CH or hepatobiliary disease. More liver specific signs such as icterus, ascites, or hepatic encephalopathy tend to occur later still in the end-stage of CH.

Diagnosis
The diagnostic approach for a dog with increased serum liver enzyme activities is discussed in a previous lecture. It is important to remember that dogs with CH can have normal results upon serum bile acid testing and may not have abnormalities of the liver upon abdominal ultrasound examination. Chronic hepatitis is definitely diagnosed by histological evaluation of a liver biopsy specimen. Sometimes this also allows an etiological diagnosis to be made. Idiopathic CH is diagnosed by excluding underlying causes of chronic inflammation. The clients should be carefully questioned to rule out hepatotoxins. It is important to rule out copper accumulation by histological staining for copper and copper quantification. Liver tissue and bile should be submitted for bacterial culture (in my experience bacterial growth is uncommon). If histology reveals granulomatous hepatitis, further testing for geographically relevant infectious agents is indicated (e.g. Bartonella sp, Heterobilharzia americana, systemic fungal diseases).
Anti-inflammatory drugs

There is limited information supporting the use of the anti-inflammatory drugs in the treatment of idiopathic CH. A retrospective study of 151 dogs with chronic hepatitis of various causes found that those treated with corticosteroids survived longer than those that were not. However, these results should be interpreted with caution as the retrospective design of this study meant that it was susceptible to bias as the clinicians may have decided to start corticosteroids in dogs that were more likely to have a favorable response. The results of a more recent retrospective uncontrolled study of 36 dogs with idiopathic chronic hepatitis found that hepatic inflammation decreased and coagulation parameters returned to normal after 6 weeks prednisolone treatment and in some dogs the stage of hepatic fibrosis remained the same or improved. However, the majority of these dogs had a recurrence of clinical signs or residual disease at the end of treatment. Randomized placebo controlled clinical trials are needed before definitive recommendations can be made. Currently, when there is histological evidence suggesting a significant component of inflammation I consider using anti-inflammatory drugs such a prednisolone. Typically, a dose of 1–2 mg/kg/day PO is initially started and is then gradually tapered. It should be noted that high dosages of prednisolone/prednisone often cause vacuolar hepatopathy and other side effects, which can be detrimental to these dogs. Monitoring response to treatment is difficult because liver enzyme activities usually increase after starting treatment with prednisolone. Some authors recommend a second liver biopsy procedure 6 weeks after initiation of treatment. Other anti-inflammatory drugs that are sometimes used in place of prednisolone include azathioprine (2 mg/kg PO q48 hours) and cyclosporine (5–10 mg/kg/day PO). Neither has been proven to be beneficial and azathioprine can be hepatotoxic.

The use of “hepatoprotectants” in canine chronic hepatitis

Nutraceuticals and other hepatoprotectants are often used in the management of dogs with CH and other liver disease. Unfortunately, there are very few clinical trials in dogs that have assessed their efficacy. This can make it difficult for clinicians to know when to use them. Justified. By understanding how these agents work it is easier to make rational treatment decisions. It is important to state that these agents are not a substitute for treating the underlying cause of hepatic disease.

Because of its central role in metabolism the liver is very susceptible to oxidative damage. Oxidative damage is important in the pathogenesis of a range of hepatic diseases, including CH. S-adenosylmethionine (SAMe) is a precursor of the important hepatic antioxidant glutathione. The main rationale for using this agent is that it helps prevent oxidative damage by preventing depletion of hepatic glutathione. It has also been purported that SAMe may have anti-inflammatory properties, modulate apoptosis, and be anticarcinogenic. However, these effects have not been documented in dogs. At the recommended dose of 20 mg/kg PO q12 hours SAMe has rarely been reported to have side effects in dogs other than occasional vomiting after dosing. S-adenosylmethionine is indicated in a range of liver diseases where oxidative stress is believed to be a contributing factor, including CH. However, it is important to note that there is currently little evidence supporting the efficacy of SAMe in dogs. Oral administration of SAMe has been shown to reduce oxidative stress but not histological changes consistent with vacuolar hepatopathy in dogs receiving prednisone.

Silymarin is extracted from the milk thistle plant. Silibinin is the most biologically active component of silymarin. Silymarin is believed to have antioxidant effects by scavenging free radicals and reducing lipid peroxidation. It is also believed to have anti-inflammatory and antifibrotic properties. Additionally, silymarin may be a choleretic agent. At commonly used doses silymarin does not appear to cause side effects although its bioavailability is low. Potential indications for silymarin include acute liver injury and CH. Again, there is very limited evidence to support its efficacy in the veterinary literature. In one study of Beagles administered Amanita phalloides toxin, 11 dogs treated with intravenous silibinin survived whereas four out of twelve control dogs died. In a study of dogs being treated with the chemotherapy agent lomustine, dogs treat with a product containing silymarin, SAMe, and phosphatidylcholine (Denamarin) were shown to have smaller increases in serum ALT and ALP activities than those that were not, suggesting a hepatoprotective effect.

Ursodeoxycholic acid (UDCA) was found to be the active compound in the traditional Chinese remedy of dried black bear bile. Ursodeoxycholic acid is a hydrophilic bile acid that is believed to have multiple beneficial properties including: choleretic effects, displacement of other more toxic bile acids from the circulating pool, an antiapototic effect, and immunomodulatory effects. When used at a dose of 15 mg/kg/day PO this drug has few side effects other than causing occasional diarrhea. Because of its choleretic effect and the displacement of more toxic hydrophobic bile acids it makes sense to use this drug in dogs with intra or extrahepatic cholestasis. Due to its claimed immunomodulatory and antiapoptotic there is a theoretical reason to use UDCA in dogs with CH. However, evidence supporting the use of UDCA in dogs is limited to a few case reports.

Vitamin E is actually a family of eight lipid soluble vitamins. The main role of vitamin E is as an antioxidant, protecting phospholipids from oxidative injury by scavenging free radicals. Generally, vitamin E is well tolerated and side effects are not observed. Because of these properties I consider using this supplement in dogs with liver diseases that can lead to oxidative damage, such as, copper associated CH. However, it is important there is no clinical evidence supporting the efficacy of vitamin E in dogs with hepatobiliary disease.
Supportive care
Supportive care is also important for these dogs. Dogs with hepatic disease, especially those with portal hypertension are at increased risk of gastroduodenal ulceration. Consequently, treatment with omeprazole (1 mg/kg PO 12-24 hours) is indicated if GI bleeding is suspected. The development of ascites in dogs with CH is a poor prognostic indicator. Furosemide can lead to hypokalemia and metabolic acidosis both of which can precipitate HE in humans. The aldosterone receptor antagonist spironolactone (2 to 4 mg/kg PO q12 hours) is therefore a better initial choice. If this is ineffective furosemide can be added starting at a low dose (1 mg/kg PO q12 hours). Severe protein restriction is no longer recommended for dogs with hepatic encephalopathy (HE) as this can lead to protein malnutrition. It is important to also note that dogs with liver disease that do not have signs of HE likely do not benefit from dietary protein restriction. Non-meat protein based diets are sometimes recommended for dogs with HE. Once the signs of HE are controlled with a commercial hepatic support diet, it is recommended to add non-meat protein to the patient’s diet to help prevent protein malnutrition. Lactulose can be given orally to patients with chronic HE. It is usually started at a dose of 1 to 3 mL per PO per 10 kg of body weight every 6 to 8 hours. The dose is then adjusted until the patient passes three to four soft stools per day. Neomycin is a poorly absorbed aminoglycoside antibiotic that is sometimes used to treat HE in dogs. The gastrointestinal absorption of neomycin is very low, but can be increased in patients with decreased gastrointestinal motility or bowel wall damage. Substantial systemic absorption can cause ototoxicity and nephrotoxicity. Metronidazole is another antimicrobial that is sometimes used for the treatment of HE in dogs. Metronidazole is usually given at a dose of 7.5 mg/kg PO q8–12 hours in dogs with HE.

Anti-fibrotic medications
Colchicine impedes microtubule polymerization during mitosis and therefore is believed to inhibit fibrosis and inflammation. Colchicine is frequently associated with gastrointestinal side effects in dogs and aside from a few case reports from which it is very difficult to prove efficacy there is little evidence to support its use. I therefore do not recommend using this drug.

References/further reading
Increased serum liver enzyme activities, especially alkaline phosphatase (ALP) activities, are commonly identified in dogs. These increases represent a diagnostic challenge to clinicians for a number of reasons. Firstly, sometimes increased serum liver enzyme activities occur due to primary hepatobiliary disease and other times they can occur secondary to extrahepatic disease. This may be because tissues other than the liver also produce these enzymes. Additionally, the liver plays a major role in the metabolism and the excretion of drugs, as well as exogenous and endogenous toxins. The liver is perfused by the portal circulation, whereby a large proportion of its blood supply comes from the splanchnic circulation via the portal vein. Consequently, the liver is susceptible to injury caused by a variety of toxins, diseases in other parts of the body, as well as ischemia. Thirdly, sometimes increased liver enzyme activities can occur due to benign processes, such as hepatic nodular hyperplasia or can be due to conditions that are progressive and require early intervention to have an optimal outcome, such as chronic hepatitis. This can make it difficult for clinicians to know how aggressive to be when working up these dogs. Performing extensive diagnostic evaluation, including invasive tests such as liver biopsy, is costly causing some clients to be reluctant or unable to proceed. Sometimes in depth evaluation of dogs with increased serum liver enzyme activities is not required. For example, when there are mild increases in ALP activity. Despite these obstacles and uncertainties, using a logical approach clinicians can prioritize which dogs require liver biopsy, which require investigation for extrahepatic disease, and which can be managed less aggressively.

Hepatic enzymology

Alanine aminotransferase (ALT) is found primarily in the cytosol of hepatocytes. ALT is released when the cell membrane permeability of the hepatocytes increases or if there is hepatocyte necrosis. Although this enzyme is found in a variety of tissues, increased serum ALT activities are considered to be relatively liver specific. The exception to this is that rarely ALT activity can increase in patients with severe muscle injury. Alanine aminotransferase is considered to be a sensitive marker of liver injury. Aspartate aminotransferase (AST) is found in the mitochondria and cytosol of hepatocytes. The cytosolic fraction is released when the cell membrane permeability of the hepatocytes increases or if there is hepatocyte necrosis, whereas the mitochondrial fraction is only released when there is necrosis. Increases in AST generally parallel those in ALT but muscle disease can cause an increase in serum AST activity. Because of this, AST is considered less liver specific than ALT.

The hepatic, bone, and steroid induced ALP isoenzymes can all contribute to serum ALP activity in dogs. In the liver, this enzyme is bound to the membranes of the hepatocytes that form the bile canaliculi and the sinusoidal membranes. When there is cholestasis, this membrane bound ALP is released into the circulation and the synthesis of this enzyme is induced. Alkaline phosphatase is therefore considered to be a sensitive marker of cholestasis in dogs. Because of the two non-hepatic isoenzymes mentioned above, ALP is not liver specific. Serum ALP activities can be increased when there is increased osteoblast activity e.g. growing dogs or dogs with osteolytic disease e.g. osteosarcoma. Synthesis of the steroid induced ALP isoenzyme is induced by both exogenous and endogenous glucocorticoids. It is also important to note that increased serum ALP activities have been reported in a family of apparently healthy Siberian Huskies and also in some apparently healthy Scottish Terriers. Vacuolar hepatopathy due to excess adrenal production of androgens is suspected to be the cause in the latter. Gamma-glutamyltransferase (GGT) is an enzyme that is found bound to the hepatocytes that comprise the bile canaliculi and bile ducts. Increases in serum GGT activity generally parallel those in ALP as both are considered to be relatively sensitive markers of cholestasis. In general increases in GGT are considered to be less sensitive but more specific for the presence of hepatobiliary disease than those of ALP.

Initial patient evaluation

There are many causes of increased liver enzyme activities, so it very important for clinicians to go from a list of all the possible causes to a list of all the causes that are “probable for that patient on that day”. Information collected during history taking and physical examination is often very helpful when doing this. The patient’s signalment can help refine the differential list. For example, very young dogs are more likely to suffer from congenital conditions, e.g., congenital portosystemic shunts (CPSS) or certain infectious diseases, e.g. infectious hepatitis than neoplasia or inflammatory conditions, e.g. chronic hepatitis. Some breeds, i.e. Bedlington terriers, Skye terriers, West Highland white terriers, Dalmatians, and Labradors are predisposed to copper associated chronic hepatitis. Doberman pinchers and Cocker spaniels are predisposed to idiopathic chronic hepatitis. The breeds of dog predisposed to CPSS include the Maltese terrier, Yorkshire terrier, Havanese terrier, pug, and miniature schnauzer. Increased serum liver enzyme activities are more concerning in these breeds. When taking a history, it is very important to ask specifically about exposure to hepatotoxins such as cycads, blue green algae, amanita mushrooms, aflatoxins, heavy metals, xylitol, or chlorinated compounds. A variety of drugs can also be hepatotoxic, these include: ketoconazole, various antimicrobial agents, azathioprine,
carprofen, lomustine, acetaminophen, ketoconazole, mitotane, and phenobarbital. It is important to specifically ask about any herbal remedies that the dog is receiving as many of these have been reported to be hepatotoxic, including: herbal teas, pennyroyal oil, and comfrey. Ascertaining the dog’s vaccination history is also worthwhile as leptospirosis and canine adenovirus-1 can cause hepatic injury. Early in the course of liver disease dogs may not have any clinical signs. The earliest clinical signs seen in dogs with liver disease are often non-specific and include: icterus, ascites, poor body condition, stunted growth, hepatomegaly, or signs of hepatic encephalopathy. It is important to emphasize that dogs with hepatobiliary disease do not always display clinical signs or have abnormal findings on physical examination. Physical examination may also reveal findings that are suggestive of extrahepatic disease. For example, bilateral symmetrical alopecia is consistent with hypothyroidism or hyperadrenocorticism.

**Routine laboratory testing**
Other changes on a serum biochemistry panel can provide important clues as to the cause of increased serum liver enzyme activities. When serum concentrations of albumin, cholesterol, glucose, and urea are below the lower limit of the reference interval or towards the lower limit of the reference interval, this is consistent with decreased hepatic function. It is important to remember that these changes are not specific for hepatobiliary disease. For example, the serum bilirubin concentration may also be increased when there is hemolysis. Additionally, due to the large hepatic functional reserve capacity, liver disease must be severe before these changes are seen. Patterns of serum liver enzymes activities can be suggestive of certain pathologies. For example, during cholestasis the serum activity of ALP is dramatically increased and is higher relative to that of ALT. There may also be evidence of extrahepatic diseases. Analysis of a complete blood count can suggest inflammatory conditions, rule out hemolysis, and if microcytosis is present this is consistent with portosystemic shunting (or iron deficiency). Urine specific gravity can be decreased in patients with hepatic insufficiency or portosystemic shunts. Excessive bilirubinuria in dogs implies hemolytic or hepatobiliary disease. Urate urolithiasis seems to be more common in patients with portosystemic shunts than those with other types of hepatic dysfunction. However, it should be noted that urate crystalluria is not specific for hepatobiliary disease.

When do you recommend further diagnostic testing?
Once basic diagnostic evaluation of the dog has taken place the decision whether or not to pursue further diagnostic testing should be made. Every case is different so it is difficult to make universal recommendations. However, I can offer the following general guidance:

- If there are clinical findings or other laboratory test results that are suggestive of primary hepatobiliary disease, further diagnostic testing should be pursued.
- If there are clinical findings or laboratory tests results that suggest the extrahepatic diseases that can lead to increased liver enzyme activities, further diagnostic evaluation to identify their cause is needed.
- If serum liver enzymes activities (ALP or ALT) are severely (three times greater the upper limit of the reference interval) or persistently increased (greater than twice the upper limit of the reference for more than 3 to 4 weeks), further diagnostic evaluation is needed.
- As ALT is more liver specific than ALP, increases in serum ALT activity are more concerning than increases in ALP.
- If none of these conditions apply then it is reasonable to wait and recheck the serum liver enzymes at a later date.

Further diagnostic testing
The utility of plain abdominal radiographs for diagnosing hepatobiliary disease is limited and they rarely lead to a definitive diagnosis. However, they can be used to assess the hepatic size and to rule out certain extrahepatic diseases. Abdominal ultrasound is more useful than radiology for evaluating the hepatic parenchyma and the biliary tract. It is also sometimes possible to diagnose portosystemic shunts using this modality. However, unless a disease is characterized by architectural changes of the hepatobiliary system, a definitive diagnosis cannot be made with ultrasound examination. It is also important to remember that dogs with severe liver disease may not have any changes on abdominal ultrasound examination. Despite this limitation, when primary hepatic disease is suspected, abdominal ultrasound is usually performed prior to liver biopsy. Measurement of plasma ammonia and paired preprandial and postprandial bile acids are sensitive tests for portosystemic shunting and one of these tests should be performed when this is suspected. However, because of the hepatic functional reserve capacity, these tests are not as sensitive for detecting hepatic insufficiency in the absence of shunting and normal results do not rule out severe liver disease. Therefore, performing these tests does not always alter the decision whether or not to perform hepatic biopsy.
In selected cases, hepatic cytology is useful as it can lead to a definitive diagnosis of certain diseases and can be highly suggestive for the presence of others. Indications for performing hepatic cytology are a suspicion that a round cell tumor is present, when infectious agents, for example *Histoplasma capsulatum* are suspected, and when hepatic masses are observed on abdominal ultrasound.

To make a definitive diagnosis of primary hepatic disease liver biopsy is often required. Prior to doing this the patient’s risk of hemorrhage should be assessed by measuring prothrombin and activated partial thromboplastin time, ideally measuring serum fibrinogen concentration, performing a platelet count, and performing a buccal mucosal bleeding time. Liver biopsy techniques in dogs include: percutaneous needle biopsy, laparoscopic biopsy, and surgical biopsy. Each technique has its own set of advantages and disadvantages. No matter which technique is chosen, it is important to collect multiple biopsies as well as to save a specimen for copper quantification and another for bacterial culture. Although, supportive treatment, such as the hepatoprotectant agents are important in the management of patients with hepatic disease they are not a replacement for the specific treatments, for example copper chelating agents, that may be indicated once a histological diagnosis has been made. When in doubt, if there is a suspicion of primary hepatic disease it is better to biopsy rather than to delay biopsy until the dog is in end-stage liver failure, at which point treatment is unlikely to be effective.

**When should I biopsy the liver?**

Again, it is hard to make universal rules as every case is different but I can offer the following general guidelines:

- Hepatic biopsy is indicated when a hepatic mass has been diagnosed and a diagnosis has not been made based on cytology
- Hepatic biopsy is indicated when the serum ALT activity has been greater than twice the upper limit of the reference interval for more than 3 to 4 weeks and extrahepatic disease is unlikely to be the cause.
- Hepatic biopsy should be considered when there are multiple acquired portosystemic shunts. Acquired shunts suggest that there is hepatic parenchymal disease e.g. chronic hepatitis, which requires biopsy to be definitively diagnosed. However, acquired portosystemic shunts occur late in the course of disease and are irreversible. Consequently, the prognosis for these dogs is poorer so some clients may not wish to proceed. They can also occur due to pre hepatic portal hypertension e.g. due to portal vein thrombosis.

**References/further reading**


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Protein Losing Enteropathy: Improving Outcomes
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Protein losing enteropathy (PLE) is defined as the loss of protein from the intestines due to intestinal disease. Often, this results in hypoalbuminemia, and may be accompanied by hypoglobulinemia. Strictly, any condition leading to abnormal protein loss from the intestines is a PLE. However, if the patient’s serum albumin is not decreased, this protein loss often goes un-noticed. Any intestinal disease, if severe enough, can result in PLE. The underlying mechanisms for this are disruption of the intestinal mucosal barrier and/or lymphatic dysfunction. Protein losing enteropathy is therefore classified as being a syndrome rather than a disease. Protein losing enteropathy is much more commonly diagnosed in dogs than cats. Diseases that have been found to cause PLE in dogs include: intestinal lymphangiectasia (IL), inflammatory bowel disease (IBD), intestinal neoplasia (especially lymphoma), fungal infections, intussusception, and gastrointestinal (GI) parasites. Protein losing enteropathy can lead to some important consequences in our patients, which can even be life threatening.

Clinical presentation
Due to the diverse range of underlying conditions that cause PLE, any age, breed, or sex of dog may develop PLE. However, some breeds have been demonstrated to be particularly at risk. These include Yorkshire Terriers, Rottweilers, German Shepherd Dogs, Soft Coated Wheaten Terriers, Norwegian Lundehunds, and Basenjis. The most common clinical signs of PLE are diarrhea, vomiting, and weight loss. It is important to remember that some dogs with PLE may not vomit or have diarrhea, these dogs usually but not always present with weight loss. Other clinical signs may be due to the loss of serum proteins, especially albumin, including ascites, edema and pleural effusion. Occasionally, dogs suffering from PLE develop respiratory distress due pulmonary thromboembolism. During PLE, blood proteins including antithrombin is lost into the intestines potentially making them hypercoagulable. However, a study using thromboelastography showed that all 15 dogs with PLE enrolled were hypercoagulable but this was not entirely explained by decreases in antithrombin. Ionized hypocalcemia can occur secondary to hypovitaminosis D. This hypocalcemia may be manifested as muscle tremors or even seizures.

Physical examination may reveal weight loss and poor body condition due to mal-nutrition. Thoracic auscultation may reveal decreased lung sounds due to pleural fluid. Swelling of the legs and/or ventral parts of the body due to edema may be present. Abdominal palpation may reveal a fluid wave, abdominal masses, enlarged organs, enlarged lymph nodes, or thickened bowel loops.

Diagnostic approach
As discussed before, many animals with PLE have non-specific signs of gastrointestinal disease. However, some dogs will present with weight loss alone or decreased serum albumin may even be noticed as an incidental finding. A serum biochemistry panel will confirm hypoalbuminemia. Globulin concentration can be decreased, normal, or increased, so it is the albumin concentration that is more important to consider.

Once hypoalbuminemia has been documented it is tempting to diagnose PLE in any dog with gastrointestinal signs. However, there are other possible causes of hypoalbuminemia and these can be associated with clinical signs of diarrhea, vomiting, and weight loss. The possible causes of a serum albumin concentration below 2.0 g/dL are hepatic insufficiency, severe dermatological disease, protein losing nephropathy, and protein losing enteropathy. To diagnose PLE, the three other possible causes should be ruled out (or increased fecal protein loss should be demonstrated). The possibility of liver disease is investigated by interpretation of a serum chemistry panel and possibly measuring a pre- and postprandial bile acid concentrations. The presence of severe dermatological disease is easily determined during physical examination. Protein losing nephropathy is investigated by measuring the amount of protein in the urine. This can initially be done by urinalysis and if urinary protein loss cannot be ruled out this way, a urine protein to creatinine ratio should be performed. It is also very important to consider hypoadrenocorticism as a cause of low albumin. When PLE cannot be diagnosed by ruling other causes of severe hypoalbuminemia it may be helpful to document fecal protein loss using the fecal alpha-1 protease inhibitor test.

Once PLE has been confirmed, efforts should be made to find the underlying cause. Abdominal ultrasound is frequently the most useful imaging modality for assessing the gastrointestinal system of small animals. Changes on abdominal ultrasound, such as hyperechoic mucosal striations, can often be observed with PLE patients, but these seldom give us a definitive diagnosis. Microscopic evaluation of intestinal biopsy specimens is often the most informative test. Intestinal biopsies can be collected via endoscopy, via an open abdominal surgery, or via laparoscopy. Each technique has its advantages and disadvantages. At Texas A&M, endoscopy is often performed initially. It is very important to perform an upper and lower gastrointestinal tract endoscopy so that the stomach, duodenum, ileum, and colon can all be biopsied. In most cases when combined with the clinical presentation, laboratory tests and imaging
findings, histopathologic analysis of endoscopic intestinal biopsies can lead to at least a provisional diagnosis, and guide initial treatment. However, sometimes a final diagnosis is only made after assessing the patient’s response to treatment.

**Common causes**

Intestinal lymphangiectasia is characterized by dilation of the lymph vessels of the intestines. It is believed to be the most common cause of PLE in dogs. Intestinal lymphangiectasia occurs in both a congenital form (primary IL) and an acquired form (secondary IL). Primary IL is a developmental abnormality that leads to an insufficiency or malformation of the lymphatics. This condition may affect other parts of the body as well as the intestines. Secondary IL is due to obstruction of lymph flow. This develops either due to physical blockage of the lymphatics, or high venous pressure. The lacteals can be physically blocked by inflammation or cancer of the intestines. When the lymph flow is obstructed, high protein lymph leaks out into the intestinal wall. This leakage of protein contributes to the intestinal disease as it can cause inflammation and secondary granuloma formation, which can further impede lymph flow.

Because of the inflammation that develops secondary to IL, it can be very difficult/impossible to differentiate primary IL and IL secondary to chronic enteropathy. Yorkshire Terriers, Maltese Terriers, Rottweilers, and Norwegian Lundehunds are predisposed to IL (probably primary IL). During endoscopy, distended lacteals may be visible as multiple white spots on the intestinal mucosa.

Inflammatory bowel disease is defined as a group of idiopathic, chronic gastrointestinal disorders characterized by mucosal inflammation. It is diagnosed on the basis of observing inflammatory cell infiltrates in the bowel wall on intestinal biopsies and ruling out other causes for the inflammation. These other causes of chronic enteropathy in dogs include dietary intolerance or allergy, antibiotic responsive enteropathy (intestinal disease). In idiopathic IBD, inflammatory cells accumulate in the bowel wall for an unknown reason. The inflammation is often classified according to which cell types are most abundant. Lymphoplasmacytic inflammation is the most commonly identified variety of IBD in dogs and cats. IBD must be severe in order to result in intestinal protein loss.

Certain breeds of dog are predisposed to getting distinct forms of PLE. Soft Coated Wheaten Terriers often develop a hereditary PLE characterized by IL and inflammation of the intestinal wall with a concurrent protein losing nephropathy. Basenji’s can develop what is described as an immunoproliferative enteropathy. Lundehund syndrome is characterized by gastritis, IL, and IBD.

Lymphoma can affect many parts of the body, including the intestines. Roughly 75% of dogs with intestinal lymphoma will have hypoalbuminemia. Lymphoma can be diagnosed based on intestinal biopsies, although endoscopic biopsies may not be deep enough to distinguish this cancer from intestinal inflammation. Laboratory tests such as immunophenotyping and PCR for antigen receptor rearrangements can help differentiate lymphoma from IBD. Adenocarcinoma and other tumors may also cause PLE due to blood loss and mucosal ulceration. These tumors may be confined to one section of the intestine. Because of this it may not be possible to reach them with an endoscope. Consequently, surgical biopsy or fine needle aspiration of any mass that is present may be required. Abdominal ultrasound can help in selecting which sampling technique is best to use.

*Histoplasma capsulatum* is a fungal organism that can cause gastrointestinal disease in dogs. It is unusual for Histoplasma to affect the gastrointestinal tract of cats, but it is reasonably common in dogs, although usually other organs are also affected. Diagnosis can often be made on cytological evaluation of rectal scrapings, lymph node aspirates, hepatic aspirates, or splenic aspirates, depending upon which organs are affected. Occasionally the diagnosis is made on intestinal biopsy, but this is not usually necessary. A urine/serum antigen test (Histoplasma EIA, Mira Vista Laboratories) is also available.

**Treatment**

As there are many underlying causes of PLE in the patients there is no single treatment protocol for this syndrome, every patient has different needs. The aims of therapy are to treat the underlying cause and to support the patient. Treatment of IL and IBD as well as supportive care are discussed below.

The mainstay of treatment for IL is feeding an ultra-low-fat, highly digestible diet. Reducing the dietary fat content decreases the amount of fat that needs to be transported in the intestinal lacteals, thereby to some extent reducing the problem of the lacteal obstruction. The commercial diets with the lowest fat contents are Royal Canin Gastrointestinal Low-Fat, Purina EN Low Fat, and Hill I/D Low-Fat. An alternative is to use an ultra-low-fat home cooked diet, for example, boiled turkey breast and rice. This diet can be used initially and if the patient responds after 2–3 weeks, it must be supplemented in order to make it nutritionally balanced. Commercial weight-loss diets are not suitable for dogs with PLE because although they have a fairly low fat content they are calorie restricted and usually these dogs are already severely malnourished. Some very sick PLE patients may benefit from being fed an elemental diet such as Vivonex T.E.N. Elemental diets very easy to digest, as they contain amino acids rather than proteins. If using an elemental diet, it is important to select one with a very low fat content. They are expensive and so are typically used to provide short-term nutrition. In many cases of IL there is a component of inflammation. The presence of inflammation is determined by evaluation of intestinal biopsy specimens. If this is the case, the patient may benefit from anti-inflammatory medications. Common choices are prednisone, chlorambucil, and cyclosporine (see below). The justification for these medication in cases of IL is to prevent the development of lymphatic granulomas, which can impede lymphatic drainage.
As previously discussed, IBD must be severe in order to cause PLE. Consequently, aggressive treatment is needed. Anti-inflammatory drugs are the main treatment for IBD. Common choices are prednisone (2 mg/kg PO per day), chlorambucil (2-4 mg/m² PO q24 hours) cyclosporine (5 mg/kg PO q24 hours), and azathioprine (2 mg/kg PO q48 hours). Prednisone and prednisolone are the most commonly used anti-inflammatory drugs for treating IBD, and are cheap and frequently effective. However, these drugs often have unwanted side effects when used at higher doses. Azathioprine is often used in conjunction with prednisone to provide additional immunosuppression, or so that a lower dose of prednisone can be used. This drug can have serious side effects (it can affect the liver and the bone marrow) and should NOT be used in cats. Furthermore, it takes up to 2-3 weeks of treatment before it is fully effective. This is an important consideration for a sick patient. Cyclosporine is a newer immunosuppressive agent that is generally well tolerated and acts quickly. It is expensive, especially for larger dogs, and there is currently not much data to support its use in IBD, although the preliminary data is encouraging. A recent retrospective study suggested that a combination of prednisolone and chlorambucil was more common than a combination of prednisolone and azathioprine for treating dogs with chronic enteropathy including those with PLE. Dietary management is also important in dogs with IBD. Often dogs with IBD and PLE have not undergone a diet trial prior to biopsy, so a diet trial with a novel antigen or hydrolyzed antigen diet is frequently worthwhile. However, if microscopic evaluation of the intestinal biopsies suggests or confirms a diagnosis of IL, I advise feeding an ultra-low-fat diet.

Supportive care
Because of their hypoalbuminemia, gastrointestinal disease, and their potential to develop spontaneous blood clots PLE patients are fragile. Supportive care is therefore extremely important.

Providing adequate nutrition is vital. Patients, who are vomiting or have severe gastrointestinal disease, may not willingly eat. Often, treatment of nausea with anti-emetics such as maropitant (Cerenia; 1 mg/kg SQ/IV q24 hours) is the first step to take. If this is not effective, tube feeding may be needed. In small to medium sized dogs an esophageal feeding tube is my preference, whereas I typically place gastrostomy tubes endoscopically in larger dogs, and in giant breeds or deep chested breeds surgical placement of a gastrostomy tube may be needed. Forced syringe feeding is not a practical solution. The dog’s resting energy requirement (RER) should be estimated. It is advisable to initially feed half or one third of the estimated RER during the first 24 hours before gradually increasing the amount. With emaciated dogs eventually it is important to feed more calories than the estimated RER so that they gain weight. One method is to estimate a target weight for the dog and feed the RER calculated based on this.

When necessary administration of intravenous fluids that provide oncotic support can help reduce fluid accumulation and therefore stabilize the dog’s circulation. This can be important prior to or during anesthesia. However, often these dogs have had chronic hypoalbuminemia and so they have compensated for their low blood oncotic pressure and do not require colloidal support. Synthetic colloids (e.g. Hetastarch or Vetastarch) are currently my preference for providing colloidal support to dogs with PLE. Human albumin solutions are available and are sometimes used in severely hypoalbuminemic dogs but can be associated with severe side effects due to immune reactions. Canine albumin has intermittently been available in the US and in theory should avoid these problems. Plasma transfusion is not a practical way to provide increase a dog’s serum albumin in most cases as relatively large amounts need to be given, which for all but very small dogs is cost prohibitive.

Measures to reduce the risk of thrombosis include giving ultra-low dose aspirin (0.5 mg/kg PO q24 hrs) or preferably clopidogrel (Plavix; 1–3 mg/kg PO q24 hours), meticulous care of IV catheters, not placing unnecessary IV catheters, and frequently encouraging the patients to move.

Dogs with PLE often have low serum vitamin B12 (cobalamin) concentrations as they cannot absorb this vitamin from their small intestine. Supplementing cobalamin by subcutaneous injection or orally can correct this, and may improve the patient’s gastrointestinal signs, as well as their appetite. It is important to use cyanocobalamin and not vitamin B mixtures, as these do not contain sufficient cobalamin. Supplementation with vitamin K (1 mg/kg SQ q24 hours for 3 to 5 days) is indicated in patients with prolonged clotting times. In dogs with symptomatic or severe ionized hypocalcemia, supplementation with calcium and calcitriol is indicated.

References/further reading
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 tools and tips to help guide owners through the decision process and to provide them with guidelines to do what is best for the pet and the family.

**Proceedings**

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:

- 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.”)
- What do you hope the life expectancy of your pet will be? What do you think it will be?
- What is the ideal situation you wish for your pet’s end of life experience? (at home, pass away in her sleep, etc.)
- 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 goodbye.

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 perception 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 difficulty 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**
   - Desire to be with the family has not changed.
   - Interacts normally with family or other pets (i.e., no increased aggression or other changes).

2. **Natural Functions**
   - Appetite has stayed the same.
   - Drinking has stayed the same.
   - Normal urination habits.
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
   • 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.
   • 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.
   • 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.

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
   • 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.
   • 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.
   • 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 wellbeing. (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.

**Resources**

Almost every time the door opens, I am greeted with a crying client. As a veterinarian that limits my practice to in-home hospice and euthanasia, it is something I encounter daily with my families. In those moments, I give a warm smile, a gentle handshake and in many cases, a big hug.

Although the majority of our appointments are for euthanasia, we also offer veterinary hospice care to our concerned pet parents. However, veterinary hospice is still very misunderstood, even within our profession. I am often asked, "What is veterinary hospice?" at clinics and conferences. It is important to first understand what hospice is NOT: It is not prolonging suffering nor is it euthanasia or natural dying. Hospice simply is a medically supervised service dedicated to providing comfort and quality of life for the pet (and the owners) until euthanasia is elected or natural death occurs.

A great deal of families wish to keep their pet alive for as long as possible while also maintaining a good quality of life but simply don’t know how and feel helpless. As a veterinary hospice practitioner, I am able and willing to help extend life as long as pain and anxiety are controlled, but this is always preceded by a lengthy discussion on the progression of the disease process present and a clear “stop point” which we agree is the ending of a good quality of life. Communication, preparation, and more communication is the hallmark of a successful hospice case.

At Lap of Love, many of our clients are referred to us from veterinary specialists – mostly oncologists, cardiologists and internists. While much of veterinary hospice is ideally done in the home, where the pet is most comfortable, many discussions and treatments should be started at the clinic with their primary veterinarian who has enjoyed a long term relationship with the pet parent. With that being said, I am sad to report that approximately 40% of our clients have not taken their pet into the clinic within the last 2 years. I strongly believe that we can help a great number of pets if we are able to educate owners on the aging process and also the progression of the specific diseases their pets are facing.

Veterinary medicine focuses a lot on ‘Senior Wellness’ but I think we are asleep at the wheel when it comes to geriatric pet care. Caring for a geriatric pet is a completely different experience than caring for the 8 yr old ‘senior dog’. C are giving for the elder pet can be emotionally and physically exhausting and it is vital to support the owners through this time. Our philosophy of the way we care for these pets, in most cases, needs to shift from curing but to simply caring. This quote from Jurassic Park is a favorite of mine, “Just because we can, doesn’t mean we should”. Too often owners tell me that they are scared to continue with their regular veterinarian because they are simply forced into x-rays or bloodwork. Often hospice is simply a tool to help the owners grasp the idea that their pet’s life will be ending soon. It may be a month or even just a day – at this point radiographs are pointless – but pain medication, education, communication and preparation is priceless.

“Doc when is it time?” – Boy do I wish I had a dollar for every time I was asked that! However, this is a good question and deserves a lot of attention and time discussing it with your clients. It is not as simple as saying ‘When he stops eating, it’s time’ or ‘He will give you ‘the look’ and you will know’. Although at times those are good indications of it being time, often, it is not. The 13 year old Labrador with osteoarthritis may still be eating and looking excited when his owner comes home, yet can barely get up, falls down the stairs and is sitting in his own feces half the day. Assessing quality of life is an important part of the hospice appointment. There are many tools available that can assist owners with evaluating quality of life. We have a Quality of Life scale available on our website www.lapoflove.com under the ‘Quality of Life’ tab. We also developed a more sophisticated tool where owners can create a profile for their pet, select a specific ailment and are asked questions based on the ailment. They can also make journal entries, chart weight or body condition score and attaching pictures every day. This is free to the public at www.pethospicejournal.com.

Although providing hospice in the home can garner a lot of information that may be missed at the clinic, the discussion and treatment can start with you. Setting up a hospice program in your clinic is actually very simple.

It is most important to help the family understand the disease process their pet is facing. Although we cannot predict exactly what will happened in the future, we can use our medical training and experience to give each family facing an end-of-life experience with their pet a possible and probable progression of their pet’s disease process. As doctors, this is the most important piece of information we have to give them and the most valuable tool families have in the decision making process. We must, to the best of our ability, explain the most likely “natural” method of death if left unattended. This educated approach to the physicality of death is essential to veterinary hospice care; by providing the family with knowledge and expectations, we give them the ability to make an informed decision based on their personal wishes for their pet with the gentle guidance of their veterinarian.

By using the word ‘Hospice’ with your clients, it redirects their thoughts from curing their pet to caring for their pet and preparing themselves for death and grieving. Then, you can tailor your medical management appropriately to make sure the pet is kept comfortable and safe.
Some hospice services your clinic can offer:

- **Consultations** - This is our most common and requested hospice service. You may be surprised at how appreciative the client is for 30 minutes with a veterinarian discussing what to expect and how to manage their pet’s disease and progression.
  - When a client calls us for a euthanasia but says, “It’s not time yet – but I want to be in your system” – to me, this is a call for help. Their pet is bad but not quite ready for euthanasia. This is the perfect opportunity to offer a consultation.
- **Pain and anxiety management** – The amount of pets that I see that are not on any pain medication is staggering. Providing adequate pain medication is vital and evaluating its effectiveness is just as important. I also equip the owner with “emergency intervention” they can do themselves. For example, the client with a dog with Osteosarcoma should leave your clinic with a dose of injectable pain medication and the knowledge of how to administer it in case of a pathologic fracture. That way, the pet can have some relief while the next steps are organized.
  - Many dogs are up all night panting and pacing, with many owners awake as well. Providing medications that help them sleep through the night helps the anxiety level and is appreciated by everyone in the house.
- **In home technician visits and care** – seeing the pets in their own environment is key as they act differently in their surroundings. More importantly, modifications can be made that may have been overlooked and treatments can be done in the home without a distressing trip to your clinic.

The most common ailments we see at Lap of Love are; Osteoarthritis, renal disease, heart failure and a variety of cancer. But one that is often overlooked or put into the osteoarthritis category is sarcopenia. Age related muscle atrophy is a huge problem in our geriatric pets. Owners need to be educated on this ailment and taught how to manage it in the home, how to properly exercise their pets and provide supportive care. In most of those patients, pain medications and NS AIDS are of no benefit, leaving the owner frustrated. Having a hospice package and plan for the above ailments is a great way to start offering hospice at your clinic.

**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:

Disease sheets with detailed information about the illness affecting the pet, including end-stage clinical signs
- 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.
- “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.
- Adjunctive services you support and trust (preferably mobile) in the area, such as acupuncture, massage, mobile grooming, in-home pet sitting.
- Local pet loss groups or grief counselors, contact local human hospice for a good referral source.
- 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.
- Emergency clinics in the local area, if your clinic does not offer 24-hour emergency care.
- Specific euthanasia information, including:
  - When and how to schedule euthanasia at your clinic, and if your clinic offers euthanasia in the home.

While offering veterinary hospice may not provide the largest avenue of revenue – the immeasurable benefits are great. The satisfaction your clients will have with the full circle of veterinary care at your clinic will be priceless. This will lead to positive word of mouth, referrals, and repeat business with other pets from that client when necessary and most importantly – it is what is best for the pet.

Veterinary hospice is here to stay. When families have a better end of life experience with their pets, they heal more quickly from the debilitating emotional loss. They are better able to cope with their decisions, feel confident in their ability to care for their pets, and more quickly open their homes and hearts to pet ownership again.
Veterinarians have a variety of tools to keep puppies and kittens healthy as they grow, and we are well prepared to help our aging patients as they reach their senior years. The care and management of a geriatric pet, however, is very different for both the patient and the owners alike. As pets reach advanced ages and enter into this last life stage, owners are faced with a myriad of physical and emotional concerns (for both the pet and themselves). There is so much more that can be done within the veterinary profession to properly recognize this geriatric stage, keep the patient comfortable, and help owners deal with their delicate, aging family members.

Veterinary hospice is rapidly gaining traction and typically focuses on the terminal or chronically ill pet. Before and during this last stage, there is much we can do to help pets live a comfortable life as a geriatric. Our abilities to recognize and manage pain, anxiety, hygiene, and other symptoms that may limit quality of life has advanced in recent years and our profession is seeking ways to identify these unique client and patient needs, communicate effectively, set realistic expectations, and help guide pet parents with the care and management of their aging geriatric companion animal.

The goal of proper and effective geriatric pet care is to enhance the quality of life for the pet and the owners, empower them to properly care for their pet during this delicate life phase, and maintain the strength of human-animal bond. The goal of this presentation is to offer the methodology, tools, and soft skills that are essential to properly caring for this age group.

It will empower veterinarians to embrace the geriatric pet, know how to handle the symptoms that plague them as well as assist owners with the care and management.

Aging is the inevitable decline in the body's resiliency both mental and physical. Over time, cell production decreases, leaving fewer cells which are less capable of repairing wear and tear on the body. The immune system is compromised and therefore more susceptible to infections, less proficient at seeking out and destroying mutant cells, many older pets succumb to conditions they could have resisted in their youth.

The aging process is incredibly complicated and it can be difficult to distinguish between changes that are the result of ‘age’ and those that come with common medical conditions.

Below are the top 6 symptoms that we will cover in the presentation – plus more!

**Eyes**

Lenticular/Nuclear Sclerosis: All geriatric dogs (starting at about 6-7 years old) develop a hardening of the lens. However, it does not become noticeable until about 10. The lens is added onto throughout life, gaining layers of protein. As the new layers of protein are added, inner layers are compacted together and become harder. The hardening of the lens fibers makes it difficult for the lens to change shape – needed for focusing. Near vision is therefore reduced – just like in middle-ages people who need reading glasses. Pets become hesitant going down stairs and more difficult when catching small treats or toys.

**Ears**

Presbycusis, also known as age related hearing loss. Mid to high frequencies are affected first followed by progressive loss at all frequencies. Onset is typically in the last third of a breed’s typical lifespan and will eventually progress to complete deafness.

Four types of presbycusis are described in humans and in dogs but the most common seen is the sensory presbycusis which is characterized by loss of hair cells and degeneration of the organ of Corti.

Although the loss is progressive, owners usually report an acute onset because of the ability of the animal to compensate for hearing loss until nearly complete deafness occurs. Age related hearing loss most often occurs in both ears, affecting them equally.

**Skin**

Dull Skin and Coat: An older animal’s skin and hair may look dull and lusterless due to the decreased production of natural oils by the sebaceous glands. This can also cause the skin to appear dry and flaky. Continued brushing will help stimulate the skin to produce the oily secretion and an excessively dry coat may benefit from implementing a fatty-acid supplement. The skin also loses elasticity as pet’s age and is more susceptible to infections. The worst side effect of a skin infection is that the pet smells and therefore is shunned out of the bedroom or living area.

**Muscles – Can’t get up or down easily**

Sarcopenia is defined as the progressive loss of lean body mass in aging animals in the absence of disease. As muscle tissue mass decreases so does muscle strength which is why older people are less steady or have difficulty catching their balance. Our pets may exhibit similar signs such as changes in their movements reflected in difficulty getting up or reluctance to jump up.
Lungs:
The elastic fibers in a dog’s lungs allow them to expand and contract with each breath. As a dog grows older, some of these fibers are replaced with fibrous scar tissue diminishing the ability to breathe as efficiently as possible. Pet owners should recognize that an older animal can’t exercise in extreme temperatures as well as they did when they were younger. Jogs or walks with your pet may need to become slower or shorter as they progress through their older years.

Trouble at night – Panting and pacing
Some older dogs may become restless at night and stay awake pacing throughout the house or panting. There are many reasons an older dog may have difficulty sleeping at night including both medical and anxiety or behavioral related causes. Dogs do get cognitive dysfunction which is similar to dementia in people. Cognitive dysfunction is also referred to as sundowner syndrome and is categorized as a slow, degenerative and progressive disorder in our aging pets.

Sundowning is a syndrome in Alzheimer’s patients of recurring confusion and increased agitation in the late afternoon or early evening. The hallmarks of this syndrome in dogs are progressive confusion, reversal of day-night wake-sleep patterns and poor adaptability to new situations. The exact reason for this change in our geriatric pets is unknown.

This is just the tip of the iceberg when it comes to the ailments and common symptoms our pets face when they age. Dogs do get cognitive dysfunction which is similar to dementia in people. Cognitive dysfunction is also referred to as sundowner syndrome and is categorized as a slow, degenerative and progressive disorder in our aging pets.

Providing in home evaluations can also provide you with insight to how the pet manages in their home and also how the owner is managing the pet. Both are very important. 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.

Assessing quality of life
When dealing with an aging pet – the topic of ‘when is time’ is bound to come up. Giving your clients ways to evaluate quality of life will be key in helping them deal with that questions.

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! Many Quality of Life tools are discussed at Lapoflove.com

Geriatric wellness plan
Similar to wellness plans for younger patients, clinics can create Geriatric Wellness plans to encourage owners to consistently bring their pets in for exams. Bundling services and avoiding services that may not be necessary at this life stage is the foundation. An example of bundling services is offering 4 visits per year for a discounted rate (i.e., if your typical office visit cost is $45– offer 4 visits for a discounted rate of $135 instead). At the geriatric stage, diseases and symptoms progress fast; thus, warranting the need for multiple visits a year. Bundled service discounts are a great way to maximize compliance for pets in need by incentivizing for a visit every quarter.

Offering unique services is another component of a Geriatric Wellness Plan. For instance, geriatric pet sitting, monthly “sanitary shaves”, Fear-Free nail trims, laser therapy, physical therapy, and geriatric boarding/day care are a few ideas that can be incorporated into the plan.

At this stage in life, many pets will also need specialized accessories or products to help manage their daily activities. This can be done by offering a retail space within the clinic, or if that is unfeasible, simply by providing information sheets to clients on useful items and where to order them.

In summary
As a profession we have been well educated and equipped for marketing and caring for the senior pet. For those fragile, advanced aged geriatric pets there is an opportunity to provide better care as they enter their golden years, and support the families as they struggle alongside their pet. Marketing specifically to this group helps to highlight the symptoms the pet will encounter while also
focusing on the challenges the caregiver may face. Overall, this confirms to the caregiver that you empathize with their plight, gains their trust, and encourages them to reach out for assistance with their pet when needed.
What is Aging?
While most of the specific alterations and mechanisms are not well defined, on a cellular level, cumulative DNA damage leading to genomic instability, oxidative damage and telomere shortening play roles. Specific organ systems are often evaluated extensively, both on an individual basis (tracking renal function), and on a population basis (looking at the prevalence and risk of chronic renal disease within an age group).

Since the process of aging ultimately results in the demise of the patient, much attention has been given to the trends of aging and relative life expectancy for particular breeds, and in relation to dog size, or healthy weight range for a breed or individual. Since excess weight, taken to the extreme in obese pets, is known to have direct impact on the health of an individual, this should also be taken into account when evaluating a pet.

Relative Age
For an owner, and the veterinary staff, the first step is to determine the relative age of the pet, as compared to human years. The old adage of one dog year equaling 7 human years can provide an estimate, but dogs and cats age at different rates, primarily based on their size. . The designation of senior should be applied to a pet that has reached 75% of its expected life span. The AAFP has provided a Life Stages Chart for Cats, with the senior status reached from 11 to 14 years of age. Smaller pets have longer expected life spans and giant breeds are often considered senior at 5-6 years of age. Several resources have tables that allow you to determine your pets’ relative age – but each animal is an individual, so these are starting guidelines to assess their senior status. Keep in mind, if the pet was adopted as an adult, there is a chance that the age on record might be an estimate, sometimes on the low side, when trying to determine their relative age.

Starting the care early – wellness from maturity
And just because a pet is growing older doesn’t mean those twilight years can’t be healthy ones. Think of the concept of “healthspan” when dealing with mature pets, not just “lifespan”: recognizing changes that are within normal limits for the pet and dealing with those changes that are not healthy. Within that “healthspan”, it is important to determine when the level of care for the different life stages needs to be adjusted. According to the AAHA Senior Care Guidelines for Dogs and Cats¹, the comparable start to the senior years in humans is around 56 – 60 years of age, or approximately the last 25% of the expected lifespan of the pet. The clinical screening of healthy pets prior to this stage can set baselines for comparison when the pet’s systems being to experience changes. From a chart estimating the relative age of a dog or a cat in human years, this senior stage is reached at around 10 years of age for pets up to 20 pounds, 9 years of age for those between 21 and 50 pounds, 8 years for those between 51 and 90 pounds, and 7 years for dogs over 90 pounds.

Body Condition and Senior Nutrition
Many practices now enjoy assessment of the Body Condition Score (BCS) to determine if a patient is in its correct weight range. Certainly, excess body weight can be accompanied by higher risks of osteoarthritis, diabetes and other metabolic diseases. On the other hand, with aging pets, weight loss can be a significant issue as well. While the basal metabolism rate of dogs continues to slowly

decline with age, at around 11 years of age in cats, that decline changes to an increase in BMR, and an increased need for high quality nutrients. This is another reason, besides chronic disease, to also monitor a patient’s lean body mass or LBM. By tracking both BCS and LBM, nutritional adjustments can be made for that particular patient’s needs. In some older cats, increased protein of a high quality might be recommended in the absence of renal failure. In fact, even in renal cases, dietary protein levels do not cause or alter the course of kidney disease. Low dietary protein only decreases the symptoms associated with kidney failure, not slow it or cure it. Geriatric pets require the same or more protein than younger animals, especially active seniors. Old pets may be special, but not with regards to protein.

Other conditions may require nutritional adjustments as well, from sodium restriction in cardiac disease to special gastrointestinal needs or an increase in antioxidants. Unfortunately, there are no specific guidelines from AAFCO for senior nutritional needs (as there are for growth vs maintenance), so the wide variation in nutrients can be quite confusing.

Mobility/exercise/enrichment

If a patient has a high BCS, managing the diet might be accompanied by increased exercise, but it is important to do a full evaluation on the musculoskeletal health of the individual. If they are already overweight, osteoarthritis may limit their mobility, and this has been identified in many dogs and even cats. Starting an exercise program gradually, with supplements or medications to ameliorate any discomfort can help that patient reach an ideal weight much more quickly. Exercise and environment enrichment (that can also adjust food intake appropriately) is also thought to help with the patient’s overall health and attitude.

Cognitive Dysfunction

As many pets age, there can be a noticeable change in activity and attitude and in some pets, certain signs may not be attributable to a medical cause, and “he’s just getting old” isn’t enough of an explanation. Just as in humans, dogs and cats can experience diminished cognitive function, beyond what can be expected in the normal aging process. The DISHA acronym found in many publications can help alert you and the client to potential issues:

- **D** – Disorientation – may appear lost. confused
- **I** – Interaction – may not respond to familiar faces, or be clingy
- **S** – Sleep-wake cycles – sleep more during day, less during night
- **H** – Housetraining – eliminates inappropriately
- **A** – Activity levels – aimlessly wanders or decreased focus

Cognitive Dysfunction Syndrome (CDS) can be devastating to a family, when their life-long friend is disoriented, gets ‘lost’, forgets housetraining, doesn’t interact with others, or has a disrupted sleep cycle that can impact everyone. Most of the previous studies and data have been focused on canine patients, and while many of the signs are similar, excessive vocalization, irritability and decreased self-hygiene seem to be more prevalent signs in felines. This new emphasis on feline patients is supported by a recent study that investigated cognitive decline in cats. Behavior modification, environment enrichment, various supplements and even prescriptions can help decrease some of these signs, but the best results are found with earlier intervention.

Dental Health

Dental care and senior care often go hand-in-hand, as dental disease can affect appetite, comfort levels and associations with organ disease. Using the need for dental care is a reason to complete diagnostic recommendations, and when a thorough senior health care check has been done, that can be a good time to catch up on dental care.

Senior Care Programs

There are many recommendation for starting and implementing senior care programs, but one important aspect that is often overlooked is the ability to measure how well your program is performing, or if you have met the goals you set at the beginning. Here is an example of one approach:

- **Bi-annual exam**
- **Annual CBC, U/A, chemistries**
  - Mini-chem at 6-7 years of age (or at ‘mature’ status)
• Full chem at 8-10 years of age (or at ‘senior’ status)
• Add in Thyroid profile for cats at 8, dogs at 10

• Add in disease related
  o Chest radiographs, ECG

• Behavior and Nutrition counseling
  o If you are just starting a senior wellness program, trying to do too much at once can be challenging and some client might be resistant.

• Sudden introduction with additional costs may be challenging to implement
  o Phased-in program with Mature Wellness first
    • “Silver Elite” status
  o Step-up to Senior Wellness with more comprehensive evaluation and testing
    • “Gold Elite”
  o Promotion to Geriatric Care – likely with disease related therapy
    • “Platinum Elite”

Diary – daily functions
• Body condition, skin condition, masses - photos
• Appetite – increased, decreased, change in food type preference, difficultyprehending, chewing, swallowing?
• Water consumption/elimination – increase, decrease, change in habits?
• Activity – amount, frequency, type
  o Encourage interaction
• Alertness – Cognition or sensory (sight, hearing?)
• Sleep patterns – increased, decreased, change patterns, vocalization
  o Resting parameters – respiratory rate, cardiac rate
• Senses – sight, hearing
• Comfort level – watch gradual changes, response to medication
• Regular pictures for comparisons

Client Education and Involvement
The key for having a successful senior care program – and healthier senior patients – is getting the clients involved with every stage of patient care; and that takes education. Discussing wellness and preventive care throughout the pet’s life stages will help prepare the owner for the increased needs as their pets’ age. Using the tools such as the Daily Diary will keep the owner aware of gradual, subtle changes, and can help prepare them when those changes add up to conditions that need management. Working as a team, with the owners’ input and clinical diagnostics and therapies, will help provide optimal care for your senior patients.
A veterinary clinic’s curb appeal does not stop at the clinic door. It extends all the way into exam room and, most importantly, to the entire team! Every person our clients interact with will receive a “snap judgment” from their first impression. How long does this take? For years the general rule has been 7 seconds, but a few years ago a group of psychologists found that it takes about one tenth of a second to form an impression of a stranger, simply from their face (1). They also found that longer exposure to the stranger does not significantly alter the impression; it only boosts confidence in the initial judgment.

What does this mean to a veterinary team? It means that we have a very, very small amount of time to make a positive impression on our clients. This positive impression is not only essential from a business standpoint (you want them to come back!), but also from a medical one. Our clients need to trust us; they need to believe that we care about their pet the same way they do. Without the belief and trust that the client and the doctor have the same desired outcome, trust and rapport will not be established and the client may not accept the treatment plan that the veterinary professional team has offered. Which is, after all, the reason we are in business; to care for, treat, heal, and support animals.

Of course, the importance of body language or non-verbal communication is not a new concept. The “7-38-55 Rule” was first developed in 1971 by UCLA psychology professor Albert Mehrabian (2): 55% of what we convey when we speak comes from our body language, 38% from our tone of voice, and a mere 7% from the words we choose. This study has been widely misinterpreted by stating “97% of what we convey is non-verbal” instead of garnering a greater understanding of vocal (tone, cadence, etc.) and body language cues, which are inappropriately combined to come up with the “97%”.

Mehrabian more clearly states the following on his website:

\[
\text{Total Liking} = 7\% \text{ Verbal Liking} + 38\% \text{ Vocal Liking} + 55\% \text{ Facial Liking}.
\]

Please note that this and other equations regarding relative importance of verbal and nonverbal messages were derived from experiments dealing with communications of feelings and attitudes (i.e., like–dislike). Unless a communicator is talking about their feelings or attitudes, these equations are not applicable.

Although this landmark study is riddled with criticism and misinterpretation, it remains an important and highly cited illustration of the value of nonverbal communication. Many other studies have arisen since, each with a new methodology, and with the continued conclusion that non-verbal cues are 3 to 4 times more influential than verbal cues.

Before we dive into the real content of this talk, it’s important to understand that reading body language is not the same as mind reading. This is the difference between “observation” and “evaluation.” Reading someone’s non-verbal cues is about observation; we want to find natural tendencies in someone’s physical behavior (called their “baseline”), then look for deviations from their baseline, and finally ask open ended questions to find the root cause of the change.

For example, you may walk into a room and find two people seated, both have their arms crossed while one has both feet flat on the floor and the other has her legs crossed at the knee. You might assume that the closed off body postures means they are both upset, and perhaps the female is even more upset because her legs are crossed as well. This may be true, but probably not. Jumping to conclusions so quickly and, for example, immediately putting your guard up or responding with your own closed off body language may start you off on a bad foot (no pun intended) by eliciting defensive behavior from these clients. In this example, crossed arms might be this gentleman’s natural baseline, and the female may simply be cold!

Remember, reading body language is about observing someone’s baseline, finding where there are deviations from that baseline, and using powerful questions to find the underlying cause of the deviation.

The basics

The basics of body language are pretty simple. Across species lines, animals (human and non-human), use adaptations to increase or decrease their physical presence. A bear stands on his back legs to appear taller, cobras expand their hood when they are threatened, and the mantis lifts her front limbs while displaying a conspicuous eyespot in order to scare or distract a predator.

Humans present similar non-verbal “tells” by puffing their chest and standing taller when an attractive woman walks by or throwing both hands up in the air after accomplishing a huge milestone (even humans who have been blind since birth exhibit these behaviors).

The opposite is true as well; a dog cowers in the back of a cage or tucks his tail, an embarrassed child covers her face. We tend to minimize our physical presence when we want to disappear!

Each unique area of our body displays our emotions differently. The face is the most important when it comes to first impressions, and the feet most important when you want to know whether a negotiation is being tipped in your favor.
Personal curb appeal

When you want to make the most positive impression possible on a client, there are 4 main areas to consider: Initial facial expressions, the introduction to the client, non-verbals while speaking, and physical appearance. Each of these areas have been proven to influence the impression someone has on another person.

1. Facial expressions

Judgments based on facial appearance or expression play a very powerful role in how we get treated (2). In fact, in a court of law, it’s been shown that “mature faces” receive harsher judicial outcomes than those with a “baby-faced,” and having an face that is thought to be “competent” (as opposed to trustworthy or likable) may be highly predictive of whether a person gets elected to public office (3). Also, like it or not, attractive people are more favorably viewed in general, leading to overall better outcomes in life in addition to being thought of as more trustworthy (4).

What is a good way to use your facial expressions to improve your curb appeal? Smile. Yes, simply smile. Of course we have all been subjected to the “fake smile” versus “genuine smile”! This distinction has been researched for quite some time; so much so that a genuine smile is now described with the name “Duchenne smile” after the French physician Guillaume Duchenne, who studied the physiology of facial expressions in the nineteenth century (5). The Journal of Personality and Social Psychology described the difference from the anatomical perspective (5):

A. The Duchenne smile involves both voluntary and involuntary contraction from two muscles: the zygomatic major (raising the corners of the mouth) and the orbicularis oculi (raising the cheeks and producing crow’s feet around the eyes).

B. A fake smile involves the contraction of just the zygomatic major since we cannot voluntarily contract the orbicularis oculi muscle.

Interestingly, the fake smile is controlled by the motor cortex while more complicated emotion-related expressions, like the Duchenne smile, are controlled by the limbic system.

Yes, our clients can tell the difference! A genuine, warm, sincere expression of happiness that conveys a welcoming greeting is related to emotion, while the cheesy grin is simply a forced muscle action. So make sure your greeter (whomever that might be) smiles because they are happy to be there, not because they are forced to!

2. The non-verbals of introduction

Upon being greeted with the warm, genuine smile, the customary introduction ensues. Even if this is a long-standing client, there is still a formal greeting ritual we all engage in. The first 7 seconds may be too long for a first impression, but it’s the perfect amount of time for a good introduction.

In our current Western society, the handshake occurs first and, as long as it’s a good one, is the universally accepted sign of professionalism, politeness, and confidence. A good handshake is an art! Whether you’re the veterinarian or the support staff, make sure you initiate the handshake before the client does to show a confident welcome. Remember, they are coming into your “home” (the clinic) and you want them to feel that you genuinely appreciate their presence. Make hand contact palm-to-palm, web-to-web (the “web” is the flap of skin between your thumb and pointer finger) while keeping the angle of your hand either perpendicular to the ground, or palm facing slightly up. Palm down in a handshake indicates power. Don’t squeeze too tightly, nor too loosely, and maintain consistent tension as you say your greeting. Also, make sure to shake everyone’s hand in the pet’s family, not just the primary owner, even the children. (What a way to inspire a new generation of veterinarians!)

While shaking the client’s hand, maintain good eye contact and introduce yourself, even if you believe they know your name (but not with close friends of course!). They may have forgotten your name since their last visit, and setting your client up for success by knowing your name helps build their confidence. (More on verbal techniques, including how to say the client’s name, in another lecture.)

Since the introduction is about 7 seconds long, make sure it’s meaningful. Step in front of the receptionist’s desk to shake their hand, use a two-handed handshake (both of your hands around their one hand), lean gently forward to show appreciation for them coming in, and/or bend down to pet their dog (cats may not appreciate this though!).

3. Non-verbals to gain rapport

After you’ve made an amazing first impression, followed by a confident introduction, it’s times to complete the circle so that the client builds the trust, rapport, satisfaction, and connection with the entire veterinary team. These skills all enforce the concepts of active listening, engaged interaction, and supporting the client’s concerns.

These concepts are broken into 3 anatomical areas, top, middle, and lower body regions.

A. Body language in the top ⅓

Eye contact is incredibly important! But how much is too much? At what point does it start to become creepy? One study in the Royal Society Open Science (6) found that, when asked to stare at a video of an actor staring back at them, participants had a “preferred gaze duration” of 3.3 seconds (give or take 0.7 seconds). They also found that the rate of pupillary dilation (an automatic reflex) was a good indicator of how long they wanted to gaze; the longer their preferred gaze, the faster their pupils expanded. (Don’t
get too attached to this difference, however. The change was so subtle that it was only seen with eye tracking software, which would be awkward to follow in real life!

Make your eye contact consistent by looking only inside the imaginary triangle between the two points about 1 inch above each eye and the tip of the nose; going further down to the mouth or chin is more indicative of a social or amorous relationship.

Aside from the eyes, do not bite, tense, purse, or conceal your lips. Janine Driver, re-known body language expert, says “when we don’t like what we see or hear, our lips disappear.” This is evidenced by turning both lips into our mouth, similar to spreading Chap Stick once it’s been applied.

When nodding your head, a gentle, 1 second nod implies active listening, whereas faster head nods may tell your listener “hurry up, I don’t have time for this.” Make your nods slow and small with a closed mouth (which indicates you are listening).

Hands and arms are the second component of this category. Many of us will find ourselves wringing our hands or picking at our fingernails at any given moment. This may increase when we are nervous and evolve from a normal, baseline behavior into what is considered “pacifying” behavior. This is a normal reaction to nervousness or discomfort. (Again, we don’t know WHY someone may be nervous or uncomfortable, but we can simply make the observation then follow up with a powerful question.)

On the deeper meaning of hand positions, Adam Kendon, Gesture: Visible Action as Utterance, says:

Gestures of the Open Hand Prone or “palm down” family are used in contexts where something is being denied, negated, interrupted or stopped, whether explicitly or by implication. Open hand Supine (or “palm up”) family gestures, on the other hand, are used in contexts where the speaker is offering, giving or showing something or requesting the reception of something.

When auditing the body language of your own hands and arms, use open, offering palms when escorting a client to an exam room, offering to take their coat, or asking if there’s “anything else you need?”

B- Body language in the middle ⅓

Where someone’s torso is facing may be one of the most important indications of where they want (or don’t want!) to be. The “Belly Button Rule” dates back to the 1930s. Since then, numerous scientists and body language experts have reinforced the theory. Most notably, Dr. Albert Mehrabian, professor Emeritus of Psychology and UCLA has said “the belly button rule is the most important indicator of reading a person’s intention.”

During an introduction, face your belly button towards them. This indicates genuine interest and engagement. While you’re writing in the patient’s chart as they actively describe their pet’s history (or anything else they feel is important to you), you may turn your shoulders slightly away in record notes as long as your belly button remains mostly pointed towards the person that is talking.

C- Body language in the lower ⅓

Many experts feel that it’s easier to read someone’s feelings by looking at there feet than any other part of their body. In fact, this concept especially applies to interactions when one party is attempting to “conVINce” another, which can be the case when a veterinarian (or anyone else on the team) is presenting an estimate to a client. Studies have actually shown that crossed legs can have a devastating effect on a negotiation.

In How to Read a Person Like a Book, authors Gerard I. Nierenberg and Henry H. Calero reported that the number of times settlements were reached increased greatly when both negotiators had uncrossed their legs. In fact, they found that out of two thousand videotaped negotiation transactions, not one resulted in a settlement when even one of the negotiators had his or her legs crossed.

So what is “good” body language in this lower part of the body? Since building a rapport with clients is our main goal, you want to be perceived as interested and actively listening. Uncross your legs, both feet flat on the ground, sit on the edge (but not too far) of the seat, and lean slightly forward. (This is a great stance to take when writing the clinical history while listening to the client.) For the best effect possible, don’t jiggle your feet, wrap your toes around the edge of the chair, or cross your legs or your ankles. And if you see the client doing any of these unwanted behaviors, it might be a good time to audit your own body language or other communication styles (tone or phrasing, more in another lecture on these) in order to compensate for the potential misalignment. Of course the client might simply be cold!

4. Physical appearance

You may not be into fashion or up on the latest trends, but that’s not what having a “nice” appearance is all about. Being well dressed has everything to do with appearing put-together, not being a mannequin for the latest crop top or fringe boots. Just as our clients will judge the veterinarian’s surgical skills by the neat row of sutures, the will also judge our entire team’s knowledge, professionalism, compassion, and overall trustworthiness by the way we choose to dress ourselves that morning.

We’ve all heard the saying “dress for the job you want” or “clothes make the man.” Well, those sayings have real research, and lots of it, to back them up! In 1955 a group of researchers had a man cross a city street against traffic (8). When this man was dressed in a suit, 3.5 times as many people followed him as when he was wearing a “work shirt and trousers.” Regardless of background demographics, a business suit is universally seen as a form of authority.

Taking this one step further, not only is being well-dressed seen as a reason for others to follow you, but also a reason for others to do what you ask them to do. In another study (9), an experimenter would stop someone on the street, point to a person about 50 feet away (this person far away was an accomplice), and say “You see that guy over there by the parking meter? He’s over parked but
doesn’t have any change. Give him a dime!” The experimenter would then leave. When dressed in a uniform (anything relating to authority), most people complied with the instruction to give the other person money. When dressed in regular clothes, however, compliance was less than 50%.

But how does this translate into the exam room? What about the white coat hypertension we hear so much about? It appears this may be an overreaction, making it the exception, not the norm. In a written survey in 2005, patients were asked to review pictures of physicians in four different dress styles, then answer questions relating to their preference as well as their willingness to discuss sensitive issues (10):

On all questions regarding physician dress style preferences, respondents significantly favored the professional attire with white coat (76.3%, P <.0001), followed by surgical scrubs (10.2%), business dress (8.8%), and casual dress (4.7%). Their trust and confidence was significantly associated with their preference for professional dress (P <.0001). Respondents also reported that they were significantly more willing to share their social, sexual, and psychological problems with the physician who is professionally dressed (P <.0001). The importance of physician’s appearance was ranked similarly between male and female respondents (P = .54); however, female physicians’ dress appeared to be significantly more important to respondents than male physicians’ dress (P <.001).

The conclusion from this study was obvious: “Respondents overwhelmingly favor physicians in professional attire with a white coat. Wearing professional dress (ie, a white coat with more formal attire) while providing patient care by physicians may favorably influence trust and confidence-building in the medical encounter.”

More recently in 2015 (11), a comprehensive international review of studies on physician attire was published in the British Medical Journal Open, adding to the previous study’s findings. The authors confirmed the idea that, yes, most people prefer their doctor to be dressed formally, and also stressed that how you feel about your doctor’s attire can depend greatly on your age and/or culture. For example, in general, Europeans and Asians of any age, and Americans over age 50, trusted a formally dressed doctor more, while Americans in Generation X and Y tended to accept less-dressy physicians more willingly. Doctors in other roles, such as surgery or emergency however, appear more insulated from this effect and patients much more willing to see their doctor in scrubs.

Even if you are not the doctor, pick your attire carefully. What you chose to put on your body says more to the client about your professionalism and trustworthiness than you may think!

Conclusion
Curb appeal does not stop at the clinic’s entrance. And fortunately for veterinary professionals, those clinic doors are human sized, not small doggy-doors (until pets earn a monetary income, this will be the case)! We have to interact with, connect with, and ultimately, win the trust of our clients if our professional knowledge is to be put to good use. Without that rapport with our clients, something every person of the veterinary team is responsible for upholding, our treatment plans may not be accepted and/or compliance may not be achieved. Only through immediate, consistent, and appropriate maintenance of this bond will the patients receive the best possible medical care, and our clients happy to see us again!

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Veterinary medicine is one of the greatest professions. Sadly, it is can also be one of the most emotionally draining. We are automatically at ‘risk’ for compassion fatigue due to the nature of our job (high stress, long hours, long weeks, and most importantly, not enough funding), in addition to our personalities (perfectionists, high achievers, and highly compassionate). Knowing why you’re struggling is important, so it’s not helpful to label every negative experience in the veterinary profession as compassion fatigue. When I took an honest look at how I was feeling, I wasn’t running out of compassion. My fatigue stemmed from making ethical decisions within the boundaries of clients’ (often) illogical values or unreasonable budgets.

My friend and mentor Alice Villalobos, DVM, once asked me if I experienced compassion fatigue in my veterinary hospice work. “No,” I quickly responded. “I don’t believe I’ll ever run out of compassion.”

I went on to explain my thoughts on compassion fatigue and how I think it’s an overused term in the veterinary industry. I told her that I felt more drained in emergency work than in hospice care, and that’s when Dr. Villalobos said something that will stick with me forever: “I believe what we really struggle with in our profession is not so much compassion fatigue as ethical fatigue.”

Her assessment landed perfectly in my mind. In emergency medicine, I was rarely given full financial rein to do the very best for my patients. More often I was required to make decisions based on someone else’s monetary budget, decisions that could arguably border on ethical dilemmas:

- Do I run blood work or spend this money on immediate treatment?
- Do I use minimal sedation to suture up a pet so I have funds left over for pain medication and antibiotics?
- Do I tell this family their pet has a 5 percent chance of living a few more months, knowing they will drain their savings account or go into debt just to get a little more time?
- Do I euthanize this sick kitten that was just dropped off because we don’t have any foster homes?

These are ethically based decisions, not compassion-based. We all know that if we do not choose to negotiate this path with some clients, they will make a drastic decision (like euthanasia elsewhere or perhaps an unfair negative online review), which may lead to the pet not being helped at all. And to me, that is the ultimate failure.

In contrast, according to the Oxford English Dictionary, compassion fatigue is an “indifference to charitable appeals on behalf of those who are suffering, experienced as a result of the frequency or number of those appeals.” Basically, it means you stop caring because you’re required to care so often. Anyone in any kind of long-term caregiving capacity should be able to understand compassion fatigue, including parents. And of course, this problem definitely exists in our profession. It’s a very sad, destructive, and dangerous. It should be handled with extreme care. We can also take a look at what we consider compassion fatigue to be, what it initiates it, and whether or not we can re-categorize the feelings in a more productive way.

While I personally understand compassion fatigue in all its clinical glory, I just don’t relate to it. I’ve cared for my infant child who didn’t sleep through the night for 11 months straight. I’ve attended to nine in-home euthanasias in one day. I’ve dealt with case after case in the ER, day after day. We all know the feeling of peeling off our socks and lying down in bed after more than 24 hours on the job, only to be awakened by a text or phone call from the clinic (or even a friend) with a question about another pet.

To me, this isn’t compassion fatigue. This is being overworked, under slept, burned out or simply really, really exhausted. In such moments, I’m not lacking compassion. I’m lacking rest (or a Snickers bar!).

The overuse of the term “compassion fatigue” can be a dangerous path for our profession, which is why the distinction between it and “ethical fatigue” has been so inspiring to me. Instead of using compassion fatigue to describe any negative emotion we experience on the job, diving deep and understanding our unique stressors offers a much more holistic approach to a healthy working environment. The idea of being drained of compassion makes the experience personal, which can take a heavy toll on anyone.

Conversely, the concept of being ethically fatigued puts the problem between the pet and the tough decision that needs to be made instead of between the pet and our personal inability to feel compassion. This was an important distinction for me early on in my career and has made a huge difference in my emotional health.

Years ago, when a pet owner presented me with my fourth parvo puppy of the night and then proceeded to become angry because she believed I was “just in it for the money” and that I “must not care about animals,” I did feel like my compassion had been completely drained. I thought, “How can I possibly care when this person doesn’t realize the dedication and drive it takes to deal with people like her—especially when parvo is completely preventable with a $15 vaccine?”

Interactions like these used to drain me. But as I watched this woman get angry with me, her 8-year-old daughter sitting in the exam room wearing clothes that clearly hadn’t been clean in some time, telling me she would beg on the side of the road to pay for her puppy’s treatment and that I was a terrible person for making her do that, I realized her anger had nothing to do with me. With all the
struggles, she must have had in her life, it took bravery to bring her pet into a clinic where she surely knew she couldn’t afford treatment.

I looked at her and said, “I want to thank you for bringing Piper here, and I’m so sorry if you feel judged by me or my team. Let’s figure something out together.”

Her anger subsided and she calmed down. I realized there was something worse in this world than being berated by a client’s misdirected anger. It was that puppy dumped and left to die on the side of the road with no person. It was letting that little girl feel that a veterinarian didn’t want to help. Seeing this woman as a human being instead of someone who was “out to get me” changed my perception and changed what was possible.

Immediately, the compassion I had for the puppy drove my decisions because my self-defensive guard was down. Instead of being overwhelmed by a lack of desire to help the woman, I was able to concentrate on the need to help the patient. And most importantly, I began to realize something about myself—you can’t give what you don’t have, and as long as I was my own source of compassion, I would never run out.

I realize this may not apply to everyone, but as a woman and a mother, I believe I have an endless supply of compassion. In hospice practice I work with people every day who need empathy and support, and I never feel drained by them. Do I feel tired, stressed, overworked, under slept and perhaps a little burned out at times? Of course, but that’s not compassion fatigue. It’s having an empty tank, and it can be refilled by engaging in things that bring us joy.

Simply put, it was not an overdraft on my compassion supply that led me to feel fatigued. It was the immense responsibility to make the right decision within the boundaries of someone else’s (often) illogical values or unreasonable budget. That is not compassion fatigue; it’s ethical fatigue.
Verbal Communication During Euthanasia: What to Say when You Don’t Know What to Say
Dani McVety, DVM
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As veterinarians, we have two parties to serve in almost all areas of our profession; first, the owner/client, and second, the patient. In human medicine, the client and the patient are generally the same entity. Even in pediatric care, 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 choice to removal of “life support” and initiate euthanasia 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!

As we move through this discussion, remember that veterinary medicine these days is more like pediatrics than the “horse mechanics” we were generations ago. Pets are family now. They have moved from the barnyard to inside the home to our bedroom… and even under our sheets! Even the survey conducted by the American Veterinary Medical Association (AVMA) (1) found that of 63.2% considered their pets to be family members. Another 35.8% considered their pets to be pets or companions and only the remaining 1% considered their pets to be property.

In the previous lecture, Body Language - Your Personal Curb Appeal, we discussed non verbal communication and its role in the interaction we have with clients. This next part of the communication picture relates to the words we say, and how we say them.

The use of pet pronouns
The words we choose to use when describing pets must be reflective of the importance they hold in the family. Sure, some people may view their pet as “just a dog,” but those people will be only slightly offered by your endearing use of the word “baby” as compared the owner that refers to herself as “Charlie’s mom,” she will be much more offended by the use of the pronoun “it”!

Through many discussions with 1000’s of veterinary professionals, veterinary students, veterinary receptionists, and our highly trained team of care coordinators, I have personally come to the conclusion that more than half of us are wiling to say the word “baby” when referring to a client’s pet. Of course, that doesn’t mean we all prefer this term. Personally, I’m not completely comfortable with its use, but I’ve adopted it based on the reaction from pet parents (also a phrase gaining traction). In our Support Center, we use the term “baby” (not fur-baby, I personally don’t like this one at all), only before we have the name of the pet, which we ask for immediately. Once the pet’s name is known, there is no greater word than the name given to him/her by his owners.

Along these same lines, we have adopted the use of “pet parent” in our practice, but still generally use the word “owner” when referring to the case amongst colleagues. Again, “pet parent” is not my personal first choice, but the upside of conveying we understand the importance of the pet in the family is much more beneficial than risking the downside of appearing “cold” or “rude.” It’s rare that someone is genuinely offended by the use of these overly fluffy words, even if it’s not their first choice either. But with 84% of pet owners referring to themselves as “mom” or “dad,” this doesn’t seem too far off the mark (2).

Tone of voice
Cats and dogs both use different vocal tones at different times of stress, attraction, play-seeking, or almost any other behavior. Humans also deepen their voice while making their speech sound “more pleasant” when talking to someone they find attractive. A recent study illustrated this point (3):

We examined how individuals may change their voices when speaking to attractive versus unattractive individuals, and if it were possible for others to perceive these vocal changes. In addition, we examined if any concurrent physiological effects occurred when speaking with individuals who varied in physical attractiveness. We found that both sexes used a lower-pitched voice and showed a higher level of physiological arousal when speaking to the more attractive, opposite-sex target. Furthermore, independent raters evaluated the voice samples directed toward the attractive target (versus the unattractive target) as sounding more pleasant when the two voice samples from the same person presented had a reasonably perceptually noticeable difference in pitch.

The idea of using a lower pitched voice to influence others in a multitude of ways has been known for quite some time. Even Margaret Thatcher was known to have too “shrill” of a voice at the beginning of her career; so much so that she was not allowed on
party broadcasts. But before her election in 1979, she worked with a speech coach to help lower her pitch and develop her infamously calm, authoritative tone. Her biographer Charles Moore later wrote, “Soon the hectoring tones of the housewife gave way to softer notes and a smoothness that seldom cracked except under extreme provocation on the floor of the House of Commons.” (4)

Aside from lowering the vocal tone, a common mistake is the use of “up-speak.” A frequent mistake in women (though men can do this as well!), this offender ends every sentence on a higher note than the rest of their speech. Doing this makes everything that’s said sound like a question and, most importantly, gives up the confidence we wish to convey to our clients. Some professionals feel this kind of tone is very “California-Valley-Girl.” With the perception this speech pattern makes its users appear young, immature, and overall uncertain. Instead of ending a statement on a high note (literally, not figuratively), try ending it on a consistent, or even lower pitch (NOT softer), to convey a strong sense of confidence.

Salutations
We’ve all been there, the typical “hi, how are you” followed by the “great, how are you?” and then, it’s really bad, one more “I’m great, how are you…” and then you’re lost. When responding to the customary “how are you?” find and use (and re-use!) a phrase that you really love: “loving life and living the dream!” or “this is the best day of my life” or “it couldn’t be better, I get to play with animals all day!” Any of these will leave the client feeling happy (hopefully), and at minimum, spark a curiosity in them that may lead to an interesting conversation.

Sounding persuasive
Though there are 100’s of tips on sounding persuasive, we have chose our top 3 below: Talk moderately fast, use just enough pitch, and use powerful pauses.

Rate of speech
Speaking at a regular rate, perhaps even moderately fast, has been shown to be positively correlated with perceived intelligence. “Interviewers who spoke moderately fast, at a rate of about 3.5 words per second, were much more successful at getting people to agree than either interviewers who talked very fast or very slowly,” said Jose Benki, a research investigator at the U-M Institute for Social Research (ISR) (5). Throw in a bit of humor, and you have a recipe for winning someone over!

Pitch variation
Some researchers have shown that the more active the pitch and variation, the more energetic and engaging someone may appear. This isn’t always the case, however; “We found only a marginal effect of variation in pitch by interviewers on success rates. It could be that variation in pitch could be helpful for some interviewers but for others, too much pitch variation sounds artificial, like people are trying too hard. So it backfires and puts people off” said Benki (5).

Powerful pauses
“When people are speaking, they naturally pause about 4 or 5 times a minute,” according to Benki. “These pauses might be silent, or filled, but that rate seems to sound the most natural in this context. If interviewers made no pauses at all, they had the lowest success rates getting people to agree to do the survey. We think that’s because they sound too scripted. People who pause too much are seen as disfluent. But it was interesting that even the most disfluent interviewers had higher success rates than those who were perfectly fluent (and did not use pauses).”

Particularly in a high paced, knowledge based profession like veterinary medicine, you are best to make your verbal deliveries with minimal variation, focusing instead on tone, include natural…. steady… frequent… pauses!

Sounding honest
In Alex Peatland’s book, “Honest Signals: How They Shape Our World,” the authors point out three to keep your eye on (6):

1. Speech mimicry and behavioral mimicry. Are they using the same words you use? Speaking at a similar speed and tone? Are they sitting the way you sit? Is a subtle, unconscious game of follow-the-leader going on? This is a sign the other person feels emotionally in sync with you. It can be faked but that’s rare and difficult to pull off consistently across a conversation.

2. Consistency of emphasis and timing. This is a sign of focus and control. Someone who is less consistent is less sure of themselves and more open to influence.

Win them over again
If all else fails, what are 2 things you can do to win someone over? Robert Cialdini, author of the must-read book “Influence,” provides these important tips:

1. Give Honest Compliments. It may not be easy, especially if the person has been distancing themselves from you for a while. But if you’re objective, they probably have some qualities you admire. If you take a positive action and compliment them, it may well break the ice and make them re-evaluate their perceptions of you.

2. Ask for Their Advice. Cialdini notes this strategy – which involves asking for their professional advice, book suggestions, etc. – comes from Founding Father Ben Franklin, a master of politics and relationship building. “Now you’ve engaged the
rule of commitment and consistency,” says Cialdini, in which they look at their actions (giving you advice or a book) and draw a conclusion from it (they must actually like you), a surprisingly common phenomenon in psychology. “And suddenly,” says Cialdini, “you have the basis of an interaction, because now when you return it, you can return it with a book you think he or she might like.”

Verbal communication is, indeed, extremely important in the communication we have with clients. The delivery, consistency, and accompanying non-verbal cues give the client the feeling that we are either listening and engaged or detached and uninterested. We have a choice, and with proper education, we can be in a better position to choose the best route for our patient, our client, and our team.

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Honey bee disease etiologies
  • Bacterial
    o American Foulbrood
    o European Foulbrood
  • Fungal
    o Chalkbrood
    o Nosema
  • Parasitic
    o Varroa mites
    o Tracheal mites
    o Small Hive beetles
    o Tropilaelaps
  • Viral
    o Over 20 different viruses
  • Toxicities
    o Pesticides
  • Idiopathic
    o Colony Collapse Disorder
  • Environmental
    o Starvation
    o Excess Humidity

Varroa destructor
  • Foreign Animal Disease in USA prior to 1987
  • Now very common across USA
  • If bees sick, this is the first differential
  • Reproduce well with honeybees
  • Carry diseases
  • Complex control methods, no silver bullet
  • Bee keepers... Bee havers... Bee hadders
    o Life b.m.
    o Tracheal mites 1984
    o Varroa mites 1987

Varroa destructor – life cycle
  • Mites on bees = Phoretic mites
  • Female leaves bee, enters brood about to be capped
  • Lays eggs starting 60 hours after capping

Varroa life cycle
  • First egg = male (haploid)
  • Lays egg every 30 hours
  • Rest are female
  • Feed on pupa
  • Mate as adults in cell
  • Emerge with adult bee
  • Drone brood preferred
    o ~15 days available
    o Worker ~ 12 days
    o Extra 2 mites per time in brood
Population dynamics
- Stop adding bees
- Keep adding mites
- More bees removed
  - “worked to death”
- Higher mites per bee
- More disease spread because more chance an infectious mite bites a bee.

Varroa – What problems caused?
- Puncture the exoskeleton of the bee
- Wound caused, and immuno-suppressors injected.
- Feed on fat bodies
- Dirty mouth parts? Contaminated saliva? Opportunistic invaders?
- Now over 20 viruses recognized in bees, most are spread by Varroa mites
- Immuno-suppression
- Viral Loads
  - More mites, more spread
  - Infective Dose varies by route of administration
  - Enhancement by mite of viral infectivity?
  - Types of viruses seen – DWV, BQC, IAPV, CPV
- Decreased lifespan
  - More disease, more ‘accidents’, less exercise tolerance (oxygen carrying capacity)
- ‘Birth’ Defects?

Varroa treatments
- Originally: burn hives until they were eradicated
- Then: Treat with harsh chemicals 1x or 2x per year
- Now:
  - Monitor level of mites.
  - Treat if level too high.
    - Soft chemicals or hard chemicals.
  - Continue other control methods.
  - Recheck mite levels

Varroa mite monitors
- Visual – bad!
- Drone brood exam
- Sugar Shake
- Ether Roll/alcohol wash
- Sticky Boards

Varroa control - Chemicals
- Integrated Pest Management
- Chemicals often seen as last resort.
- Chemicals often used to treat everything whether needed or not.
- Concerns:
  - Kill mites, not bees
    - Some side effects – bees, brood, queens, sperm
  - Honey is a food product
  - Wax is considered a chemical “sponge”
    - Studies show wax has a lot of residues, many are beekeeper applied miticides.

Varroa mite control
- Natural Methods
- Drone Comb Removal
- Remove breeding mites
  - Breeding resistant bees
    - Russian bees, Varroa Sensitive Hygiene, “ankle-biters”
- Swarming
- Splitting
  - More time without brood where mites hide and reproduce. Phoretic mites exposed to bees.

**Common clinical presentations**
- My bees were very strong, now they are all gone
- I was new this year, so I did not have to worry about mites. Why are my bees all dead?
- I have honey in the hive, and a small cluster of dead bees.
- I treated in August and now in November my bees are all dead.
- I looked for mites, but didn’t see them so I didn’t do any treatments.
- I am a natural beekeeper, so I did not treat them.

**Tracheal mites (Acarapis woodi)**
- 1919 First found in Isle of Wight in English Channel
- US Honey Bee Act of 1922 prevented import
- 1984, tracheal mites found in USA.
- Extensive losses, breeding efforts, treatments, now hardly recognized
- Microscopic mite that lives in breathing tubes and is transmitted bee to bee.
- Control was chemicals (menthol) and disruption of the bee to bee transfer (grease)

**American foulbrood**
- Paenibacillus larvae
- Easily spreads
- Contaminated comb, honey, clothing, tools & hands
- Kills pupa
- Ropes
- Clears milk
- Smells

**AFB – How to diagnose**
- Piece of comb 2 inches square
- Wrap in newspaper
- NOT in ziplock or plastic wrap
- Not molded or rotten
- Clinical signs
- ELISA tests
- Milk Test
- Laboratory
  - Aerobic culture on blood sagar
  - Also do sensitivity
    - Kirby Bauer OTC and TYL
  - USDA or veterinary labs?

**Antibiotics to treat?**
- Vegetative Stage - clinical disease
- Spore Stage – able to spread disease, but not visible
  - Able to be inactive for several decades
- Prevention of AFB vs Cover Up?
- Radiation?
- Once a beekeeper gets on antibiotics, they are loathe to stop. Many books advocate antibiotics once or twice a year.
Shook swarm method
- AFB not extensive
- Shake all bees off frames into a box
- Burn Frames and scorch wood of hive bodies
- Place bees on new frames with bare foundation
- Bees use honey to power wax glands, excrete any spores outside instead of storing contaminated honey
- Feed antibiotic while building comb
- Variable success depending on severity.
- Most states do not allow this for AFB treatment.

American foulbrood
- Is it best to suppress clinical signs by feeding antibiotics?
- Is it best to allow it to show up and then burn it to rid the apiary of spores?
- Overall, maybe 1% incidence
- Historically a lot higher
- 40% in New York State mid 1900s
- Reason most states have bee inspectors
- Many beekeepers and inspectors fear a massive outbreak once antibiotics are not available OTC.
- Others think this will help weed out bad beekeepers.

European foulbrood
- Melissococcus plutonius
- Kills larvae
- Bacteria outcompetes larva for food and larva starves.
- Much more common than AFB
- Stress association
  - Forage availability
  - Humidity
  - Population of adults

EFB diagnosis and control
- Clinical signs
- ELISA
- Culture
  - Frequent overgrowth with other bacteria
- New York State:
  - EFB is changing? Seeing it without characteristic upturning of larvae
- State inspector calling it AFB, lab confirms EFB
- Requeen
- Feed
- Increase population
- Feed Oxytetracycline
  - Resistance??
- Shook swarm method is advocated in Europe
- Recurrence common, regulatory effort is minimal

Chalk brood
- Ascosphaera apis
- Fungus
- Very common but not very severe problem
- Often spring time when low bee numbers per brood and have chilled brood
- Clears as colony strengthens and temperatures warm up
- Requeen if problem.
Nosema
- Caused by a single cell protozoan
- Two different types
  - Nosema apis
    - Diarrhea disease of early spring
    - May affect packages and weak colonies
  - Nosema ceranae
    - Newly recognized in US
    - Colony loss and dwindling of population
    - Bees tend not to demonstrate diarrhea
    - Significance not clear (2009)
    - Can be a severe problem (2010)
- Infects cells lining of midgut
- Less efficient use of protein and greater energy consumption due to poor absorption of nutrients
- A strong colony may not show symptoms
- Like other bee diseases, stressors make Nosema symptoms much worse.
  - Poor quality pollen
  - Cool damp weather
  - Small colonies
  - Failing queens
  - Long periods without cleansing flights

Diagnosis
- Microscopy for spore counts
- Counts vary widely between bees and by season
- Healthy prolific colonies will experience decreasing Nosema levels
- Spore counts
  - Crush a number of bees with known water, Mix thoroughly
  - Count with hematocytometer
  - -OR-
  - Smash single bee onto slide
  - Look for spores as high or low numbers
  - Problem is if 3 or more out of 10 have high numbers

Diarrhea does not equal Nosema
- Normal feces
- High ash feed
  - Non-digestible feeds
  - Darker honey
- Lack of flying weather
- Amoeba?

Treatment
- Nosema is a spore former
- Treatment may be necessary
- It is not appropriate to treat blindly though many do
- Little correlation between spore counts and illness
- Fumagilin ????
  - Nosema comes back worse after treatment?
- Reason to rotate frames out more often?
- Acetic acid or ozone fumigation of old combs?

Fumagilin
- Antibiotic mixed in sugar syrup.
- Do not mix in hot syrup.
- Apply to bees after or before honey flow, not during honey flow.
- No active NADA in the USA.
- Product imported from Canada and FDA CVM uses enforcement discretion to allow its import.
- Not on list of human medically important antibiotics, so still OTC

**Small hive beetles**
- Aethina tumida
- Africa origination
- Egg, Larva, Pupa, Adult complete metamorphosis
- Pupa is outside hive in sandy soil
- Emerging as adult, can fly miles to find a bee hive.
- Fast movement
- Look for dark places to hide
- Can overwhelm weak hives
- Bees usually “jail” them by chasing to a hiding spot and not allowing them to leave
- Traps rely on this – bees chase beetles into trap, oil in bottom, so beetles fall in and can’t get out.
- Beetle larvae destroy honey
- Extract honey as soon as removed from hive
- Treatments and control
- Oil Traps
  - Check Mite (coumaphos) hidden in piece of cardboard, mite accesses to hide, bees can’t follow
    - Off the market currently
- Ground treatments – GuardStar
  - Kill pupating mites
  - Beetles seek ground beyond the treatment
- Off label treatments as beekeepers experiment?

**Wax moths**
- Strong populations of bees keep control.
- Dead or weak hive problem
- Brood combs destroyed
- Confused with Small Hive Beetle larvae
- Paradichlorobenzene control in stored equipment.

**Colony collapse disorder**
- Colony of bees decreases from populous to brood with few adults and a queen without explanation
- Neonicotinoids
- Fungicides
- Drought, mono-culture ag, yards – lack of forage
- Nosema
- Varroa and viruses
- Poor management

**Pesticides**
- Pickup pesticide from crop or exposed by spraying or forage on broken bags of pesticides
  - Large pile of dead bees in the hive/at hive entrance
  - Collect sample of bees, freeze them.
  - Call your state pesticide contact.
  - USDA pesticide screen ~$300 per sample.
- Neonicotinoids – systemic pesticide
  - Safe for mammals
  - In pollen or nectar?
  - Sub-lethal effects on bees are an added stressor
Foreign animal diseases

- Tropilaelaps mites
  - Similar life cycle to Varroa mites
  - Asian Honey Bee mite
    - Apis dorsata
- Brachypeplus basalis
  - Beetle similar to Small Hive Beetle
  - Now found in USA in commercial colonies on west coast

Emerging diseases?

- Sepsis and Hemocyte Loss in Honey Bees (Apis mellifera) Infected with Serratia marcescens Strain Sicaria
Veterinarians and bees
- FDA now mandating veterinary involvement for antibiotic use
- Veterinarians need to jump up and seize the opportunity!
- USDA does not look to veterinary involvement
- Bees need
  - Preventive Care
  - Parasite Control
  - Disease Diagnosis
  - Disease Treatment
  - Beekeeper education

Veterinarian in the apiary
- For a valid VCPR, the veterinarian needs to visit the apiary (bee yard)
- Moving bees to the veterinary office is difficult
- Dead hives may be brought to the office?
- Smartphones and pictures?
  - What can you learn on site?
  - How strong are the hives
  - Where are they kept
  - Water available?
  - How does the beekeeper keep the bees?
  - How does the beekeeper act around the bees?
  - Unrecognized conditions in other hives?
  - When visiting, you should have a veil that will protect your face and head
- You should have a clean hive tool and smoker
- It may be advisable to have the beekeeper provide these for you
- You should not wear leather gloves. We find Nitrile milker gloves work well
- Have sampling supplies available – plastic bags, bee sampling bottles, newspaper
- Walk and conduct yourself in a slow deliberate manner to avoid upsetting bees.
- Establish a list to examine so you can do a thorough physical exam
- Clean up when you leave – bucket of water, chlorinated cleaner like Comet, steel wool, torch to heat hive tool
- History – why are we here? What have you done?
- External examination – environment, hive appearance, behavior of bees at entrances, soil
- Internal examination – behavior of bees, size of hive, spacing of frames, smells, presence of other insects, abnormalities of the bees
- Internal examination – brood frames, color, type of brood, pollen, honey stores, wax moths, beetles, larval diseases, brood pattern
- Internal examination – what is on the bottom board or sticky board

Commercial beekeepers
- Sign a prescription or VFD so I can do what I always do.
- Disease sampling
- Disease recognition
- Disease management
- Protocols for when to use treatments
- All the stuff listed for Hobbyists

Hobbyist beekeepers
- Education
- How to work bees
• What is normal?
• What caused this problem?
• Do I have a problem?
• What do I do about it?

**Veterinarian in the apiary**
• Veterinarians are highly trained in physiology, anatomy, disease, diagnosing, treatment and preventing. The FDA recognizes this and is why they require a veterinarian before antibiotics are used.
• Many beekeepers are older and have done bees for a lot of years. They can still learn about bees!
• People earn PhDs in beekeeping
• There are a lot of stories in beekeeping that may or may not be true. They also may or may not apply to the situation at hand.
• “More is missed for not looking than not knowing” Thomas McCrae

**Veterinarian in the apiary**
• Subjective
• Objective
• Assessment
• Plan
• Works as well in the apiary as it would in the hospital
• Medical records prove VCPR if any issues with VFD or prescriptions

**Antibiotics in honey bees**
• Commercial beekeepers
  o Move bees into crops for pollination
  o Poor biosecurity
  o High disease exposure
  o Feed antibiotics to kill whatever they can to prevent bringing home disease
• Commercial beekeepers
  o Weak hive treatments and prevention
  o Queen mating hives
• Any beekeeper
  o Prevention of American Foulbrood
  o Should we? Are we just covering up previous bad management?
• Veterinary Client (Beekeeper) Patient (beehive) Relationship
  o Physical presence at apiary
  o Yearly? Every 6 months?
  o Personally acquainted with husbandry
  o Willing to accept responsibility for medical management and follow up
  o Beekeeper willing to follow directions
    ▪ There is a lot of non-labeled product going into beehives.
    ▪ Can you control this behavior?
• 3 antibiotics with Honey Bee labels
  o Oxytetracycline, Tylosin, Lincomycin
• All are given to bees by mixing powdered antibiotic with powdered sugar or mixing in a grease patty
• Apply 2 Tablespoons to hive by spreading around outside of hive.
• Do not apply to brood
• Oxytet has VFD and RX forms
• Others have only RX forms

**Residue avoidance**
• Antibiotics will appear in honey
• Honey is tested for antibiotic by some packers
• No reporting requirement to regulatory bodies
  o Milk is different from honey
• Beekeepers feed antibiotics before any honey being collected or after removed honey for human consumption
  o Honey for humans usually collected late spring though early fall.
• ELISA tests available for on farm or in plant testing

Antibiotics in honey bees
• Prior to writing VFD or RX, do I need a diagnosis?
• Prevention, Control, Treatment
  o FDA terms without definitive definitions
  o Control means there is disease and you are keeping it from spreading in others that you treat
• What is the quality of a diagnosis?
  o Physical signs
  o In house/hive side testing
  o 3rd party laboratory testing

Hive side or in clinic ELISA tests
• About $13 each
• AFB or EFB
• Highly purified monoclonal antibodies used to detect antigen of bacteria
• Mix samples in bottle of fluid, mix well, place sample on well, wait 5 minutes
• Line on C=control T=test

Culturing honey bee bacteria
• Paenibacillus larvae
  o Grey white colonies on agar. Orange possible also
  o Spore forming and motile rods
  o G+, but difficult to stain
  o Catalase -, esculin +
  o Also consider wet mount. Only bee pathogen that shows Brownian motion.
• Melissococcus plutonius
  o Small white, shiny, well defined colonies
  o Non motile cocci in pairs or chains
  o Difficult to grow, other associated bacteria also grow

USDA lab – Beltsville MD
• Brownian motion on wet mount
• Culture on blood agar
  o Maybe heat shock
• AFB makes a spore
• AFB fluorescent under UV light
• Smell
• Kirby Bauer Disk for resistance
• 5microgram Oxytetracycline disk
• Most isolates clear at least 50mm zone
• Less than 50mm zone is resistant

Dead hive necropsy
• Lots of causes of dead hives. Diagnosis of cause of death or ruling out a cause may help beekeeper apply limited resources to prevention
• Starvation
• Varroa Mites
• American Foulbrood
• Freezing

The Necropsy
• Are any bees present
• Honey present?
• Bees present?
• Organized cluster?
• Brood present?
• Fecal staining?
• Testing

Using deadout resources
• If AFB and Nosema can be ruled out combs can be reused
• Honey can be fed
• Combs will greatly aid packages or nucs
• Moldy combs
• Secure resources before robbing/wax moths
• Many beekeepers will destroy these valuable resources in case a low incidence disease caused the deadout.

Parasitic mite syndrome
• Guanine deposits = Mite poop

European foulbrood
• Curled larvae
• Out competed for food
• Spotty brood patterns
• Culture
• ELISA
• Confused with AFB
• It is a great service to diagnose correctly as this does not need to be burned!

Diagnosing conditions
• Laying worker
• All drone brood
• Worker did not get inseminated so only lays haploid eggs and cannot reach bottom of cells
• Treatment
  o Add a queen via a nuc
  o Combine with other queenright hive
• “My hive swarmed two weeks ago, now there are no eggs or queen, how do I introduce a new queen?”
• Maybe a queen is not needed.
• By knowing normal bee biology, we know swarm leaves between a week prior to new queen emerging and the day of emergence, it takes her a week to mature, then she goes on another week of mating flights.
• At 2 weeks post swarm, no eggs or queen normally. New queen may start laying soon.

Chilled brood
• Expand brood nest
• Cold weather, so bees cluster and cannot keep brood warm
• Beekeeper “reverses” hives and splits brood
• A lot of resources for bees to clean this up

Overheated bees
• Bees are a heat producer
• Need ventilation
• Nucs or packages or hives
• In transport, usually netted, not locked in
• Queen’s sperm storage may kill future fertility.

Euthanasia of bees
• Sprays require contact and are effective
• Sprays are short lived
• Sprays tend not to get carried into a hive
• If bees are inside a cavity, sprays won’t work. You are better to open the cavity and remove the combs and bees.
• Overturned trucks, ground bees (yellow jackets)
• Soapy hot water
• Firefighting foam
• Surfactants that break the surface tension and allow liquid to enter trachea of bees.

**Microscopy**

• Tracheal Mites
• Nosema

**Veterinarians and beekeepers**

• Veterinarians are highly trained biologists that can systematically examine a system for abnormalities.
• Veterinarians are highly trained in disease diagnosis, treatment and prevention
• More is missed for not looking than not knowing
• People earn PhDs in beekeeping
• There is a lot of superstition out there. The FDA is looking for veterinarians to bring the science.
Honey Bees and Veterinarians
Christopher Cripps, DVM
Betterbee
Greenwich, NY

Chris Cripps
• Started beekeeping with the Boy Scout Beekeeping Merit Badge in mid 80’s in New Hampshire
• Cornell – classes in Bee Biology with labs
• Ohio – DVM OSU
  o Inspector for 2 counties (Columbus area)
  o Make appointment with owners, look over hives for diseases, take samples for lab as needed, report health status to owner and recommend any treatment needed.
  o Moved 6 hives to a horse farm in Ohio from NH
• New York – Greenwich
  o Arrived in 1995 with 6 hives, all my stuff, and $10 in the back of a U-haul
  o Kept up to 12 hives, sold honey, moved bees for pollination
  o Worked as dairy veterinarian at Battenkill Veterinary Bovine
• September 2012 – bought Betterbee (bee supply business) and left veterinary practice?? Now partners with veterinarians Joe Cali and Jack Rath and 2 others.

Betterbee
• Beekeeping supply business that sells bee hives, frames, foundation, extractors, queen rearing supplies, fencing, honey bottles, bears, caps, books, classes
• Beekeepers Serving Beekeepers
• Teach beginner & intermediate beekeeper classes
• Answer questions from customers
• Supply scientific information about beekeeping

Honey bee veterinary practices
• With 3 veterinarians, I thought we had the largest group of veterinarians in honey bees
• Visited Wilbanks Apiary in Claxton, Georgia
• 7 veterinarians worked there one year- Argentinians

Honey bees
• Veterinary curricula in many countries include honey bees
• USA and Canadian schools do not include bees
• Don’t worry, you did not miss it, it is not offered
• New antibiotic labeling and registration requires a veterinarian to write an order for any antibiotic to be fed to animals
• Honey Bees are Food Producing Animals that have been fed antibiotics
• Only insect listed as food producing animal

How did it work previously?
• Antibiotics with labels for Honey Bees:
  • Oxytetracycline
  • Tylosin
  • Lincomycin
• Indications: for the control of American or European Foulbrood
• Self-reporting survey 2015 of ~5000 beekeepers about 1/14 reported antibiotic use
• They averaged ~900 hives

Honey bees and antibiotics: How did it work before?
• Commercial beekeepers feed antibiotics to prevent disease
  o Mostly oxytetracycline mixed in sugar that has been over the counter
• Little understanding of bacteriology
  o Oxytet to start
  o Tylosin used if oxytet doesn’t work
  o Lincomycin seldom used
• Gather hives in highly populated areas for pollination – California almonds, Maine blueberries
“Cesspools of disease” that cannot be avoided because the pay is so good and may mean the difference between having a profit or loss for the year.

**Honey bees and antibiotics: How does it work now?**
- Prior to transportation, must obtain a certificate of inspection
- Issued by state apiarist
- Bee inspectors visit apiaries, remove brood from a percent of hives (~10%). Submit samples to USDA if any concerns or issue health certificate if no concerns
- Veterinary epidemiologist might help – what percentage of frames should be inspected to find disease assuming 1% prevalence?
- Positive American Foulbrood (AFB) may invoke state-mandated burning of affected hives
- Long lasting spores are contagious to other hives within 3 mile radius
- Burning beehives worth $500 each makes beekeepers wary of outside inspection

**Beekeepers like other food producers**
- Frugal
- Expect value for money spent
- Veterinarians sell what?
  - Knowledge and disease control techniques
  - Signature on antibiotic orders that they did not previously need to have
- If you come to the table to offer services, make sure you offer value
  - Get Educated!

**Important points in bee biology**
- Complete Metamorphosis
  - Eggs, Larvae, Pupae, Adult
  - 1 to 2,000 eggs per day
- Worker egg to adult
  - 21 days
- Queen egg to adult
  - 16.5 days
- Drone egg to adult
  - 24 days
  - Important for Varroa Mites!
- 3 distinct castes of bees:
  - Queen (diploid) – fertile female that lays eggs
  - Workers (diploid) – infertile females that care for young (brood), gather food, clean and defend hive.
  - Drones (haploid) – males that breed queens

**Bee sex and genetics**
- Queen emerges from cell
- After a week of maturing in the hive, starts mating flights
- Mates with 8-20+ drones over next week
- Never mates again
- Does not mate with drones in hive
- Sperm must live many years in queen’s spermathecal
- Langstroth 1852
- “Bee Space” = 3/8 inch
- Movable Frame Hives

**Wax production**
- Carbohydrate drives glands on abdomen in young workers
- Feeding sugar can push more wax production
- Wax used to make all comb
- Paper used by wasps and hornet, not bees!
Lipophilic chemicals persist in wax
  o Chronic pesticide exposure

Lots of brood leads to a lot of bees which lead to a lot of honey
  • Package of bees is 3 lbs
    o Common way to start
  • Contains ~10,000 bees
  • Colony that survived winter will be small and grow through the year

Starter bee colonies
  • Swarm
  • Package
  • Nucleus Hive

Honey bee food storage
  • Protein = pollen
  • Need variety of pollens for a balanced Amino Acid profile
  • Carbohydrates = nectar
  • Perhaps 20% solids converted to 17% water
  • Invertase, dehydration
  • Mixture, ferment = bee bread
  • Do fungicides hurt bees?

Honey bees, directions and food
  Drifting
    • Bees make orientation flights to determine where their hive is before foraging.
    • Bees may return to wrong hive if many in same area
    • Welcomed if they have food
  Robbing
    • Strong hives defend themselves from robbing
    • Weak hives robbed by strong hives
    • Drifting and Robbing can lead to disease spread

Winter time
  • Honey bees keep cluster heated throughout the winter
  • ~95deg F when raising brood in January
  • Hornets and wasps overwinter on or underground as individuals
  • Bees don’t defecate in hive
  • Process honey to keep hive warm
  • Need to fly to defecate
  • “Cleansing Flights”
  • Once below 50°F, cannot operate muscles, fall out of air

Protective clothing
  • Veils
  • Blinking attracts bees
  • Gloves
    o Leather cannot be cleaned
    o Nitrile gloves
  • Coveralls and jackets with incorporated veils

Smokers
  • Calms bees
  • Burn many different Materials
  • Light with a propane torch
• Store safely if driving between apiaries
• Clean bellows well to avoid disease spread

Bee stings
• Stinger is modified ovipositor
  o Drones do not sting
• Worker sting barbed
  o Rip insides of bee out
  o Only one sting then dead
• Swelling normal reaction
• More times stung, less swelling
• Anaphylaxis possible
  o Epinephrine
  o Diphenhydramine

Beekeeper behaviors
• Work bees in a calm manner
• Sudden jarring motions elicit defensive behavior
• Falling barometric pressure, rain increase bee defensiveness
• Black colored clothing not good
• Wool or fleece not good
• Bees get legs caught
• “Suit of armor” and go hard at bees
• Wear nothing but veil and be gentle
• Crushing bees can lead to more disease issues

Veterinarians and beekeepers
• Diseases for which antibiotics may be used
• American Foulbrood
• European Foulbrood
• Other disease treatments are Over the Counter
• EPA regulated mite treatments
• Non-medically important antibiotic

To order antibiotics: 2 choices
Prescription
• Requires VCPR in most states
• Typical form used in clinics
• Oxytetracycline, tylosin or lincomycin as water soluble forms
• Send to licensed pharmacist or give to client
• ELDU is allowed
• Keep records per state law
• Expiration max???

Veterinary feed directive
• Requires VCPR
• New to most veterinarians
• Oxytetracycline only
• Tylosin, Lincomycin have no VFD approval for bees
• Send to licensed medicated feed mill or distributor
• ELDU prohibited, but not enforced.
• Keep original records for 2 plus years
• Expiration max 6 months

Extra label drug use
• Labeled antibiotics
• For the CONTROL of …
  o Indicates there is a diagnosis of AFB or EFB
  o Treating other hives to prevent spread
• Most antibiotic use will be for PREVENTION or TREATMENT
  o No antibiotics labeled in honey bees for these indications
• VFD cannot be written for ELDU but RX can
  o New FDA announcement allows ELDU in minor species with some restrictions. Check these out first!
  o Still too limiting to help beekeepers???

Honey bee prescription issues
• Identification
  • Beehives tend not to be individually identified
    o No state tags
  • Apiaries identified by address
  • Most states say you must personally visit the apiary for a valid
  • VCPR
  • Follow up may be by other means once valid
  • VCPR is established
    o Telephone
    o Pictures/Email
  • AVMA PLIT will acknowledge your work with bees
  • Products available
  • Feed mills may make product for VFD fulfillment
  • Need not be antibiotic licensed feed mill
  • Type A medicated article (Terramycin) comes in bag for 8000 treatments
  • Prescription product of oxytetracycline is in much smaller packets
  • Should we be using antibiotics?
    • European foulbrood
      o Can be a problem and antibiotics can be convenient treatment
      o Small queen rearing hives
    • American Foulbrood
      o May cover up the clinical signs and hide disease
      o Still contagious
      o Transfer disease to people buying used equipment
      o Many people think it should not be used
      o Europe prohibits

Veterinarians and bees
• Who is in a better place to provide this than veterinarians?
• To provide those service, gain knowledge
• If you understand the disease and its nuances, the beekeeper may view you as a source of knowledge rather than another bill
• Join the Honey Bee Veterinary
  o New website to help beekeepers find veterinarians
The human-animal bond
The AVMA defines the Human-Animal bond as: “A mutually beneficial and dynamic relationship between people and other animals that is influenced by behaviors that are essential to the health and well-being of both. This includes, but is not limited to, emotional, psychological, and physical interactions of people, animals, and the environment.” (1) Perhaps a simpler definition of the human-animal bond might be the strong emotional attachment people feel for their pets. Numerous scientific studies have demonstrated the physiological and psychological health benefits people derive from the presence of animals in their lives. (2-6)

Why is the human-animal bond important to veterinary teams?
The human-animal bond is the very reason we can practice medicine and surgery at the level we do today and it is key to our personal, professional, and financial success. The veterinary profession has seen a drastic shift in the way our clients perceive their pets over the last 5 – 7 decades. That, along with technological advances and the willingness to spend money on that technology has allowed us to practice medicine at a higher, more fulfilling level professionally. At the same time, the daily challenges veterinarians and their teams face in practice take a huge toll on us mentally emotionally, and physically. There are many stressors on the veterinary professional including but not limited to: economic debt, long hours, and perfectionism. In addition, veterinary caregivers have an innate, deep connection to the human-animal bond. This connection brings us great fulfillment while at the same time exacting a huge emotional toll. Clients inability to pay for care, animal suffering, economic euthanasia, difficulty in maintaining clinical distance along with other factors, all play a part in this toll on veterinary caregivers. These factors can all contribute to burn out, compassion fatigue, and even suicide. But while the HAB is perhaps part of the reason for this emotional toll, re-connecting to the bond – the very reason that most of us entered this profession, may also be part of the answer to these issues.

People seek veterinary care because of their feelings for their animal companions, and they make purchasing decisions based on those feelings. When we show clients how much we care, they trust our recommendations & trust that we have their best interests at heart. The astute practitioner understands that our best clients are those that have a strong bond. Studies show that practices that use tools and techniques to incorporate the HAB into their practices have increased patient visits and increased income.(7) The astute practitioner understands that our best clients are those that have a strong bond. A strong human-animal bond drives our profession and it is imperative that we embrace, nurture, and promote this bond in our practice.

Applying and prescribing the human-animal bond in practice
While most practitioners have an awareness of the HAB and an understanding of why it’s important to our practices, many struggle with how to create or implement a bond-centered practice. It is important for veterinarians to realize that a Bond-Centered Practice is not just a system of protocols put in place, nor a group of tools or services to be implemented, but rather a philosophy that defines their practice. At the heart of a bond-centeredpractice is an understanding that we must consider the emotional needs of the owner and how our treatment plan will impact the owner and the HAB.

The following list is not complete but merely represents one practitioner’s thoughts on how veterinary teams can begin the journey to meet the emotional needs of pet owning families and develop a mastery of HAB science.

Be aware of the bond
A bond is being presented at every appointment. And because 50% of that bond is on the human side of the leash, we need to consistently think about both the emotional needs of the owner and how the treatment plan impacts the owner. Likewise, we have to think about how our treatment plan impacts our patient and the bond itself. All veterinary team members have an innate sense of the HAB but often the logic and science of our training along with the realities of practice life tends to push that feeling out of our consciousness. Time constraints and the very nature of our task-oriented profession also gets in the way of our awareness of the bond.

Acknowledge the bond with clients
The bond between an owner and his/her pet has the potential to deepen and grow. Acknowledging the connection is a simple way to validate and strengthen the HAB. This acknowledgement is especially important when an owner is grieving. Our client’s feelings of grief are not always validated by friends, at work, or even at home. In fact, it is not unusual for these feelings to be minimized with comments like: “Why are you so upset? It’s just a dog!” or “Why are you still grieving? It’s been three months!” As a practitioner of the HAB, simply validating owner’s grief and checking in later can have a tremendous impact for a client highly bonded to his/her pet.
Address behavior in your practice
Behavioral issues lead to several negative emotions and will always negatively impact the human-animal bond - sometimes resulting in the ultimate fracture in the HAB – relinquishment to a shelter. Providing behavioral services – either in house or through referral services – is imperative for a practice that wants to become bond-centered. In a bond-centered practice, providing services that help pet owners solve a behavior problem is as important as diagnosing diabetes. For a well-patient visit, asking open-ended questions about a pet’s behavior is as important as asking about appetite and water drinking for a vomiting patient.

Educate pet owners about the bond- Embrace the pet effect!
Educating pet owners on all aspects of pet care is important at every visit but it is also equally important to educate owners on the health benefits of the HAB. In September of 2016, HABRI released findings from a comprehensive survey of pet owners that showed that when educated on the scientific research documenting the health benefits of owning a pet, owners were motivated to take better care of their pets – resulting in a significant, positive impact on veterinary care. (8) The Pet Effect Campaign, is a multi-pronged campaign aimed to introduce pet owners to the health benefits of having a pet in their lives employs a web site, videos, and client education materials to promote this research.

Embrace the Fear Free℠ movement
Fear Free℠ addresses the emotional health of pets as well as addressing the emotional welfare of client and team members. The focus of Fear Free℠ is to eliminate negative experiences with veterinary visits. Techniques to reduce the stress, anxiety, and fear that many pets experience at our hospitals include: limiting food before a veterinary visit, carrier work, music, Feliway/DAP, non-slip surfaces, minimizing waiting room time, and pharmacology to address fear, anxiety, and stress. Currently, certification is available at the individual level but will be available for the entire practice in 2018.

Celebrate the bond in your practice
The journey and philosophy of guiding your practice to becoming bond-centered is an ongoing process that has no endpoint. It becomes part of the daily interactions with all clients and their pets. Discussing and celebrating positive treatment outcomes from the standpoint of how the veterinary team has positively impacted the HAB is an important way to promote the HAB in your practice. Likewise, sharing positive on-line reviews, cards, and letters from clients with all team members is a way to keep the focus on the HAB. It is this author’s opinion that team members tend to respond more favorably to discussing how we’ve touched/improved the lives of our patients’ and clients rather than sharing financial data and goals.

Don’t get in the way of the bond
There are many ways in which veterinarians negatively impact the human-animal bond. We can inadvertently negatively impact the human-animal bond by simply asking owners to do things they don’t want to do or cannot do. These are separate and distinct issues. For example, an elderly client may not have the functional ability to pill their dog or cat or even open a pill vial. A younger client may be able to perform the task but because of lifestyle, simply does not want to be burdened with the responsibility. We also need to be aware of unintentional consequences impacting the HAB when choosing a therapeutic protocol. The act of medicating a pet may create a negative experience where a normally loving and constant companion now runs and hides from its owner. This can be significant if chronic medications are required. Likewise, missed or forgotten doses can create a negative experience with owners feeling guilty. Side effects from medications may significantly impact the human-animal bond as well. For example, therapeutic corticosteroid use often creates issues with inappropriate urination, behavioral changes, and polyphagia. Be aware of how medication side effects may impact the HAB.

Compliance
Compliance is a common problem in veterinary practice that is often overlooked or underestimated by practitioners. The 2003 AAHA Compliance Study indicated that the majority of veterinarians overestimate the rate at which their clients comply with diagnostic and therapeutic recommendations (9). For medications that are prescribed twice daily, this study indicated that only 30% of clients give them as prescribed – well under what most practitioners estimate. In addition, it has been shown that multiple doses per day increase the likelihood of pet owners missing or delaying a dose (10). Other studies have found similar lapses in compliance (11-13). The impact of poor or non-compliance can be significant in our practices and in our clients and patients’ lives. Poor compliance results in poor treatment outcomes. A poor treatment outcome means our patient continues to suffer. For our clients, a poor treatment outcome may result in anger and/or frustration due to additional costs and their pet’s continued suffering. Unhappy clients in turn impact the reputation and bottom line of our practices as irritated clients often leave the practice permanently or to seek another opinion.

Impact on the human-animal bond
Many pets are difficult for their owners to medicate or dislike receiving treatments. Likewise, many clients either cannot, or prefer not to medicate their pets. When we prescribe medications in these situations we may inadvertently impact compliance and damage the bond between an owner and their pet. Being aware of what impact a treatment protocol may have on the relationship between an owner and their pet will aid the practitioners in making good choices that will not negatively impact compliance or this bond. Therefore, an awareness of the human-animal bond should be an important factor in our treatment choices.
The consequences of both poor compliance and negatively impacting the human-animal bond can be avoided through awareness and open-ended questioning. When taking a history, ask open-ended questions and pay attention to extenuating circumstances that may forewarn potential compliance issues and/or damage to the bond. Do not assume that all pet owners can or will medicate as prescribed. Be aware that the act of medicating a pet may create a negative or stressful encounter. Additionally, this stress may be accentuated with missed or forgotten doses as the pet owner experiences anxiety or feels guilty about their role in their pet’s recovery. Replace closed questioning such as, “Can you medicate you’re your pet?” With open-ended questions such as, “Tell me about your experiences medicating your pet”. Open-ended questioning of the pet owner is imperative in determining the best treatment option for a successful outcome. To the closed-ended questioning, many pet owners will often answer yes, even when extenuating circumstances are present that will preclude compliance. All of us want to appear to be “good” pet owners and certainly able (and willing) to give medication to our pets. With open-ended questioning the owner may share information that will steer the direction of treatment when choices are available. For instance, once daily dosing may be attainable whereas twice daily just won’t work. Again, look for clues and listen carefully regarding how your treatment plan may impact the bond between owner and pet. We must avoid facilitating an interruption in the human-animal bond between our patients and our clients and we should be vigilant that we are not inadvertently responsible for our clients feeling bad because of our therapeutic choices.

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The Pet Effect: Wonder and Why of Connection
Joe Hotzhauer, DVM
Zoetis
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For many years, there has been a strong notion among pet owners and non-pet owners alike that the mere act of having a pet influences one’s physical and mental health in a positive way. In fact, many have claimed that simply being in an animal’s presence can have a calming, ‘peace of mind’ inducing, and in some cases, even a curative effect on humans. (Halm, 2008) A recent example in the June 23, 2017 AVMA SmartBrief highlighted a Duke University study looking at using an 11 yr old golden retriever therapy dog named Aspen to calm pediatric patients undergoing echocardiograms in order to achieve better diagnostic images without the need for sedation. While not everyone is an “animal person,” many in society today appreciate these benefits of pet ownership as pet census numbers continue to rise, particularly among younger generations. A quick look at social media and we can see daily examples demonstrating the various ways simply being with these creatures help us, improve our outlook on life and how we are often happier because of them. In this country, where pet owners spent ~$350M in 2016 on pet Halloween costumes alone (APPA Survey), many of us truly love to spoil our pets—but why?

Before moving on to a discussion about the data available regarding the positive aspects of pet ownership, we must address the ‘why’. As scientists, we need to examine the putative physiologic mechanism(s) by which this might be accomplished. The intuitive feeling that many express (“I like having my pet near me and I feel better when they are”) is not evidence in itself.

Since the 1990’s, oxytocin has risen in prominence (and arguably, importance) from an occasionally mentioned subject in the medical literature to being crowned “the hormone of love.” While it’s possible that oxytocin may get more credit than it deserves for all things associated with love and bonding, there are increasing data to show that it may, in fact, be the underpinning of pair bonding and trust, even between species. Perhaps, even a significant mediator of our domestication of various animals—especially dogs.

In her book, “Made for Each Other”, Meg Daley Olmert makes a strong and supportable case for this. Olmert believes that in some way, wolves found their way into our lives and it was most likely the individuals on either side that were experiencing the highest levels of oxytocin (and possibly lower levels of vasopressin) that were the ones to touch a hand to a wet nose first. We now know that oxytocin is a strong source of bonding felt by new mammalian mothers both human and otherwise. In some cases, new mothers are not particular about the baby, whose it is, or even the species of the baby. As our human brains began to grow extensively, we began to create and draw, and most of what we drew were animals, our favorite subject.

Fast forward to today and we know that oxytocin is not produced in the hypothalamus, as was once thought, but is produced in multiple areas deep in the brain. These are the oldest areas of the brain. So old, in fact, that they lack the capacity for speech. These are areas driven exclusively by emotion. This is easily demonstrated by thinking of someone you love and trying to explain why. “There aren’t words,” is a common expression and given the discreteness of these brain functions...there often quite literally aren’t words to describe such an emotion.

The amygdala is one of the key structures in the brain that affects our stress axis, whether we bond or not, and is likely a key component to this question of mental and physical health for humans as it relates to animals. The amygdala is the source of stress, worry and mistrust. Oxytocin has been shown to calm the amygdala. It re-establishes what Olmert calls the ‘calm-connect’ system in the brain. As the levels of oxytocin are increased (through touching, petting, hugging, etc), measurable differences can be detected in heart rate, respiratory rate and blood pressure, all decreasing to a level more consistent with calm. All these effects are very beneficial to a new mother, but have even farther reaching implications in the discussion of how animals can help make us healthier and maybe even helping to keep them healthier.

A study conducted in South Africa (Odendaal, 2003) set out to see what effect and measurements they could assess with regards to oxytocin in people who played with dogs they knew. The study consisted of separating dogs from their companion humans before taking baseline oxytocin levels. Subsequently, each pair was allowed to reunite and play with each other as they typically do. When oxytocin levels were measured again after 20 minutes of play, both the human’s and the dog’s oxytocin levels had doubled compared to baseline. Using this study and others like it, we have entered an era where researchers can truly begin to look at co-regulation between species as a byproduct of just being with each other, and from there begin to look at the science of the healthy connection.

As time has progressed, more and more research goes into evaluating this idea of a “joint health.” Our understanding of this co-regulation has brought the concept of One Health to the foreground—and that term is now frequently used by both physicians and veterinarians. We continue to understand more and more about specifics concerning our connections with other creatures and that those connections are far older than we ever thought.

Greg Berns at Emory University has been able to train dogs to lie perfectly still without sedation in fMRI scanners so that their brain activity can be assessed. In January 2015, he published a paper studying various scents that light up dog’s pleasure centers...
(specifically, their caudate nuclei). He looked at both human and canine scents, as well as familiar and unfamiliar scents. This broke down into 4 scents, the familiar human scent being that of the dog’s companion human (Berns, 2015).

In all 12 dogs, the caudate nucleus only activated with the smell of the familiar human. This means that only that scent, as opposed to other conspecifics, activated the reward centers in those dogs. They actually feel some form of pleasure in the simple act of smelling their human’s scent. Often people lament that, as close to and important as their dog is to them, their dog will never know their human’s name. Is it possible to say now that not only do dogs know our ‘name’ in a way that even we cannot detect, they know our name given to us by nature, they know our name at a deeper level than even we may ever perceive…our “scent name!”

Clearly, there is a continuing need to evaluate and expand our knowledge of potential health benefits not only for humans, but for the animals involved as well. To begin to accomplish this, a commitment to producing high quality, well-funded, peer-reviewed publications on this subject is needed. One of the groups dedicated to this cause is the Human Animal Bond Research Initiative Foundation (HABRI). They are a non-profit organization, partnered with Purdue University, with the goal of being a central point of information for interested companies, organizations, and individuals with the common goal of advancing the human animal bond. HABRI’s mission is to: “Encourage informed decisions and actions that support the presence of pets and animals in society by advancing the science that demonstrates the positive roles they play in the integrated health of individuals, families and communities.” Zoetis is proud to be a founding sponsor and partner with HABRI.

They are part of many ongoing studies looking at various aspects of human and animal health including cardiovascular disease, depression, Autism, allergies and even cancer. Some examples of studies underway include: “Animal Assisted Social Skills Training for Children with Autism Spectrum Disorders”, “The Canines and Childhood Cancer Study: Examining Behaviors and Stress in Therapy Dogs” and “Animal-Assisted Intervention for Post-Traumatic Stress Disorder (PTSD).

As a final offering of data in support of potential real health benefits or pet ownership, a survey of 1,000 family physicians (the largest survey of its kind to date, not yet published) from August, 2014 is offered. Consisting of 28 questions, the survey conducted by HABRI in partnership with Cohen Research Group, asked physicians about their opinions on the subject of pets and overall health. The results were not difficult to interpret. 97% of them reported that they believe there are health benefits that result from pet ownership. 60% of them said that they have recommended getting a pet to a patient, 17% said that they made that recommendation for a specific condition. 75% said they had seen one or more of their patients’ overall health improve and 87% said their patients’ mood or outlook had improved. Most interestingly, 74% of the physicians said they would prescribe a pet to improve overall health if the medical evidence supported it, 8% said they would prescribe a specific pet for a specific condition. In a recent article in the New York Times, author Richard Schiffman writes:

“Some intriguing early research suggests links between the microbes that our animal companions bring into our homes — and that we breathe in and swallow — and the microbes that thrive in our digestive tract. “Exposure to animal bacteria may trigger bacteria in our gut to change how they metabolize the neurotransmitters that have an impact on mood and other mental functions,” Dr. Gilbert said, although he cautioned that research into how pet microbes affect the human gut microbiome remains at an early stage.

Netzin Steklis, a biologist at the University of Arizona who is working on a study of the elderly to learn more about how living with dogs changes their skin and gut microbiomes, says that pet owners have long known that animal companionship can lift our mood. “But it is not just an oxytocin story anymore,” she said, referring to the brain chemical often called the hormone of love. She suspects that the physiological effect of their bacteria in our guts may contribute to the well-known antidepressive benefit of pet ownership.

“Dogs have been with humans for 40,000 years,” she said. “But we are only now looking to find out how living with them impacts our health. We’ll know more soon.” (Schiffman 2017)

Though pet ownership (the bulk of the data supporting cat and dog, and likely horse ownership) certainly seems to help many humans in both mental and physical health realms, there are people that may remain unaffected or arguably worsened by pet ownership for reasons discussed in other sources. Even though we are only beginning to understand some of the basics of the neuro-chemistry of social bonding, just as with synthetically produced therapeutic molecules, not everything works for everyone so we need to approach the topic from a realistic, but also an enthusiastic point of view. The large amount of interest, research and funding that continue to support this topic is exciting and should be encouraged.

There is a tremendous potential everyday for us to embrace and enhance the pet-owner and pet bond. And we know that according to an AAHA study, irrespective of geography or socioeconomics, a key driver of practice success and growth is when that practice makes the human-animal bond its “keystone bond” of focus. Do you ever think about what your treatment plan for a pet will potentially do to the bond at home? How can we, as veterinary staff, find ways in our waiting areas, our exam room procedures, our communication and discharge instructions and even our product choices to enhance the pet owner-pet bond? Have you considered if it will enhance or potentially harm the bond and do I have another option? We will likely continue to discover fascinating things about our connections to all living creatures. We need also to be ready to accept that much of what we learn may in fact be a form of re-learning. We may re-learn things that we as a species knew thousands of years ago and have long forgotten. In the meantime, love your pet and don’t be afraid of that slobbery kiss from your dog; it may just do you some good in more ways than one!
*I would like to thank Dr. Patrick Flynn and Dr. Josh Schulz for their significant contribution to these lecture notes.

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Cervical hyperesthesia (painful response to a noxious stimulus) is a common presentation in clinical practice. The causes are variable ranging from benign to sinister. More often, than not, these cases have a positive outcome if the underlying etiology can be identified and addressed.

First, it is important to recognize the cervical pain is not spinal cord pain. The neuroparenchyma itself has few nocioceptors. Pain originates from the structures outside/adjacent to the spinal cord including the annulus fibrosis of the intervertebral disc, the meninges, the periosteum, the nerve roots (radicular), the muscle and the joints. Recognizing where the source of the hyperesthesia emanates from are important for both pathologic understanding and choice of therapeutics.

Dogs with cervical hyperesthesia may have other neurologic deficits but often do not. This is in contrast to thoracolumbar disease which frequently co-presents with proprioceptive or motor abnormalities. The predominant thought is that it relates to the larger canal/cord ratio in the neck than the back. It also speaks to why cervical disease can be much more severe and refractory to medical management when pain is the sole clinical sign.

Identifying cervical hyperesthesia is often as simple as noting a low head carriage at the walk. These dogs often have muscle fasciculations in the neck and shoulders. In more chronic cases, neurogenic atrophy can be noted in the supraspinatous muscles. Lateral motion of the neck may elicit a pain response but in the author’s experience ventroflexion and lateral palpation are most effective. The dogs may be ataxic in all limbs and postural reaction deficits may also be noted. The thoracic limbs may be spastic/hypertonic with normal reflexes in C1-6 myelopathies that have upper motor neuron disease. The limbs are flaccid/hypotonic with poor reflexes and atrophy in C6-T2 myelopathies with lower motor neuron disease. Dogs with C6-T2 myelopathies may also have poor motor component to their cutaneous trunci and Horner’s syndromes.

Several etiologies implicated in cervical hyperesthesia are to follow. Prioritizing the differential diagnosis list is based on a combination of signalment, acuteness of onset, severity of hyperesthesia and progression. From a diagnostic standpoint, cervical radiographs are often non-specific but indicated. They are useful in ruling out traumatic disease or severe discospondylitis and osteolytic neoplastic disease.

**Intervertebral disc disease (IVDD)**
With a high prevalence in chondrodystrophic breeds, cervical IVDD is nearly as common as thoracolumbar disease. Although survey spinal radiography may reveal changes suggestive of IVDD, these changes are seen in predisposed breeds and are not indicative of causal pathology. Conservative therapy is a combination of supportive care, anti-inflammatories and analgesics. 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. Surgical therapy has a >90% success rate and is generally considered a rapid recovery with infrequent complications.

**Atlantoaxial subluxation (AAS)**
AAS is a relatively frequent cause of C1-6 myelopathies in toy breeds of dogs, although and can occur in any age or breed. In suspected cases, care should be taken with manipulation of the neck. 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.

Radiographs are often diagnostic for AAS and on lateral projection, an increase in the space between the dorsal arch of C1 and the spinous process of C2 (> 4 mm) is noted. That said, advanced imaging is often recommended prior to surgery because of the high co-presence of cranio cervical malformations such as caudal occipital malformation syndrome.

Treatment for the condition is most frequently surgical. Conservative management has a 50% success rate and the challenges of external coaptation near the mouth and around the neck. Surgery can be performed by either dorsal or ventral techniques and is most often associated with a positive outcome.

**Cervical spondylomyelopathy (CSM)–“wobblers disease”**
A bimodal disease that affects either young giant breed dogs or older large breed dogs. The giant breed dogs have an osseous stenosis that causes a dorsolateral compression. Large breed dogs, most specifically Dobermans have a ventral “Disc associated” compressive syndrome. Both of these conditions can be constitutively present or only compressive in dynamic positions (predominantly extension). Survey radiographs are less helpful in these conditions and advanced imaging is indicated. Conservative approaches have been described but this too is often considered a surgical disease with varying reported success rates.
Meningitis/Meningomyelitis
See lecture “CSF-Who, what, when…”

Neoplasia
Any neoplasm that compresses the pain sensitive structures can result in cervical hyperesthesia. There is a high predominance of intradural tumors in the cervical spine including meningiomas and malignant nerve sheath tumors. Soft tissue sarcomas can be causal as can round cell tumors that affect bone such as plasma cell tumors. The tumors can be acute or chronic on presentation based on the affect they have to the vertebral bones. Survey radiographs are considered specific but not sensitive in that over 60% of the bone must be demineralized before radiolucency is noted. Therapies are variable and some tumors may respond to chemotherapeutic (round cell tumors) or radiation therapy (meningioma) while other don’t respond to either ancillary therapy. Surgical therapy is valuable for decompressive and diagnostic purposes but is often not curative in isolation because of the difficulty in attaining complete excision.

Key drugs, dosages and indications
Pharmacologic treatment of the patient with neck pain
Adopted from J Rossmeisl DVM, MS DACVIM (SAIM, Neurology)

<table>
<thead>
<tr>
<th>Tier</th>
<th>Drug choices</th>
<th>Dose</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tier 1*</td>
<td>Carprofen</td>
<td>2.2 mg/kg PO q 12 hrs</td>
<td>PO, SC</td>
</tr>
<tr>
<td></td>
<td>Prednisone</td>
<td>0.5 mg/kg/day</td>
<td>PO</td>
</tr>
<tr>
<td></td>
<td>Acetaminophen/Codeine</td>
<td>Dose to codeine equivalent of 0.5–1 mg/kg q 6 hrs</td>
<td>PO</td>
</tr>
<tr>
<td>Tier 2</td>
<td>Tramadol</td>
<td>2–5 mg/kg PO q 8–12 hrs</td>
<td>PO</td>
</tr>
<tr>
<td></td>
<td>Methocarbamol</td>
<td>22–44 mg/kg PO q 8 hrs</td>
<td>PO</td>
</tr>
<tr>
<td></td>
<td>Diazepam</td>
<td>0.25–0.5 mg/kg PO q 8 hrs</td>
<td>PO, IV</td>
</tr>
<tr>
<td></td>
<td>Gabapentin</td>
<td>15 mg/kg q 8hrs</td>
<td>PO</td>
</tr>
<tr>
<td>Tier 3</td>
<td>Morphine</td>
<td>0.25–1.0 mg/kg q 4–6 hrs</td>
<td>IM, SC</td>
</tr>
<tr>
<td></td>
<td>Oxymorphone</td>
<td>0.1–0.2 mg/kg q 4–6 hrs</td>
<td>IM, IV, SC</td>
</tr>
<tr>
<td></td>
<td>Hydromorphone</td>
<td>0.1–0.4 mg/kg q 4–6 hrs</td>
<td>IM, IV, SC</td>
</tr>
</tbody>
</table>

* Drug choices in this tier should not be used in combination.
Cerebrospinal fluid is an ultrafiltrate of blood that bathes the neuroparenchyma. The CSF is created predominantly by the choroid plexi in the ventricles of the brain. The fluid circulates through the ventricular system and into the subarachnoid space around the spinal cord. The CSF is predominantly drained through the arachnoid villi. Because of its proximity to the neuroparenchyma and meninges, cytology can be revealing regarding infectious/inflammatory and neoplastic conditions of the central nervous system. CSF changes may also be seen when nerve roots are inflamed even if there is no direct CNS involvement, because the proximal nerve roots are encased by the meninges. The common clinical example of this is polyradiculoneuritis, which can be idiopathic or due to infectious agents such as Neospora or Toxoplasma.

In cases where meningoencephalitis or -myelitis is thought likely; CSF collection should be considered. CSF collection can be performed in isolation but often follows advanced imaging, specifically MRI. The reason it is often performed after imaging is related to its primary contraindication. Space occupying masses or other causes of elevated intracranial pressure can predispose to brain shift and herniation with CSF acquisition. Further, the process of collecting CSF itself can cause later imaging artifacts. Other contraindications include atlantoaxial subluxation or severe overlying pyoderma.

CSF collection is easier to perform at the cisterna magna and less likely to result in blood contamination. However, the risk of iatrogenic injury is higher. Lumbar CSF acquisition is more challenging and can be performed at the L6-7 space in dogs/ L7-S1 space in cats. There is consideration for increased safety of lumbar punctures in cases of high intracranial pressure as there is more compliance between the sight of puncture and elevated pressure.

With both techniques, CSF should be allowed to drop freely into the collection tubes. CSF needs to be analysed or preserved ideally within 1 hour of collection.

General CSF cytology includes total protein count, nucleated cell count and nucleated cell differential. The total red blood cell count is noted as is general fluid color and presence of foreign material. Other parameters occasionally measured include creatinine kinase and glucose.

CSF cytology is considered a sensitive but non-specific indicator of CNS disease. It is rare that CSF is pathognomonic for any singular condition (<2%). Specific etiologies that could be identified include neoplasms (lymphoma or choroid plexus tumors) or infectious disease (Cryptococcus). More often than not, cell differentials give non-definitive hints of the underlying etiology.

A neutrophilic pleocytosis (cell predominance) is most suggestive of bacterial meningitis or steroid responsive meningitis arteritis. Bacterial meningitis is often associated with pyrexia, vomiting, inappetance, severe hyperesthesia and poorly localizing neurologic signs. It is rare and difficult to diagnose as bacteria can contaminate cytology and yet are difficult to culture from the CSF. Ribosomal RNA PCR has been performed to identify bacterial subunits but is rarely performed. Treatment is with antimicrobials that cross the blood brain barrier. SRMA is a more commonly encountered disease most frequently found in Beagles, Boxers, Pointers, Bernese Mountain dogs and Nova Scotia Duck Tolling Retrievers. They frequently present for cervical hyperesthesia and although they may have pyrexia are more frequently systematically normal. As the name implies they are respond well with high doses of immunosuppressive steroids. In cats, a neutrophilic pleocytosis is frequently associate with feline infectious peritonitis.

A lymphocytic/plasmacytic pleocytosis is more suggestive of viral or the meningoencephalitides of unknown etiology (MUE). Distemper, rabies and other viral etiologies should always be considered, particularly in unvaccinated young animals. Serum titers are often coupled with CSF and are often paired with known viruses vaccinated for. Other non-viral infectious agents include rickettsial agents which are typically identified through serology. The MUE is a class of immune mediated diseases that includes the frequently seen granulomatous meningoencephalitis and the lesser seen necrotizing polioencephalitis and leukoencephalitis. The MUE are frequently encountered in small breed female dogs, though they are seen in a variety of breeds and both sexes. These conditions are more challenging to treat and often involve a combination of steroids in conjunction with other immunomodulators.

An eosinophilic pleocytosis is most suggestive of protozoal, fungal or immune mediated etiologies. Toxoplasmosis and Neosporosis are most easily identified through serologic testing. Fungal agents, with the exception of Cryptococcus which has sensitive and specific serologic testing, are often identified by urine antigen testing.

Albuminocytologic dissociation is a term characterizing an elevation in CSF protein with a normal cell count. A very non-specific finding, this is often seen in cases of Generalized Tremor Syndrome; White Shaker Dog syndrome; These dogs present with diffuse, fine, whole body tremor. Dogs with white hair coats (Maltese terrier, West Highland White terrier) are more commonly affected, however, dogs with other coat colors are also affected. Additional neurological abnormalities include nystagmus, menace response abnormalities, proprioceptive deficits, and seizures. No underlying etiologic cause has been identified but these dogs are also responsive to steroid/immunomodulatory therapy.
Dizzy Now?
Vestibular Syndromes
Fred Wininger, VMD, MS, DACVIM
Veterinary Specialty Services
Manchester, MO

Function of the vestibular system
The vestibular apparatus is a sensory system essential for 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 CN VII 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 though the foramen magnum can compress these cell bodies. There is a direct connection between these nuclei and the “vestibulocerebellum” made up of the flocculonodular 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 bilaterally 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 CN VII 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 CN VII) 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.
### Clinical Sign

<table>
<thead>
<tr>
<th></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>
</tr>
</tbody>
</table>

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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.
Imaging the nervous system is a difficult task because of its residence in the hard bone of the skull and vertebral column, as well as its opaque parenchyma and minimal variation in tissue density. The introduction of computerized tomography (CT) and then magnetic resonance imaging (MRI) revolutionized veterinary neurology because of the ability to detect structural disease despite these obstacles. Cross sectional imaging has the advantage of improved resolution over 2-dimensional imaging by eliminating summation and permitting partial thickness intra-cranial reconstruction. Further, techniques that utilize protons rather than density take advantage of the more robust chemical differences present in neuroparenchyma. CT first and now MRI are the gold standard in structural imaging of the brain and spinal cord. That said, each technique has indications for different presentations and choosing the correct modality can mean the difference in arriving at a successful diagnosis.

Although cross sectional imaging has changed veterinary neurology for the better with improved diagnostic power, it is important to remember structural assessments of the nervous system cannot replace the functional evaluation. The primary functional assessment of the nervous system is a thorough physical and neurologic examination. Other functional assessments include electrophysiological techniques (EMG, EEG, ERG, BAER) and newer hemodynamic techniques (fMRI and Infrared spectroscopy). Often structural disease is not accompanied with functional disorders (ie. Chiari-like malformations or hydrocephalus). The inverse is also true in that functional disease may not have a structural correlate (idiopathic epilepsy). For this reason these functional and structural assessments complement each other but cannot be evaluated in isolation.

The term tomogram refers to any cross sectional modality. These techniques include CT and MRI, but also ultrasound and advanced metabolic imaging techniques such as SPECT or ultrasound.

**CT principles**

CT uses the simple techniques of 2D radiography and through complex computations is able to reconstruct these images in cross sectional and 3D studies. Images are acquired by rapid rotation of an x-ray tube 360 degree around a patient. The transmitted radiation is then measured by a ring of radiation sensitive detectors on the opposite side of the subject. An image is generated from these measurements on the premise that the density of the structures within the cylinder can be calculated when evaluated in combination. Complex mathematical algorithms allow each voxel to be assigned a numerical value based on the level of attenuation occurring within that volume of tissue. These units are known as Hounsfield units (HU).

Hounsfield units range from -1000 for air and +3095 for the densest object than can be imaged by CT (compact bone). Soft tissues of clinical interest generally range from -100 to +100. In neuroparenchyma, the grey matter is +35HU and the white matter +45HU, explaining why the technique has difficulty discerning the contrast of these structures. However, adjacent structures such as the ventricles can be easily imaged (HU=0).

The human eye can perceive 32 shades of grey. Therefore, for highest contrast resolution the goal is to maximize those shades to evaluate the tissue of interest. Window level defines the middle of the tissue of interest. Window width describes the range of HU displayed. The larger the window, the more HU represented by each shade of grey, the lesser the contrast. Soft tissue windows are smaller because the range of densities is lower when compared to bone. In other words, a shade of grey in a soft tissue window may represent 3 different HU, while in bone a shade may represent 100HU or more.

Contrast resolution is defined as the ability to display an image of an object that differs only slightly in attenuation compared to its surrounds. CT has 10x better contrast resolution over radiographs. However, the contrast resolution of CT for discerning neuroanatomical structures from each other and disease is poor.

**MRI principles**

MRI is different from other imaging modalities in that it utilizes protons (predominantly water content) to create images rather than density. This is particularly of value in the brain and spinal cord, where density differences are minor compared to differences in water content.

At rest in the normal environment, the protons of the body are moving towards entropy in a random arrangement. MRIs created large magnetic fields, which cause these protons to align along an axis. Though aligned, these protons are still rotating around their aligned axis, a process called precession. The speed at which they precess is the same for all protons of the same valence as defined by the larmor equation. Though precessing at the same speed, the protons are all out of phase with each other. When a radiofrequency wave is applied to the protons, they are jarred out of alignment together and all develop the same phase. With time, they return to their resting state in the magnetic field. The speed at which this return to rest is dependent on the chemical characteristics in which the proton resides and can be measured.

- The speed at which the proton returns to its resting axis is known as either longitudinal, spin-latice or T1 relaxation.
The speed at which the proton comes out of phase with the adjacent protons around it is known as transverse, spin-spin or T2 relaxation.

The ability to weight the images on these and other proton characteristics allows for interrogation of the nervous system, to make the tissue or lesion of interest more conspicuous.

**CT indications compared to MRI**

Although MRI has become the gold standard in neuro-imaging, there are several scenarios in which CT is indicated and perhaps preferable. CT has greater spatial resolving abilities when imaging bone. Because of this and its ability to create very thin slices, fractures can be easily identified. 3D reconstructions are superior with CT and allow for more global imaging of the bone as well as aid in surgical planning. CT is also excellent at imaging hemorrhage, which will appear as hyper-attenuating 3 hours after a bleeding event and remain so for 3 weeks as the HU is directly proportional to the PCV of the bleed. CT is inherently less expensive to purchase and maintain which translates to less expense and greater availability to the client. Although MRI has become quite fast, acquisition times are shorter with CT which is essential for critical patients or those that can not tolerate anesthesia. CT is less susceptible to metal artifacts compares to MRI, which can be important in projectile/shrapnel wounds.

CT has some inherent deficiencies aside from its poor contrast resolution. CT requires relatively large doses of ionizing radiation; Not frequently a concern in veterinary medicine. CT is poor at imaging the caudal aspect of the skull/brainstem because of beam hardening artifacts. In this case, the low energy photons do not pass through the petrous temporal bone proportionally to the high-energy photons and the computer gets conflicting information casting dark streaks across the brainstem.

**Low field vs. high field MRI**

Magnets what produce a field 1T or greater are generally considered high field, with low fields being anywhere between 0.2-0.5T. High field MRI have a superconducting magnet, which requires a cryogen to keep the system cooled and maintain low electrical resistance in the coil. These magnets requires special housing systems both to eliminate radio interference but also for protection from their attractive properties. This size restriction and cost of maintenance limits their availability. Low field MRIs are less costly and cheaper to maintain.

The main advantage to High field MRIs over their low field counterparts is the time required for image acquisition. The high magnetic field promotes faster proton relaxation and therefore multiple signals and averages are acquired in minutes. High field MRI images have more information and higher resolution with smaller voxels. Theoretically, low field MRI could create similar quality diagnostic images, but with acquisition times nearing hours to days! There have been few studies in veterinary medicine comparing these different fields, but the human field literature demonstrated the poor sensitivity and specificity of low field MR over two decades ago and it is rarely used for neural imaging. However, low field imaging maintains a role in extremity imaging for humans, as it does not require a closed bore system. (An advantage for the claustrophobic patient). This advantage does not translate to the anesthetized veterinary patient.

CT and MRI have their own inherent advantages and struggles. Our experience is that they often work best in tandem or when one modality fails. Future imaging techniques with MRI and PET are embracing the idea of combined structural and functional imaging.
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>
</tr>
</tbody>
</table>

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-Characterised 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.

Neurolocalization basics
Animals with neurological disease often present for problems walking, moving, and standing. This discussion will focus upon the clinical diagnosis of these abnormalities in dogs and cats.

Neuroanatomy of gait
Gait is commonly defined as a regularly repeating series of leg movements during walking or running. The nervous system controls the actions of the muscles, bones, joints and associated connective tissue important for walking. Normal walking is produced by the recruitment of stepping reflexes, which alternate between the extensor and flexor muscles. The vestibulospinal and reticulospinal tracts are facilitatory to the extensor muscles, important for maintenance of the body tone against gravity (the stance or propulsive phase). The corticospinal and the rubrospinal tracts are facilitatory to the flexor muscles, important for the protraction (flight) phase of limb movement.

Locomotion is thought to be controlled at the level of the brain stem; however, a discrete anatomic gait center (nucleus) has not been identified. Supratentorial (forebrain) structures are important for voluntary initiation of movement. The cerebellum, while not necessary for the initiation of movement, is important for coordination of movement. Cerebellar influences coordinate and smooth body movements by controlling rate, range, and force of limb motion. The cerebellum helps to "smooth" movements.

Clinical evaluation of abnormalities of gait
Clinical evaluation of gait usually involves observation of the animal's movements during walking, and, when indicated, running. This is best accomplished by having a handler walk the animal over a flat, non-slippery area (such as concrete or carpet). An overall assessment is made of how the animal moves and clues are obtained to abnormalities present.

General abnormalities of gait
Ataxia
Ataxia literally means lack of an axis, and is sometimes described as incoordination. Ataxia can result from a variety of anatomic lesions within the nervous system, most commonly of the cerebellum, vestibular system and spinal cord sensory pathways. Ataxia, without motor involvement (paresis), usually implies cerebellar or cerebellar pathway disease. Sensory ataxia due to loss of joint position sense is often made worse by blindfolding the animal, effectively preventing visual compensatory mechanisms.

Dysmetria
Dysmetria is improper estimation of distance during muscular activity. Dysmetria includes both hypo- and hypermetria. With hypermetria, voluntary muscular movement overreaches the intended goal; with hypometria, voluntary movement falls short of the intended goal. Hypermetria is more commonly recognized than hypometria. Both of these abnormalities are most often associated with lesions of the cerebellum or cerebellar pathways. In the instance of hypermetria, for example, the loss of cerebellar input, which normally stops the flexion phase of gait, results in the exaggerated movement.

Spasticity
Spasticity is a state of increased muscle tone and commonly results from upper motor neuron (UMN) lesions. Spasticity is observed in the gait as a lack of normal flexion or floating (failure to adequately flex the limbs during gait).

Stiffness
Stiffness associated with decreased step length is commonly seen with diseases of the peripheral neuromuscular apparatus [lower motor neuron (LMN) cell body, nerve roots, peripheral nerve, neuromuscular junction and muscle]. Dogs with neuromuscular disease may also have a stiff, stilted, choppy gait due primarily to muscle weakness. These abnormalities may be episodic and occur, as in the case of myasthenia gravis, as the level of exercise increases. A similar appearance may occur in dogs with pain, primarily from musculoskeletal disease.
Paresis
Paresis is derived from the Greek word for relaxation and suggests neurological weakness without complete paralysis (implies that some voluntary motion remains). Varying degrees of paresis can occur with some animals retaining the ability to walk while others are unable to support their own weight and stand. Paresis may be observed at gait as dragging of the toes or feet. Abnormal toenail wear may suggest underlying paresis.

Paresis at gait first occurs with lesions in the midbrain caudal to the level of the red nucleus. The severity of the gait impairment increases as the lesion occurs progressively more caudally in the central nervous system. For example, supratentorial lesions may result in significant hemiparesis when postural reactions are tested; however, gait remains relatively normal. With brain stem or spinal cord lesions, the associated paresis usually results in obvious gait impairment.

Lameness
Lameness [decreased or non-weight bearing on a limb(s)] is usually associated with pain of the limb from musculoskeletal disease. A similar clinical abnormality can also occur with nervous system dysfunction (and presumably pain), referred to as nerve root signature. This abnormality often occurs in a single thoracic limb due to cervical spinal compressive disorders (intervertebral disk extrusion). The same phenomenon may affect a pelvic limb. Often, the affected limb may appear painful upon manipulation, mimicking an orthopedic problem.

Clinical assessment
Important clues as to the cause of the gait abnormality can be obtained by assessing the step distance (i.e., the distance between where both thoracic or both pelvic limbs are placed in relation to each other). This determination is best made when viewing the animal from a lateral direction and when the animal is perpendicular to the examiner's line of sight.

Generally, animals with UMN neurological disease have normal or increased step lengths, while dogs with orthopedic or neuromuscular disease have shortened step lengths. Animals with UMN disease may appear irregular and uncoordinated, having a tendency to sway from side to side; some may fall to the side. Their feet may contact the ground with increased force. Doberman pinchers with cervical vertebral malformation - malarticulation, for example, may overflex the hock joints during weight-bearing, presumably the result of motor or sensory weakness.

Dogs with LMN disease may hold the head low (possibly because of neck muscle weakness) and take short ("choppy") steps. Some have a kyphotic posture. They may tire easily and appear unwilling to perform. Animals with orthopedic disease may have a similar gait abnormality wherein the limb step length is short. Normal conscious proprioception in the presence of a shorten step distance should suggest underlying orthopedic disease or neuromuscular weakness. Poor limb perfusion, such as from a partial bilateral iliac arterial thrombus or from a right-to-left PDA may also result in a short, choppy pelvic limb gait. The clinical signs may worsen with exercise (similar to myasthenia gravis) and differential cyanosis may be seen with the latter disease. Dogs with vertebral pain may also be short-strided and reluctant to move.

Depending upon where within the nervous system the lesion occurs, gait may be altered differently. Supratentorial disease will often not affect gait. Wide circling toward the side of a unilateral forebrain lesion is common. Occasionally, spasticity may be noticed, with the limb (primarily a thoracic limb) appearing stiff, floating and over-reaching. Also, apparent hypermetria may be found, usually in the thoracic limb opposite a supratentorial lesion.

Lesions of the brain stem and cervical spinal cord will often have dramatic effects on gait, frequently impairing the ability to stand and generate a gait. If the lesion occurs unilaterally, ipsilateral hemiparesis is noted. If pathways are affected bilaterally, tetraparesis will be present. Spinal reflexes should reflect the UMN nature of the lesion.

Neurologic abnormalities resulting in abnormal gait

Intracranial disease
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Spinal cord abnormalities
Animals are commonly presented for problems involving the spine and spinal cord. Often, these clinical signs are reflected as problems with walking or moving (limb motion). If there is abnormal function of a limb, the first determination made is whether there is, in fact, a neurologic problem. If a neurologic problem is encountered, then a determination of the location of lesion or lesions is made. Once the location of the lesion is determined, an appropriate differential diagnosis and diagnostic plan can be formulated.

The spinal cord can be functionally divided based upon the spinal segments and associated clinical signs of disease affecting these segments. The following is a summary of the clinical signs associated with lesions of selected spinal segments.
The functional division of the spinal cord is primarily dependent on the presence of either upper motor neuron (UMN) or lower motor neuron (LMN) clinical signs to the limbs. The presence or absence of these characteristic signs is based upon the concept of local reflex (does not require conscious control) function and the normal control of these reflex functions from higher nervous system centers.

**Lower motor neuron disease**

Technically, the lower motor neuron includes the motor neuron cell body, the motor (efferent) peripheral nerve, the neuromuscular junction, and the muscle.

**References**

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**
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**
  - neuoproective 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\(_A\) receptors
  - Possible NMDA antagnosit 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

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**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
- **Side Effects**
- **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.

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**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
- **Metabolism/Excretion**
  - **Dose**
    - 2-4mg/kg PO BID

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**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
Selecting diets for healthy pets and pets with medical conditions will be discussed by a board-certified veterinary nutritionist. Strategies for determining when a therapeutic food is indicated will be discussed. Strategies to increase client adherence to nutritional plans will also be discussed. Case examples will be included to illustrate tips and strategies.

What is the difference between a therapeutic diet and an over the counter (OTC) diet?
Beyond requiring approval from a veterinarian, veterinary therapeutic diets are formulated to help manage one or more disease processes in cats and dogs. Because of this special formulation, not all veterinary therapeutic diets will be complete and balanced (i.e., meeting the minimum and maximum nutrient levels put forth by the American Association of Feed Control Officials [AAFCO]). Some medical conditions alter the nutrient requirements of pets such that they have different needs from healthy pets. Depending on the nutritional goals, modifications to diets may be necessary that are outside the AAFCO guidelines, and thus these diets will have nutritional adequacy statements such as ‘for intermittent or supplemental use only’ or ‘for use under supervision by a veterinarian.’ In some diseases, nutritional management with veterinary therapeutic foods is critical due to these altered nutritional requirements and providing the level of nutrients appropriate for a healthy pet would be detrimental to their medical condition. Communication to owners about the difference between veterinary therapeutic and OTC diets can promote adherence, especially in cases where the diet must be fed exclusively for benefit.

Situations where veterinary therapeutic diets are indicated
AAFCO guidelines
Veterinary therapeutic diets may be most useful in instances where disease conditions require nutrient modification outside AAFCO guidelines and peer-reviewed studies have shown a benefit with specific diet modification. For example, renal disease is one instance where almost every pet requires phosphorus restriction below AAFCO guidelines as their disease progresses. Pets with severe to moderate renal disease (i.e., International Renal Interest Society Stage 3 or 4 chronic kidney disease) often require protein restriction below AAFCO guidelines as well. Other example disease conditions (and the nutrients of concern) that often require modification beyond AAFCO guidelines include: protein-losing-nephropathy (protein), hyperthyroidism (iodine), copper-associated hepatopathy (copper), and urate urolithiasis (purines).

Processing and formulation
In other instances, special processing of veterinary therapeutic diets may be required that is not available in OTC foods. For example, when conducting a dietary food allergy trial, ensuring quality control in protein sources is critical to successful diagnosis and treatment. Studies have shown OTC limited ingredient foods can be contaminated with other protein sources and not appropriate to use for dietary trials. Veterinary therapeutic diets containing hydrolyzed proteins or those intended for use as novel protein diets undergo extensive testing and strict processing methods. Though many OTC foods may be marketed as limited antigen, they are not always formulated to be used as a diagnostic tool.

Situations where veterinary therapeutic diets may be indicated
Other diseases require case by case assessment as to whether a pet’s condition requires modification of a diet that would be available in OTC foods (e.g., obesity, gastrointestinal disease, cardiac disease, etc.). In the case of urinary conditions, peer-reviewed studies have shown benefit of a veterinary therapeutic diets for specific urolithiasis management (e.g., struvite dissolution). While some OTC diets may also have properties that reduce the risk of various urolith formation, if a diet has not been tested for its ability to alter the environment of the urine in a clinical or research setting, the efficacy is simply not known. Furthermore, not all urinary conditions can be treated similarly. For example, in Feline Lower Urinary Tract Disease (FLUTD) or Feline Idiopathic Cystitis (FIC), studies have shown stress management and moisture intake are critical to disease management, which does not require veterinary therapeutic foods.

Strategies for success while using therapeutic diets
Do: Include treats and medication administration
Almost all owners give treats and studies show up to 60% of owners provide medication with food items. While some veterinary therapeutic diets come in treat forms, this is not always possible. Without proper client communication, owners may also have special treats they are convinced won’t affect treatment. Adherence can be increased by including treat and medication administration options that also meet nutritional goals (e.g., an alternative option to high sodium peanut butter, cheese or deli meat for administering pills is banana slices).
**Don’t: Interchange flavors and formulations**

Canned and dry versions of the same veterinary therapeutic foods will not always have similar nutrient profiles. For example, while a dry version of gastrointestinal diet may be quite low in fat, the canned version of the same food may be moderate or high in fat, which would be a significant problem for a pet with pancreatitis. Nutrient levels for macronutrients like fat or protein and for micronutrients like sodium and phosphorus may be drastically different between flavors or canned/dry versions. Be mindful of nutrient profiles and alert owners that only the specific formulations and flavors recommended should be used without consulting with their veterinarian first.

**Additional clinical considerations**

**Growing pets**

Until skeletal maturity (at least 12 months of age), cats and dogs must be fed a food formulated to meet AAFCO guidelines for growth or undergo AAFCO feeding trials for growth. Some veterinary therapeutic foods have been modified outside AAFCO guidelines, but have undergone feeding trials to ensure they are still suitable for growing animals. In disease conditions where nutritional management contradicts the needs of growing animals (i.e., protein losing nephropathy in a 7 month old puppy), a board-certified veterinary nutritionist should be consulted to properly formulate a nutritional plan (www.ACVN.org).

**Multi-pet households**

Pet owners often ask if veterinary therapeutic foods can be fed to healthy pets, especially if there are multiple pets in the house. If the food meets AAFCO guidelines or has undergone AAFCO feeding trials, it is likely appropriate for a healthy pet. However, it should never be assumed all other pets in the house are healthy and each pet should have a nutritional assessment to ensure dietary recommendations are appropriate. In general, veterinary therapeutic diets formulated for dental disease, gastrointestinal disease, and heart disease are likely to meet AAFCO guidelines, but each pet should be individually assessed to ensure appropriate dietary recommendations.

**Refresher on AAFCO statements**

Every food (but not treats) should have an AAFCO statement that describes if the food is complete and balanced and what lifestage the food is appropriate for. These statements will tell you three things:

- **Is this food complete and balanced?**
  - If not, it will say (often in very small print) “this product is intended for intermittent or supplemental feeding only.” This means it does NOT have all the essential nutrients a healthy pet needs.
  - Veterinary therapeutic diets often have this statement due to their modifications for diseases.
- **How did the company determine the food was complete and balanced?**
  - Companies can either do non-invasive feeding trials or perform an analysis of their product to determine the food is complete and balanced.
  - Feeding trials will state “Animal feeding tests using AAFCO procedures substantiate that ___ food provides complete and balanced nutrition...”
  - Nutritional analysis only will state “___ food is formulated to meet AAFCO nutrient profiles...”
  - Feeding trials ensure pets have eaten this food and done well, but ideally, companies have tested their foods by both methods to ensure it is safe for pets.
- **What lifestage does this diet provide complete and balanced for?**
  - AAFCO provides nutrient profiles and feeding trial requirements for growth, reproduction, and adult maintenance. (Note: there are NO senior guidelines!)
  - Foods that say all life stages must meet minimum levels of both growth and adult.
  - Starting in 2017, look for additional notation on foods for growth that specify whether the food is appropriate for large breed dogs (those expected to be 70 lbs or larger at mature weight).

**Summary**

Some medical conditions can be nutritionally managed with diets that are found over-the-counter. However, many medical conditions require adjusting the nutrient levels in diets below what is recommended for healthy pets (i.e., meeting the Association of American Feed Control Officials (AAFCO) minimum and maximum nutrient guidelines to be complete and balanced food). Each pet should be assessed individually to determine their nutritional goals based on their disease condition and nutrient requirements.

**General pet nutrition resources**

- American College of Veterinary Nutrition (ACVN) Website: www.acvn.org
  - Resources for pet owners, veterinarians, and a listing of all board-certified veterinary nutritionists.
- World Small Animal Veterinary Association Nutrition Toolkit: www.wsava.org/nutrition-toolkit
  - Note that this site has resources for pet owners and for veterinarians on pet nutrition topics.
• Tufts Clinical Nutrition Service Petfoodology Website: www.petfoodology.org
  o University website created by board-certified veterinary nutritionists with frequently updated blogs on pet nutrition.
Core strategies for communication
There are four core strategies for communication that will be applied to discussing nutrition with owners: 1) open-ended questions, 2) reflective listening, 3) non-verbal communication, and 4) empathy (Shaw, 2006). While it may seem most effective to ask closed-ended questions to quickly obtain necessary information, open-ended questions can evoke the owner’s perspective as well as information that will help develop a nutritional plan. One study revealed veterinary clients were only able to speak for a median of 11 seconds before being interrupted, which could leave owners with a sense of mistrust in their healthcare team. After asking open-ended questions, summarizing responses with reflective listening can let owners know they are being listened to, but also can redirect owners who may be getting lost in details back to the concern at hand. While having a discussion with clients, particularly in a controversial and emotional topic such as nutrition, non-verbal communication is critical to be aware of. If the discussion is positive, but an individual is frowning and crossing his or her arms, then there are mixed messages that should be explored further to ensure clear communication. Lastly, empathy with a focus on education in lieu of judgement on any previous decisions a pet owner may have made can greatly build trust in the exam room. One study revealed genuine empathy, which included providing positive feedback and normalizing concerns, was expressed in only 7% of over 200 veterinary visits studied. Employing these four core strategies while discussing nutrition with clients can help owners feel valued and respected, which can lead to increased trust in the healthcare team and improved outcomes for patients. The following sections will describe how to apply these core strategies throughout the visit in an efficient manner.

Getting information: saving time on diet histories
Start by obtaining a complete diet history, where all food items fed (including treats and table food) should be included to allow for an accurate estimate of daily caloric intake or to possibly help guide further diagnostics and treatment. For example, a dietary history of chicken jerky treats may warrant further discussion about renal disease and any related clinical signs. Owners may not volunteer information if not directly asked, so it is very important to spend time accurately assessing the current diet, including supplements, treats, rawhides, dental chews, and foods used to administer medications, etc. Pet owners may also not realize that chews or bones are sources of calories, or that some human foods can be toxic to pets. Obtaining a full and accurate diet history can be time consuming, so it is helpful to have owners pre-fill out a diet history form at home (where they can easily access the names of their pet’s foods and treats) and bring it to the appointment, or even fill it out while waiting in the lobby. This allows the healthcare team to incorporate reflection by summarizing the history and they can ask any pertinent follow up questions. Examples of diet history forms that can be given to owners are in the World Small Animal Veterinary Association Nutrition Toolkit (available at: www.wsava.org/nutrition-toolkit). Applying the core strategies by asking open-ended and non-judgmental questions such as “What treats does your pet enjoy?” may yield a more positive and forthcoming answer than “Do you feed your pet treats?” Also during this information gathering session, the healthcare team can assess the stage of change an owner may be in, especially if nutritional changes will be required for the pet (e.g., Are they interested in making changes at all for an overweight pet? Have they altered their pet’s food to a recommended therapeutic renal diet but then stopped after having challenges?). Some owners who are not ready to make any nutritional changes for their pet may benefit from education only, while others who are ready or have already started making changes may benefit from specific guidance and troubleshooting when they experience setbacks.

Giving information: clear and specific guidance
After obtaining a diet history and assessing the client’s needs, clear and specific nutritional recommendations should be provided. One study showed that a little more than half of pet owners agreed that their veterinarian communicated in language they understood. Avoiding jargon and asking owners “How can we best communicate information?” can help to decide whether diagrams, written pamphlets, or demonstrations will be the most effective method of providing information. Employing non-verbal communication can help decipher if an owner does not understand or may need rephrasing of the information being provided. Avoid vague statements such as ‘Your cat could lose a few pounds,’ and replace them with clear and specific statements such as ‘Your cat is 4 pounds overweight, which predisposes her to conditions such as joint problems and diabetes. We can work together to adjust her diet to keep her healthy and happy for as long as possible.’ This describes the medical problem, the consequences, and sets up a team-based
approach to reach a common goal, keeping the cat healthy and happy. Feeding directions can have wide ranges and may not be applicable to each individual pet, so pet owners should be told how many calories their pet needs per day, given a specific diet recommendation, and prescribed a specific amount to meet those calorie needs, incorporating treats if requested. These calculations can be done quickly with the use of recent toolkits from nutritional guidelines.

Giving information: utilizing resources
The World Small Animal Veterinary Association Nutrition Toolkit has non-branded handouts on calorie charts for cats and dogs, body condition scoring charts, and educational handouts on how to select a pet food and how to evaluate nutrition information from the internet. Many owners find the depth and variety of information on the internet overwhelming and rarely know how to find trusted sources of credible and evidence-based nutritional information. Providing owners with vetted websites minimizes confusion and inadvertent non-adherence because of misinformation owners may have garnered from their own online searches. Hospitals can create their own frequently asked questions handout/website on ‘hot topic’ areas such as raw food, ingredient questions, calories, and treats. One example is the Tufts Clinical Nutrition Service Petfoodology website (www.petfoodology.org).

Developing a plan as a team
If a pet requires a change in their diet and owners are ready to initiate change, understanding the pet-owner relationship can help individualize the plan. Owners who are highly attached to their pets may be more apt to provide more time or financial resources to their care, however, that attachment may also result in reluctance to change food or treats that define that relationship with their pet. Asking about the relationship between the pet and all members of the household may elicit ‘non-negotiable’ aspects of the human-animal bond that can be included in the plan to increase adherence. Example questions such as ‘Is there anything you feel strongly about including in the plan?’ or ‘Could you describe your daily routine with your pet?’ can help veterinarians develop a plan both the owners and healthcare team feel comfortable with. Using a dialogue rather than a lecture format helps owners take an active role in their pet’s health. Continued use of reflective listening and empathy will help to create a treatment plan that further strengthens the owner’s commitment and facilitates adherence.

Ongoing support and follow up
All members of the veterinary healthcare team can help to create and reinforce a nutritional plan. For example, after a veterinarian and pet owner decide on a therapeutic diet, technicians can further discuss client expectations (e.g., increased stool production if a higher fiber diet), and front desk staff can set up delivery of the diet or follow up appointments to renew prescription refills. Active efforts to reach out to clients will reinforce recommendations and show support while strengthening the bond between clients and the veterinary healthcare team.

Summary
Understanding the impact of effective communication, communication strategies, and nutrition-specific applications can lead to improved client adherence and patient care.

References
General pet nutrition resources
American College of Veterinary Nutrition (ACVN) Website: www.acvn.org
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Parenteral or Enteral Feeding?
Nutrition for Hospitalized Patients
Deborah Linder, DVM, DACVN
Tufts Cummings School of Veterinary Medicine
North Grafton, MA

Nutritional assessment and strategies for feeding hospitalized pets will be discussed by a board-certified veterinary nutritionist. Strategies for selecting parenteral or enteral options for nutritional support will be discussed. Strategies for optimal support of critical care patients will also be discussed. Case examples will be included to illustrate tips and strategies.

Terms useful for nutritional assessment of pets
- Anorexia: Complete loss of appetite, where a pet is not eating or ingesting any calories.
- Hyporexia: Decreased appetite where a pet is eating, but not enough to meet their daily calorie requirements (at least resting energy requirements, RER).
- Dysrexia: Change in food preferences, where a pet is eating, but not appropriate food (e.g., an unbalanced diet or foods not appropriate for a specific medical condition).
- Body condition score: Assessment of a pet’s fat stores only. Ideal body condition is described as ribs that are palpable without excess fat covering (tip: ribs should be no more padded than the back of your hand).
- Muscle condition score: Assessment of a pet’s muscle condition by palpation of the spine, skull, scapulae, and wings of the idea.

*Note: Handouts and helpful non-branded charts of body condition scoring and muscle condition scoring can be found in the nutrition toolkit developed by the World Small Animal Veterinary Association (WSAVA), available at: http://www.wsava.org/nutrition-toolkit.

Why is nutritional support important?
During normal weight loss, the body of a healthy pet will adapt to calorie restriction and break down fat. However, when a pet has a medical illness, if calorie, and especially protein, needs are not met, the body will not adapt. Instead, pets will break down their own muscles to meet their nutrient needs and cause muscle wasting. Body condition scoring, muscle condition scoring, and assessing the risk for malnutrition is critical in pets not meeting their calorie or nutrient needs.

Who needs nutritional support?
Assessing risk of malnutrition can alert the healthcare team when to intervene for a pet and consider additional nutritional support:
- Low risk: Previously healthy pets with no conditions that would increase protein loss (e.g., protein-losing enteropathy), who have been hyporexic or anorexic for 3 days or less. Examples would be elective surgery or a trauma.
- Moderate risk: Non-debilitated pets with conditions that increase protein loss, who have been hyporexic or anorexic for 3-4 days. Examples would be septic patients or a foreign body removal that required intestinal resection.
- High risk: Debilitated pets with chronic conditions that have experienced muscle loss, weight loss, have higher than normal nutrient needs (e.g., puppies or kittens), or have been hyporexic or anorexic for 4 or more days.

Note: For pets that are hospitalized, always assess the duration of hyporexia or anorexia including time at home before hospitalization!

When should nutritional support be started?*
For patients with low-moderate risk of malnutrition, consider duration of hyporexia or anorexia:
- 1-2 days: monitor food intake and clinical condition daily.
- 3-4 days: develop a plan for nutritional support, consider placing a feeding tube if pet is undergoing anesthesia.
- 5 or more days: nutritional support is necessary, place a feeding tube or initiate parental nutrition.

For patients with moderate to high risk of malnutrition (or are already malnourished):
- Nutritional support should be initiated as soon as hemodynamically stable regardless of duration of hyporexia or anorexia.
- *For a helpful handout on when to intervene nutritionally with hospitalized patients, see the WSAVA nutrition toolkit available at: http://www.wsava.org/nutrition-toolkit.

How should nutritional support be delivered?
- Strategies and the level of invasiveness for nutritional intervention depends on risk of malnutrition for each pet.
Low risk of malnutrition
Coax feeding: Coax feeding should only be attempted in pets that are at low risk of malnutrition and the goal should be to minimize stress and make feeding an enjoyable experience. Providing pet owners with hospitalized pets a private and quiet room to feed their pet, especially cats, can minimize stress and encourage eating.

Consider diet history: A full dietary history is crucial to knowing food preferences for each pet (e.g., dry, wet, flavor, texture, etc.) and offering them foods that are familiar to them. There are many foods available now in stew forms, pate, loaf, shredded, chunks, with dried chicken bits, and in various shapes like doughnuts, stars, pyramids, etc. Ask owners to keep a diary of their pet’s preferences, which can be useful to guide diet selection if needed.

Food aversion: Altering food temperature can also be helpful – for those with nausea, placing food in a refrigerator may reduce smells that induce nausea and make food more palatable. For pets with food aversions, using new dishes each time or disposable dishes can reduce the chance of them smelling traces of an old food that they are averse to.

Palatability enhancers: Palatability enhancers can be used with caution in pets, keeping in mind altered nutrient needs of pets with medical conditions and calories content to not unbalance the diet. Reserving 10% of the pet’s total calorie intake for treats or palatability enhancers can minimize risk of unbalancing the diet. Some popular palatability enhancers include shredded chicken breast (200 kcal/cup) for pets without protein restrictions, homemade chicken broth (store-bought is usually high in sodium and frequently contains onion or garlic), low fat and no salt added cottage cheese (200 kcal/cup), and honey or maple syrup (60 kcal/tablespoon), which is especially helpful for dogs with kidney disease or liver disease. Note: cats do not have taste receptors for ‘sweet’ foods and sugary items are not as effective as a palatability enhancer.

Moderate to high risk of malnutrition
Once a pet has become moderate or high risk (hyporexic or anorexic for 3 or more days) and previous strategies have been unsuccessful, a nutritional intervention plan should be developed to provide adequate nutrition. Assisted feeding can include a variety of short term or long term enteral or parenteral options. Enteral options (feeding tubes) are always preferred if tolerated as intestinal cells can atrophy without direct nutrition through ingested food. Parenteral nutrition is indicated if the gut is not functional (e.g., intestinal stasis, incontrollable vomiting). However, parental nutrition should only be initiated in settings with 24/7 supervision and taking into consideration the potential mechanical and metabolic complications of parental nutrition. The WSAVA Nutrition Toolkit not only has easy-to-use calorie charts, but also provides example feeding orders, monitoring templates, and a helpful flow chart for how to intervene nutritionally for each pet (available at: http://www.wsava.org/nutrition-toolkit).

How much should be fed?
The goal for all hospitalized pets should be to maintain current weight. Weight loss plans should never be initiated when pets are recovering from an illness and providing too many calories (i.e., using an illness factor to determine energy requirements) has been associated with adverse effects (such as metabolic complications) in human studies. Providing Resting Energy Requirements (RER) is a starting point that can be adjusted to ensure weight remains stable where RER = 70 (Body Weightkg)^0.75. Adjusted body weight should be used for pets with a body condition score greater than 7/9 on a 9 point scale. Methods for adjusting body weight can vary, but one method is to use ideal body weight and add on 25% of the excess body weight (e.g., a 20 kg dog with an ideal weight of 16 kg would have an adjusted body weight of 16 kg + 25% (20 kg – 16 kg) = 17 kg adjusted body weight).

What should I feed?
Selecting a diet for a hospitalized pet should be based on each individual pet’s nutrient needs, including energy requirements, lifestage (such as growth or gestation), nutrient intolerances from current conditions, palatability preferences (such as dry or canned foods), and logistical considerations (such as feeding tubes that require liquid or blenderized diets).

Summary
Assessing each pet for their risk of malnutrition will help guide the type and level of nutritional intervention needed to meet nutrient and calorie goals.

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The VOHC acceptance system for treats and foods will be discussed as well as the AAFCO guidelines on dental products by a board-certified veterinary nutritionist. Example cases in both dogs and cats will also be covered that include how to select a dental food or dental treats, especially when the pet has other nutritional needs (disease, age, etc.).

**Resources available to help consumers select dental products and foods**

Two organizations provide guidance for dental products and foods for cats and dogs. One has regulatory impact, AAFCO, and the other is a voluntary acceptance system, VOHC.

**AAFCO guidance for dental products and foods**

According to their website (www.aafco.org), AAFCO is a ‘voluntary membership association of local, state and federal agencies charged by law to regulate the sale and distribution of animal feeds and animal drug remedies.’ This is achieved by developing and implementing standards, guidelines, and definitions that aid in regulating the manufacturing, labeling, distribution and sale of animal feeds. AAFCO provides guidelines that states can then adopt into law as they see fit to regulate pet food in each state. It is important to note that AAFCO does not enforce laws and they are not regulatory themselves, but provide significant impact on regulations that states may adopt. AAFCO provides the nutrient profiles that determine if a pet food is complete and balanced for each lifestage (e.g., adult, growth), and also provide definitions for ingredients.

In general, any product that has a drug claim (e.g., ‘this product treats the following condition’) is required to have approval from the Food and Drug Administration (FDA). Within the realm of products marketed for dental health, AAFCO has made three provisions:

1. ‘Foods and treats implying drug claims or any mechanism other than mechanical must have FDA approval.’
   a. Note that if the mechanism is mechanical, it does not require FDA approval.
2. ‘Must be safe (GRAS).’
   a. GRAS is defined as ‘Generally Regarded as Safe’ by the FDA, where general recognition of safety is based on ‘common use in foods and a substantial history of consumption for food use by a significant number of consumers.’
3. ‘Foods and treats bearing claims to whiten or cleanse/freshen teeth by abrasive or mechanical actions are not objectionable.’
   a. This sentence has been taken to mean that these types of products are not AAFCO’s priority and they are unlikely to pursue possible false health claims.

In summary, AAFCO provides minimal guidance on how foods can be labeled and marketed for dental health, and it does not specifically address efficacy of products.

**VOHC guidance for dental products and foods**

VOHC was founded by a panel of veterinary dentists, dental scientists, and representatives from organizations such as the American Dental Association, the American Veterinary Medical Association, and the American Animal Hospital Association. According to their website (www.vohc.org), VOHC ‘recognizes products that meet pre-set standards of plaque and calculus (tartar) retardation in dogs and cats.’ Products that meet these standards are allowed to display a VOHC seal of acceptance following review of trials and data. It’s important to note that the VOHC does not conduct the testing of products, but supplies protocols for trials. Companies can then voluntarily conduct the trials and apply for the VOHC seal of acceptance.

VOHC seal of acceptance allows for plaque or tartar (calculus) reduction claims. In order to receive the seal, companies must produce data from two trials of healthy pets using the product in the way it is marketed to be used (e.g., daily, twice daily, etc.). There are strict protocols for control groups, scoring, randomization, and statistical analysis. Both trials must show that the dental product or food results in a 15% difference in mean mouth score (“mean of all tested teeth for all animals in the group”) between the treatment and control group. Safety is not specifically tested in the protocol, but VOHC seal of acceptance is contingent on annual confirmation that there is no information at the time that the product is unsafe. Unsafe is defined by the VOHC as “major extra-oral or body-wide issues such as toxicity, esophageal or gastro-intestinal obstruction or perforation, or gross nutritional imbalance; trauma to oral tissues, such as fracture of teeth or laceration or penetration of oral mucosa” (www.vohc.org).
Summary
AAFCO guidelines do not provide consumers much guidance in selecting dental health products. However, VOHC awards products a seal of acceptance that have proven minimal efficacy and no reports of unsafe side effects. There are a variety of foods, treats, and products that have been awarded the VOHC seal to date (see www.vohc.org for the most recent list). Studies have shown that brushing pet’s teeth is still the most effective method of preventing and reducing periodontal disease in cats and dogs, and dental products should be used in addition to this standard of care.

Dental health resources
Association of American Feed Control Officials (AAFCO) Website: www.aafco.org
Note that the full AAFCO guidelines are available online for a fee.
Food and Drug Administration (FDA) Website on GRAS information: www.fda.gov/Food/IngredientsPackagingLabeling/GRAS/
Veterinary Oral Health Council Website: www.vohc.org
American Veterinary Dental College Website: www.avdc.org
General pet nutrition resources
American College of Veterinary Nutrition (ACVN) Website: www.acvn.org
Resources for pet owners, veterinarians, and a listing of all board-certified veterinary nutritionists.
World Small Animal Veterinary Association Nutrition Toolkit: www.wsava.org/nutrition-toolkit
Note that this site has resources for pet owners and for veterinarians on pet nutrition topics.
Tufts Clinical Nutrition Service Petfoodology Website: www.petfoodology.org
University website created by board-certified veterinary nutritionists with frequently updated blogs on pet nutrition.
To Cook or Not to Cook?
When to Choose Home-Cooked Diets for Pets
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A review of the benefits, risks, and strategies for selecting home-cooked diets for pets will be discussed by a board-certified veterinary nutritionist. Strategies for assessing whether a diet is balanced will be discussed. Strategies for discussing home-cooking with pet owners and available resources will also be discussed. Case examples will be included to illustrate tips and strategies.

What is the evidence behind home-cooking for pets?
Cooking for pets can be very appealing to pet owners and there are anecdotal stories that tout benefits of home-cooked diets for cats and dogs. However, there is no evidence in the form of peer-reviewed clinical trials to support claims that home-prepared diets are healthier than commercial diets in general. Very few pets actually need to be fed a home-cooked diet because of health reasons (i.e., there is not a commercially available option that meets their needs) and an improperly prepared home-cooked diet can be quite harmful, especially for a growing kitten or puppy.

Indications for home-cooked diets
1. Medical necessity
Many medical conditions require adjusting the nutrient levels in diets outside of what is recommended for healthy pets (i.e., meeting the Association of American Feed Control Officials (AAFCO) minimum and maximum nutrient guidelines to be a complete and balanced food). There are a growing number of high quality veterinary therapeutic diets that have been formulated for various disease conditions. However, some pets may require a combination of nutrient modifications that are not available in commercial diets or in some cases, require restriction of nutrients below any commercially available diet (e.g., pets with severe protein-losing enteropathy may require fat restriction beyond the lowest fat veterinary therapeutic diet on the market). In these instances, formulating a customized home-cooked diet recipe is indicated for optimal management of the pet’s disease condition.

2. Pet preference
Even with palatability enhancers, some pets refuse to eat commercially available diets due to preference. This can sometimes happen when pets are given a large amount of table scraps and then slowly reject commercial diets especially if pet owners offer other human foods at the first sign of refusal. There are an increasing number of complete and balanced diets that have a consistency similar to human foods (i.e., stews, tubs, etc.) and these can be tried along with palatability enhancers such as adding home-made chicken broth (be careful with store bought broth as it often contains onion or garlic powder). Beyond pet preferences, some medical conditions or medications can cause altered appetite and food preferences. In these situations, home-cooked diets can be helpful due to their high palatability and ability to be customized to each pet’s preferred flavors or ingredients.

3. Owner preference
For healthy pets, and most pets with medical conditions, there is no medical necessity to provide a home-cooked diet. However, as long as the recipe is formulated by a board-certified veterinary nutritionist to be complete and balanced, home-cooking solely based on owner preference is a perfectly acceptable alternative to commercial diets. Before initiating a home-cooked diet, client communication about common myths or misconceptions of commercial pet food may alleviate some misinformed fears and ensure pet owners are making well-informed decisions for their pets.

Risks of home-cooked diets
1. Unbalanced recipes and diet drift
Home-cooked diet recipes on websites and in books (even those created by veterinarians) are often vague, out-of-date, or lack essential nutrients that are required for a diet to be complete and balanced for pets. Various studies have shown common recipes for healthy pets or pets with medical conditions have deficiencies that would be harmful. Many recipes are too vague (“use a vitamin supplement”) and lead to confusion and risk of missing essential nutrients in the right proportions. A general guide is if two people using the same recipe would not make identical diets every time, the recipe is not specific enough. Additionally, even recipes that have been formulated by board-certified veterinary nutritionists may be harmful if not followed exactly as directed. Owners sometimes make substitutions or changes to the recipe (i.e., ‘diet drift’) without consulting with their nutritionist and these changes can quickly unbalance the diet leading to excesses or deficiencies of essential nutrients.

2. Expense
In most cases, it is significantly more expensive to prepare a nutritionally complete diet at home than to purchase a good quality commercial diet, especially for a large dog. Additionally, most owners do not factor in the costs of proper supplements for home-cooked diets, which can cost up to several dollars per day, depending on the size of the pet, on top of the costs of other ingredients.
3. Quality control
Having a diet formulated by a board-certified veterinary nutritionist ensures that the recipe will meet AAFCO minimum and maximum nutrient guidelines to be a complete and balanced food (or otherwise meet a pet’s modified nutrient needs due to medical conditions). However, good quality commercial foods also undergo extensive quality control testing, digestibility trials, and feeding trials to assess for bioavailability and nutrient adequacy. Because home-cooked diets do not undergo this testing for safety and nutritional adequacy like most commercial diets, even healthy pets eating home-cooked diets should have more frequent veterinary visits and laboratory tests (blood work, urine testing) than similar pets eating commercial diets to ensure the diet is meeting their needs, which can also add to the expense of home-cooking.

Balancing home-cooked diet recipes
Due to quality control concerns as well as nutritional variability in many whole food ingredients, it is almost always recommended to use concentrated vitamin and mineral supplements in diets rather than attempt to meet all nutrient requirements using only whole foods. This approach makes the diets easier to prepare and ensures that pets receive adequate amounts of all essential nutrients. It is extremely challenging to create a recipe made of only whole foods that will consistently provide exact amounts of all essential nutrients. Additionally, a common misconception among owners is that they can ‘just add a multivitamin’ to a variety of human foods the pet is currently receiving to balance the diet. Protein sources vary in their nutrient profile and many of the common vitamin supplements on the market for humans or pets provide either too much or too little of the over 30 essential nutrients that pets require. Cooking for pets is a more precise science than the art of cooking for ourselves and should only be done with a specific recipe formulated by a board-certified veterinary nutritionist to ensure it is balanced.

Contraindications for home-cooked diets
Growth and gestation
Pregnancy, lactation, and growth are the most nutritionally demanding times in an animal’s life. Nutrient concentrations that meet the needs of adult animals at maintenance could cause serious harm to a pregnant or growing animal. In fact, severe health problems have been reported due to nutritionally unbalanced diets in growing puppies and kittens fed home-prepared diets. This is especially true for large and giant breed puppies, who have a more narrow range of acceptable dietary calcium and phosphorus concentrations. Because of the narrow margin of error and the potential risk of lifelong repercussions, home-cooked diets for animals that have not yet reached skeletal maturity (at least 12 months of age), that are pregnant, or that are lactating should only be recommended when strictly medically necessary. However, once a dog or cat is at least one year of age or has completed lactation, a nutritionally-balanced home-cooked diet is an acceptable alternative.

Obesity
Logistically, properly balanced home-cooked diets tend to be difficult to feed to weight loss patients due to their constantly changing energy and nutrient requirements during weight loss. Unlike with commercial diets, the amounts fed of home-cooked diets cannot be as easily adjusted without altering the nutrient profile due to the multiple ingredients and supplements required. Using a veterinary therapeutic diet that is formulated to be nutrient (but not calorie) dense for active weight loss is recommended. However, once a pet reaches ideal or goal body weight, a nutritionally-balanced home-cooked diet can be an acceptable alternative.

Summary
While there is no evidence that home-cooked diets are healthier than commercial diets, a properly prepared home-cooked diet that was formulated by a board-certified veterinary nutritionist can be an acceptable alternative or a part of disease management in some cases where it is medically necessary.

General pet nutrition resources
American College of Veterinary Nutrition (ACVN) Website: www.acvn.org
Resources for pet owners, veterinarians, and a listing of all board-certified veterinary nutritionists who consult with owners and can formulate home-cooked diets.
World Small Animal Veterinary Association Nutrition Toolkit: www.wsava.org/nutrition-toolkit
Note that this site has resources for pet owners and for veterinarians on pet nutrition topics.
Tufts Clinical Nutrition Service Petfoodology Website: www.petfoodology.org
University website created by board-certified veterinary nutritionists with frequently updated blogs on pet nutrition.
Traditional and non-traditional strategies for successful weight loss will be discussed by a board-certified veterinary nutritionist. This talk will cover weight loss basics, but will focus on troubleshooting difficult cases. Strategies to approach different types of pet owners will be discussed. Case examples will highlight strategies.

**Definition and diagnosis**

Obesity is one of the most common health problems affecting dogs and cats, with up to 59% of dogs and cats being overweight or obese. The most common and clinically applicable method of diagnosing obesity is a body condition scoring (BCS) system. It is important to remember that BCS only assesses body fat, while muscle condition scoring should be used to quantify muscle wasting (i.e., an obese pet, which is a BCS 9/9, could also have severe muscle wasting). Each BCS is generally defined as a 10-15% increase or decrease from ideal body weight. While definitions of obesity vary, a general consensus describes overweight as 10-20% above optimal body weight (BCS of 6-7) and obese as 20% or more above optimal body weight (BCS of 8-9). The 2014 American Animal Hospital Association Weight Management Guidelines for dogs and cats recommend that body weight, body condition scoring, and muscle condition scoring be documented in the record at every visit.

**Clinical consequences and considerations**

Obesity has been associated with numerous diseases, including osteoarthritis, dermatologic disease, diabetes, respiratory tract disease and shortened lifespan. Obesity and its effects should be taken into account in the general management of pets. Drug dosages and fluid requirements are a topic of interest in current research. Calculating requirements based on current obese body weight may increase risk of side effects or toxicity, while using optimal weight is more subjective and could potentially lead to decreased efficacy. Each pet and medication must be considered on an individual basis. Obesity in all species is more easily prevented than treated, and veterinarians play an important role in educating clients before the pet becomes obese. A discussion of body weight, body condition, and feeding amounts is an important part of the initial puppy and kitten visits. This should be reinforced at the time of spay/neuter when energy requirements are known to decrease by up to 30%.

**Traditional weight loss plans**

Start by obtaining a complete diet history, where all food items fed (including treats and table food) should be included to allow for an accurate estimate of daily caloric intake. Owners do not always volunteer information if not directly asked, so it is very important to spend time accurately assessing the current diet, including supplements, treats, rawhides, dental chews, etc. If current intake can be estimated, then calories should be restricted to 80% of current intake to encourage weight loss. If current intake cannot be determined, caloric restriction should start out initially at the calculated resting energy requirement (RER: 70 x Body Weight(kg)^0.75) for the estimated ideal weight and then be adjusted accordingly. (Note: there is a wide range of recommendations for initial energy restriction – most important is that pets receive follow up and the plan is adjusted as needed). The optimal nutrient profile for a weight loss diet should be based on the preferences and lifestyle of the owner and the pet, as well as the pet’s tolerance of the diet (some are intolerant to particular ingredients or to the high fiber content of some diets). Studies have shown diets with adequate protein may reduce risk of lean tissue loss and diets with increased nutrient density may reduce risk of nutrient deficiency. Research has revealed varying results on fiber content and moisture content (i.e., canned vs dry), suggesting these factors may be different for each patient. The 2014 American Animal Hospital Association Weight Management Guidelines provide guidance for minimum protein levels and other dietary factors. If owners are used to giving treats, adherence may be increased by allowing treats, but reserve no more than 10% of the total desired daily calories as treats. Exercise can be helpful in weight loss plans, but it should be noted exercise alone is highly unlikely to cause significant weight loss. Brisk walks over increasing distances and swimming can be great activities for dogs, while food dispensing toys, hiding kibble around a room, and toys or laser pointers can be great ways to stimulate activity in cats.

**Follow up**

An important aspect of weight management in dogs and cats is follow up. Food amounts should be adjusted 10-15% at each check in until a goal of 1% body weight loss per week is seen. Up to 2% of body weight loss per week has been shown to be safe, but higher rates of weight loss can result in rebound weight gain after reaching ideal weight, as well as increased loss of lean body mass. Exceptions to 1% weekly weight loss include dogs and cats with comorbidities where a more gradual rate of loss (0.5%) is more safe or realistic. Once ideal weight has been reached, the majority of cats and dogs will continue to need calorie restriction and a low caloric density diet.
Strategies for successful weight loss

While traditional diet and exercise plans can work well in some simple weight loss situations, many pet owners and veterinarians find weight loss a challenge and come to the conclusion that more intensive management and intervention is required. While some argue obesity should be considered a medical disease, others argue it is solely a psychological disease of the owner. The complex relationship between owners and their pets must be understood in order to achieve successful weight loss.

Readiness to change

It is helpful to consider the stage of change that an owner may be in (e.g., Are they interested in making changes at all? Have they made changes and stopped after having challenges?). One article details the theory of stages of change and how to apply this to veterinary weight loss cases (Churchill, 2010). To initiate a discussion with owners, open phrases can be useful such as “It sounds like you are concerned about your pet’s weight affecting his ability to jump and play like he used to”, or “I know I’d like a cookie over kibble, but pets can get sick like us if they ate only candy all day. Would you like to discuss a balanced diet plan with food and treats your pet likes?” Some owners who are not ready to initiate a weight loss plan may benefit from education only, while others who are ready or have already started making changes may benefit from specific guidance and troubleshooting when they experience setbacks.

The complex pet-owner relationship

If owners are ready to initiate a weight loss plan, understanding the pet-owner relationship can help individualize nutritional management. Owners who are highly attached to their pets may be more willing to provide time or financial resources to their care. However, that attachment may also result in a strong emotional bond in which they are reluctant to withhold food or treats which they feel define their relationship with their pet. Asking about the relationship between the pet and all members of the household may uncover ‘non-negotiable’ aspects of the human-animal bond that can be discussed and included in the plan to increase adherence. Example questions such as ‘Is there anything you feel strongly about including in the plan?’ or ‘Could you describe your daily routine with your pet?’ can help veterinarians develop a plan both the owners and healthcare team feel comfortable with.

Summary

Traditional caloric restriction and physical activity weight loss programs may not be appropriate or successful for all owners. Incorporating strategies to assess owners’ stage of change and including important aspects of the human-animal bond can individualize plans to each owner-pet relationship and increase adherence.

References


Pet obesity and general pet nutrition resources


Association for Pet Obesity Prevention Website: www.petobesityprevention.org

American College of Veterinary Nutrition (ACVN) Website: www.acvn.org

Resources for pet owners, veterinarians, and a listing of all board-certified veterinary nutritionists.

World Small Animal Veterinary Association Nutrition Toolkit: www.wsava.org/nutrition-toolkit

Note that this site has resources for pet owners and for veterinarians on pet nutrition topics.

Tufts Clinical Nutrition Service Petfoodology Website: www.petfoodology.org

University website created by board-certified veterinary nutritionists with frequently updated blogs on pet nutrition.
How to Diagnose and Treat Adrenal Tumors
Sandra Bechtel, DVM, DACVIM
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Often, adrenal tumors are found incidentally upon imaging of the abdomen for other medical reasons. Once an adrenal mass is discovered, a thorough understanding of adrenal physiology will aid the veterinarian in guiding clients through diagnostic and therapeutic planning. The adrenal gland is divided into the cortex and medulla. The adrenal cortex has three functionality distinct layers that release different hormones under the control of separate stimulatory mechanisms: the zona glomerulosa releases mineralocorticoids under the control of the renin-angiotensin-aldosterone system, serum potassium and ACTH; the zona fasciculata releases glucocorticoids under the control of ACTH; and the zona reticularis secretes sex hormones. The adrenal medulla acts as a modified post ganglionic sympathetic neuron and releases epinephrine (EPI) and norepinephrine (NE).

Adrenal masses may be benign or malignant, and functional or non-functional. Determining the functionality of an adrenal tumor is imperative to provide the most appropriate supportive care prior to definitive therapy. In canine necropsy studies, the most common adrenal masses were benign adrenocortical tumors, followed by - in decreasing frequency- adrenocortical carcinomas, adrenal medullary tumors (pheochromocytomas) and metastatic lesions to the adrenal gland. In cats, tumors metastatic to the adrenal glands is the most common adrenal tumor, although cats can be diagnosed with adrenocortical tumors and pheochromocytomas. The most common metastatic tumor to the adrenal glands in both dogs and cats is lymphoma.

Incidental adrenal tumors
As an adrenal mass discovered during abdominal imaging for other reasons can be disruptive to the diagnostic and therapeutic decision making process, outlining differential diagnoses and prioritizing diagnostic tests is important for patient care. The first step is to determine whether or not the mass is functional (an adrenal cortical mass producing mineralocorticoids or glucocorticoids, or an adrenal medullary mass producing EPI or NE). Therefore, blood pressure, fundic exam, and endocrine testing as outlined below to rule out functional tumors should be considered. Determination of tumor functionality will also help determine pre-operative supportive care. Staging for other tumor types is also indicated since metastatic lesions to adrenal glands are common, particularly in cats.

Therapy for incidentally found adrenal tumors is based on functionality, size, and evidence of invasion. If the mass is determined to be nonfunctional and is less than 2cm with no invasion, repeating an abdominal ultrasound regularly is recommended. Adrenalectomy should be considered for functional tumors, locally invasive tumors, or those larger than 2.5cm.

Tumors of the adrenal cortex
While hyperadrenocorticism (HAC) is a common endocrine disease in older dogs, it is most often pituitary dependent and the result of excess synthesis and secretion of ACTH, which results in bilateral adrenal cortical hyperplasia and resulting bilateral adrenal gland enlargement. Adrenal dependent HAC caused by a benign or malignant adrenal cortical tumor is relatively uncommon, representing only 15-20% of HAC cases. The result of a functional adrenal cortical tumor is usually excess glucocorticoid secretion leading to the typical HAC presentation. Dogs tend to be older with a median age of 11 years and clinical signs are consistent with HAC - polyuria, polydipsia, polyphagia, weakness, lethargy, dermatologic changes, abdominal enlargement, and panting. In addition to these clinical signs, dogs with HAC are prone to urinary tract infections, protein losing nephropathy, and a high percentage of dogs are reported to be hypertensive (complicating differentiation of cortical versus medullary adrenal tumors).

To determine if an adrenal mass originates in the cortex resulting in adrenal dependent hyperadrenocorticism (ADH), endocrine testing should first confirm the presence of HAC. A urine cortisol-to-creatinine ratio from urine collected in the home environment is a good screening tool as it is very sensitive - a normal result rules out HAC but a positive requires further confirmation. Other tests for confirmation include the ACTH stimulation test and/ or low-dose dexamethasone suppression test. Endocrine tests used to differentiate ADH from pituitary dependent HAC include the low dose dexamethasone suppression test (in some cases), high dose dexamethasone suppression test, and endogenous ACTH. 3

Typical findings of HAC on minimum database include neutropenia, monocytosis, lymphopenia, thrombocytosis, hypercholesterolemia and elevated alkaline phosphatase. Abdominal imaging in cases of pituitary HAC may reveal bilaterally enlarged adrenal glands, but in the case of ADH, abdominal ultrasound or CT may reveal a unilateral adrenal mass. An adrenal adenoma tends to be small -less than 2cm- and well encapsulated or partially encapsulated. In contrast, adrenal carcinomas tend to be greater than 2cm in diameter and may invade into the cava. Forty -50% of adrenal carcinomas metastasize and the liver and lungs are the most commonly reported sites.4 5

Once HAC is confirmed via endocrine testing and imaging, the treatment of choice for tumors of the adrenal cortex is adrenalectomy; this should be considered in all cases with functional tumors, locally invasive tumors, or those larger than 2.5cm. Surgery is diagnostic, as histopathology will confirm benign versus malignant and determine if follow-up monitoring is required, and
Therapeutic. Complications reported with surgery are cardiovascular events and hemorrhage. Potential post-operative abnormalities are adrenocortical insufficiency, pulmonary thromboembolism, pancreatitis, renal failure, and wound dehiscence. Perioperative mortality rates range from 9-60%. Post-operatively, ACTH stimulation testing within 24 to 48 hours should be considered as hypoadrenocorticism may result as the remaining adrenal gland may be atrophied, in addition to monitoring of glucose and electrolytes. Heparin and glucocorticoids may be indicated in addition to mineralocorticoid supplementation in some dogs.

The median survival time in dogs with adrenocortical carcinomas treated with surgery alone is 778 days; in dogs that survive the peri-and post-operative period and are discharged from the hospital, the median survival time is reported as 992 days in one study and 17.5 months in another. If surgery excision is not an option, medical therapy should be considered for those cases with clinical signs of HAC. Medical therapy for functional adrenal tumors include mitotane, although much higher doses are required than when used for pituitary dependent HAC and relapses are common (up to 60% of dogs); untoward side effects of mitotane occur in greater than 50% of dogs, not including dogs that develop mineralocorticoid deficiencies. The median survival time reported is 16.4 months. Trilostane can also be used for medical management of HAC, where the quality of life improves and clinical signs decrease however the size of the adrenal tumor will remain the same or increase.3,6 In dogs severely debilitated due to HAC medical therapy may be instituted as supportive care prior to consideration of surgery.

Conn’s syndrome (primary aldosteronism) is an aldosterone secreting adrenocortical tumor increasingly recognized in cats. Cats tend to be middle-aged or older. Both adenosomas and carcinomas can cause this syndrome and masses may be unilateral or bilateral. The most common clinical sign is muscle weakness resulting from hypokalemia; additional findings are arterial hypertension and, uncommonly, hypernatremia. A normal plasma aldosterone level in the face of hypokalemia is supportive of but not diagnostic for Conn’s syndrome and an oral fludrocortisone suppression test or aldosterone: renin ratio may be more useful for definitive diagnosis. Adrenalectomy is the treatment of choice with unilateral disease and outcomes reported are good, even if invasion into vena cava is present. Medical management with potassium supplementation, antihypertensive drugs, and spironolactone (an aldosterone antagonist) can be used for non-surgical patients.7-9

The adrenal cortex is also responsible for the secretion of sex hormones, and adrenal tumors with produce excessive sex hormones have been reported in the dog and cat with or without excessive glucocorticoid secretion. Tumors of the adrenal medulla

Pheochromocytomas are functional tumors of the adrenal medulla derived from chromaffin cells. Normal chromaffin cells are stimulated by sympathetic nerve fibers to secrete epinephrine (EPI) and norepinephrine (NE) into the blood stream. Pheochromocytomas are not innervated, so catecholamine release is not initiated by neural impulses and therefore their release is variable and unpredictable. Pheochromocytomas are uncommon in dogs (0.01-0.1% of all canine tumors) and rare in cats. In dogs, the median age of diagnosis is 11 years with no sex predilection. Most often, pheochromocytomas are unilateral, slow growing, vascular, and malignant. Vascular invasion is reported in up to 82% of cases; and metastasis to the liver, spleen, lung, regional lymph nodes, bone, and central nervous system is reported in up to 40% of cases. Clinical signs are often absent or vague and episodic in nature due to the intermittent release of catecholamines. The most common clinical signs associated with pheochromocytoma are weakness and collapse. Additional clinical signs are tachypnea, anxiety, restlessness, exercise intolerance, decreased appetite, weight loss, polyuria/ polydipsia, and seizures. Acute collapse may also occur due to tumor rupture and hemorrhage.10-11 Physical examination may be normal, or non-specific: tachypnea, panting, tachycardia, weakness, pallor, arrhythmias, and hypertension may be found. Hypertension is documented in 40-50% of dogs with pheochromocytoma and should be suspected with a systolic blood pressure of greater than 160mmHg; note that a normal blood pressure does not rule out a diagnosis of pheochromocytoma, as catecholamine release is episodic in nature. Ocular examination may reveal mydriasis, retinal hemorrhage, or, less commonly, retinal detachment. Neurologic examination may reveal spinal pain, rear limb ataxia, or head tilt.10-11

Thoracic radiographs may demonstrate cardiomegaly due to concentric hypertrophy, distension of the caudal vena cava from tumor thrombus, and evidence of pulmonary metastasis. Abdominal ultrasound detects an adrenal mass in most cases in addition to vascular invasion and tumor thrombus. Advanced imaging using CT or MRI provides more imaging detail in regards to the presence and location of the adrenal mass, the size, shape, and architecture of the adrenal glands, mass invasion, and metastasis to other abdominal organs.12 Plasma metanephrine has moderate sensitivity and excellent specificity, and normetanephrine has excellent sensitivity and specificity, in differentiating pheochromocytoma from dogs with adrenal cortical tumors. Urine normetanephrine: creatinine ratio may also be helpful in the diagnosis of pheochromocytoma. Of note, false positive results for both serum and urine metanephrine and normetanephrine measurements can be obtained if the dog is on phenoxymenzamine.12-13 Definitive diagnosis of pheochromocytoma requires histopathology and immunohistochemistry. Chromogranin A will differentiate between pheochromocytoma and adrenal cortical carcinoma, as chromogranin A is present the in secretory granules of endocrine cells. Pheochromocytomas will also stain with synaptophysin, a membrane component of synaptic vesicles in neurons and neuroendocrine cells.10-11

Adrenalectomy is the treatment of choice for pheochromocytoma in both the dog and cat. However, prior to consideration of surgery for a suspected pheochromocytoma, phenoxybenzamine, a non-selective alpha-1 and -2 receptor blocker, is recommended to
decrease blood pressure, decrease ventricular arrhythmias, and support blood volume expansion. Pre-treatment with phenoxybenzamine reduced the overall mortality rate in dogs undergoing surgical resection of pheochromocytoma; treated dogs were six times more likely to survive than dogs that did not receive phenoxybenzamine as a pre-treatment (mortality rate 13% versus 48% in untreated dogs). Potential complications of adrenalectomy include hypertension, hypotension, cardiac arrhythmias, and hemorrhage.9-11

Medical management is indicated for dogs when surgical resection is not a viable treatment option. Phenoxybenzamine is recommended long term for medical management of catecholamine release. Beta blockers, such as propranolol or atenolol, may be considered for severe tachycardia to prevent unopposed alpha stimulation and uncontrolled hypertension. Overall, the median survival time following surgical resection of a pheochromocytoma is 1 year, with some dogs surviving 2 and 3 years post-operatively. Actual metastatic rates are difficult to determine as, histologically, the appearance of pheochromocytomas are variable and not predictive of malignancy. Dogs with tumors larger than 5cm, evidence of metastasis, or thrombosis have a worse prognosis than dogs without these factors. Adjuvant chemotherapy has not been studied in dogs and is not thought to be helpful in people; radiation therapy has also not been studied.10-11

Conclusion

Once an adrenal mass is detected in the dog or cat, clients should be counseled on the potential differential diagnoses and management. Endocrine testing should be performed in addition to a minimum database, retinal examination, and blood pressure. If the mass is functional, invasive, or greater than 2.5 cm surgical resection should be discussed. Appropriate pre-operative supportive care will be dependent on the results of the examination and recommended diagnostics.

References

3 Melian et al in Ettinger and Feldman eds., Textbook of Veterinary Internal Medicine 7th ed.1816-1840.
6 Eastwood JM, Elwood CM, Hurley KJ. J of Small Ani Practice 2003 Mar, 44(3) 126-31.
Lymphoma is a common hematopoietic cancer diagnosed in both the dog and cat. The etiology, presentation, location, and prognosis in the dog and cat differ, and certain anatomical sites carry a better or worse prognosis. In most cases, lymphoma is considered a systemic disease and therefore is treated with systemic chemotherapy, although exceptions are discussed below.

**Canine lymphoma**

Lymphoma (LSA) is one of the most common canine malignancies, and is the most common hematopoietic tumor in dogs. Breeds with a higher risk of LSA include the Boxer, Basset, Scottie, Airedale, and Bulldog. Clinical signs of disease are variable, ranging from no signs to severe illness. The most common anatomical form of LSA in the dog is multicentric, with ~80% of dogs presenting with a primary complaint of generalized lymphadenomegaly. Other primary LSA sites in the dog are craniomediastinal (which often is associated with pleural effusion), alimentary, cutaneous, and primary extranodal sites such as eyes, central nervous system, nasal cavity, testes, bladder, and heart.

**Multicentric lymphoma**

On presentation, dogs with multicentric LSA have enlarged peripheral lymph nodes, often without clinical signs. Differential diagnoses for peripheral lymphadenomegaly include disseminated infections such as toxoplasmosis, leishmania, rickettsial, fungal, parasitic, bacterial, immune mediated disease such as pemphigus or lupus, tumors that have metastasized to locoregional lymph nodes, or other cancers.

Since most canine LSA are large cell (lymphoblastic), diagnosis is usually achieved by fine needle aspiration and cytology of an enlarged lymph node. Cytology is inexpensive and has a rapid turn-around time, and is therefore recommended as the initial diagnostic for enlarged lymph nodes. A diagnosis of lymphoma can be made when greater than 50% of cells are lymphoblasts (in most cases, greater than 80% of cells are lymphoblasts); plasma cells and inflammatory infiltrates are uncommon. On cytology, reactive lymph nodes have a heterogeneous population of mixed sized lymphocytes, and plasma cells with other inflammatory cells are common.

In some cases, particularly small cell lymphoma when the majority of lymphocytes are small and mature, diagnosis may not be possible with cytology alone. Therefore, many other tools are available to confirm a diagnosis of LSA. Lymph node biopsy or excision with histopathology can be performed; in addition, immunohistochemistry can be used on the formalin fixed tissues to differentiate B cell (CD79a, CD20 positive) from T cell (CD3 positive) LSA. Polymerase chain reaction receptor rearrangement (PARR), which is performed on blood or lymph node aspirate samples/slides, can also be used for LSA confirmation. PARR is an assay that determines if the majority of lymphocytes being tested have the same immunoglobulin for B cell or receptor for T-cell gene. If a majority of cells are clones, this indicates neoplasia compared to multiple genes in the majority of lymphocytes, which is consistent with a reactive population of lymphocytes (infectious/inflammatory causes). Of note, *Ehrlichia canis* infection may cause a false positive result within a T cell clonal population. For determination of phenotype without histopathology and immunohistochemistry, flow cytometry may be used.1

Staging can be utilized for evaluation and monitoring of response to treatment. Staging includes complete blood count (28% of dogs with LSA will have circulating malignant cells), chemistry panel, urinalysis, chest radiographs, abdominal radiographs, abdominal ultrasound, and bone marrow aspirate (57% of dogs will have evidence of LSA infiltration in the bone marrow). A minimum database is required prior to use of chemotherapy. The WHO clinical staging scheme is quite useful, and is defined as the following:

- **Stage I:** Single node or lymphoid tissue in a single organ, excluding BM
- **Stage II:** LN in regional area (cranial or caudal to diaphragm)
- **Stage III:** Generalized lymphadenopathy (both cranial and caudal to diaphragm)
- **Stage IV:** Liver and/or spleen involvement (+/- Stage III)
- **Stage V:** Blood/bone marrow or other organ systems +/− stages I-IV

Substage a: no clinical signs and b: clinical signs

Prognostic indicators are helpful when discussing an individual dog’s expected outcome with treatment with clientele. The following have been considered reliable prognostic indicators: substage, with substage a better than b; location, with cutaneous (diffuse), alimentary, mediastinal, and central nervous system having a worse prognosis; immunophenotype, with B cell better than T cell; prior glucocorticoid therapy which is a negative prognostic indicator due to the potential development of multidrug resistance; and response to treatment.2
**Paraneoplastic syndromes**

Anemia is the most common paraneoplastic syndrome, and it is usually nonregenerative, normocytic, normochromic, and also non-clinical. Through the production of parathyroid hormone related protein and other mechanisms, hypercalcemia occurs in approximately 15% of dogs with LSA overall, and in 40% of dogs with mediastinal or T cell LSA. Other frequent paraneoplastic syndromes are fever, monoclonal gammapathies, neuropathies, and cachexia. When clinical signs of LSA are present, they can include weight loss, fever, anorexia, lethargy, polyuria, polydipsia, anorexia, and other systemic signs of illness.

**Therapy**

Treatment of canine multicentric LSA is often rewarding - although cures are rare, most dogs respond quickly to therapy and can lead an excellent quality of life. Chemotherapy remains the mainstay of treatment, and many combinations of therapy have been studied. With the use of glucocorticoids alone, the median survival time (MST) is reported to be 4-8 weeks – the MST is the point at which 50% of dogs are alive, and 50% are not. Important to discuss with clients is that use of glucocorticoids prior to chemotherapy may lead to tumor cell upregulation of the p-glycoprotein pump, which causes resistance to the chemotherapeutics doxorubicin and vincristine. Single agent doxorubicin (usually with prednisone) is often used, given once every 3 weeks for 5 treatments. This provides a remission rate of approximately 75%, with a median remission time of 4-5 months. However, the ideal treatment is combination chemotherapy, using multi-drug protocols with drugs having different mechanisms of actions and effective at different parts of the cell cycle. This achieves more efficient cell kill and the development less drug resistance. CHOP and L-CHOP based protocols are the most frequently used for canine LSA:

- C: cyclophosphamide (Cytoxan®)
- H: hydroxydaunorubicin (doxorubicin, Adriamycin®)
- O: Oncovin® (vincristine)
- P: prednisone
- L: +/- asparaginase (Elspar®)

A CHOP protocol will provide 80-90% overall remission rate with a MST of 12 months. Alternatively, a COP protocol can be used, which provides a 70% remission rate for a of MST 6 months (similar to single agent doxorubicin).3,4 If a CHOP protocol has been completed and the dog remains in clinical remission for a period of time, when LSA relapses initiation of CHOP is again recommended with successful results. Overall, when used as a first line treatment, CHOP provides a median remission of 9.6 months; when retreated with CHOP following relapse, a 78% complete response is achieved with median remission duration of 5.3 months. The CHOP protocol is recommended as long as the patient continues responding to it.

Rabacfosadine (Tanovea™) was recently conditionally approved by the FDA for the treatment of canine lymphoma. Response rates as a single agent in naïve lymphoma are promising (79% partial or complete response) and when used alternating with doxorubicin overall response rate was 84%.10-11

Once refractory to CHOP, many rescue agents have been used with the general overall response rate of 40-50%, with median duration 1-3 months. Common protocols are CCNU alone or with asparaginase; LOPP (CCNU, vincristine, procarbazine, prednisone); MOPP (mechlorethamine, vincristine, procarbazine, prednisone); Doxorubicin + dacarbazine; vinblastine single agent, and mitoxantrone.

As LSA is a systemic disease, surgery is infrequently used for treatment. However, in some situations, surgery may be appropriate. These situations include solitary (early stage I) or solitary extranodal (cutaneous) LSA. Radiation therapy can be useful in localized for definitive therapy or in palliative situations, including nasal, oral, mediastinal, and cutaneous.

**Non-multicentric lymphoma**

Alimentary lymphoma in dogs carries a poor prognosis. One study reports a median survival time of 13 days with surgery, chemotherapy, or a combination of therapies and another report revealed a median survival time of 77 days with combination chemotherapy. Cutaneous lymphoma also carries a poorer prognosis than multicentric lymphoma; epitheliotrophic lymphoma has an overall response rate (partial and complete) of 83%, but the median duration is only 3 months. Central nervous system lymphoma is treated ideally with a combination of radiation therapy and chemotherapy that crosses the blood brain barrier (such as cytosar arabinoside), but duration of response is short. Dogs with primary hepatic lymphoma have a median survival time of only 2 months, although 4 months is reported in 44% of dogs that achieved complete remission with a doxorubicin based protocol.2

**Feline lymphoma**

Lymphoma is the most common hematopoietic tumor in cats. Unlike the dog, immunophenotype (B vs T cell) is not prognostic. Furthermore, the etiology of some lymphoma cases in cats is better defined than in the dog. For example, one direct causative agent of LSA in cats is Feline Leukemia Virus (FeLV); other contributing etiologies are Feline Immunodeficiency Virus (FIV), tobacco smoke in the household, immunosuppression (10% of cats with renal transplants and subsequent immunosuppressive therapy develop LSA), and chronic inflammation (such as inflammatory bowel disease). FeLV plays a direct role in tumor formation, and cats with FeLV have a 60 fold increased risk of developing LSA compared to FeLV- cats; 25% of cats that are FeLV+ develop LSA. FeLV+ cats are younger and predisposed to the mediastinal, multicentric, and spinal LSA locations. As opposed to the direct role that FeLV...
plays, FIV plays an indirect role in tumor formation by causing immunosuppression, and therefore increases the risk of lymphoma development. As rates of FeLV and FIV infection have decreased, LSA associated with these diseases has decreased as well.

**Alimentary lymphoma**

Most cats with lymphoma are older, with a median age of 11 years, and are FeLV and FIV negative. The alimentary form is most common location and is increasing in occurrence. It can be solitary or diffuse through the intestinal muscle layers and submucosa causing complete or partial obstruction. Clinical signs commonly include anorexia and weight loss, in addition to vomiting and diarrhea. On presentation, many cats will have palpable abdominal mass or thickened bowel loops, and 75% will be anemic. A definitive diagnosis is achieved with cytology or biopsy and histopathology of abnormal areas. Staging includes minimum database, chest and abdominal imaging, and a bone marrow aspirate if significant cytopenias are present.

Most cats have high grade lymphoblastic LSA, and with combination chemotherapy (CHOP) complete response rates range from 50-70% and median survival times range from 7-10 months. Approximately 25-30% of cats will have a remission of 1 year or longer. If a COP protocol is used, the median remission time decreases to 5 months. Positive prognostic factors in cats are a complete remission with therapy, FeLV-, early clinical stage, anatomic location, and addition of doxorubicin in chemotherapy protocol. Immunophenotype is not prognostic in cats as it is in dogs. Recent research has shown a possible survival benefit when abdominal irradiation is used following chemotherapy.6-8

**Small cell lymphoma**

A subset of cats have small cell (lymphocytic) lymphoma of the gastrointestinal tract. For diagnosis of small cell lymphoma, a biopsy sample (usually full thickness), is required. Treatment is much less aggressive, and involves only chlorambucil and prednisone. Response rates are high, with 70% of cats achieving complete remission, and the median survival time is 17 months.6,7 Even cats achieving partial remission can enjoy a good quality of life.

**Non-alimentary LSA**

Mediastinal LSA arises from the thymus, mediastinal lymph nodes, or sternal lymph nodes. Pleural effusion is common, and most are young, FeLV+, cats. Diagnosis is achieved by cytology of the pleural fluid or the mass. The median survival time is 2-3 months with CHOP treatment in FeLV+ cats. Radiation therapy is quite useful helpful for palliation of clinical signs associated with the mediastinal mass and pleural effusion, and is often used with or prior to chemotherapy in critical cases. Multicentric (nodal) LSA is much less common in cats than dogs. Many cats are FeLV positive, and important differential diagnoses are reactive lymph nodes, hyperplastic lymph nodes, infection, or FIV. Renal LSA can be primary, or associated with alimentary LSA. About 25% are FeLV+, and extension to the central nervous system is common. The kidneys are usually uniformly enlarged, and the median survival time is 3-6 months with chemotherapy; a chemo therapeutically that crosses the blood brain barrier is added into a protocol due to rates of the CNS extension. Nasal/paramosal LSA is a form that is usually localized (unless FeLV+) and represents 30-50% of feline nasal tumors. Most are FeLV negative and the LSA is of intermediate to high grade. This form of LSA has the best prognosis in cats, with a median survival time reported at 18 months to 2 years if localized and treated with radiation therapy.9 In contrast, large granular LSA, with cells that have abundant cytoplasm with prominent azurophilic granules (thought to be NK cells or cytotoxic T cells), have a very poor response to chemotherapy and short survival times.6,7

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How to Diagnose and Treat Thyroid Carcinomas

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The most important gland in metabolism control is the thyroid gland. Thyroid tissue is formed of follicles; the lumen is filled with colloid, the main storage form of thyroid hormones. Follicular cells produce and secrete triiodothyronine (T3) and thyroxine (T4). The formation of thyroid hormones is as follows: iodine is converted to iodide in the gastrointestinal tract and transported to the thyroid gland, where follicular cells trap iodide via active transport; iodide is then oxidized, and oxidized iodine attaches to tyrosine (which is a part of the thyroglobulin formed within the follicular cell) via thyroid peroxidase. Two iodide molecules attached to tyrosine yield diiodotyrosine; and attachment of 2 diiodotyrosine yield tetraiodothyronine (thyroxine, T4) and one moniodotyrosine and one diiodotyrosine yield triiodothyronine (T3). The T3 and T4 are stored in colloid for 2-3 months. The ultimate function of thyroid hormones is to increase metabolism, and high quantities can increase the basal metabolic rate by 60-100%. Outside the follicles are parafollicular cells (also called C-cells), which secrete calcitonin. Calcitonin has a minor role in calcium regulation (release in states of hypercalcemia, it will temporarily inhibit parathyroid hormone induced bone resorption); chronic increases or decrease in physiologic calcitonin do not result in long-term changes to biologically active plasma calcium.

Hyperthyroidism may also be present including polyuria, polydipsia, polyphagia, weight loss, and muscle wasting. Canine thyroid carcinoma is euthyroid, 30% are hypothyroid (likely due to destruction of normal thyroid tissue or suppression of TSH), and 10% are hyperthyroid. Approximately 35-40% of dogs with follicular thyroid carcinoma will have detectable metastasis at presentation, but up to 80% will develop metastasis. The most common metastatic sites are regional lymph nodes - mandibular, retropharyngeal, cranial cervical - lungs, and late in stage of disease, liver. Thus, staging procedures indicated include loco-regional lymph node palpation and cytology and thoracic radiographs to investigate for metastasis. Abdominal ultrasound should be performed if indicated. Parafollicular (medullary, C-cell) thyroid tumors have a lower metastatic potential than follicular thyroid tumors, although the distinction between follicular and parafollicular origin cannot be made without histopathology.

Canine thyroid carcinoma

The definitive etiology of spontaneously occurring thyroid tumors in dogs is not known. Interestingly, thyroid tumors retain thyroid stimulating hormone (TSH) receptors, and a study of hypothyroid beagles that did not receive thyroid supplementation revealed an increased incidence of thyroid tumors. Therefore, TSH stimulation of thyroid gland growth without normal negative feedback is theorized to play a role in thyroid tumor development. Thyroid irradiation is associated with an increased incidence of thyroid carcinoma in people, rodents and dogs. An iodine deficient diet is also hypothesized to increase TSH, therefore potentially increasing the risk of thyroid tumor development.

Dogs are middle aged to older at diagnosis, with the median age of 9 to 11 years. Overrepresented breeds include Golden retrievers, beagles, boxers, and Siberian huskies. The left and right thyroid glands are affected with equal frequency and up to 60% of thyroid carcinomas in dogs are bilateral. Most often, thyroid carcinomas are large, infiltrative, fixed masses in the ventral cervical region. Of particular note is that well circumscribed and freely moveable thyroid carcinomas – those that are quite amenable to surgery - are reported in 30% of dogs. Additionally, ectopic thyroid tumors can be found anywhere between the base of the tongue and the base of the heart, with the tongue base, cranial mediastinum, and heart commonly reported.

A palpable cervical mass is the most common presenting complaint and exam findings. Differential diagnoses include an abscess, granuloma, salivary mucocele, lymphatic metastasis of a tonsillar squamous cell carcinoma, carotid body tumor, or sarcoma. When clinical signs are present they may include cough, tachypnea, dyspnea, dysphagia, dysphonia, laryngeal paralysis, Horner’s syndrome, and facial edema. Acute hemorrhage can occur if the cervical vasculature is invaded by tumor. Although uncommon in dogs, signs of hyperthyroidism may also be present including polyuria, polydipsia, polyphagia, weight loss, and muscle wasting.

Initial diagnostic usually include fine needle aspiration and cytology of the mass, noting that non-diagnostic samples are common due to hemodilution, since thyroid carcinomas have a high vascular density. If cellular, cells of neuroendocrine origin suggest thyroid carcinoma in a dog with a large cervical mass. Ultrasound guidance for fine needle aspiration can be helpful. Histopathology will allow for definitive diagnosis using biopsy or surgical excision.

Additional diagnostics include a complete blood count, chemistry panel, and urinalysis – although these do not reveal changes specific to thyroid carcinoma, baseline assessment of organ function is helpful for determining therapeutic options. Furthermore, a thyroid panel (total T4, free T4, TSH) is recommended for all dogs with a thyroid mass. Approximately 60% of dogs with thyroid carcinoma are euthyroid, 30% are hypothyroid (likely due to destruction of normal thyroid tissue or suppression of TSH), and 10% are hyperthyroid. Approximately 35-40% of dogs with follicular thyroid carcinoma will have detectable metastasis at presentation, but up to 80% will develop metastasis. The most common metastatic sites are regional lymph nodes - mandibular, retropharyngeal, cranial cervical - lungs, and late in stage of disease, liver. Thus, staging procedures indicated include loco-regional lymph node palpation and cytology and thoracic radiographs to investigate for metastasis. Abdominal ultrasound should be performed if indicated.
Advanced imaging is indicated in determining a definitive treatment plan. Cervical ultrasound can guide fine needle aspiration or biopsy and confirm thyroid gland involvement. CT scan or MRI are will aid in determining if the mass is surgically resectable and for surgical planning. However, it is important to note that should a client be interested in $^{131}$I treatment, the iodinated contrast commonly used for CT scans will interfere with thyroid scans and $^{123}$I treatment. Thus, CT with iodinated contrast should be avoided when $^{131}$I treatment is a consideration.

Nuclear scintigraphy is a useful tool to determine if $^{131}$I is a potential treatment option for canine thyroid carcinomas, and the dog does not need to be hyperthyroid for this treatment to be considered. If a thyroid carcinoma is capable of trapping $^{99m}$Tc-pertechnetate or if the tumor can trap and organify $^{123}$I or $^{131}$I, the primary tumor can be visualized on scintigraphy and $^{131}$I may be a valid treatment option, even if the tumor cannot complete the remaining steps for synthesis and secretion of functional thyroid hormone. If the dog is not a good candidate for $^{131}$I due to inadequate uptake on thyroid scan or if the owners do not want to consider this therapy, a contrast CT scan can then be pursued.

Numerous treatment options are successful in the treatment of canine thyroid carcinomas. Some thyroid carcinomas are amenable to surgery at time of diagnosis. With surgery alone to treat freely movable tumors, the median survival time reported is 3 years. If the mass is considered invasive, the median survival time decreases to 6 to 12 months. Potential complications of surgery (dependent on tumor size and invasiveness) are laryngeal paralysis, megaesophagus, hypocalcemia (due to removal of parathyroid glands with the thyroid), and upper airway distress.

Histopathology following surgery or biopsy will determine the cell of origin. Most canine thyroid carcinomas are of follicular cell origin, which is further subdivided histologically into papillary, follicular, compact (solid), mixed or anaplastic. While these subtypes are prognostic in people, they have not been found to be prognostic in dogs and these subtypes are treated similarly. Para follicular or medullary (C-cell) carcinomas of the thyroid gland are less common, representing approximately 36% of thyroid carcinomas. If a diagnosis of neuroendocrine tumor is obtained without a distinct cell of origin, immunohistochemistry (IHC) should be pursued to confirm thyroid gland origin, then cell of origin since it is prognostic. Follicular carcinomas will stain positive for thyroglobulin and parafollicular carcinomas will stain positive for calcitonin, calcitonin gene-related peptide, chromogranin A, and neuron specific enolase. While both follicular and parafollicular thyroid carcinomas will stain positively for thyroid transcription factor-1, parafollicular thyroid carcinomas will be negative for thyroglobulin.

For thyroid tumors that are unresectable or those incompletely excised with surgery, external beam radiation therapy and $^{131}$I radiotherapy are commonly used. Using external beam radiation therapy in the face of palpable tumor, the progression free survival is 80% at 1 year and 72% at 3 years post-therapy. Also of note is that reported times to maximal tumor reduction ranges from 8 to 22 months following completion of radiation therapy. Fine fractionation protocols are preferred due to potential of late side effects to larynx, esophagus, and trachea. Early effects of radiation therapy, which resolve with supportive care, include alopecia, erythema, and mucositis. Late effects are uncommon with currently utilized protocols but may include skin fibrosis, alopecia, chronic tracheitis, or dry cough. Median progression free survival and survival times are reported at 24-45 months. Similarly, $^{131}$I can be used effectively post-operatively or in the face of bulky disease, if scintigraphy reveals that the thyroid carcinoma is capable of trapping or organifying $^{99m}$Tc-pertechnetate or $^{123}$I, respectively. Reported median survival times are 28-34 months if metastasis is not present, and 12 months with evidence of metastasis. The most common side effect of $^{131}$I is myelosuppression, and, although asymptomatic in most cases, complete blood counts are recommended weekly for 4-6 weeks following treatment.

Chemotherapy can also be considered for dogs with non-resectable tumors. Response rates of 30-50% are reported with cisplatin or doxorubicin chemotherapy. The role of chemotherapy following local disease control with surgery, radiation therapy, and/or $^{131}$I is not yet well defined. Tyrosine kinase inhibitors may also play a role in the treatment of non-resectable disease. Canine thyroid carcinomas express VEGFR2, PDGFR, and KIT, which are inhibited by toceranib. One large study of toceranib included 15 dogs with bulky thyroid carcinoma, and reported 26% partial response and 53% stable disease.

The metastatic potential of thyroid carcinomas increases with increasing tumor size (> 23cm$^3$) and the presence of bilateral disease. Although canine thyroid carcinomas have high metastatic rates (up to 80% overall), metastases tend to be slow growing and dogs can enjoy a long term good quality of life even when metastases are present. In one study investigating hypofractionated external beam radiation therapy, the overall median survival time was 1.8 years for 13 dogs, 5 of which had pulmonary metastatic disease. Metastatic disease has also been reported to respond to $^{131}$I in some cases, and chemotherapy in others.

**Feline thyroid carcinoma**

Bilateral feline thyroid hyperplasia and adenomas are present in ~70% of feline hyperthyroidism cases, and hyperthyroidism is the most common endocrinopathy of cats. Common clinical signs include weight loss, polyphagia, palpable goiter, heart murmurs and tachycardia. Diagnosis is usually made with high normal to elevated total T4 measurements. Thyroid carcinomas are uncommon, representing only 1-3% of hyperthyroid cats, and follicular carcinoma is the most common type diagnosed. Nonfunctional and nonsecretory thyroid tumors are rare in cats. The clinical presentation is similar to cats with hyperthyroidism caused by thyroid adenomas. Nuclear scintigraphy with $^{99m}$Tc-pertechnetate may reveal patchy or irregular uptake within the thyroid, extension or...
invasion of the thyroid mass, or uptake at metastatic sites. However, scintigraphy is not a sensitive diagnostic tool for determining benign versus malignant disease.

In contrast to most adenomas, carcinomas tend to recur following surgery and do not respond to the lower doses of $^{131}$I used to treat adenomas. Thyroid carcinomas are firm, fixed, and invasive, and potentially involve multiple masses. Definitive diagnosis requires histopathology. Metastasis is reported in up to 70% of cases, usually to the lymph nodes and lungs. Staging should include a minimum database and thoracic radiographs, with abdominal ultrasound if appropriate. The treatment of choice for thyroid carcinomas in cats is surgical resection followed by high dose $^{131}$I, with survivals reported to range from 10 months to 41 months. $^{131}$I has also been used as the sole treatment with a median survival time of 814 days. Higher doses of $^{131}$I are utilized in thyroid carcinomas compared to adenomas as cats with thyroid carcinomas have larger tumor burdens, and malignant cells may not trap and retain iodine as efficiently as adenomas. Medical management with methimazole may also be used to control clinical signs in cats when surgery or $^{131}$I are not viable treatment options.14

Conclusions

Thyroid tumors in the dog are most often large, invasive, and do not produce T4. Despite high rates of metastasis, metastases tend to be slow growing and many dogs can enjoy a good quality of life for long periods of time. Treatment options are numerous including surgery, external beam radiation therapy, chemotherapy, tyrosine kinase inhibitors and radiiodine therapy - overall treatments are well tolerated. Lack of hyperthyroidism does not exclude dogs with thyroid tumors from $^{131}$I therapy and nuclear scintigraphy can identify potential candidates. If $^{131}$I is a therapy clients will consider, a contrast CT should be avoided until nuclear scintigraphy is completed; if a CT scan is performed a washout of up to 4-6 weeks may be recommended prior to nuclear scintigraphy and $^{131}$I therapy. Cats most often have benign thyroid tumors easily treated with $^{131}$I, and if malignant higher dose $^{131}$I may be utilized.

References

Research and development of new immunotherapies is rapidly expanding in both human and veterinary oncology, some therapies showing remarkable results. The immune system plays multiple roles in the prevention and the development of cancer. For example, the immune system fights and cures infectious agents that can directly cause cancer, resolves inflammation that promotes cancer, and recognizes and kills tumor cells when they form. It is therefore not surprising that underlying immune dysfunction exists in cancer patients, both human and veterinary. Furthermore, the immune system has a pro-tumor role, as chronic inflammation in the presence or absence of infection promotes carcinogenesis when unregulated. The immune system can additionally support tumor formation by selecting cancer cells that can best survive in an immunocompetent host. The tumor itself evades and even utilizes the immune system to its advantage by secreting molecules that result in systemic immunosuppression to protect itself from the body’s natural defenses. Based on this knowledge, and that gleaned from both human and veterinary studies, a further understanding of the immune system’s role in cancer is important to utilize targeted therapies and manipulate immunity back to the advantage of the patient. As the conflicting roles of the immune system in cancer become better understood, immunotherapy is becoming more prevalent in the veterinary clinical trial and clinical practice settings.1

Background of immune dysfunction and loss of immune surveillance in cancer patients

The cellular component of the innate immune system is comprised of neutrophils, macrophages, dendritic cells (DCs), and natural killer cells (NKs); these are rapidly acting and not specific. The adaptive arm of the immune system is quite specific to the insult, and responds more robustly when exposure is repeated; adaptive immunity consists of T and B lymphocytes. Both arms of the immune system are critical for defense against cancer.

Immune surveillance is the system by which the immune system recognizes cancer cells as foreign, and triggers their elimination. The proof of this concept lies in murine model studies, where mice lacking interferon-γ (IFNγ) responsiveness or adaptive immunity are more susceptible to both spontaneous and carcinogen induced cancer. This theory is further supported by findings in other species with underlying immunodysfunction and increased cancer risk. For example, dogs with immune mediated thrombocytopenia have a higher incidence of lymphoma compared to dogs without immune mediated thrombocytopenia, and cats with renal transplantation and long term cyclosporine administration had a six fold higher risk of developing lymphoma.2,3 Dogs with cancer have reduced neutrophil oxidative burst, NK proliferation, lymphokine activated killer function, and blunted inflammatory response to bacterial pathogen associated molecular patterns compared to normal dogs.4-6 Furthermore, dogs with cancer have increased percentages of T regulatory cells compared to normal dogs, particularly dogs with carcinomas.7

Tumors that form in a deficient immune host are more immunogenic than tumors that form in the immunocompetent. In fact, the immune system may help shape clonal selection and the immunogenicity of the tumor itself. For example, the immune system can promote tumor formation by selecting for cells best adapted to survive in a normal immune system environment or by creating conditions within the tumor microenvironment that facilitate tumor growth. Cancer immunoediting refers to the elimination of cancer by the immune system, the selection of less immunogenic tumor cells during the anti-tumor response, and/or evasion of the tumor from the immune system. Mechanisms of tumor evasion of the immune system are loss of tumor antigens, down-regulation of antigen presenting molecules, and tumor resistance to cytotoxic pathways including over-expression of anti-apoptotic molecules. Tumors secrete immunosuppressive molecules such as transforming growth factor β (TGF-β) and interleukin-10 (IL-10). TGF-β binds to its receptors on lymphocytes, altering the lymphocyte phenotype and therefore its cytokine secretion profiles. These cytokine profiles are implicated in the generation of T regulatory cells that inhibit other T cells. IL-10 binds to its receptor expressed on immune cells to upregulate genes responsible for preventing maturation of DCs; furthermore, IL-10 has direct effect on CD4+ T cells and inhibits their proliferation and their production of cytokines. Further study evaluates the ability of cancer to cause immunosuppression to improve its chances of survival.1 The function of myeloid cells (monocytes, macrophages, DCs) when exposed to soluble factors excreted from canine tumor cells was significantly decreased; phagocytic activity was blunted leading to decreased tumor antigen uptake, decreased MHC class II expression lead to decreased tumor antigen presentation, and diminished stimulation of the adaptive immune system due to decreased CD80 expression was found.8

The role of inflammation

Inflammation is an important physiological response against injury or infection; inflammation is also highly regulated and short lived in those with intact immune systems with the help of anti-inflammatory mediators. However, when inflammation is not well controlled and becomes chronic, it contributes to carcinogenesis. A cell component of the chronic inflammatory response is macrophages, producing tumor necrosis factor α (TNF-α). TNF-α will coordinate the inflammatory response by inducing a range of
effector molecule release, and many of these effector molecules are proteins that perpetuate inflammation. Furthermore, leukocytes produce reactive oxygen species (ROS) and nitric oxide (NO) to help fight infection, however, these both cause DNA damage (through the formation of peroxynitrite). So chronic inflammation, leading to increased ROS and NO production, leads to an increase in DNA damage and increased DNA mutation rates. The increase in DNA mutations rates leads to an increased risk of cancer.1,9

Several pro-inflammatory products, including TNF-α, interleukins, and chemokines, mediate processes that are known to be critical in tumor formation: cellular proliferation, apoptosis, angiogenesis, and metastasis. The final link between inflammation and cancer is the transcription factor NF-KB, a transcription factor that regulates the expression of genes that encode for these pro-inflammatory products. NF-KB is induced by macrophages, target cells of inflammation, and cancer cells. It will inhibit apoptosis, promote angiogenesis, and promote metastasis. In addition to inducing anti-apoptotic protein production (Bcl-xL, c-Flip), promoting angiogenesis (vascular endothelial growth factor), and various cytokines, NF-KB activates cyclin D, which stimulates the cell to progress through the cell cycle and expression of pro-inflammatory genes, such as cytokines and COX-2. Overall, NF-KB helps maintain the inflammatory response and promotes metastasis.1

Cancers linked to chronic inflammation are ocular sarcoma and injection site sarcoma in the cat, urinary bladder transitional cell carcinoma, squamous cell carcinoma, and osteosarcoma in the dog, and lymphoma in the dog and cat. Cancers linked to chronic infection in the cat are lymphoma (FeLV), sarcoma (FeSV), sarcoid (bovine papilloma virus), and squamous cell carcinoma (papilloma virus). In the dog, two cancers are associated with chronic infection: esophageal sarcoma (Spirocirca lupi) and squamous cell carcinoma (papilloma virus). In addition, there is a suspected link between lymphoma and inflammatory bowel disease in both the dog and cat.9

Biologic response modifiers (non-specific immune stimulation)
The goal of non-specific immune activation is to stimulate the innate and adaptive immune system to recognize tumor cells as non-self. Immune stimulators are, for the most part, innate immune stimulators as increased antigen presenting cell activity (DCs, macrophages) will lead to more effective T and B cell responses.10 Immunootherapy must overcome many of the mechanisms of tumor escape described above, however, immunotherapy has been successfully carried out and shown efficacy, alone and in combination with other therapies. Evidence of a blunted immune system and overcoming this is described in dogs with osteosarcoma. Dogs with localized osteomyelitis following a limb-sparing procedure had nearly double the survival time of dogs with no infection following surgery.11 A follow up study using the murine model of canine osteosarcoma revealed that bacterial osteomyelitis elicited non-specific tumor growth inhibition mediated by NKs, inflammatory monocytes, and tumor associated macrophages.12 These results are encouraging for the development of future immunotherapies, and, in fact, systemic immune stimulation is successful in the treatment of other canine cancers.

The bacillus of Calmette and Guerin (BCG) is a modified strain of Mycobacterium bovis. Infusion of BCG into the bladder is an integral treatment for human non-invasive bladder cancer; unfortunately, dogs and cats develop invasive bladder cancer and BCG is ineffective. However, intravesical BCG has been used successfully in the treatment of equine sarcoidos and bovine ocular squamous cell carcinoma.10 Immunocidin™ is a formulation of mycobacterial cell wall that is USDA licensed for use intra-lesionally in canine mammary cancer. A large clinical trial has not been published at the time of these proceedings, but preliminary data from the licensing study appear promising. Liposome-encapsulated muramyl tripeptide (L-MTP-PE) is composed of mycobacterial cell wall components encapsulated in a liposome. It activates monocytes and macrophages with the result of increased production of pro-inflammatory cytokines, include IL-1, IL-6, IL-7, IL-8, IL-12, and TNF-α. When used intravenously following amputation in stage II canine osteosarcoma as the sole adjuvant treatment, survival was significantly prolonged compared to dogs not treated with adjuvant therapy. The response with combined L-MTP-PE and cisplatin chemotherapy post-op showed no benefit when administered concurrently. However, dogs treated with surgery and cisplatin/ L-MTP-PE sequentially did have a survival advantage compared to those dogs receiving adjuvant cisplatin alone. A survival advantage was also found in dogs diagnosed with splenic hemangiosarcoma – following surgery, dogs treated with doxorubicin, cyclophosphamide, and L-MTP-PE had higher serum levels of TNF-α and IL-6 and longer survival compared to dogs treated with surgery, chemotherapy, and empty liposome. Availability of this product, however, is limited.10

Liposomal DNA complexes are composed of bacterial DNA motifs which stimulate the activation of DCs. Antitumor activity is mediated by NKs, DCs, and the release of pro-inflammatory cytokines. These complexes can also be used for gene delivery, and when administered IV with a gene encoding for IL-2 to dogs with pulmonary metastatic osteosarcoma, potent immune activation and NK cell activity was noted in dogs, with an increase in overall survival.13

Oncolytic viruses are viruses that preferentially replicate within cancer cells, and result in lysis of the tumor cells. Oncolytic viruses are tumor selective and can directly kill tumor cells; additionally, the y can be used for targeted delivery of drugs, cytokines or genes. Currently, canine adenovirus and canine distemper virus are under investigation for this technique.10
Cytokine therapy (non-specific immune stimulation)
Interleukin-2 activates DCs, macrophages, and B cells in addition to inducing local expansion of T cells and stimulating NK cells. Therefore, it plays an important role in immune system stimulation and is a promising therapeutic intervention. Although side effects are considerable when IL-2 is used in people, IL-2 has been used with success in the treatment of canine cancer. For example, in dogs with carcinoma of the urinary bladder or urethra, intravesical IL-2 was administered into the mass or post-operatively into the tumor bed, with some responses. Intravenous gene delivery using liposome-DNA IL-2 complexes is safe in canine osteosarcoma and systemic immune activation resulted; 3 of 20 dogs had partial or complete regression of lung metastases; an inhalational formulation of liposomal IL-2 was also found to be safe, with some evidence of preliminary efficacy. Interleukin 12 (IL-12) also has pronounced stimulatory effects on both the innate and adaptive immune system, in addition to having an anti-angiogenic effect. IL-12 and IL-15, which stimulates NK cells and promotes proliferation of T cells, are under investigation for the treatment of canine cancer.

Recombinant canary pox virus expressing feline IL-2 has been successfully utilized in cats with injection site sarcomas. When administered post-operatively and combined with brachytherapy, time to recurrence was improved compared to cats treated with surgery and brachytherapy alone.

Cancer vaccines (specific and targeted immune stimulation)
The adaptive immune system detects tumors through tumor associated antigens, recognized by cytolytic T cells and antibodies. Tumor associated antigens can be specific for a particular tumor, arise from a mutated gene product, or they can be normal cellular antigens that are overexpressed. The ultimate goal of a tumor vaccine is an antitumor response that results in regression of the tumor. Because the adaptive immune system takes longer to induce than the innate immune system, a response to cancer vaccination may take several months. The currently available vaccine for canine cancer treatment is Onccept® for the treatment of melanoma. This particular vaccine is a xenogeneic DNA vaccine; each dose contains plasmid DNA with the gene encoding for human tyrosinase. This is administered transdermally every 2 weeks for 4 treatments, then once every 6 months. Studies have shown that this treatment is safe, and is promising for prolonging survival in dogs with loco-regional disease control (surgery and/or radiation therapy). Of note, when using this vaccine, it is important that local disease be controlled.

Other approaches to tumor vaccination are the use of autologous vaccines - whole tumor cell and tumor cell lysate vaccines (these are made directly from the patient’s tumor cells or from a cell line of the same tumor type and administered with an adjuvant for immune stimulation). This has shown promise in combination with standard care in canine hemangiosarcoma and canine lymphoma. Many clinical trials are ongoing at various institutions to evaluate production, safety, and efficacy of these vaccines. Dendritic cell vaccination is also under investigation due to their potent antigen presenting ability. An attenuated recombinant *Listeria monocytogenes* expressing chimeric human Her2/neu ((Her2/neu is a tyrosine kinase receptor overexpressed in pediatric and canine osteosarcomas) with the goal of inducing Her2/neu specific immunity was reported in a small number of dogs following amputation and carboplatin chemotherapy (administered every 3 weeks, for 4 treatments). The results revealed that Her2/neu immunity was induced and the median survival time was prolonged compared to historical controls. Further investigation is ongoing.

Monoclonal antibodies, commonly used in the treatment of cancer in people as an adjunct to standard therapy, are not often used in veterinary oncology. Although a B and T cell monoclonal antibody were developed and USDA approved for used in dogs with lymphoma, further unpublished work was reported as disappointing, and development of monoclonal antibodies with better specificity are ongoing.

Numerous research projects are ongoing with the ultimate goal of a safe, effective, and affordable immunotherapy for dogs and cats with cancer.

Conclusions
The immune system is defective in patients with cancer, and both the innate and adaptive arms of the immune system are affected. Current research is further elucidating mechanisms of immune system dysfunction in dogs and cats with cancer. Promising research is ongoing to determine how we can manipulate the immune system, and, with combined our current standards of care, improve the prognosis of dogs and cats with cancer.

References
Canine mammary tumors

Mammary tumors usually occur in older female dogs, with an average age of 10-11 years. The development of mammary tumors is hormone dependent, and time of ovariohysterectomy (OHE) is correlated with the incidence of mammary tumors. Dogs spayed prior to the first estrus have the lowest risk of tumor development at 0.05%; this increases to 8% if OHE is performed after the first estrus and rises to 26% if OHE is performed after the 2nd estrus. The 50/50 rule for canine mammary tumors states that about half of canine mammary tumors are benign, and half are malignant. Of the malignant tumors, roughly half will metastasize. Therefore, to determine the best course of treatment, detailed histopathology and multiple prognostic factors are used.

Most mammary masses are presented as incidental findings on examination or following identification by clients. A notable exception are inflammatory mammary carcinomas – dogs with this cancer tend to present with mammary masses that are erythematous, warm to the touch, ulcerated, painful, and lymphedema in addition to systemic signs of disease including anorexia, weight loss, and lethargy. Upon discovering a mass in the mammary chain, fine needle aspiration and cytology should be pursued. Although cytology usually cannot distinguish between benign and malignant epithelial tumors, cytology can rule out non-primary mammary gland malignancy, which will change the approach for disease control. For example, subcutaneous mast cell tumors can occur in the mammary region and pre-operative supportive care will be quite different. If a malignant tumor is suspected or confirmed, staging should be performed prior to definitive treatment and includes investigation of common metastatic sites, namely regional lymph nodes, lung parenchyma, and abdominal lymph nodes. Other less common metastatic sites are bone, liver, kidneys, adrenal glands, spleen, pancreas, and diaphragm. Thoracic radiographs, abdominal ultrasound, and lymph node aspiration and cytology are the first steps in staging a malignant or suspected malignant mammary tumor.

The WHO modified staging system is used for canine mammary tumors:

Stage I represents a tumor less than 3cm in size, with no lymph node or distant metastasis.
Stage II is a tumor that is 3-5cm maximum, with no lymph node or distant metastasis.
Stage III is a tumor > 5cm in size, with no lymph node or distant metastasis.
Stage IV is any tumor size with lymph node metastasis.
Stage V is any tumor with distant metastasis, +/- lymph node metastasis.

Stage is prognostic in dogs with malignant mammary tumors. Dogs with tumors of less than 3cm in size, especially those that are well circumscribed, have a better outcome than dogs with tumors larger than 3cm or those with invasive or ulcerated masses. Histopathologic type and subtype are also important - most canine mammary tumors are carcinomas; sarcomas are much less common. The histologic subtypes of well differentiated, complex, or tubular/papillary carcinoma have a better prognosis then poorly differentiated, simple, solid, anaplastic or inflammatory carcinomas; mammary sarcomas are also a poor prognostic indicator. In addition, tumor grade, including indices of proliferation and evidence of lymphatic or vascular invasion can provide valuable information for treatment decisions. Evidence of lymph node involvement or distant metastasis is also predictive of outcome. A recent study found that with surgical treatment of mammary tumors, lymphatic invasion, ulceration, and incomplete surgical margins were negative prognostic indicators for overall median survival time.

Surgery is the initial treatment of choice for canine mammary tumors, with the exception of dogs with inflammatory mammary carcinoma or distant metastasis. The primary goal of surgery in the treatment of canine malignant mammary tumors is to use the simplest procedure to remove the entire tumor. All tumor tissue should be submitted for histopathology and in the case of multiple masses, all masses should have histopathology performed as both benign and malignant lesions may occur in the same dog and in the same mammary chain.

- Lumpectomy is indicated for masses that are known to be benign and are superficial, not fixed, and < 0.5cm in diameter.
- A mammectomy is used for masses that are >1cm and are centrally located within the gland.
- Depending on individual anatomy, however, a regional mastectomy may be a simpler procedure.
- Major lymphatic connections exist between glands 1 and 2, and glands 4 and 5. Glands 1, 2, and 3 drain to the axillary and cranial sternal lymph nodes and glands 3, 4, and 5 to the superficial inguinal lymph nodes. Surgically, glands 1, 2, and 3 or 4 and 5 can be removed en bloc with extensive mammary chain involvement.
- Radical unilateral or bilateral staged mastectomy is reserved for those tumors in which complete removal is not achievable with a less invasive procedure.
- If accessible, axillary and/or inguinal lymph nodes should also be excised and submitted for histopathology.
Chemotherapy, although not well studied, is used for dogs with mammary tumors that have a high risk of metastasis. This includes mammary sarcomas, mammary carcinomas of the subtypes anaplastic, simple, poorly differentiated, or solid, those with lymphatic or vascular invasion, high grade, and those with lymph node metastasis present. Cyclophosphamide and 5-fluorouracil (5-FU) in combination were used successfully in a small number of dogs with Stage III disease, and prognosis was improved with the use of this treatment regime. Doxorubicin and carboplatin have also studied in cell culture with efficacy against canine mammary carcinoma; however, there is another report of efficacy using doxorubicin in two dogs with distant metastasis of mammary adenocarcinoma. Water soluble paclitaxel (Paccal®Vet) has demonstrated preliminary efficacy in dogs with measurable disease in a small number of cases of mammary adenocarcinoma (1CR, 2PR, 1PD), although availability is currently limited. Radiation therapy is not well studied for canine mammary tumors given the general success of surgery, but is considered a viable option for tumor palliation in dogs with non-resectable tumors.

Staging and chemotherapy should be considered following surgery in those dogs determined to have a tumor with the high risk of metastasis. Poorly differentiated carcinomas treated with surgery alone have reported median survival times of 2.5 months, compared to 21 months in dogs with adenocarcinoma, and 16 months in dogs with solid carcinoma. Tumor size is also important – even with invasive tumors dogs with a mass of <3cm have a better prognosis than dogs with masses of >3cm. Dogs diagnosed with mammary sarcoma have an overall poorer prognosis; most will die or be euthanized due to disease progression within 9-12 months of diagnosis.

Inflammatory carcinomas have a very poor prognosis – most are rapidly progressive and become too large and infiltrative for surgical excision. In addition, many of these dogs are systemically ill and disseminated intravascular coagulation is frequent (reported in 21% of dogs). These tumors are generally not surgically resectable due to size, lymphedema, and associated disseminated intravascular coagulation. Short-term palliation may be achieved in some cases with non-steroidal anti-inflammatory drugs, radiation therapy, or chemotherapy, but the overall prognosis remains poor.

Factors that help predict prognosis include tumor size, histologic type and subtype, metastasis (lymph node or distant), tumor grade, degree of nuclear differentiation, degree of invasion, and lymphatic or vascular invasion. Surgery is the treatment of choice for canine mammary tumors, and the surgical procedure elected should be the simplest procedure that can be performed to achieve complete tumor margins. In high risk tumors, chemotherapy is recommended following surgery; 5-FU and cyclophosphamide, carboplatin, or doxorubicin are those most commonly recommended.

Feline mammary tumors
In contrast to canine mammary cancer, over 90% of feline mammary tumors are malignant. Mammary cancer is locally aggressive in cats, and due to extensive communication in vasculature between feline mammary glands, multiple gland involvement is common. Metastasis occurs through both the lymphatic and vascular routes, so common metastatic sites are lymph nodes, lungs, liver, and pleura, in addition to less common sites such as bone, kidneys, and adrenal glands. The Siamese breed may have an increased risk of metastasis. Poorly differentiated carcinomas treated with surgery alone compared to cats with a mass of >3cm. Other prognostic factors include extent of surgery, presence and location of metastasis, Ki-67 index and high AgNOR count.

Although only representing 10-20% of feline mammary tumors, benign lesions are a differential for a mammary mass. Benign lesions include duct papillomas, simple or complex adenomas, and fibroadenomas. Fibroadenomatous hyperplasia is an exaggerated proliferation of mammary tissue and is likely hormone dependent. This condition may develop during puberty, in pregnant or pseudopregnant queens, or in cats on hormonal therapy (megestrol acetate or maderoxyprogesterone acetate). Treatment of choice is removal of hormone source, usually OHE performed by a flank approach. Antiprogestins have also been used in cases where OHE is not desirable.

Malignant tumors are much more common than benign mammary tumors in cats, and carcinoma is the most frequent type diagnosed. Common subtypes of mammary carcinoma are tubular, papillary, ductular, anaplastic, and solid. Histologic type of carcinoma is prognostic, in that cats with papillary or tubular mammary carcinomas have much longer survival times than ductular carcinomas, and anaplastic carcinomas are considered to have the worst prognosis. Additionally, tumor size is important in determining prognosis. Cats with a mass of less than 2 cm or 2-3cm in size (WHO stages I and II respectively) have a better prognosis with surgery alone compared to cats with a mass of >3cm. Other prognostic factors include extent of surgery, presence and location of metastasis, Ki-67 index and high AgNOR count.

On physical examination, a discrete, palpable, and sometimes moveable mass or masses are palpated in the mammary gland. If advanced, ulceration, erythema, and edema may be present. Fine needle aspiration and cytology will rule out other malignancies and may potentially provide a diagnosis.

Staging in cats should be performed prior to surgery due to the high and often rapid rates of metastasis. Staging should include a minimum database (complete blood count, chemistry panel, urinalysis), aspiration of regional lymph nodes, thoracic radiographs and abdominal ultrasound. Cytology of pleural fluid if present or any suspicious lesions (including enlarged lymph nodes) on ultrasound should be performed.
The WHO modified staging system is used for feline mammary tumors:

Stage I: Mass <2cm, no nodal or distant metastasis
Stage II: Mass 2-3cm, no nodal or distant metastasis
Stage III: Mass >3cm OR nodal metastasis, no distant metastasis
Stage IV: Distant metastasis

Surgery remains the best first step in the treatment of feline mammary cancer. Cats have 4 pairs of mammary glands, 2 cranial that drain to the axillary lymph node and 2 caudal that drain to the superficial inguinal lymph node. There also exist small veins in all mammary glands that cross midline, which may allow spread of malignant tumors between pairs of mammary glands. Therefore, aggressive surgery is aimed at getting wide and complete margins. Most often, radical unilateral mastectomy is recommended, and, in some cases, staged bilateral mastectomy. These aggressive surgical procedures have been shown to increase disease free intervals compared to less aggressive surgeries.2,6

Chemotherapy is used post-operatively in cats with malignant tumors with the goal of extending disease free intervals and overall survival times. Doxorubicin based chemotherapy protocols are among the most commonly studied and used; doxorubicin single agent for 5 treatments (MST 448 days) or doxorubicin and cyclophosphamide combinations.7,8 In addition, mitoxantrone single agent compared to doxorubicin single agent (every 3 weeks, 4 treatments) has been studied following unilateral or bilateral mastectomy, with similar median survival times of 1.2-2 years were reported in both groups.9 Chemotherapy has also been used in situations where aggressive surgery is not possible. Using a combination of doxorubicin and cyclophosphamide, a 50% partial response rate was noted in 14 cats with mammary carcinoma in one study and 45% in another.10 Therefore, chemotherapy may be useful in cases where surgery is not feasible, and a decrease in tumor volume may help preserve the cat’s quality of life.

Overall, prognosis for cats diagnosed with mammary carcinoma is dependent on tumor size, grade, and histopathologic subtype. When treated with aggressive surgery followed by doxorubicin chemotherapy, cats with stage I and II mammary carcinoma have median survival times of 1.2 years and cats with a tumor of >3cm 6 months. With the addition of chemotherapy after surgery, the 2 year survival rate increases from 15-20% with surgery alone to 37% with surgery and chemotherapy. Cats with complex carcinomas have a longer median survival time (32.6 months) compared to cats with other carcinoma types (15.5 months). The presence of metastatic disease is a negative prognostic indicator.

Over 80-90% of feline mammary masses are malignant carcinomas, and diagnostic steps should include cytology, thoracic radiographs, abdominal ultrasound, and regional lymph node sampling to determine if metastasis has already taken place. Aggressive surgery with unilateral or staged bilateral radical mastectomy is recommended for local disease control, and chemotherapy should be considered following surgery due to the high potential for metastasis to occur.

References
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Canine osteosarcoma (OSA) is the most common primary bone tumor in the dog, and represents most cancers in the skeleton. The median age at diagnosis is 7 years, although a bimodal distribution exists with an increase in incidence at 18-24 months. Large to giant breeds are most commonly affected and higher risk breeds are the St Bernard, Great Dane, Irish Setter, Doberman pinscher, Rottweiler, German Shepherd, and golden retriever. Most OSAs occur in the appendicular skeleton; more specifically in the metaphyseal region of long bones. The distal radius and proximal humerus are the two most common sites overall and the distal femur and distal and proximal tibia are the most common sites in the hindlimb; less than 10% of OSAs are multicentric at the time of diagnosis.

The etiology of OSA has been studied, and many different factors have been found that influence the incidence of this disease. Physical factors, such as major weight bearing bones adjacent to late closing physes in heavy dogs may have multiple minor trauma and subsequent injury in cells leading to mitogenic signals, increasing chance of mutant cells. An association with metallic implants, chronic osteomyelitis, and even fractures without internal fixation has been found. Other causes are exposure to ionizing radiation (plutonium, radium), or a rare, late complication of ionizing radiation: 3% of dogs with OSA were treated for a soft tissue sarcoma with radiation therapy in that region. Molecular and genetic factors involved in OSA development are p53 (a tumor suppresser gene) that was mutated and non-functional, alterations in growth factors, cytokines, and hormone signaling systems, alterations in matrix metalloproteinases, which are matrix degradative proteins that may allow disease progression and metastasis, blood vessel density – which is greater in those dogs presenting with metastasis and increased plasma vascular endothelial growth factor (alterations in blood vessel formation), and telomerase reverse transcriptase gene upregulated in some OSA cell lines allowing cell immortalization.

OSA is a malignant mesenchymal tumor of primitive bone cells that produces extracellular matrix of osteoid, which is the basis of histological diagnosis. Subclassifications, although not definitively prognostic, include osteoblastic, chondroblastic, fibroblastic, telangiectatic, and poorly differentiated. Radiographically, these are aggressive locally, causing bone production and bone lysis; soft tissue swelling common; and pathologic fracture can occur. It is rare for an OSA to cross the joint surface as collagenase inhibitors limit progression through the synovium. OSA is aggressive systemically as well, with metastasis being common and the cause of loss of life in those dogs with adequate control of local disease and pain. Although less than 15% of dogs have radiographically detectable metastasis at diagnosis, 90% will die of metastatic disease. The most common sites of metastatic disease are lung, other bones, and other soft tissue sites. As a sarcoma, OSA moves through the hematogenous route and movement through lymphatics to local lymph nodes is rare; but when lymphatic movement does occur the prognosis declines dramatically. Clinical signs when metastasis occurs may be vague and hypercalcemia is very rare with osteosarcoma.

Presentation and differential diagnoses

Most dogs present with a history of lameness, sometimes following trauma, and swelling at primary site. Pain is caused by microfractures and disruption of peristeme induced by osteolysis of cortical bone with tumor extension from the medullary canal. Diagnosis is first suspected on physical examination and radiographs of the lesion. Common radiographic findings are cortical lysis, soft tissue extension, soft tissue swelling, and new bone (tumor or reactive bone). Codman’s triangle is a finding often seen with OSA; as tumor invades the cortex, the periosteum is elevated and new bone is laid down leading to a triangular appearing deposition of dense new bone on the cortex, at the periphery of the lesion. These findings are consistent with but not definitive for OSA. Differential diagnoses are other primary bone tumors (chondrosarcoma, fibrosarcoma, hemangiosarcoma), metastatic cancer to bone (transitional cell carcinoma, prostate carcinoma, mammary carcinoma), multiple myeloma or lymphoma of bone, systemic mycosis with bone localization, bacterial osteomyelitis, or bone cysts.

Diagnosis and staging

Diagnosis of OSA is confirmed by histopathology, although cytology is less invasive and can support the diagnosis. Unlike tumors in locations other than bone, it is important, regardless of method chosen, to sample the center of the lesion to obtain the best diagnostic sample. At the lesion periphery, reactive bone is present and may hinder confirmation of OSA. Staging is an important consideration for prognostic information, and may also determine treatment course. Although lymph node metastasis is rare, local draining lymph nodes should be palpated and aspirated if palpable. Thoracic radiographs, three views, are recommended to detect pulmonary nodules indicative of metastasis. A bone scan is a highly sensitive tool used to detect areas of bone metastasis; because a bone scan is so sensitive, radiographs of any suspicious area is recommended for confirmation. Any region of osteoblastic activity, including tumor, arthritis, or infection, will be identified on a bone scan.
The staging scheme used for osteosarcoma is the following:

- Stage I: low grade lesion, no metastasis (rare)
- Stage II: high grade lesion, no metastasis
- Stage III: regional or distant metastasis

**Therapy**

When discussing potential treatment options with owners whose dogs have been diagnosed with osteosarcoma, it is important to separate the treatments for location and systemic disease. Treatment of local disease is often the most pressing, as uncontrolled pain causes a decline in the quality of life. The first and foremost treatment option still discussed with owners is amputation, which takes away the major source of the dog’s pain. Many dogs do quite well following surgery, and a significant improvement is often reported post-operatively, as dogs become pain free and adjust to life with 3 limbs. Complete forequarter amputation or coxofemoral disarticulation amputation is recommended for complete tumor excision. While amputation is still considered the standard of care for local control of osteosarcoma, not all dogs are necessarily good candidates due to concurrent orthopedic or neurologic disease. However, many treatment options exist with the goal of palliation of pain and improvement in quality of life.

**Surgical limb spare**

Limb sparing surgery is one such option, if the lesion is of the distal radius or ulna. Limb sparing surgery in other locations is not usually recommended as arthrodesis of the associated joint is required, and arthrodesis of the scapulohumeral, coxofemoral, stifle, or tarsal joints results poor function and a high complication rate. Additional criteria include OSA clinically and radiographically confined to the leg, where the primary tumor affects <50% of bone, no pathologic fracture, less than 360 degree involvement of soft tissues, and a firm/ definable mass (not edematous). Surgical options for replacement of the bone, following tumor excision, include frozen allograft (bone harvested into a bone bank), metal endoprosthesis, pasteurized tumor autograft (excision of the tumor, followed by pasteurization at 65C for 40 minutes, followed by reimplantation), longitudinal bone transport osteogenesis, or ulnar transposition limb sparing of a distal radial OSA². Potential complications of limb sparing techniques include infection, implant failure, and tumor recurrence.

**External beam radiation therapy**

Should limb spare not be an option, radiation therapy has been used with success in palliation of pain for the treatment of OSA. There are no site restrictions, and radiation therapy can be used to treat one or multiple sites in the palliative setting. A bone scan can be recommended prior to treatment to complete staging and to treat multiple sites if indicated. Protocols reported include accelerated, 2-, 3-, and 4-fraction protocols. Overall, palliative protocols provide 50-93% response rates (pain relief) for a median duration of analgesia of 53-180 days.³ Radiation therapy has also been used in a definitive treatment setting. The goal of definitive radiation therapy is complete local tumor control. A median survival time of 209 days for fractionated high dose radiation and chemotherapy for systemic therapy was reported.⁴ Stereotactic radiation therapy has also used to treat canine osteosarcoma. The advantage to stereotactic radiosurgery is the ability to deliver very high doses of radiation therapy to the tumor, while sparing normal tissues. In this report, acute side effects (hair loss, irritation) were mild to moderate, and in a small group of dogs the median survival time of stereotactic radiation therapy with follow up chemotherapy was 363 days. The main complication was pathologic fracture of the treated site.³

**Samarium (Quadramet®)**

Samarium-153-ethylenediamine-tetramethylene-phosphonic acid (¹⁵³Sm-EDTMP) is a bone seeking radioisotope administered intravenously. This treatment is commonly used in people for palliation of metastatic cancer to bone. It is used with success if the ratio of tumor dose to surrounding tissues is favorable – determined using a bone scan. This particular treatment provides pain relief, and in some cases, tumor growth delay. Administered intravenously, it is particularly helpful in cases of multiple bone site involvement, or in cases of other tumors metastasizing to bone. One important side effect to note is transient bone marrow depression following treatment, which will delay chemotherapy. This particular treatment also requires isolation in a radiation facility for 5-7 days before release. ⁴

**Bisphosphonates**

One final treatment option for localized disease and pain control is pamidronate or zoledronate, which are bisphosphonates that inhibit bone resorption by binding to hydroxyapatite crystals, inhibiting further calcium and phosphorous dissolution. Furthermore, they block osteoclastic activity and induce apoptosis of osteoclasts. The result is inhibition of bone resorption. This particular treatment can also target multiple OSA sites and can be used in combination with the above mentioned treatments. Pamidronate and zoledronate are renally excreted and usually administered monthly; renal parameters should be checked prior to each monthly infusion.⁵
Pain control palliation
The last and least aggressive option for treatment of local disease in dogs diagnosed with OSA is pain control alone with a combination of non-steroidal anti-inflammatory drugs and opioids. These can be used alone for those pet owners not wanting to pursue more aggressive therapy or in combination with the other treatments discussed above.

Pathologic fractures
When any of the above limb sparing options fail or disease progresses, amputation is the treatment of choice. However, in some dogs, this is still not a viable option. A recent study reported internal and external fracture repair in dogs with a pathologic fracture due to osteosarcoma or undifferentiated sarcoma of the appendicular skeleton. Some dogs experienced pain relief associated with fracture fixation, and, should amputation be ruled out due to concurrent disease or owner’s wishes, this may provide an alternative for improvement in quality of life.11

Therapy to delay metastasis – chemotherapy and immunotherapy
Chemotherapy is used with the goal of delaying the onset to OSA metastasis. Although not effective at controlling local disease, chemotherapy has been shown to extend median survival time in both amputees and limb spares. Doxorubicin, carboplatin, cisplatin, or combinations of doxorubicin with a platinum agent are those most commonly recommended in practice at this time. One of the most commonly used protocols is carboplatin every 3 weeks for 4 treatments. With local disease control using amputation or limb spare followed by a course of chemotherapy using a platinum containing drug, MSTs range from 8-10 months. In contrast, the prognosis with amputation alone is MST 4-5 months.6-10

Past research has determined that osteosarcoma is an immunogenic tumor; namely, previous work identified that dogs undergoing limb sparing surgery followed by chemotherapy and experiencing osteomyelitis after surgery had longer survival times than dogs treated the same way that did not experience infection post-operatively. This is an exciting new area of research and multiple clinical trials are ongoing investigating the efficacy of controlled immunotherapy in dogs with osteosarcoma.

Prognostic factors
Many prognostic factors are helpful to discuss with owners in regards to potential and expected outcomes following suspicion of, or confirmation of osteosarcoma. The first prognostic indicator is location – while the mandible, orbit, and distal to carpus and tarsocrural joint are considered positive prognostic locations compared to appendicular, other locations including maxilla, rib, scapula, soft tissue, mammary, and vertebral are negative prognostic factors.

Additional negative prognostic factors are stage III disease (metastatic disease), and dogs with lymph node metastasis (MST of 59 days with surgery and chemotherapy compared to a MST of 318 days in dogs without lymph node metastasis). Elevation in alkaline phosphatase (ALP) is a consistent poor prognostic factor in many different studies. A pre-operative elevation in total ALP (>110 U/L) or the bone isoenzyme (>23 U/L) revealed a shorter disease free interval and MST; furthermore, dogs with increased ALP pre-operatively that did not return to normal within 40 days post-operatively failed earlier than dogs with normal ALP due to metastatic disease. In dogs with appendicular OSA, those dogs less than 5 years of age had a shorter survival than dogs older than 5 years when treated with amputation alone. And in OSA of the flat bones, small dogs with complete excision have a better prognosis than larger dogs.

Conclusions
Presentation and radiographs can lead to an increased index of suspicion for OSA, and cytology or histopathology can be used to confirm suspected diagnosis. Overall, important considerations to discuss with dog owners when faced with treatment decisions are the facets of localized disease control and systemic disease control. Local disease control to provide pain relief and quality of life is the most important aspect of treatment. Although amputation is still our current standard many different treatment options are available for pain and tumor control. These options include limb spare (if a distal radial or ulnar lesion), external beam radiation therapy, Samarium, and bisphosphonates. In addition, systemic disease control using chemotherapy should also be discussed to maximize quality of life time. The prognostic factors discussed above can be very useful in aiding pet owners in the decision making process.

References
Mast cell tumors (MCT) are the most common canine cutaneous tumor, representing 16-21% of all cutaneous tumors in dogs. While MCT tend to occur in older dogs, with a mean age of 9 years at diagnosis, they have been diagnosed in dogs much younger. Breeds reported to be at increased risk for the development of MCT are Boxers, Boston terriers, Labrador retrievers, golden retrievers, beagles, schnauzers, pugs, cocker spaniels, Staffordshire terriers, Rhodesian ridgeback, Weimaraners, and Shar-peis. There is no sex predilection. MCT arise from mast cells in the dermis, and the granules of both normal and neoplastic mast cells contain histamine, heparin, and proteases. Thus, the typical MCT are red, raised, hairless dermal masses that may have a history of waxing and waning in size. Bleeding and licking/chewing at the mass may also be a part of the dog’s history. These clinical signs are due to histamine release and resultant erythema, swelling, and pruritus. However, it is important to note that dogs may not have this characteristic history. MCT can have a wide range of appearances on clinical examination, and can easily misdiagnosed as lipomas or other non-neoplastic masses if cytology is not obtained. Also of note is that approximately 10% of dogs with one MCT will have multiple MCT.

**Diagnosis**

MCT are usually diagnosed easily on cytology, which reveals a population of small to medium round cells with deeply basophilic cytoplasmic granules. Infiltration of eosinophils is common and reactive appearing fibroblasts may be present. Mast cells may degranulate, or some MCT have granules that will not pick up the typical Romanowsky stains commonly used in practice, which may complicate the diagnosis. A Wright-Giemsa or toluidine blue stain will often reveal the presence of granules in cases of marginal staining. Additionally, some high grade and anaplastic MCT may not have granules present, and histopathology with immunohistochemistry is necessary in these cases for definitive diagnosis.

**Staging**

If palpable, it is reasonable to consider cytology of the draining lymph node in all MCT cases – even if the lymph node palpates normally, metastatic disease may be present. In cases with negative prognostic indicators – such as location (mucosal, subungual, large or rapidly growing, in a location not amenable to wide surgical excision, recurrent, etc.), full staging may be recommended prior to therapy. If the mass is amenable to wide surgical excision with no determined negative prognostic factors present, the necessity of full staging will be determined based on grade of the MCT post-operatively.

Full staging includes a complete blood count, chemistry panel, and urinalysis to determine overall health status of the dog. A buffy coat analysis to investigate for circulating mast cell has been used in the past, but is not sensitive or specific for mast cell metastasis to the bone marrow and is therefore often not recommended. Fine needle aspiration and cytology of the draining lymph node should be performed if accessible (a normally palpating lymph node may still have metastasis present). Although thoracic radiographs will rarely reveal pulmonary metastatic disease from MCT, they are useful to determine lymph node status within the thoracic cavity and any concurrent conditions or concurrent metastatic neoplasia that may change the pet’s prognosis and therapy. Abdominal ultrasound is performed for evaluation of lymph nodes and abdominal organs and in some cases fine needle aspiration of the liver and spleen is recommended even if ultrasonographically normal, as MCT metastasis may still be present. A bone marrow aspirate to detect MCT metastasis is not routinely performed; it should be considered in cases of visceral MCT or in cases where the index of suspicion for MCT metastasis is high.

**Prognostic factors**

Histologic grade strongly predicts outcome in canine MCT. The Patnaik system has been used historically, with grade I representing low grade and well-differentiated MCT, grade III representing high grade and undifferentiated MCT, and grade II representing a more variable and intermediate differentiation. While most therapy studies in the past have used this particular grading system, the...
variability in the prognosis for dogs with grade II MCT made prediction of the best therapy recommendations difficult. Furthermore, a high percentage of dogs (up to 75%) were diagnosed with grade II MCT and the agreement among pathologists as to grade of the same MCT varied as much as 37%. These confounding factors made counseling clients difficult at best. Further research indicated that mitotic index - number of mitotic figures per 10 high-powered fields (hpf) may be helpful in determining which grade II mast cell tumors were more likely to behave aggressively, with a mitotic index of 5 used as a cut-off.²

A two-tiered grading scheme was developed to improve the agreement of pathologists in regards to grading of MCT and to hopefully improve the clinician’s ability to guide dog owners on the best therapy options. In this system, which utilizes only two grades – high and low – a high grade MCT has at least one of the following characteristics: at least 7 mitotic figures in 10 high powered fields, at least 3 multinucleated cells in 10 hpf, at least 3 bizarre nuclei in 10 hpf, and/or karyomegaly. When investigating survival times in dogs treated with surgery alone and using the two-tiered grading scheme, the median survival time was less than 4 months in dogs with high grade tumors and greater than 2 years in dogs diagnosed with low grade tumors.³

Despite these findings, it is important to note that a small proportion of low grade MCT may behave aggressively. A recent study comparing metastatic rates at the time of diagnosis found evidence of metastasis in 5.8% of grade I (Patnaik system), 16.5% of grade II (Patnaik system), and 14.9% of low grade (Kiupel system) MCT. This emphasizes the importance of using not just grade, but other prognostic factors to determine staging and therapeutic approach.²

Additional testing on biopsy samples can be performed to determine prognostic factors. These include immunohistochemistry for cell proliferation markers, such as Ki67. Determination of c-kit status, with the presence of c-kit mutations indicating higher grade tumors. WHO clinical stage – stages 0 and I have a better prognosis than higher stages. Furthermore, location is a known prognostic factor. For example, subungual, oral, and mucous membrane MCT are associated with higher grade tumors; in addition preputial and scrotal tumors are associated with a worse prognosis. Visceral MCT or bone marrow metastasis is also associated with a worse prognosis. MCT of the muzzle have higher rates of lymph node metastasis, although this may not affect prognosis if treated aggressively. Tumors that are slow growing or present for months prior to diagnosis without rapid change tend to be lower grade, as do MCT in Boxers, while MCT that are rapidly growing tend to be higher grade. Dogs with signs of systemic illness due to MCT (melena, black tarry stools, vomiting, and anorexia) may have a higher grade MCT. The complexity of MCT overall can make staging and therapy recommendations challenging to make. However, basic guidelines can aid in the overall decision making process.

**Therapy**

In general, local disease is treated locally (surgery and/or radiation therapy) and systemic disease is treated systemically (chemotherapy and/or small molecule inhibitors). However, there are instances in which systemic therapy can be used to downstage disease prior to more definitive therapy.

In a dog presenting for therapy for a MCT in a site amenable to wide surgical excision, with no evidence of negative prognostic factors on history or examination, surgical excision with 2 cm margins and 1 fascial plane deep is a reasonable starting point. This procedure will be both diagnostic and therapeutic. If the loco-regional lymph node is palpable, cytology is recommended prior to surgery.

- **Low grade, complete surgical margins:** In this situation, routine follow-up and monitoring for new mass development is recommended.
- **Low grade, incomplete surgical margins:** Consider revision of the surgical scar to achieve complete margins, radiation therapy, or regular follow-up to monitor for recurrence and new masses.
- **High grade, complete surgical margins:** Recommend staging and follow-up chemotherapy.
- **High grade, incomplete surgical margins:** Recommend staging, revision of the surgical scar or radiation therapy, and follow-up chemotherapy.

If a dog with MCT and negative prognostic factors presents for evaluation and therapy complete staging prior to therapy is recommended. The presence or absence of metastasis will aid in developing an appropriate treatment plan.¹ If a dog with MCT has an unresectable mass, consider staging and systemic therapy, including prednisone alone with the goal of shrinking the mass for surgical resection, or any of the systemic therapies discussed below.

While numerous chemotherapy protocols have been investigated in small studies, vinblastine and prednisone is one of the most commonly utilized protocols at this time. Additional protocols are numerous and include the use of CCNU, vinblastine/CCNU/prednisone, and chlorambucil/prednisone among others. When used following surgery to treat Patnaik grade III MCT or MCT with high metastatic risk, vinblastine and prednisone provided a MST of 3 years in one study, and 57% survival at 1 and 2 years in another.³ ⁴

Small molecule inhibitors are another potential systemic treatment options for dogs with MCT. Toceranib, which is FDA approved, is a tyrosine kinase inhibitor active against Kit, vascular endothelial growth factor receptor 2, and platelet derived growth factor receptor beta among others. Mutations in c-kit were found in some higher grade MCT resulting in constitutive activation (activation of the cell surface receptor and stimulation of cell signaling without the appropriate growth factor) and thus toceranib was
studied, and approved, for the treatment of recurrent grade II or III MCT in dogs. The overall response rate in the initial trial was 42.8%.5

References
Radiation therapy is an effective treatment tool for many types of cancers. Ionizing radiation can be administered by an external source, termed teletherapy, and is most often delivered by a linear accelerator. Other, less often utilized in veterinary medicine, delivery options for ionizing radiation are brachytherapy – administration of a radioactive source directly into the tumor, or systemic injection of a radioisotope.

A brief overview of the mechanisms of teletherapy (external beam radiation therapy)

Linear accelerators are most commonly used for external beam radiation treatment. Linear accelerators deliver megavoltage radiation (x-rays and gamma rays that have energy over a million electron volts) which spares the skin and has good penetration. Radiation dose is also evenly distributed through the tissues. Most radiation therapy treatments are based on CT images to provide a 3-dimensional plan and deliver a precise dose to tumors and calculate doses delivered to surrounding normal structures using gross tumor volume and clinical target volume (a margin of predicted microscopic disease). The target of ionizing radiation is DNA - with both direct and indirect damage. A secondary electron results from absorption of an x-ray photon. Then, direct DNA damage is caused by an electron interacting with DNA and indirect damage is caused by an electron interacting with water, producing a free radical (hydroxyl radical) that then damages DNA. Most damage is caused by indirect action.1,2

Total radiation dose is delivered in small doses, called fractions, for a number of reasons. Because tumors have areas within that are not well oxygenated, and oxygen is necessary for radiation to work, delivering small doses frequently will allow less-oxygenated areas of the tumor to become better oxygenated during treatment, and therefore respond. It also allows redistribution of tumor cells throughout the cell cycle, as cells in G2 and M are most sensitive to irradiation. And timing of fractions also allows normal rapidly dividing cells, like mucosa, to repair but theoretically not enough time for tumor tissues to repair. Fractionation into small treatments also decreases the risk of late (and potentially life-limiting) side effects of radiation therapy, including necrosis or secondary cancer formation.1,2

Early (acute) side effects associated with radiation therapy can occur in any normal structure that is included in or near a radiation field. They occur in tissues with rapidly proliferating cells including mucosa, skin, small intestines, and bladder mucosa. These effects usually start toward the mid-to end of the radiation course and subside within 2-4 weeks. Mucositis is the most common side effect associated with radiation of the oral cavity; pain, redness, irritation, and ulceration may occur. Supportive care includes decaffeinated tea rinses, topical pain control and systemic pain control. Oral antibiotics are indicated if secondary infection occurs. A soft, palatable, low salt diet is also helpful. Side effects to the skin include moist desquamation, alopecia, and erythema. Therapy is supportive and prevention of self-trauma is essential for the healing process. An e-collar is almost always required for the 2-3 weeks that side effects are present. Side effects to the nasal cavity can include mucositis and nasal discharge, to the eye blepharitis, blepharospasm, conjunctivitis, corneal ulceration, and uveitis.1,2

Late side effects are those that occur in cells that do not divide often, such as nerves and bone and are usually changes to the connective tissues, stroma, and vasculature. Late side effects typically occur years after radiation and are minimized by administering small doses of radiation more frequently (fine fractionation); coarse (sometimes termed palliative) radiation therapy, which utilizes larger doses given less frequently, will increase the risk of late side effects. Fibrosis, non-healing ulcers, fistulas, cataracts, and secondary cancers are examples of late side effects.1,2

Tumor types treated with teletherapy

In dogs, many oral tumors are responsive to radiation therapy, providing a decrease in tumors size for comfort and quality of life, or sterilizing the tumor bed to delay or prevent tumor recurrence. Acanthomatous ameloblastoma (also called acanthomatous epulides) are locally aggressive tumors without reported metastasis. If surgical excision is not an option or is incomplete, full course radiation therapy has high rates of tumor control – for example, one report revealed a 3 year progression free survival of 86% in dogs with tumors less than 4 cm in size (this decreased to 30% in tumors over 4 cm in size). Oral squamous cell carcinomas also have high response rates to radiation therapy (unless located in the tonsil or at the base of the tongue, where metastatic rates are higher). Oral melanoma are also highly responsive to radiation therapy in the dog. Coarse fraction (higher doses given less frequently) radiation therapy is often used due to the high metastatic rates of oral melanomas, however, the reported response rates are above 90% for local tumor response. Oral fibrosarcomas are less responsive than ameloblastoma, squamous cell carcinoma, and melanoma, and marginal resection prior to radiation therapy will likely improve overall tumor control if complete resection is not possible.3

Nasal tumors are also effectively treated with radiation therapy and in fact is the standard of care. In dogs, nasal carcinoma is the most commonly diagnosed nasal tumor. With no therapy, the reported median survival times is 95 days. Utilizing megavoltage irradiation and CT computer planning, the median survival time in dogs treated with radiation therapy is 11-19 months. In cats,
lymphoma is the most commonly diagnosed nasal tumor followed by carcinomas. When lymphoma is localized to the nasal cavity, radiation therapy yields high response rates and durations and may be combined with chemotherapy to yield median survival times of greater than 1 year. With carcinomas, the median survival time is similar to the dog at about 12 months.\(^4\)

Brain tumors are also effectively treated with radiation therapy, either as a stand-alone therapy or post-operatively. The overall median survival times reported with radiation therapy as the sole treatment range from 1-2 years. Radiation therapy can also be used to treat pituitary macroadenomas, and although neurologic signs associated with a space occupying mass in the brain are expected to improve, the signs of endocrinopathy (hyperadrenocorticism) will likely not.\(^5\)

Additional tumors effectively treated with radiation therapy include mast cell tumors, soft tissue sarcomas, and injection site sarcomas. Radiation therapy can be used as the sole treatment or pre- or post-operatively. When used with surgery, long disease free intervals and excellent local control may result. For example, median survival times of dogs with soft tissue sarcomas treated with surgery followed by radiation are reported at 2270 days. Similarly, low grade mast cell tumors treated with surgery and radiation therapy have greater than 90% tumor control 1 and 2 years post-therapy. Injection site sarcomas in cats are also treated aggressively, with a combination of wide surgical margins and radiation therapy, have extended median disease free intervals compared to surgery alone. Apocrine gland anal sac adenocarcinomas in dogs also have extended disease free intervals when treated with both surgery and radiation therapy; radiation can be used alone in the palliative setting.\(^5\)

**Radiation therapy for pain control**
Teletherapy can also be very effective in providing pain relief, and therefore quality of life, for many veterinary patients. For example, over 90% of dogs with osteosarcoma treated with radiation therapy experience pain relief utilizing a coarse fractionation protocol. In dogs or cats with mediastinal lymphoma, radiation therapy often provides rapid, albeit short-term, response. In these cases, therapy is used to downstage disease and relieve the discomfort associated dyspnea while definitive chemotherapy is pursued. Urinary bladder and prostate tumors may also be treated with either fine fractionation or coarse fractionation radiation therapy to provide tumor control and pain relief.\(^2,5\)

**Evaluation of potential patients for radiation therapy**
Radiation therapy can be very successful in the treatment of many different tumor types, but a thorough evaluation is necessary prior to recommending this treatment. A thorough physical examination can detect abnormalities that may indicate concurrent disease. A minimum database is used to assess the overall health of the dog or cat. Thoracic radiographs will assess for pulmonary metastatic disease in addition to assessing heart and lungs prior to multiple sedation or anesthetic episodes needed for treatment. For some dogs and cats, abdominal imaging may be recommended to assess for metastatic or concurrent disease prior to the time and financial investment of radiation therapy. Palpation and cytology of the locoregional lymph node is also often indicated to assess for metastasis and potential inclusion in the radiation therapy field.\(^2\)

**Conclusion**
Radiation therapy is an excellent tool that can be used in the management of cancer in dogs and cats. When combined with surgery, it is a powerful tool to prevent the recurrence of many cancer types. Alone, it can manage and provide quality of life for long periods of time for dogs and cats with cancer. And radiation therapy can be used in the palliative setting, to provide pain control, and improve a pet’s quality of life.

**References**
\(^3\)Liptak JM and Withrow SJ. In Small Animal Clinical Oncology 5th ed 2013; 381-398.
\(^5\)LaRue SM and Gordon IK. In Small Animal Clinical Oncology 5th ed 2013; 180-197.
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. This makes sense in that while a patient is walking bone is loaded longitudinally; however, in many cases bone fractures occur due to a transverse load.

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 are important as these are the very forces that must be overcome when choosing the appropriate fracture fixation method.

Fractures occur when the sum of the forces to the bone are greater than the ultimate strength of the bone. This 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 interfractionary 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 interfractionary strain has to be <2% and the interfractionary 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.
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.

Physseal fractures are classified by the Salter Harris (SH) classification scheme. SH I fractures are through the physis itself, while SH II fractures are through physis and into metaphysis. SH III fractures are through physis and into the epiphysis and are considered intra-articular. SH IV fractures are through physis 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.

In summary to be able to fix a fracture requires one to be able to correctly diagnosis and classify a fracture. Remembering bone characteristics, biomechanics, and healing all play in decision making for fracture fixation. In terms of classification it is important to describe the fracture with the anatomical location, severity, configuration, displacement, contamination, and if and what type of growth plate fracture may be present. The biggest piece of advice with fracture diagnosis is to take orthogonal radiographs to create the full picture.
In part I we discussed the classification and diagnosis of fractures as well as basic bone healing. 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 not able to do. I will also never commit to a certain type of fixation as my plan may change intra-operatively. 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. “Should I fix this fracture” means 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 (see below) 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.

Kapler M, Dycus DL. A practitioner’s guide to fracture management: Part II: Selection of Fixation Technique and External Coaptation. Todays Veterinary Practice, September/October;23-30, 2015.
External coaptation

External Coaptation is defined as the use of bandages, splints, casts, etc. to aid in the stability and support for soft and osseous tissues. It is useful for the management of wounds, edema reduction, and fracture management. The central theme for any patient with external coaptation is comfort and function. External coaptation for fracture management can be as the primary fixation, temporary fixation, and ancillary fixation. For primary fixation the bandage and splint will be the sole means of fixation where as with temporary fixation the bandage/splint may be used to cover open fractures or stabilize a fracture until definitive surgical correction. Ancillary fixation with external coaptation is useful for additional support such as after bone plating to prevent implant breakdown as seen with distal radius and ulna fractures.

To utilize external coaptation for primary fixation one must be able to stabilize the fracture a joint above and below the fracture. This leaves only fractures that are distal to the elbow or stifle amenable for external coaptation. Thomas-Schroeder splints are never recommended. These are traction devices that are constructed of a wire frame and soft bandage material. The splint does not adequately immobilize the shoulder or hip, and, therefore, is considered contraindicated in humeral and femoral fractures. The advantages external coaptation for fracture management are that it does not disturb the fracture site, there is minimal risk of contamination, no risk of implant break down, it is easy to apply and there may be a decreased expense. However, there are associated costs for frequent rechecks and bandage changes which tend to add up very quickly especially if complications develop. The disadvantages for using external coaptation for primary fixation are that it is limited to fractures that are distal to the stifle or elbow.

External coaptation wont counteract all the forces on a fracture and there is the possible need for eventual surgery if the fracture fails to heal. If there is fracture instability this could result in delayed, nonunions, or malunions. Frequent bandage changes will be needed which will require cost and frequent visits by the owner. Furthermore limb stiffness can occur from prolonged immobilization, which can lead to disuse atrophy and fracture disease. Probably the biggest reason I really don’t like bandages and splints is just by placing a bandage there is a 63% morbidity associated with it, which will lead to costs to the owner and potential delays in fracture healing.1 When determining if external coaptation is an option certain patient factors need to be considered. For example ideal candidates for external coaptation are younger animals with green stick fractures. These are fractures that are often incomplete, minimally or non-displaced and have an intact fibula or ulna which will increase stability. Breed and confirmation are very important as chondrodystrophic breeds and obese patients can be challenging to incorporate an appropriate splint. Patients that have suffered poly-trauma may not be the best candidates, as they may need internal fixation to promote early limb use. Concurrent diseases need to be considered, as immuno-suppressed patients may take longer to heal and thus require longer immobilization. Temperament needs to be considered such as aggression. If the patient is going to require multiple bandage changes then this may prove challenging. Another consideration is patient assessment. Breed is a consideration such that small and toy breed dogs with radial/ulnar fractures are not a good candidates for external coaptation. The blood supply when compared to large breed dogs is decreased and there is a higher risk of complications. In fact in small breed dogs with radius and ulna fractures treated with external coaptation are at an 83% chance of malalignment or nonunion.2-4 Another consideration when choosing external coaptation, as a primary means of fixation is fracture assessment. External coaptation will counteract bending and rotational forces as long as the joint above and below are immobilized. The goal with external coaptation is once the splint is applied orthogonal radiographs need to be taken to asses the fracture. Try to follow the 50/50 rule which states that cortical positioning of the fracture ends should have 50% contact to expect fracture healing. Note that this is for fracture healing to be possible, not probable. The goal should be for 100% reduction. Furthermore, rotational alignment is imperative meaning the joint above and below the fracture is aligned and there is no rotational issue that may lead to angular limb deformities or gait abnormalities or progression of OA. If these goals are not met then external coaptation should not be used and internal fixation should be considered.

External coaptation when used as a temporary means of fixation can help improve comfort and reduced swelling while the patient awaits definitive repair. It can also act as a protective covering with open fractures. No matter how clean your hospital is, hospital bugs are much worse than what the bone was exposed to in the environment. I tend to use extreme caution placing a temporary splint on humeral and femoral fractures, as it is very hard to fully stabilize it. Furthermore, the splint can act as a fulcrum and cause worsening pain or malalignment of the fracture. I tend to keep these in a crate with analgesic relief while awaiting fixation. External coaptation for ancillary fixation is designed to add additional support. I tend to use this with splint management following radius/ulna fractures. Along with additional support it will also help minimize cycling of the implants to help prevent premature breakdown.

The quick and dirty technique for external coaptation is to sedate or anesthetize the patient for fracture reduction and alignment. I prefer to use custom made fiberglass splints rather than preformed plastic ones as I feel that the comfort is better improved if the splint follows the patient’s anatomy. The splint is typically applied to the lateral aspect for the hind limb and either the lateral or palmar aspect for the front limb. Remember to splint a joint above and below the fracture. It’s important to provide enough padding to prevent pressure sores and movement. Leave the middle two digits exposed. To encourage weight bearing and to minimize trauma to the articular cartilage from prolonged immobilization it is important to splint them in a functional standing angle. As has been previous mentioned take orthogonal radiographs after splint application to evaluate the reduction and alignment.
In terms of radiographic healing ideally we will consider it completely healed with disappearance of the fracture line. Technically we want 3 of the 4 possible cortices to show evidence of bridging before returning the patient to normal activity. Ideally, within about 5-7 days there will be slight widening of the fracture gap; this is normal as the bone tries to maintain interfragmentary strain. There may be some evidence of “smudging” of the fracture edges. Around 10-12 days we can see the early stages of a bony callus beginning to form. In some cases around the 30-day mark there is the beginnings of disappearance of the fracture line, then around 90 days there is complete healing and remodeling of the callus. This is the time point when many can return to normal activity.

Complications with any type of fracture fixation or implant as much as they suck, happen to everyone, even the best. The big 3 are infection, implant failure, and poor bone healing. The easiest thing to do is blame someone else. Don’t be quick to blame the owner, the dog, or the particular plate. Many times the reason for the issue is standing right in front of you if you were to look in a mirror.

Infection can occur due to hematogenous spread, direct inoculation from an open fracture or surgical contamination or less commonly direct spread from a focal soft tissue infection. In the acute phase after a bone has fractured the vascular channels are comprised which results in ischemia. Bone ischemia is a major predisposing factor for osteomyelitis. Around the bone ischemia is a reactive hyperemia that is associated with an increase in osteoclast production. Along with increased osteoclast production there is also periosteal irritation that leads to periosteal reaction. The aggressiveness of the infection is noted to parallel that of the periosteal reaction seen on radiographs. The damage and ultimately the response to treatment of bony infections are dependent on the viability and stability of the fixation, the virulence and antibiotic sensitivity of the organism and the condition of the soft tissue envelope. The most common type of osteomyelitis is from direct inoculation, which is also known as post-traumatic osteomyelitis. The staph species dominate with \textit{S. intermedius} being the most common. Some gram-negative bacteria can be associated with osteomyelitis. Fungal organisms are usually due to hematogenous spread. For an infection to occur the bacteria must contaminate and colonize the bone and surrounding tissues. Its important to note that a stable fracture will heal in the face of infection, an unstable fracture will not heal in the face of infection and will perpetuate the persistence of infection.

Poor bone healing is broken into delayed union, non-union, or malunion. Delayed union is healing of a bone that takes longer than expected to heal. The normal healing time frame for a bone is 8-12 weeks versus a nonunion, which is where the bone fails to heal regardless of healing time or if a delayed union is not addressed. A malunion is characterized by a healed fracture in an improper alignment. This may be noted as shortening of a limb, malalignment of the joint surfaces, rotational abnormalities, or varus and valgus deformities. Nonunions are further broken down into viable and nonviable. Viable nonunions can be classified as hypertrophic where there is considerable callus formation but no bone healing, this is sometimes referred to as an “elephants foot”, or it can be moderately hypertrophic where there is a lesser degree of callus known as a “horses foot”. Both types of hypertrophic viable nonunions are typically caused from motion in the fracture site; therefore more rigid fixation is needed. An oligotrophic viable nonunion is hard to distinguish from a nonviable nonunion due the fact there is no radiographic evidence of healing. Its cause is due to lack of cellular activity which are typically due to loose implants in the area of the fracture such as loose cerclage wires. Nonviable nonunions can be classified as dystrophic where there is nonviable bone on either side of the fracture, necrotic where there is an infected section of bone such as a sequestrum, defect where there is a gap at the fracture site that is too large to heal, or atrophic where there is removal of dead bone by the host with no healing and often times resorption of the bone.

References

The decision in choosing internal versus external fixation is dictated by many factors that have been discussed in parts I and II. After considering the various factors such as the fracture configuration/classification, patient, client, and veterinarian factors then remember to run through the below chart in deciding on external fixation (as discussed in part II) versus internal fixation. If internal fixation is chosen then the decision has to be made as to which type.

### Invasiveness & Stability

<table>
<thead>
<tr>
<th>Least invasive &amp; Most unstable</th>
<th>Primary Fixation</th>
<th>Ancillary Fixation</th>
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<tbody>
<tr>
<td>External Fixation</td>
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<tr>
<td>External coaptation (cast or splint)</td>
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<table>
<thead>
<tr>
<th>Internal Fixation</th>
<th>Lag screws</th>
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<tbody>
<tr>
<td>IM pin and/or K-wires</td>
<td></td>
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<tr>
<td>External skeletal fixator (ESF)</td>
<td>Full cerclage</td>
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<tr>
<td>Interlocking nail (ILN)</td>
<td>Hemi cerclage</td>
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<tr>
<td>Bone plate and screws</td>
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</table>

**Note:** Even though ESF and ILN are listed above bone plates and screws, the last 3 primary fixation methods offer complete stability depending on the fracture configuration, however, in general, ESF and ILN are less invasive than bone plates and screws.

Kapler M, **Dycus DL**. A practitioner’s guide to fracture management: Part II: Selection of Fixation Technique and External Coaptation. Today’s Veterinary Practice, September/October;23-30, 2015.

Internal fixation is ideal for allowing early return to function, maintenance of joint function, and counteracting all the forces on the fracture. It is especially important for fractures that are subject to compression, shearing, and tension as well as comminuted or long oblique fractures. Also, if a fracture could be treated with external coaptation can’t be reduced appropriately by following the 50/50 rule then open reduction and internal fixation (ORIF) should take place. Remember the 50/50 rule states that if 50% of the fracture ends must be in contact for fracture healing to be possible not probable. So the long skinny on internal fixation is to hold the fragments rigidly until they are healed while allowing the patient to move the limb and bear weight. The health of the limb, and the joints surrounding the fracture, is optimized when the muscles continue to function and joint motion is able to maintain cartilage nutrition. Bone will heal; however, maintaining joint function is paramount so don’t get tunnel vision on just the fracture.

However, the advantages of internal fixation over external fixation don’t go without some considerations. First it requires a surgical approach, which results in increased tissue damage. Fragment manipulation may prolong healing. Years ago where the carpenter approach was used there was significant fragment manipulation, which affected healing. With newer approaches and considering the gardener approach there is less fragment manipulation and thus we see quicker tissue healing. The implants do remain inside the body and thus can potentiate infection. Patients with implants are at risk for a surgical site infection (SSI) up to 1 year after surgery, and some may even develop infections longer than that. The advantages and disadvantages of the various approaches must be balanced to optimize an individual patients fracture, so remember there is no “orthopedic cookbook” of one size fits all.

When considering the types of internal fixation to utilize, several factors need to be considered. We have discussed various factors when approaching fractures and many of those factors still play a role when deciding on the type of internal fixation. We need to consider the fracture classification (think part I of this series), along with the bone affected, any concurrent injuries, and the forces acting on the bone. Furthermore, several important questions need to be asked: can we reconstruct the shape of the bone, if so then can the bone share the load of the implant, or will the implant have to bear the load of the weight?

**IM pin/Cerclage wire**

Intramedullary Pins (aka IM Pins) are typically smooth round 316L stainless steel rods. Steinmann pins are most commonly used and they are available from 1/6 to 1/4 of an inch in diameter. They come in either trocar or chisel points and the end may have threads. Note that the threads do not improve holding and in fact have a tendency to fail prematurely because the thread shaft interface creates a point of weakness. K-wires appear just as Steinmann pins but their size if much smaller ranging from 0.035-0.062 inches in diameter. IM pins do a great job of resisting bending forces, but are very poor at resisting rotational forces. They can be used as a primary or ancillary fixation with external skeletal fixation or bone plating. They should never be used alone as the only source of fixation. If using an IM pin for primary fixation then cerclage wires should be added. Cerclage wires are designed to counteract axial and rotational forces while providing interfragmentary compression. Cerclage wires do not damage the blood supply or interfere with healing unless they become loose. Cerclage is French for encircling hoops placed around wooden barrels. It is essentially orthopedic wire that is placed around bone. There are various sizes available from 22 gauge, which is used for small dogs and cats up to 18 gauge, which is used in large dogs. In general cerclage wire is used in conjunction with other fixation methods especially with IM pins. It should only be used alone in non-weight bearing bones such as the mandible. **Cerclage wire when used appropriately does not**
damage the blood supply. The major blood supply is the medullary artery, which is not suppressed by the wires; furthermore, the periosteal arterioles are not blocked by cerclage wires. The wires do not block venous efflux as they have very minimal contact with the bone. Also, cerclage wires don’t interfere with healing unless they become loose or have been applied inappropriately.

The advantages of IM pin and cerclage wire fixation are maintenance of axial alignment with resistance to bending and rotation. In some cases there may be less cost to the client and it is technically easier than some of the other internal fixation methods. The disadvantages as with any internal fixation are it requires an open surgical approach, as previously mentioned there is a high likelihood of failure if the IM pin is used without cerclage wire. There is limited control of axial loads. Also, there is a risk of pin migration or implant breakdown.

IM pin and cerclage wire fixation should only be considered and used with long oblique or spiral fractures of the humerus, femur, or tibia. The IM pin should be placed into the medullary cavity of long bones using either a drill or Jacobs chuck. It can be placed either normograde or retrograde. If using and IM pin for primary fixation the goal is to use an IM pin that is 70% of the medullary canal. If using the IM pin for ancillary fixation for example with a bone plate then the goal is to use an IM pin that fills about 35-45% of the medullary canal. Normograde IM pin insertion involves reducing the fracture first and starting the pin at one end of the bone. The IM pin is then driven across the fracture line and the pin is seated into the opposite bone segment. This is the preferred method in the femur and tibia to avoid injury to the sciatic nerve or penetrating the joint. Retrograde IM pin insertion involves starting the pin at the fracture site, driving the pin out one of the fracture fragments, reducing the fracture, then driving the pin back across the fracture and seating it into the opposite segment. When using cerclage wires a minimum of 2 should be used. They should be placed approximately 1 cm apart beginning at least 0.5 cm from the beginning and end of the fracture line. It is very important that the cerclage wire be placed perpendicular to the bone; otherwise they will not become tight. The cerclage wires must be tight to provide adequate interfragmentary compression. When placing cerclage wire don’t entrap soft tissue. In conical shaped bone such as the tibia a notch in the bone can be placed or a k-wire can be used to create a hemi-cerclage wire. As you twist pull the wire away from the bone, which will create equal tension to ensure an interlocking twist. Cutting the twist cerclage in and of itself will reduce the wire tension by about 21%, be sure to leave at least 3 twists. After cutting the cerclage wire don’t bend it over, this will reduce the wire tension by 70% and set up a scenario for wire loosening.

When using IM pin and cerclage wire we want to avoid the articular surfaces; when using this fixation in the tibia we also want to avoid damaging the insertion site of the cranial cruciate ligament. Never place an IM pin into the radius; there is a high likelihood of joint penetration into the carpus or articular surface of the radial head. Also, avoid stack pinning (placing multiple small pins to replace one large pin) as this does not provide any additional rotational support. Complications of IM pin and cerclage wire fixation are improper placement where damage to the joints or soft tissues occur, specifically sciatic entrapment. There can be implant failure or migration. If there is abnormal rubbing of the soft tissues then a seroma can form. Continued instability of the fracture site will lead to delayed or non-unions of the fracture site. Improper case selection is also consideration when complications arise. Placing an IM pin and cerclage wire for a transverse, short oblique, or radial fractures is a guaranteed bet for a complication. Other complications such as using undersized wire use of a single wire along with technical errors such as insufficiently tightening or bending over wires. Ensure wires are placed perpendicular to the bone. Inadequate fracture reduction will also increase chances of implant loosening and complications. Failure to check all wires is very important as many times once a second wire is placed the first wire will become loose.

External skeletal fixation (ESF)

ESF involves transcutaneous placement of threaded pins or wires into bone, which are secured by clamps, rods, rings, or epoxy. Linear ESF use transfixation pins or K-wires that are attached to a linear connecting bar using a clamp. Pins can be placed in one of two ways, either half pins or full pins. Half pins penetrate the skin on one side but go through both cortices of the bone while full pins penetrate the skin on both sides and go through both cortices of the bone. The classification for linear ESF is described as Type I through Type III with a few subtypes. The strength of the ESF is stronger as the types increase. A type Ia is considered unilateral and uniplaner with 1 connecting bar and a half pin. A type Ib is considered unilateral but biplanar such that two type Ia ESF’s are 90 degrees to each other. A type Iia is considered bilateral and uniplaner consisting of 2 connecting bars and full pins. This differs from a type Iib in that there are 2 connecting bars but a combination of full and half pins. A type III ESF is the strongest of the linear ESF in that it is a combination to type I and type II. It is considered bilateral and biplanar consisting of 3 connecting bars and a mix of full and half pins.

ESF is diverse in its use in that it can be applied in a variety of fracture scenarios. It is especially useful in patients with open contaminated fractures. It will allow for concurrent wound management, it keeps implants away from the fracture site, and the implants are removed after the bone has healed thus allowing resolution of infection. Furthermore, ESF is able to counteract all the forces acting on a fracture. They can be used as either primary or ancillary stabilization. It can be used with an IM pin to help control rotation.
The advantages of ESF are that it may be applied in a closed manner thus avoiding disrupting the fracture hematoma and soft tissues. It is good for most fracture configurations being that it is versatile and well tolerated. They are able to be destabilized over time to allow the healing bone to adapt. ESF are relatively easy to apply and remove. There are numerous configurations and types so if you like to construct things then ESF is for you. The disadvantages are pin loosening that happens very frequently, pin tract sepsis, limited function of soft tissues if the pins are passed through large muscle groups. The biggest issue with ESF is the postoperative care of the frame, which will require work on the owner’s part.

When using ESF the goal is to use pins that are about 25-30% of the bone diameter. The pins are inserted through stab incisions and should penetrate both the cis and trans cortex. The minimum to have a stable ESF is to use at least 2 pins above and below the fracture. My mentors always told me that 3 is better and 4 is the best. When inserting the pins try to avoid the fracture line, nerves, and vessels. Additionally, minimize muscle penetration to cut down on post-operative drainage. When inserting the pins use low speed power insertion to reduce thermal necrosis. Using power insertion will reduce the wobble versus when inserted by hand. Thermal necrosis is a huge contributor to pin loosening. Once finished bandage the frame postoperatively to prevent it from damaging the owners home. Scrub sponges can be used to help with bandaging and they will have some chlorhexidine residue on them. They can also be placed in the freezer and used as post op cold packs around the frame. Don’t use smooth or negative profile pins with ESF. If you must use smooth pins they should be inserted at a 70-degree angle to improve holding power. Negative profile pins have a weakness in the pin at the thread pin interface since the threads are cut into the shaft of the pin, thus the diameter of the threaded region is smaller than the diameter of the rest of the pin. I try to use positive profile pins with ESF. These pins have the threads rolled onto the diameter of the pin, such that the diameter of the pin in the threaded region is the same as the rest of the pin.

**Bone plate and screws**

In the simplest terms using a bone plate and screws involves securing a bone plate to the bone via screws. Depending on the plate type the material may be stainless steel, titanium, etc. There are two big categories of bone plates: locking or non-locking. Bone plates and screws resist all forces a bone undergoes, but is weakest in regards to bending. Because of this the plate is placed on the tension surface of the bone. Non-locking plates allow the plate to be held in close contact with the bone by the screws. Once the patient begins to walk the axial load through the bone creates a shearing force at the screw-bone interface. This shearing force is counter-acted by friction generated at the plate-bone interface. Therefore, for non-locking plates to provide the best stability they must be contoured and applied directly to the bone with no soft-tissue in-between. Locking plates are considered a fixed angle system and behave more like an ESF. It does not rely on the friction between the bone and plate. Rather, the axial force the patient creates when walking is converted to and creates a compressive force at the screw-bone interface. In theory, locking plates are stronger and stiffer. Also, they do not have to be contoured to the bone. Up to 2 mm offset is considered acceptable which may improve biologic osteosynthesis.

Bone plates can be used in 3 primary ways. A neutralization plate is such that the fracture can be anatomically reconstructed and the bone/implant will share the load of the weight. Bridging or buttress plating is where the fracture can’t be anatomically reconstructed and the bone does not share the load, the implant must withstand the forces. Bridging and buttress are commonly used interchangeably; however, buttress plating is technically reserved for metaphyseal fractures to keep articular surfaces from collapsing while bridging plating is reserved for diaphyseal fractures that can’t be reconstructed. Compression plating is used with specific plates called DCP or LCP plates. Compression plating can be used when the fracture can be anatomically reconstructed to enhance stability.

The two basic screws are cortical and cancellous. As the name implies the screw is designed for the type of bone the screw is placed in. Cortical screws have a smaller pitch and less depth to the thread, which help with engaging the dense cortical bone. Cancellous screws have a larger pitch and more depth to the thread, which are designed to engage the spongy cancellous bone. Furthermore, screws can be self-tapping, or non self-tapping. Self-tapping screws have cutting flutes on them, which cut the thread into the bone and are designed to speed insertion of a screw. While nice to have, they do have less overall surface area of the bone-screw interface so the flutes have to be driven at least 2 mm past the trans cortex. Non self-tapping screws must have the threads cut into the bone prior to inserting the screw. Furthermore, the screw diameter used should be about 25% of the bone diameter. Ideally, one must engage at least 5 cortices on either side of the fracture but try to shoot for 6. Exceptions to this rule can be made on rare occasions such as with young dogs, ilial fractures, or with use of locking plates.

The advantages of bone plates and screws are that they counteract all forces, typically allow early return to function, they can be placed in a minimally invasive way and they come in a variety of sizes and configurations so that most fractures are amenable to bone plating. The disadvantages are that it requires open fixation, there is the risk of infection and thus implant removal. Furthermore, there can be implant breakdown, as well as the cost of the implants and the cost to the owner.
Traditional Chinese Veterinary Medicine (TCVM), although relatively new to the Western world, is a medical system that has been used in China to treat animals for thousands of years. It is an adaptation and extension of Traditional Chinese Medicine (TCM) used to treat humans. Speaking broadly, Chinese Medicine is a complete body of thought and practice grounded in Chinese Daoist philosophy. Though it can be traced back over two millennia in recorded history, it, like any medical system, continues to evolve today, and current research on acupuncture and herbal medicine is beginning to shed light on its mechanism of action.

Chinese medicine theory

Chinese Medicine is based on the Daoist worldview that the body is a microcosm of the larger, surrounding universe. As such, the cosmic laws and forces that govern the external world also govern the body’s internal environment. Just as life-energy or “Qi” is an innate force of the universe, it too is a fundamental force of the body, driving its every action and transformation. Yin-Yang theory, which is central to Daoist philosophy, also features prominently in Chinese Medicine. This theory describes how opposing forces of the universe - light and dark, hot and cold, etc. - mutually create and transform each other, and play a key role in the characterization of physiological function and disease.

The Ancient Chinese observed yearly cycles through five seasons – spring, summer, late summer, autumn, and winter, which they corresponded to the Wu Xing, or Five Elements, consisting of Wood, Fire, Earth, Metal, and Water. Just as the Earth cycles through these five seasons, the body, too, passes through the five phases in its own life cycle. In this way, a young pup is said to be in its Wood (or spring) phase of life, while an old mare is said to be in its Water (or winter) phase. Moreover, the bodily organs have also been mapped to the five phases, and the Five Element Theory is used to explain the functional relationships between organ systems. For instance, the Kidney, corresponding to the Water element, is the “mother” of the Liver, a Wood element organ, because Water generates Wood in the way that watering a tree makes it grow.

Disharmony and disease

In Chinese Medicine theory, disease is understood as an imbalance in the body, and diagnosis proceeds through identifying the underlying “pattern” of disharmony. Pattern diagnosis differs from conventional Western medical diagnosis in that it takes into account not only disease signs but how these signs relate to the individual patient. Thus, TCVM practitioners will consider the temperament, sex, age, activity, and environment of an animal along with the animal’s particular disease signs. This approach stems from the belief that the body is as an interconnected system of forces and functions so that disease and disharmony must be examined with respect to the whole patient. For this reason, Chinese Medicine is often regarded as more holistic than conventional Western Medicine.

The four branches of TCVM

Once a particular type of disharmony or disease pattern is identified, treatment often proceeds through a combination of treatment modalities. Though the terms Chinese Medicine and acupuncture are often used interchangeably in the West, acupuncture is actually only one modality or “branch” of TCM and TCVM. There are actually four branches of TCVM – Acupuncture, Herbal Medicine, Food Therapy and Tui-na (Qi-gong, a form of Chinese meditative exercise, is a fifth branch of TCM that is excluded from TCVM because it cannot be performed by animals).

1) **Acupuncture** is a treatment that involves the stimulation of points, typically achieved through the insertion of specialized needles into the body. Acupuncture points typically lie along the body’s Meridian Channels along which Qi flows. Most veterinary acupuncture points and Meridian lines are transposed to animals from humans, though knowledge of some “classical points” defined on particular species have been retained and are used to this day.

2) **Herbal Medicine** utilizes herbal ingredients listed within the Chinese Herbal Materia Medica in particular combinations or formulas to treat particular disease patterns. Herbal formulas are administered orally and are typically given in powder form to horses and other large animals and in tea pill or capsule form to cats and dogs.

3) **Food Therapy** is the use of diet to treat and prevent imbalance within the body. It utilizes knowledge of the energetics of food ingredients to tailor diets for individual animals.

4) **Tui-na** is a form of Chinese medical massage in which different manipulations are applied to acupoints and Meridians to promote the circulation of Qi and correct imbalances within the organ systems.
“Integrative” medicine: TCVM and Western veterinary practice
TCVM is often viewed as a form of complementary therapy, and is best when used in conjunction with Western Veterinary Medicine (WVM). Both TCVM and WVM have their own strengths and weaknesses. TCVM is a holistic approach that is suited to assessing the well-being of the whole patient, and treatments are generally non-invasive with few side effects. However, TCVM lacks the tools necessary to pinpoint illness to specific disease-causing agents like pathogenic bacteria or viruses, and treatments are better suited for chronic conditions than acute ones. On the other hand, WVM utilizes the tools of modern science to diagnose disease with great precision, and Western drugs and procedures are powerful and fast acting. However, its insistence on detailed diagnosis may come at the expense of getting the larger picture. In many ways, TCVM and WVM each has what the other lacks. Thus, the best medical system involves the integration of the two systems, so that the strengths of one can compensate for the weaknesses of the other.

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Matt Brunke, DVM, CCRP, CVPP, CVA
North County Veterinary Referral Practice
Glen Falls, NY

Kinesiology taping was developed by Dr. Kenzo Kase in Japan during the 1970’s. Although it has been around for over 40 years now, it is just emerging as a modality for canines and felines. It has been used in people and horses, and with the advent of more effective tape it can be used on small animals.

Kinesiology tape is completely different from traditional types of sports tape in the fact that it stretches along its length (but not across its width), allowing it to contour around body parts and allows joints to move through a full range of motion. Traditional tapes have been used primarily to limit movement, but the goal of using kinesiology tape is to promote proper movement.

Most tape is woven from a blend of cotton and nylon fibers in a pattern that allows it to stretch 180% along its length, but not at all across its width. It uses an acrylic, latex-free, essentially hypoallergenic medical adhesive on one side with a paper backing that allows it to be applied by practitioners without touching the adhesive.

Tissue decompression has two primary effects on the body. First, it relieves pressure from the free nerve endings in the tissues that are responsible for nociception (pain), so it can immediately reduce perceived pain. Secondly, the decompression action of the tape allows better circulation to and from the area taped.

The second major effect of kinesiology taping is the stimulation it provides to the variety of sensory nerves in the skin and underlying tissues. The skin and the connective tissue beneath it is filled with sensory receptors that are responsible for feeling light and heavy touch, ne point discrimination, pain, temperature and pressure. Additionally, some of these receptors serve a proprioception role, meaning they contribute to the brain’s sense of where the body’s parts are in space and throughout movement.

With kinesiology tape there is an alteration of the afferent signals going from the taped area to the brain. As a result, this changes the brain’s response to the incoming information, altering the efferent signals returning to the taped area. This neurological effect of taping is responsible for many of the beneficial effects of using kinesiology tape.

Contraindications
- Ingestion of the tape – potential GI obstruction
- Local infection/open wounds of the area
- Tumors
- Cardiac decompensation

Taping for pain mitigation
Goal
To provide adjunct pain relief, after other modalities such as laser therapy, joint mobilization, massage, etc..

Procedure
A three step process is used. 1) Stretch the body part, 2) apply a stabilization strip and 3) apply a decompression strip.
- Apply maximal pain free stretch to the body part to be taped. Apply an anchor end with no stretch. Then mild stretch in a longitudinal direction, again anchoring.
- The decompression strip is applied perpendicular to the stabilization strip. This is applied with moderate stretch in the center of the tape, and then with minimal stretch in either direction.
- For stifles and shoulders, flexion of the joint is needed for initial application

Taping for inflammation/Edema
Goal
To reduce inflammation from affected area as quickly as possible, enabling faster recovery and return to function.

Procedure
Cutting strips of tape into “fingers” or “tentacles” to provide space for fluid to leave the swollen region. Creating a cross-hatch or lattice design may be needed. Work circumferentially around the area when possible (as with distal limbs). An anchor strip around the tentacle strips may be needed.

Taping for neurosensory awareness/Posture
Goal
To improve sensory awareness, improve body posture.

Procedure
Application of tape along the dorsal surface of affected metacarpal and or metatarsal areas with mild stretch. This can provide a non-painful incentive to place the foot appropriately.
Additionally, “connecting the dots” can be done and taking tape to mimic the path of the nerve that is in dysfunction

As with any procedure, proper safety is advised. Having an assistant appropriately restrain a patient may be needed to avoid injury to the rehab practitioner.

Depending on the reason for taping, kinesiology tape should be left on 12 hours – 3 days. Replication for some patients will be necessary.

- Using a waterproof tape is useful for those patients then undergoing underwater treadmill therapy.
- If a patient licks or chews at the tape, an Elizabethan collar is recommended.

To remove the tape, gentle rolling of the tape in the direction of the hair can be done. Ripping the tape off will not be pleasant to the animal.

- Rubbing alcohol or acetone may be needed to release the acrylic adhesive.

Repeating palpation of sore areas to determine the effectiveness of the tape is advised.

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Elbrond, VS, Schultz, RM, Myofascia - The unexplored tissue: Myofascial kinetic lines in horses, a model for describing locomotion using comparative dissection studies derived from human lines... Medical Research Archives, 2015, Issue 3


The “down dog” can arise (pun intended) from many causes. Some are orthopedic (bilateral CCL rupture) but the majority are neurologic in nature. They can be further broken down into acute and chronic conditions.

Acute down dog examples are IVDD, FCE, tick paralysis, trauma and metabolic conditions. Chronic origin examples are lumbosacral disease, degenerative myelopathy and diskospondylitis.

A thorough history and physical examination is a requirement to further understand these patients. A minimal data base (CBC/Chem/UA, plus Thyroid or Urine Culture if indicated) should be done on all patients. Survey radiographs can be extremely helpful in providing practical information. Referral for advanced consultation, imaging (CT/MRI) or other studies (nerve conduction) and surgical intervention (if indicated) may also be needed.

The rehabilitation practitioner may encounter these patients in the peracute setting, or after specialist intervention. Regardless of timing, they must be able to provide a baseline of practical and safe care to assist the patient. General practitioners can also provide a standard of care, and should also be aware of referral to a rehabilitation practitioner (CCRP or CCRT) or boarded rehab specialist (ACVSMR).

Establishing a baseline and then proper communication with the owner is vital. These cases can be “down” for 1-2 days or 1-2 months, or permanently. Understanding prognosis, as well as financial and emotional commitment to these patients is critical. The author recommends working in 2 week intervals, and not committing anyone to a 3 month timeframe. Continuous reassessment and communication is vital.

The team approach to these patients is critical. Establishing a relationship with the patient and the owner will allow for better understanding of the case, and of each patient’s needs. Some cases may be managed as outpatient, while some may require inpatient care. With outpatient, it is recommended to have the patient left for the majority of the business day, so as to slowly work with them. Inpatient care should be discussed and the owner made aware of who will monitor the patient overnight, and care for them on weekends.

Fundamental nursing care is essential for these patients. Changing recumbency every 4 hours (and attempting to keep sternal during daytime) is vital for both physical and mental well being. Clean soft bedding, with appropriate padding (to minimize risk for pressure sores) is needed.

Progressing to standing, items such as carts, harnesses or hoists can be quite useful to save the staff members. Working through assisted standing, partial assistance and then eventually standby assisted standing exercises will depend on each individual patient.

In between standing exercises (done 4-6x a day, or more) the patient should rest. Additional modalities to be used when the patient is resting includes, but is not limited to: LASER, passive range of motion, massage, thermotherapy, cryotherapy, and electrical stimulation.

Bladder and bowel expression may be needed. If so, it should be done in a safe manner. The author recommends avoiding urinary catheters (indwelling or temporary) after the initial 72-96 hours, so that determinations can be made on patient progress. Additionally, catheters provide an access point for infection. Urinary medications (phenoxybenzamine, bethanechol) should be used carefully, and after ruling out any infection.

Bowel expression may needed as well. This can be done in a variety of ways, including manual emptying. The important thing to remember is to give the patient TIME (in a harness, hoist, etc.) to void on their OWN. The author has found that many clinicians and especially owners are not patient enough to give these patients the time they need to void on their own.

For bowels, enemas, oral lactulose and movement (walking) can help facilitate proper bowel movements.

Referral from a general practice/ER to either a rehab practitioner or boarded sports medicine-rehabilitationist should be considered early for these challenging cases. The large breed dogs themselves present unique problems that may not be readily fixable in most practice settings.

Progress can be made with these patients slowly and with proper communication to the owner, reasonable goals can be set and attempted to meet.

Selected references
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Therapeutic Exercises in Veterinary Rehabilitation
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Therapeutic exercises are the real “meat and potatoes” (or tofu and potatoes for you veggie folks) of veterinary rehabilitation. They consist of a variety of focused exercises that are intended to mimic real life work. Consider this to be the occupational therapy aspect of vet rehab.

The goals of these exercises are:
- Improve active pain free ROM
- Improve muscle mass and strength, balance, performance of daily function, aerobic capacity, prevent further injury.
- Reduce weight (when indicated) and lameness
- Important method to best return of function

Remember that prior to starting any exercise, signalment, history and a complete physical examination (including assessment and plan) MUST be done. Certain patients will have limitations that you need to be aware of. This is not a “cookie cutter” approach.

You can be creative in mimicking activities, for example, if you have agility equipment available, then setting up an easier course and having the dog WALK through that is building up exercise. Cavaletti rails, leashes, weave poles, balance boards are just some of the tools that can be used for ther ex.

Remember to use your environment as well.
- Stairs (traction, not scary)
- Couches (cushions)
- Air Mattresses
- Work outside (hills, sand dunes, tall grass, snow)
- Owner limitations? Have them sit at the table

I start with introducing myself to the patient, and not being in a rush. I find it easiest to start with the “down” patient (whether from orthopedic or neurologic issues) and progress through these exercises to the fully ambulatory patient that is need of fine tuning.

For those down patients remember the fundamental aspects of down dog care (clean, dry bedding, changing recumbency, etc.)

For completely down dogs we start with assisted standing exercises. Goals here are to: strengthen the patient, aid in proprioception, improve circulation and respiration, give them a chance to eliminate, are good for their mental well-being. Maximal assistance is needed to provide support 75-100% of the patient’s body weight. They cannot independently stand, and require a team effort. Place feet appropriately and use a sling, towel, Help Em Up Harness to achieve a “normal” position. Adjust for tolerance, but start with 20-30 seconds, per stand, 15 reps per set, 2-4x a day. Slowly increase the standing from 30 second to 5 minutes per session, pending how the patient improves.

Active assisted standing exercises follow, they get stronger let them do more, requiring <75% effort from us. Just enough support to maintain standing, physiorolls great for this, as are carts, hoists, etc.

Standby assisted standing exercises follow. Now has strength and motor to support against gravity, but are still ataxic or weak. You are right by their side, only there to prevent a fall. Once they can achieve rising and holding upright on their own, they may be ready for ambulation. Remember that during ALL of the phases of standing exercises you are doing proprioceptive training. That means that EVERY standing exercise the feet are placed appropriately, providing sensory feedback to the CNS.

For those patients that did not lose ambulation, this is where most of them come in. Proprioceptive training starts with the patient standing independently – time to do it right. Exercises here include 1) weight shifting, 2) unloading of a limb, 3) balance board and 4) exercise balls and rolls. Sit to stand and sit to down to stand exercises with good form are a great home exercise at this time. The patient is actively participating in their recovery.

Dynamic ambulation (aka WALKING) comes next. All walks must be on leash, with adult supervision. The pace must be dictated by the patient, but the handler must encourage the pace (not out smelling the roses, think power-walking). The handler may need to adjust their stride, we want walking, not running. This means for small breed dogs that people must walk extremely slowly, allowing the patient to use all four legs. Otherwise they will run to catch up, and not weight bear and strength train on the limb (think about your FHO patients).

As the patient improves, variety can be taken with different exercises, based on the patient’s needs and goals. This can include:
- Egg-crates, Foam Rubber, Air Mattress
- Couch cushions (shifting balance)
- Stairs – 5-7 steps, 2-4x a day for starters (on leash)
- Pole weaving, tunnels, pulling weight
- Ankle weights
- Syringe cap (on contralateral foot)
- Cavaletti rails

Start with 3-4 simple exercises per session, and always introduce it to the patient, then the owner, before having the owner do it at home. If the patient is improving, increase EITHER the time to a particular exercise by 10-20% each week.

If they are painful? Pause, address, start back up slowly. It may be related to their surgery or be a consequence from their lack of ambulation for a prolonged period of time. Objective outcomes are key, so re-measure – girth, stance analyzer, goniometer. Smartphone apps can be used by owners to track how far/fast they are walking the dog.

A practical, multimodal approach to therapeutic exercises will result in a better patient.

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My dog doesn't have Obamacare and can't wait for the government to make up its mind: What can I do about the torn cruciate?

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My dog doesn't have Obamacare and can’t wait on the government to make up its mind; what can I do about the torn cruciate?

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 us, statements that we 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

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 animal's 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 peri-articular 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 stay 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.

Option 3 is the grey zone. It may be the morbidly obese dog that needs to lose weight before surgery. It may be the family that is saving up for the TPLO, but wants to do something prior to surgery. Or it can be any other number of reasons. The focus in this area is education. Client education is critical with regards to many aspects of their pet. This includes obesity reduction (through diet and exercise), pain mitigation (through NSAIDS and other oral medications), chondroprotection (through oral supplementation of Glucosamine, Chondroitin, Omega-3 FA, etc..). Seeing the big picture with these pets is needed to achieve a favorable outcome. Without it, you get a fat, immobile dog in chronic pain.

Obesity: Checking thyroid function on all overweight dogs is recommended. If it is normal, then using a prescription diet tailored for obesity and arthritis is recommended. (MB uses Hills Metabolic and Mobility in his practice). Once initial inflammation is reduced (usually with NSAID, 1-2 weeks after injury) a rehab program of underwater treadmill walking is initiated. The goal here is to use the buoyancy of the water to reduce stress on the unstable stifle. This will allow for reduction of muscle atrophy and caloric burn without further injuring the patient. A protocol of 10-30 minute walks 2-3x a week (progressively building) is recommended. If possible, a photobiomodulation (therapy laser) treatment protocol is also initiated at these visits, with the goal being to reduce inflammation and promote tissue healing.

An at home program can include short leash walks, but a focus here on therapeutic exercises is used instead. Once initial inflammation is reduced, working on appropriate sit to stand exercises with the stifle in appropriate position is attempted. 10 reps per set, 10-2 sets per day. Initially we aim for 5/10 reps to be square, and then each week increasing our goal by one. The affected stifle should be against a wall, so as to minimize outward rotation. The goal here is fairly quick succession throughout the set. As soon as the patient ischium touches the ground the dog should be asked to stand again and repeat the exercise. Stretching and range of motion can begin at 1-2 weeks post injury, and then continue daily through week 8. The goal here is to maintain range of motion of the joint and minimize contraction.

The walks should be at a slow pace, on leash and on flat surfaces with good traction, Hill work and turning quick corners is not added until weeks 4-6.

Around weeks 4-6 as the fibrosis is being achieved, core strengthening with balance disks and wobble boards can be done in a slow safe and professionally administered manner.

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My issues with stifle orthotics are as follows:

1. **Tolerability**: I can 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 refuse 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 an owner pay the expense for a brace and then the dog won’t tolerate it.

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 it 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%.

Cruciate disease is a complex problem that does not have a clear cut answer. Ultimately, surgical intervention may be needed in any case that is initially managed without it. Proper education of the client is critical to establishing favorable outcomes.

**References**

Hip dysplasia (HD) was originally described in 1935 by Gerry Schnelle and has become one of the most common orthopedic conditions that leads 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.

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.2

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 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 is it currently to a hip score of 5, which is equal to an OFA excellent grade.

Physical rehabilitation has a multimodal approach within itself for managing HD. Physical modalities, manual therapies and therapeutic exercises can all be used to achieve relief from HD. Goals of rehab for the patient include: maintaining or improving muscle mass, building muscle support around the lax or arthritic joint (and all joints), reducing pain and weight loss (via exercise, when indicated).

Physical modalities can include thermotherapy (the use of cold and warm packs). The benefits of cryotherapy are established (pain relieving, vasoconstriction, etc.) and warm compresses can be used to relieve pain, cause vasodilation and also help to warm up stiff, tight tissues to begin other exercises.

Therapy LASERs (Light Amplification by Stimulated Emission of Radiation) have become very popular in recent years. There are different wavelengths, amplitudes, treatment times and other factors that must be considered. This process has also been called photobiomodulation. It has been proposed to activate cytokines and other tissue factors, decrease pain and inflammation and increased wound healing. Always use goggles for both the humans and patient to avoid damage to the eyes. It cannot be used over pregnancy or cancer.

Manual therapies are skilled hand movement techniques intended to: improve issue extensibility, increase range of motion (ROM), induce relaxation, mobilize or manipulate soft tissues and joints, modulate pain and reduce swelling and inflammation. These can include massage and joint mobilizations. The basic principles of joint mobilizations work from physiologic motions and accessory motions. Physiologic motions are normal active motion that is available at a joint. Examples: flexion, extension, abduction, internal rotation, etc. Accessory Motions are movements that cannot be performed actively. Examples: distraction, compression, glides, spins and rolls. There are 4 grades of mobilization, and the manipulation (used in chiropractic) is a 5th grade. Grades 1-4 are passive movements, with 1 and 2 not reaching initial resistance of the joint end feel. Grade 3 moves through the initial resistance to the end feel, but does not exceed it. Grade 4 mobilizations are compact with in the first and second resistance points. Grade 5 (manipulations) exceed the normal end feel of a joint.

Therapeutic exercises are the “meat and potatoes” of rehabilitation. These are designed to work a patient from a recumbent position back to normal (or as close as possible) activity following injury or insult. Exercises in this group can include cavaletti rails, working on balance boards, disks or other core strengthening equipment. Once walking on a flat non-slip surface is achieved, adding varying degrees of difficulty (up hills, through different traction, etc.) can be included. Sit to stand exercises and core strengthening with dancing exercises are also helpful. The key is to keep the patient moving and building.

Land treadmills – Can be useful devices for providing exercise. Small and medium dogs will work well on a human machine, but larger dogs will benefit from a canine treadmill. This is due to stride length and length of the belt. Having the dog walk on an incline will help build up the pelvic limbs.

Underwater treadmills – Can be used as both a diagnostic and therapeutic tool. The buoyancy of water will allow severely affected animals to utilize their limbs. There are also studies showing the benefit of underwater treadmill therapy for reducing obesity in dogs. With water at the level of the hock, there is a 9% reduction in perceived body weight, a 15% reduction with water at the stifle, and 62% reduction when at the greater trochanter. The non-slick, safe, contained surface an underwater treadmill provides is superior to walking in ponds, lakes or swimming in pools, in the author’s opinion.

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 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.

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
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.1,2 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 hyaluronan acid (HA) and other enzymes.

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.

In summary of OA inflammation: Osteoarthritis is a chronic progressively destructive disease that involves the entire joint. Inflammation is a key component of both joint destruction and pain. Acute pain resolves after the initial injury heals. Chronic pain involves structural changes of the dorsal horn, is more intense than acute pain and more difficult to control. Treatment considerations for osteoarthritis should address inflammation as well as pain.

Diagnostic approaches to osteoarthritis: 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.

A multimodal approach to OA management is needed. Non-Steroidal Anti Inflammatory Drugs (NSAIDS) represent the cornerstone of therapy, but other modalities include: nutrition, chondroprotectants, additional analgesics, physical rehabilitation, weight control, exercise, an EPA rich diet and many new and emerging options. Let’s look through these individually.

Obesity is a growing issue in veterinary medicine. The effects of obesity on OA are twofold. Biomechanical stress contributes to clinical signs and progression of disease. Adipokines secreted by white fat cells contribute to the progressive inflammation of osteoarthritis. Leptin levels are increased in obese dogs. In humans with osteoarthritis, increase leptin levels correlate with elevated MMPs and NO in synovial fluid. Adiponectin is anti-inflammatory, but levels are low in obese dogs. In human patients with knee osteoarthritis there is a significant correlation with adiponectin: leptin ratios.

Humans with increased body mass index (BMI) experience OA in non-weight bearing joints, which resolves with weight loss. Decrease in BMI, is associated with symptomatic relief from knee OA in man. Systematic review of canine studies found that preventing obesity decreases incidence of OA and weight loss reduces signs of OA. Additionally, diets rich in Omega-3 fatty acids have shown to be beneficial for both dogs and cats with OA. Additional nutritional supplements such as glucosamine, chondroitin, methylsulfonylmethane (MSM) and others have been shown potentially beneficial for our patients.

Physical rehabilitation has a multimodal approach within itself for managing OA. Physical modalities, manual therapies and therapeutic exercises can all be used to achieve relief from OA. Goals of rehab for the DJD patient include: maintaining or improving muscle mass, building muscle support around the arthritic joint (and all joints), reducing pain and weight loss (via exercise, when indicated).

Physical modalities can include thermotherapy (the use of cold and warm packs). The benefits of cryotherapy are established (pain relieving, vasoconstriction, etc.) and warm compresses can be used to relieve pain, cause vasodilation and also help to warm up stiff, tight tissues to begin other exercises.

Therapy LASERs (Light Amplification by Stimulated Emission of Radiation) have become very popular in recent years. There are different wavelengths, amplitudes, treatment times and other factors that must be considered. This process has also been called photobiomodulation. It has been proposed to activate cytokines and other tissue factors, decrease pain and inflammation and increased wound healing. Always use goggles for both the humans and patient to avoid damage to the eyes. It cannot be used over pregnancy or cancer.

Therapeutic exercises are the “meat and potatoes” of rehabilitation. These are designed to work a patient from a recumbent position back to normal (or as close as possible) activity following injury or insult. Exercises in this group can include cavaletti rails, working on balance boards, disks or other core strengthening equipment. Once walking on a flat non-slip surface is achieved, adding varying degrees of difficulty (up hills, through different traction, etc.) can be included. Other modalities in this group can include walking on treadmills or underwater treadmills.

Disease modifying agents for OA are next to be discussed. Polysulfated glycosaminoglycan is FDA approved, disease modifying osteoarthritis drugs; for dogs and horses; water-based, for intramuscular injection Dosage: 2 mg/lb body weight, IM, twice weekly for up to 4 weeks (maximum of 8 injections). MOA: specific is not known; osteoarthritis drugs; for dogs and horses; water-based, for intramuscular injection Dosage: 2 mg/lb body weight, IM, twice weekly for maintenance injections have bene anecdotally reported for both dogs and cats. Clinical studies on PSGAGs showed both good efficacy and safety. Treated dogs had statistically significant improvement in range of motion and total orthopedic score over placebo treated control dogs. 2.1% of dog had adverse reactions including: transient pain at the injection site (1 incident), transient diarrhea (1 incident each in 2 dogs) and abnormal bleeding (1 incident). These effects were mild, self-limiting; did not require interruption of therapy. Do not use in dogs showing hypersensitivity to PSGAG, or in dogs with known or suspected bleeding disorders. Use with caution in dogs with renal or hepatic impairment.

Adjunct analgesics for OA are numerous. They are used in addition to or replacement for NSAIDS. Research is scant on some of them. Amantadine – only drug studied to treat canine osteoarthritis. In dogs with osteoarthritis pain refractory to an NSAIDs, addition of amantadine improved physical activity. Amantadine might be a useful adjunct therapy for the clinical management of canine osteoarthritic pain. It can be dosed at 3-5mg/kg SID. Gabapentin – Calcium channel modulator – 5-10mg/kg SID-TID. Amitriptyline 0.5-1.0mg/kg SID-BID – cats and dogs. Local anesthetics – Lidocaine, bupivacaine, mepivacaine. Acetaminophen can be used in dogs.
but not cats. Opioids – morphine, meperidine, methadone, oxymorphone, hydromorphone, fentanyl, fentanyl patches, butorphanol, pentazocine, nalbuphine, buprenorphine, codeine and tramadol.

Tramadol’s metabolism and elimination is rapid and variable among dogs. When administered orally or intravenously to the dog, metabolism of tramadol and all metabolites is rapid. There is much variability between dogs, possibly breeds. Pain control did not necessarily correlate with plasma levels of the active metabolite (O-desmethyltramadol). Tramadol effects on α-adrenergic or serotonin receptors may contribute to analgesic effects in the dog. Regardless of mechanism of action, studies suggest oral dose should be 5 mg/kg q 6 hours or 2.5 mg/kg q 4 hours. In the author’s opinion this is a very challenging drug to utilize effectively in practice due to these variables.

Galliprant is a first-in-class non-cyclooxygenase (COX) inhibiting, non-steroidal anti-inflammatory drug (NSAID) in the piprant class. Piprants are a newly recognized drug class, established and defined by the World Health Organization in 2013 as prostaglandin receptor antagonists (PRA). Unique mechanism of action by antagonizing the prostaglandin E2 (PGE2) EP4 receptor. PGE2 its physiologic effects through binding of four different receptors, EP1, EP2, EP3 and EP4. The EP4 receptor has been identified as the primary receptor responsible for mediating pain and inflammation associated with osteoarthritis. Galliprant selectively blocks the EP4 receptor, thus blocking PGE2 elicited pain.

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
9. Adequan prescribing information. NADA 141038, Novartis animal Health, US, INC.
1. Introduction
   a. Many veterinarians fear the treatment of older dogs and cats with analgesics and anesthetics.
   b. Fear of use of these agents results in suffering and untreated conditions for which anesthesia is necessary
2. Things to consider
   a. Being old doesn’t mean that anything is wrong
   b. Being old means that there are a number of patients with subclinical organ function
      i. Renal, hepatic, and cardiac are the most common
   c. Body composition itself can have a pharmacodynamics effect in geriatric patients
      i. Decreased lean body mass
      ii. Drug receptors
      iii. Neurotransmitters
3. Decreased Resilience
   a. It is not unusual for geriatric dogs and cats to have reduced recovery times from physiological disturbances such as those brought on by anesthesia or sedation.
   b. Geriatric animals may have heightened response to both pain and other stimuli that a younger animal would not have.
4. Concurrent Treatments
   a. Many Caregivers give OTC drugs or supplements
      i. They often don’t consider them important enough to tell you
      ii. St. John’s Wort is common
5. Behavioral Considerations
   a. Environmental changes
      i. Hospitalization can bring on signs of cognitive dysfunction
      ii. Can cause decreases in willingness to eat, drink or interact
      iii. Encourage caregivers to bring items from home
   b. Nursing Care
      i. Pay special attention to food and water intake
      ii. Padded bedding as most have OA
      iii. Socialization is important during time in clinic
6. Diagnostics
   a. Always check for concomitant disease.
      i. Lab work
      ii. Radiographs
      iii. Urinalysis
7. Treatment
   a. Treatment of long term pain can be challenging.
      i. Many causes are not curable; caretaker should understand that the goal is palliative not curative.
   b. Easy to give drugs, impossible to take them back
      1. Start with lower doses and monitor for effect of drug and adverse events
   c. Try to use reversible drugs
8. Cardiopulmonary Issues
   a. As many as 58% of geriatric dogs show evidence of valvular disease
   b. Both cats and dogs have decreased alveolar plasticity
   c. Both of these issues mean less oxygen availability to tissues
9. CNS
   a. Mean alveolar concentration requirements decrease with age by an unknown mechanism(s).
      i. Always use lower settings for older animals
10. Hepatic Issues
    a. Total hepatic mass decreases with age and subsequently the ability of the liver to metabolize drugs.
11. Metabolic Issues
As most animals age, their muscle mass decreases. At the same time body fat increases

i. Affects both water soluble and fat soluble drugs

b. Many older animals are either hypothyroid (dogs) or hyperthyroid (cats)

12. Renal Issues
a. Many geriatric animals have a decrease in renal function, which can impair their ability to excrete or clear certain drugs.

b. This can be complicated by cardiac issues

13. Pain drugs for chronic use
a. NSAIDs
   i. The side effects of NSAIDs in geriatric animals are similar to those of other age groups
      1. Monitoring for ulcer formation may be a challenge in older dogs that eat sporadically
   ii. Use of NSAIDs in cats with renal disease
      1. Over 90% of cats of any age have some DJD
      2. Renal disease in cats is inflammatory in part
         a. OK to give in cats with renal disease
         b. Follow other precautions
         c. Use is off label in the U.S.

b. Amantadine
   i. Amantadine works on the NMDA pathway
   ii. Important to use in dogs that are refractory to NSAIDs alone

c. Gabapentin
   i. Exact mechanism is unknown, no big studies in dogs or cats
      1. Encouraging reports on its effectiveness
   ii. Cleared by kidneys
      1. Decrease dose with possible renal impairment
      2. Start at half the recommended doses.

d. Maropitant
   i. Confusing recommendations to use for pain
   ii. Use it for vomiting
      1. Given the day before, may relieve nausea as well

e. Acetaminophen
   i. Bad in cats
   ii. OK for dogs
      1. Pharmacodynamics are uncertain, should never be sole source of pain relief

f. Grapiprant
   i. New class of drugs, piprant class, approved for use in dogs
   ii. Blocks the prostaglandin E2 at the EP4 receptor
   iii. Has had pilot studies in cats

14. Other drugs
a. No evidence to support:
   i. Oral tramadol
   ii. Oral hydrocodone
   iii. Oral Oxycodone

b. All risk, no benefit

15. Physical Modalities
a. Rehabilitation
b. Acupuncture
c. Massage
d. Weight Loss
e. Myofascial Pain Treatments
f. Hot and cold therapy
References


1. NSAIDs are the most widely used analgesics in veterinary medicine
   a. Crucial in the treatment of acute pain
   b. Cornerstone of treatment in chronic painful conditions
2. Review of COX pathway
   a. Cox-1 not used in veterinary medicine
      i. Considered constitutive or for physiologic function
   b. Cox-2 popular NSAIDs are Deramaxx, Metacam, Previcox and Rimadyl
      i. Work on pain and inflammation
   c. Work by stopping production of prostanoids from prostaglandins by inhibition of cyclooxygenase pathway
   d. Prostanoids cause pain and inflammation through stimulation of PGE receptors at both the nociceptor and dorsal horn of the spinal cord
3. Adverse effects of NSAIDs in Dogs
      i. Most studies were not randomized, controlled and blinded
      ii. Most did not use a clinical population of dogs
   b. Had sufficient data to compare carprofen, deracoxib, ketoprofen, meloxicam and rebenacoxib
      i. Most common Ae’s
         i. Vomiting and diarrhea
         ii. Melena and fecal blood
         iii. Colitis
         iv. Abdominal Pain
         v. Icterus
         vi. GI ulceration and perforation
      d. Results
         i. Difficult to discern if a significant difference exists between NSAIDs regarding safety
         ii. AE’s were similar to placebo when only the highest quality studies were included.
   a. Events per 1 million oral NSAIDs administered to DOGS
      i. Renal insufficiency 44
      ii. Emesis  170
      iii. Anorexia 74
      iv. Lethargy 83
      v. Death 113
   b. Events per 1 million oral NSAIDs administered to CATS
      i. Renal insufficiency  122
      ii. Emesis  254
      iii. Anorexia 180
      iv. Lethargy 172
      v. Death  164
5. New NSAIDs for Dogs
   a. Onsior injectable
      i. Injectable same for dogs and cats
      ii. Refrigeration required
      iii. Use within 12 weeks of broaching vial
      iv. 2 mg/sq
      v. Maximum use 3 days
   b. Onsior Tablets
      i. 2 mg/kg
      ii. Indicated for post-surgical pain
iii. Maximum 3 days
iv. Fast absorption
   1. Tablets 30-60 minutes
   2. SQ 1-2 hours
v. Rapid Clearance
   1. Tablets 1 hour
   2. Injection 1-4 hours
vi. Onsior persists longer at the site of inflammation than in the blood, so mechanism of action is 24 hours despite short clearance time
vii. Efficacy
   1. Injectable $p=0.0055$
   2. Tablets $p=0.0188$
   3. $P$ value < 0.05 is good
viii. Adverse Reactions
   1. Similar to all NSAIDs, diarrhea, vomiting, decreased appetite
c. Galliprant (Grapiprant)
   i. Classified as a “Non-COX inhibiting NSAID” by the FDA
      1. Actually belongs to its own class, the piprants
   ii. Inhibits the EP4 receptor which is responsible for pain
      1. NSAIDS block the entire COX pathway
   iii. Tested in dogs, without significant pathology other than OA
   iv. AE’s
      1. Vomiting and diarrhea more common in grapiprant than in NSAIDs
         a. However most resolved within a few days and without intervention
         b. No further AE’s developed (i.e. ulcer or gastric perf)
         c. Was given at 15 times the package dose daily for 9 months, no dogs developed an ulcer or gastric perforation
      2. Efficacy
         a. $P=0.0315$
6. Chronic Use of NSAIDS in Cats
   a. Not approved for long term use in cats in the U.S.
   b. Mode of Action in Cats
      i. Like dogs, inhibits COX
   ii. Metabolism of Meloxicam
      1. Oxidation, which is a good thing. More reliable in cats than glucuronidation
      2. Half-life about 24 hours but variable from cat to cat
   iii. Metabolism of Robenacoxib (Onsior)
      1. Degrades to form $\gamma$-lactam
         a. Extensively metabolized by liver
      2. Half-life is 1½ hours
         a. Persists in tissues longer
c. Approved use
   i. Meloxicam single injection
   ii. Onsior 3 days
d. Potential problems with long-term administration
   i. Dosing: Label dose of meloxicam is not appropriate for long-term administration
   ii. Half-life in meloxicam is near or exceeds 24 hours
      1. Can be cumulative at higher doses
7. Why chronic use in cats?
   a. Transitional Cell Carcinimal
   b. Degenerative Joint Disease
      i. High prevalence od DJD
         1. Lascelles study showed 92% rate of OA in cats from 6 months to 20 years
   c. No other drugs licensed in the US for the chronic treatment of OA in cats
8. Treatment of OA in cats
   a. Clinicians scared away
      i. Potential for toxicity
      ii. Off label Use
      iii. Black Box warning
   b. AAFP Guidelines
      i. Before starting any cat on a long term NSAID perform PE, Blood Pressure, CBC, Blood Chemistry, Urinalysis
      ii. Routinely monitor the patient every 3-6 months
      iii. Base dose on lean body mass
      iv. Give drug with or after food
      v. Feed moist food to insure good fluid intake
      vi. Educate owners on potential side effects
      vii. Stop drug if cat stops eating
      viii. Titrate to lowest effective dose
      ix. Reduce the dose if other drugs are being used
      x. Never give with corticosteroids
   c. Cats with renal disease
      i. Renal disease in cats has an inflammatory component
      ii. Most cats with renal disease will benefit from the administration of meloxicam, both pain and renal function
         1.
Myofascial Pain Therapy and Massage for Successful Surgical and Pain Treatment Outcomes
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1) Abbreviations and Definitions
   a) MPS Myofascial Pain Syndrome
   b) MTrP Myofascial Trigger Point
      i) A contracted region of muscle
   c) TB Taut Band
      i) The band of muscle which is tight because of the MTrP

2) DN Dry Needling
   a) The invasive method by which MTrP’s are treated, using an acupuncture needle

3) Twitch Response
   a) An involuntary muscle reaction that occurs as a result of dry needling. Involves spinal pathways.

4) History of Muscle Pain
   a) Janet Travell
      i) Cardiologist in the 1940’s
      ii) Took a strong interest in the amount of muscle pain seen in many of her cardiology patients
      iii) Rediscovered myofascial pain
      iv) Along with David Simons wrote the Trigger Point Manual
         (1) Still used as text today
   b) Interest has slowly but steadily grown in the human field
      i) Barely known in the veterinary field
   c) Past History
      i) French physician Guillaume de Baillou (1538-1616) published book on “muscular rheumatism”
      ii) Thomas Sydenham (1624-1689) published book in 1676 that included description of MPS
      iii) British Physician Balfour (1816) wrote: “Patients as having a large number of nodular tumours and thickenings which were painful to the touch and from which pain shot to neighbouring parts”

5) Has been called…..
   a) Fibrositis (Gowers, 1904)
   b) Fibromyositis (Telling, 1911)
   c) Myofasciitis (Albee, 1927)
   d) Myofibrositis (Murray, 1929)
   e) Perineuritis (Clayton & Livingstone, 1930)
   f) Idiopathic myalgia (Gutstein-Good, 1940)
   g) Rheumatic myalgia (Good, 1941)
   h) Myofascial Pain Syndrome (Travell, 1948)
   i) Myodysneuria (Gutstein, 1955)
   j) Fibromyalgia (Yunus, 1977)

6) Trigger Point Development
   a) Primary
      i) Acute injury after trauma
      ii) Chronic muscle overload
      iii) Poor mechanics
      iv) Repetitive movements

7) Secondary Causes: Most common
   a) Underlying disease or inflammation from any chronic painful condition
   b) Even when the primary cause is not muscular, central excitatory effects tend to expressed in the muscles, making this a frequent complication accompanying other sources of pain
   c) Satellite MTrPs: Primary trigger points may cause or may induce and maintain referred pain in the form of satellite
      i) MTrP’s elsewhere.
      ii) Example of Satellite MTrP from Human literature
         (1) Pain from MTrPs in the upper trapezius may induce and maintain MTrPs in the anteror temporalis or masseter muscle
         (2) Dry needling of the trapezius can reduce the irritability of satellite MTrPs
   d) Stress
      i) It is known that stress can activate trigger points in humans.
      ii) In animals??

8) Characteristics of Muscle Pain
   a) Human description
i) Usually a cramp or an ache, sometimes hard to localize.
ii) Animals cannot tell us
b) Cortical structures unique to muscle pain are activated
   i) Inhibited more strongly than other types of pain by descending pain modulating pathways
   c) Activation of muscle nociceptors are much more effective than other types of nociceptors at inducing changes in the spinal cord’s dorsal horn
9) 3 Types of electrical contractility
   a) Normal
   b) Abnormal
   c) Pathological
      i) More on this later
10) Acetylcholine (ACh) is necessary for all contractures, normal, abnormal or pathological
    a) Increased ACh action can be present for a variety of reasons
       i) Lack of acetylcholinesterase
       ii) Sensitized receptors
       iii) Excess ACh
       iv) Low pH
    b) Excess calcitonin gene related peptide
11) Action Potential
    a) Release of acetylcholine causing an impulse via T tubule and release of Ca++ into the sarcoplasmic reticulum
12) Contraction
    a) Ca++ binds to troponin and exposes active site of actin
       i) Allows bridge between myosin and actin to form
    b) ADP released
    c) ATP necessary to detach the myosin-actin bridge
13) Pathophysiology
    a) There are several components that make up the pathophysiology of the trigger point. They are:
       i) The Motor Endplate Component
          (1) The motor endplate is the place where alpha motor neurons synapse with target muscle fibers
          (2) These neurons cause the release of acetylcholine (ACH) which through a cascade of reactions causes myosin and actin to bind causing sarcomere contraction
          (3) A decrease in acetylcholinesterase results in the inability of the contraction to “release.”
       ii) The Motor Component
          (1) Muscle contraction compresses local sensory nerves, which reduces the axoplasmic transport of molecules that normally inhibit ACH release
          (2) Muscle contraction also compresses local blood vessels resulting in a decrease in oxygen at a time when the muscle contraction requires an increase in the amount of oxygen
          (3) This results in a rapid depletion of ATP resulting in an energy crisis ATP is needed to turn off ACH release. This results in a vicious cycle of continued muscle contraction and ATP depletion
       iii) Sensory Component
          (1) Micro-sampling from within the MTrP revealed elevated concentrations of inflammatory substances:
          (2) Protons, bradykinin, serotonin, substance P, norepinephrine, tumor necrosis factor and interleukin-1b
             (a) Persistent barrage of nociceptive signals from MTrPs may eventually cause central sensitization leading to allodynia or hyperalgesia
             (b) These changes can become permanent
       iv) Autonomic Component
          (1) Autonomic phenomena associated with MTrPs may include vasoconstriction/vasodilation and pilomotor activity.
          (2) The autonomic nervous system may indirectly exacerbate MTrP formation via visceralosomatic reflexes.
             (a) Again, another reflex arc where MTrPs stimulate the ANS which causes disturbances in the viscera which then can increase the central sensitization
14) Theory to Therapy
    a) Deep digital pressure? Only increases compression and worsens the condition
    b) Affected muscles that cross an articular surface can reduce the functionality of that joint via decreased muscle length
    c) Constant pressure on the joint increases sensitization which then sends constant nociceptive signals to the CNS which responds with further activation of the MTrPs
    d) Needling with botulinum toxin type A prevents release of ACh
    e) Dry needling in animals is very effective
15) Why that muscle?
    a) The Cinderella Hypothesis
       i) Works on the idea that within any muscle, certain fibers are always the first recruited during contraction and the last released during relaxation
       ii) This is very significant during activities that do not require full muscle contraction, but only subtle contraction
iii) e.g. Chronic cruciate rupture or hip dysplasia results in very slight and subtle contraction to take just a little weight off the affected leg.

16) What is a trigger point?
   a) Not all trigger points are active.
      i) We have latent trigger points, either through lack of a triggering event or because it has been treated
   b) A trigger point is an area of disturbed motor function
      i) As described in the previous slides, there are areas of unrelenting myosin and actin binding
      ii) This results in the “taut band” an area of muscle fibers that are abnormally shortened and which mechanically impair the action of the muscle AND reduces joint space in the joint the muscle crosses resulting in restricted range of motion.

17) Biochemical changes in trigger points
   a) Shah J, Phillips T M, et al. analyzed substances within a trigger point
   b) Use of a microdialysis needle capable of continuously collecting extremely small samples of physiological saline after exposure to the trigger point
   c) Findings
      i) Protons
      ii) Bradykinin
      iii) Calcitonin gene-related peptide
      iv) Substance P
      v) Tumor necrosis factor -Alpha
      vi) Interleukin -1 beta
      vii) serotonin
      viii) norepinephrine

18) Muscle weakness
   a) Results because of the disturbed motor function
   b) Vasoconstriction often results secondary to the muscle compression of the taut band but can be an autonomic response
      i) This happens at a time when the muscle needs MORE oxygen to generate ATP molecules

19) Pain
   a) The affected muscle is painful
   b) Referred pain may be present
   c) Muscle Cramps can be induced by irritation of the latent MTrPs
      i) Glutamate injected into both latent trigger points and into normal muscle
         1) Normal muscle had zero muscle cramps
         2) Trigger point injections resulted in 93% of the muscles having cramps
         3) Pain propagation of MTrP after glutamate
      ii) Mechanical stimulation of latent MTrP
      iii) Pain propagation to latent MTrP

20) What causes Trigger Points in the clinical setting?
   a) Mechanical Stresses
      i) Most common perpetuating factor in dogs
   b) Chronic Muscle Overload
      i) Contributes to the muscle mechanisms that develop taut bands by different causes all which can result in:
      ii) Low level sustained muscle contractions
      iii) Direct trauma
      iv) Eccentric muscle contractions
      v) Submaximal concentric muscle contractions
      vi) Maximal concentric contractions
   c) Orthopedic Injury
   d) Post Operative surgical trauma and pain
   e) Neuropathy
   f) Joint dysfunction
   g) Acute Trauma
      i) Acute trauma may activate MTrPs but does not perpetuate them. Sudden activation of muscles resulting in
         1) Muscle strain
         2) Joint sprain
      ii) Fractures
      iii) Direct trauma
      iv) Excessive or unusual exercise
      v) Acute trauma seen most commonly in
         1) Performance dogs
         2) Hit by car
         3) Falls of any kind
         4) “Weekend Warrior”
   h) Osteoarthritis
Osteoarthritic joint dysfunction will lead to postural changes

Postural changes result in muscle mechanisms causing

Low level muscle contraction

Eccentric muscle contraction

Unaccustomed muscle contraction

Visceral somatic Pain

It is known in humans that visceral pain will perpetuate MTrPs in the area of referred pain

Dorsal horn of spinal cord receive input from viscera and from receptors in the skin and deeper tissues

Muscle pain due to visceral nociceptive activation of dorsal horn probably causes MTrPs in dogs as well

Diseased organs and nerves can manifest themselves in both skin and muscle problems

Occasionally I see dogs that have MPS for no apparent reason but they also have Inflammatory Bowel Syndrome

Nutritional Inadequacies

Cobalamine and folate is a perpetuating factor

Not known to cause issue in dogs

But: Often present in bowel disease in dogs which often have concurrent MPS

Possible future link?

Iron deficiency

Recognized as a perpetuating factor in people

No evidence in dogs as of yet

Metabolic

Hypothyroidism in humans causes

Muscle pain and weakness

Cramps

Pain

Not known to cause MTrPs in dogs

Nerve impingement

MTrPs are formed in the extremity corresponding to the involved spinal cord segment

Stress

Stress and tension in humans can cause trigger points to form.

How do we measure this in dogs?

Neurologic Conditions

There can be postural changes and weight shifting brought about by any neurologic dysfunction

Mechanical stresses result

Eccentric contractions

Unaccustomed concentric contractions

Low level sustained contractions

Development of MTrPs in muscles innervated by injured or damaged nerves

Action potentials are generated at the site of compression in both directions

Radiculopathy

Most common in thoracic limb

Usually a C6-T2 spinal cord injury causing MTrPs in long head of triceps

Treatment Techniques and Patient Evaluation

Identify and control perpetuating factors

Then and only then apply specific trigger point therapies

Treating the trigger points without treating the causes will result in temporary pain relief at best

Dry needling seems to be the most efficacious treatment in veterinary medicine

Palpation of taut bands just requires practice

Needling technique is different than needle placement for acupuncture

Steps in Finding and Treating MTrPs

Palpate muscles to locate taut bands and trigger points

Look for all of classic signs of painful palpation. Depends on dog and personality...licking lips, turning and looking, vocalization, jump, menace response

Dry needling the points

Twitch response is the desired response to treatment

Don’t confuse with a jump response, the animal voluntarily reacting to the painful palpation

You cannot feel the trigger point itself. You are looking for the taut band of muscle

You might find the trigger point by palpating along the taut band: The taut band is painful. The trigger point is very painful
v) Humans can report referred pain from palpation of the trigger point

28) Client Education
a) Most veterinarians don’t know about MPS, even more true of your clients
b) Explain role of perpetuating factors
c) Always form a treatment outline and the rationale and expected outcome measures
d) Enlist the owners help to modify the animal’s environment or routine
e) Explain importance of schedule, medications exercises etc. etc.
f) I try as hard as possible to make the next appointment before the client leaves my office.
g) Usually weekly to start

29) The veterinarians role
a) Identify and treat perpetuating factors
b) Apply specific therapies as indicated (DN, medication, acupuncture, etc)
c) Re-evaluate patient about every three weeks and reconsider diagnosis of perpetuating factors if poorly responsive

30) Palpation
a) 4 kg of pressure on a muscle should not be painful
   i) If it is, then it is probably a trigger point
b) Algometer
   i) Works great in people. Dogs quickly learn to think that the algometer is causing the pain
c) Palpation Techniques
   i) For most muscles, a pincer technique is used.
      (1) No matter the technique, never apply more than 4 kg of pressure
   ii) For muscles such as the triceps that can be grabbed
   iii) Single finger or flat hand palpation
      (1) For muscles that don’t lend them selves to “grabbing”
      (2) Infraspinatus, tensor fascia latae
   iv) Spade hand palpation
      (1) Iliopsoas

31) Dry Needling
a) You know you have got the spot when you get a localized twitch response
   i) This is actually a spinal reflex
b) MTrPs are often clustered together. You keep pecking away at the same spot over and over. I find it very common to get 15-20 twitch responses in the same spot.
   i) The same needle can be re-sheathed and used over and over until it starts to feel dull or bend
c) Dry needling also causes micro trauma, increased local blood flow helping to relieve the energy crisis and release of inflammatory agents.
d) Muscles with taut bands feel different than normal muscles when you put a needle in them. There is a heavy “clay” feel to the muscle. Some people describe it as feeling “gritty.”
e) Accurate needle placement produces a twitch response. The needle is moved in and out of the MTrP until the twitches stop.

32) Other Treatment Techniques
a) Massage
   i) Massage brings some instant relief to any trigger point.
   ii) The relief is temporary compared to dry needling.
   iii) Twitch response is seldom attained
   iv) Massage compresses muscles
      (1) These muscles are already compressed along with blood vessels
   v) Massage further moves blood from the muscle...all at a time when there is low blood flow, low oxygenation of the tissue and lack of ATP
   vi) Does it make it worse in the long run?
b) Stretch
   i) Stretching the muscle provides relief from trigger points.
   ii) It usually involves a “spray and stretch” technique wherein a coolant is sprayed on the skin and the affected muscle is then stretched
      (1) The coolant is really just a distraction for the patient
      (2) Just as in dry needling, a twitch response should occur
   iii) As in massage, I question the wisdom of muscle compression when the muscle is already compressed, along with blood vessels.
   iv) This technique (without the coolant) is mostly used by physical therapists working with animals
c) Procaine Injections
   i) Local anesthetic injected into trigger points allows longer periods of time between treatments.
   ii) It is necessary to know exactly where the trigger point is, not practical to inject procaine along the length of every taut band.
      (1) In humans, this is easy because they can tell you when you are exactly on the most painful “spot.”
      (2) We can’t get the same feedback in dogs
(3) I don’t know of anyone who is using it in animals
d) Botulinum Toxin Injections
   i) Not used in veterinary medicine
   ii) As in the case of procaine injections, it is easy to identify the taut band in the dog, but unlike in people, it is difficult to impossible to identify the most painful trigger point
   iii) Doesn’t allow for treatment of key points
   iv) Toxic doses of botox can occur trying to treat all painful points
   v) In theory, injection of a trigger point with botox would inactivate it for several months
   vi) The U.S. Food and Drug Administration has approved the use of botox for migraine headaches that are the result of MPS
   vii) There is not strong evidence for the use of botox outside of migraine treatments.
   viii) More due to lack of good studies, lots of clinical evidence of its effectiveness.

33) Massage Techniques.
a) There are three techniques of massage.
   i) Petrissage
   ii) Effleurage
   iii) Tapotement
b) I recommend the book Medical Massage by Narda Robinson to learn these techniques
c) Massage is one of the oldest forms of manual therapy used to employ pain relief.
d) Common treatment
   i) Muscle Damage
   ii) Degenerative Myelopathy
   iii) Pain and relaxation, especially in cancer patients
1. Philosophy of the pain practice
   a. As much a state of mind as it is about a set of skills
   b. The Five Freedoms
      i. Freedom from thirst hunger and malnutrition by ready access to fresh water and a diet to maintain full health and vigor
      ii. Freedom from discomfort by providing a suitable environment including shelter and a comfortable resting area
      iii. **Freedom from pain, injury and disease by prevention or rapid diagnosis and treatment**
      iv. Freedom to express normal behavior by providing sufficient space, proper facilities and company of the animal’s own kind
      v. Freedom from fear and distress by ensuring conditions that avoid mental suffering
   c. Euthanasia is the last pain treatment not the first
      i. Hospice is an option
      ii. People do get angry and will go elsewhere. But if enough of us say “no” the message will get across
2. Consideration of Pain in Animals
   a. Common pain misconceptions
      i. My dog doesn’t cry out, he isn’t in pain
      ii. My pet is just old
      iii. Animals don’t feel pain like we do
      iv. There is nothing we can do
      v. Post-op pain is a benefit
      vi. OHE’s, neuters and minor procedures don’t need pain meds
      vii. It is too dangerous to give sick and debilitated animals pain medication
      viii. My clients won’t pay for “that.”
3. A Pain Practice means a team approach
   a. Every person on staff needs to be on board
      i. If they are not, clients can tell
   b. Receptionist
      i. Should be handing out a chronic pain survey to every animal that is 6 years or older and walks into your door
      ii. Should be handing out the same questionnaire whenever a client voices a pain concern
   c. Technician and assistants
      i. Don’t underestimate their power of observations
   d. Kennel Help
      i. Sometimes they are the only ones that see an issue when the animal lets it’s guard down
4. Chronic Pain Questionnaire
   a. Many signs of pain not observable in clinical situation
      i. People write it off as “age”
   b. Use questionnaires designed for chronic pain, not acute pain
   c. If you get answers that you cannot reconcile with your own observations, go over the survey with the owners.
5. The Pain Exam
   a. Observe for gait abnormalities
      i. Take the time to walk the dog on a non-slip surface, often that means a parking lot or sidewalk
   b. Exam room
      i. Choose your quietest room
      ii. Consider a non-slip floor
         1. Elephant bark
      iii. Exam best done on the floor, not the table
         1. Small dogs cats and aggressive dogs may need a table
      iv. Make it easy to get to the exam room
1. If you have slippery tiles, roll out a carpet or a roll of yoga mat

6. The Pain Patient  
   a. Examination techniques will vary based on the number of modalities you are familiar with  
   b. Range of motion  
      i. The flexion and extension of a joint  
      ii. Normal ranges and “end feels” of the joint can be hard to determine unless you have rehab experience.  
         Even still, if you look at the painful limbs of every dog, you will start to learn normal and abnormal feels  
   c. Cruciate disease  
      i. Learn the anterior tibial thrust technique to look for partial tears of the CCL  
      ii. Look for medial buttressing in the cases of chronic rupture  
   d. Myofascial exam. One of the most important exams to look for underlying pathology  
      i. Really a two day lecture  
      ii. In a nutshell, constant overuse of muscles because of underlying pathology will cause painful areas  
         called trigger points to appear within a band of muscle  
      iii. These areas are painful to palpation and only the most stoic dog will not react to firm palpation of the  
         muscle.  
      iv. These trigger points are almost always a symptom of underlying pathology. Finding them and having  
         the owners see the dog’s reaction to palpation will invariably convince the owner that further diagnostics  
         are necessary  
   e. Direct digital pressure on joints  
      i. Usually unrewarding unless there is joint infection or osteosarcoma  
   f. Age should be considered when looking for an etiology  
      i. Young dogs congenital disease and trauma  
      ii. Old dogs, acquired disease and trauma  
   g. Radiology. If you don’t look, you won’t know. Many cases of CCL have the precipitating cause of an  
      osteosarcoma. Always radiograph suspect areas of pain  
   h. Blood Work is necessary to look for concomitant issues and should always be done BEFORE the start of long  
      term NSAID therapy  
   i. Cats. History becomes more important and conversations about what the cat used to be able to do compared to  
      what it can do now are essential  
      i. History  
         1. Jumping  
         2. Sleeping  
         3. Grooming  
            a. Self grooming and being brushed can be painful  
         4. Being picked up and held by the owner  
      ii. Distant observation. Hair coat, mats, spindly legs  
      iii. Palpation  

7. Don’t forget other conditions  
   a. Neurological conditions can mimic pain  
   b. Neuropathic pain  
      i. See notes on neuropathic pain lecture  

8. Treatment Goals and Outcome measures  
   a. The key to success  
   b. If you don’t ask what they hope to achieve, how can you even start treatments  
      i. If you think YOU did everything right but you didn’t get to the owner’s goal, they will be unhappy  
   c. Ask the owner what they hope to accomplish  
      i. Discuss if their desire is reasonable  
      ii. Give time frame for re-evaluation  
         1. This enlists the owner as part of the pain team and gets them to work with you.  
      iii. Fill out the pain questionnaire at regular intervals and compare results.  

9. Finding Pain Patients  
   a. Satisfied clients will be the #1 source of pain referrals  
   b. Veterinary referrals  

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Most veterinarians will not refer
1. Afraid of losing clients
2. Think that having three NSAIDs and two glucosamine products on the shelf make them pain vets

c. Dedicated pain website.
   i. Include testimonials, written or video
   ii. Services
   iii. General pain information

10. Learn new skill sets
    a. Acupuncture
    b. Rehabilitation
    c. Become a Certified Veterinary Pain Practitioner with the IVAPM
    d. Learn Myofascial pain diagnostic and treatment techniques (www.myopainseminars.com)
    e. Attend Pain seminars
       i. Listing on www.ivapm.org

11. Learn when to admit you are in above your head and refer. Working with a pain veterinarian, like working with an internist or ophthalmologist only instills confidence with your clients.
Using Cannabidiols to Treat Pain
Michael Petty, DVM, CVPP, CVMA, CCRT, CAAPM
Animal Pain Center
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1. Hemp v Marijuana
   a. Marihuana
      i. High THC (5-30+%), low CBD
      ii. No known use in veterinary medicine
   b. Hemp
      i. Low THC, (<0.3%) high CBD, other cannabinoids and terpenes
      ii. Suggested uses in veterinary medicine

2. Both Marijuana and Hemp
   a. Medicinal, rope, cloth, canvas, paper and traditionally used for caulking ships

3. Marijuana
   a. Schedule I
   b. Can’t prescribe in veterinary medicine
   c. Research largely prohibited
      i. Federal regulation

4. History
   a. China
      i. Evidence of use as long ago as 5000 years
   b. Book of Exodus 30:23
      i. Moses was instructed by God to use hemp
   c. Egyptians
      i. Evidence of use 3500 years ago
   d. Indians (Atharvaveda)
      i. 3000 years

5. Mechanism of Action
   a. Endocannabinoid system THC
      i. Receptors in the brain and PNS
   b. Physiological effects
      i. Appetite
      ii. Pain
      iii. Nausea
      iv. Mood
      v. Inflammation
   c. Endocannabinoid Receptors CBD
      i. Mostly in the PNS
      ii. Especially immune system
      iii. In CNS as well
   d. THC and CBD
      i. Deeply involved in communication or neurotransmission
      ii. Act as modulator; telling some transmissions to speed up and others to slow down
      iii. Purpose it to return the body to a normal state

6. So why CBD?
   a. Was probably the active ingredient in marijuana that made it work
   b. Not psychoactive
   c. Higher safety levels than THC
   d. Many attributes given to CBD

7. Client Attitudes
   a. Are they using it?
      i. Many people that visited the Canna-Pet website bought it
   b. Why do they use it?
      i. Pain
      ii. Sleep aid
      iii. Anxiety
      iv. Nervous system support
      v. Reduce inflammation
      vi. Seizures
      vii. Nausea
   c. Perceived Problems with use
i. Increased appetite
ii. Lack of Energy
iii. Panic Reactions
iv. Dry mouth
v. Sedation
vi. Nausea
vii. Increased Seizures
d. How did it stack up compared to conventional medications?
   i. Better than any 19.3%
   ii. Better than most 24.7
   iii. Better than some 18.4%
   iv. As well as some 20.8%
   v. As well as most 9.3%
   vi. Worse than many 2.8%
   vii. Worse than any 2.6%
   viii. Worse than most 2%

8. Safety
   a. Over 1000 research papers on CBD
      i. Most all are human
   b. CBD found to be non-toxic
   c. Rare side effects
      i. Possible interference with cytochrome P450
      ii. Ivermectin? Discontinue CBD for 2-3 days
d. Not addictive, actually anti-addictive
e. In humans can be used as adjunct treatments to addictions such as tobacco, alcohol, opiates

9. Evidence
   a. Mostly from human research
   b. Anecdotal in animals
c. Anxiety
d. Stress areas of brain (e.g. amygdala) are rich in CBD receptors
e. Noise aversion
f. Separation anxiety
g. Fear of strangers
h. Cognitive Dysfunction

10. CBD’s in particular
    a. Cognitive Dysfunction
    b. Neuroprotective
c. Anti-inflammatory
d. Antioxidants
e. Regenerate new neurons in the part of the brain responsible for memory and can improve memory.
f. Autoimmune disorders
    i. Autoimmune thyroiditis
    ii. Immune Mediated Hemolytic Anemia
    iii. Immune Mediated Thrombocytopenia
    iv. Pemphigus
    v. Lupus
g. Bone Health
    h. Helps Heal Fractures
    i. Cancer
    i. Manage signs of cancer
    ii. pain
   iii. nausea
   iv. Reduce inflammation
    v. Induce cancer cells to die
   vi. Slow cancer growth
   vii. Inhibit neovascularization of tumors
   viii. Protect non-cancerous cells
    j. Inflammatory Bowel Syndrome
    i. Reduces mobility and inflammation
    k. Degenerative Myelopathy
    i. No evidence in dogs
       1. Works well in ALS
    l. Glaucoma
i. Actual cat study!
   1. Unfortunately they used CBG cannabigerol
   2. Relieves pressure
m. Degenerative Joint Disease
   i. Reduces inflammation and pain
   ii. Inhibits release of TNF
n. Inflammation is the underlying basis of a number of diseases
   i. Pain suppression through attaching to receptors in parts of the brain responsible for pain reception
o. Reduction of neuropathy
11. Endocannabinoid Deficiency Syndrome
   a. For some animals, there may be a problem where the endocannabinoids fail to do their job
   b. Supplementation may help
   c. Acupuncture has been shown to help as well
12. Sources of CBD
   a. Hemp plants absorb heavy metals, toxins and radiation from the soil at a high rate
   b. China produces 1/4 of the world’s hemp
      i. Very high percentage has heavy metal contamination
   c. US hemp is high quality but not allowed to be used commercially
   d. European hemp is the best and safest option
13. Production of Hemp
   a. Cold Press extraction produces low CBD
   b. CO2 extraction is best
      i. Most effective and safest extraction method
      ii. Similar to decaffeinating coffee
      iii. Expensive method
To ensure the health and well-being of pet dogs and cats, examination of feces for parasite eggs, oocysts, and cysts is an important part of the daily routine for most veterinary practices. Many different procedures and techniques are used, each with its own advantages and limitations. Direct fecal smears are useful for detecting motile protozoa, and sedimentation examinations are useful for recovering heavy (e.g., Physaloptera spp) or operculated (e.g., fluke) eggs that do not float well because of the hypertonic effects exerted by the flotation solution. The methods most frequently used to recover parasite eggs, oocysts, and cysts are flotation techniques that rely on the differences in the specific gravity (SG) of the egg(s), fecal debris, and flotation solution.

The SG of most parasite eggs is between 1.05 and 1.23. For parasite eggs to float, the SG of the flotation solution must be greater than that of the eggs. Ideally, all helminth eggs and protozoan cysts and oocysts would float and still maintain their morphologic integrity while fecal debris would sink in the chosen flotation solution. Flotation solutions are made by adding a measured amount of salt or sugar to a specific amount of water to produce a solution with the desired SG. Common flotation solutions include saturated sodium chloride (NaCl; SG 1.18), sugar (Sheather’s solution; SG 1.27 to 1.33), sodium nitrate (NaNO3; SG 1.18 to 1.2), magnesium sulfate (MgSO4; SG 1.2), and zinc sulfate (ZnSO4; SG 1.2). These solutions are effective, easy to make or commercially available, and relatively inexpensive.

Flotation procedures vary from the simple to the complex. The simplest procedure involves mixing a small amount of feces with flotation solution in a cylinder (shell vial or centrifuge tube) and adding solution until the cylinder is nearly full. The preparation is then allowed to stand until the eggs float to the top, and a sample from the top is removed to a microscope slide using a tool such as a wire loop, straw, needle hub, or glass rod. A refinement of this method involves filling the cylinder until a slight positive meniscus is formed and placing a glass coverslip over it. Again, the cylinder is allowed to stand until the eggs have had time to float to the top, and the coverslip is then removed to a microscope slide and examined. Several commercial apparatuses that use a screen to retain debris from floating to the top are variations of the simple shell vial technique.

A further refinement of the flotation technique involves centrifugation to spin down the debris and allow the eggs to float to the surface of the solution where they can be recovered. If a fixed-angle centrifuge head is used, the centrifuge tubes cannot be filled completely and thus should be removed from the centrifuge after spinning and placed vertically in a test tube rack. If a swing-head centrifuge is used, the tubes can be filled to a slight positive meniscus and covered with 18- or 22-mm² coverslips before centrifuging. When tubes are spun with coverslips in place, care should be taken not to open the centrifuge before it stops spinning, or the coverslips can shift and ruin the preparation. Veterinary hospitals usually use one or more of these methods based on cost, ease of use, availability of hardware, or simply tradition.

The Ovassay method with 1.1-SG ZnSO4 solution readily floats, hookworm (A. caninum) eggs (SG 1.0559); however, ascarid (T. canis) eggs (SG 1.0900) may not be recovered and whipworm (T. vulpis) eggs (SG 1.1453) are virtually impossible to float with such a solution. This points out the necessity for using care in weighing the salts and measuring water when preparing flotation solutions and for assuring proper SG by testing the solution with an SG hydrometer. When the SG of the salt solution (ZnSO4) is raised to 1.2, T. vulpis, and T. canis eggs are recovered in the Ovassay but in fewer numbers than with a centrifugation method using either ZnSO4 or sugar. A centrifugation method will recover significantly higher fecal counts compared with the Ovassay method. For A. caninum, a centrifugation method using 1.2-SG NaNO3 solution results in significantly higher fecal egg counts than the simple flotation method, which is allowed to stand for 5 or 10 minutes. A 15- or 20-minute simple flotation method recovers significantly similar fecal counts as compared with the centrifugation method. With low numbers of T. vulpis eggs the 5’ and 10’ simple floats can miss eggs in 2 out of 3 samples.

Over the past decade a number of studies have been conducted to evaluate and compare the performance of various fecal diagnostic techniques. From 2000 to 2004, students at KSU evaluated 206 fecal samples known to contain hookworm (A. caninum) eggs. When all hookworm data were combined, the direct smear technique failed to detect hookworm eggs 72.82% of the time. The Ovassay and centrifugation techniques yielded false-negative results 4.85% and 0.97% of the time, respectively, and recovered more than 50 eggs/slide 36.41% and 74.76% of the time, respectively. When all ascarid data were combined, the direct smear technique failed to detect ascarid eggs 85.38% of the time. The Ovassay and centrifugation techniques yielded false-negative results 32.02% and 4.93% of the time, respectively, and recovered more than 50 eggs/slide 36.41% and 74.76% of the time, respectively. When all whipworm data was combined, the direct smear technique failed to detect whipworm eggs 92.61% of the time. The Ovassay and centrifugation techniques yielded false-negative results 32.02% and 4.93% of the time, respectively, and recovered more than 50 eggs/slide 2.96% and 23.65% of the time, respectively.
Evaluations of centrifugation fecal techniques and IDEXX SNAP® Giardia fecal antigen test kits of puppy fecal samples by 2nd year veterinary students showed that almost half (56/116) of the fecal samples were recorded as positive for Giardia. The direct smear technique detected the fewest number of positives with students recording only 4 positive samples. This data may be artificially low since the fecals were collected several hours prior to laboratory and trophozoites may have been dead by time of examination. Students recorded that the SNAP® Giardia fecal antigen test identified 55 of 116 samples as Giardia positive and ZnSO₄ centrifugation technique recorded 45 of 116 samples as positive.

At a wet lab conducted at the Central Veterinary Conference in 2005 twenty-seven (27) participants returned completed fecal data forms. When a centrifugation fecal flotation technique was compared to passive flotation technique the data demonstrated that centrifugation with either 1.18 sp. gr. ZNSO₄ or 1.27 sp. gr. Sheather’s sugar solution routinely recovers more eggs and oocysts than the passive Ovassay technique. Not only did the centrifugation technique recover more eggs and oocysts in addition the participants recorded many more samples as positive with the centrifugation technique. Strikingly only once (T. canis – Ovassay - ZNOSO₄) did the Ovassay technique recover all parasites in all samples, while only once did the centrifugation technique fail to recover all parasites in all samples. In the group that used 1.18 sp. gr. ZNSO₄ solution only 2 of 14 participants recovered Taenia sp. eggs. While in the group using 1.27 sp. gr. Sheather’s sugar solution all 13 participants recovered Taenia sp. eggs using.

Even though the participants knew the samples were positive for Giardia recovery and identification of Giardia sp. oocysts was problematic for the 27 participants regardless of technique. Only 6 of the 27 participants were able to recover and identify Giardia sp. oocytes from a known positive sample. One participant each using the Centrifugation with ZNSO₄, Ovassay with ZNSO₄ and Ovassay with Sugar was able to recover and identify Giardia sp. cysts. Three participants using the Centrifugation with Sugar were able to recover and identify Giardia sp. cysts. All 27 participants had a positive SNAP® Giardia fecal antigen test on the mixed sample.

As part of a weeklong clinical Parasitology training program, veterinarians participated in a wet-lab evaluating fecal examination techniques.³ Three classes were offered during 2010, 2011 and 2012, for a total of 9 classes that included 56 participants. Fecal samples were collected from dogs at the local animal shelter, verified as positive for various parasite diagnostic stages and mixed to form composite samples. While species of parasites in fecal samples varied, all 9 classes evaluated samples that contained A. caninum, T. canis and T. vulpis eggs. Each participant conducted a direct smear, an Ovassay using a 1.18 sp. gr. ZnSO₄ solution, a centrifugation procedure using 1.18 sp. gr. ZnSO₄ solution and a centrifugation procedure using 1.24 sp. gr. sugar solution. Using the direct smear technique, participants recovered T. canis, T. vulpis and A. caninum eggs 30.4% (17/56), 26.8% (15/56) and 30.4% (17/56) of the time, respectively. The Ovassay recovered T. canis, T. vulpis and A. caninum eggs 57.1% (32/56), 41.1% (23/56) and 87.5% (49/56) of the time, respectively. The centrifugation procedure with ZnSO₄ recovered T. canis, T. vulpis and A. caninum eggs 94.6% (53/56), 85.7% (48/56) and 100% (56/56) of the time, respectively. The centrifugation technique with the sugar solution recovered T. canis, T. vulpis and A. caninum eggs 96.4% (54/56), 100% (56/56) and 100% (56/56) of the time, respectively. When the Ovassay technique was only used, 33.3%, 11.1% and 44.4% of the time did every participant recover T. canis, T. vulpis and A. caninum eggs, respectively. When the participants used the centrifugation procedure with sugar solution, every participant in every class recovered eggs of T. vulpis and A. caninum and 77.8% of the time every participant recovered eggs of T. canis.

Addition of Fecal Antigen Testing to your Standard Diagnostic Procedures. New methodology for detecting protein biomarkers secreted or excreted by nematodes in the intestinal lumen. Unique biomarkers now available for ascarid, hookworm, and whipworm. These biomarkers are produced by the worms and not the eggs. Beneficial because they can overcome issues with misidentification, spurious eggs from coprophagy and even prepatent periods.⁹

REFERENCES


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What Are We Going To Do About All These Ticks!

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While often the same products are used to combat ticks as are used to combat fleas, there are substantial differences between flea and tick control. One of the major differences is in the number of species that confront a dog. While there is one predominant flea species that infests dogs in North America, the Cat flea (Ctenocephalides felis), there are at least 10 different tick species that may be encountered. There can be remarkable regional variability in the number and diversity of tick species that infest dogs. \(^1\) While practitioners in Hawaii may only deal with one tick species infesting dogs (Brown Dog tick, Rhipicephalus sanguineus), practitioners in New Mexico may encounter three different species, in California six different species and in Kansas up to seven different tick species. This wide diversity in tick species means that ticks occur at different times of the year, are associated with different reservoir hosts and carry and transmit different diseases.

Over the past few decades there has been a change in the distribution and abundance of certain tick species in North America. \(^1\) Two of the best documented are Amblyomma americanum and Ixodes scapularis. \(^2\) \(^4\) Since both these ticks are important vectors of human and animal pathogens these changes in distribution and abundance have had a marked effect upon both human and animal health. Various factors have contributed to tick population movement including; changes in agricultural practices, reforestation, wildlife conservation, relocation and restocking, climate fluctuations and decreased environmental pesticide application.

Specific factors that have contributed to the increased range of A. americanum include increased habitat via reforestation and its wide host range that includes deer, small mammals, birds and man. \(^3\) \(^4\) The White-Tailed Deer is considered a preferred host for A. americanum, and all life stages will feed successfully upon White-Tailed Deer. Another species that utilizes similar habitats and is an excellent host for larvae and nymphs is the wild turkey. Areas with high White-Tailed Deer and wild turkey populations can have remarkably large populations of A. americanum. Similar to A. americanum the distribution of I. scapularis is linked to the distribution and abundance of the white-tailed deer. \(^5\)

Ixodes scapularis is widely distributed in the Eastern and Central U.S. in at least 35 state. \(^5\) \(^6\) Its distribution is from Florida to Maine, west into far eastern South Dakota, and south through eastern Kansas into central Texas. Ixodes scapularis is also located in central and eastern Canada.

Seasonal activity varies by geographic region, but larval activity is generally highest in August and September. Larvae attach to and feed on a wide variety of small mammals, including mice, chipmunks and shrews. Larvae also feed on birds and lizards. The white-footed mouse (Peromyscus leucopus) is of particular importance in the tick life cycle and disease transmission, because it serves as a good host for larval I. scapularis and it is a major reservoir of Borrelia burgdorferi.

Immature ticks typically engorge for 2 to 4 days before dropping off to molt in moist protected areas such as under leaf litter in forested habitats. Larvae over-winter and then molt to nymphs in the spring. Nymphs will feed for 3 – 4 days on a variety of hosts including mice, squirrels, chipmunks, raccoons, opossums, skunks, shrews, cats, birds, and humans. Nymphs occur primarily from May through July in the North. Adults occur most commonly from October through December. Adults that do not find a host will quest again, typically from March to May. Adults feed for 5 – 7 days, primarily on white-tailed deer, but also on bobcats, cattle, coyotes, dogs, foxes, horses, humans, opossums, raccoons and other mammal.

While recent pharmaceutical advances have been made in control of flea reproduction, such advances in the area of tick control are lacking. With the exception of the brown dog tick Rhipicephalus sanguineus, our ability to manage tick reproduction is limited, if not almost non-existent. As discussed previously in most flea infestations we have the opportunity to control flea reproduction by either killing fleas before they can reproduce or killing flea eggs. However, it is not just because we have effective residual insecticides, insect growth regulators or insect development inhibitors that we are successful, it is also due in large part to the fact we can often target the primary reproductive host, the flea infested dog or cat. And interestingly, failures in flea control often occur when flea infested feral pets or flea infested urban wildlife invade the owners yards.

But when dealing with most 3-host ticks the problem is that the majority of the reproducing ticks are not on the dogs or cats, but on their nature wildlife hosts. Since we are limited in our ability to manage ticks on wildlife, reinfestations are a common occurrence and protracted use of acaracides as preventives is routine in many areas.

Since tick control can be extremely difficult and because they are vectors of a variety of bacterial and protozoal diseases veterinarians should have an understanding of the ecology of the tick(s) encountered in the area in which they practice. Veterinarians need to be educated on the various aspects of tick ecology, disease transmission and control methodologies so that they can then educate their staff and pet owners.

Numerous studies demonstrate the high level of efficacy of the various acaracides but the residual activity is rarely 100% and the efficacy of products varies between and as well as within species, even in the same laboraotry. \(^5\) \(^16\) Evaluations of acaracides under natural or field conditions further illustrates that while efficacy is good it is not 100%.
In a field efficacy trial conducted in Kansas U.S.A, an imidacloprid (8.8% w/w)-permethrin (44.0% w/w) formulation was evaluated on dogs against naturally occurring populations of *Amblyomma americanum*. When dogs were walked in a naturally tick infested environment the 48-hour post-exposure efficacy of imidacloprid-permethrin formulation was 93.5%, 98.9%, 94.6%, 94.1% and 96.6% on days 3, 7, 14, 21 and 28 respectively, post-treatment.\(^{14}\)

Variation in product efficacy occurs. In two studies conducted at K-State, different results were found when evaluating the efficacy of acaricides against *Dermacentor variabilis* infestations in dogs from two different regions of the USA.\(^{9,12}\) In the first study, the efficacy of imidacloprid–permethrin and fipronil–(s)-methoprene formulations were evaluated against a *D. variabilis* isolate from California. The 48-h post-infestation efficacy on day 30 post-treatment was 92.0% and 83.2%, respectively, for the imidacloprid–permethrin and fipronil–(s)-methoprene formulations. In the second study, the 48-h post-infestation efficacy on day 30 for the imidacloprid–permethrin and fipronil–(s)-methoprene formulations against a *D. variabilis* isolate from Oklahoma was 17.5% and 75.7% respectively.

One combination spot-on product that produces more prolonged and pronounced efficacy is fipronil-amitraz. In a study conducted at K-State, the efficacy against *Dermacentor variabilis* 30 days after treatment was 99.4%.\(^{13}\)

Recently a new class of insecticide/acaricide has provided the first orally administered approach to tick control. Afoxolaner, fluralaner and sarolaner are members of the isoxazoline class and work by inhibiting insect GABA and Glutamate-gated chloride channels leading to hyper-excitation and death of insects and arachnids.\(^{17-19}\)

While product efficacy is often excellent in most studies, significant variation in efficacy can occur and 100% control is rarely achieved. Therefore, it can be expected that under natural conditions in areas where dogs are being frequently exposed to ticks pet owners will see ticks on treated dogs. We might also expect that efficacy in real world situations might be lower due to such factors as bathing and swimming, differences between dog breeds and haircoat types and frequency and correctness of product application.

Since 100% tick kill is rarely achievable, perceived efficacy of acaricides may be directly related to the numbers of ticks to which dogs are exposed. If a dog is treated with one of these highly efficacious acaracides and encounters just a few ticks it is likely all those ticks will be killed. However, if tick exposure is considerably larger, we can expect a few ticks to be observed on these dogs and pet owners may perceive a lack of efficacy. Therefore, in areas where tick populations are increasing the perception may be that the products are not as effective as they once were.

Pet owners often view tick infestations of their pets differently than flea infestations.\(^{12}\) Whether this is due to concerns about tick transmitted diseases or simply a phobia, the presence of a couple of ticks on the pet often elicits a more pronounced negative reaction than the presence of a couple of fleas. A 95% effective flea product may provide great client satisfaction while a similarly effective tick product may be perceived as a failure. Therefore, it is not uncommon that label recommended application of a product does not appear to control the problem. This may be real or perceived, based upon pet owner expectations of product performance. Given pet owner concerns, a need to reduce tick borne disease and lack of 100% efficacy; occasionally additional control measures are needed.\(^{12,14}\) If additional control measures are deemed necessary, pet owners need to be educated as to why additional control measures are necessary and notations made in the pets record before extra label uses are conducted.

One of the most common practical attempted solutions to this problem in dogs is to increase the frequency of application. Here increased residual efficacy is the expected outcome, since you are increasing the residual acaricides levels with the shorter application intervals. Additionally, with many 3-host ticks destruction of tick habitat can reduce exposure pressure. Areas that serve as refuge for ticks and wild mammals such as grass, weeds, and brush piles, between runs and along buildings, can be eliminated or treated with an approved acaricide.

In some situations, especially in tropical and subtropical regions and in climate controlled kennels brown dog ticks may infest buildings with ticks crawling up walls, curtains and throughout the home or kennel.\(^{14-15}\) In these situations acaricides may need to be sprayed indoors into cracks and crevices, behind and under furniture or cages and along walls and the ceiling. Following application, make sure the acaricide is dry before you allow animals or humans back into the premises to minimize toxicity problems. Finally, restricting pet access from tick-infested environments may be necessary.

It is apparent that the range and local density of certain tick species has increased in many areas. Whatever the factors it must be recognized that tick infestation pressure may be much higher and associated tick transmitted diseases may be more prevalent in some locations today than in the past. The increase in tick populations means that pets are encountering ticks more frequently, are exposed to more ticks per encounter and clients may be seeing more ticks on their pets than in the past. Since tick products do not kill or repel all ticks instantly, clients may get the false impression that the products are not performing as well as in the past. These situations necessitate that veterinarians set client expectations, before clients set their own unrealistic expectations of control.

References


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10. Dryden MW, Payne PA, Smith V, et al. Evaluation of an imidacloprid (8.8% w/w)–permethrin (44.0% w/w) topical spot-on and a fipronil (9.8% w/w)–(S)-methoprene (8.8% w/w) topical spot-on to repel, prevent attachment, and kill adult Ixodes scapularis and Amblyomma americanum ticks on dogs. *Vet Ther* 2006, 7(3):173-86.


To understand the relationship of Wolbachia to Dirofilaria immitis, we must have a detailed understanding of the life history and pathogenesis of canine heartworm.

Adult *Dirofilaria immitis* naturally in pulmonary arteries and occasionally the right heart; occasionally aberrant migrations to other locations in the body. Showing adult worms in right ventricle of a dog’s heart to a client may be an effective tool but it is typically a post-mortem finding). Female *D. immitis* in pulmonary arteries and occasionally the right heart deposit microfilariae into circulation. Microfilariae may survive up 3.5 years in the vascular system. Mosquitoes (>70 species worldwide; approximately 25 North America) I.H. become infected when they feed on an infected dog and consumes blood containing the microfilariae (L3). Microfilariae then develop first in mid-gut & then in malphgian tubules of the mosquito from the L1 – L2 – L3 infective stage within 13 to 30 days. L3 infective larvae then migrate to the salivary glands of the mosquito. Infected mosquito bites dog and deposits infective L1 larvae in or around the bite wound. L1 reside in subcutaneous tissues and molt to the L2 (1.5mm) in subcutaneous tissues within 3 – 12 days. L2 reside in subcutaneous tissues or muscle of abdomen or thorax and molt to the adult juvenile adults 1.2-1.5cm) within 45 – 70 days (Kotani & Powers, 1982). Immature adults (2 – 4cm long) migrate to pulmonary arteries and heart by 70 – 90 days P.I. Worms mature (12 – 30cm) and then male and female *D. immitis* mate and females begin depositing microfilariae (L1) within 6 months (rarely) but more commonly 7 – 9 months P.I. In dogs adult *D. immitis* may live 5 – 7 years

Pathogenesis – caused by adults is initially primarily a lung disease. Progressive pulmonary hypertension associated with pathological changes in the pulmonary arteries. Vascular endothelium damaged by repeated embolisms of defunct adults and inflammation associated with antigens released from worms. Platelets and WBC adhere to damaged vascular endothelium and release platelet derived growth factor. The interna and medial smooth muscle proliferate to cause arterial obstruction and increased vascular permeability with leakage of plasma into the lung. Arterial walls of smaller arteries and arterioles thickened. The pulmonary arteries dilate, become tortuous, and lose their normal tapering. Pulmonary artery obstruction, pulmonary hypertension, endarteritis, obstructive fibrosis and right ventricular hypertrophy. Prolonged presence of the parasites and, more likely the pulmonary arterial morphologic lesions are required to produce pulmonary hypertension (adults).

The Relationship of Heartworm, Wolbachia and Doxycycline.

Most filarial nematodes, including all *D. immitis*, harbor symbiotic, intracellular, gram-negative bacteria *Wolbachia pipientis* (Rickettsiales). These bacteria are symbionts that may be necessary for functional reproduction in female *D. immitis* and development in immature stages. *Wolbachia sp*. proteins have been implicated in the pathogenesis of heartworm disease in dogs and cats. *Wolbachia pipientis a* symbiotic, gram negative intracellular bacteria that is closely related to rickettisa. Identified in human and animal filarial nematodes. Heartworm endosymbiont - All *D. immitis* parasites harbor *Wolbachia*. *Wolbachia* organisms are maternally transferred from one filarial generation to the next. Bacteria are present in all life stages of the parasite. *Wolbachia* are released in large numbers at death of parasite and during production and release of microfilariae. Generally, filariae free of *Wolbachia* after treatment with tetracyclines show inhibition of maturation, survival and reproduction. It has been shown that a combination of doxycycline/ivermectin is microfilaricidal and adulticidal. Weekly doses ivermectin (6 mcg/kg) + doxycycline 10 mg/kg/day from weeks 0-6, 10-12, 16-18, 22-26, 28-34. No microfilaraemia after week 12. Antigen levels decreased in some dogs by week 24. Ivermectin/doxycycline 78.3% adulticidal effect. Much higher than ivermectin or doxycycline alone (Bazzocchi et al. 2008). In a separate study Italian study – dogs naturally infected with heartworm were administered doxycycline (10 mg/kg SID x 30 days) and ivermectin (6 mcg/kg every 15 days for 180 days). 100% microfilaria negative by day 90 & 72.7% antigen-negative by day 300 (Grandi et al. 2010). In yet another study Doxycycline administered at 10 mg/kg BID for 30-day periods to determine efficacy against larval and immature stages. Group 1, day 0-29; Group 2, day 40-69; Group 3, day 65-94; & Group 4, untreated controls. Dogs in group one 0 no microfilariae and no adult heartworms. Group 2 & 3 98.4% and 69.6% reduction in total worm burden, respectively (McCull 2011). L3 from mosquitoes fed on dogs treated with doxycycline. Normal appearance and motility, but unable to develop in dogs. Prevented further transmission of disease (McCull 2008). Effect of pretreating with doxycycline before melarsomine. Dogs experimentally infected with adult HW. Group 1 doxy 20 mg/kg SID x 30 days, melarsomine as above, ivermectin 6 mcg/kg monthly for 24 wks post infection. Group 3 melarsomine alone. NO pulmonary thromboembolism in any dogs treated with doxycycline. In dogs treated with doxycycline and ivermectin, arterial lesions almost completely absent (Kramer et al. 2011).


Dirofilaria immitis – See migrating heartworm proceedings
Toxoplasma gondii—See Toxoplasma: microscopic monsters proceedings

Paragonimus spp. – Lung fluke
- Life cycle 1st IH – snail, 2nd IH - crayfish and crabs (crustaceans) DH - mammals (dogs, cats)
- PPP – 1-2 months
- Adults – 7- 12 x 4 - 6 mm
- Eggs – 75-118 x 48 - 65 μm
- Diagnosis - Sedimentation/Sugar Float

Clinical Signs/Pathologic changes
- Respiratory problems
- Cough
- Lethargy
- Pneumothorax

TREATMENT
Praziquantel – higher dose/repeated
Fenbendazole – 14 days
- CONTROL/PREVENTION
- Uncooked Crayfish/Crab

Metastrongyloidea- “Lungworms”
- males with a caudal copulatory bursa
- buccal cavity small
- usually leave the definitive host as larvae rather than eggs
- usually live in the lungs of mammals
- life cycles commonly indirect (snail(slug intermediate hosts typical)
- migratory in definitive host
- Clinical signs
  - Coughing
  - Moderate to severe dyspnea
  - Loud breath sounds
  - Fever

Diagnosis- Zinc sulfate float and/ or Baermann exam

Dictyocaulus - large lungworm
D.H. - cattle, sheep, goats, horses, and other herbivores
D. viviparum - cattle
D. filaria – sheep, goats
D. arnfieldi – equids (donkeys)

Muellerius - hair lungworm
- I.H. - snails, slugs
- D.H. - sheep, goats
  - ♂ 11 - 14 mm
  - ♀ 19 - 23 mm
- L1 250-300 μm

Aelurostrongylus – feline lungworm
- I.H. - snails and slug
- Paratenic Hosts- amphibians, reptiles, birds, rodents
- D.H. - cats (felidae)
-♂ 4 - 6 mm    ♀ 9 - 10 mm
-L1 350-400 μm
-PPP- 6 weeks
-Treatment (EXTRA LABEL)
  -Fenbendazole (Panacur -10 days)
  +/- Ivermectin/Selamectin
  -Advantage Multi (Moxidectin) & Profender (Emodepside/Praziquantel)
  -Prednisone (1 mg/kg PO BID for five days)

**Angiostrongylus vasorum**
-fox lungworm/ French Heartworm
-I.H. - snails and slugs (mollusks)
-Paratenic Hosts- amphibians, reptiles, birds, rodents
-D.H. – fox, dog
-♂ ,♀ 14 to 20 mm (♀ barber pole)
-L1 310-400 μm - anterior cephalic button with a dorsal spine
-PPP- 7 weeks

**Capillarids**
-Eggs with polar plugs Size 50 - 80 x 20 - 40 μm - often mistaken for *Trichuris* spp eggs
-Clinical signs include sneezing, coughing , respiratory distress
-Diagnosis fecal float
-*Eucoleus aerophilus* – lungworm
  D.H. - dogs, cats, foxes, raccoons
-*Eucoleus boehmi* - nasal worm
  D.H. - dogs, fox, (cat)
-Treatment– Ivermectin, Fenbendazole, Moxidectin

**Miscellaneous migrating larvae**
-Ascarid spp in general have a lung migration during larval phase. High level of infection in young animals can lead to respiratory disease. Can attempt fecal float for diagnosis but may be during pre patent period so may obtain negatives on fecal floats
-Toxocara canis-dog
-Toxocara cati-dog
-Ascaris suum – pig
-Parascaris -equine
Migrating Heartworm: Diagnostics and Treatment
Richard Gerhold, DVM, MS, PhD
University of Tennessee
Knoxville, TN

Dirofilaria immitis – heartworm
I.H. – mosquitoes
D.H. - dogs and wild canidae, marine mammals, ferrets, cats
♂ 12-22 cm (6-9 inches)
♀ 25-31 cm (12-14 inches)
Mf 300 - 325 x 6 - 7 μm
PPP 6 months

Life cycle
- Juvenile worm matures to adult over next 3 months in dog.
- Microfilaria produced by young adult worms 6 months post infection (6 month Life Cycle)
- Male worms 6-9 inches, females 12-14 inches
- Lifespan is 5 to 7 years in the dog
- Average infection is 14 worms but can be in excess of 100

Clinical signs
- Cough
- Dyspnea
- Tiring on exercise
- Weight loss
- Classic patient: Active middle-aged dog
- Ascites
- Anemia
- Eosinophilia and thrombocytopenia
- Glomerulonephritis and proteinuria

Reasons for a dog to be AG positive and Knott’s/Filter negative
- 5 month old worms (too young –rem PPP)
- All female worms (single sex)
- Immunological Occult
- Prophylaxis/Drug induced
- Few mf present

Reason for a dog to be MF positive and AG negative
1. Adults dead/mf circulating
2. Ag sequestration/antigen antibody complexes

Time of testing
- The earliest heartworm antigen is detected is 5 months post infection
- With low worm burdens or animals on macrocyclic lactone preventives, antigenemia can be delayed to 9 mos.

What tests are recommended during annual physical exam?
1. Serology for heartworm antigen AND
2. Microfilariae concentration test
   a. Same two diagnostic tests are recommended for dogs displaying clinical signs suggestive of heartworm disease
   b. Notes on testing recommendations from AHS
- Antigen testing - most sensitive diagnostic method when screening an asymptomatic dog or seeking verification of a suspected heartworm infection
- But a study conducted on shelter dogs found a 7.1 percent false negative rate because of formation of antigen-antibody complexes.
- AHS now recommends mf testing in tandem with AG to detect dogs that are AG- but mf+.
What would you do before treatment?

- Evaluate the dog
  - Already have results from Knott’s & Ag test
- Radiography to assess severity of cardiopulmonary disease
  - Enlarged, tortuous, and often truncated peripheral intralobar and interlobar branches of the pulmonary arteries, particularly in the diaphragmatic (caudal) lobes
  - Pulmonary parenchymal disease, right heart enlargement etc
- Echocardiography

Stabilize dogs presenting with clinical heartworm disease

Treatment AHS/CAPC -3 immiticide dose regimen

- Safety
- Efficacy
- Decreased possibility of needing further melarsomine treatment
- By initially killing fewer worms and completing the treatment in two stages
  - Reduces cumulative impact of worm emboli on severely diseased pulmonary arteries and lungs

Current treatment protocol for positive dogs

First month

- Start macrocyclic lactone (preventive) and continue monthly for life
- Rx Doxycycline 10mg/kg bid for 4 weeks

If dog can not tolerate dose, reduce to 5mg/kg

(Wolbachia nos will remain low for 3 to 4 mos)
  - If dog symptomatic, Rx Prednisone 1mg/kg reducing weekly during 1st month.
  - Begin exercise restriction.

Second month

- Give second dose of heartworm preventive.

Third month

- Give third dose of heartworm preventive.
- Give one injection melarsomine (Day 61).
- Rx Prednisone 1mg/kg reducing weekly.
- Decrease activity level even further. Cage rest in more severe cases.

Fourth month

- Give fourth dose of heartworm preventive
- Give second and third melarsomine injections (Day 90 & 91).
- Rx Prednisone 1mg/kg reducing weekly for four additional weeks.
- Continue exercise restriction for 6 to 8 weeks after last melarsomine injections.
- Antigen test in 6 months
- Knott’s test or other test for microfilariae in 6 months
- Any treatment method utilizing only macrocyclic lactones as a slow-kill adulticide is not recommended!!!
- New information about resistance also prompted the AHS to place additional emphasis on the importance of year-round administration of heartworm preventives.

Diagnostic tests in cats

- Use both antigen and antibody test
- Antigen Test Kits
  - Only detects adult female worms.
  - Average worm burden in the cat is 1-2 worms and is frequently only males.

“Asthma” like syndrome occurs 3-4 months post infection. Antigen test incapable of confirming HW as etiology

- Antigen tests:
  - Detect antigen produced by adult worms (Produced by adult, female worms)
  - First detection at 5- 8 months P.I.
- Antibody tests:
  - Detect antibody produced against larval and adult worms
  - First detection at about 3 months
Feline heartworm treatment goals

- Relieve acute signs (usually respiratory) May be due to adult or larval infection
- Control chronic signs (respiratory, vomiting)
  - Prednisone (2mg/kg-decreasing doses one month)
- Prevent reinfection- prophylaxis
- Rid patient of Adults via surgery (possible? advisable?)
Neospora, Toxoplasma, and Coccidia: Microscopic Monsters
Richard Gerhold, DVM, MS, PhD
University of Tennessee
Knoxville, TN

Hosts/Disease
- Cats serve as definitive hosts and numerous mammals and birds are the intermediate hosts
  - Most cats in the wild become infected shortly after weaning
  - Mice are the usual intermediate host and a normal predator-prey relationship exists between the cats and mice that enhances transmission
- Causes toxoplasmosis

Morphology
- Oocysts are unsporulated in fresh feces
- 12 x 10 μm (11 – 13 x 9 -11)
- Sporulated oocysts contain two sporocysts each with 4 sporozoites (2 x 4 architecture)
- In the environment, sporulation occurs in 1 – 5 days; under favorable conditions, sporocysts can survive about 18 mos.; can survive in fresh and salt water

Life cycle stages
- Sporozoites- form within oocysts
  - IH ingests oocysts, one means of infection
- Tachyzoites (“fast” merozoites)-form rapidly in tissues of intermediate host(s)
  - Tachyzoites form first in epithelial tissues of intestine
  - Disseminate to other tissues for further rapid development
  - Initial, acute infections
  - Within host cells, tachyzoites are contained within a parasitophorous vacuole
- Bradyzoites (“slow” merozoites)- develop slowly as immunity develops
  - immune cytokine production is thought to induce differentiation from tachyzoites to bradyzoites
  - form slowly in tissues of intermediate host(s)
  - Can remain viable for life of host, chronic infection (quiescent)
  - Found in large, cyst-like accumulations
  - Bradyzoites are infective upon ingestion

Pre-patent period (PPP) in cat
- Varies with stage ingested
  - 3-5 days when bradyzoites are ingested
  - 5-10 days when tachyzoites are ingested
  - 20-24 days when oocysts (sporozoites) are ingested

Epidemiology
- Seroprevalence of T. gondii
  - Serology is not useful in predicting shedding of oocysts by cats → oocysts shed prior to antibody formation
- Infection routes for cats
  - Carnivorism (primary)
  - Transplacental
  - Oocyst ingestion (lowest)
  - Cats can be both definitive and intermediate hosts
    - If intermediate host, usually see lung infections and pneumonia in cats

Human epidemiology
- Fecal-oral ingestion of oocysts (primary way humans are infected in US)
- Ingestion of tachyzoites and/or bradyzoites in undercooked meat and raw milk (goat’s milk esp., unpasteurized), congenital
- Organ transplant

581
• Blood transfusions (much less common)

Pathology & pathogenesis
• Pathology varies with strain of parasite, age of host, organs invaded, immune status of host, species of host
  o Enteritis
  o Hepatitis
  o Pneumonitis
  o Myocarditis
  o Chorioretinitis
  o Encephalitis
  o Placentitis
  o abortion

Clinical signs of congenital infection
• *T. gondii* naïve woman stands a 20-50% probability of passing infection to fetus if infected during pregnancy
• Earlier infection, more damaging to fetus

Diagnosis
• Intestinal (cat)
  o Oocysts in fresh feces (possible diarrhea)
  o Few cats shedding at any one time
• Serologic Dx
  o **Positive serological result does not correlate with shedding in cats**!
  o Can use serology for other mammals and birds performed at UTCVM
  o Acute v. past; significant increase in IgG titer in 2-3 week time span- look for rising titers

Control
• Keep cats indoors
• Discourage feral cat colonies and educate owners about *Toxoplasma* risks due to predation of intermediate hosts
• Keep cats away from livestock
• Keep cats away from sand boxes & public parks, and beaches
• Adequately cook meat
• Freeze meats before eating- freezing kills tissue cys

*Neospora caninum*

Hosts/Disease
• Causes neosporosis
  o CNS disease in dogs, cats, cattle, sheep, etc.
• In 1998, strain in cattle found to use dogs as DH; dogs can also be infected with tissues stages
• No human cases to date, not considered a human pathogen, not zoonotic
• Causes abortion in cattle, sheep, goats, etc.

Oocysts
• Identical to *Toxoplasma*
  o Remember only cats shed *Toxoplasma* oocysts in feces
• 11 x 12 μm

Life cycle sequence
• Sporozoites (in dog feces, ingested by IH)
  ↓
• Tachyzoites (travel to various tissues via blood)
  ↓
• Bradyzoites (develop in various IH tissues, cysts in brain only)
  ↓
  » Cyst wall thicker than *T. gondii*
• DH (dog) eats IH with bradyzoites

• Bradyzoites initiate asexual schizogony (tachyzoites), eventually a sexual

• Dogs shed few oocysts, make it difficult to study, much to be learned yet about life cycle

Pathology/Pathogenesis of neosporosis
• CNS & systemic disease in dogs, cats, cattle, sheep, etc. (not humans)
• Can be fatal in dogs, esp. congenitally infected dogs

Clinical Signs (dogs)
• In transplacentally infected puppies hindlimb paresis & weakness are typical presentations
• In adult onset disease:
  o Nodular dermatitis
  o Pneumonia
  o Urine and fecal incontinence
  o Hepatitis
  o Myocarditis
  o Myositis

Clinical signs (cattle)
• Clinical signs seen in calves; only clinical sign in cows is abortion
• Major cause of abortion in U.S.,

Diagnosis
• Oocysts in feces of dogs (canids)
• Serology;
  o ELISA
  o IFA
  o Neospora agglutination test (NAT)
  o Also used on bovine sera
• PCR

Public health
• Not considered a human health concern
Tick-borne diseases are extremely important and emerging diseases in the United States in both humans and animals. The area in which you live will influence the diseases that are circulating in the environment. Although diseases such as Lyme disease has received a great deal of attention, other important diseases including ehrlichiosis, Rocky Mountain spotted fever, anaplasmosis and cyauxzoonosis have been emerging in various areas. A good travel history is imperative given various species of ticks and tick-borne diseases are more common in certain geographical areas. More information on tick-borne disease distribution can be found at http://www.capcvet.org/parasite-prevalence-maps/

Identification of ticks
Tick bodies are divided into two primary sections including fused head and thorax and abdomen. All adult and nymphal forms have 4 pairs legs and no antennae and all larval forms have 3 pairs of legs. The importance of determining larvae vs other stages include to determine the likelihood of tick being infected with various pathogens. Unless transovarial transmission occurs, larvae are unlikely to be infected with pathogens, while nymphs and adults have higher likelihood include with pathogens in transstadial transmission. Whereas hard ticks have scutum, soft ticks do not have scutum. Ticks are great vectors due to their ability to be persistent blood-suckers which attach firmly & feed slowly, long life spans, may be geographically widespread, resistant to environmental conditions, high reproductive potential, and can pass infective agents through egg to next generation and/or through successive stages. Ticks bites in themselves can lead to wounds and inflammation from salivary proteins. Secondary infection and disease can be due to toxicosis, local necrosis, and tick paralysis. Tick bites predispose animals to secondary attacks by myiasis-producing flies.

Soft tick have no scutum are soft, tough, leathery body, do not stay attached—instead take multiple small volumes of blood, and often feed at night.

Soft ticks include Otobius megnini (Spinose Ear Tick) transmits relapsing fever caused by a Borrelia spp. (different than Borrelia burgdorferi which causes Lyme Disease). Spinose ear ticks are more common in western states that are west of 100th meridian

Hard Ticks is largest family of ticks has a scutum (dorsal, hardened plate) that covers entire dorsum of males and forms an anterior shield in females. Hard ticks remain attached until engorged and then fall off to molt or lay eggs. General life cycle include:

- Egg → 6-legged larva → 8-legged nymph → 8-legged adult
- Oviposition (egg laying) occurs off of the host

Nymphs and adults can be identified based on visual exam but often unable to distinguish larvae without microscopic exam

Nymphs and adults are more likely to harbor pathogens than larvae—this is why you need to be able to distinguish larvae (6 legs) from nymphs/adults (8 legs).

Tick species

All dermacentor spp.
- Ornate ticks with eyes
- Basis capitulum (mouth part) is rectangular if viewed from above and has stubby palps
- Resembles Rhipicephalus (both have 11 festoons, small rectangular patterns on posterior abdomen)

Dermacentor variabilis (American dog tick)
- Eastern half of U.S. and west coast, but rare in Central US
- Dogs, cats, humans, horses, cattle, fox, rodents, and other mammals
- Can cause tick paralysis in humans, dogs, etc.
- May take as little as 3 months, with favorable conditions, or up to 2 years
- Principal vector of Rickettsia rickettsia - Rocky Mountain spotted fever (RMSF) and others in Spotted Fever Group
- Infrequent vectors of tularemia, anaplasmosis, Babesia canis, Cyauxzoon fells

Rhipicephalus sanguineus (brown dog tick)
- Wide distribution
- Rhipicephalus ticks are similar in appearance to Dermacentor, except they have a hexagonal basis capitulum. All stages parasitize on dogs and will attach to other animals, but usually not humans
- Can survive indoors for months to possibly years without a blood meal
- Domestic & kennel problem due to tropical nature of tick and because it cannot survive outdoors in North America
- Vectors Babesia canis voglei, tularemia, Ehrlichia canis, RMSF—Very important vector of RMSF in humans and dogs in southwestern US
All *Amblyomma* spp.
- Ornate ticks
- Long mouth parts & commonly 11 festoons—allows one to differentiate from *Ixodes* spp which lack festoons

### Amblyomma americanum (Lone Star Tick)
- Wide distribution, but mainly in southern U.S.
- Large silver spot at apex of scutum on females—hence name “lone star”
- All stages feed on wild & domestic animals, birds, & humans and is significant pest for humans & animals
- Can transmit *Coxiella burnetii* (Q-fever), *Ehrlichia chaffeensis*, *Ehrlichia ewingii*, RMSF,
- Vectors agent of Southern Tick Associated Rash Infection (STARI) in humans
- Cause of STARI is currently unknown—may actually be the host reaction to tick saliva—leads to swelling and pain at bite region in people.

### Amblyomma maculatum (Gulf Coast Tick)
- Southeastern US in Gulf coast region, but has expanded range recently
- Ornate scutum—often confused with *Dermacentor*—examine mouth parts to differentiate
- Adults attack nearly all animals & humans and can transmit *Hepatozoon americanum*—dog must eat tick to be infected with *Hepatozoon*

### All *Ixodes* spp.
- Inornate ticks and No festoons, has distinct anal groove anterolateral to anal orifice
- Used for identification in NON-ENGORGED tick but can’t see groove in engorged ticks—use mouth parts instead

### *Ixodes scapularis* (Black-Legged Tick)
- Wide distribution, in East, South, and Midwest U.S. Highest populations in upper Midwest and New England/midatlantic states
- Primary Lyme disease (*Borrelia burgdorferi*) vector in Eastern US and Midwest
- Vectors *Babesia microti*, *Anaplasma phagocytophila*

### *Ixodes pacificus* (California Black-Legged Tick)
- Primary Lyme disease vector in the West Coast

### Tick-borne diseases

#### Tick paralysis
Potentially fatal reaction to a paralyzing neuromuscular toxin secreted in the saliva of a female tick late in her feeding. Cattle, sheep, horses, dogs, and humans seem to be most affected.

Clinical signs include: headache, vomiting, general malaise, loss of motor function and reflexes, followed by paralysis that starts in the lower body and spreads to the rest of the body

Respiratory failure and death can result. Signs disappear rapidly when tick is removed, suggesting that the toxin is rapidly excreted or destroyed

#### Lyme Borreliosis
- Agent: *Borrelia burgdorferi*
- Animal health: Major cause of canine and equine disease, including endocarditis and joint pain. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.
- Human health: Acute and chronic diseases including joint pain, heart disease, and neurological disorders. Most cases occur in the spring and summer, during nymphal emergence, and in late fall and winter, during adult emergence.

#### Rocky Mountain Spotted Fever
- Agent: *Rickettsia rickettsia*
- Sometimes placed in “Spotted Fever” disease group
- Vector: *Dermacentor variabilis*
- Geographical distribution: Eastern US mainly. Most frequently reported tick borne disease in the eastern US. Other agents other than *R. rickettsia* can lead to spotted fever group disease in humans. Clinical signs include flu like symptoms as well as petechial hemorrhage.

#### Cytauxzoon felis
- Piroplasm of cats. Bobcats are reservoir host that is transmitted by *Amblyomma americanum*. Clinical signs: fever, dehydration, icterus, lymphadenomegaly, and hepatosplenomegaly. Treatment with atovaquone plus azithromycin.
Diagnosis: PCR, blood smear (negative blood smear does not rule out infection) since early stage only see schizonts in macrophages. Prevention: Keep cats indoors!! Use preventative for tick infestation
Anaplasma phagocytophilum:
- Intracellular rickettsia that causes human granulocytic anaplasmosis
- Infects granulocytes and leads to bleeding, fever, leukopenia,
- Clinical signs/symptoms may be worse with co-infection with Lyme or Babesia
- Vectored by *Ixodes scapularis* so same geographical distribution as Lyme Disease. Can be transmitted by blood transfusion.
- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in granulocytes.

Ehrlichia canis
- Intracellular rickettsia that causes canine ehrlichiosis
- Infects monocytes and leads to fever, anorexia, lethargy, thrombocytopenia, lymphadenopathy, edema, bone marrow suppression.
- The acute stage is mainly due to a vasculitis. *E. canis* replicates in monocytes. The infected monocytes bind to vascular endothelial cells and leads to a vasculitis
- Transmitted by *Rhipicephalus sanguineus*—worldwide distribution
- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in monocytes
- Don’t treat animals that are clinically normal but are only seropositive—potential false positive due to positive predictive value.
- Treatment with doxycycline or minocycline
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- Diagnosis: clinical signs, PCR (acute cases), serology (chronic), CBC to look for leukopenia, Blood smear to look for morulae in granulocytes.
Wildlife Diseases Important for Practitioners to Know

Richard Gerhold, DVM, MS, PhD
University of Tennessee
Knoxville, TN

Wildlife diseases can be important for both wildlife populations as well as potentially important for livestock or domestic animal and public health. A subset of diseases will be discussed that practitioners should know

a. Chronic wasting disease (CWD) or cervids- important prion disease of deer family (cervids) and leads to transmissible spongiform encephalopathy. Only cervids can be naturally infected and incubation periods can be months to years depending on host genotype. Clinically signs include weight loss, salivation, ataxia, poor hair coat, etc. Diagnosis can be performed by ELISA or immunohistochemistry. Current models suggest that CWD can lead to significant focal population decreases in various cervid species and every effort should be made to minimize the chance of the disease being introduced into areas where it does not exist.

b. Tularemia- bacterial disease of rabbits, beavers, and various rodents. The disease can progress quickly and lead to death. Lesions consists of pinpoint areas of necrosis in the liver and abscesses of the lymph nodes. The bacteria can infect humans and can be quickly fatal. Transmission can occur by aerosolization, ingestion, and vector borne.

c. Plague- Similar to Tularemia but seen in western US in ground squirrels and similar species. Cat, dogs, and humans can be infected by direct contact or via aerosolization, ingestion, and vector borne.

d. Hemorrhagic disease of cervids. Caused by Epizootic hemorrhage disease (EHD) and bluetongue (BT) viruses. Multiple serotypes of both viruses exist. The virus is transmitted by Culicoides midges. Clinical signs include edema, hemorrhage, conjunctivitis, lethargy and death. Lesions can include erosions of oral cavity and rumen. Can lead to large focal population declines in certain regions of US where disease does not occur regularly. Disease is seen in later summer. Evidence exists that cattle can be infected with the EHD virus and lead to blisters and vesicles. Sheep are susceptible to BT virus. No evidence of human infection

e. West Nile virus- Virus found in birds (particularly corvids –crows and blue jays). Can lead to sudden death or chronic disease depending on the bird species and likely other factors. Virus is transmitted from mosquitoes so care to reduce mosquito breeding areas is important in controlling the disease. WNV is zoonotic and can lead significant morbidity and mortality in humans. Dead bird surveillance is often important in determining geographical hot spots of the virus.

f. Baylisascaris procyonis- roundworm of raccoons but dogs can also act as definite host and shed eggs in feces. Aberrant hosts that ingest larvated eggs can have visceral and neural larval migrants and can lead to neurological disease and death. The parasite has been a major impact in restorations of Allegheny wood rats. Chickens, quail, mice, rodents and other animals can have disease. Furthermore humans can be infected and several humans mortalities have occurred due to the parasite. Efforts should be made to make areas around houses and livestock to be unattractive to raccoons. These efforts include not leaving pet food outside, keeping compost piles away from house and have lid on compost pile, keeping houses and attics closed off from wildlife and educating clients not to feed wildlife and not to keep wildlife as pets

g. Echinococcus spp.– Tapeworm parasitic disease of ruminates and carnivores or of rodents and carnivores. Eggs are shed by carnivores and are zoonotic if eggs are ingested. Eggs are identical to Taenia tapeworms of dogs. Lesions in ruminants consist of fluid filled sacs in the lungs and liver. The geographical distribution of the parasite may be expanding.

h. Avian influenza- viral disease of birds. Waterfowl are the natural host for low path avian influenza viruses. Viruses may mutate to high path and cause infection in poultry leading to significant mortality and morbidity. There is potential for zoonotic infection of flu in humans and there is continued concern about avian influenza virus mutating to become an epidemic level disease of humans.

i. Feeder diseases of wild birds: includes salmonella, avian pox, aspergillosis, and trichomonosis. Lesions can consist of caseous material in the oral cavity and confirmatory testing is needed to determine exact cause. Feeders and waterers should be cleaned every two weeks with 10% bleach solution to minimize disease transmission.
RACE required objectives

1. Describe the wildlife reservoirs for rabies in the United States
2. Discuss the importance of different strains of rabies in different hosts
3. Review the typical routes of transmission of rabies to domestic animals and humans
4. Describe the active role the practicing veterinarian plays in public health regarding rabies prophylaxis and response

Rabies is still a public health issue in the United States, despite the growing misperception among the public and some working in veterinary medicine, that it has disappeared. Veterinarians have a large role to play in the continued control and management of this nearly 100% fatal disease. Successful management of potential exposures in humans and companion animals depends largely upon good communication between the public health department, the practicing veterinarian, and the animal owner. Additionally, a thorough understanding of the basic fundamentals of the disease characteristics is a must for public health officials and veterinary medical professionals.

Key etiologic and pathophysiologic points
All warm-blooded mammals are susceptible to infection with rabies, however susceptibility is not equal among all species. Different wildlife reservoirs maintain rabies in different regions of the United States: skunk, fox, coyote, raccoon, and bat.

Rabies should always be on a differential list for an animal presenting with neurological symptoms – until a definitive diagnosis is made. Universal precautions should be mandatory for all animals of unknown vaccine history, especially those with a known potential wildlife exposure. Rabies has no effective clinical treatment or cure to date. Response centers on prevention of the onset of clinical symptoms with aggressive prophylaxis in humans and exposed pets. Vaccination prior to exposure is more effective than prophylaxis post-exposure.

Once symptoms appear, rabies is nearly 100% fatal. Exposure to potentially rabid wildlife does not guarantee illness if prophylaxis is up-to-date and post-exposure boosters are administered.

Summary
1. Rabies is still a threat to human health and to animals in the United States.
2. There is still no effective treatment or cure for rabies following the onset of clinical disease.
3. Routine communication between veterinarians and public health officials is necessary for appropriate rabies prevention.
4. Veterinarians need to recognize and communicate honestly about the risk of rabies to staff and clients.
5. Veterinarians should be vigilant in their own protection and follow-up with recommended titers and boosters.

References/Suggested reading
If You Cage Creatures Enough… How to Handle Escapes

Jenifer Chatfield, DVM, DACZM
Dade City, FL

The law of averages would dictate – if one puts something in a cage long enough, it will eventually get out. Escapes range from simple – a small bird flies out the door as the keeper enters – to complex – 3 adult orangutans have used a broken tree limb following a windstorm to bridge the moat in their exhibit and all three are out on zoo grounds with an elementary school class also on grounds. No matter how complex, the approach to response is fundamentally the same – the animal got out of its cage/enclosure, so we will put it back.

Escapes are much more likely to overwhelm zoo resources subsequent to a natural disaster. During an escape response, it is important to first determine how many animals are escaped, what species, and their relationship to each other (is it a family group, rival males, different species, prey/predator species, etc.) Then, the veterinarian(s) responding should work with the husbandry staff to establish an incident command structure and a capture plan.

The capture and restraint of zoo animals is one of the most critical skills necessary during a disaster response. Whether the animal has escaped or needs to be restrained for a clinical evaluation following a disease outbreak response protocol, the selected method of restraint is crucial to a positive outcome. When restraining any animal, the first priority must be to avoid human injury. Human injury could include bites and scratches or zoonotic disease transmission, thus, appropriate personal protective equipment (PPE) should be worn at all times when restraining animals. The second priority is to avoid animal injury, including disease transmission from the handler to the animal. Appropriate use of PPE should prevent this as well. Typically, if the animal is considered “Code Red” or extremely dangerous, a shoot team as well as a capture team will be deployed. The shoot team will be armed with live ammunition weapons to dispatch the animal if human injury is imminent. The capture team will employ chemical or physical methods to recapture/restrain the animal. Examples of typical “Code Red” animals are tigers, elephants, rhinos, bears, great apes, etc. Chemical methods of restraint for recapture are typically administered via remote delivery system. When possible and prudent, physical methods of capture and restraint are preferred to chemical methods due to the inherent risk of anesthesia to the animal.

The most common remote delivery option for chemical restraint is a self-injecting dart shot from either a blow-pipe, pistol, or rifle. Many variables are important in order to dart animals effectively and consistently. Wind, humidity, distance, species targeted, dart weight, etc., all play an important role and can make or break a shot. Consistent and frequent practice are necessary to become proficient at darting multiple species at varying distances. However, some basic principles will serve the practitioner well when faced with the situation under less than ideal circumstances.

Human safety is of primary consideration – if the dart misses the targeted animal, is there a danger of hitting a human? Animal safety is considered – ideally the dart should enter a large muscle mass such as the upper arm, thigh or rump. When possible, dart only isolated animals rather than one of many in a group. All responding team members should remember that the anesthetic process is the same and induction will generally take 5-10 minutes and in some cases up to 20 minutes. The dart is not a bullet; The drug takes time to be absorbed and to have an effect. Educate team members so that the animal is not rushed prematurely as people can be injured. It is important to have team members who are more experienced with darting animals perform this procedure. Bear in mind, the darted animal may attempt to flee the area after darting, or it may try to retaliate towards the shooter or any other visible team member. Additionally, the animal may run towards water. Drowning can be a real threat. It is best to try to steer/herd the animal away from bodies of water during induction. If the animal is in a tree, try to safely break any fall. Overall, darting can be difficult, but is a useful procedure when animals need to be captured from open areas.

Physical restraint is a good choice for immobilizing animals during a escape because immobilizing drugs may be in short supply and anesthetic risk is increased when an animal is stressed (such as following escape). Hand-grabbing, or hand catching, animals carries great risk for human injury and animal injury and so should only be executed by experienced personnel. However, it can allow for the best opportunity for a veterinary exam as the animal is not anesthetized and the veterinarian can lay hands directly on the animal. It is best to hand grab animals only in enclosed areas. A squeeze chute is utilized for hoofstock and can be used for primates. It also allows for direct contact during examination. A net can be used to catch small mammals and birds and to restrain them for an exam. All physical restraint methods present a risk for the animal and the person and so should only be executed by experienced personnel when appropriate. Each of these methods is best employed when an animal is already otherwise contained, either in a room, building, paddock, pen, etc. If the animal is free-roaming, then chemical restraint utilizing a remote delivery system is likely the best option for capture and restraint.

Many zoos maintain venomous (or hot) and non-venomous reptiles. Some facilities also store anti-venom on hand. If you are not sure if a reptile is “hot” or not, it is best to assume that it is and handle it as such. The buddy system is a must when handling reptiles. Never attempt to capture or handle a reptile alone. If you get bit, your buddy can ID the reptile to medical staff, but your buddy can also make sure that the reptile remains caged after the bite or is killed. Capturing a loose snake does present a risk, so a decision must be made whether to recapture loose snakes or simply kill on sight. During an escape, it is good to remember that reptiles are cold-
blooded. So, they will be attracted to warm areas during cold times and vice versa. Additionally, temperature control is very important to maintaining reptile health post-capture.

Many zoos hold non-human primates of varying species. Personnel safety and safety of any general public should be a primary consideration – which is why initial removal of public from any area for capture procedure is paramount! Appropriate personal protective equipment (PPE) should be worn by all personnel when handling primates.

**Recommend further study and reference**

Kreger TJ and Armento JM. *Handbook of Wildlife Chemical Immobilization.*
Leptospirosis is one of the most exciting re-emerging pathogens in the USA. In fact, in the last 10 years, research indicates that the old dogma regarding risk of leptospirosis infection has been reversed. For example, recent investigations indicate that a risk factor for infection is a dog living in an urban area, rather than a rural dwelling dog as was historically thought to be at bigger risk. Additionally, small breed dogs have been identified as being at greater risk as well. Because of the zoonotic risk of leptospirosis, and the possibility of dogs becoming carriers, effective client communication and timely definitive diagnostics are important for regular practitioners to embrace.

Diagnostic options for leptospirosis include culture and antibody titer. However, as the initial presenting symptoms of leptospirosis are those of a simple urinary tract infection and most first-line empirical antibiotics are effective, most clinicians do not perform diagnostics. The advent of benchtop testing in recent months has made the option of definitive diagnosis more reasonable in regular practice.

Summary
1. Leptospirosis is a real infectious disease issue facing companion animals
2. Leptospirosis vaccination should be performed based on a risk assessment for each animal
3. Presenting symptoms of leptospirosis could mimic a simple urinary tract infection
4. Effective communication with veterinary practice staff and clients regarding zoonotic disease is important.

References/Suggested reading
Tuberculosis in Captive Wildlife

Jenifer Chatfield, DVM, DACZM
Dade City, FL

Tuberculosis is an emerging concern among captive and free-ranging wildlife. The disease can have significant financial implications and poses a problem for regulatory officials for control. Compounding the issue is the fact that several species of wildlife can act as maintenance hosts for Mycobacterium bovis. Many captive species are also impacted by Mycobacterium tuberculosis which poses a more significant zoonotic threat.

Clinical diagnostics
More than 10 different diagnostic procedures exist to identify Mycobacterium infection in captive wildlife. This is because none of them is good. Most are indirect, measuring the body’s response to the presence of the pathogen rather than directly identifying it. Of course, these sorts of indirect identification procedures also deliver a “positive” result when the animal has merely been exposed to the pathogen in the absence of infection. Certainly, the absence of a reasonable, reliable diagnostic procedure for a regulatory disease is problematic to say the least.

Typically, because of the regulatory nature of Mycobacterium, depopulation is promoted by officials as the response to a positive test result. In higher profile species, such as great apes and elephants, where depopulation for regulatory reasons is not as politically palatable for the public, multimodal antibiotic therapy can be attempted. Typically cost is a prohibitive component, however, and animal movement is severely restricted following resolution.

The prognosis for captive wildlife infected with Mycobacterium sp. is complex. Naturally, regulatory-driven depopulation has a poor prognosis. Therapy, if attempted, is typically long-term and complicated, carrying movement restrictions for the life-time of the animal. Additionally, therapy is not successful in 100% of cases due to antimicrobial resistance, animal compliance, cost of therapy, risk of exposure to caretakers, etc.

Summary
1. Mycobacterium sp. infect may different commonly held captive wildlife species.
2. There is no good ante mortem test for tuberculosis in any species.
3. Antimicrobial therapy for tuberculosis infection in captive wildlife is complicated, at best.
4. Practitioners should have a good understanding of the complications associated with administering tuberculosis diagnostics, regardless of the result.

References/Suggested reading
3. USDA - Bovine Tuberculosis. Found at: https://www.aphis.usda.gov/wps/portal/aphis/ourfocus/animalhealth/sa_animal_disease_information/sa_cattle_health/sa_tuberculosis/ct_bovine_tuberculosis_disease_information?utm/p/a0/04_Sj9CPykssy0xPLMnMz0vMAFgjzOK0_D2MD0MjDz0gy1DDe9wxc8LXz0Mj09TPQLsh0VZdilHg1/. Accessed 11/5/15.
Conservation efforts target increasing overall population numbers in general, but is this always appropriate? Conversely, are concerns about limited space a legitimate reason to stifle reproduction among exotic animals? What role does veterinary medicine and chemical or surgical sterilization play? Veterinarians have an ethical obligation to provide options to animal owners. Captive breeding programs have long-served as a safe repository for species whose native habitat is under threat from anthropological events, such as war or famine, to stochastic naturally occurring events such as a cyclone or a tsunami. The captive populations not only serve as a potential source of animals for reintroduction once the threats in the native habitat have been mitigated, but these captive animals also serve as a genetic bank of sorts to preserve genetic diversity among a species. Opponents of captive breeding programs are concerned that support of captive populations detracts from in situ conservation efforts such as habitat preservation. However, 21st century conservation efforts require that an integrated approach be embraced to include both in situ efforts and a robust captive breeding program. Genetic isolation (inbreeding) can produce specimens that are not of great fitness and can, over time, decrease the overall long-term viability of a population or species. Successful captive breeding programs require free movement of animals between collections to avoid the phenomenon of genetic isolation. Successful breeding of captive endangered species should also provide for appropriate levels of care for specimens which may include expansion of possible pool of locations for collections.

References
Influenza transmission is through aerosolized droplets and other respiratory secretions. Influenza survives in the environment, even under some less favorable conditions. Animals and humans are able to shed influenza prior to the onset of clinical symptoms for up to 72 hours, making biosecurity difficult to perform effectively.

**Diagnostic and therapeutic points**

Diagnosis can be based on clinical presentation or in conjunction with a variety of diagnostic tests. Supportive care shouldn’t be overlooked in non-food animals. Early supportive care and isolation from cohorts should be definitive. Additionally, personnel should be careful and maintain good hygiene practices to prevent cross-species transmission. Antivirals are sometimes effective in animals, but must be given very early in infection to be effective. Some resistant influenzas do exist. Vaccinate!

Early response to supportive care with less severe fever, decreased respiratory secretions, etc., are generally good prognostic indicators. Treatment in commercial production operations is not possible and all influenza infections in animals should be treated as significant.

Given the likelihood that the next influenza pandemic will be the result of a recently adapted animal strain infecting humans, veterinary medical professionals should be vigilant in their own disease prevention and get routine annual influenza immunizations.

**“Take home” points**

1. Influenza is unpredictable and cross-species transmission does occur.
2. Virus shedding is possible for 48-72 hours prior to the onset of clinical symptoms, so good biosecurity should be practiced when bringing new animals into an existing group.
3. Influenza is not new – it has just evolved.
4. Antivirals are most effective when initiated early in disease onset.
5. Veterinarians and their staff should be vaccinated annually to prevent influenza transmission.

**References/Suggested reading**

What is alternative? According to Webster’s Dictionary it refers to something that is “different from the usual or conventional”. Depending on where you went to school, some of these techniques may not be “alternative” to you as they have (slowly) become more main stream. Many of these techniques are now included in some veterinary schools’ curricula.

So why worry about this? Spay/neuter surgeries have evolved considerably over the last 10-15 years. High volume spay/neuters in shelters and humane societies, low cost spay/neuter clinics, and mobile spay/neuter clinics have contributed to an evolution in the way that these surgeries are performed. It is important to keep up with the way that the profession is changing and realize that a different approach is not always wrong: And after all, you may be seeing some of these patients for follow up care and may even deal with (rare) complications post-surgery.

Canine neuter

- Pre-scrotal approach – is the traditional method taught in most veterinary schools. In this procedure the testicles are individually advanced cranially to the scrotum, the skin and facial layers are incised over the testicle, and the testicles are individually removed and double ligated. It is important to incise directly over the testicles to prevent inadvertently damaging the urethra. There are different methods for closure; in our practice we close in two layers – the deep facial layer and the skin. To the author’s knowledge, canines are the only species where this approach is utilized.

- Scrotal approach – this technique has been used in high volume spay/neuter surgery for a number of years and is now taught at several veterinary schools. In this procedure an individual testicle is stabilized and the incision is made directly over the scrotum. The testicles are then individually removed and double ligated or ligated with a single Miller’s knot. The facial/subcutaneous layer is then closed. The incision may be left “open” to allow drainage and healing by second intention or the incision may be closed with cyanoacrylate tissue adhesive. Proponents of the use of tissue adhesive argue that the use of this substance will provide an immediate barrier to surgical wound contamination and has hemostatic and bacteriostatic properties. A study published last year in Veterinary Medicine shows that the scrotal approach for canine neuters is faster than the traditional method and actually had a lower complication rate than the pre-scrotal approach.

- Suture-less scrotal castration – is used in pediatric and juvenile canines in numerous high quality – high volume spay/neuter facilities. For this procedure a single scrotal incision is made on ventral midline and both testicles are accessed through this incision. Opening of the vaginal tunic versus performing a closed castration is left to the discretion of the surgeon. The testicles are individually exteriorized and a hemostat is then directed parallel to the spermatic cord, pointed in a proximal direction. The hemostat is twisted to form a simple overhand knot in the spermatic cord; the cord is cut distal to the hemostat and the cut end slipped over the end of the hemostat to complete the knot. The scrotal skin incision is closed using cyanoacrylate surgical skin adhesive although some surgeons may elect to leave the incision open to allow drainage and healing by second intention. A recently completed, not yet published, study with Oregon State University’s College of Veterinary Medicine and the Oregon Humane Society demonstrated that this technique is a safe and efficient method for pediatric and juvenile canine castration. This study will be discussed along with preliminary results.

Feline spay

- Double ligating the ovarian pedicle – is the traditional method taught in most veterinary schools. There is absolutely more than one way to spay a cat (which is the point of this portion of the lecture) but this is the method that we teach fourth year Veterinary students at Oregon State University: A ventral midline celiotomy is performed and the uterus is located using a spay (or Snook) hook. After a hemostat is placed on the proper ligament, the suspensory ligament is torn using a hemostat or cut with a scalpel blade to allow the ovary to be well exteriorized. A window is then created within the broad ligament to facilitate isolation of the ovarian pedicle. A hemostat is placed across the vascular pedicle just proximal to the ovary. Two separate absorbable suture ligatures are then placed around the ovarian vascular pedicle prior to transection. After this procedure is performed on the other side, the uterine body is then exteriorized and double ligated; the use of a transfixing suture should be considered at the discretion of the surgeon. After the removal of the uterus and both ovaries the incision is closed. There is more than one way to close these incisions: At this time we are teaching our 4th year students to close the body wall in a simple continuous pattern and the close the skin using an intradermal suture pattern. We also use cyanoacrylate surgical skin adhesive to aid in surgical would closure and to provide an immediate barrier to contamination of the surgical site.
Pedicle tie – is a procedure where the ovarian pedicle is “auto-ligated” in the same way that the spermatic cord is tied on itself in feline castrations. A ventral midline celiotomy is performed and the uterus is located using a spay (or Snook) hook. After a hemostat is placed on the proper ligament, the suspensory ligament is torn using a hemostat or cut with a scalpel blade to allow the ovary to be well exteriorized. A window is then created within the broad ligament to facilitate isolation of the ovarian pedicle. For the pedicle tie procedure a hemostat is then directed parallel to the ovarian vascular pedicle, pointed in a proximal direction. The hemostat is then twisted to form a simple overhand knot in the vascular pedicle; the pedicle is cut distal to the hemostat and the cut end slipped over the end of the hemostat to complete the knot. After this procedure is repeated on the opposite side, the uterine body is then exteriorized and ligated. Upon removal of the uterus and both ovaries the incision is closed. Again, there is more than one way to close these incisions: At this time we are closing the body wall in a simple continuous pattern and the closing the skin using an intradermal suture pattern. We also use cyanoacrylate surgical skin adhesive to aid in surgical closure and to provide an immediate barrier to contamination of the surgical site. A study with Oregon State University’s College of Veterinary Medicine and the Oregon Humane Society evaluating this technique was published in 2016 in the Journal of Feline Medicine and Surgery. In this paper, after evaluating more than 2,000 surgeries, the authors concluded that this method is safe and almost 30% faster than double ligating the ovarian pedicle.

Ovariectomy
Most veterinary schools in the U.S. and Canada still teach ovariohysterectomy (OHE) as the preferred procedure for sterilizing female dogs and cats. In many countries in Europe the ovariectomy (OVE) is the preferred technique. According to several published papers the short term and long term complications of ovarioectomies and ovariohysterectomies are similar.

In one paper published in Veterinary Surgery in 2006 the authors evaluated the literature and found that there was no significant difference in long-term urogenital problems, including endometritis/pyometra, urinary incontinence, or neoplasia in dogs that underwent ovariectomy vs. ovariohysterectomy. The authors concluded that OHE is technically more complicated, time consuming, and is probably associated with greater morbidity vs. OVE. In this paper the authors also concluded that OHE is likely associated with a larger incision, more intraoperative trauma and would result in increased discomfort for the patient. Finally, they pointed out that uterine tumor formation is relatively rare (with 0.003% risk of malignancy) versus OHE specific complications (e.g. distal ureteral ligation). They concluded that there is no scientific reason that OHE should be preferred over OVE for castrating female dogs.

In a prospective clinical trial published in the Journal of the American Veterinary Medical Association in 2011 researchers evaluated 20 dogs that underwent OHE and 20 dogs that underwent OVE. They evaluated for blood loss, erythema, swelling, discharge, dehiscence, and postoperative pain. This paper reported no statistically significant difference in complication rates between dogs in the OHE group and dogs in the OVE group. Additionally, there was no significant difference in surgical time and no difference in pain scores between the two groups.

Conclusion
Spay/neuter surgeries have and will continue to evolve. It is, therefore, important that we conduct research to evaluate these new techniques to determine their safety and relevance. Also, newer graduates may be coming into practice with an enthusiasm for trying some of these newer techniques and may have been taught some of these methods in veterinary school. So again, it is important to keep up with the way that these procedures are changing and recognize that different is not always bad. In fact the research presented here demonstrates that different can be safe and efficient.

References and suggested reading


Animal Hoarding:
An Overview
Kirk Miller, DVM, DABVP
Oregon State University
Portland, OR

An animal hoarder is defined as someone who has accumulated a large number of animals that overwhelm his or her ability to provide a minimum of care, including adequate nutrition, sanitary conditions, and veterinary care.

The key point here is that it is not about the number of animals, it is about what happens to the animals. In fact, that is one of the challenges of trying to deal with animal hoarding through the legal system and/or municipal codes. The number of animals is not the problem; the problem is that it has overwhelmed the person's ability to provide a minimum of care.

There are several key characteristics that have been described which can be helpful in identifying an animal hoarder:

- Obsessive attempts to accumulate or maintain a collection of animals in the face of progressively deteriorating conditions
- Failure to provide minimal standards of sanitation, space, nutrition, and veterinary care for the animals
- Inability to recognize the effects of this failure on the welfare of the animals, human members of the household, and the environment
- Denial or minimization of problems and living conditions for people and animals

Types of hoarders
There are several distinct types of hoarders that have been described by Patronek and others. There is often some overlap and a given hoarding situation may not fit entirely into one category. Rather, these are guidelines that are useful in how to approach a hoarder and whether or not this person is likely to be amenable to receiving help.

- The Overwhelmed Caregiver
  - The overwhelmed caregiver is someone who initially provided adequate care but has found that conditions have deteriorated over time. This person may understand that there is a problem (may try to minimize it) and will tend to have fewer issues dealing with authority and accepting intervention.

- The Rescuer Hoarder
  - The rescue hoarder is described as a person with a compulsion that is based on a strong need to rescue animals from possible death or euthanasia. They will actively acquire animals (in the face of deteriorating conditions) and believe that they are the only ones who can care for them. They will tend to avoid authority but may work within a network of animal “welfare” people.

- The Exploiter Hoarder
  - The exploiter hoarder is indifferent to the harm that their actions have caused animals in their care and acquire animals to satisfy their own needs. They will deny that there is a problem and reject authority figures or outside help. Skilled at presenting excuses and explanations they may come across as charming and articulate. They will lie, cheat and steal without remorse to achieve their goals. Puppy mills and people illegally selling animals for research sometimes fall into this category.

Interventions based on typology
It should be emphasized that the types of hoarders described above are theoretical, working definitions. However, these descriptions may give some insight into which type of intervention will work best in a given situation.

- The Overwhelmed Caregiver
  - Since this type of person likely understands that there is a problem there is a higher likelihood that she/he will accept intervention and assistance. They are generally more likely to surrender the animals, accept spay/neuter assistance, and respond to other offers of help. Counseling and other therapeutic interventions may be of assistance.

- The Rescuer Hoarder
  - The rescue hoarder is distrustful of authorities and therefore less likely to accept intervention and assistance. “Mission” driven, this type of person will avoid authorities and believes that she/he is the only one that can care for the animals. Counseling and other therapeutic interventions may be of assistance but prosecution and monitoring would likely be appropriate.

- The Exploiter Hoarder
At the other end of the spectrum the exploiter hoarder may have genuine sociopathic tendencies and is unlikely to accept intervention and/or assistance. Aggressive prosecution and monitoring may be the only way to inhibit this behavior.

Dangers of hoarding
Animal hoarding will adversely affect the animal victims as well as any people in the environment. The animals often suffer from malnourishment, overcrowding, and general neglect. Stress, starvation, and infectious diseases are commonly encountered and after rescue the animals may be beyond help due to medical and/or behavior problems. Health effects for people in the environment include poor sanitation, zoonotic diseases, self-neglect and child/elder neglect. The environment is often severely contaminated with urine and feces to the point of causing structural damage.

Prevalence
- Reports
  - There may be up to 7,000 reports each year in the U.S. Multiple reports on same person can occur which can make it difficult to know the true number of reports each year.
- Cases
  - Approximately 1500 new cases of animal hoarding are opened each year in the U.S. This translates to approximately 250,000 animal victims of hoarding each year.
- Trends
  - According to the Pet Abuse Database from 2000 to 2006 reporting increased 5 fold. The Animal Legal Defense fund reports that the number of cases doubled in the last few years. Certainly there is greater public awareness of hoarding, both object hoarding and animal hoarding, due to the media attention that this condition has generated. That may be more of a factor in the increase in reports and cases than an actual increase in the number of people engaging in this behavior.

Demographics
Although anyone can become involved in this the perpetrators of animal hoarding are often middle-aged to older females.

Causes/Etiology
At this time we do not know what causes someone to become an animal hoarder. There are numerous theories but no definitive answer. Some have speculated that childhood trauma and/or attachment disorder can result in an unhealthy devotion to animals. Some have proposed an addictions model and in some cases this theory will fit including a preoccupation with animals, denial of problem/excuses, and self-neglect. Other possible causes include Delusional Disorder, Dementia (documented in some cases), Borderline Personality Disorder, Obsessive-Compulsive Disorder, and Insistent Caregiving. While there is anecdotal evidence to support each of these models, more study is needed.

Legal aspects
For prosecuting these cases State animal cruelty laws are often utilized which center around the failure to provide proper care. This is a crime of omission or neglect and considered a misdemeanor in most states. Those that are found guilty may face fines, animal forfeiture, (rare) jail time, and bans on pet ownership. One of the biggest problems in dealing with animal hoarding in the long term is that psychological diagnosis, treatment, and counseling are often lacking. Perhaps even more importantly, monitored probation is often lacking. Thus, offenders are usually free to re-engage in this behavior. Some states, Illinois and Hawaii in particular, have passed specific laws against animal hoarding.

Related laws
- Forfeiture and Bond Laws
  - Forfeiture – animals are considered evidence and cannot be adopted until prosecution is complete.
  - This is very expensive. It also means that these animals will occupy valuable shelter space while the case is working through the legal system.
  - Bond law = legislation requiring animal owners to post a security or bond for the care of the seized animals. This is helpful not only for defraying the costs associated with providing care for the animals but can be used as a negotiation tool as well.
- Community Ordinances
  - May be useful in states that don’t have animal hoarding specific laws. These are community ordinances that outlaw animal hoarding and it should be noted that they are different than pet limitation ordinances.
Prosecution

- Phases of the case
  - Complaint
  - Investigation
  - Triage
  - Animal Removal and Transport
  - Custody/Temporary Sheltering
  - Placement/Adoption
  - Expert Witness/Trial

Treatment/Prevention

Unfortunately, the lack of a known cause or etiology makes treatment of animal hoarding difficult, if not impossible. At this time the recidivism rate is thought to be around one hundred percent. The legal system needs to make this a priority and allocate resources – in particular mandated treatment for animal hoarders and monitored probation. The old adage that an animal hoarder will likely pick up another animal on the way out of the courtroom appears to be accurate.

References


Anorexia in Cats
Kirk Miller, DVM, DABVP
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Anorexia is defined as the lack of or the loss of the appetite for food. The importance of anorexia, especially in felines, really cannot be overstated. It is often one of the first things that an owner will notice when a pet is ill and is a frequent reason for veterinary visits. It is also a major problem in cats in animal shelters.

Mechanisms
There are numerous reasons that a cat will stop eating. Thinking of the possible mechanisms can inform the clinicians’ diagnostic and therapeutic plans.

- Psychological – psychological reasons for anorexia include stress, fear, and pain. A recent study published in JAVMA demonstrated a strong relationship between stress, weight loss, and upper respiratory infections.
- Pathophysiologic
  - Degenerative – conditions that result in the deterioration of cells and/or organs over time. These diseases may be congenital or acquired.
  - Anatomic – can include oropharyngeal diseases and foreign bodies.
  - Metabolic – encompasses a large array of disorders. These generally result from major organ dysfunction. Clinically, these are typically acquired disorders.
  - Neurologic – any condition that alters mentation may contribute to anorexia. It may be difficult to discern neurologic causes of altered mentation verses other causes of altered mentation (ie metabolic dz.), all of which can lead to anorexia.
  - Neoplastic – cancer cachexia, sterile inflammation, or space occupying mass are just a few of the ways that neoplasia can contribute to anorexia.
  - Infectious, Inflammatory – often considered together, any condition that can lead to fever will often lead to anorexia.
- Drugs – can lead to anorexia in numerous ways. Some drugs will adversely affect the stomach leading to nausea and/or delayed gastric emptying. Others can alter the smell or tasted of food leading to anorexia.
- Influence of neurotransmitters
  - Serotonin – decreases appetite. Cyproheptadine is a medication that acts as a serotonin antagonist.
  - Gamma-aminobutyric acid (GABA) – stimulates the hunger center or inhibits the satiety center. Valium will lead to increased GABA activity and inhibit serotonin release.
- Influence of hormones
  - Insulin – acts to decrease appetite, which explains why diabetics will often have an increased appetite.
  - Cortisol – acts to increase appetite. Think of the patient with Cushing’s disease versus the patient with Addison’s disease and their response to food.
- Gastrointestinal factors
- Environmental and sensory factors – the texture, shape and smell of food can have a profound impact on appetite. Likewise, patients may exhibit preferences for foods with different flavors. Acquired behaviors, ie eating people food, can lead to anorexia when the preferred type of food is no longer offered or available.

Causes
This is another way of thinking about (roughly) the same thing. I find this schema to be more clinically friendly and relevant.

- Primary Anorexia – think of this as problems that involve the head.
  - Neurological dysfunction
  - Psychological disorders
  - Loss of smell
- Secondary Anorexia – think of this as problems that involve the body. This is a major cause of anorexia and these conditions may also be associated with nausea and vomiting.
  - Pain – inhibits the appetite center.
  - Abdominal organ disorders
  - Toxic agents – can act centrally or through organ destruction.
  - Endocrine – ie Addison’s disease.
  - Neoplasia
Infectious disease
- Miscellaneous – cardiac failure, ketosis, motion sickness, etc.
- Pseudoanorexia – these are problems with the mouth (sort of). Basically, anything that inhibits the ability to pick up, chew, or swallow food.
  - Disorders of the oral cavity
  - Hypoglossal paralysis
  - Mandibular paralysis
  - Maxillary or mandibular fractures or dislocations
  - Retrobulbar disease
  - Other

Effects
Prolonged and/or severe anorexia can have deleterious effects on almost all body systems. Protein-energy malnutrition, increased metabolic rate and protein catabolism, and alterations in fat metabolism are just a few of the changes that can take place. Additional problems include the potential for immune system compromise, hepatic dysfunction, and intestinal alterations. In felines, the consequences of prolonged anorexia, hepatic lipidosis in particular, can be worse than the inciting disease or event.

Clinical approach
The clinical approach to the anorexic patient begins, of course, with a detailed patient history. Duration of clinical signs and the presence or absence of other clinical signs can give important clues to the underlying etiology. Special attention should be paid to any changes in the environment – new diet, change in feeding location/routine, new people or pets in the house – as many of our feline patients are particularly sensitive to stress.

Likewise, the importance of a good physical exam cannot be overemphasized when dealing with an anorexic patient. Disorders of the mouth (pseudoanorexia) can often be ruled in or out on the basis of a physical exam. Some of the secondary causes of anorexia, ie abdominal masses, may be identified on physical exam. At the very least, the physical exam can give clues as to the most appropriate course in pursuing a diagnosis.

Primary anorexia is usually a diagnosis of exclusion: Pseudoanorexia is often diagnosed on physical exam. Diagnostic testing is therefore most useful for ruling in or out the secondary causes of anorexia. A complete blood count, chemistry panel, and urinalysis will allow the clinician to evaluate many organ systems. Liver disease, renal failure, and diabetes are just a few of the causes of anorexia that can be evaluated with bloodwork. Radiographs, ultrasonography, and even exploratory surgery are other diagnostic modalities that should be considered when appropriate.

Treatment
Treatment of anorexia should center around treating the cause, if it is known. Primary anorexia, particularly stress, may be solved utilizing some of the following treatments; these may also prove useful in patient support during the initial workup.

- Encourage eating – warming the food, small meals, treats, and baby food are often used to tempt cats to eat.
- Appetite stimulants
  - Cyproheptadine – is an antihistamine that has been shown to stimulate appetite in cats. Although it is metabolized by the liver and excreted by the kidneys it appears to be (clinically) fairly safe.
  - Mirtazapine – is an antidepressant in people that appears to be effective as an appetite stimulant in cats. It is also useful as an anti-nausea drug and an antiemetic. Impaired excretion in CRF cats.
  - Diazepam – a classic! Use with caution in patients with hepatic or renal disease. Typically administered IV for appetite stimulation – oral administration associated with rare cases of liver failure.
  - Midazolam HCL (Versed) – another benzodiazepine – can be given IM.
  - Propofol – yes, the anesthetic. Published report of efficacy as an appetite stimulant in dogs. Much anecdotal evidence of its’ usefulness as an appetite stimulant in cats.
  - Megestrol Acetate?, Steroids?
- Alternative therapies
  - Pheromones
  - Acupuncture
- Feeding
  - Enteral
    - Nasoesophageal or nasogastric tube
    - Pharyngostomy or esophagostomy tube
    - Gastrostomy tube
Jejunostomy tube
  o Parenteral
    ▪ Total Parenteral Nutrition (TPN)
    ▪ Partial Parenteral Nutrition (PPN)

In our shelter, this would be a typical treatment progression that an average case may follow. It is important to note that this may change depending on the condition of the patient at presentation and/or the response to treatment. Dehydration will contribute to anorexia and, if detected, should be treated early and aggressively.

**Typical treatment progression**
- Treat URI and/or stress
- Encourage eating
- Appetite stimulants
  - Mirtazapine (or Cyproheptadine)
  - Diazepam or Propofol
- Monitor Weight
- Monitor Liver Enzymes and T. Bili.
- Feeding Tube

**Conclusion**
Anorexia has the potential to cause significant morbidity and mortality in felines, especially in shelters where time and financial resources may be in short supply. Understanding the potential reasons that cats may stop eating will inform the clinicians’ diagnostic and therapeutic plans and allow these patients to be treated in the most efficient manner possible.

**References and suggested reading**
Respiratory Infections in Dogs and Cats
Kirk Miller, DVM, DABVP
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Canine infectious tracheobronchitis
- Acute, highly contagious respiratory infection
- Paroxysmal cough, variable expectoration, +/- nasal or ocular discharge

Why is this condition important?
Canine Infectious Respiratory Disease Complex (CIRDC) is considered among the most prevalent infectious respiratory diseases in dogs. Outbreaks of this disease are relatively common, especially in high density environments like animal shelters, pet shops, and boarding kennels. Most of the pathogens that we will discuss are spread via dog to dog contact or airborne secretions.

Pathogens
- Viruses
  - Canine Parainfluenza Virus (CPIV) – In these cases infections are typically restricted to the upper respiratory tract. Edema in the vocal folds will cause the “honking” cough commonly associated with CIRDC. Transmission occurs via aerosolized droplets; incubation typically lasts about 3-10 days and viral shedding can go for about 7 days post infection.
  - Canine Adenovirus 2 (CAV-2) – In these cases the virus replicates in nasal mucosa, pharynx, tonsillar crypts, trachea, and bronchi so it tends to be a disease of the lower airways. Peak infection will occur 3-6 days post exposure which often occurs via oronasal transmission. This agent is capable of producing a single-agent viral pneumonia.
  - Others:
    - Canine Respiratory Coronavirus
    - Canine Herpesvirus
    - Pneumovirus
    - Canine Distemper
    - Influenza A: H3N8
    - Influenza A: H3N2
- Bacteria
  - Bordetella bronchiseptica – Is a principal agent involved in CIRDC. It tends to be involved in more significant clinical disease and is a major player in multi-agent infections. Transmission is via direct contact, aerosolization of microparticles, and fomites (dishes, hands, etc.). Incubation for about one week is typical although the pathogen may not be cleared for up to 3 months.
  - Mycoplasma – Remember that Mycoplasma is group of tiny simple intracellular bacteria without cell walls. This will impact the types of antibiotics that will be effective against these organisms (no Beta-lactams!). They are considered to be a primary cause of secondary pneumonia in Canine Infectious Respiratory Disease.
  - Streptococcus equi var zooepidemicus – A nasty and, thankfully, rare bug that has been involved in only 3 confirmed outbreaks. Susceptible to numerous antibiotics.

Diagnosis
The diagnosis of Canine Infectious Respiratory Disease Complex is often based on a history of exposure combined with clinical signs. Bloodwork, in particular a CBC, may be useful: Chest radiographs are often helpful to confirm or rule out a secondary pneumonia although this modality is usually reserved for patients with more severe respiratory symptoms. In our shelter we will occasionally use bacterial cultures and/or viral PCR, especially in cases that have not responded to empirical treatment or have implications for our population as a whole.

Treatment
Our current protocol for treating kennel cough centers around the use of Doxycycline. The reason for this is two-fold: We use periodic cultures to monitor the types of pathogens that we encounter and find that we are often dealing with Mycoplasma – sensitive to Doxycycline. Also, we occasionally have pets that come back to the shelter after being treated by local practitioners for a pneumonia that is not resolving. Often these patients have been on Clavamox which will not be effective against Mycoplasma organisms. The majority of these patients have their condition resolve after Doxycycline is used.

Patients that are dehydrated or anorexic will often benefit from parenteral fluid therapy. This may involve subcutaneous fluids or IV fluids. Additionally, patients may benefit from the use of coupage and/or nebulization.
**Prevention**

Vaccination of all dogs upon arrival at the shelter is the mainstay of prevention. Despite this, some dogs will invariably become ill while in the shelter or soon after adoption. Isolation/segregation of sick dogs is ideal although space constraints may limit the ability of some shelters to achieve this goal. Stress reduction, avoiding overcrowding, and adherence to disinfection protocols will decrease the level of these diseases within the population. Finally, proper ventilation will help reduce transmission of airborne infectious organisms. Currently, a minimum of 10 – 12 air exchanges per hour is recommended for proper ventilation.

**Feline upper respiratory infections**

- AKA “URI”

**Why is this condition important?**

Like Canine Infectious Respiratory Disease Complex (CIRDC), Feline URI is considered among the most prevalent infectious respiratory diseases in cats. Outbreaks of this disease are also relatively common, especially in high density environments like animal shelters, pet shops, and boarding kennels. URI rates can reach 30-90% in shelters and it is a common reason for euthanasia in shelter cats. Fortunately, we can do a lot to prevent this disease if we understand more about how it is spread and how it can be prevented.

**Pathogens**

- **Viruses**
  - Feline Herpesvirus 1 – Herpesvirus is the most important pathogen in “background” URI in shelters. A large percentage of patients with herpes will already have it on arrival to the shelter and it is reactivated by stress. Therefore, in a shelter setting herpes control often centers around decreasing stress.
  - Feline Calicivirus – Calicivirus is highly variable and mutates frequently so controlling calicivirus, especially during an outbreak, can be challenging. Ulceration, especially of the tongue, mouth, and nose are the most common clinical signs. Occasionally cats with Calicivirus will present with a shifting leg lameness secondary to polyarthrits. This is usually a transient problem that responds well to NSAID’s.
  - Influenza – Various strains of influenza virus, including H3N2, can be of concern in animal shelters. Late 2016/early 2017 saw an outbreak of H7N2 (“bird flu”) in New York.
  - Systemic Virulent Calicivirus – Preceded by typical FCV signs these patients will go on to develop edema and the extremities with alopecia and ulceration. It has also been associated with hepatocellular necrosis and the mortality rate can reach 50%. Adult cats are severely affected and it may impact vaccinated and unvaccinated cats. It is important to note that Calicivirus is common and that Systemic Virulent Calicivirus is relatively rare.

- **Bacteria**
  - Secondary Invaders – Classically, secondary bacterial infections will cause a change in the character of nasal or ocular discharges. This will result in more mucopurulent material which, when present, indicates that antibiotics may be appropriate.
  - Chlamyphila – Will often present as a unilateral conjunctivitis with minimal respiratory signs. It should be noted that cats can be carriers of C.felis.
  - Mycoplasma
  - Bordetella

**URI prevention**

Perhaps one of the most important things we can do to help prevent URI is to help control stress. Providing places to hide, cage covers, decreasing noise (especially from barking dogs) and avoiding moving cats from cage to cage can all help to minimize stress. Other steps that we can take to help prevent URI include sanitation/handling of cats, segregation of ill vs. healthy cats, and vaccination of all cats on arrival to the shelter.

**URI diagnosis**

The diagnosis of Feline Upper Respiratory Infection is almost always based on a history of exposure combined with clinical signs. Clinical signs of URI in cats include sneezing, nasal discharge, and ulcers/sores on the nose, lips or tongue. Fever, lethargy, and loss of appetite may also be present but can be signs of other disease conditions as well. Gingivitis and stomatitis may be seen in cats with Calicivirus as well as a shifting leg lameness secondary to polyarthrits. As with kennel cough in dogs we will occasionally use bacterial cultures and/or viral PCR, especially in cases that have not responded to empirical treatment or that have implications for our population as a whole. Culture and PCR may also be useful in cases with legal implications (ie hoarding cases) for the purposes of documentation.

**URI treatment**

A large portion of URI cases will be due to viral disease: Thus, treatment will be centered around supportive care. Ensuring that the patient is eating and remaining hydrated is critical. Patients that develop mucopurulent nasal or ocular discharge likely have a secondary bacterial infection: Antibiotics will be appropriate in these cases. At our shelter we perform periodic cultures to see what
pathogens we are dealing with and what antibiotics will be most successful in treating these infections. Currently, we are using Doxycycline for treating these patients.

Patients with ulcerations of the tongue or mouth, secondary to calicivirus, are particularly vulnerable. Monitoring these patients carefully for evidence of pain and/or anorexia is important. Pain medications may be needed to keep these patients comfortable and eating. We have had good success with using transmucosal buprenorphine for this purpose.

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“Canine: Infectious Respiratory Disease Complex (a.k.a Kennel Cough)” Koret Shelter Medicine Information Sheets last edited July 2015.
The Spay/Neuter Debate:
Why, When, What If…
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Why talk about this
There has been a lot of research around the area of spay/neuter within the last 15-20 years and especially within the last 5 years. Some of this research has raised good points while other studies are fraught with potential bias and erroneous conclusions. Additionally, the lay press (and the internet!) will sometimes take parts of these studies and use them to further their own agendas. It is important that we, as Veterinarians, know what information is out there so that we can help our clients make sound decisions based on the best science available.

Why spay and neuter
Traditionally, spaying and neutering dogs and cats has been advocated for the greater benefit of society, specifically to help reduce pet overpopulation. Currently, it is estimated that about 10,000 animals, mostly dogs and cats, are euthanized in U.S. shelters every day; that’s about one animal every 11 seconds. Statistics on shelter populations can be difficult to track for a variety of reasons but it appears that great strides have been made within the last 40 years to reduce the number of animals euthanized in animal shelters. It has been estimated that in 1973 that about 13.5 million dogs and cats were euthanized in shelters (or about 20 percent of pets). By the early nineties that number had been reduced to 4-5 million dogs and cats euthanized in U.S. shelters (or about 5.3 percent of the pet population). It is likely that spaying and neutering has played a role in the reduction of shelter euthanasia. In fact, in some parts of the country animal shelters don’t have enough dogs available for adoption and are importing adoptable dogs from other areas.

“Early” spay/neuter or prepubertal gonadectomy has been around since the early 1990’s. The main reason to perform these procedures at a younger age is to allow humane organizations and animal shelters to adopt out animals after they have been spayed or neutered. Gonadectomized animals rarely reproduce: Incentives to encourage owners to have their pet spayed or neutered after adoption were marginally successful at best, allowing animals that were adopted while sexually intact to contribute to the pet overpopulation problem.

Potential benefits of spay/Neuter
Overall, spayed and neutered pets are likely to have a longer life-span. This is due in part to changes in behavior from reductions in sex hormones and related activity (roaming, fighting, etc.). Spayed and neutered animals will also have a reduced risk of certain cancers and complications related to the retention of sexual organs (ie pyometra). Many of the benefits can be summarized as follows:

- **Male Dogs**
  - Decreased BPH, prostatitis, prostatic cysts, Brucellosis, TVT Eliminates risk of testicular tumor
  - Decreased risk of perineal hernia, perianal adenoma, male-male aggression

- **Male Cats**
  - Decreased risk of roaming, fighting, urinating in house? Decreased urine odor

- **Female Dogs**
  - Reduced risk of Brucellosis and TVT: Eliminates risk of pyometra, ovarian neoplasia
  - Decreased risk of mammary neoplasia

- **Female Cats**
  - Eliminates risk of pyometra, ovarian neoplasia. Eliminates estrus behaviors
  - Decreased risk of mammary neoplasia (> 90 % are malignant!)

A recent study has led some to question the protective effects of spaying dogs with regard to mammary tumors. There are, however, some questionable aspects of the design of that particular study. Additionally, others have shown that the incidence of malignant mammary tumors is much higher in European countries (Italy, Denmark, etc.) where there are more sexually intact dogs. Until there is more convincing evidence to the contrary we should continue to have every confidence that spaying dogs will reduce the incidence of mammary cancer.

Potential risks of spay/Neuter
Recent studies have looked at the risk of spaying and neutering dogs, beyond the normal concerns about anesthesia and surgery. It is fairly well accepted that spaying and neutering will have an effect on metabolism resulting in a tendency for these animals to gain weight. Obesity is a very real concern that can predispose these patients to diabetes and various orthopedic issues: With good client education obesity is a preventable disease. Some studies have found a correlation between spay/neuter status and other diseases. As
mentioned earlier some of the evidence is better...some is less than convincing. In order to be as concise as (reasonably) possible I’ll divide these potential risks into one of three categories — neoplasia, orthopedic concerns, and other.

- **Neoplasia** — The vast majority, if not all, of the studies looking at a link between spay/neuter status and the development of cancer have been on purebred dogs. The fact that purebred dogs are predisposed to certain cancers is of no surprise to anyone in the veterinary field: Generalizing findings from studies in certain purebred dogs to all dogs is a fallacy. Additionally, finding a correlation between spay/neuter status and cancer does not prove a cause and effect relationship. One study looking at the long-term effects on spaying and neutering Golden Retrievers compared to Labrador Retrievers found little or no relationship between spay/neuter status and the increased incidence of cancer in the Labrador Retriever group - thus proving that the effects of confounding variables (like genetics) are more powerful than the effects of spay/neuter status in the development of cancer.

- **Orthopedic diseases** — A major concern is that the removal of the influence of gonadal hormones will result in the delayed closure of growth plates resulting in changes in the skeletal structure that can lead to certain conditions or diseases. In contrast to the idea that spay/neuter causes cancer this is a more biologically plausible theory. Predominately, studies are looking at hip dysplasia and cranial cruciate ligament rupture in dogs.
  - **Hip dysplasia** — As with neoplasia, the majority of studies looking at the impact of spaying and neutering dogs on the development of hip dysplasia involved pure bred dogs that were seen at referral institutions: So it is questionable that this information can be applied to all dogs. One very large study that looked at multiple breeds of dogs did find a correlation between spay/neuter status and the development of hip dysplasia. The authors noted that owners with spayed and neutered animals may be more likely to seek veterinary care for orthopedic injuries which could skew the data. Unfortunately, this study did not control for weight or body condition score which is likely more of a factor in the development of this condition. Certainly, more study is needed in this area.
  - **Cranial Cruciate Ligament rupture** — Several studies have looked at spay/neuter status as a risk factor for CCL rupture. The greater length of long bones and changes in the tibial plateau angle of spayed and neutered dogs, again, provides a biologically plausible theory for this risk. Larger dogs, older dogs, and obese dogs are at greater risk for this condition and studies that do not control for body condition score should be given less than full consideration. One study looking at the medical records from a first-opinion veterinary practice found that neutering (and spaying) was not associated with an increased risk of cranial cruciate ligament rupture. Again, more study is needed in this area.

- **Other**
  - **Urinary incontinence** — Acquired urinary incontinence post spay has been well documented although the reports show a prevalence that ranges from 5% to 20% of spayed female dogs. A recent report found that the prevalence was at the low end of this range. The majority of these patients will respond to medical management.

**What’s the bottom line?**

- **Male Cats** — this is a classic “no brainer”. The pros far outweigh the cons and we should neuter every male cat as soon as possible. There is no known medical reason not to neuter cats and to neuter them early.
- **Female Cats** — still, “just do it”! Again, the pros far outweigh the cons. And a recent study shows that spaying before the cats’ first heat (so do it early) will greatly reduce the risk of malignant mammary neoplasia.
- **Male Dogs** — for most small and medium sized dogs the pros of neutering far outweigh the cons. For responsible owners of large and giant breed dogs or breeds that are predisposed to orthopedic problems discussing the option of waiting until the dog is over 1 year of age may be prudent, if not overly cautious.
- **Female Dogs** — until there is further evidence to the contrary, the concern about mammary tumors is very real and most dogs should be spayed before their first heat. Certainly this is valid for small and medium sized dogs: After discussion of the pros and cons of waiting owners of large and giant breed dogs may elect to delay alter.

**Conclusion**

Currently, the pros far outweigh the cons when it comes to spaying and neutering the average pet. More study is needed in this area due to the large number of conflicting reports and inadequate data. It is important to keep in mind that millions of animals are still euthanized in shelters every year in this country: It would be irresponsible to undermine the efforts of humane organizations and shelters at reducing pet overpopulation when the information that we have regarding the potential negative consequences of spaying and neutering animals is far from conclusive.
References


The Veterinarian’s Role in Recognizing and Reporting Animal Abuse
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Veterinarians have a unique role to perform and an ethical duty to recognize and report animal abuse. Increasingly, the lay public is aware of the connection between animal abuse and interpersonal violence. This increasing awareness is elevating the seriousness of animal related crimes in the minds of the public and law enforcement, resulting in more laws related to mandatory reporting of animal abuse on the part of Veterinarians. In fact, in 2016 the Federal Bureau of Investigation began tracking cases of animal abuse.

In this talk I will describe and define six types of animal cruelty and the laws that pertain to these areas. Additionally, I will cover how Veterinarians can help and what Veterinarians should know about reporting. Due to the fact that this Conference draws participants from throughout the Midwest and beyond, we will not be able to cover the specific laws of your state or municipality. Many of the examples given will be based in Oregon, my home state.

Anticruelty laws

- **Neglect** – Involves intentionally, knowingly, recklessly or with criminal negligence failing to provide minimum care for an animal in one’s custody or control. There are many reasons that people will neglect animals including ignorance, stress, apathy, poverty, and psychological barriers to normal behavior. It is important to note that in most cases of animal cruelty the perpetrator can be held accountable for their actions: Ignorance is not a defense and mental illness is generally not an acceptable excuse. If the person understands the charges against them and are mentally able to participate in their defense they can face prosecution despite a diagnosis of mental illness. We will discuss degrees of neglect and the types of cases seen in practice.

- **Hoarding** – An animal hoarder is defined as someone who has accumulated a large number of animals that overwhelm his or her ability to provide a minimum of care, including adequate nutrition, sanitary conditions, and veterinary care. The key point here is that it is not about the number of animals, it is about what happens to the animals. In fact, that is one of the challenges of trying to deal with animal hoarding through the legal system and/or municipal codes. The number of animals is not the problem; the problem is that it has overwhelmed the person’s ability to provide a minimum of care. There are several key characteristics that have been described which can be helpful in identifying an animal hoarder:
  - Obsessive attempts to accumulate or maintain a collection of animals in the face of progressively deteriorating conditions
  - Failure to provide minimal standards of sanitation, space, nutrition, and veterinary care for the animals
  - Inability to recognize the effects of this failure on the welfare of the animals, human members of the household, and the environment
  - Denial or minimization of problems and living conditions for people and animals

We will discuss the types of animal hoarders that have been identified and review a few cases that have been seen at the Oregon Humane Society.

- **Abuse** – Animal abuse is defined as intentionally, knowingly, or recklessly causing physical injury to an animal. There are degrees of abuse which increase with the severity of injury to the animal and result in increased penalties for the perpetrator. This is, roughly, how animal abuse is currently defined and prosecuted in Oregon:
  - Animal Abuse in the First Degree:
    - Intentionally, knowingly, or recklessly causing serious physical injury to an animal, or cruelly causing the death of an animal.
    - Animal abuse in the first degree is a misdemeanor in Oregon.
  - Aggravated Animal Abuse in the First Degree:
    - The perpetrator maliciously kills an animal
    - OR
    - Intentionally or knowingly tortures an animal
    - Mandatory veterinary reporting in Oregon
    - Aggravated animal abuse is a felony in Oregon
  - Animal Abuse in the Second Degree:
    - Intentionally, knowingly, or recklessly causing physical injury to an animal.
    - Animal abuse in the second degree is a misdemeanor in Oregon.
We will discuss animal abuse in some detail. It defies logic but people who abuse and neglect animals sometimes take them to the Veterinarian! It is important that practitioners are aware of abuse and neglect – we won’t find it if we are not looking for it.

- Abandonment – Abandonment is defined as intentionally, knowingly, recklessly or with criminal negligence leaving a domestic animal at a location without providing for the animal’s continued care. It is specifically written into Oregon law that it is a crime to abandon an animal at or near an animal shelter, vet clinic, or other place of shelter.
- Fighting - Involvement in animal fighting, participation in animal fighting, and possessing animal fighting paraphernalia are all separate crimes: This enables prosecutors to elevate the seriousness and penalties for involvement in animal fighting. Most crimes involving animal fighting are Class C felonies in Oregon: Penalties for a Class C felony are up to five years of imprisonment and/or a maximum fine of $125,000. Interestingly, being a spectator at a cockfight is now a felony in Oregon. It is important to note that with animal fighting there is often other criminal activity taking place in the vicinity. Guns, drugs, money laundering, and even prostitution may be encountered in this environment. For law enforcement officials who are ambivalent about animal welfare issues this may provide an incentive to pursue these criminals.
- Sexual Assault - A person commits the crime of sexual assault of an animal if the person:
  - Touches or contacts, or causes an object or another person to touch or contact, the mouth, anus or sex organs of an animal or animal carcass for the purpose of arousing or gratifying the sexual desire of a person; or
  - Causes an animal or animal carcass to touch or contact the mouth, anus or sex organs of a person for the purpose of arousing or gratifying the sexual desire of a person

How veterinarians can help
- Recognize – Recognizing that there is a problem is certainly the first step. Awareness of animal related crime involves looking for it with a healthy degree of suspicion. For example, is the pet emaciated, have overgrown nails and matted fur? These may be signs of neglect. Does the story that the owner is telling you fit with the injuries that you see? If not, this may be a case of abuse.
- Prevent – Prevention of the crime of neglect, for example, may involve client education. Grooming, proper feeding and watering, and dental care often seem obvious to people in our profession. Depending on the education level or mental status of your client these may or may not be so obvious. In some cases prevention is not possible or appropriate – you should go straight to reporting. Our Humane Investigations Officers actually spend more time educating people on the proper care of animals (and following up on these cases) than arresting people.
- Address/Report – Please see below for information about reporting.
- Medical Expert Witness – Noted animal forensics expert Dr. Melinda Merck once said that “Veterinarians are the Golden Retrievers of witnesses…we’re used to people liking us”. Unfortunately, a court of law can be a contentious environment due to our adversarial legal system and Veterinarians should be prepared for this. A medical expert is a vital part of a successful prosecution. Explaining evidence, interpreting data, and answering questions should be expected. In particular, the defense attorney may challenge the case on legal or scientific grounds. Defense attorneys will often have their own expert witness: Lack of an expert medical witness for the prosecution may result in an unsuccessful prosecution.

What veterinarians should know about reporting
- The Law – What crimes are you mandated to report in your state? You might be surprised to learn that in some states, Veterinarians are mandatory reporters of child abuse. It is incumbent upon us as professionals to know, at the very least, what crimes must be reported.
- What to Record – The bottom line is that good medical records are necessary. Medical history, exam findings and any laboratory/radiographic findings. Keep in mind that your records will be a part of any legal proceedings that take place.
- What to Report – An account of the situation or incident is a vital first step. A detailed description of animal, physical exam findings, medical records and observations of the client are also necessary. If possible a detailed description of suspect, including (if known) their name, address, phone number, license plate number, and a physical description of the person or persons involved. Fortunately, a lot of this information will be obtained when the client makes an appointment or checks in.
- Where to Report – Where to report depends on your location. Local animal control officials, the local SPCA or Humane Society, or local law enforcement are all good resources. Looking into where to report ahead of time is advisable.

Conclusion
Veterinarians have a professional obligation, an ethical obligation, and sometimes a legal obligation, to report animal abuse. We are often in the unique position to help animals in the early stages of abuse, preventing more serious harm in the future: This is especially
important in light of the link between animal abuse and domestic violence. Knowing what to report, where to report, and how to report animal abuse are important – and being prepared in advance is imperative because these can be dynamic, emotionally charged situations. Please take the time to find out where to report animal abuse in your area.

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Anesthesia and Common Surgical Procedures in Small Ruminants

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Preanesthetic considerations

Small ruminants, as well as other ruminants, are susceptible to complications associated with anesthesia and recumbency. Positioning of these animals in dorsal or lateral recumbency for surgery allows for the weight of the abdominal viscera to shift ventrally and cranially, causing the diaphragm to be pushed further into the thoracic cavity, thereby reducing the functional residual capacity of the lungs. As a result, as increased ventilation/perfusion mismatch may lead to significant hypoventilation and hypoxemia during anesthesia. In addition, the weight of the abdominal viscera may compress great vessels such as the vena cava leading to decreased venous return, cardiac output, and arterial blood pressures. Therefore, close monitoring of cardiovascular and pulmonary functions and institution of appropriate treatments to ensure normal arterial blood pressure and adequate ventilation are important parts of perioperative anesthetic management.

Ruminal tympany, bloat, regurgitation, and aspiration pneumonia are common problems associated with general anesthesia in farm animal species that should be anticipated and addressed with proper precautions. Ruminal fermentation continues even in anesthetized animals. Normal, awake animals are able to relieve the gas produced by fermentation through eructation. Sedatives and anesthetics tend to inhibit GI motility and prohibit eructation, thus allowing gas to accumulate in the rumen. An average rumen capacity of small ruminants has been reported to be 15-18L. Bloating, especially in non-fasted animals, can occur during anesthesia and compromise the cardiopulmonary systems by increasing intra-abdominal pressure resulting compression of the diaphragm and great vessels thus further complicating the already compromised cardiopulmonary function resulting from abnormal positioning required by the surgery. Fasting of these patients prior to anesthesia reduces the amount of gas produced by fermentation and minimizes its detrimental effect on the cardiopulmonary systems.

Regurgitation and aspiration of stomach content can occur in farm animal species during anesthesia, especially in non-fasted animals. The risk of regurgitation decreases significantly when water is withheld for 6-12 hours and feed is withheld for 12-24 hours prior to anesthesia in small ruminants. Domesticated ruminants have a large rumen that is usually full of liquid materials which does not empty completely even after 24-48 hours of fasting. Regurgitation can occur during either light (active regurgitation) or deep (passive regurgitation) anesthesia in ruminants despite preoperative fasting and withholding of water. If the airway is not protected, a large amount of ruminal materials can be aspirated into the trachea and reach the small airways. Aspiration of acidic stomach fluid causes immediate reflex airway closure and destruction of type II alveolar cells and pulmonary capillary lining cells. Consequently, pulmonary edema and hemorrhage, hypoxemia, and arterial hypotension develop due to a loss of alveolar and capillary integrity leading to reflex airway closure, bronchospasm, dyspnea, hypoxemia, and cyanosis. The greatest impact of aspirating rumen contents lies in the amount of bacterial microflora and solid food materials aspirated. Animals may die before an endotracheal tube can be placed to protect the airway in extreme cases. Preoperative withholding of feed and endotracheal intubation with an adequately inflated cuff immediately following induction of anesthesia are recommended in all anesthetized farm animals.

Ruminants normally salivate profusely during anesthesia. Total amounts of saliva secretion in conscious adult sheep have been reported to be 6-16L per 24 hours. In the past, anticholinergics like atropine were used routinely as part of the anesthetic induction regimen in an effort to prevent salivation. However, atropine only reduces the water content of the saliva, thus causing the saliva to become more viscous and increasing the potential of airway obstruction, particularly in neonates. If the trachea is left unprotected during anesthesia, large amounts of saliva may be aspirated. Therefore, tracheal intubation with appropriate inflation of the cuff immediately following induction should be instituted to protect the airway. Placing a sandbag or rolled-up towel under the neck of a small ruminant patient to elevate the throat latch so that the mouth opening is lower that the occiput allows saliva to escape, avoiding the potential for aspiration. This technique also helps to minimize the flow of passive regurgitation during deep anesthesia.

Preanesthetic preparation

When possible, small ruminants should be fasted for 12-24 hours and water withheld for 8-12 hours before induction of anesthesia. Preanesthetic fasting may not completely prevent regurgitation, but it will decrease the amount of solid matter in the rumen. Fasting also does not prevent bloating during anesthesia, but it reduces the rate of fermentation, thus reducing the amount of gas formation, the severity of bloating, and its effect on ventilation. Neonatal ruminants or those less that 3 weeks of age, can be treated as monogastrics. Fasting of young ruminants less than 4 months of age is not recommended because of the potential for hypoglycemia and prolonged recovery. Fasting may not be possible under emergency situations, and precautions should be taken to avoid aspiration of gastric fluid and ingesta. Prevention of regurgitation and aspiration of ruminal content can be achieved effectively by placing the small ruminant patient in sternal recumbency and endotracheal intubation instituted immediately following induction. Tracheal intubation is more difficult in small ruminants as compared to large ruminants and other species because their mouth do not open widely, the intermandibular space in narrow, and the laryngeal opening is distant to the thick base of the tongue. The technique used for tracheal
intubation of small ruminants is easier when the animal is placed in sternal recumbency immediately after induction of anesthesia. Intubation is best accomplished with the help of a guide tube/stylet and long-bladed laryngoscope (250-350 mm). Hyperextending the animal’s neck helps visualization of the larynx. A cuffed endotracheal tube should be used to provide an adequate seal between the tube and the tracheal mucous membrane so to prevent aspiration of saliva and regurgitated ruminal contents. The animal should be maintained in sternal recumbency until the cuff is inflated. Blind intubation, similar to that used in horses, has been used for intubation in sheep and goats; however, it may require multiple attempts in order to successfully place the endotracheal tube in the trachea. Another technique described as “stick intubation” has been used effectively. With the animal in lateral recumbency, a small-diameter rod made of wood or stainless steel can be used as a stylet to stiffen the endotracheal tube. One hand occludes the esophagus, and the other hand manipulates the endotracheal tube into the trachea. Care and gentle maneuvering should be used to prevent initiating laryngeal spasm and to minimize trauma to the oral mucous membrane.

Castration
Castration of the normal, young male is among the most commonly performed surgical procedures in small ruminants. An emerging trend is to delay castration in some animals (meat goats and pets) who are fed very high concentrate diets due to concerns of urolithiasis. Although bloodless surgical techniques such as banding and use of the Burdizzo emasculatome are commonly used, it may be necessary to perform surgical castration. Surgical castration is performed by excision of the distal one half to one third of the scrotum with a scalpel blade or using a Newberry knife to leave two flaps of scrotal skin (cranial and caudal). The testicles are exposed and removed after making the skin incision of choice. Many clinicians may prefer to place ligatures on the spermatic cord or to crush the cord with an emasculator, to ensure appropriate hemorrhage control. Care should be taken in using an emasculator routinely used for larger species such as horses in that an emasculator that functions well in equine castration may not adequately crush the smaller cord of the small ruminant. Therefore, ligatures may be preferred which does not take little additional time and no significant expense. The spermatic cord is then transected distal to the ligature. The testicles may be removed by traction, as is commonly done in calves, but this method has been associated with herniation almost immediately after castration in some cases. If the traction method is used, pressure should be applied over the respective external inguinal ring with fingers while pulling the testes with the other hand to minimize trauma to the ring and subsequent herniation. Most animals will require a minimum of sedation and local anesthesia; older animals typically require general anesthesia. An alternative to traditional castration equipment is the Henderson castration tool which uses a twisting action to effectively ligate the cord. Local anesthesia may be performed by blocking the scrotal using a line block of local anesthetic proximal to the incision in the scrotal skin as well as another dose of local anesthetic into the center of each testicle.

Dehorning
Kids older than 2-3 weeks of age, those whose previous dehorning resulted in the growth of abnormal horn tissue (scur), and adult goats are all suitable candidates for dehorning. Disbudding should be considered for kids less than 2-3 weeks of age. It is recommended that general anesthesia be used for dehorning in adults, particularly in males with large horns. However, sedation (e.g. xylazine) and a cornual nerve block may also be effective. Because of anatomical differences, the cornual nerve block in goats requires at least two injection sites per horn versus the one injection site commonly used in cattle. In goats, the cornual nerve is a branch of the zygomaticotemporal nerve that lies halfway between the lateral canthus of the eye and the base of the horn. The horn base in goats is also heavily innervated by the cornual branches of the infratrochlear nerve which exits the orbit at or in close proximity to the medial canthus. Because of the widespread branching, the nerve is best blocked using a line block midway between the medial canthus of the eye and the median horn base. Alternatively, a ring block around the base of the horn may also be used for anesthesia and dehorning.

Owners should be warned that this procedure can be quite bloody. In addition, it may take 4-6 weeks to heal, result in secondary sinusitis, leave holes that never completely heal, or possibly result in brain abscess. The skin around the horns should be clipped and surgically prepared. The clinician makes a circular incision through the skin 2 mm outside of the horn-skin junction. The strip of skin between the two horns should be left intact in order to improve healing and shorten healing time. An obstetric wire is then placed into the incision and a helper/technician holds the head to prevent excessive motion. With the surgeon standing in front of the animal. The cut should be made in a rostral-ventral direction. Alternatively, a small Barnes dehorner may be used to cut or snip off the horn tissue; the cut should be made to avoid injuring the thin skull. Hemorrhage may be controlled by cautery, pressure, or pulling the bleeding vessels with hemostats. If the animal has a small horn base, the surrounding skin can be undermined and stretched over the opening created by horn removal. Closing the skin over the surgical site allows for quicker recovery but is rarely possible in adult males without removing some of the frontal bone. An antibiotic ointment can be applied and a gauze pad or other absorbable material can be placed over each removal site. The pads can be held in place by tape wrapped around the head or by a piece orthopedic stockinette pulled over the animal’s head with holes cut for the eyes and ears. Animals may be given antibiotics, tetanus prophylaxis/antitoxin, or
Before the catheter is deflated and removed. The Foley catheter should not be removed before day 7 after surgery to
avoid complications than restraint in dorsal recumbency. The rumen is dorsal aspect up with this positioning which serves to help retain
abdominal viscera within the abdominal cavity. Should the dam bloat in left lateral recumbency, the rumen will have more room to
dilate before respiratory compromise develops, and it can be decompressed if needed. Although, experience suggests that this is not a
frequent problem. If bloat does occur, the practitioner usually is wise to quickly complete the surgery and return the dam to sternal
recumbency rather than being overly concerned with rumen distention. The muscular body wall is relatively thin in small ruminants in
comparison to other large animal species. Thus, the utmost care is required in making the body wall incision to inadvertently
damaging deeper structures (rumen). The gravid uterus usually can be well exteriorized in small ruminants to allow packing off of the
uterus with sterile towels before the uterus is opened. If possible, both uterine horns should be exteriorized; although, this may be
difficult in some cases (multiple large fetuses). The uterine incision is best made on the greater curvature of the uterus in an easily
accessible area between the ovary and the cervix. This incision can be made over the hindlimbs of the fetus. However, it is frequently
easier to make the incision over the head of the kid with care taken not to make a laceration on the face of the fetus. The practitioner is
best served by making an incision over each respective fetus, but occasionally two fetuses in the same uterine horn can be removed
through one uterine incision. However, it is difficult to maneuver a fetus from one uterine horn into the other to permit removal though
one uterine incision. So, it is often best to make at least one uterine incision per uterine horn when fetuses are present in both uterine
horns. Any placenta that is not firmly attached to the uterus should be removed before closure of the uterus. The uterine incision
should be closed with absorbable suture in an inverting pattern (e.g. Utrecht, Cushing, Lembert) to achieve a fluid-tight seal. One-
layer closure is usually sufficient; although, a second layer can be added if the integrity of the first layer is in question. The uterus
should then be cleaned of any blood or debris before it is replaced into the correct position in the abdominal cavity. The muscular
body wall is closed with absorbable suture in a manner chosen by the practitioner. Closure of each muscle layer separately is
recommended, but in the interest of time, layers may be combined without detriment which will be discussed in further detail.

Cesarean section
Cesarean section is the most common abdominal surgery performed in small ruminants. Although aseptic technique in a hospital
setting is ideal for better results and fewer complications, the procedure can certainly be performed in the field with good results.
Cesarean section can be performed with the animal in dorsal recumbency using a ventral midline approach, but is more commonly
performed with the animal in right lateral recumbency through a left paralumbar fossa incision. Local anesthesia obtained with the use
of a paravertebral or line block is adequate. One must remember to limit the dose of lidocaine used for local anesthesia to no more
than 6 mg/kg of body weight. Diluting the 2% lidocaine from the bottle with an equal volume of saline will create a 1% solution,
which achieves adequate anesthetization of the surgical site without causing toxicity. When necessary, a 1 part lidocaine: 2 parts saline
creates a 0.5% solution may be used and has also offered adequate anesthetization. The body wall of small ruminants is relatively thin,
so the local infiltration does not need to be administered as deeply as in cattle. Some animals may need mild sedation, but most
patients are easily restrained in lateral recumbency without sedation. Regional anesthesia using a lumbosacral epidural is a viable
alternative for cesarean section in small ruminants. The recommended dose is 2 mL of 2% lidocaine per 10 kg (22 lb) body weight. An
18- or 20- gauge, 3.8 cm (1.5 inch) needle is sufficient in most animals. Onset of anesthesia occurs within 5-15 minutes and generally
lasts 60-120 minutes.

The left paralumbar fossa approach allows the dam to remain in lateral recumbency which leads to far fewer respiratory
complications than restraint in dorsal recumbency. The rumen is also dorsal aspect up with this positioning which serves to help retain
abdominal viscera within the abdominal cavity. Should the dam bloat in left lateral recumbency, the rumen will have more room to
dilate before respiratory compromise develops, and it can be decompressed if needed. Although, experience suggests that this is not a
frequent problem. If bloat does occur, the practitioner usually is wise to quickly complete the surgery and return the dam to sternal
recumbency rather than being overly concerned with rumen distention. The muscular body wall is relatively thin in small ruminants in
comparison to other large animal species. Thus, the utmost care is required in making the body wall incision to inadvertently
damaging deeper structures (rumen). The gravid uterus usually can be well exteriorized in small ruminants to allow packing off of the
uterus with sterile towels before the uterus is opened. If possible, both uterine horns should be exteriorized; although, this may be
difficult in some cases (multiple large fetuses). The uterine incision is best made on the greater curvature of the uterus in an easily
accessible area between the ovary and the cervix. This incision can be made over the hindlimbs of the fetus. However, it is frequently
easier to make the incision over the head of the kid with care taken not to make a laceration on the face of the fetus. The practitioner is
best served by making an incision over each respective fetus, but occasionally two fetuses in the same uterine horn can be removed
through one uterine incision. However, it is difficult to maneuver a fetus from one uterine horn into the other to permit removal though
one uterine incision. So, it is often best to make at least one uterine incision per uterine horn when fetuses are present in both uterine
horns. Any placenta that is not firmly attached to the uterus should be removed before closure of the uterus. The uterine incision
should be closed with absorbable suture in an inverting pattern (e.g. Utrecht, Cushing, Lembert) to achieve a fluid-tight seal. One-
layer closure is usually sufficient; although, a second layer can be added if the integrity of the first layer is in question. The uterus
should then be cleaned of any blood or debris before it is replaced into the correct position in the abdominal cavity. The muscular
body wall is closed with absorbable suture in a manner chosen by the practitioner. Closure of each muscle layer separately is
recommended, but in the interest of time, layers may be combined without detriment which will be discussed in further detail.

Tube cystotomy
Placement of a Foley (16 to 24 French) catheter into the bladder and exiting through the ventral abdomen allows for continual
drainage of urine. By routing urine flow through the catheter, the urethra is allowed to rest in order to decrease inflammation and
promote healing. A small skin incision is made lateral to the paramedian incision and inserts the catheter subcutaneously, where it
enters the abdomen and then the bladder. A purse-string suture is placed in the bladder wall to position the Foley. A small stab
incision is made in the middle of the purse-string and the balloon end of the Foley catheter is placed into the bladder, after which the
purse-string suture is tightened. After inflating the Foley catheter with saline, the bladder is tacked to the body wall with minimal
tension. A one-way valve can be made from a finger of a latex glove and placed over the end of the catheter to create a type of
Heimlich valve which helps decrease the incidence of ascending infections. The celiotomy site should be closed in three layers with
an absorbable suture, and the subcutaneous tissues and skin closed in routine fashion. Sutures can be removed in 10 to 14 days. Many
goats will attempt to pull or chew the tubes, so belly bandages, Elizabethan collars, and close monitoring should be used to help
ensure catheter placement. Clamping the catheter should begin on the fourth day after surgery to allow for normal urination. This
should be done in a dry stall and with increasing duration until a full-stream urination is achieved. Normal urination should occur for
1 to 2 days before the catheter is deflated and removed. The Foley catheter should not be removed before day 7 after surgery to
reduce the chances of urine leaking from the bladder. This bladder defect is allowed to heal spontaneously.
Bladder marsupialization

Bladder marsupialization offers excellent long-term survival and is a good technique for non-breeding animals and those whose perineal urethrostomy sites have strictured to the point of preventing urine flow. The animal is anesthetized and placed in dorsal recumbency. The skin is clipped and aseptically prepared. An 8 to 12 cm paramedian incision is made in the caudoventral abdomen parallel and 2 to 4 cm lateral to the prepuce. The apex of the bladder is exteriorized, the bladder decompressed, stay sutures placed 4 to 5 cm apart, and the cystotomy incision is made between the stay sutures. A second abdominal incision is made on the opposite side of the prepuce. The site of the second abdominal incision is chosen to minimize urine scalding of the surrounding skin. The apex of the bladder should be pulled or lifted into the second abdominal incision by the stay sutures. All four corners of the bladder should then be sutured to the abdominal wall. The seromuscular layer of the bladder should be sutured to the fascia in a circumferential fashion using a horizontal mattress pattern with absorbable suture. The bladder margins are sutured circumferentially to the skin with an absorbable suture. The original abdominal incision is closed. Urine is voided from the bladder through the marsupialized site. Therefore, the incision should be large enough to allow urine flow but not large enough to allow bladder eversion or prolapse. Systemic antibiotics should be administered prior to surgery and for as long as 14 days post-operatively (procaine penicillin G 22,000 IU/kg IM BID, ceftiofur 2.2 mg/kg IM SID to BID). Short-term complications include bladder prolapse and cystitis, and over time fibrotic stromal closure over the marsupialization site may occur.

References available upon request.
Pregnancy toxemia
Pregnancy toxemia (ketosis) affects does during late gestation. It occurs most commonly in either fat or thin animals that carry two or more fetuses. The condition develops when the doe cannot ingest enough nutrients to meet both the glucose requirements of the growing fetus and her own body’s metabolism. During early gestation, the dam’s increased appetite is enough to encourage her to compensate for the increased nutrient needs. However by late gestation, the growing fetuses are occupying more space in the dam’s abdomen which leads to the dam being physically incapable of eating enough to meet her nutritional needs unless more nutrient-dense feeds are provided. If adequate energy is not available to the gestating doe, she can metabolize body fat to meet her own nutrient requirements. When fatty acids are metabolized at high rates, ketone bodies are produced, which can be dangerous in high levels. The condition where excess ketones are present in the bloodstream, known as ketosis, results in depression and anorexia until the doe becomes too weak to stand.

Pregnancy toxemia is a common indication for the use of dextrose-containing solutions over a period of time. In sheep and goats, a variety of metabolic derangements have been documented as part of the pregnancy toxemia, including hyperketonemia, ketonuria, metabolic acidosis, hypocalcemia, hypoglycemia, and decreased liver function from hepatic lipidosis. Hypoglycemia is an inconsistent finding in cases of pregnancy toxemia. Often, the does may have normal blood glucose or be hyperglycemic.

Pregnancy toxemia can prevented by properly managing the weight of does throughout the year, and especially prior to breeding and during gestation. Does should be body condition scored at breeding, as overweight and excessively thin does are at a higher risk for ketosis. An ultrasound can also be performed during gestation to determine fetal number. Animals gestating multiples can be fed and managed differently than those with singletons. Whenever possible, does should be divided into different groups or pens so that they can be managed differently during gestation to minimize their risk of toxemia. While it is acceptable for overweight does to lose weight during the first two trimesters, they should be gaining weight by the third trimester. Feeding grains with increased energy density during the third trimester, or about six weeks prior to kidding, will help to prevent pregnancy toxemia. Providing higher quality hay is also a good idea for gestating ewes or does.

Hypoglycemia
Hypoglycemia is most commonly seen in as a cause of weakness and depression in neonates. However, infectious causes of depression and weakness should always be considered as a potential contributing factor to these clinical signs. Hypoglycemia is relatively easy to diagnose with the aid of an inexpensive, portable glucometer. Kids typically develop hypoglycemia due to weakness, shock, environmental stress, disease of kid or dam, and poor nutrition. Administration of 50 mL/kg of dextrose (or 5% of body weight) in warm milk replacer or 1 mL/kg of 50% dextrose, either intravenously or orally (diluted to 5% dextrose), should provide ample energy to correct hypoglycemia. Intravenous administration may be necessary if gut motility is absent. If the kid does not regain an appetite, then follow-up therapy may be necessary.

Hypocalcemia
Hypocalcemia can be a problem in does most commonly shortly before or after parturition. However, the disease occurs more common after parturition. Before parturition, there is an abrupt demand for calcium in the last 3-4 weeks before parturition in does with more than one fetus due to the calcification of fetal bones. With the demands of lactation, there is a abrupt demand for calcium. The body may require 1 or days to accrue the necessary enzymes capable of mobilizing bone stores of calcium. High dietary intake of calcium, phosphorus, or some cations (potassium and sodium) decreases the production of parathyroid hormones. During decreased parathyroid function, less 1,25-dihydroxycholecalciferol is produced. Lack of this hormone results in lowered absorption and mobilization of calcium from the intestines and bones. Low dietary calcium or increased amounts of dietary anions enhances the production and release of parathyroid hormones.

Early in the course of the disease, animals most commonly present with a stiff gait, tremors, and tetany with decreased rumen motility. They may also be ataxic or constipated. As the disease progresses, animals begin to have increased heart and respiratory rates, bloat, and depression. Corneal and pupillary light reflexes are normal initially but may become depressed before disappearing entirely. Diagnosis is usually made based on history and signalment suggestive of an animal at risk of development of hypocalcemia. Serum calcium concentrations less than 4-5 mL/dL in goats are diagnostic. If animals are showing clinical signs of disease, the immediate attention is required. Intravenous administration of calcium borogluconate (50-100 mL of a 23% solution) is most commonly used. Oral calcium administration of a calcium gel designed for cattle but based on goat body weight may help prevent relapse. If using an oral calcium gel, the author has found it helpful to rinse the oral cavity with water following administration of the gel to help prevent oral irritation. Subcutaneous administration of calcium may also be used but solutions containing dextrose or other
Electrolytes should be avoided, if possible, as some have been associated with abscess formation. Cardiac monitoring is necessary during treatment; therapy should be slowed or stopped if arrhythmias occur. If the treatment is successful, the animal will usually stand, urinate and/or eructate within 20 minutes.

**Metabolic acidosis**

Metabolic acidosis can be seen in goats as a result of absorption of D-lactate from the gastrointestinal tract (e.g., with grain overload or enterocolitis) and sodium loss with secretory diarrhea. Sepsis or other causes of septic shock may also cause metabolic acidosis due to L-lactate accumulation as a result of poor tissue perfusion. Animals with pregnancy toxemia may also experience metabolic acidosis. Isotonic sodium bicarbonate (1.3% NaHCO₃) and hypertonic sodium bicarbonate (5% or 8.4% NaHCO₃) may be used alone or added to other solutions to directly correct metabolic acidosis.

The base deficit is calculated by subtracting the measured bicarbonate from the normal total bicarbonate (approximate normal bicarbonate is 25 mEq/mL). The amount of bicarbonate to administer is calculated as follows:

**Neonates**

\[ \text{mEq bicarbonate needed} = \text{base deficit} \times \text{body weight in kg} \times 0.6 \]

**Adults**

\[ \text{mEq bicarbonate needed} = \text{base deficit} \times \text{body weight in kg} \times 0.3 \]

The constants 0.6 and 0.3 represent the approximate proportion of extracellular fluid volume relative to total body weight, which is different for neonates compared with mature animals. From this formula, the total milliequivalents of bicarbonate needed to completely correct the acidosis can be calculated. In some cases of neonatal diarrhea or severe grain overload, it may be necessary to administer the entire amount of calculated base deficit of bicarbonate in order to correct the acidosis. However, most cases will resolve with partial correction with administration of approximately half of the deficit over 2-4 hours. This will then be followed by complete correction of the deficit after fluid therapy which allows the normal physiologic compensatory mechanisms to function in the animal.

References are available upon request.
The basics: *Haemonchus contortus* and *Trichostrongylus* sp.

Internal parasites of small ruminants are one of the most significant health problems facing both owners and veterinarians. *Haemonchus contortus* and *Trichostrongylus* sp. are the two nematode parasites that are commonly identified on routine fecal examinations. *Haemonchus contortus* pierces the mucosa of the abomasum and causes anemia and protein loss. The eggs are deposited onto pasture where they develop into first and second stage larvae within the fecal pellet. The infective third stage larvae migrate up grass blades where they become suspended in dew droplets and are then ingested by the small ruminant. These larvae then mature into adults in the abomasum. The adult parasites live in the abomasum with each adult can produce 5,000 to 10,000 eggs per day. The pre-patent period, or time from ingestion of the third stage larvae to the time mature adults produce eggs that are passed in the feces, is about 21 days. Clinical signs of infestation with *H. contortus* may include unthriftiness, rough hair coat, pale mucous membranes, submandibular edema, and death. Diarrhea is not a common clinical manifestation of infestation with *H. contortus*.

*Trichostrongylus* sp. inhabit the small intestine and cause diarrhea and protein loss but not anemia. Many veterinarians and producers use the FAMACHA parasite control program for parasite management. However, it is important to remember that the FAMACHA program will miss severe infestations of *Trichostrongylus* sp.

Anthelmintic resistance

Parasite resistance to anthelmintics has become an increasingly common and serious problem for the last several years. Few anthelmintics are approved for use in small ruminants in the United States, and due to the tremendous investment needed by pharmaceutical companies; new discovery of anthelmintics is unlikely. The three common classes of anthelmintics used in small ruminants and camels are: 1) Benzimidazoles which include albendazole (Valbazen®), fenbendazole (Safe-Guard®, Panacur®), and oxfenbendazole (Synanthic®); 2) Imidathiazole/Thetrahydropyrimidine which include levamisole (Tamisol®, Levasol®), morantel tartrate (Rumate®), and pyrantel pamoate or tartrate; and 3) Macrolides which include ivermectin (Ivomec®), moxidectin (Cydectin®), eprinomectin (Eprinex®), and doramectin (Dectomax®). Only four drugs have been approved by the Food and Drug Administration (FDA) for use in goats. These drugs include morantel, thiabendazole (no longer marketed), fenbendazole, and phenothiazine. The FDA does allow for extra-label use of other anthelmintics as an exclusive privilege of the veterinary profession and only when a *bona fide* veterinarian-client-patient relationship exists and an appropriate medical diagnosis is made.

Resistance to benzimidazole, levamisole, and other imidazothiazole anthelmintics has been documented for many years. Even more recently, documented cases of resistance to ivermectin and other macrolides has been reported with increasing frequency. Although moxidectin (Cydectin®) has been considered the drug of last resort in many cases there has been a recent increase in the number of cases with resistance to moxidectin. It is important to remember that resistance to one drug in a class usually means resistance to all drugs in that class. The efficacy of moxidectin in the face of resistance is only due to increased potency. Therefore, it is necessary that both producers and veterinarians practice prudent use anthelmintics and learn alternative practices for controlling intestinal parasites in small ruminants and camels.

Resistance to anthelmintics has become a problem for many reasons. Some reasons include inappropriate dosing and administration of anthelmintics including rotational use of anthelmintics, and inappropriate use of pasture management after anthelmintic use.

Diagnosing resistance

A quantitative method to determine parasite burden and aid in diagnosis of resistance is extremely important. The modified McMaster’s technique is one of the most commonly used tools to determine fecal egg counts and, thus, serves as a good aid in diagnosing resistance to anthelmintics. The modified McMaster’s technique is easily performed but does require a McMaster’s slide. When determining resistance, a modified McMaster’s is performed to determine the number of eggs per gram of feces. The animal is then administered a given anthelmintic, and then 10-14 days later another modified McMaster’s technique is performed. If there is no resistance to the anthelmintic administered to the animal, one should expect to see a 95-99% reduction in the number of eggs per gram of feces. If the anthelmintic was properly administered and post-treatment egg counts are still high, then resistance to the anthelmintic administered can be concluded. As previously mentioned, it is important to remember that resistance to one drug in a class usually implies resistance to all drugs in that class. It is necessary to obtain a representative population on the farm due to individual animal variations.

Another method used to diagnose resistance of anthelmintics is a larval development assay or DrenchRite® assay. This is a test that is only performed in the laboratory of Dr. Ray Kaplan at the University of Georgia. The Drench Rite® Assay is an *in vitro* test that uses collected feces to “grow” larvae from which numerous anthelmintics are tested to determine resistance. This assay is more...
expensive than the modified McMaster’s technique but provides so much more information to the practitioner and client regarding resistance issues on the farm.

Management and control of parasites

FAMACHA®

FAMACHA® is a copyrighted parasite control program that was developed in South Africa to identify severely parasitized sheep and goats. This farm-based system uses visual inspection and analysis of the mucous membrane pallor as an indication of anemia and, thus, degree of parasitism with Haemonchus contortus. In any given flock or herd, 20-30% of the animals harbor 70-80% of the parasites. The FAMACHA® program allows producers to treat only the most severely parasitized and anemic animals on a farm which reduces the number of dewormings and helps prevent the development of resistance. FAMACHA® is based on the concept of refugia. Refugia refers to all of the parasite eggs and larvae already on pasture and all the parasites in the animals that have not seen anthelmintics. Thus by only deworming the most severely parasitized and anemic animals, there is a larger population of naïve parasites that have not exposed to anthelmintics (large refugia). This is how FAMACHA® helps prevent anthelmintic resistance. The most important factor responsible for widespread development of anthelmintic resistance is the common practice of treating all animals in a herd at one time. This practice leaves no parasites in refugia which leads to resistance; meaning that the only eggs deposited onto the pasture for the next several weeks are those that survived anthelmintic treatment or the resistant parasites. Those animals needing multiple treatments should be culled from the herd. Over a period of generations, the genetics of the herd favor animals that are parasite resistant. However, it is important to remember that the FAMACHA® program should only be applied to adults because anemia in kids and lambs can progress quite rapidly. Due to the periparturient rise in parasites, decreased immunity to gastrointestinal nematodes and high nutritional demands, periparturient does and does in lactation must also be monitored very carefully.

The FAMACHA® program is easy to use but requires intensive training and education of producers. This training can be accomplished by going through a program that is given by members of the American Consortium for Small Ruminant Parasite Control (www.wormx.info). Dates for training workshops and additional information can be found at www.scsrcpc.org. It is important to remember that the FAMACHA® program is only evaluates the level of anemia due to Haemonchus contortus. Therefore, periodic fecal examinations are necessary to evaluate the level of infestation with other parasites.

Grazing practices

Another extremely important point that is imperative for good parasite control involves preserving pastures. Ideal stocking rates for small ruminants is 6-8 head per acre. If enough pasture is available, producers should remove animals from the pastures for 3-6 months to allow the larvae on pasture to die. Other practices that to reduce parasite burdens include alternating or co-grazing pastures with horses or adult cattle, and after deworming, keep the animals on a dry lot before putting on a new, clean pasture. In addition one should keep in mind that goats are natural browsers. Allowing animals to utilize browse areas reduces parasite transmission because the forage is further from the ground.

Nutrition

Nutrition is an important aspect of herd health that can help promote resistance to parasites. Appropriate levels of protein in the diet may be one of the most important factors because it helps promote good immune function. It is also thought that phosphorus may help protect animals from the negative effects of parasites as parasites pull phosphorus from the host. Other minerals that are important to immune function and thus may help promote resistance to parasitism include selenium and copper. The best means to ensure proper mineral intake is to offer a free-choice, loose trace mineral salt. However for sheep, it is important to remember to use trace minerals specifically formulated for sheep to help prevent copper toxicity.

Quarantine

There are only two ways that resistant parasites are acquired – they are either home-grown or they are purchased. Thus, management practices must also include a quarantine program that will prevent animals harboring resistant parasites from entering the farm. New additions should be isolated from the rest of the herd and placed on a dry lot without any access to forage. These new additions should then be dewormed. Fourteen days after deworming, a fecal egg count or fecal floatation should be performed. The animal should only be allowed in herd if it is negative. Any parasites that remain after the deworming are resistant to at least one of the anthelmintics administered and should not enter the herd. This new addition to the herd can then be turned out on “contaminated” pasture so that any remaining resistant parasites will be overwhelmed by the local population of parasites.

How to deworm: A new way of thinking

As the effectiveness of the dewormer decreases, it provides less and less benefit, and once it falls to <50%, it is no longer useful as a sole treatment. Despite previous recommendations, rotating between dewormers will not prevent resistance from worsening. Therefore, it is no longer recommended. Instead, it is now recommended to use dewormers together at the same time in combination. There are two major benefits to using drugs in combination: 1) there is an additive effect with each dewormer used, thus the efficacy of the treatment increases with each additional dewormer given and 2) by achieving a higher efficacy, there are fewer resistant
parasites that survive the treatment. Therefore, there is a greater dilution of resistant parasites by the susceptible portion of the population. The more dewormers that are used in combination, the greater the efficacy of treatment will be. However, if all the dewormers individually have poor efficacy, the combination will not achieve a high level of efficacy. Once efficacy falls to 50%, even a combination of three dewormers will still fail to reach a 90% efficacy.

Before using combination deworming, there are a few precautions which include the following: 1) In New Zealand and Australia, products are sold that contain a combination of dewormers, so only one product needs to be administered. In contrast, in the USA, no dewormers are yet sold in this formulation, so the dewormers need to be bought and administered separately. This increases the cost as compared to the products available in these other countries. Additionally, the different groups of dewormers are not chemically compatible, thus they cannot be mixed together in the same syringe. Rather, they need to be administered separately, but can be given one immediately after the other; 2)

All dewormers should be administered at the full recommended dose whether administered singly or in combination; 3) When using dewormers in combination, meat and milk withdrawal times will be equal to the dewormer used with the longest withdrawal time period; 4) If using dewormers in combination, it is critical to maintain refugia; thus, one should be using a selective treatment approach based on FAMACHA© (see FAMACHA© section of the ACSRPC website for more information on this method and for further explanations of refugia). The presence of refugia is essential to realize the full benefits from combinations. In fact, if refugia are not maintained then you will not get the necessary dilution of the resistant survivors, and this will then lead to having multiple-resistant worms that can no longer be controlled with the combination treatment; 5) If the efficacy of your dewormers are >80%, it is possible you may not notice any difference in the clinical response of treatments when applied singly vs. in combination. However, the impact on the further development of resistance could be quite large; 6)

Any safety precautions that exist for a single dewormer will also exist when used in a combination; however, there are no known additional risks with using more than one dewormer at the same time.

Alternative methods and future approaches
Due to the growing and serious problem of parasite resistance to anthelmintics, alternative methods for parasite control are being considered. The ultimate goal for parasite control would be a vaccine to immunize animals against specific parasites. There are no vaccines currently in development, but this is a long-term goal. Another method includes the use of biological agents to destroy the parasite larvae. The nematode-trapping fungus, *Duddingtonia flagrans*, is a natural inhabitant of soil and has been used in this manner. Spores from this fungus are grown on grain and fed to the animals; the fungus passes unchanged in GI tract and concentrate in the feces. The feces are then deposited on the pasture where spores develop and trap the developing larvae within the hyphal loops. Currently this nematode-trapping fungus is not available for commercial use.

Some plants have the natural ability to suppress parasites in ruminants. *Sericia lespedeza* has shown the ability to reduce fecal egg counts and decrease the percentage of ova in feces that develop into infective larvae. This is a tall plant that animals browse, thus keeping their heads off of the ground and away from shorter forages where larvae can migrate up the plant. This forage contains condensed tannins which are thought to be the active compound. Condensed tannins can also be found in other plants such as *Lotus corniculatum* and Birdsfoot trefoil. Plants that are high in tannins may also be unpalatable to some animals due to bitterness. However, some of the plants, particularly *Sericia lespedeza*, are being used with some success.

Copper oxide wire particles are very potent killers of *H. contortus*. Animals receive capsules filled with copper oxide wire particles where they remain in the stomach and release copper over time. However, one should use caution when using copper oxide wire particles in sheep due to the possibility of copper toxicity. Currently there is no product that is available specifically for small ruminants which makes dosing difficult. Therefore, one should use caution when using copper oxide wire particles.

Another alternative that has received much attention is diatomaceous earth. Diatomaceous earth is composed of glass skeletons of diatoms and is thought to work by lacerating the cuticle of the parasite. However, no reports have described any efficacy with the use of diatomaceous earth.

References available upon request.
25 Big Digital Marketing Hacks
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If there is one thing that I have learned in the past 22 years of working with veterinary professionals, it is that you love education. So much so, that when you go to meetings, you are often such a sponge that by the time you are heading back home, your brains are overflowing with information. Often times this can have a crippling impact on the awaiting staff. With this in mind, I try to design my lectures with easy to follow and digest information that leaves the attendee with actionable items that can be applied immediately upon returning to the practice. Usually, this involves 3-5 techniques or concepts that are developed over 10-15 minutes of time. This talk will be a bit different. While equally digestible, we will spend about 2 minutes on each of the following 25 aspects of digital marketing to reveal why the particular service or tool is important, a reasonable result if used, and a “hack” for each that will help you be most successful. Get ready for fast paced action!

The top 25

1. Custom web design
2. Responsive web design
3. Copy that effects both user and search engines
4. Calls to action
5. Value propositions
6. Trackable conversion points
7. Mobile marketing
8. Practice app development
9. Automated reminders
10. Push notifications of reminders through a practice app
11. Texting your clients
12. Automation of review requests
13. Management of review content
14. Local Search
15. Neighborhood Search
16. iBeacon
17. Pet Activity Tracking
18. Automated surveys
19. APT
20. Customer Loyalty
21. Pay Per Click
22. Retargeting
23. Information Grabbing/Lists
24. Sales funnels
25. Video
Monitoring Digital Marketing:  
How to Check Your ROI

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You cannot manage what you do not measure
We’ve all heard the adage, but how many of us actually practice the principles of tracking and managing the numbers that should drive our efforts. My experience says not many. This session will explore how successful practices leverage the digital metrics available to manage their online marketing efforts.

Set reasonable expectations
Being reasonable about your goals and expectations could, in fact, be one of the most important aspects of setting up a digital marketing program. All too often practices set upon a marketing journey with lofty expectations that are impossible to track or achieve. Practice employees jump on the program band wagon but are left feeling as if they failed when the program does not meet the expectations that were set and eventually the practice abandons the marketing medium because it “does not work.” Try communicating with industry thought leaders about their experience in the space, reach out to peers to determine their experience, and perhaps begin a very small program with the goal of actually learning what your practice can expect from the medium. Then, with the experience in hand, future program development can be planned as you are familiar with the results and can develop reasonable (responsible) expectations.

Understand what the goal is before you develop any marketing medium
Perhaps the first step in developing the “goal” for any marketing medium is to understand a bit about who you are. Because many of your goals will (or should be) financial based, it would be helpful if you had a good understanding of your practice profit margins. It would be great if you had the time and resources to dive deep into your financials and your marketing efforts to try to track the impact that every marketing dollar has on each service’s bottom line, but that is probably not reasonable (see above). Basic information about your current gross revenue, profitability, target profitability, and minimum profit margins would be very important here as these numbers become the starting point and ultimately the foundation for a well-managed marketing plan.

How would it be possible for a practice to set a goal of increasing the profitability of a service, raising weekly gross revenue numbers, or development of a special discount/loyalty program (although I deeply despise discounting) without understanding the actual financials. If you do not know these numbers, do not be afraid, you are not alone. I frequently find myself in conversations with veterinary professionals that are in the same space. Ask your accountant for some help here.

Great goal examples are:
- Increase the profitability of a service
- Increase the frequency you are performing a service
- Increase the number of new clients
- Decrease the number of clients who lapse more than 13 months
- Increase the frequency of client visits
- Decrease the number of “open” appointment slots
- Increase the average client transaction
- Increase monthly gross revenue

What is analytics?
Analytics are bits of information that tell the story of how your digital marketing efforts are actually reaching their intended target. When used properly, they are one of the driving forces behind the success of the digital space as now we can track the performance of our efforts much more efficiently. Back in the “old days” we had to rely upon surveys and general data to demonstrate how well our marketing or advertising efforts were performing. Those days, for the most part, are gone.

The most common form of analytics are called “Google Analytics”. This is a free service available through Google, installed within the code of your website, and is used to monitor/report information about your web traffic. In most cases, your practice will need to reach out to your web development folks and have them assist with the installation of analytics. Most website projects should already have analytics installed, in fact, if you do not…you should be concerned as analytics provide the important information necessary to determine the success of the project.
A word of caution
While the code for tracking and ultimately the information developed through the analytics code is free, you should expect that a web marketing firm will charge you (or build into your monthly service fee) for a professional breakdown of your website analytics. Look at analytics much like any blood test you would run on a patient. The data from the test means much less without your professional opinion on how to proceed with that information in hand.

Examples of content that comes in analytics
Over the years I have seen thousands of reports that have been developed using Google analytics information. While a practice can get lost in the data and “push back” because of head spins, it will help if you remember the goals that were set at the beginning of the program. Analytics allows you to look at how your website performs, how users interact with the site, and ultimately how well the website “converts”.

Conversion is the act of causing a web user to perform the desired task. Good examples of conversions are requesting an appointment, clicking to call the practice from a mobile device, filling out a form, watching a video, or submitting their contact information. So, if your marketing goals were to increase the number of calls to the practice through the website, you could determine the baseline for calls after a month of performance, then monitor that metric over time. Adjustments could be made regarding the call to action, color of the “call” button, positioning of the “call” button, meta description used to describe the web page in the search engine results, and overall page layout so that optimum results are achieved.

During the lecture we will take a much deeper look at an analytics report and will explain what many of the data points mean, but to give a sneak peek at what analytics can deliver, you should know that you can determine how many people visit your site and leave immediately, how many people browse many pages of your site, where they leave, how long they stay on your site, how many people visit via desktop, how many visit on their mobile devices (even phone model numbers are available), which pages receive the most (and least) traffic, how many people click on a particular button, which keywords are most beneficial, and much more information than the format of these notes will allow.

Value of automating reminders
Thus far we have only discussed the analytic information that can be developed through Google in relation to your practice website. Please understand that additional information is available through other services or products used in the course of your practice operation.

Take, for instance, automated reminder services. Many successful practices recognize the value of automatically extracting information from their practice management software and sending text, email, or even postcard reminders for upcoming appointments, services, and those that are overdue. But few reminder services and/or practices take the information to the “next level” by sorting the clients by the average amount spent on the pet to be reminded, tracking revenue generated by reminded clients on a weekly basis, understanding the impact a missed appointment has on your bottom line, realizing how many pets are overdue and the amount of dollars that are potentially “missing” from your revenue, the dollars gained by reactivating lapsed clients by connecting the reminder with real revenue for that particular client, and much more!

We will take a deeper dive into this topic during the talk, but the key take away here is to look for opportunities for your practice to utilize the services and processes you already have in place to marry your marketing and communications efforts with actual invoices.

How to use data from analytical data
As I mentioned earlier, simply having the data is not enough. At very least you must understand what each of the metrics mean and at best, you have identified the metrics that are most beneficial to the growth of your practice and are using those metrics to guide you through future marketing efforts. With this information in hand you can expand your efforts into new and perhaps more beneficial mediums.

For example, through your website analytics you might find that most of your “organic” website traffic comes from a certain group of key words or phrases. This information can be used to develop a more robust pay-per-click campaign which will provide more data for you to track and segment. By looking deeper at the pages which get the most traffic, you might see who gave their information to you on that page. Then, by comparing and tracking reminders and client reactivation revenue, you can begin to assign values to the actual keywords and phrases used to promote your practice.

The bottom line here is that the digital space provides a huge amount of data that, if collected and understood, can provide practices like yours an extremely valuable tool for practice growth!
SEO Basics:  
How to Convert “Prospects” into “Clients”  
Bill Schroeder  
InTouch Practice Communications  
Schererville, IN

You need SEO  
Most practices understand that in order to be successful they need a solid marketing plan. However, very few practices “get” that this plan must include search engine optimization. Search engine optimization is the art of properly developing, maintaining, and positioning content and code on your practice's website and throughout the internet so that when pet owners are searching for services like yours, they are provided your website as a resource. This talk will walk you through the basics of search engine optimization and begin to discuss how to convert clients from those who have searched for services like yours, into longtime clients.

Custom designs allow you to stand out  
First, it must be mentioned that your practice's website should be custom. This is extremely important as the average pet owner does not know the difference between good or bad veterinary medicine and we will be looking for your personality when browsing the internet. Besides why would you utilize a template that many other practices could use when developing your most valuable marketing tool? Also when speaking about your practice website and its development, you should pay great attention to make certain that your website passes the Google+ mobile friendly test. During the lecture we will discuss how to evaluate your website and how to run your side through google's tool. This is extremely important as Google has been very forward about the fact that websites that are not mobile friendly will not be considered for search.

Content is still king  
With that said, we should now focus on the value of content. Since Google's spiders are only capable of reading code and content, it is extremely important that you spend time creating very high value content. By “high value” I am mean content that has been created to attract the search engines and is extremely inviting to those website users who visit your page. Care should be taken to develop a list of keywords and phrases that are most often used by those in your community searching for services like yours. Often times practices engage the services of SEO certified copywriters who are professionally trained to create the content that will best position there practice's website. During the lecture we will take a deeper dive into content by looking at a few websites from the perspective of a Google spider. This exercise will highlight the importance of keywords and the appropriate positioning of those keywords on a practice website.

Calls to action, value propositions, and conversion points  
When developing your website content you should focus on creating great calls to action, value propositions, and trackable conversion points. A call to action is simply a statement that asks a web user to perform a desired action. Great examples of calls to action are call the practice now, book your appointment today, register for our newsletter, and learn more about dentistry. Value propositions are the give and take of the transaction. They are the “if you do this”, “we will give you that”. Examples of value propositions are "call the practice now, and receive VIP priority treatment” and "Book an appointment today and receive 10% off your next boarding stay". When created properly, these calls to action and value propositions are married with clickable and trackable buttons that become conversion points.

If, while laying out your website and the content which will populate your site, you take the time and ask yourself exactly what you would like the user to do on that page, you very well could determine your call to action, value proposition, and trackable conversion point. By properly installing google analytics on your website, you should be able to monitor the web user traffic and determine how effective your call to action, value proposition, and trackable conversion points are by reviewing reports that show how many people actually visited that page and ultimately clicked on that button. If done properly, this data should be the most valuable analytics and SEO report information. Too many practices spend time focusing only on keywords and keyword positioning. In the end, it is quite possible that you can have great keyword ranking without any conversions. This would mean that you have spent a tremendous amount of effort getting your website to be seen but once pet owners arrive, they are not performing the desired action. The bottom line here is that keyword rankings do not necessarily equal an increase in new pet owners or revenue.

During the lecture we will examine some great calls to action and effective placement of keywords and key phrases so they are attractive to both the search engines and pet owners. We will also take a deeper dive into the back side of a website so that you can return to your practice and determine if effective SEO techniques are being applied.
**Sales funnels**

Once you have attracted the attention of local pet owners it is important that your website is designed to route them through the journey of converting from someone who is interested in services like yours to becoming a client who will stay with you for many years. This process is most often referred to as a sales funnel. A sales funnel is wide at the top and narrow at the bottom much like a traditional kitchen funnel. The reason it is wide at the top is that the message being delivered at that level, or at the entry point of your website, is acceptable for all who would land on your site. As the pet owner's journey throughout the site becomes more specific, the funnel narrows as the message is only appropriate to a select group of people. By the time the prospective customer reaches the bottom of the funnel, they are receiving extremely specific information that is geared towards asking them to convert. During the lecture we will take a look at a few well-developed funnels and discuss how a prospective customer may walk through the funnel and what information is most appropriate at the different stages.

It is impossible for me to deliver a paper or lecture about search engine optimization without discussing the two sides of positioning your website to be found by those who are utilizing search engines like Google. These two sides are "organic" and "paid" search.

**Organic search**

This type of search happens most naturally. It involves creating great content, developing effective website code, using effective design strategies, positioning well written content in valuable on-site and off-site places, and utilizing keyword strategies that attract users through the search engine results pages and retain those who ultimately land on your site. Organic search does not involve a payment to a search engine like Google but may require a payment to a professional search engine optimization firm that is experienced at managing projects like yours. This process can often be very cost-effective but will more than likely take longer for results to be obvious when compared to paid search.

**Paid search**

Paid search or pay per click, has a much more immediate effect. This strategy involves utilizing great keyword and key phrase research along with a keen understanding of the demographics of those who may be looking for services like yours. Paid search results appear in the search engine rankings at the top and bottom of the page. Practices that use this technique are most often interested in immediate search results and are very often competing with very well established websites that rank for the desired keywords.

A word of caution: paid search can be a very slippery slope as it is quite easy to mistakenly create ads in a fashion that results in the ad being displayed to and ultimately clicked on by those who are outside of your target market. I strongly suggest that if you are interested in a paid search campaign, that you seek the guidance or services of a professional pay per click agency.

During the session we will take a deeper dive and examine the most appropriate instances where organic or paid search can be applied.
People like stories
Recently, a trend has developed within the small business marketing world that has cast a light on a technique long used by very sophisticated brands. This technique, known as storytelling, appeals to a consumer's desire to be most connected with a brand or the people that operate within a business.

Why is this important?
In the veterinary world it is easy for us to understand that the average pet owner does not know the difference between good and bad veterinary medicine. People can most easily understand emotions and feelings created by familiar situations. As a result, my experience says that pet owners respond very well to emotional-based messages that are authentically created by the people and personalities that have created and support the veterinary practice.

Storytelling is an opportunity for practices to demonstrate who they are, the experience a current client or prospective client can expect when utilizing the services of your practice, and provides a very unique canvas for practices to relate a message and provide clearer understanding of a need for desired action.

Figure out which stories should be told
Before you start crafting your story, it is important that your practice understands what types of stories need to be told. To do this, I suggest you refer to your practices marketing plan. Identify the products, services, or themes that you would like to share with your current and prospective clients, and make a list of these instances. Then, conduct some research within your client and employee base by sending out surveys and having live conversations with those who are important to your practice about the things that are important to them. From this research you should develop a list of qualities and components of your practice that are most popular. Listen for common keywords like compassion, care, customer service, attention to detail, and other distinguishable and brand worthy attributes that should become the theme of many of your stories. Then, with this information in hand, you should call upon your life experience and the experiences of those who are close to you to frame up stories that are easy to understand and demonstrate the topics discovered during your research.

Be authentic
It should be noted that the term "stories" often suggests that the message may have a fairytale or fabricated component. While some businesses and professional storytellers have been successful utilizing a bit of elaboration or fiction in their stories in order to demonstrate a point, I suggest that your stories be as authentic as possible. By doing so, they will be easy for you to tell and will parallel the natural experience your clients have when interacting with you or your staff.

Allow your clients to experience you
We had a Schroeder family St. Patrick's Day tradition that involved our family dressing up in our finest Irish wear and making an annual visit to our local McDonald's for a Shamrock shake treat. My children looked forward to it as it often became a conversation that began months before St. Patrick's Day. Several years ago, my youngest daughter Lilly was in the third grade. On that particular St. Patrick's Day we, as usual, visited our local McDonald's for dessert. Immediately upon entering the restaurant Lilly noticed her third-grade teacher was sitting with her family in the corner doing the exact same thing our family had planned. As soon as Lilly recognized Mrs. Carr, her focus shifted from getting that Shamrock shake to wanting to interact with her teacher and her teacher's family. Reluctantly, I allowed Lilly to visit her teacher’s table but instructed her to return immediately to our table so that her teacher could enjoy some privacy. After Lilly did so, we began enjoying our family tradition but as our McDonald's visit continued I found myself watching Lilly and realized that she was no longer interested in the Shamrock shake but was, in fact, completely amazed by the fact that her teacher was at McDonald's.

Lilly’s amazement caused me to contemplate the situation for a great deal of time before I realized why Lilly was so amazed. It turns out that Lilly never was able to picture Mrs. Carr outside of the classroom. The fact is that when Lilly arrived at school each day Mrs. Carr was standing at the door waiting to greet her. Then at the end of the day, Mrs. Carr waved goodbye as Lilly boarded the bus. As far as Lilly was concerned, Mrs. Carr curled up in a ball and slept in the corner of the classroom. In Lilly’s mind Mrs. Carr did not have a life outside of the third-grade classroom. After our McDonald's encounter, Lilly began to look at Mrs. Carr as a "real" person.
I suggest to you that many of your clients are unable to realize that you have a life outside of your practice. You are Mrs. Carr. Storytelling can be an excellent way for you to demonstrate the fact that you have a life outside the practice. You are dealing with life issues just like your current and prospective clients. You are a pet owner and are often faced with the same decisions they are facing. By doing so, you will experience increased compliance, decreased missed appointments, a higher level of interaction, and an overall increased amount of respect paid to the time you share with them during your appointments.

How you can use storytelling
Storytelling does not have to be literal. I am not suggesting that you create an environment whereby your employees and clients are sitting cross legged at your feet hanging on your every word. But rather, I suggest you come up with easy to understand and transfer bits of information that help clients and employees understand the subject matter at hand. This technique can be used during your appointments, when creating copy for your website, during daily social media posts, writing blogs, and when developing videos that demonstrate your practice’s offering.

Meet Kindra Hall
As a part of my introduction to and continued study of storytelling, I have followed a thought leader within the space named Kindra Hall. If you are interested in learning more about storytelling and applying it to your practice, I highly recommend you find her website and begin following her on social media. Recently, I attended a handful of Kindra's lectures. She mentioned a project that stopped me in my tracks and forever validated storytelling and the value of sharing story-like information.

Not so worthless objects
This study is known as the "significant objects" project and can be found online at significantobjects.com. In order to demonstrate the value of storytelling and the ultimate impact it can have on a consumer, a group of writers visited garage sales, thrift stores, and other similar so that they could purchase items that are both random and worthless. Examples of these items included a piece of wire, and old salt shaker, a ceramic apple, and a one eyed stuffed animal. With these items in hand the group of writers created fictional stories about the origin of each item. Most times, these stories demonstrated the owner's emotional bond to the item.

These items were then posted on auction sites like eBay and sold for values that often paralleled 100 times their purchase price. Items that were purchased for under $.50 were being sold for more than $50. This demonstrated the fact that the purchaser was more interested in relating to and owning the story then they were the random, otherwise worthless item.

We will practice storytelling
During the lecture we will spend time telling some stories. I will demonstrate how revealing portions of my personal life can help you better understand who I am and why you should pay attention to my message. Then, we will take a deeper dive into some of the situations whereby you can apply storytelling in your practice’s big picture plan and day to day routine.
Disorders of the upper airway occur commonly in brachycephalic breeds of dogs. Chief client complaints include excessive respiratory noise, reduced exercise tolerance, heat intolerance, and dyspnea. Cyanosis also may be observed. Since multiple airway abnormalities may occur in the same dog, a systematic and thorough approach to patient evaluation is essential for proper management. Brachycephalic breeds with upper airway disorders present both anesthetic and surgical challenges to the clinician. Diagnosis and management of the following upper airway disorders are reviewed: stenotic nares, elongated soft palate, everted laryngeal saccules (laryngeal collapse), and hypoplastic trachea.

**Patient evaluation**

Evaluation of the patient with upper airway disorders should include a thorough history, physical examination, radiographic examination, and pharyngoscopic, laryngoscopic and tracheoscopic evaluation in the anesthetized patient. Determine the frequency, severity, and pattern of occurrence of dyspnea and excessive noise when obtaining the history from the client. Note the occurrence of cyanotic episodes, as this may indicate the presence of more severe or multiple abnormalities.

Physical examination focuses on the cardiopulmonary systems, yet does not exclude other body systems. Inspect the external nares looking for axial deviation of the dorsolateral nasal cartilage, and evaluate function of the nares. Observing the dog at rest and breathing with a closed mouth will help determine if air is moved effectively through the nose. Placing a clean microscope slide in front of the nares with the animal breathing through the nose will also provide an estimate of air flow through each nostril. Auscultate the thorax and upper airway. Lateral cervical radiography emphasizing soft tissue detail may help delineate an elongated soft palate. Perform pharyngoscopy, laryngoscopy, and tracheoscopy in the anesthetized patient, being prepared to proceed with surgery after the evaluation.

**Pre-surgical management**

Anesthesia in the brachycephalic breeds requires diligent pre-anesthetic preparation and attentiveness to detail during and after anesthesia. Points of emphasis include avoiding regurgitation or vomition, providing pre-anesthetic oxygenation, rapidly inducing anesthesia, gaining rapid control of the airway, and administering a single dose of corticosteroid preoperatively.

Regurgitation and vomiting is observed relatively commonly following general anesthesia administration in dogs undergoing upper respiratory surgery. Regurgitation poses two risks to the patient: aspiration pneumonia and reflux esophagitis. Post-anesthetic regurgitation may be minimized by withholding food from the dog for 18 to 24 hours prior to anesthesia. Water is usually withheld for 4 to 6 hours before surgery, also. The use of pre-anesthetic metaclopramide (0.2-0.4 mg/kg, SQ) may have beneficial effects on reducing the incidence of postoperative vomiting and regurgitation.

Use of a corticosteroid (e.g., dexamethasone, 0.1-0.2 mg/kg, IV or SQ) immediately prior to pharyngeal or laryngeal surgery in the brachycephalic dog may reduce postoperative swelling. The author embraces such practice, as it seems to positively impact ventilation in the immediate postoperative period.

**Anesthesia**

Oxygen (5 L/minute) should be administered via face mask or rebreathing hose for at least 5 minutes immediately prior to anesthetic induction to dogs that do not resist its delivery. Anesthesia should be rapidly induced with an injectable agent (e.g., propofol), to enable rapid and atraumatic endotracheal tube placement. Brachycephalic breeds are not good candidates for the use of inhalation anesthetics as induction agents, because they can develop significant difficulties before the endotracheal tube is placed. Employ adequate laryngoscopic viewing to efficiently accomplish endotracheal intubation. Choice of endotracheal tube size (diameter and length) should reflect the diameter of the trachea and the length of cervical region. Avoid excessively large or long tubes. When properly placed, the endotracheal tube should extend from the tip of the nose to the thoracic inlet and be slightly smaller than the tracheal diameter. After minimally inflating the endotracheal tube cuff, secure the tube with a section of roll gauze tied behind the animal's head. Maintain endotracheal intubation for as long as possible during recovery from anesthesia.

**Diagnosis**

**Stenotic nares**

The nose contributes 80% of the total airway resistance during inspiration. Diagnosis of stenotic nares is made by physical examination. Reduced air flow through the stenotic nostrils is noted when the dog is closed mouth breathing. Reluctance to breathe through the nose may also be noted. The wing of the nostril may be mildly, moderately, or severely deviated medially. Affected dogs often are restless and anxious, especially when restrained.
Elongated soft palate
An elongated soft palate usually produces signs of excessive respiratory noise, particularly when the animal is sleeping. The redundant soft tissue in the pharynx projects into the larynx and causes stridor in symptomatic dogs. Make a definitive diagnosis of elongated soft palate during pharyngoscopic evaluation. An elongated soft palate extends more than 3 mm caudal to the tip of the epiglottis. The elongated soft palate often is thickened and has an inflamed tip.

Everted laryngeal saccules (Stage I laryngeal collapse)
Everted laryngeal saccules are a secondary disorder of the upper respiratory tract. Everted saccules produce excessive noise, primarily on inspiration and possibly dyspnea. This abnormality seems to develop due to the generation of strong negative intra-laryngeal pressures during inspiration. Everted laryngeal saccules may accompany any or all of the other brachycephalic upper respiratory disorders. History is often not particularly helpful in presumptively diagnosing everted laryngeal saccules, although recent evidence of exercise or heat intolerance may be noted. Laryngoscopic examination in the anesthetized patient reveals redundant mucosa just rostral to the vocal folds near the floor of the larynx. Everted saccules are usually white and glistening and obscure the vocal folds. Their prominence may vary with the degree of inspiratory effort. The laryngeal opening is smaller, particularly its ventral aspect.

Hypoplastic trachea
Hypoplastic trachea is observed fairly commonly in Bulldogs and some other brachycephalic breeds. If the trachea is severely hypoplastic, the animal's respiratory function may be almost continuously compromised. Diagnosis is made following lateral cervicothoracic radiographs and/or tracheoscopy.

Surgery
Stenotic nares
Surgical repair of stenotic nares involves the excision of a portion of the lateral alar fold and approximation of the adjacent tissues. A vertical (or elliptical) wedge of tissue is excised from each lateral alar fold. Hemorrhage is controlled with local pressure and reapposing the wound edges. Simple interrupted or mattress sutures (e.g., 3-0 poliglecaprone 25) are placed to enlarge the size of the nostril.

Elongated soft palate
Repair an elongated soft palate via an oral approach with the intubated patient positioned in sternal recumbency. A properly placed mouth gag and suspension of the patient's upper jaw assist the surgery. A soft palate is considered to be elongated if its free end extends beyond the caudal border of the palatine tonsils. Alternately, the end of the soft palate should not interfere with the movement of the epiglottis. Excise the redundant portion of the soft palate after placing stay sutures in its lateral edges near the proposed level of resection. These stay sutures help minimize trauma to the tissues and guide the line of excision. Use Metzenbaum scissors to incise part of the soft palate starting laterally and proceeding medially. After incising about one-half of the width of the soft palate, begin placing sutures in the incised edge of the palate to appose oral and nasal epithelial surfaces of the cut edge of the soft palate. A synthetic absorbable suture (e.g., 4-0 poliglecaprone 25) is placed using a simple continuous pattern to achieve mucosal apposition. Incise the remainder of the width of the soft palate, discarding the redundant portion of the palate, and complete the closure. Verify accuracy of excision prior to the removal of the stay sutures and mouth gag.

Everted laryngeal saccules
Excise everted laryngeal saccules through an oral approach. Use a mouth gag and properly position the patient to assist the surgery. The author prefers to maintain tracheal intubation, provided that the dorsally-deviated endotracheal tube permits adequate visibility and access to the ventral larynx. Long handled instruments (Debakey tissue forceps and fine-tipped Metzenbaum scissors) aid the procedure. Temporary removal of the endotracheal tube or tracheostomy endotracheation may aid visibility and surgical efficiency. Grasp the everted mucosa with Debakey tissue forceps, excise the redundant mucosa as near its base as possible using Metzenbaum scissors, and discard the excised tissue. Inspect for any evidence of remnants of the saccule; remove such remnants in a similar fashion. Hemorrhage is usually minimal.

Hypoplastic trachea
There is no clinically tested surgical treatment for hypoplastic trachea. Owners should be notified that respiratory signs may persist in patients with severely hypoplastic trachea, even following successful repair of other disorders. The veterinary clinician should also be prepared to modify endotracheal tube selection in those anesthesia patients with hypoplastic trachea.

Postoperative considerations
Brachycephalic patients require close observation during their post-anesthetic recovery. Recovery should be as smooth and controlled as possible. Preoperative tranquilization (e.g., 0.1 mg/kg acepromazine, IV) may help prolong the recovery process. Delay endotracheal tube removal as long as possible. A temporary tracheostomy may be necessary in selected patients, if pharyngeal or laryngeal swelling is experienced. Offer water a few hours after the patient is fully awake. Delay offering food until the next day.
Summary
The anesthetic and surgical management of the brachycephalic dog with upper airway disorder(s) require proper planning and diligence to be successful. Such planning and attentiveness to detail should include complete patient evaluation, proper selection and use of perianesthetic drugs, efficient, atraumatic surgery, and close observation during the anesthetic recovery period.

References
Pre-surgical considerations
Traumatic diaphragmatic hernia should be suspected in any animal with known or suspected trauma, recent or not, that is presented with dyspnea. Pathologic effects of the diaphragmatic hernia are due to negative impact on cardiorespiratory dynamics and on herniated organs themselves. Physical examination of a patient with traumatic diaphragmatic hernia of recent origin often reveals signs of shock. Treatment of shock, including use of intravenous fluids is generally the first therapeutic intervention. Thoracic auscultation of the animal with traumatic diaphragmatic hernia often reveals abnormal breath sounds and possibly other sounds (e.g., borborygmus) over at least part of the thorax. Heart sounds may be either louder or more muffled than normal, depending on the position of the heart and the presence of tissue interposed between the heart and ribs. Thoracic percussion may also reveal areas of reduced resonance. Abdominal palpation may accentuate the animal's dyspnea and reveal an ‘empty’ abdomen. Elevation of the animal's forequarters may result in improved ventilation due to shifting of abdominal viscera back into the abdomen.

Radiology or ultrasonography (particularly in patients with pleural effusion) often reveals loss of a distinct diaphragmatic line, increased pleural density, and displacement of abdominal viscera. The single-most useful view for diagnosis of diaphragmatic hernia is the lateral projection. Gas within the displaced viscera may be noted. Pleural effusion (blood with recent trauma or a modified transudate with a more long-standing hernia) may also be noted. Pulmonary contusion occurs commonly in patients with recent trauma.

Pre-surgical considerations for the patient with traumatic diaphragmatic hernia include blood volume replacement and support, ventilatory support, possible antimicrobial use, and close observation. Repair of a traumatic diaphragmatic hernia is undertaken as soon as the patient has been adequately stabilized after the initial injury. The author’s target is 24 to 48 hours after injury, unless herniation of the stomach is acutely negatively affecting ventilation. Diaphragmatic hernia has a higher priority than definitive fracture repair. Intravenous fluid replacement may not be as aggressive as in other shock patients because of the risk of pulmonary edema. Volume of fluid replacement is guided by cardiovascular parameters (e.g., capillary refill time, pulse quality, mucous membrane color, and central venous pressure) and respiratory parameters (e.g., ventilatory rate and effort, auscultatory findings, and pulse oximetry). Ventilatory support, including housing the animal in a high oxygen environment and positioning the patient with the forequarters elevated is important prior to surgical repair. Removal of pleural effusion may also improve ventilation in selected cases. Antimicrobials may be used perioperatively to prevent infection-related pulmonary problems. Every patient with traumatic diaphragmatic hernia needs close observation, since rapid changes in ventilatory function may occur. Patients not responding to pre-surgical management or experiencing deterioration despite appropriate management may be surgical candidates within 36 hours of injury.

Anesthetic considerations
Anesthetizing the patient with traumatic diaphragmatic hernia often presents challenges. The reduction in ventilatory reserves caused by the trauma and the presence of abdominal viscera within the thorax makes controlling patient ventilation throughout the anesthetic episode both necessary and critical. Specifically, the pre-induction administration of oxygen is beneficial, provided the patient tolerates such administration without resistance. Rapid intravenous induction of anesthesia, maintenance of a patent airway through rapid, atraumatic endotracheal intubation, assisted (or controlled) ventilation, and appropriate patient positioning are indicated in the traumatic diaphragmatic hernia patient. Severe hypoventilation resulting from inattentiveness to patient positioning and reliance on spontaneous patient ventilation may occur shortly after anesthetic induction. Maintaining slight elevation of the patient's forequarters during surgical preparation and surgery is also helpful. Anesthetic induction using inhalation agents is not recommended because of the precarious ventilatory status of the patient.

Surgical management
After a standard preparation of the ventrum of the patient (extending from manubrium to the pubic pectin), drape the ventral abdomen to expose the midline. Create a midline skin incision from the xiphoid process to caudal to the umbilicus. Continue controlled patient ventilation during the surgical procedure, since communication between the pleural cavity and the atmosphere occurs on entry into the abdomen. Consider administration of an appropriate volume of intravenous fluids (e.g., 10 ml/kg/hr) and, particularly in long-standing hernia situations, an aqueous corticosteroid (e.g., 4 mg/kg dexamethasone or 30 mg/kg methylprednisolone) prior to the manipulation of abdominal viscera. Such pre-treatment may reduce the incidence of re-expansion pulmonary edema after surgery.

Incise the abdominal wall on the midline, and excise the fat-filled falciform ligament. Consider extending the abdominal wall incision paracostally along the affected side, particularly when repairing defects in the dorsal diaphragm. Visibility of the diaphragmatic defect is enhanced by placing a pediatric Balfour retractor over the towel-protected abdominal incision and using
malleable retractors to manipulate the abdominal viscera. Remove abdominal or pleural fluid by suction. Carefully retract the abdominal viscera from the thorax using gentle traction. Delay final, definitive positioning of the viscera until the diaphragmatic defect is closed. Inspect the lungs and pleural cavity after the herniated contents have been retrieved from the pleural cavity. Avoid immediate re-inflation of remaining atelectatic areas of the lung, as this may enhance the potential for re-expansion pulmonary edema. Thoroughly evaluate the entire diaphragm to determine the extent and number of rents present.

Close the diaphragm using a simple continuous pattern of synthetic absorbable suture material (e.g., 2-0 polydioxanone). Start at the least accessible part of the defect, taking care to avoid traumatizing or constricting the caudal vena cava, hepatic veins, aorta or esophagus. On closing the diaphragmatic defect, remove residual air from the pleural cavity either by thoracentesis performed through the diaphragm (e.g., 14 or 16 gauge over-the-needle catheter) or by placing a thoracostomy tube. Explore the abdomen, with particular attention given to the previously displaced tissues. Repair lesions as needed. Close the abdomen after definitive replacement of the abdominal viscera.

**Postoperative considerations**
Postoperative considerations include ventilatory support, analgesic administration, and close observation. Continue ventilatory support until the patient is adequately ventilating spontaneously. Take particular care when administering positive pressure ventilation during and after surgery, because trauma to the lungs from overzealous ventilation is a distinct possibility. Re-expansion pulmonary edema following re-oxygenation of chronically collapsed lungs is a major cause of perioperative death, particularly in animals with long-standing (chronic) diaphragmatic hernias. Reperfusion injury, with release of superoxide radicals that are not effectively scavenged, is thought to result in increased pulmonary capillary permeability and pulmonary edema. Assist spontaneous ventilation in the postoperative patient by maintaining the patient in a forequarters-elevated position and the appropriate use of analgesics. Nasal oxygen administration may also be beneficial.

Opioid analgesics smooth the immediate postoperative recovery period and assist ventilation by controlling pain. Close observation of the postoperative diaphragmatic repair patient is indicated to react appropriately to the patient's changing status. Offer small feedings on the first postoperative day. Limited patient activity is indicated at least until suture removal.

**Prognosis**
Approximately 15% of animals die before presentation for anesthesia and surgical correction. Induction is a critical phase of anesthesia. Inadequate ventilation is a potential cause of intraoperative death. Survival to discharge approximates 90% of cases in recent case series. Complications may be observed in up to 50% of patients, with cardiorespiratory issues seen soon after surgery and issues relating to herniated tissues (e.g., gastrointestinal perforation or obstruction) being more delayed.

**References and suggested reading**
Conditions of the external ear
The primary indications for external ear surgery in dogs are unresponsive or recurrent otitis externa (especially with otitis media), polyps or neoplasia of the external ear canal, and trauma and avulsion of the auricular and annular cartilages.

Otitis externa
Base the type of ear surgery to perform on the extent and severity of otic disease present as determined by otoscopic and radiologic (including computerized tomographic [CT]) examinations. Rupture of the tympanic membrane on otoscopic exam or evidence of otitis media on radiologic (CT) exam is usually treated with a total ear canal ablation with lateral bulla osteotomy (TECA with LBO). Palpation of the external ear canal determines the presence of calcification or proliferation of the otic cartilages. Calcification of otic cartilages is usually an indication for performing a TECA with LBO.

Owner’s expectations also may influence the decision as to which procedure to perform. Those clients seeking to avoid the continued need to treat their pet’s ears should be appraised of a TECA with LBO. Functional hearing loss after bilateral TECA with LBO may be complete; however, little change from the pet’s preoperative status may be noted.

Lateral ear resection
Lateral ear resection is the least invasive surgical procedure of the external ear canal; it has been used in dogs for over 40 years. It may be indicated in the patient with chronic non-proliferative otitis externa that has failed to respond adequately to appropriate medical management. Lateral ear resection may also be used to gain access for excisional biopsy of masses involving the vertical ear canal. The goal of lateral ear resection is to provide access to the horizontal ear canal. The medial wall of the vertical ear canal and the entire horizontal ear canal remain after lateral ear resection.

Following surgical preparation of both sides of the pinna, ear canal and lateral periotic region, drape the site to expose the entire pinna. Measure the level of the horizontal ear canal by inserting a forceps or probe into the vertical portion of the ear canal. Create a "U"-shaped skin incision so that its width just exceeds that of the vertical canal and its length extends approximately 2 cm below the junction of the vertical and horizontal ear canals. Dissect the skin flap and leave it attached dorsally to assist in the cartilage incisions. Bluntly and sharply dissect the subcutaneous tissue and the parotid salivary gland to expose the lateral wall of the vertical ear canal. Create parallel incisions in the lateral cartilage to the level of the horizontal canal using scissors. Start the rostral incision at the tragoheletic incisure, and start the caudal incision at the intertragic incisure. As most canine ear canals curve slightly rostrally, curve both cartilaginous incisions slightly rostrally to maximize exposure of the horizontal canal. Deflect the incised flap of vertical canal ventrally, trim it to the appropriate size, and suture the edges of the cartilaginous flap to the skin. Carefully appose the skin and ear canal epithelium with monofilament, nonabsorbable sutures (e.g., 3-0 polypropylene).

Vertical ear canal ablation
The vertical ear canal ablation also has the goal of providing access to the horizontal ear canal, but it results in excision of the entire vertical ear canal. Indications for performing a vertical ear canal ablation are similar to those for lateral ear resection, but also include proliferative (granulomatous) otitis externa involving only the vertical ear canal, neoplasia of the vertical ear canal only, and possibly traumatic separation of the auricular and annular cartilages. Because a vertical ear canal ablation results in removal of more diseased tissue compared to that removed by a lateral ear resection and potentially reduced postoperative pain compared to a lateral ear resection, a vertical ear canal ablation may be preferable to a lateral ear canal resection. The vertical ear canal ablation procedure seems to be technically easier to perform than a lateral ear resection, and a more precise opening to the horizontal canal is created more consistently.

Prepare and drape the affected ear as described for lateral ear resection. Create a vertical skin incision directly over the vertical ear canal to the level of the horizontal ear canal. Make a circular incision surrounding the opening of the ear canal and connect the vertical incision. Make this circular incision to include the proliferative tissue that surrounds the opening of the ear canal. Take care when incising the skin and cartilage of the anthelix so just the cartilage and not the deeper tissue is incised. Dissect the entire vertical ear canal from the surrounding tissue to the level of the horizontal canal using blunt and sharp dissection. The horizontal ear canal begins just proximal to the junction of the auricular and annular cartilages. Excise the vertical ear canal by scalpel, creating an opening into the horizontal ear canal. When incising the ventral aspect of the cartilage, create a small drain board, as apposing the horizontal canal cartilage and skin is aided and potential for stricture of the opening of the ear canal is reduced. Appose the epithelium of the horizontal ear canal as accurately as possible to the surrounding skin with monofilament, nonabsorbable sutures (e.g., 3-0 polypropylene). Close the dorsal aspect of the skin incision in the shape of a "T", using the same suture material.
Surgical correction of traumatic separation of the auricular and annular cartilages results in the creation of a horizontal opening in the horizontal ear canal, similar to that resulting from a vertical ear canal ablation. Locate the blind end of the horizontal ear canal (proximal end of the annular cartilage) by sharp and blunt dissection. Flush the secretion-filled horizontal ear canal, and appose the epithelium of the horizontal ear canal to the surrounding skin with sutures. The vertical ear canal, which is separated from the rest of the ear canal, may be left undisturbed or excised. The horizontal ear canal in cases of traumatic separation of the auricular and annular cartilages tends to be shorter than that following a standard vertical ear canal ablation. Thus, tension on the suture line between skin and otic epithelium tends to be greater, making postoperative stricture more likely.

**Total ear canal ablation with lateral bulla osteotomy**

Total ear canal ablation is the most invasive surgery of the external ear, yet it is most likely to be curative when combined with a lateral bulla osteotomy procedure. Indications for performing a TECA with LBO include proliferative otitis externa involving both the horizontal and vertical ear canals, most cases of periotic abscessation, severe, non-responsive otitis externa accompanied by otitis media, calcification of the otic cartilages (especially annular cartilage), and neoplasia of the horizontal ear canal. Total ear canal ablation results in excision of the entire external ear canal. The goals of this procedure are to surgically excise all diseased tissue (infected and/or neoplastic), including epithelium of the tympanic bulla, and to eliminate clinical signs associated with otitis externa and media.

Prepare and drape the affected ear as described for lateral ear resection. The skin incision and initial dissection are identical to those for the vertical ear canal ablation. Continue dissection around the entire horizontal ear canal until the junction of the annular cartilage and the skull is reached. Take great care when dissecting around the horizontal ear canal, particularly in the calcified ear canal, to avoid damaging the facial nerve. Positively identify and protect the facial nerve, which courses rostrally just ventral to the horizontal ear canal. Carefully excise the ear canal from the skull with scalpel blade, chisel, or rongeurs. Since otic epithelium continues into the bony ear canal for 5 to 10 mm before the tympanum is reached, excise all remaining otic epithelium. Use rongeurs, bone curette, and alligator forceps to remove all of the epithelial tissue. Use rongeurs to remove the ventrolateral wall of the osseous bulla and bony ear canal. Access to the middle ear cavity (tympanic cavity) enables flushing and gentle curettage of this area. This step is critical to a long-term successful outcome. Completely evacuate the tympanic cavity. Close the incision after administering a bupivacaine splash block.

Possible complications following TECA with LBO include temporary or permanent facial nerve injury (resulting in partial or complete facial paralysis), sympathetic nerve injury (resulting in Horner’s syndrome), hearing loss, and partial pinna necrosis.

**Results**

Results following otic surgery depend, largely, on proper patient selection, avoiding technical errors, identification and treating systemic disease(s), accurately assessing the extent of ear disease and conscientious postoperative management. Failure following lateral ear resection has been shown to be as high as 47%. Long-term results following vertical ear canal ablation may be more favorable than those for lateral ear resection. Results following total ear canal ablation without bulla osteotomy have been discouraging, with recurrence of periotic abscessation being a common complication. Results following total ear canal ablation with lateral bulla osteotomy are quite favorable.

**Conditions of the middle ear**

The primary indications for middle ear surgery in dogs include unresponsive otitis media (usually in concert with otitis externa) and removal of inflammatory polyps. Pain is the primary clinical sign associated with otitis media; however, head shaking, ear scratching, otic discharge, head tilt, vestibular signs, and Horner’s syndrome also may be observed.

Physical examination, including otoscopic evaluation and radiologic (including computerized tomographic [CT]) evaluation confirms the diagnosis. Otoscopically, attempt to visualize the tympanic membrane prior to and after irrigating the external ear canal. Exudate or masses within the external ear canal may obliterate the tympanic membrane. The tympanic membrane may be reddened, bulge into the ear canal, or be ruptured. Radiographs of the skull should be made to evaluate the tympanic bullae. Lateral, ventrodorsal, oblique, and open-mouth projections of the skull can be made. Radiographic views which provide helpful information are the oblique and open-mouth projections. Computerized tomographic evaluation of the tympanic bulla is particularly helpful with distinguishing fluid density from that caused by tissue.

The anatomy of the tympanic bullae in dogs includes a connection between the tympanic cavity and the pharynx by the auditory tube. The normal tympanic bullae appear as a thin shell of bone with an intraluminal air density. With disease and chronicity, the tympanic bullae become sclerotic and thickened, and the tympanic cavity may contain a fluid- or tissue-density. Sympathetic fibers in the middle ear are present on the dorsal aspect of the bulla; they are relatively unresponsive to surgical trauma in the dog.
Otitis media
Otitis media is a relatively common disease that may go unrecognized. It frequently accompanies chronic otitis externa. Otitis media may be diagnosed on otoscopic exam by seeing changes in the tympanic membrane. Such changes may include loss of integrity, change in color (becoming more opaque and grey), and change in shape (becoming more convex and bulging). Radiographic (especially CT) assessment of the tympanic bullae is quite helpful in diagnosing otitis media. Radiographs are more helpful in assessing duration of middle ear disease, as tympanic bullae become sclerotic with chronicity.

Inflammatory polyps
Inflammatory polyps are benign, pedunculated growths that may occur rarely in the middle ear or external ear canal of dogs. Site of origin is believed to be the mucosa of the tympanic cavity. Inflammatory polyps may extend from the tympanic cavity through the tympanic membrane into the external ear canal. Otitis media accompanies inflammatory polyps within the tympanic bulla, but it is unclear whether the infection initiated the polyp growth or is secondary to its presence. Clinical signs observed depend on the size and location of the polyp. Otic polyps result in signs of otitis externa and/or otitis media. Thoroughly evaluate the external ear canal otoscopically and by palpation.

Treatment
Treatment of otitis media often involves a combination of medical and surgical techniques. Accurate microbiologic assessment of the tympanic cavity is critical to success. If otitis media accompanies chronic otitis externa, a total ear canal ablation with lateral bulla osteotomy is usually indicated. If primary otitis media is present, a myringotomy may be indicated. Myringotomy may be considered both a diagnostic and a therapeutic procedure. Puncture the pars tensa portion of the tympanic membrane at the five or seven o’clock position using a spinal needle or a small Steinmann pin. Use a 3.5 F polypropylene catheter to aspirate fluid from the tympanic cavity for culture and susceptibility testing. Gently flush the tympanic cavity with warm saline until the escaping fluid is clear. Choose an appropriate systemic antimicrobial agent based on susceptibility testing results. Continue therapy for approximately 4 weeks.

Surgical treatment of otitis media usually involves performing a bulla osteotomy. The tympanic bulla may be approached surgically through two approaches: lateral or ventral. The lateral bulla osteotomy is usually reserved for cases with concurrent otitis externa that require a total ear canal ablation. Ventral bulla osteotomy is usually performed as a separate procedure. Ventral bulla osteotomy is more challenging in the dog than it is in the cat, but it provides better visualization and exposure of the ventral aspect of the tympanic cavity than does the lateral approach.

Position the paramedian skin incision midway between the angular process of the mandible and the level of the wings of the atlas. Dissect between the digastricus muscle (laterally) and the hypoglossal and styloglossal muscles (medially). Identify the tympanic bulla as a raised structure between the angular process of the mandible and jugular (paracondylar) process of the skull. Penetrate the tympanic bulla with a Steinmann pin (3/32 inch) and enlarge the opening with rongeurs. Obtain microbiologic and histologic samples from the tympanic cavity. Flush the tympanic cavity with warm saline solution, and carefully remove tissue and exudate by gentle curettage. Close the incision routinely.

Horner’s syndrome is an uncommon complication after ventral bulla osteotomy in dogs. Most signs of Horner’s syndrome will resolve within a few days postoperatively. Head tilt toward the affected side may persist postoperatively.

Summary
The middle ear of the dog can be accessed surgically via a lateral or ventral approach. The lateral approach is most often used in combination with total ear canal ablation, while the ventral approach is used as a stand-alone procedure. A ventral bulla osteotomy is more challenging to perform in the dog vs. the cat. Prognosis for resolution is better for inflammatory polyps than it is for neoplastic conditions. The prognosis for otitis media resolution depends, in part, on the duration of disease.

References and suggested reading
Surgery of the perineum of the dog is performed most frequently to remove tumors or to repair a perineal hernia. Perineal herniorrhaphy is the primary focus of this session.

**Perineal neoplasia**

Neoplasms of the canine perineum include anal sac tumors, including apocrine gland adenocarcinoma of the anal sac, tumors of the perineal skin, including mast cell tumors, and perianal gland tumors. Surgical excision may be challenging due, in part, to the proximity of the anus, rectum and structures essential for fecal continence (e.g., caudal rectal nerve and anal sphincter muscle).

**Perineal hernia**

Perineal hernia is a failure of the supporting structures of the pelvic outlet to contain the viscera of the abdominal cavity and pelvic canal. Successful perineal herniorrhaphy is dependent on knowledge of the anatomy of the perineum. Goals include achieving adequate exposure, avoiding injury to vital structures, maximizing potential for success, and minimizing postoperative complications. Options for repairing a perineal hernia include the traditional technique and the internal obturator muscle transposition technique. Additional techniques that may be utilized include colopexy, cystopexy, deferent duct fixation, and use of prosthetic implants, including porcine small-intestinal submucosa [SIS] or polypropylene mesh.

**Surgical anatomy**

The perineum covers the pelvic outlet and surrounds the openings to the anal and urogenital canals. The principal structure of the perineum is the pelvic diaphragm, consisting of the coccygeal and levator ani muscles. Structures of surgical importance in the perineum include levator ani, coccygeal, internal obturator, and external anal sphincter muscles, pudendal nerve and vessels, sacrotuberous ligament, sciatic nerve, and caudal gluteal vessels. The levator ani and coccygeus muscles form the lateral boundary of the rectum and the medial boundary of the pelvic diaphragm. The coccygeal muscle lies just lateral to the levator ani muscle. Sutures are placed in the levator ani and coccygeus muscles when repairing a perineal hernia. The internal obturator muscle covers the obturator foramen and ischial table and forms the ventral aspect of the pelvic diaphragm. The pudendal nerve and vessels course caudomedially across the dorsal surface of the internal obturator muscle. The pudendal nerve supplies the caudal rectal nerve, the sole motor supply to the external anal sphincter muscle. The sacrotuberous ligament extends from the lateral sacrum to the lateral angle of the ischiatic tuberosity. Suture incorporation of the sacrotuberous ligament may be used in either the conventional repair or the internal obturator muscle transposition technique. This ligament is also a surgical landmark for the location of the sciatic nerve, which lies just ventrolateral to it. The caudal gluteal vessels may be traumatized during either the conventional or internal obturator muscle transposition technique, particularly when placing sutures in the sacrotuberous ligament or transecting the tendon of the internal obturator muscle.

**Clinical presentation**

The typical presentation for dogs with perineal hernia is a middle-aged to older male dog (intact or neutered) with a unilateral or bilateral perineal swelling. Perineal hernia is much less commonly observed in female dogs or cats. With unilateral herniation, the contralateral pelvic diaphragm will often feel weaker than normal on rectal palpation. Hernial contents may include any of the following: serosanginous fluid, fat, rectal wall, prostate, urinary bladder, and, rarely, small intestine. Rectal abnormalities associated with perineal hernia include rectal deviation (flexure), rectal dilatation (sacculation), or rectal diverticulation. Definitive repair of the rectal abnormality at the time of perineal herniorrhaphy may be necessary in cases of diverticulation and excessive dilatation.

**Preoperative preparation**

Avoid use of enemas in the immediate preoperative period, as intraoperative contamination with liquid feces compared to formed feces is more likely. After anesthetic induction, digitally remove feces from the rectum. The anus may be closed with a purse-string suture after placement of a rectal tampon or left open for placement of a syringe case per rectum at surgery. The latter technique assists in identification of rectal abnormalities and the external anal sphincter muscle. Perioperative antimicrobials (e.g., cefazolin - 22mg/kg IV) may be used.
Traditional technique

Make a curvilinear skin incision lateral to the anus from the level of the tail head to that of the ischiatric tuberosity on the affected side. Expose the lateral rectal wall by bluntly dissecting medial to the levator ani muscle. Dissect adhesions between the hernia contents and surrounding structures. Identify and protect the pudendal artery, vein, and nerve. Reduce the hernia contents and maintain reduction using a gauze sponge placed on an Allis tissue forceps. Occasionally, excision of hypertrophied retroperitoneal fat or cystocentesis is necessary to achieve hernia reduction.

Preplace sutures to improve visualization during their placement. Sutures should be synthetic, monofilament and either absorbable (e.g., polydioxanone or polyglyconate) or nonabsorbable (e.g., polypropylene or monofilament nylon) and of relatively large diameter (size 2-0 or 0). Place sutures between the levator ani and coccygeus muscles (dorsolateral), the external anal sphincter muscle (medially), the internal obturator muscle (ventrolateral), and possibly the sacrotuberous ligament (laterally). Incorporate fascia, if possible, when placing sutures. Five or more sutures usually are placed during this technique. Take care to avoid the sciatic nerve and caudal gluteal vessels when placing sutures in the sacrotuberous ligament. Take care to avoid the anal sac and rectal mucosa when placing sutures in the external anal sphincter muscle. Close the subcutaneous tissue with synthetic absorbable sutures (e.g., 4-0 monocryl) and skin with synthetic nonabsorbable sutures (e.g., 3-0 polypropylene).

Castration of the intact patient is performed because of its potential beneficial effect on prostatic size. In dogs in which fixation of the deferent ducts is to be performed, castration is performed prior to the ventral midline approach, and ligation techniques are modified as described below.

Internal obturator muscle transposition technique

The most common complication after perineal herniorrhaphy is recurrence of the hernia. Relatively recent techniques have been used to help reduce the incidence of recurrence after perineal herniorrhaphy. These techniques include transposition of the internal obturator muscle, recognition and repair of rectal abnormalities, colopexy, cystopexy, fixation of the ductus deferens, and use of supporting materials (e.g., SIS or polypropylene mesh). Each technique is not applicable to every case of perineal hernia; however, selected use of one or more of these techniques seems to reduce the potential for recurrence.

Transposition of the internal obturator muscle has been associated with a lowered incidence of recurrence compared to that seen with the conventional repair technique. The dorsally transposed internal obturator muscle fills the defect left after reducing the hernia contents. The dorsolateral aspect of the transposed internal obturator muscle (including its tendon) is sutured to the levator ani and coccygeus muscles, its medial aspect is sutured to the external anal sphincter muscle, and its lateral aspect is sutured to the sacrotuberous ligament. Tension on tissues, particularly the external anal sphincter muscle, appears to be less compared to when the traditional technique is used. Also, simultaneous bilateral repair using the internal obturator muscle transposition technique results in lower patient morbidity immediately postoperatively than that seen with the traditional technique.

Additional techniques

Rectal abnormalities that may accompany perineal hernia include deviation (flexure), dilatation (sacculation), and diverticulum. Recognition and correction of these problems may reduce postoperative recurrence rates following perineal herniorrhaphy. Diagnosis of rectal abnormalities is presumptive after rectal examination, although confirmation may require radiologic evaluation (i.e., modified barium enema). Rectal deviation is usually treated effectively by combining a colopexy with repair of the hernia. Rectal sacculation is an outpouching of the rectal wall with enlargement of the rectal lumen. Repair involves inversion or excision of the redundant tissue and an inverting closure using synthetic absorbable sutures. Rectal diverticulum is protrusion of rectal mucosa through a defect in the muscularis. Repair involves inversion or excision of the protruding mucosa and closure of the muscular defect in the rectal wall using synthetic absorbable sutures.

Colopexy is performed via a ventral midline celiotomy. Its purpose is to stabilize the colon in a cranial position to prevent perineal herniation of the rectum and colon. Apply cranial traction to the colon, create a 3- to 5-cm longitudinal seromuscular incision near the antimesenteric border of the distal descending colon. Make a similar incision through the transversus abdominis muscle on the left abdominal wall > 5 cm lateral to the ventral midline. Suture the colon to the left dorsolateral body wall using 5 to 6 interrupted sutures of synthetic nonabsorbable material (e.g., 3-0 polypropylene) in partial thickness fashion.

Fixation of the ductus deferens is performed to provide fixation of the prostate and urinary bladder within the abdomen. This technique is particularly useful when repairing a perineal hernia that contains a retroflexed urinary bladder. Fixation of the ductus deferens is performed only in neutered patients. If the patient is to be neutered at the time of fixation of the ductus deferens, modify the orchietomy technique such that the ductus deferens is ligated separately. Perform a ventral midline abdominal approach. If the urinary bladder is herniated, return it to its normal position. Identify each ductus deferens near its termination in the prostate gland. Retract each ductus from the inguinal canal, and provide moderate cranial traction to each deferent duct. Affix the deferent ducts to the ventrolateral abdominal wall using PDS or polypropylene suture. Perform a cystopexy by suturing the urinary bladder to the right abdominal wall. Incise the seromuscular portion of the urinary bladder as described for a colopexy (above). Make a similar incision.
through the transversus abdominis muscle on the right abdominal wall > 5 cm lateral to the ventral midline. Place 3 to 5 interrupted partial thickness sutures of synthetic nonabsorbable material to complete the cystopexy.

Prosthetic implants used during perineal herniorrhaphy include SIS and synthetic (polypropylene) mesh. Results with either material have been favorable, and complications have been minimal. Suture an ovoid piece of mesh in place over the transposed internal obturator muscle with synthetic absorbable sutures. Securely attach the mesh to the fibrous tissue adjacent to the ischium (especially ventromedial) to improve security of closure. Use of prosthetic implants may be reserved for recurrent hernias.

Postoperative management
Analgesia and sedation are provided immediately postoperatively to smooth the recovery from anesthesia. A low-residue diet can be used for the first few days after surgery. Stool softeners are recommended long-term to minimize stress on the repaired perineum during defecation. An Elizabethan collar or side brace may be necessary to control self-inflicted trauma.

Summary
Successful perineal hernia repair is facilitated by knowledge of the perineal anatomy. Hernial recurrence is minimized by identifying and treating accompanying rectal abnormalities, achieving a tension-free closure of the defect, usually through transposition of the internal obturator muscle, utilization of appropriate additional surgical techniques, and assuring effective long-term elimination of soft feces.

References
Surgical approaches

Intercostal thoracotomy and median sternotomy are the two most commonly performed approaches to the thorax in small animals. Be sure to prepare a large enough area to allow placement of a thoracostomy tube such that it exits at least two intercostal spaces from the primary thoracotomy incision.

Intercostal thoracotomy

Use this approach to access a specific region of a hemithorax. This approach provides good access to the pulmonary hilus, heart, and limited portions of the mediastinum and ipsilateral thoracic cavity. While the third through the tenth spaces are theoretically accessible, the fourth through the sixth intercostal spaces provide the most reliable access to lung lobes and the heart.

Use the lateral thoracic radiograph to help determine the appropriate intercostal space to incise. Center the approach over the hilus of the affected lung not over the lesion (cranial lobe - 4th or 5th, middle lobe - 5th, caudal lobe - 6th intercostal space). Use a 4th intercostal thoracotomy incision to expose the heart in the dog. Use the 8th intercostal space to expose the caudal esophagus or thoracic duct.

Incise the skin parallel to the ribs from just ventral to the costovertebral junction to just dorsal to the sternum. Incise the latissimus dorsi muscle with scissors parallel to the skin incision. Verify intercostal space identification by counting caudally from the first rib. Incise the serratus ventralis muscle parallel to its fibers. Incise the intercostal muscles midway between ribs. Bluntly puncture the pleura to allow the lungs to fall away from the lateral thoracic wall before extending the intercostal incision with Mayo scissors. Insert rib retractors over laparotomy sponges to protect skin and muscle.

Alternatively, a muscle-sparing technique for lateral thoracotomy has been described. This technique is comparable in efficiency and visibility but results in less pain than the traditional technique. A Balfour retractor was used to provide retraction, with the side blades providing retraction of the ribs and the center blade providing dorsal retraction of the latissimus dorsi and serratus ventralis muscles. The author has no clinical experience with this approach.

Place a thoracostomy tube as described below. Close the intercostal space by pre-placing heavy (usually ‘0’ suture) absorbable sutures circumcostally. Close the serratus ventralis and scalenus muscles as a separate layer (3-0 polydioxanone suture). Close the latissimus dorsi muscle separately while incorporating its fascia (3-0 polydioxanone suture). Close the subcutaneous tissue and cutaneous trunci muscle together (4-0 poliglecaprone 25 suture), and the skin (3-0 polypropylene).

Median sternotomy

Use this approach when exploring both sides of the thoracic cavity. Structures in the dorsal thoracic cavity (e.g., pulmonary hilus) are more difficult to reach through this approach. Median sternotomy may be combined with a ventral midline celiotomy or a caudal cervical approach. Exposing cranial or caudal mediastinal masses and performing a more complete subtotal pericardiectomy are potential indications for performing a median sternotomy. Avoid incising the entire length of the sternum, as postoperative sternal instability and pain seem to be increased compared to leaving at least one sternebra intact at either end.

Incise the skin, subcutaneous tissues and pectoral muscles over the sternum with a scalpel. Cut the sternum on midline with an oscillating saw, taking care to limit penetration of the saw blade. Protect the tissues with moistened laparotomy sponges, and position retractors (e.g., Finochietto retractors) to achieve adequate visibility and access.

Place a thoracostomy tube prior to closing the incision. Close the sternal incision by pre-placing stainless steel wire (approximately 20 gauge) in a figure-eight pattern to appose each incised sternebra. Close the pectoral muscles and subcutaneous tissues in separate layers. Close the skin in routine fashion.

Thoracostomy tube placement

A thoracostomy tube may be placed associated with a thoracotomy or as a separate procedure. Place a thoracostomy tube before closing the thoracotomy incision. Place the tube so that its skin exit point and thoracic wall entry point are off set. Cut additional holes in the thoracostomy tube near its end. Do not position the thoracostomy tube in the primary incision. Match the size of the tube to the patient size and its intended use (tube size in patients with pleural effusions generally should be slightly larger than those in patients with pneumothorax). Plan to have the tube enter the thoracic wall two intercostal spaces from (usually caudal to) the primary lateral thoracotomy incision. Tube placement during median sternotomy is more challenging, as tunneling of the tube can be more difficult. Position the fenestrated end of the thoracostomy tube at the level of the second sternebra and ventrally. Connect the exterior of the thoracostomy tube to a three-way stopcock using an adaptor. Use suture to secure the tube to the skin using a friction...
suture pattern. Position a C-clamp on the tube below the three-way stopcock for added safety. Cover the thoracostomy tube with a bandage.

Commercially available trochar chest tubes are easier to insert because they contain a metal stylet. Risk of injury to underlying tissues may be increased when thoracostomy tubes are placed as a separate procedure compared to placing them in conjunction with a thoracotomy. Create a small skin incision slightly larger than the tube diameter at the dorsal aspect of the caudal thorax (usually 9th to 11th intercostal space). Advance the tip of the tube subcutaneously about two intercostal spaces before inserting the tube into the pleural cavity. Use a controlled thrust to insert the end of the tube just through the chest wall. Remove the trocar, and advance the tube so that its end is level with the second intercostal space and ventrally positioned. Quickly attach an appropriately-sized adaptor and 3-way stopcock in the end of the tube. Place a friction suture to hold the tube in place securely. Evacuate the pleural cavity, and bandage the thoracostomy tube in place.

Commercially available small bore wire-guided chest tubes are quite easy to insert as either a separate procedure or at the time of thoracotomy. Tube insertion is via the catheter-over-guidewire (modified Seldinger) technique. Such tubes include a normally closed bi-directional valve with luer adapters that prevents air from entering the chest.

**Conditions of the thoracic wall: trauma and neoplasia**

Rib fractures occur in small animals, particularly after blunt trauma. Most rib fractures are treated non-surgically with analgesics and bandage application. Penetrating thoracic wounds from dog fights often result in hidden trauma to underlying tissues. Many cases of penetrating thoracic wall trauma are treated non-surgically, in part, because of the potential for having severely traumatized deeper tissues that are questionable for closure. A traumatic event that may require surgical intervention is flail chest. Flail chest occurs when several adjacent ribs are fractured in at least two places and results in an unstable chest wall segment. The unstable (flail) segment moves paradoxically compared to the rest of the thoracic wall (i.e., in on inspiration and out on expiration). Stabilization of the unstable portion of chest wall improves ventilation. Place percutaneous sutures around the ribs and attach them to an external fixation device that spans the traumatized area.

Surgical resection with wide margins of grossly normal tissue is the treatment of choice for thoracic wall neoplasia. Full thickness resection of multiple ribs may require surgical reconstruction of the thoracic/abdominal wall by using synthetic material (e.g., polypropylene mesh). With tumors of the caudal thoracic wall, the diaphragm can be advanced cranial to the resection site. Such advancement may offer more options for closure of the surgical site.

Position an appropriately-sized (slightly larger than the defect) and shaped piece of polypropylene mesh to cover the defect. Suture the mesh to muscles at the edges of the defect, taking care to draw the mesh tightly across the defect. If the mesh is positioned over the thorax, place a thoracostomy tube, and cover the mesh with either thoracic wall musculature or an omental pedicle flap. Close the subcutaneous tissue and skin in a routine fashion.

**References and suggested reading**


The signalment of most cases of GDV include large to giant breed dogs in the age range of 10 months to 14 years. There is no sex predilection. Clinical signs are most commonly non-productive retching and abdominal distension but animals may present severely debilitated depending on the amount of time they have been affected. Physical exam findings include a distented, painful abdomen, nausea and or active retching. Other signs of illness depend on whether they are in compensatory, endotoxic or noncompensatory shock.

**Diagnosis**

Radiographs are considered the gold standard for diagnosing a GDV but should not be undertaken without medical stabilization of the patient. A right lateral position is the most illustrative view highlighting the “double bubble” or “smurf’s hat” of the stomach being split into two chambers. Other important findings on radiographs would include any evidence of free gas or mesenteric volvulus.

Blood work abnormalities include acid-base derangement, hemoconcentration, hyperlactatemia, a stress leukogram and a hypercoagulable state. Clinical chemistry changes that may be encountered include evidence of hepatocellular damage, biliary stasis, prerenal/renal insufficiency, blood loss, impaired glucose control and impared electrolyte balance.

**Preoperative medical management**

Medical treatment initially consists of gastric decompression, IV fluid resuscitation and pain management. The goal is to stabilize the cardiovascular, respiratory and renal systems. Severe gastric damage is related to more severe gastric distension (not malpositioning) so decompression is of the utmost concern. Fluid therapy is initiated after placement of 1-2 large gauge IV catheters and usually begins with bolus shock therapy (45-90ml/kg) of crystalloids in combination with colloids (10-20ml/kg). Blood pressures should be monitored and if necessary supported with vasopressor administration to protect the renal system from ischemia. Ventricular arrhythmias are commonly seen in dogs suffering from GDV therefor a continuous EKG is applied on admission and postoperatively for at least 24-48 hours.

Gastric Decompression can be accomplished by two techniques and should be performed within 15-20 minutes of the patients diagnosis.

Orogastric tube decompression- this technique requires intubation of the patient to ensure protection of the airway. After intubation, a roll of tape is placed in the mouth caudal to the canines and an orogastric tube of appropriate diameter has lubricant applied to the outside and is gently passed down the esophagus. With the trachea intubated there is no chance of accidentally moving down the trachea. There will be significant resistance with the lower esophageal sphincter is encountered. Gentle rotation of the tube and blowing into the end of the tube can help overcome the pressure. Gastric contents should be seen and or smelt from the tube. Empty the stomach and leave the tube in place to keep the stomach decompressed until surgery.

Trocar decompression- Choose a large gauge over the needle catheter (10-14g). Clip and clean an area on the left or right cranial dorsolateral abdomen that has a tympanic feel when compression is applied. Avoid the spleen by feeling for a more solid structure during palpation. Insert the catheter with an abrupt pop and remove the stylet. An acrid odor will confirm placement into the stomach. Hold the hub firmly against the body wall while pushing inward to facilitate decompression. Remove the needle when no further air is escaping.

Pain management should also be initiated early on during the diagnosis and preoperative medical management as these patients are in severe discomfort. The use of opioids (Hydromorphone, Oxymorphone, Methadone) is recommended and can be easily administered once IV access has been achieved.

**Surgical management**

The goals of surgical treatment are to correct gastric and splenic positioning, determine gastric and splenic viability, and to prevent gastric malpositioning in the future.

- Surgical explore begins with derotation of the stomach. Upon entrance into the stomach, omentum is found overlying if the stomach is in the most common rotated position (180° clockwise around the long axis of the patient). To correct the rotation, the surgeon stands on the right side of the patient, the right hand grasps the stomach wall adjacent to the left body wall and left hand grasps the stomach adjacent to the right body wall. The right hand then pulls ventrally and toward the right body wall while the left hand pushes the stomach dorsally and to the left body wall. The gastro-esophageal junction is carefully palpated and visualized to ensure there is no remaining rotation. If a tube is not already in place one is placed now to aid in full decompression of the stomach.
Assessment of the stomach and spleen is then performed to ensure viability of these organs. The stomach is palpated for any areas of thinning and the color is assessed. Time is given for the color to improve as it commonly does once the rotation has been corrected. Gastric wall resection can be performed with stapling devices or by hand. The splenic vasculature is palpated for pulses. Once repositioned, the spleen should also be given 5-10 minutes for observation of improvement. If a splenectomy is deemed necessary this can be accomplished with vessel sealing devices, stapling devices or hand ligatures.

Once the stomach and spleen have been evaluated, a gastropexy is performed. Recurrence is reported to be as high as 80% for cases that are re-positioned alone. There are 10 different types of gastropexy possible.

- Incisional
- Tube
- Grid
- Belt-loop
- Circumcostal
- Incorporating
- Laparoscopic-assisted
- Laparoscopic
- Percutaneous
- Fundopexy

Incisional gastropexy is the most common and will be discussed in detail

- Technique relies on the healing of the edges of the gastric seromuscular incision to the edges of the peritoneum-transverse abdominal muscle.
- A 3-5 cm incision is made with a scalpel perpendicular to the long axis of the patient, or at an angle on the right ventrolateral body wall 6-8 cm lateral to the celiotomy.
- The incision extends through the peritoneum and the transverse abdominal muscle.
- A 3-5 cm incision in the serosa of the gastric wall at the antrum parallel to the longitudinal axis of the stomach is made next.
- The most cranial incision lines are sutured first with 2-0 to 0 delayed absorbable or nonabsorbable taper needle in a continuous pattern originating dorsally.

Emergency temporary gastropexy has been reported if definitive surgical treatment cannot be provided for a delayed period of time. This entails heavy sedation with local anesthetics or preferably general anesthesia. The patient is clipped and prepped over the right paracostal region and a 10 cm incision is made through the skin. The external abdominal oblique muscle is separated along muscle fiber lines, the internal abdominal oblique and transverse abdominal muscles are also separated. The stomach wall is identified and grasped and pulled towards the skin incision. The gastric wall is then sutured to the skin incision with 2-0 to 3-0 monofilament suture the full circumference of the incision. An incision is made in the center of the stomach and gas, fluid and particulates allowed to escape. The skin is coated liberally with TAB or sterile lubricant to prevent scalding from gastric contents.

Postoperative Management includes aggressive fluid therapy and continued analgesics. Monitoring for arrhythmias, hypotension, electrolyte and acid-base derangements are vitally important. Gastric protectants (H2-receptor antagonists, coating agents) for mucosal damage and anti-emetics (centrally or peripherally acting) are also administered. Food is usually withheld for 24 hours. Prokinetic medications such as metoclopramide and or erythromycin can be given to combat ileus. Recurrence of dilatation can still occur after gastropexy but no recurrences of volvulus have been reported with the incisional technique.

Postoperative complications include ongoing necrosis of the stomach leading to a septic abdomen, progression of preoperative shock leading to MODS, SIRS or ARDS, and even death.

Prognosis is mostly affected by early recognition and treatment. The need for gastric resection and poor lactate clearance have been associated with poor prognosis in some studies. Survival rates have been as high as 98% in patients without gastric necrosis and 66% in dogs with gastric necrosis.
With the new EPA regulations the types of rodenticides seen in practice are changing. Bromethalin is on the upswing along with the non-second generation anticoagulants.

**Anticoagulants**

Anticoagulants in use as rodenticides today are almost all second-generation derivatives. They inhibit the activity of vitamin K epoxide reductase, which converts vitamin K epoxide to the active reduced form. This reduced vitamin K is crucial to activation of clotting factors II, VII, IX, and X.

Any exposure > 0.02 mg/kg of a second generation anticoagulant requires treatment and evaluation. Emesis can be induced if ingestion has occurred within the last 4 hours. If little or no bait is recovered, administration of activated charcoal is next. Another option is to institute Vitamin K1 therapy (2.5-5 mg/kg/day) or monitor PT tests. Because the body has several days’ worth of active Vitamin K stored in the liver (the site of the re-activation activity), there is a delayed onset of effect on blood clotting after ingestion of an anticoagulant. Factor VII has the shortest half-life, so we can get the earliest valid estimate of effect by checking the prothrombin time (PT). The PT is expected to elevate within 24-48 hours post ingestion.

Early signs of anticoagulant toxicosis are vague, and depend on the site of a bleed. Lethargy, non-productive cough, intermittent lameness, mild anemia, or even sudden collapse can be seen. Petechiae and ecchymoses are more often seen later in the course of illness, after the platelet numbers have been depleted in smaller bleeds. Diagnosis is based on signs, history of possible exposure, and coagulation studies.

If the animal is actively bleeding, start vitamin K1 and give clotting factors via a whole blood transfusion, fresh frozen plasma, or fresh plasma. Minimize physical activity throughout therapy.

**Bromethalin**

Bromethalin is a neurotoxin that uncouples oxidative phosphorylation in CNS mitochondria. This results in lack of adequate ATP concentration and insufficient energy for maintaining Na⁺-K⁺ ion channel pumps. Loss of pump activity results in cerebral and spinal cord edema and a demyelination injury to long nerves.

Bromethalin is rapidly absorbed from GI tract. Cats are far more sensitive to this agent than are dogs. Dogs seem to have both a low-dose and a high-dose syndrome. With lower doses signs may not appear for 72-96 hours, and include hind limb ataxia and paresis, decreased proprioception, loss of deep pain response, vocalizations, patella hyper-reflexia, CNS depression progressing to coma, vomiting, and fine muscle tremors. At or above the mean lethal dose, signs can begin within 12-24 hours and include severe tremors, hyperthermia, extreme hyperexcitability, running fits, hyperesthesia and seizures.

Treatment of clinical signs is directed to controlling cerebral edema, and is mostly frustrating and non-productive. Mannitol, corticosteroids and diazepam may be used. Animals with sub-lethal doses will require good nursing care.

**Cholecalciferol**

Cholecalciferol is a Vitamin D₃ analog. It alters calcium metabolism in the body, increasing intestinal absorption and renal tubular reabsorption of calcium and stimulating bone resorption. Clinical signs of intoxication usually develop within 12-36 hours. Early signs include lethargy, weakness, anorexia, polydipsia, polyuria, and vomiting, often with blood. Biochemical alterations include hyperphosphatemia within 12 hours and hypercalcemia within 24 hours of exposure and azotemia (both renal and pre-renal). The elevated calcium levels result in calcification of many tissues, notable renal tubules and walls of blood vessels. The elevated calcium also has a direct effect on kidney function, sometimes causing acute renal failure even without mineralization.

Diagnosis of toxicosis is based on history of exposure, clinical signs, serum chemistries and urinalysis. Run baseline chemistries as soon as possible after a known exposure. Pursue GI decontamination if within several hours of ingestion, or if there is evidence of ingestion (chewed box) at unknown time but a still asymptomatic animal. Multiple doses of activated charcoal and cholestyramine can help decrease absorption.

Treatment is aimed at lowering the serum calcium and phosphorus levels, preventing a rise in these values if still normal, and stopping further calcium mobilization from the bones. IV normal saline at twice maintenance, prednisone and furosemide all enhance calciuria. Monitor serum calcium, phosphorus, BUN and creatinine daily to judge effectiveness of therapy. If calcium levels are rising despite calciuresis, best choice is pamidronate (Aredia™). Unlike salmon calcitonin, it needs to be given only once, with a repeat dose possibly at about 5-7 days. It acts at the level of the osteoclast and is deposited in the bone itself. Once the pamidronate has been administered, it is important to taper the initial treatments (prednisone, furosemide) and decrease the rate of fluid administration.
Continue to monitor calcium, phosphorus, and kidney values during this time. End of therapy will be marked by a return to normal of kidney values and the decrease of calcium x phosphorus levels (in mg/dl).

**Zinc phosphide**

Zinc phosphide is an old rodenticide posing as a new one. The phosphide salts are unstable in an acid environment. At gastric pH they degrade rapidly to form phosphine gas. Phosphine gas, when inhaled, results in acute non-cardiogenic pulmonary edema. When absorbed systemically, it is thought to block cytochrome C oxidase, leading to formation of highly reactive oxygen compounds. It is these reactive compounds which cause most of the tissue injury, most severe damage is in tissues with the highest oxygen demand – brain, lungs, liver and kidney.

Lethal doses for cattle, sheep, pigs, goats, dogs, and cats range between 20-50 mg/kg. For a 55 pound (25 kg) dog, that would be between 10 grams (0.35 ounce) and 25 grams (just under an ounce) of 5% bait. Severely poisoned animals may die in 3-5 hours. Those who survive longer than 48 hours have a pretty good chance.

Initial signs may vary by species, as well as by the dose. Onset of signs is normally between 15 minutes to 4 hours post ingestion. Vomiting, often with blood, is common. Dogs may show lateral recumbency with whole body tremors and salivation. Other signs may include anorexia and lethargy. Rapid deep breathing may signal the onset of the pulmonary changes. Abdominal pain, ataxia, and weakness leading to recumbency may follow. Hyperesthesia and seizures may develop that resemble the signs of strychnine toxocosis.

Metabolic acidemia ensues. Other biochemical changes may include depressed serum calcium and magnesium. If there is survival beyond 48 hours an elevated blood urea is common. Hepatic and renal damage often may be detected 5-14 days later.

Initial decontamination is tempered by the wish to keep the stomach pH as high as possible to prevent the formation of phosphine gas. If there has been no spontaneous vomiting, it may be better to induce emesis with apomorphine rather than hydrogen peroxide. Giving food, commonly done in order to improve gastric emptying and the response to peroxide, will trigger release of gastric acid and increase the rate of production of phosphine. If you are going to perform gastric lavage, add an alkalizing component like a magnesium and aluminum hydroxide gel to your lavage liquid. Also consider mixing into your activated charcoal preparation.

Supportive care includes IV fluids to maintain blood pressure renal perfusion, and gastroprotectants. Seizures may respond to diazepam, or may require barbiturates or full anesthesia. Since the most severe injury is probably due to action of the oxygen radicals, use of an antioxidant may be useful – consider vitamin C or n-acetylcysteine.

Caution: Phosphine gas released from vomitus or stomach washings can cause significant illness in veterinary personnel assisting animal. Phosphine has been describes as having a spoiled fish or garlic odor. It is detectable at 1-3 ppm in air; maximum allowed in air in occupational situations is 0.3 ppm, so if you can smell it, you are being exposed to a concentration that can be harmful.
Common Household Hazards
Tina Wismer, DVM, DABT, DABVT
ASPCA Animal Poison Control Center
Urbana, IL

“The good”
Desiccants
Desiccant packs are included as moisture absorbents. They are found in shoeboxes, electronics, medications and food. Silica gel, one of the most common desiccants, is a white powder or a lustrous granule. Silica gel comes in paper packets or plastic cylinders. Packages of silica gel are attractive to pets because of the rustling noise, and the packages are easy to bat around. Most ingestions will not cause clinical signs, although a mild gastrointestinal upset may occur. If a large amount is ingested, there is potential for osmotic diarrhea occurring. Ingestion of the intact packet may cause a gastrointestinal obstruction.

Food products often contain desiccants composed of iron. Deli meats, pepperoni, etc. are likely to have this type of desiccant. The iron content can range from 30-60%. Once the iron has oxidized, the resulting compound (iron oxide) is inert and non-toxic. Vomiting is very common.

Ant and roach baits
Ant and roach baits are common objects found in households. They are also referred to as hotels, traps, or stations. The insecticides used most commonly in these baits are sulfurlamid, fipronil, avermectin, boric acid, and hydramethylnon, all of which are of low mammalian toxicity and present in very low concentrations within the baits. The baits also contain inert ingredients such as peanut butter, breadcrumbs, fats and sugar to attract the insects; these agents are also sometimes attractive to pets. Exposures of pets to these types of ant baits usually do not require decontamination or treatment.

Birth control pills
Birth control pills generally come in 28 tablet packs with 21 hormone tablets (estrogen +/- progesterone) and 7 placebo tablets. Most hormone pills contain 0.035 mg of estrogen or less. In general, estrogen doses of less than 1 mg/kg are not of concern. At higher doses, bone marrow suppression may be seen.

Non-ionic and anionic detergents
Non-ionic and anionic detergents are found in a wide variety of household products, including body soaps, shampoos, dishwashing detergents, various household cleaners, etc. These products are gastrointestinal and ocular irritants with few to no systemic effects. Clinical signs consist of hypersalivation, vomiting, and diarrhea, and are generally mild and self-limiting, although ingestion of large quantities may result in more severe vomiting (+/- blood) requiring veterinary intervention.

Toilet water (tank drop-Ins)
Tank "drop in" products typically contain anionic/nonionic detergents, cationic detergents, bleach, and/or acids. However, when a tank "drop in" cleaning product is used in a toilet, the actual concentration of the cleaner is very low in the bowl. With dilution by the bowl water, the cleaning agent is just a gastric irritant. Common signs seen with ingestion include mild vomiting.

Glow-in-the-dark sticks and jewelry
Glow-in-the-dark items (glo-sticks, necklaces) are popular novelty items that are sold at fairs, carnivals, novelty stores and skating arenas. The primary luminescent agent in these types of products is dibutyl phthalate (n-butyl phthalate), an oily liquid that is also used as a plasticizer and insect repellent. Dibutyl phthalate is of low toxicity (LD50 >8000 mg/kg in rats) so serious problems are unlikely. Even though the extremely unpleasant taste of dibutyl phthalate may limit exposure, some very dramatic signs may be seen. Signs generally occur within seconds of the pet biting into the item. Compared to dogs, cats tend to have a much more exaggerated reaction to the taste of dibutyl phthalate. Cats may display profuse salivation and foaming, with occasional retching and/or vomiting. In all cases, signs are generally self-limiting and should resolve once the pet gets the taste of the product out of their mouth. The exposure is managed by diluting the taste of the dibutyl phthalate using milk or highly palatable food (e.g. canned tuna). Any chemical that has gotten on skin or fur should be bathed or wiped off to prevent re-exposure when the animal grooms themselves; taking the pet into a darkened room will aid in identifying the luminescent chemical on the skin or coat.

“The bad”
Acids
Products containing acids include cleaning agents (e.g. toilet bowl cleaners), anti-rust compounds, etching compounds, automotive batteries, and pool sanitizers. The relative toxicity of an acid is related to its concentration and decreases with dilution. Acids produce localized coagulative necrosis of tissue and generally produce immediate pain upon exposure, which helps to limit ingestion. In most cases, clinical signs occur almost immediately upon exposure. Oral exposure results in oral pain, vocalization, dysphagia, vomiting (+/- blood), abdominal pain, and irritation or ulceration of oral and/or esophageal mucosa. Lesions often appear milky white to gray initially, then gradually turn black. Esophageal lesions are less common than with alkaline products. With high levels of exposure, gastric ulceration is also possible. Dermal exposure results in dermal irritation or ulceration, accompanied by intense local pain.
Inhalation of acid fumes may result in dyspnea, pulmonary edema, tracheobronchitis or pneumonitis. Ocular exposure may result in corneal erosion or ulceration.

Attempts to chemically neutralize with a weak alkali are contraindicated, as this may stimulate an exothermic reaction that will exacerbate tissue injury. Treatment of oral exposure includes immediate dilution with water or milk. Gastric lavage and induction of emesis are contraindicated due to the risk of increasing corrosive injury. Activated charcoal is ineffective for caustic agents and should not be used. Treatment of oral lesions is symptomatic, and should include antibiotics to prevent infection; pain management (opioids), sucralfate slurries to treat oral, esophageal or gastric ulcers; intravenous fluids to maintain hydration; and provision for nutritional support (e.g. gastrostomy tube). Dermal exposures should be treated with copious flushing with clear water for 15 minutes. For ocular exposures, eyes should be flushed with room temperature water or sterile saline solution for 15 minutes. Fluorescein staining of the eyes should be performed, and corneal erosion or ulceration should be treated as needed. Animals with significant respiratory signs (coughing, dyspnea, etc.) should be monitored for a minimum of 24 hours for the development of pulmonary edema. Supplemental oxygen or other respiratory supportive care should be used as needed.

Alkalis

Alkaline products include sodium or potassium hydroxide, ammonium hydroxide, sodium or potassium hydroxide, and potassium peroxymonosulfate. Common sources of alkaline products include drain openers, automatic dishwasher detergents, alkaline batteries, toilet bowl cleaners, swimming pool products and radiator cleaning agents. Agents with pH greater than 11 should be considered to be capable of causing significant corrosive injury. Alkaline agents penetrate local tissue rapidly and deeply, causing liquefactive necrosis. Unlike acidic products, very little pain may be evident upon initial contact with an alkaline product, which may encourage further contact and ultimately result in more extensive exposures.

Clinical signs may not develop immediately, and it may require up to 12 hours for the full extent of tissue damage to become apparent. Acute signs are similar to acid ingestions but may also include significant hyperthermia (>104°F). Esophageal and/or pharyngeal ulceration may occur. Treatment is the same as with acid exposures, emesis should NOT be induced and activated charcoal should not be given. Evidence of oral discomfort and inflammation generally develop within 2 to 4 hours, although the full extent of injury may not be evident until 12 hours post exposure. Esophageal lesions may take weeks to heal and there is risk of stricture formation, leading to impairment of esophageal function.

Cationic detergents

Cationic detergents are contained in fabric softeners, some potpourri oils, hair mousse, algaecides, germicides and sanitizers. Cationic detergents are more toxic than non-ionic/anionic detergents and can cause extensive systemic and local effects at levels as low as 2% or less. Local tissue injury caused by cationic detergents resembles that seen with exposure to alkaline products (see Alkali section). In addition, cationic detergents can cause systemic toxicity including CNS depression, coma, seizures, hypotension, muscular weakness and fasciculations, collapse, pulmonary edema, and metabolic acidosis; the mechanism of these signs is not known. Treatment of local exposure is similar to that for alkaline products (see Alkali section). Systemic signs should be treated symptomatically (i.e. fluids for hypotension, diazepam for seizures, etc.).

Pennies

Pennies minted since 1983 contain 99.2% zinc and 0.8% copper, making ingested pennies a rich source of zinc. Other potential sources of zinc include hardware such as screws, bolts, nuts, etc., all of which may contain varying amounts of zinc. In the stomach, gastric acids leach the zinc from its source, and the ionized zinc is readily absorbed into the circulation, where it causes intravascular hemolysis.

The most common clinical signs of penny ingestion are vomiting, depression, anorexia, hemoglobinuria, diarrhea, weakness, collapse and icterus. Secondarily, acute renal failure may develop. Clinical laboratory abnormalities will be suggestive of hemolysis (elevated bilirubin, hemoglobinuria, hemoglobinemia, regenerative anemia) and may also indicate the development of kidney failure. Serum zinc levels may be obtained—blood should be collected in all plastic syringes (no rubber grommets) and shipped in Royal blue top vaccutainers to minimize contamination with exogenous zinc. Radiography of the abdomen may reveal the presence of coins or other “hardware” within the stomach.

Treatment for recently ingested pennies would include induction of vomiting. Activated charcoal is not indicated, as it is of little benefit in binding metals. Removal of zinc-containing foreign bodies via endoscopy or gastroscopy/enterotomy may be required. Treatment for symptomatic animals should include blood replacement therapy as needed, intravenous fluids, and other supportive care. The use of chelators may not be necessary in cases where prompt removal of the zinc source is accomplished. If chelation therapy is instituted, careful monitoring of renal parameters is important for the duration of therapy.

Polyurethane adhesives

Isocyanate glues (Gorilla Glue®, Elmer’s ProBond Polyurethane Adhesive®) are expanding wood glues that have been associated with gastric foreign bodies (FB) in dogs. These products contain isocyanates. When ingested (chewing a 2 oz bottle of adhesive has been sufficient) the adhesive polymerizes into a large, friable FB that can form a cast of the gastric lumen. The adhesive is hygroscopic, absorbing water from the stomach as it expands and the warm body temperature may also play a role in expansion. Dogs
licking small amounts off of the floor or ingesting paper towels soaked with the product generally had mild, transient GI signs but no FB. Attempts to dilute recently ingested glues with food or liquids have not prevented FB development. Do not induce emesis due to risk of expanding FB in esophagus. Radiographs can be performed to determine the presence of a FB in the stomach (looks like kibble). Sometimes the FB is large enough to palpate. Evidence of a foreign body has been seen as early as 4 hours post-ingestion, but radiographs at 24 hours post-ingestion are likely to be more reliable. If present, the FB will require surgical removal.

**“The tasty” Chocolate**

There are a wide variety of chocolate and cocoa products to which pets may be exposed, including candies, cakes, cookies, brownies, and cocoa bean mulches. The active (toxic) agents in chocolate are methylxanthines, specifically theobromine and caffeine. Methylxanthines stimulate the CNS, act on the kidney to stimulate diuresis, and increase the contractility of cardiac and skeletal muscle. The relative amounts of theobromine and caffeine will vary with the form of the chocolate (see table).

The LD₅₀’s of theobromine and caffeine are 100-300 mg/kg, but severe and life threatening clinical signs may be seen at levels far below these doses. Mild signs have been seen with theobromine levels of 20 mg/kg, moderate signs have been seen at 40-50 mg/kg, and seizures have occurred at 60 mg/kg. Clinical signs occur within 6-12 hours of ingestion. Initial signs include polydipsia, bloating, vomiting, diarrhea, and restlessness. Signs progress to hyperactivity, polyuria, ataxia, tremors, seizures, tachycardia, PVC’s, tachypnea, cyanosis, hypertension, hyperthermia, and coma. Death is generally due to cardiac arrhythmias or respiratory failure. Because of the high fat content of many chocolate products, pancreatitis is a potential sequela.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Theobromine</th>
<th>Caffeine</th>
</tr>
</thead>
<tbody>
<tr>
<td>White Chocolate</td>
<td>0.25</td>
<td>0.85</td>
</tr>
<tr>
<td>Milk Chocolate</td>
<td>58</td>
<td>6</td>
</tr>
<tr>
<td>Semi-sweet Chocolate chips</td>
<td>138</td>
<td>22</td>
</tr>
<tr>
<td>Baker’s Chocolate (unsweetened)</td>
<td>393</td>
<td>47</td>
</tr>
<tr>
<td>Dry cocoa powder</td>
<td>737</td>
<td>70</td>
</tr>
</tbody>
</table>

Management of chocolate ingestion includes decontamination via emesis. Activated charcoal may be given in some instances. Intravenous fluids at twice maintenance levels will help maintain diuresis and enhance urinary excretion. Because caffeine can be reabsorbed from the bladder, placement of a urinary catheter is recommended. Cardiac status should be monitored via EKG and arrhythmias treated as needed; propranolol reportedly delays renal excretion of methylxanthines, so metoprolol is the beta-blocker of choice. Seizures may be controlled with diazepam or a barbiturate. In severe cases, clinical signs may persist up to 72 hours.

**Bread dough**

Raw bread dough made with yeast poses mechanical and biochemical threats to animals ingesting it. The warm, moist gastric environment stimulates yeast growth, resulting in expansion of the dough mass, resulting in gastric distention, which if severe, can result in respiratory and vascular compromise. Perhaps more significant is the release of alcohol from yeast fermentation, resulting in profound metabolic acidosis, CNS depression and death. Early clinical signs may include unproductive attempts at emesis, abdominal distention, and depression. As alcohol intoxication develops, the animal becomes ataxic and disoriented. Eventually, profound CNS depression, weakness, recumbency, coma, hypothermia may occur. Management of exposure includes decontamination and treatment for alcohol toxicosis. Because emesis is often unsuccessful, gastric lavage with ice water may be needed.

**Moldy food (tremorgenic mycotoxins)**

Tremorgenic mycotoxins produced by molds on foods are a relatively common, and possibly under-diagnosed, cause of tremors and seizures in pet animals. Because of their relatively indiscriminate appetites, dogs tend to be most commonly exposed to tremorgens. These toxins are produced from a variety of fungi that grow on practically any food, including dairy products, grains, nuts, and legumes; compost piles may also provide a source of tremorgens.

Clinical signs include fine muscle tremors that may rapidly progress to more severe tremors and seizures. Death generally occurs in the first 2 to 4 hours and is usually secondary to respiratory compromise, metabolic acidosis or hyperthermia. Other signs that may be seen include vomiting (common) hyperactivity, depression, coma, behavior alterations, tachycardia, and pulmonary edema. Asymptomatic animals exposed to moldy foods should be decontaminated via emesis or lavage followed by activated charcoal and cathartic. In symptomatic animals, control of severe tremors or seizures has priority over decontamination. Seizures may respond to diazepam, tremors respond best to methocarbamol (Robaxin®; 55-220 mg/kg IV to effect). Supportive care should include intravenous fluids, thermoregulation, and correction of electrolyte and acid-base abnormalities. In severe cases, signs may persist for several days, and residual fine muscle tremors may take a week or more to fully resolve. Testing of stomach content, suspect foods, or vomitus for tremorgens is available through the Animal Health Diagnostic Laboratory, Michigan State University (517-355-0281).
Macadamia nuts
Macadamia nuts are cultivated from *Macadamia integrifolia* trees commonly found in Hawaii and Australia. After ingesting macadamia nuts, dogs develop weakness, depression, vomiting, ataxia, tremors, transient paresis, and hyperthermia. The mechanism of macadamia nut toxicosis in dogs is not known. Signs develop within 12 hours and most dogs return to normal with minimal care within 48 hours. Clinical signs are reported at ingestions as low as 2.4 g/kg body weight. Treatment of clinical signs includes fluids and thermoregulation. Prognosis of macadamia nut intoxication is good.

Xylitol
Xylitol is a sugar alcohol. It is used in sugar-free products such as gums and candies as well as for baking. It doesn’t cause significant increases in blood glucose or insulin in humans. However, in dogs, xylitol causes a rapid, dose-dependent insulin release followed by potentially significant hypoglycemia. Signs can include vomiting, weakness, ataxia, depression, hypokalemia, seizures, and coma. Some dogs have developed liver dysfunction or failure following ingestion of xylitol although the mechanism of action is unknown.

Treatment of xylitol ingestion by dogs should include emesis, if asymptomatic. A dog can show signs of hypoglycemia in as few as 30 minutes. Activated charcoal does not bind xylitol. Frequent small meals or oral sugar supplementation may be used to manage dogs not showing signs. If clinical signs of hypoglycemia develop, a bolus of IV dextrose followed by a dextrose CRI should be used to control moderate to severe hypoglycemia. Hypokalemia, likely secondary to insulin-induced movement of potassium into cells, should be treated if significant. Treatment should continue until blood glucose normalizes. Liver enzymes should be monitored for 24 hours.
Prevent toxicant absorption
Decontamination should be instituted only after the animal has been fully stabilized. If there could be possible legal action, seal with tape and initial/date sample. It is important to maintain records of chain of custody of samples (vomitus, carcass, etc.).

Ocular exposure
Ocular exposures may cause irritation or corrosion of the ocular tissues depending on the substance, the concentration, the exposure time and the sensitivity of the patient. With any ocular exposure, the eyes should be flushed repeatedly with tepid water or saline solution for a minimum of 20-30 minutes. An eyedropper may be used for smaller patients. With a larger patient, fill a plastic cup and slowly pour over the ocular area, or a medicinal syringe may be used. Patients may be given a mild sedative prior to flushing if needed and if the health of the patient will allow. If not sedated, the patient should be allowed to rest at regular intervals during the flushing to minimize stress. Fluorescein staining should be performed after flushing and repeated at 12 – 24 hours post-exposure to check for corneal ulceration. Treatment with lubricant ointments should follow staining, and topical medications applied as indicated.

Dermal exposure
Dermal exposures may occur to a large variety of substances including petroleum products, pesticides and insecticides, corrosive or irritating materials and substances that are sticky (tar, asphalt, sap and glue). Removal of dermal substances may be less stressful if the patient is sedated. Sedatives should only be used if the health of the patient will allow. If not sedated, the patient should be allowed to rest at regular intervals during the bathing to minimize stress.

Bathing
For dogs and cats, bathing in a mild liquid dishwashing detergent (e.g. Dawn) and warm water is recommended. Baths may need to be repeated to completely remove the toxicant. Afterwards, the animal should be rinsed well with warm water and towel dried to prevent chilling. These patients should be kept in a warm environment away from drafts until completely dry. Dermal substances can be removed from very small animals such as birds, reptiles or rodents by misting with room temperature water in a warm environment. Misting should continue until the product can no longer be detected on the coat or feathers by odor or touch. If misting is insufficient at removing the product, a liquid dishwashing detergent (e.g. Dawn) should be diluted in the misting bottle and applied, making sure to avoid the eyes. After removal of the substance, the animal should be rinsed via misting with clear water until all soap is removed. With heavy exposures, the animals may be bathed with liquid dishwashing detergent and rinsed well, with care taken not to over-stress the animal. After misting or bathing, the animal should be wiped with a dry towel and kept in a warm environment away from drafts until completely dry.

Sticky substances
When dealing with sticky substances (e.g. gum, glue traps, tar, etc), the use of solvents should be avoided as solvents may cause dermal irritation or burns. To remove sticky substances from mammals, trim the fur to remove as much of the substance as possible. Then work a small amount of vegetable oil, mineral oil, mayonnaise or peanut butter through the rest of the substance until it breaks down into "gummy balls". Afterwards, wash with liquid dishwashing detergent as described above. For birds, do not trim the feathers, just utilize vegetable oil, mayonnaise or peanut butter and then bathe.

Oral exposure
Dilution
Dilution with milk, water, or liquid from water-packed tuna fish is recommended in cases of ingestion of corrosive or irritant products, exposure to toad secretions, or taste reactions due to topically applied products (e.g. “foaming kitties” following flea spray application). Dilution with milk may also aid in relief of oral discomfort secondary to chewing on plants that contain insoluble calcium oxalates in their leaves (e.g. Philodendron spp.). For birds and reptiles, juicy fruits and vegetables can be fed to accomplish dilution.

Emesis
Emetics generally empty 40-60% of the stomach contents and are assumed to be more beneficial than gastric lavage. Dogs, cats, ferrets, and potbellied pigs are examples of animals that can vomit. Emetics should not be used in rodents, rabbits, birds, horses, and ruminants. Induction of emesis is contraindicated with ingestion of corrosive agents or hydrocarbons. Pre-existing conditions (e.g. seizure disorders, severe dyspnea) may also cause use of an emetic to be contraindicated. Emesis should not be attempted if the animal
has already vomited or is exhibiting significant clinical signs. Potential complications from emesis induction may include aspiration, persistent gastritis, and transient bradycardia (due to vagal stimulation).

Emesis is most productive if performed within 2-3 hours post-ingestion. In some cases, such as ingestion of chocolate, large numbers of sugar-coated tablets, grain-based rodenticides, or plant material, emesis may be effective even after 2-3 hours due to formation of boluses of product in the stomach (chocolate, tablets) or delay in gastric emptying (grain-based products, plants). Feeding the animal a small meal prior to inducing vomiting can increase chances of an adequate emesis.

Three percent (3%) hydrogen peroxide is a preferred emetic, especially if emesis is to be induced at home by the owner. Peroxide is readily available (needs to be “fizzy,” not flat), easy to administer, and often highly effective, especially in dogs. The dosage is 1 teaspoonful/5 lbs body weight, not to exceed 3 tablespoons. Vomiting usually occurs within 10-15 minutes and the dose can be repeated once if not initially successful. In the process of foaming (which triggers the vomiting) the peroxide is converted to water and oxygen, so if no vomiting occurs there is no concern about adverse effects from the retained peroxide. Overdosing with hydrogen peroxide should be avoided, as it may result in gastritis that may take days to resolve.

Apomorphine hydrochloride may also be utilized as an emetic in dogs. The recommended dosage is 0.04 mg/kg IV, SQ. Reversal of the CNS depression from apomorphine may be accomplished through the use of naloxone. An alternative route of administration is to instill apomorphine conjunctivally. The eye should be rinsed well after the animal has vomited. Anecdotally, the latter method results in less CNS depression than injection. Xylazine or dexmedetomidine can be used as an emetic in cats. It will cause significant hypotension, bradycardia and CNS depression, but these effects can be reversed with yohimbine or atipamezole.

Other emetics have been used including salt, liquid dishwashing liquid, syrup of ipecac and powdered mustard. Salt that is not vomited up may result in hypernatremia, causing severe neurological derangements. Syrup of ipecac generally has a delay in onset of action of up to 40 minutes in dogs and if not vomited up can cause myocardial depression and hypotension; the FDA has withdrawn ipecac as an emetic for human use due to questions of efficacy and safety. Powdered mustard does not appear to be an effective emetic in dogs or cats.

Adsorbents
Activated charcoal adsorbs toxicants and facilitates excretion via the feces by capturing the toxicant molecules in its micro-porous matrix. Activated charcoal is available in powder, liquid, gel and capsule forms. Activated charcoal capsules are not uniformly broken down in the GI tract of animals, and many will pass through the digestive tract intact. For this reason, if capsules are to be given, they must be cut open and the charcoal from multiple capsules pooled and then mixed with liquid to be administered. Activated charcoal tablets used for breath freshening or “anti-gas” are not appropriate forms of charcoal for decontamination.

Activated charcoal is contraindicated in animals that have ingested caustic materials. Chemicals that are not effectively adsorbed by activated charcoal include ethanol, methanol, fertilizer, fluoride, petroleum distillates, most heavy metals, iodides, nitrates, nitrites, sodium chloride, and chlorate.

The recommended dose of activated charcoal for all species of animals is 1-3 gms/kg body weight. Repeated doses of activated charcoal every 4-8 hours at half the original dose may be indicated when enterohepatic recirculation of the toxicant is known to occur, or if ingestion of sustained release medications has occurred. See precautions regarding electrolyte disturbances under Cathartics below.

Kaolin-Pectin (Kaopectate) has also been recommended as an adsorbent in some instances. Kaolin is a form of clay (hydrated aluminum silicate) and pectin is a purified carbohydrate derived from fruits. Unfortunately, many of these kaolin-pectin products have recently been reformulated to contain salicylates, which makes their use in small animals less desirable. Another clay, bentonite (colloidal hydrated silica) has been used historically, but in most instances activated charcoal is a superior absorbent to the clays.

Cathartics
Cathartics enhance elimination of substances, including activated charcoal, by moving them through the gastrointestinal tract. Without cathartics, the toxicant bound by activated charcoal can eventually be released and absorbed by the GI tract. Cathartics are not to be used if the animal has diarrhea or is dehydrated. There are saline, osmotic and bulk cathartics.

Caution: Saline and osmotic cathartics may result in electrolyte disturbances (most notably hypernatremia) if overdosed or used in small, dehydrated or debilitated animals. Occasionally, hypernatremia may develop in animals with no apparent predisposition. Animals developing tremors, fasciculation, disorientation or other neurologic signs within 1-3 hours of receiving activated charcoal should have their electrolytes evaluated.

Bulk cathartics can be utilized in mammals and birds. One such cathartic is psyllium (Metamucil®). The dose for dogs and cats is 1 teaspoonful mixed with food every 12 – 24 hours. Psyllium is dosed in birds as follows: mix ½ teaspoon with 60 ml of baby food and give via a dosing syringe or eyedropper. Boiled white rice or unspiced, canned pumpkin may also be used in cats and dogs. Dilute peanut butter, fruit or vegetables can be utilized in birds and reptiles. Timothy hay can be utilized in rabbits.

Osmotic cathartics, like sorbitol, pull electrolyte-free water into the gastrointestinal tract. Sorbitol is commonly combined with activated charcoal in prepared products. The dose is 3ml/kg. Osmotic cathartics can be utilized in mammals, birds and reptiles.

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Saline cathartics include sodium sulfate (Glauber's salts) and magnesium sulfate (Epsom salts). Saline cathartics act by stimulating gastrointestinal motility. The dose is 250 mg/kg mixed in water or activated charcoal. Saline cathartics should not be used in birds or reptiles.

Enemas
Enemas can be helpful when elimination of toxicants from the lower gastrointestinal tract is desired. The general technique is to use plain warm water or slightly soapy warm water. Enemas are not recommended for birds. In reptiles, enemas may be useful since ingested materials often lag for prolonged periods in the colon.

Lavage
Gastric lavage is used in mammals to remove recently ingested toxicants. Gastric lavage should not be used to remove caustic substances or hydrocarbons. Rabbits have very thin stomachs wall so use great caution when performing gastric lavage in this species. Gastric lavage is generally considered to be less effective than emesis in removing toxicants from the stomach, but may be indicated in cases where induction of emesis has been ineffective or is not possible (e.g. seizing animals). General anesthesia must be maintained when performing gastric lavage. A cuffed endotracheal tube should be in place to prevent aspiration. Body temperature water or physiologic saline (preferred for small patients) should be instilled via gastric tube at 10 ml/kg BWT. Use only gravity to instill and to drain the liquid, repeat until lavage fluid runs clear. Inclining the patient head-down at 20-degree angle will facilitate fluid removal, and use of large bore tubes and multiple flushes may yield better success. Body temperature should be monitored closely, as animals may become hypothermic even when body temperature water has been used. Potential complications from gastric lavage may include gastric or esophageal perforation, aspiration, and hypothermia.

Enterogastric lavage is sometimes recommended when potentially lethal oral exposure has occurred and there is need for evacuation of more than just the stomach. Gastric lavage is performed as directed above and an enema administered. A pre-anesthetic dose of atropine (0.02 mg/kg), if not already given for anesthesia and not contraindicated, may aid in intestinal relaxation and prevent abdominal distention. With the stomach tube left in place, the enema tube is attached to a water faucet and digital pressure around the rectal orifice is used to seal the tube in place. Low pressure, body temperature water is allowed to slowly fill the intestinal tract until water flows from the stomach tube; gently massaging the intestines may via abdominal palpation may enhance the water passage. Once the water from the stomach tube flows clear, the process is complete. Potential complications from this procedure include intestinal rupture, profound hypothermia, and gastroenteritis.

Gastrotomy may be indicated for agents that will not readily pass through the gastrointestinal tract on their own. This may include ingestion of pennies and iron supplements (both of which tend to adhere to gastric mucosa), raw yeast bread dough, expandable polyurethane wood glues, lead objects, and ingestion of large amounts of toxic plant material, pill vials or medication tubes.

Crop lavage is used in birds to remove recently ingested toxicants. Frightened and fractious birds should be anesthetized prior to crop lavage. An endotracheal tube should be placed to prevent aspiration. The crop should be flushed gently with warm saline and aspirated. This should be repeated 3 – 4 times. Crop lavage should not be performed in cases of caustic or petroleum distillate ingestion.

Miscellaneous
For patients with cheek pouches (e.g. many rodents), the cheek pouches should be emptied in cases of oral exposure to a toxicant.

Intralipids
Intralipids are lipid emulsions. Lipid emulsions are commonly used as a fat component for parenteral nutrition. While more studies are needed, lipid therapy is very exciting new treatment for lipid soluble toxicoses. Lipid use is based on human research investigating bupivacaine overdoses. The possible mechanism for lipid rescue is that the lipids bind to the fat soluble toxin (“lipid sink”) and bound toxin is inactive. Liposyn, or any other 20% lipid solution, can be given through a peripheral catheter and is relatively inexpensive. A bolus of 1.5 ml/kg is given (over 1 minute if cardiac arrest, slower otherwise), followed by 0.25 ml/kg/min for 30-60 minutes. This is repeated in four hours if the serum is clear. Lipid therapy can hasten recovery time in some cases. There are possible complications to lipid therapy: significant lipemia, pancreatitis, transiently increased liver enzymes, volume overload and lipids can also remove antidotes and other therapies.

Cholestyramine
Cholestyramine is an anion exchange resin available by prescription only. It is used to lower cholesterol in patients who have not responded to normal therapies. Cholestyramine has been used in human medicine to aid in the treatment of toxicoses (amiodarone, digitoxin, chlordane, methotrexate, piroxicam, vitamin D, warfarin, blue-green algae, indomethacin). It binds with bile acids in the intestine, preventing their reabsorption. This stops enterohepatic recirculation. Cholestyramine is not absorbed out of the digestive tract, so it has no systemic effects, but constipation and mild liver enzyme elevation may be seen. The dose is 0.3 – 1 g/kg TID for
several days (depends on toxin ingested). For our patients, the powder should be given or mixed with canned food. Cholestyramine is cost effective with a price around $50-80 for 240g.

Inhalation exposure
“Decontamination” of animals exposed to inhaled toxicants primarily involves removing them from the source of the inhalant and administering oxygen as needed. Depending on the type of inhalant (e.g. smoke, chlorine gas, etc.), monitoring for up to 24 hours for the development of noncardiogenic pulmonary edema is recommended.

Ancillary support
General supportive care includes maintaining hydration, ensuring adequate urine output, monitoring of respiratory, cardiac and neurologic status, and managing clinical signs as they develop. Recumbent or comatose animals require careful monitoring and thermoregulation. Gastrointestinal protectants or anti-emetics may be required (e.g. NSAID overdosages). Management of secondary hepatic or renal injury is imperative.
Marijuana
Over the past few years there has been an increase in the number of marijuana intoxicated pets presented to veterinary clinics. It is unknown if this is truly an increase in cases, if people are more willing to seek veterinary care due to changing attitudes about marijuana or if more potent forms of marijuana are prompting pet owners to seek medical attention.

Marijuana (Cannabis sativa) is used both recreationally and medicinally by people. It is thought to be the most commonly used illegal substance worldwide, with nearly half the population in the United States reporting at least one time use. Marijuana has been used as an anti-emetic, analgesic, anticonvulsant, muscle relaxant, appetite stimulant and to decrease intra-ocular pressure in glaucoma. Currently in the United States, there are 25 states that legally allow cannabis for medical use and four states (Alaska, Colorado, Oregon, Washington) and the District of Columbia, have legalized small amount of cannabis for recreational use by adults age 21 years of age and older. However, it is still a Schedule I controlled substance under the US Controlled Substances Act.

The main toxic principle of marijuana is a resin called tetrahydrocannabinol (THC), but the plant contains over 60 cannabinoids and cannabinoiids. The amount of these resins will vary with plant variety, sex of plant (female plant, “sensemilla” more toxic), geographic location, and growing season. THC acts via stimulation of cannabinoid receptors throughout the body. Cannabinoid receptors found in the pain pathways of the brain and spinal cord mediate its analgesic effects. The antiemetic properties are thought to be secondary to the effect of cannabinoid receptors within the central nervous system. d-9-THC also affects dopaminergic, cholinergic, noradrenergic, serotonergic, and GABA sites. There are 2 main cannabinoid receptors, CB1 and CB2. CB1 receptors, primarily found in the CNS, are associated with psychoactive effects, and peripheral CB2 receptors are associated with the immune system, responsible for the immunomodulatory effects of cannabinoids.

In the past, most pet exposures to marijuana were ingestions of plant material from baggies or joints. This has changed and now edibles (cookies, brownies, etc.) and concentrates (oils, waxes, shatters) have become more popular. Through selective breeding, THC levels have become higher than ever. The University of Mississippi Potency Monitoring Project has reported that THC levels have more than doubled over the last 25 years. THC levels in plant material ranges from 1-8%, extracts 28%, and hash oil up to 50%. Another change has been the increase in marijuana butter based edibles. THC butter is made by heating marijuana in butter to extract the lipophilic THC. This butter is then used to make the baked goods. While both dogs and cats willing ingest plant material, dogs are the most likely to consume edibles. Many of the edibles also incorporate chocolate and this can increase the toxicity.

Another issue with THC containing products is quality control. In one study, 75 products were evaluated to determine the amount of cannabidiol and THC found in the various products. The results indicated that 17% of products were accurately labeled, 23% were under labeled and 60% were over labeled with respect to THC content.

The most common clinical signs after ingesting marijuana are ataxia, lethargy, and urinary incontinence. However, about 25% of patients may present stimulated instead. Hyperesthesia and disorientation are also frequently seen along with bradycardia, hypothermia, mydriasis, and tremors. Animals that get into concentrates or THC butter products may become comatose and hypotensive. Clinical signs can be seen as soon as 30 minutes after oral ingestion and may last up to 72 hours.

Urine drug screening tests have not been validated for use in dogs. Most over-the-counter urine drug tests will give a false negative result for marijuana (THC) in dog urine. This is thought to be due to different metabolites produced by dogs when compared to humans (8-OH-Δ9-THC produced by dogs vs 11-OH-Δ9-THC in humans). These different metabolites may also explain the urinary incontinence that is seen in dogs and not in other species.

As marijuana is an anti-emetic, inducing emesis may not be successful but can be tried with recent (< 30 minutes) oral exposures if the animal is asymptomatic. Activated charcoal is generally not needed. Intravenous fluid administration should be started and adjusted if dehydration or hypotension develops. Diazepam or low dose acepromazine (if normotensive) can be used for agitated patients. Monitor blood glucose levels in young animals. Many cases with plant material ingestion can be managed at home with confinement and monitoring the ability to ambulate.

For more symptomatic animals, monitor respiratory function, heart rate, blood pressure and body temperature. Keep animal warm, quiet, minimize sensory stimuli, and rotate body position q 4 hours if the animal is recumbent. Intralipids (20% solution) may be helpful for severely affected (comatose) animals because THC is lipid soluble but results have been variable. The dosing regime is a 1.5 ml/kg initial bolus (over 20-30 minutes) then a CRI of 0.25 ml/kg/min for 30-60 minutes. Repeat CRI in 4 hours provided there is no lipemia. No specific CBC or chemistry profile abnormalities are expected. There is no role for dialysis, hemoperfusion, urinary alkalization or multiple dose charcoal in the management of marijuana toxicosis. THC is highly protein bound (97% to 99%) and has a large volume of distribution (10 L/kg, with high lipophilicity), and thus dialysis or hemoperfusion have no theoretical benefit.

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Toxicity is dose-related, however, there is a wide-range of variability among individuals. Patients with hepatic impairment may be more sensitive. A lethal dose has not been established in dogs or cats, but it only takes a small amount to cause clinical signs. Fortunately death is rare. There are published reports of two dog deaths after ingesting edibles and a 12-week-old ferret after ingesting plant material. If appropriate treatment is implemented, the prognosis is good and no permanent effects should be anticipated.

**Synthetic cannabinoids**

Synthetic marijuana, or more precisely synthetic cannabinoids, are chemicals synthesized in laboratories and mimic the effects of delta-9-tetrahydrocannabinol (THC), the main psychoactive ingredient in marijuana. They may also be called cannabinoid receptor agonists or tetrahydrocannabinol (THC) homologs. These compounds are not structurally related to classic cannabinoids or THC. However, they primarily interact with the CB1 receptors within the CNS, resulting in cannabis-like effects. Synthetic cannabinoids have shown a higher binding affinity for receptors than THC. They have been divided into 7 major groups based on their chemical structure:

- Naphthyoylindoles (e.g. JWH-018, JWH-073 and JWH-398)
- Naphthylmethylindoles
- Naphthoylpyrroles
- Naphthylmethylindenes
- Phenylacetylindoles (i.e. benzoylindoles, e.g. JWH-250)
- Cyclohexylphenols (e.g. CP 47,497 and homologues of CP 47,497)
- Classical cannabinoids (e.g. HU-210)

These compounds are sprayed on plant material and sold as potpourri or herbal incense labeled "not for human consumption." These products are often referred to as “herbal highs” or “legal highs” because of their hazy legal status and purported natural herbal make-up. They may also be sold as a liquid for use in e-cigarette type vaporizers. These products are available through the internet, gas stations, liquor stores, and head shops (see Table 1 for common brand names).

These compounds first began appearing in Europe in 2004 and have since come to the United States. Their original use was as a research tool to investigate analgesic and anti-inflammatory properties of cannabinoids but without the psychotropic effects. These substances have no accepted medical use in the United States.

There have been over 40 different synthetic cannabinoids identified and classified as Schedule 1 controlled substances by the DEA. This makes it illegal to manufacture, distribute, possess, import, or export these cannabinoids. As the older generations of synthetic cannabinoids are made illegal, new ones take their place. These ‘new synthetic cannabinoids’ may use the same or similar product labels while having a higher intensity or longer-lasting high putting the user at more risk. Other contributing factors to synthetic cannabinoid toxicosis include potential unidentified contaminants (e.g., sympathomimetics), or bath-to-bath variability of an individual THC homolog among products. The specific composition of these products is constantly changing as individual chemical constituents are banned. In addition, there is very little known regarding the herbal mixtures used as the delivery vehicle; the herbs themselves may also have additive psychoactive properties.

Use of synthetic cannabinoid receptor agonists has been associated with adverse psychiatric, cardiovascular, renal, pulmonary, and neurologic effects in humans. Clinical signs in dogs are similar to traditional marijuana exposures: ataxia, lethargy, hypothermia, bradycardia, vomiting, urinary incontinence, hyperesthesia, mydriasis and disorientation. However, cardiac signs appear to be more prevalent and seizures have been reported. One fluorinated synthetic cannabinoid (XLR-11) has been associated with AKI in humans.

Treatment for synthetic cannabinoids is the same as for marijuana. Monitor respiratory function, heart rate, blood pressure and body temperature. In severe cases, intralipids may be helpful. No specific CBC or chemistry profile abnormalities are expected.

The toxic dose is variable, depending on the specific THC homolog. The duration of effects can be shorter or longer than THC when smoked, but there is no kinetic information about ingestion of these products.

So why would a person chose synthetic cannabinoids over the real thing? In many cases it has to do with drug testing. The typical screening tests on blood and urine samples will be false negative. This is due to a difference in metabolites. However, some private labs have now developed tests to detect the metabolites of certain cannabinoids in blood or urine.

**Cannabinoids**

Cannabinoids (CBDs) do not have the psychoactive properties of THC and have been suggested for pain control and appetite stimulation in pets. CB2 selective agonists have been shown to be effective in the treatment of pain, various inflammatory diseases, and osteoporosis in humans. CBD even has anticonvulsant-activity. At this point more research is needed in this area. There is no known appropriate dose for pets and the ASPCA Animal Poison Control Center has had dogs develop the same signs as THC ingestion after ingesting CBD only products (quality control?).

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Salvia divinorum
Salvia is a plant in the mint family that has been used historically for religious rituals and herbal healing. Salvia contains salvinorin A which is hallucinogenic. In a double blind, placebo-controlled trial, healthy humans experienced hallucinations without a significant change in HR or BP. In humans with inhalation exposures, hallucinations occur within seconds and may last for 30 minutes. There is some absorption through the oral mucosa, but hallucinogenic effects are not seen after oral ingestion. Salvia is not scheduled by the DEA but some states have restrictions. ASPCA APCC data shows dogs exposed to Salvia divinorum develop signs similar to TCH (anxiety, ataxia, bradycardia, hyperthermia, nausea, panting, sedate, urinary incontinence).

Table 1. Common names of synthetic cannabinoid products

<table>
<thead>
<tr>
<th>Spice</th>
<th>K2</th>
<th>Skunk</th>
<th>Moon Rocks</th>
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<td>Black Magic</td>
<td>Spike</td>
<td>Mr. Nice Guy</td>
<td>Exclusive</td>
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<td>Sensation</td>
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<td>Blaze</td>
<td>Red x Dawn</td>
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<td>Fire</td>
<td>Crazy Clown</td>
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<td>Scooby Snax</td>
<td>Bizzaro</td>
<td>Silver</td>
<td>Aroma</td>
<td>Genie</td>
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References
Toxicity of Human Medications
Tina Wismer, DVM, DABT, DABVT
ASPCA Animal Poison Control Center
Urbana, IL

Aspirin
Aspirin (acetylsalicylic acid, ASA) is the salicylate ester of acetic acid and is a weak acid derived from phenol. It is available as tablets and capsules (65, 81, 325, and 500 mg), powders, effervescent tablets and oral liquid preparations. Aspirin reduces pain and inflammation by reducing prostaglandin and thromboxane synthesis through inhibition of cyclooxygenase. At very high doses, aspirin and other salicylates uncouple oxidative phosphorylation leading to decreased ATP production. Salicylates also affect platelet aggregation.

Aspirin is rapidly absorbed from the stomach and proximal small intestine. Aspirin is metabolized in the liver and excreted through the urine. The elimination half-life increases with the dose. Cats are deficient in glucuronyl transferase and have prolonged excretion due to decreased metabolism. Elimination is also slower in neonates and geriatric animals.

Signs may include vomiting (+/- blood), hyperpnea, respiratory alkalosis, metabolic acidosis, gastric hemorrhage, central lobular liver necrosis, and bleeding diathesis. Fever and seizures may be seen due to the uncoupling of oxidative phosphorylation. Renal insufficiency is uncommon with salicylate toxicoses.

Emesis can be performed in the asymptomatic animal, unless contraindicated. Activated charcoal adsorbs aspirin and repeated doses may be used with large ingestions. A cathartic should be used, unless the animal is dehydrated or has diarrhea. Liver values, glucose, acid base status and electrolytes should be monitored. Maintain hydration and start GI protectants (sucralfate, H2 blockers, +/- misoprostol, +/- omeprazole). Gastric protectants should be continued for 5 - 7 days, longer in the symptomatic patient. Antiemetics should be used to control vomiting. Alkalization of the urine results in ion trapping of salicylate in the kidney tubule and increases its secretion. Ion trapping should only be used in cases where the acid base balance can be monitored. Assisted ventilation and supplemental oxygen may be required if the animal is comatose. Seizures should be treated with diazepam. Fluids, whole blood, and electrolytes may be needed to control hypotension and hemorrhage, manage acute bleeding ulcers, and correct electrolyte abnormalities. Acid base imbalances should be corrected. Hyperpyrexia should be treated conservatively as aggressive cooling (ice baths or cold water enemas) may result in hypothermia. Prognosis is good if the animal is treated promptly and appropriately. The development of hepatic necrosis is considered to have a poor prognosis. With hepatic damage, treatment may need to be continued for weeks.

Other salicylates
Salicylates are found in many products. Bismuth subsalicylate (Pepto-Bismol®, Kaopectate®) contains 9 mg of salicylate in 1 ml (2 tablespoons = 325 mg aspirin). Topically applied salicylates (arthritis, psoriasis, teething, wart removal) can be absorbed through the skin and cause systemic problems. Oil of wintergreen is used as a flavoring for candy and contains approximately 98% methyl salicylate.

Acetaminophen
Acetaminophen (Tylenol®, non-aspirin pain reliever, APAP) is a synthetic non-opiate derivative of p-aminophenol. Acetaminophen is rapidly and almost completely absorbed from the GI tract. Peak plasma levels are seen at 10-60 minutes (60-120 min for extended release). Two major conjugation pathways are used to metabolize APAP by most species (P-450 metabolism followed by glucuronidation or sulfation). APAP-induced hepatotoxicity is due to the formation of the oxidative metabolite, N-acetyl-para-benzoquinoneimine (NAPQI). Glutathione can conjugate and neutralize NAPQI, but when glutathione stores are depleted, NAPQI binds to sulphydryl groups on the hepatic cell membrane and damages the lipid layer. Another metabolite, PAP (para-aminophenol), appears to be responsible for methemoglobinemia and Heinz body formation.

Methemoglobin values increase within 2-4 hours, followed by Heinz body formation. Clinical signs include depression, icterus, vomiting, hypothermia, methemoglobinemia, facial or paw edema, death, dyspnea, and hepatic necrosis. Liver necrosis is less common in cats than in dogs. Clinical signs of methemoglobinemia may last 3-4 days. Hepatic injury may not resolve for several weeks. Early decontamination is most beneficial. Emesis is usually unrewarding. Activated charcoal adsors APAP and a cathartic should also be used, unless the animal is dehydrated or has diarrhea. Monitor liver values and for the presence of methemoglobinemia. ALT, AST and bilirubin may rise within 24 hours after ingestion and peak within 48 to 72 hours.

Symptomatic patients need initial stabilization, including oxygen if dyspneic. Treatment involves replenishing the glutathione stores and converting methemoglobin back to hemoglobin. N-acetylcysteine (Mucomyst®, NAC) is a precursor in the synthesis of glutathione and can be oxidized to organic sulfate providing sulphydryl groups that bind with APAP metabolites to enhance elimination. An initial oral loading dose of 140 mg/kg (dilute to 5% in dextrose or sterile water) is given, followed by 70 mg/kg PO QID for 7 treatments, or longer if still symptomatic. Fluid therapy is used to correct dehydration and for maintenance needs, not for
diuresis. Whole blood transfusion may be necessary to increase oxygen carrying capacity, but cats must be monitored for volume overload. Ascorbic acid provides a reserve system for the reduction of methemoglobin back to hemoglobin; however, ascorbic acid has questionable efficacy and may irritate the stomach. Cimetidine is an inhibitor of cytochrome p-450 oxidation system but takes several days to become effective and should be avoided in cats. It has now been demonstrated that cimetidine blocks one of the only pathways that cats have to convert methemoglobin back to hemoglobin. For hepatic injury, s-adenosylmethionine (SAMe, Denosyl-SD4®) at 20 mg/kg/day shows a positive effect for treatment of APAP toxicosis. Prognosis is good if the animal is treated promptly. Animals with severe signs of methemoglobinemia or with hepatic damage have poor to guarded prognosis.

Ibuprofen
Ibuprofen (Motrin®, Advil®, etc.) is a nonsteroidal anti-inflammatory agent. Ibuprofen inhibits prostaglandin synthesis by blocking the conversion of arachidonic acid to various prostaglandins. Ibuprofen decreases secretion of the protective mucous layer in the stomach and small intestine and causes vasoconstriction in gastric mucosa. Ibuprofen inhibits renal blood flow, glomerular filtration rate, tubular ion transport, renin release and water homeostasis. Ibuprofen may also affect platelet aggregation and possibly hepatic function. Serious hepatotoxicosis is not a common problem with ibuprofen. Absorption of ibuprofen is rapid (0.1 to 1.5 h). Plasma half-life in the dog has been reported to be 2-2.5 hours, but the elimination half-life is considerably longer. Ibuprofen is metabolized in the liver and undergoes significant enterohepatic recirculation before being excreted in the urine. The onset of GI upset is generally within the first 2-6 hours after ingestion, with GI hemorrhage and ulceration occurring 12 hours to 4 days post ingestion. Renal failure often occurs within the first 12 hours after massive exposure to an NSAID but may be delayed for 3-5 days.

Emesis can be performed in the asymptomatic animal. Activated charcoal adsorbs ibuprofen and a cathartic should also be used, unless the animal is dehydrated or has diarrhea. GI protectants are very important. A combination of misoprostol, H2 blockers, sucralfate and omeprazole can be used to manage and/or prevent gastric ulcers. Animals should be started on IV fluids at twice maintenance for 48 hours if renal failure is expected. Monitor BUN, creatinine, and urine specific gravity (baseline level, 24, 48, and 72 h). Acid-base disturbances are rare and usually transient. Fluids, whole blood, inotropic agents, and electrolytes should be given to control hypotension and hemorrhage, maintain renal function, and correct electrolyte abnormalities. Assisted ventilation and supplemental oxygen may be required if animal is comatose. Prognosis is good if the animal is treated promptly and appropriately. Gastrointestinal ulceration usually responds to therapy. Acute renal insufficiency resulting from ibuprofen administration has been considered reversible.

Opioids and opiates
There are many opioids and opiates used in human and veterinary medicine. Opioids and opiates are synthetic or natural compounds derived from the opium poppy, *Papaver somniferum*, and are generally classified (agonist or partial agonist) by their ability to exert effects at the different opioid receptors (mu, kappa, delta, sigma). Partial agonists are agonists at one (or more receptors) and antagonists at others. Opioids act centrally to elevate the pain threshold and to alter the psychological response to pain. Most of the clinically used opioids exert effect at the mu receptor (mu1 subtype mediates analgesic effects, mu2 mediates respiratory depression).

Opioids are well absorbed from the GI tract, but bioavailability is variable as some opioids have a large first pass effect (i.e. fentanyl). These opioids are administered in other manners (CRI, buccal, transdermal) to reach therapeutic blood levels. Metabolism varies, but opioids generally undergo hepatic metabolism (conjugation, hydrolysis, oxidation, glucuronidation, or dealkylation). This glucuronidation may account for the sensitivity of cats (who are deficient in glucuronyl-S-transferase) to opioids.

In dogs, CNS signs include depression, ataxia, and seizures. Respiratory depression, vomiting, bradycardia, and hypotension may be seen. Cats may show excitatory behavior and urinary retention. Detection of opioids can be made from urine or serum samples.

Treatment in an asymptomatic animal may include emesis if the ingestion is recent. Activated charcoal with cathartic should be administered and the patient monitored for up to 12 hours. If the animal becomes symptomatic, naloxone (0.1-0.2 mg/kg IV, IN, IM or SQ) can be administered. As the duration of action of naloxone is much shorter than that of the opioids, repeat dosages may be necessary. Partial agonists/antagonists (i.e. butorphanol) may be used to partially reverse pure agonists if no naloxone is available. Monitor temperature, cardiac function and blood gases. Treatment times will vary with the half life of the opioid. If respiratory and cardiovascular function can be maintained then prognosis is good. For those cases that are seizing, prognosis is guarded.
<table>
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<tr>
<th>DRUG</th>
<th>ACTIVITY</th>
<th>Mu</th>
<th>Delta</th>
<th>Kappa&lt;sub&gt;1&lt;/sub&gt;</th>
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<td>Antagonist</td>
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(P = partial)

**Fentanyl**

Fentanyl suckers, lozenges and transdermal patches are becoming more frequently used in both human and veterinary medicine. The lozenges or suckers contain fentanyl citrate in a sucrose and liquid glucose base and are attractive to animals. The patches have poor absorption from the GI tract, but can be absorbed transmucosally while the animals are chewing on them. Signs are similar to other opioids with depression, bradycardia, hypotension, weakness, and pallor predominating. Treatment is as for other opioids.

**Selective serotonin reuptake inhibitors (SSRIs)**

Selective serotonin reuptake inhibitors (SSRIs) all differ structurally, but have the same ability to inhibit presynaptic neuronal reuptake of serotonin. Drugs in this class include fluoxetine (Prozac®), paroxetine (Paxil®), sertraline (Zoloft®), fluvoxamine (Luvox®), citalopram (Celexa®) and escitalopram (Lexapro®). They have little to no effect on non-serotonin neurotransmitters and thus have less anticholinergic, sedative and cardiovascular side effects than other types of antidepressants. The most common signs of overdose are depression, vomiting, anorexia, ataxia, muscle tremors, arrhythmia (tachycardia and bradycardia are possible), and hypertension. The term serotonin syndrome has been used to describe multiple signs associated with severe SSRI toxicosis, including agitation, tremors, tachycardia, and hyperthermia. Other less common signs include diarrhea, salivation, mydriasis, seizures, nystagmus, and coma.

Emesis should only be attempted with recent exposures, assuming that the patient is asymptomatic. Gastric lavage may be considered if large numbers of pills were ingested. Activated charcoal with a cathartic should be administered and may be effective several hours after exposure. Treatment consists of monitoring vital signs closely, controlling clinical signs and providing appropriate supportive care. Diuresis does not enhance excretion because SSRIs are highly protein bound, but fluid therapy should be considered to help support blood pressure and maintain renal function. Diazepam can be used to control seizures and treatment of CNS signs may also help in the control of some of the other signs such as tachycardia, hypertension, and hyperthermia. Propranolol may be used to counter tachycardia. Cyproheptadine, in addition to being an antihistamine and an appetite stimulant, is a non-selective serotonin reuptake inhibitor. Chlorpromazine or acepromazine can be used in addition to cyproheptadine to treat agitation.

**Venlafaxine**

Venlafaxine (Effexor®) is a bicyclic antidepressant; it is a potent serotonin and noradrenaline reuptake inhibitor as well as a weak dopamine reuptake inhibitor. Drugs in this class include fluoxetine (Prozac®), paroxetine (Paxil®), sertraline (Zoloft®), fluvoxamine (Luvox®), citalopram (Celexa®) and escitalopram (Lexapro®). They have little to no effect on non-serotonin neurotransmitters and thus have less anticholinergic, sedative and cardiovascular side effects than other types of antidepressants. The most common signs of overdose are depression, vomiting, anorexia, ataxia, muscle tremors, arrhythmia (tachycardia and bradycardia are possible), and hypertension. The term serotonin syndrome has been used to describe multiple signs associated with severe SSRI toxicosis, including agitation, tremors, tachycardia, and hyperthermia. Other less common signs include diarrhea, salivation, mydriasis, seizures, nystagmus, and coma.

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**Amphetamines and related compounds**

Amphetamines can be found in both prescription ADHD and weight loss medications (Ritalin®, Adderall®, Vyvanse®, Concerta®), as well as illicit substances (methamphetamine, crack). Pseudoephedrine is found in cold and allergy medications. Amphetamines and pseudoephedrine are sympathomimetic alkaloids. They stimulate alpha- and beta-adrenergic receptors, causing the release of endogenous catecholamines at synapses in the brain and heart. This stimulation causes peripheral vasoconstriction and cardiac stimulation resulting in hypertension, tachycardia, ataxia, agitation, tremors, and seizures.

Asymptomatic animals may have emesis induced and activated charcoal administered. Fluid therapy is important to enhance elimination and maintain CV stability. Agitation, hyperactivity, and tremors tend to respond best to phenothiazines. Diazepam can worsen dysphoria. Because part of the syndrome is related to serotonin excess, cyproheptadine has been used to manage some of the CNS effects. If tachycardia persists, propranolol may be used. Signs may last up to 48-72 hrs in severe cases.

**Albuterol**
Albuterol (Proventil®, Ventolin®) is a synthetic sympathomimetic amine with primarily beta-2 receptor agonist properties. It is used most commonly for the treatment of asthma. Albuterol binds to beta-2 receptors on the surface of the smooth muscle cells in many different tissues as well as in skeletal muscle, liver and cardiac tissue. Binding to the receptor initiates the conversion of ATP to cyclic AMP, which mediates a variety of intercellular responses resulting in smooth muscle relaxation, increased skeletal muscle contractility and an intracellular shift of potassium. Overdoses of albuterol may lead to effects of beta-1 stimulation, including increased inotropic and chronotropic effects on the heart.

Dogs are usually exposed by chewing on inhalers but there are also solutions, syrups, powders, tablets, and extended release tablets available. When inhalers are punctured, dogs get an inhalation plus an oral exposure. This leads to a quick onset of signs and prolonged duration signs. When inhaled, signs can begin in five minutes. Ingestions usually have a lag time of 30 minutes before clinical signs start. In dogs, signs generally resolve within 12 hours except for certain individuals who may experience signs for up to 48 hours. The most common signs seen are tachycardia, vomiting, depression, tachypnea, hyperactivity, muscle tremors, hypokalemia, and weakness. Rarely, death has been reported.

Decontamination is not advised for inhaler, solution or syrup exposure due to rapid absorption and onset of actions. Emesis (if within minutes of ingestion) and activated charcoal advised with tablet ingestion only (especially extended relief tablets). Vital signs, heart rate and rhythm, and serum potassium levels should be monitored closely for at least the first 12 hours post-exposure and longer if clinical signs persist.

Propranolol or other non-selective beta blockers should be administered if heart rates greater than 160 to 180 bpm are observed. Propranolol slows the heart rate, has direct myocardial depressant effects and helps normalize serum potassium levels. Potassium may be supplemented as needed and should be considered if serum potassium levels fall below 2.5 mEq/l. Animals with known or underlying cardiac disease may be at risk for decompensation and sudden death. Agitation can be treated with diazepam or low dose acepromazine. Prognosis in most cases is very good.

Imidazoline decongestants
Naphazoline (Clear Eyes), tetrahydrozoline, oxymetazoline (Afrin), and xylometazoline (Neo-Synephrine) are imidazoline decongestants. They are vasoconstrictors used for the symptomatic relief of rhinitis, sinusitis, or conjunctival inflammation. Imidazolines are sympathomimetic agents with primary effect on alpha-adrenergic receptors. There is little if any effect on beta-adrenergic receptors. Overdose or intoxication from oral ingestion or excessive topical administration can result in severe drowsiness with diaphoresis, hypotension or shock, bradycardia, respiratory depression, and coma.

Imidazoline decongestants are readily absorbed via the gastrointestinal tract. Most cases result when a dog punctures or chews a container and ingests the contents. Signs of intoxication may include (with decreasing frequency): vomiting, bradycardia, cardiac arrhythmias, poor capillary refill time, hypotension or hypertension, panting, upper respiratory sounds, depression/weakness/drowsiness, nervousness, hyperactivity and shaking. These signs are expected to be present within 30 minutes to 4 hours post exposure.

Emesis is generally not practical due to very rapid absorption and onset of clinical signs. Monitor heart rate and level of alertness. If no signs within 3-4 hours, do not expect to see any. Assess heart rate, rhythm and blood pressure and consider EKG if indicated. Administer intravenous fluids. If there is a significant decrease in heart rate, give atropine at a pre-anesthetic dose. Atropine may not raise the heart rate, but its use was followed by a significant rise in blood pressure. Give diazepam if significant nervous effects (apprehension, anxiousness, shaking) are present. If signs persist for several hours, assess serum electrolytes (potassium, sodium, chloride) and correct them as needed. Since this is an alpha-adrenergic agent, it is reasonable to consider using an alpha antagonist like yohimbine or atipamezole to reverse the hypotension and bradycardia.

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**Toxicity of Veterinary Medications**

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ASPCA Animal Poison Control Center
Urbana, IL

**Phenylpropanolamine**
Phenylpropanolamine (PPA, Proin®) is a sympathomimetic agent used in veterinary medicine for controlling urinary incontinence in dogs. Signs can be seen at therapeutic doses in some dogs and serious signs appear at doses above 20 mg/kg. Signs include tachycardia, hypertension, panting, excitement/hyperesthesia, piloerection, tremors, and seizures. Reflex bradycardia may occur secondary to the hypertension. Signs normally start within 30-90 minutes and may continue up to 48 hours, depending on dose. Emesis may be induced if the ingestion was within 10-15 minutes. Activated charcoal should be given if possible. Heart rate and blood pressure should be closely monitored. Nitroprusside or other pressor agents can be used to manage hypertension. Atropine is contraindicated in the management of bradycardia as it will worsen the hypertension. Phenothiazines may be used to control hyperesthesia and excitement. Animals should be put on IV fluids to promote excretion, protect renal function and help with thermoregulation. As with other stimulants, cyproheptadine may be given if signs of serotonin syndrome develop.

**Chewable NSAIDS**
Chewable NSAIDs are commonly ingested by both dogs and cats. They inhibit prostaglandin synthesis by blocking the conversion of arachidonic acid to various prostaglandins. NSAIDs decrease secretion of the protective mucous layer in the stomach and small intestine and cause vasoconstriction in gastric mucosa. They also inhibit renal blood flow, glomerular filtration rate, tubular ion transport, renin release and water homeostasis. NSAIDs may also affect platelet aggregation and possibly hepatic function. Serious hepatotoxicosis is more commonly seen with chronic dosing. The absorption of NSAIDs are rapid and plasma half-life will vary with the medication. Half-lives are generally longer in the cat and they are considered to be more sensitive to the adverse effects. Most NSAIDs are metabolized in the liver and undergo enterohepatic recirculation before being excreted in the urine. Geriatric animals and neonates, as well as animals with acute renal insufficiency, liver disease and hypoalbuminemia are at higher risk of toxicosis. Administration of NSAIDs combination with glucocorticoids, salicylates, or other NSAIDS could potentiate the adverse effects of these drugs.

Emesis can be performed in the asymptomatic animal. Activated charcoal adsorbs NSAIDs and may need to be repeated (enterohepatic recirculation). GI protectants are very important. A combination of misoprostol, H2 blockers, sucralfate and omeprazole can be used to manage and/or prevent gastric ulcers. Animals should be started on IV fluids at twice maintenance for 48 hours (or more) if renal failure is expected. Monitor BUN, creatinine, and urine specific gravity (baseline level, 24, 48, and 72 h). Acid-base disturbances are rare and usually transient. Dialysis may be necessary if unresponsive oliguric or anuric renal failure develops.

Fluids, whole blood, inotropic agents, and electrolytes should be given to control hypotension and hemorrhage, maintain renal function, and correct electrolyte abnormalities. Assisted ventilation and supplemental oxygen may be required if animal is comatose. Seizures should be treated with diazepam. Prognosis is good if the animal is treated promptly and appropriately. Gastrointestinal ulceration usually responds to therapy. Acute renal insufficiency resulting from ibuprofen administration has been considered reversible, but development of papillary necrosis is generally considered irreversible.

**Carprofen**
Dogs can develop GI ulcers at 20 mg/kg and acute renal failure at 40 mg/kg. Cats develop ulcers at 4 mg/kg and ARF at 8 mg/kg.

**Deracoxib**
Dogs can develop GI ulcers at 15 mg/kg and acute renal failure at 30 mg/kg.

**Pimobendan**
Pimobendan (Vetmedin®) is a selective phosphodiesterase (PDE) III inhibitor with positive inotropic/vasodilator ("inodilator") effects. Pimobendan and its metabolite UD-CG 212 have a dual effect. They increase sensitivity to calcium in cardiac muscle which has a positive inotropic effect. They also increase cAMP levels resulting in vasodilation. Pimobendan is used for management of congestive heart failure in dogs due to AV valvular insufficiency or dilated cardiomyopathy. The therapeutic dose of pimobendan is 0.5 mg/kg divided BID and peak plasma levels are reached within 1-4 hours. The drug and its active metabolite have a short half-life and not detectable in the plasma at 4 and 8 hours, respectively, after dosing. Overdose effects can include hypotension and tachycardia with vomiting seen at any dose. Symptomatic care includes IV fluids to control hypotension. If no response, pressor agents can be used.
Methionine
Methionine is an essential amino acid often found in veterinary urinary acidifiers. Because the formulation is often very palatable, animals may ingest these medications in great quantity. Animals with underlying hepatic insufficiency are at greater risk. Doses greater than 300 mg/kg can cause clinical signs. Signs of toxicity include ataxia, depression, lethargy, salivation, vomiting, metabolic acidosis and hepatic encephalopathy type signs (restlessness, circling, seizures, aggression, blindness, coma). Deaths are rare. Recent evidence suggests the homocysteine metabolites produced in the liver and other organs are the cause of the CNS effects. If large amounts are ingested, emesis and activated charcoal should be implemented. Monitor acid-base status. Cats can develop methemoglobinemia and Heinz-body hemolytic anemia (more commonly seen with chronic dosing). Signs can last for up to 24 hours. The prognosis is excellent if clinical signs are managed.

Avermectins
Avermectins include ivermectin, milbemycin, selamectin, doramectin, abamectin and moxidectin. In nematodes and arthropods, avermectins bind to glutamate-gated chloride channels causing hyperpolarization by enhancing the movement of chloride ions into the cell. This results in paralysis. In mammals, avermectins cause CNS effects by potentiating the release and binding of GABA in the central nervous system. Doses of ivermectin and moxidectin in heartworm medications are safe for even MDR1 (ABCB1) deficient dogs (Collie-type breeds, Australian Shepherds, etc). Problems arise when owners are giving large amounts to treat dermatologic disorders or give the equine product to their pets. In general, young animals are considered more sensitive to the effect of avermectins due to a less developed blood brain barrier. Ivermectin is well absorbed orally and the half life in the non-sensitive dog is as long as 2-3 days. Enterohepatic recirculation is suspected based on the long half life and extent of fecal excretion (98%) of ivermectin. With the ‘non-sensitive’ breeds of dogs signs may be seen at 2000 mcg/kg, but only 150 mcg/kg is needed in the ‘sensitive’ breeds to cause signs. Cats have demonstrated clinical signs at the "therapeutic dose" of 200 mcg/kg. Moxidectin is a semi-synthetic avermectin that is much more lipid soluble than ivermectin. Therapeutic levels of moxidectin have been measured 30 minutes post oral exposure. Moxidectin has a wide margin of safety in dogs when given orally. Doses of up to 300 times the therapeutic dose (300 mcg/kg) resulted in little to no side effects. Most problems are encountered when dogs ingest horse dewormer.

The most common clinical signs of avermectin toxicosis include: depression, weakness, recumbency, ataxia, and coma. Other reported signs include tremors, seizures, transient blindness, bradycardia, and hyperthermia. If the exposure has just occurred and the animal is asymptomatic induce vomiting (if an oral overdose) or consider surgical debridement if given SQ and can localize injection site in massive overdoses. If the animal is symptomatic, treatment is mostly supportive care and repeated dosages of activated charcoal. Activated charcoal/cathartic should be given q 8-12 hours (sorbitol 70% -cathartic of choice) until normal. Intralipids can be given, however efficacy is greater with moxidectin due to its higher lipid solubility. Treatment can take days to several weeks. Supportive care is very important (fluids, parenteral nutrition, frequent turning, etc.). Physostigmine can be given, but it is not an antidote. Physostigmine has a very short beneficial effect (arousal for 30-90 minutes) and should only be used in severely non-responsive dogs (not recommended for cats). The recommended dose is 0.05 mg/kg IM or IV (very slow, over 5 minutes). Prognosis depends on the speed of onset of clinical signs, the faster the onset, the worse the prognosis.

Spinosad
Spinosad is a tetracyclic macrolides anti-parasitic. It can cause vomiting and ataxia, however, if spinosad is given in conjunction with high dose ivermectin, avermectin toxicosis can develop.

Piperazine
Piperazine is an over the counter anthelminthic (roundworms only). The therapeutic dose for dogs and cats is 45-110 mg/kg PO. Signs can occur at therapeutic doses. The most common signs include vomiting, ataxia and tremors. Signs start within the first 24 hours and can last for several days. Animals should be kept in a dark quiet area with fluid support.

Amitraz
Amitraz is a centrally acting alpha adrenergic agonist with some peripheral alpha 1 and alpha 2 activity. It can be found in some veterinary dips (Mitaban®, Taktic®) and tick collars (Preventic®). Amitraz has low dermal absorption, but rapid oral absorption. Clinical signs include sedation, ataxia, vomiting, ileus, bradycardia and hypotension. Hyperglycemia can occur due to suppression of insulin release. Cats and young animals are at increased risk. Clinical signs can be reversed with α-2 antagonists (yohimbine or atipamezole). Treatment also includes a bath if the exposure was dermal. If a collar is ingested, emesis, bulking the diet or endoscopy can be attempted.

Permethrin and other concentrated pyrethrins
Permethrin is a synthetic type 1 pyrethrin. Permethrin is found in shampoos, dips, foggers, spot-ons, and sprays. Permethrins appear to be relatively safe in dogs. Smaller dogs seem to have a greater risk of toxicity and skin hypersensitivity reactions to the spot-ons. Skin
reactions can be treated with bathing +/- antihistamines or steroids. Cats are more sensitive to the toxicity of pyrethroids. The low concentration products (sprays, foggers) contain 0.05-0.1% of permethrin and do not seem to cause the signs that the concentrated (45-65% permethrin) spot-ons do. Permethrin toxicity usually occurs when the owner applies the dog product to the cat; however, cats which actively groom or engage in close physical contact with recently treated dogs may also be at risk of toxic exposure. Clinical signs of permethrin toxicity in cats include hypersalivation, depression, muscle tremors, vomiting, anorexia, seizures, and possibly death. Onset of clinical signs is usually within a few hours of exposure but may be delayed up to 24 hours. The severity of clinical signs varies with each individual. Treatment recommendations include bathing with liquid dish washing detergent and controlling the tremors. Methocarbamol works best to control the tremors. If no injectable methocarbamol is available, the oral form may be dissolved in water and given rectally. If the cat is actively seizing, barbiturates or inhalant anesthesia may need to be used. Permethrins appear to have no direct action on the liver or kidneys, but fluids may be needed to help protect kidneys from myoglobin breakdown products in actively tremoring cats. Prognosis for mildly trembling cats is usually good, but treatment may last 24-48 hours.

**Essential oils**

Essential oils have been used for flea control. D-limonene is a derivative of citrus pulp. This essential oil has minimal to moderate efficacy to control fleas. If diluted properly, this product has a high margin of safety. Application of the undiluted product can cause skin and oral irritation, lethargy, vomiting, salivation, ataxia and muscle tremors. Essential oils can penetrate the skin and cause peripheral vasodilation leading to hypotension and hypothermia. Melaleuca oil is an essential oil from the Australian tea-tree, *Melaleuca alternifolia*. It does have antibacterial and antifungal properties but the efficacy of this agent to repel or kill fleas has not been established. Inappropriate application of products not intended for topical use may result in ataxia, weakness, tremors and depression. Pennyroyal oil is derived from the leaves and flowers of the pennyroyal, squaw mint, or mosquito plants. Pennyroyal oil contains a volatile compound called pulegone, which is responsible for the toxic effects of the plants. The effectiveness of pennyroyal oil to kill fleas is unknown; however, toxicity has been reported. Exposure to pennyroyal oil may induce depression, vomiting, hepatic necrosis, diarrhea, epistaxis, seizures, and death.

Toxicity is dose-related and the possibility of severe signs is more likely if the pure oil is applied to the pet. Cats appear to be more sensitive than dogs to any of the essential oils. Treatment recommendations include bathing with liquid dish washing detergent, activated charcoal with cathartic, pain control if needed, body temperature regulation and fluids. Most essential oils have long half lives (days) due to enterohepatic recirculation.

**Metronidazole**

Metronidazole is a synthetic antibacterial and antiprotozoal agent. Signs can be seen with both chronic dosing and acute overdoses. It has been postulated, but not proven, that the neurotoxicity described during metronidazole therapy is related to conversion by gut flora to a neurotoxic thiamine analog. Signs of intoxication associated with metronidazole in dogs and cats include ataxia and nystagmus most commonly. Seizures, tremors, lethargy/depression, vomiting and hypermetria have also been reported. Signs can begin within 1-3 hours with an acute overdose. Treatment includes discontinuation of the medication (if applicable) and administering diazepam. Diazepam seems to decrease the treatment time needed for these cases to resolve. Neurologic symptoms may require days to weeks before resolving.
Baclofen
Baclofen is a centrally acting skeletal muscle relaxant that mimics γ-aminobutyric acid (GABA) within the spinal cord and causes a flaccid paralysis of skeletal muscles. At oral therapeutic levels, baclofen has virtually no CNS effects due to its poor ability to cross the blood brain barrier, but in overdose situations, CNS effects are common. The most common clinical signs of toxicosis are vomiting, ataxia and vocalization/disorientation, but the most life threatening signs are dyspnea, respiratory arrest and seizures. Dyspnea and respiratory arrest are secondary to paralysis of the diaphragm and intercostal muscles.

The onset of clinical signs varies in dogs with signs occurring anywhere from 15 minutes to 7 hours post exposure (average of 1.9 hr). Duration of clinical signs vary from several hours to several days. Signs can continue long after serum baclofen levels have returned to normal due to the slow clearance from the CNS. Dog doses as low as 1.3 mg/kg can cause vomiting, depression and vocalizing. There are no established lethal doses in animals, but per the APCC data base, deaths in dogs have occurred at doses as low as 8 mg/kg.

Due to the rapid onset of clinical signs, emesis should be considered in only the asymptomatic, recently exposed patient. Gastric lavage may be considered with large ingestions, but care must be taken to ensure that anesthesia does not compound CNS depression. Short acting induction agents such as propofol followed by inhalent anesthesia with a protected airway is preferred. All asymptomatic cases should receive activated charcoal with a cathartic. Avoid magnesium-based cathartics (Epsom salts), as they may worsen CNS depression. Exposed animals should be monitored for 12 hours for development of clinical signs.

Ventilatory support is a prime concern and endotracheal intubation and positive pressure mechanical ventilatory support may be needed for an extended time in severe cases. Diazepam is the drug of choice for centrally acting skeletal muscle relaxant induced seizures. Propofol or isoflurane may be considered in cases that are refractory to diazepam. Long acting barbiturates or other agents that produce profound or prolonged CNS depression should be used with care. Cyproheptadine (1.1 mg/kg PO or rectally) has been used successfully to reduce the vocalization/disorientation seen in some animals. Fluid diuresis is used to enhance elimination and maintain blood pressure. Intralipids have been used successfully in early intoxications. The use of CNS respiratory stimulants are of questionable value and experimental studies have failed to consistently produce positive outcomes when flumazenil was used and have potential to cause serious adverse effects (seizures). Prognosis is variable, and can depend on the availability of ventilatory support for depressed patients. Prognosis is more guarded if seizures develop.

Calcium channel blockers (CCB)
Calcium channel blockers (verapamil, diltiazem, nifedipine, etc.) slow the activity of the SA pacemaker as well as conduction through the AV node. They also cause frequency-dependent channel blockade in the AV node so that it is effective in slowing supraventricular arrhythmias. Calcium channel blockers reduce total peripheral resistance, blood pressure, and cardiac afterload. They can also cause negative inotropic effects, but this is rarely of clinical significance.

Calcium channel blockers have a low margin of safety, causing hypotension and dysrhythmias. Bradycardia and AV nodal depression are the most common dysrhythmias, although others are possible. Hyperglycemia, hyperkalemia, hypokalemia, and hypocalcemia are possible. Due to a rapid onset of signs, the induction of emesis may not be appropriate. Standard decontamination practices should be performed in cases of significant exposure. Any dose exceeding the therapeutic dose should be monitored for cardiovascular signs. Fluid replacement and calcium chloride administration may help correct blood pressure and conduction abnormalities. Calcium gluconate (1 ml/10 kg of 10% solution) may be less effective than calcium chloride but can be used. Monitor for hypercalcemia if calcium is supplemented. Atropine and isoproterenol may be used for bradyarrhythmias and may be more effective following calcium administration. If hypotension persists, norepinephrine, neosynephrine, dopamine, dobutamine, or amrinone are recommended. Insulin and dextrose infusions in a canine model improved survival following verapamil overdose. The newest treatment is intralipids. Prognosis is dependent on dosage and response to therapy. Noncardiogenic pulmonary edema has been reported in cases of massive overdose.

Digoxin
Digoxin is a digitalis glycoside that can be found in elixers (0.05 and 0.15 mg/ml), tablets (0.125, 0.25 and 0.5 mg) and capsules (0.05, 0.1 and 0.2 mg). Digitalis-like compounds (cardiac glycosides) are also found in several plants: oleander (Nerium oleander), foxglove (Digitalis purpurea), Kalanchoe sp. and lily-of-the-Valley (Convallaria majalis). These compounds inhibit the myocardial cell membrane Na-K ATPase pump. This inhibition results in increased intracellular sodium concentrations. The sodium must exit by exchanging with extracellular calcium. The sarcoplasmic reticulum binds the excess calcium and uses it to increase contractility. Digitalis is used in the treatment of congestive heart failure, atrial fibrillation or flutter and supraventricular tachycardias.
Absorption following oral administration occurs in the small intestine and is variable dependent on the oral dosage form used. Food may delay, but does not alter, the extent of absorption. Peak cardiac effects are seen in 6-8 hours. Digoxin is distributed widely throughout the body with highest levels found in kidneys, heart, intestine, stomach, liver and skeletal muscle. The half life of digoxin is 14.4 - 56 hours in the dog and 23.8 – 42.8 hours in the cat.

Adverse effects of digitalis glycosides are usually associated with high or toxic serum levels and are categorized into cardiac and extracardiac signs and symptoms. Cardiac effects may include almost every type of arrhythmia described. The more common arrhythmias or ECG changes seen include: complete or incomplete heart block, bigeminy, ST segment changes, paroxysmal ventricular or atrial tachycardias with block, and multifocal PVCs. Extracardiac effects most commonly seen in veterinary medicine include mild GI upset, anorexia, weight loss, depression and diarrhea. Hyperkalemia and hyponatremia are seen with overdosage.

In dogs the acute toxic dose after IV administration has been reported to be 0.177 mg/kg. The minimum lethal dose of cardiac glycosides is not well established, but 0.33 mg/kg orally in a dog was lethal. Drug levels may be available from a human hospital on a "stat" basis. Cats are relatively sensitive to digoxin while dogs tend to be more tolerant of high serum levels.

Emesis can be induced with recent ingestion in an asymptomatic animal. Activated charcoal decreases digoxin absorption up to 96%. In the symptomatic patient supportive and symptomatic therapy should be implemented. IV fluids should be started at maintenance rate, but do not use calcium-containing fluids like Ringer's or LRS. Forced diuresis does not accelerate the elimination of cardiac glycosides and may worsen electrolyte imbalances. Serum electrolytes, arterial blood gases, and continuous ECG monitoring should be instituted. In severe intoxication, monitor serum potassium hourly. The use of specific antiarrhythmic agents in treating life-threatening digitalis-induced arrhythmias may be necessary. Digibind® (Burroughs Wellcome Co., Research Park Triangle, NC) is a specific antagonist to digoxin. It is an immune Fab produced from specific digoxin antibodies from sheep and will bind directly to the drug, inactivating it. It is expensive however and several vials may be needed for treatment. Prognosis is guarded with large ingestions especially in patients with underlying disease and risk factors.

5-Fluorouracil (5-FU) is in the antimetabolite class of antineoplastic agents. The topical creams and solutions (Efudex, Fluoroplex, Adrucil) are extremely toxic if ingested. 5-FU destroys rapidly dividing cells, causing severe vomiting and GI irritation. It is likely that the drug, inactivating it. It is expensive however and several vials may be needed for treatment. Prognosis is guarded with large ingestions especially in patients with underlying disease and risk factors.

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The onset of clinical signs usually occurs within 0.5 to 5 hours following ingestion. 5-FU rapidly distributes to the total body water and it is absorbed by all cells. In dogs that survived, signs lasted from 18 hours to 14 days. The minimum lethal oral dose for the dog is 20 mg/kg, but signs of toxicity are seen as low as 8.6 mg/kg. Often signs begin with vomiting (with or without blood) and progress to tremors and seizures within a few hours. The vomiting isn't always seen before seizures, nor are seizures seen in every case. Seizures may require care for more than 24 hours.

Emesis, activated charcoal and cathartic can be started if the animal is asymptomatic and the ingestion was recent (less than 1 hour). Seizures and tremors are rarely controlled with diazepam. Pentobarbital, phenobarbital, gas anesthetics (isoflurane), and propofol have been used successfully. GI protectants and antiemetics should be started. IV fluids, thermoregulation, antibiotics, and pain control are very important parts of the therapy. If animals live through the severe vomiting and seizures, WBC's could start to decline in 5-20 days. Filgrastim (Neupogen) may be given for neutropenia (5-6 mg/kg SQ). Prognosis is guarded to poor once signs occur. Sixty-four percent of dogs ingesting 5-FU die or are euthanized.

5-HTP

5-hydroxytryptophan (5-HTP, griffonia seed extract) is a precursor of serotonin. 5-HTP is available over-the-counter and is used to treat a variety of disorders including obesity, depression, anxiety, insomnia, PMS, and compulsive gambling. Overdoses of 5 HTP induce "serotonin syndrome" due to overstimulation of serotonin receptors in nervous system, gastrointestinal tract, cardiovascular and respiratory systems.

In dogs the most common clinical signs include: vomiting, diarrhea, seizures, hyperthermia, hyperesthesia, depression, mydriasis, vocalization, death, blindness, hypersalivation, dyspnea, ataxia/paresis, disorientation, hyperreflexia, and coma. Signs are similar, but vary in severity, whether 5-HTP or other serotonergic drugs such as SSRIs or MAOIs are ingested.

Treatment of serotonin syndrome is largely symptomatic and supportive. Inducing vomiting is not recommended if clinical signs are present because of the increased risk of aspiration. Seizures and agitation generally respond to diazepam or phenothiazines (the drug of choice in humans), and barbiturates can be used in refractory cases. Because hyperthermia is a significant concern, cooling measures should be instituted. Diuresis does not enhance excretion, but intravenous fluids should be administered to support the cardiovascular system, aid in thermoregulation, and maintain renal blood flow. The use of cyproheptadine, a nonselective serotonin
antidepressant, has shown to be a helpful adjunct in managing serotonin syndrome in animals. Cyproheptadine may be administered at a dose of 1.1 mg/kg PO (dogs) or 2-4 mg PO (cats). In cases where the oral route is not feasible (e.g. severe vomiting), cyproheptadine may be crushed and mixed with saline to be instilled rectally. Propranolol also has some serotonin blocking effect, and may be of benefit if animals are tachycardic. Metabolic acidosis may occur and can be corrected with sodium bicarbonate as indicated by blood gas analysis. Symptomatic care to control vomiting, abdominal pain, or other signs can be instituted as needed.

**Cholecalciferol**

Cholecalciferol is a Vitamin D₃ analog. It can be found in rodenticides, oral vitamins and dermal preparations for psoriasis (Dovonex®). Cholecalciferol alters calcium metabolism in the body, increasing intestinal absorption and renal tubular reabsorption of calcium and stimulating bone resorption. Clinical signs of intoxication usually develop within 12-36 hours. Early signs include lethargy, weakness, anorexia, polydipsia, polyuria, and vomiting, often with blood. Biochemical alterations include hyperphosphatemia within 12 hours and hypercalcemia within 24 hours of exposure and azotemia (both renal and pre-renal). The elevated calcium levels result in calcification of many tissues, notable renal tubules and walls of blood vessels. The elevated calcium also has a direct effect on kidney function, sometimes causing acute renal failure even without mineralizations.

Diagnosis of toxicosis is based on history of exposure, clinical signs, serum chemistries and urinalysis. Run baseline chemistries as soon as possible after a known exposure. Pursue GI decontamination if within several hours of ingestion, or if there is evidence of ingestion (chewed box) at unknown time but a still asymptomatic animal. Decontamination consists of multiple doses of activated charcoal and possibly cholestyramine. Cholestyramine is an anion exchange resin available by prescription only. It is used as an adjunctive therapy for the lowering of serum cholesterol in patients with primary hypercholesterolemia who have not responded to diet or other measures alone. Cholestyramine is also indicated for use in the relief of pruritus associated with partial biliary obstruction. It has also been used to aid in the treatment of toxicoses in humans (amiodarone, digitoxin, chlordane, methotrexate, piroxicam, pfiesteria toxin, thyroid, Vitamin D, warfarin, blue-green algae, indomethacin).

Cholestyramine binds with bile acids in the intestine, preventing their reabsorption and producing an insoluble complex, which is excreted in the feces. Cholestyramine has been shown to decrease the toxicity of indomethacin in the dog. Animals are dosed at 0.3 – 1 g/kg TID for several days (depends on toxin ingested). The powder should be given before feeding if possible or mixed with canned food. Cholestyramine is not absorbed out of the digestive tract, so it has no systemic effects other than possible constipation. If giving with activated charcoal, alternate q 4 hours.

Treatment for cholecalciferol is aimed at lowering the serum calcium and phosphorus levels if elevated, preventing a rise in these values if still normal, and stopping further calcium mobilization from the bones. IV normal saline at twice maintenance, prednisone and furosemide all enhance calciuria. Monitor serum calcium, phosphorus, BUN and creatinine daily to judge effectiveness of therapy. If calcium levels are rising despite calciuresis, best choice is pamidronate (Aredia™). Unlike salmon calcitonin, it needs to be given only once, with a repeat dose possibly at about 5-7 days. It acts at the level of the osteoclast and is deposited in the bone excreted in the feces. Cholestyramine has been shown to decrease the toxicity of indomethacin in the dog. Animals are dosed at 0.3 – 1 g/kg TID for several days (depends on toxin ingested). The powder should be given before feeding if possible or mixed with canned food. Cholestyramine is not absorbed out of the digestive tract, so it has no systemic effects other than possible constipation. If giving with activated charcoal, alternate q 4 hours.

**Strychnine**

Strychnine is derived from the tree, Strychnos-nux vomica. It is frequently used in malicious poisonings. The approximate lethal dose in dogs is about 0.75 mg/kg. One-quarter ounce (7 gram) of bait has estimated 35 mg of strychnine, enough to kill a hundred-pound dog. Strychnine acts by competitively and reversibly antagonizing glycine at postsynaptic neuron sites in the spinal cord and medulla. Glycine is an inhibitory neurotransmitter, so this results in unchecked reflex stimulation. More powerful extensors predominate, resulting in rigidity.

Signs begin within 10-120 minutes, usually without vomiting. There is anxiety and stiffness, followed rapidly by violent tetanic seizures. Sound or touch can elicit these spells, but external stimuli are not necessary to trigger them. There is a classic saw horse stance and a strained facial grimace. Death results from anoxia, as periods between rigidity decrease. The primary goal of treatment is to prevent asphyxiation. General anesthesia (pentobarbital, inhalents, propofol) will allow control of the airway and you can perform gastric or entero-gastric lavage, followed by AC. IV fluids and forced diuresis enhance elimination. If severe tachycardia continues after the muscle activity is controlled, consider propranolol.

**Zinc phosphide**

Zinc phosphide is an old rodenticide posing as a new one. The phosphide salts are unstable in an acid environment. At gastric pH they degrade rapidly to form phosphine gas. Phosphine gas, when inhaled, results in acute non-cardiogenic pulmonary edema. When absorbed systemically, it is thought to block cytochrome C oxidase, leading to formation of highly reactive oxygen compounds. It is...
these reactive compounds which cause most of the tissue injury, most severe damage is in tissues with the highest oxygen demand – brain, lungs, liver and kidney.

Lethal doses for cattle, sheep, pigs, goats, dogs, and cats range between 20-50 mg/kg. For a 55 pound (25 kg) dog, that would be between 10 grams (0.35 ounce) and 25 grams (just under an ounce) of 5% bait. Severely poisoned animals may die in 3-5 hours. Those who survive longer than 48 hours have a pretty good chance.

Initial signs may vary by species, as well as by the dose. Onset of signs is normally between 15 minutes to 4 hours post ingestion. Vomiting, often with blood, is common. Dogs may show lateral recumbency with whole body tremors and salivation. Other signs may include anorexia and lethargy. Rapid deep breathing may signal the onset of the pulmonary changes. Abdominal pain, ataxia, and weakness leading to recumbency may follow. Hyperesthesia and seizures may develop that resemble the signs of strychnine toxocosis.

Metabolic acidemia ensues. Other biochemical changes may include depressed serum calcium and magnesium. If there is survival beyond 48 hours an elevated blood urea is common. Hepatic and renal damage often may be detected 5-14 days later.

Initial decontamination is tempered by the wish to keep the stomach pH as high as possible to prevent the formation of phosphine gas. If there has been no spontaneous vomiting, it may be better to induce emesis with apomorphine rather than hydrogen peroxide. Giving food, commonly done in order to improve gastric emptying and the response to peroxide, will trigger release of gastric acid and increase the rate of production of phosphine. If you are going to perform gastric lavage, add an alkaizing component like a magnesium and aluminum hydroxide gel to your lavage liquid. Also consider mixing into your activated charcoal preparation.

Supportive care includes IV fluids to maintain blood pressure renal perfusion, and gastroprotectants. Seizures may respond to diazepam, or may require barbiturates or full anesthesia. Since the most severe injury is probably due to action of the oxygen radicals, use of an antioxidant may be useful – consider vitamin C or n-acetylcysteine.

Caution: Phosphine gas released from vomitus or stomach washings can cause significant illness in veterinary personnel assisting animal. Phosphine has been describes as having a spoiled fish or garlic odor. It is detectable at 1-3 ppm in air; maximum allowed in air in occupational situations is 0.3 ppm, so if you can smell it, you are being exposed to a concentration that can be harmful.
Pesticides

*Cholecalciferol* is a Vitamin D₃ analog. It alters calcium metabolism in the body, increasing intestinal absorption and renal tubular reabsorption of calcium and stimulating bone resorption. Clinical signs of intoxication usually develop within 12-36 hours. Early signs include lethargy, weakness, anorexia, polydipsia, polyuria, and vomiting, often with blood. Biochemical alterations include hyperphosphatemia within 12 hours and hypercalcemia within 24 hours of exposure and azotemia (both renal and pre-renal). The elevated calcium levels result in calcification of many tissues, notable renal tubules and walls of blood vessels. The elevated calcium also has a direct effect on kidney function, sometimes causing acute renal failure even without mineralizations.

**Household and industrial**

*Ethylene glycol* (EG) is present in automotive radiator antifreeze, brake fluids, aircraft deicers, condensers, heat exchangers, home solar units and portable basketball goal post bases. Ethylene glycol may also be used to winterize toilets in RVs and summer homes in colder latitudes. Cats, rabbits and humans are the most sensitive to EG, with dogs, cattle, pigs and rodents having an intermediate sensitivity. It is important to remember that EG is a potent alcohol and many of the signs of toxicosis will relate to severe alcohol intoxication. Because of the different mechanisms involved in EG toxicosis, clinical signs frequently change throughout the course of the toxicosis. It is sometimes easier to break the clinical signs into 3 different stages, although considerable overlap between these stages may be seen and some animals will not experience each stage; death can occur at any stage. The stages are 1) neurologic—the initial inebriation due to the effects of alcohol on the CNS, 2) cardiopulmonary—due to severe acidosis and electrolyte disturbances, and 3) renal—due to renal tubular injury from calcium oxalate crystals. Treatment of EG toxicosis must be timely and aggressive. Failure to institute appropriate therapy within the first several hours may result in irreversible renal damage or death of the animal.

*Phenol* (carbolic acid, hydroxybenzene, oxybenzene) is a hydrolyzed form of benzene. Phenols are used for their antiseptic and local anesthetic properties. Dilute phenol solutions (0.1-4.5%) are found in sore-throat lozenges, throat sprays, gargles, gels, ointments, and lotions as a local anesthetic. Phenol destroys the outer layers of skin and is sometimes used as a chemical peel. Phenol is readily absorbed following inhalation, oral and dermal exposure. In dilute solutions, phenol is an irritant and inflammation may be seen at the site of absorption. In concentrations of 5% or more, phenol rapidly denatures all proteins with which it comes into contact. Dermal application of phenol can also cause systemic signs. Large doses can lead to muscle tremors, seizures, coma and death. Mortality associated with dermal exposure to phenol is greatly influenced by the surface area exposed as well as the concentration of the applied solution. Cats are more sensitive to phenol because of their limited glucuronide transferase activity. Oral phenol exposure in animals causes panting, profuse vomiting, diarrhea, salivation, and ataxia, which may progress to gastric ulcers, muscle fasciculations, and methemoglobinemia. Urinalysis abnormalities include albinuria, hematuria, green/black color, and the presence of casts.

*Pine oil* (arizole, oleum abietis, unipine, yarmor) is a component of many household cleaners and disinfectants. Pine-scented formulations contain small amounts of pine oil and have minimal toxicity compared with pure pine oil. Oral and dermal absorption of pine oil is considered to be poor. Pine oils are irritating to the mucous membranes, producing erythema of the oropharynx, mouth, and skin. Ingestion of pine oil may cause vomiting, CNS depression, tachycardia, nephritis, and fever. Less commonly seen signs include diarrhea, hypotension, bradycardia, ataxia, coma, renal failure, and myoglobinuria following large ingestions. Pulmonary toxicity may be caused by either aspiration, or chemical pneumonitis resulting from absorption of pine oil from the GI tract with subsequent deposition in the lung. Cats are deficient in certain types of glucuronyl transferase activity, making them more susceptible than other species to pine oil toxicoses. A cat that ingested about 100 ml of Pinesol® (20% pine oil, 10.9% isopropanol) developed severe depression, ataxia, unresponsive pupils, and died. Autopsy revealed pulmonary edema, acute centriflobular hepatic necrosis, and total renal cortical necrosis.

Pharmaceuticals

Nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit prostaglandin synthesis by blocking the conversion of arachidonic acid to various prostaglandins. Decreased prostaglandins mean decreased pain but also decreased secretion of the protective mucous layer in the stomach and small intestine and vasoconstriction in gastric mucosa. NSAIDs inhibit renal blood flow, glomerular filtration rate, tubular ion transport, renin release and water homeostasis. NSAIDs have a narrow margin of safety. GI ulcers and renal failure can be seen after an acute ingestion. Cats are thought to be twice as sensitive as dogs due to their limited glucuronyl-conjugating capacity.

There are many other renal toxic pharmaceuticals including: *sulfonamides, tetracyclines, amphotericin B, cisplatin* and most of the heavy metal *chelators*. *Alpha lipoic acid* is a neutreaceutical that can also cause renal failure.
Metals

**Arsenic** can be found in some fungicides, herbicides, pesticides and ashes from chromated copper arsenate (CCA) treated lumber. Arsenic interferes with a variety of enzyme systems within the body, resulting in disruption of cellular homeostasis that can result in peracute death within hours of exposure. Arsenic is readily absorbed via ingestion and has a predilection for skin, nails, hooves, feathers, sweat glands and hair. Bloody vomiting and/or diarrhea due to extensive necrosis and hemorrhage of the gastrointestinal tract characterize acute arsenic toxicosis. Damage to capillary endothelium results in fluid and blood leakage, hypovolemia, dehydration, hypotension and shock. Cardiac arrhythmias, pulmonary edema and multi-organ failure secondary to acute cardiovascular collapse are possible. Later signs may include lethargy, anorexia, fever, polyuria progressing to anuria, tremor, hypothermia, stupor and death. Treatment of arsenic toxicosis entails removal of the arsenic source, symptomatic care and, potentially, chelation with British Anti-Lewisite (BAL).

**Mercurial salts** are present in elemental, inorganic and organic forms. Inorganic mercury compounds have been used historically in diuretics, antibacterials, antiseptics, ointments, laxatives, and antisyphilitic agents. Mercury ions bind to sulfhydryl groups and also have an affinity for phosphoryl, carboxyl, amide and amine groups. This impairs the structure and function of key proteins and enzymes, and alters receptor affinities and cellular metabolism. Inorganic mercury salts are corrosive and nephrotoxic following ingestion. Salivation, abdominal pain, watery bloody diarrhea, proteinuria, and acute renal failure may occur and potentially fatal hypovolemic shock may result. Perform chelation in symptomatic patients (DMSA, BAL, d-penicillamine).

Plants

**Grapes/raisins** (Vitis sp.) can cause renal failure in dogs. At this time the mechanism of action and toxic principle are unknown. Histopathologic examination has shown proximal renal tubular degeneration or necrosis with the basement membrane remaining intact. The distal convoluted tubules are usually less frequently and less severely affected. Some dogs are exposed and never develop signs and some only develop mild GI signs and recover. Vomiting usually begins within 6 hours of ingesting the grapes/raisins. BUN and creatinine begin to elevate in 12-18 hours. Dogs developing severe oliguria or anuria generally were poorly responsive to attempts to increase urine production (mixed results with peritoneal and hemodialysis). If renal values are normal at 48 hours, the animal can be weaned off fluids and sent home.

True lilies of the *Lilium* and *Hemerocallis* genera (Easter lilies, tiger lilies, day lilies, etc.) can cause acute renal failure in cats. The water soluble toxic principle is unknown. Even minor exposures (bite on a leaf, ingestion of pollen) may result in toxicosis, so all feline exposures to lilies should be considered potentially life-threatening. It should be noted that not all plants with “lily” in the name are true lilies. Cats often begin vomiting within a few hours after exposure. Within 24 to 72 hours of ingestion, oliguric to anuric renal failure develops, accompanied by depression, anorexia, and dehydration. Elevations in BUN, creatinine, P and K⁺ are detectable as early as 12 hours post ingestion. Creatinine elevations may be especially high. Abundant casts, proteinuria, glucosuria, and isosthenuria are usually detectable on urinalysis within 24 hours of ingestion, reflecting lily-induced damage to renal tubular cells. In severe cases, death or euthanasia due to acute renal failure generally occurs within 3 to 6 days of ingestion.

*Cortinarius* sp. mushrooms contain orellanine, a nephrotoxic compound. These bright rust-brown or orange-brown mushrooms occur throughout the U.S. and Canada. Orellanine is not destroyed by drying or cooking. It inhibits alkaline phosphatase which in turn interrupts the production of ATP. Lesions are limited to the kidney (tubulointerstitial nephritis). There can be a latent period of 36 hours to 20 days before the onset of symptoms in people. Anorexia, vomiting, diarrhea, PU/PD, lethargy, and muscle pain can be followed by oliguric or anuric renal failure. Due to the long lag time, GI decontamination is limited. Monitor for renal failure, hypotension, arrhythmias, respiratory depression, hypoglycemia, electrolyte disturbances, and hypoxia. Forced diuresis should NOT be done because it may increase renal damage. Peritoneal dialysis and kidney transplants are performed in humans. Outcome is based on the amount of toxin ingested, but a great deal of variability exists. A shorter latent period correlates with a poorer prognosis.

Mycotoxins

**Ochratoxin A** (OA) is a potent nephrotoxin produced by several species of *Aspergillus* and *Penicillium* molds. Monogastric species are much more sensitive to ochratoxins than ruminants. Ochratoxicosis is usually associated with the feeding of contaminated barley, but wheat, oats, corn, beans, peanuts, hay, and green coffee beans have tested positive. OA is a competitive inhibitor of protein synthesis, induces lipid peroxidation and interferes with carbohydrate metabolism. OA causes degeneration of proximal renal tubules and bile duct proliferation. OA can be tested for in feed, liver or kidney and metabolites can be found in milk or urine. Treatment is symptomatic and supportive.

**Citrinin** is another mycotoxin that can cause renal tubular necrosis. It is commonly found with ochratoxins. Most grains including wheat, oats, barley and corn can be affected. Within a few hours, protein, and glutathione (GSH) tissue levels are decreased and the respiratory capacity (uptake of O₂) and the metabolic enzyme succinic dehydrogenase are inhibited. Treatment is symptomatic and supportive.
Miscellaneous
Any toxin that causes hemoglobinuria (pit viper venom, onions/garlic, brown recluse spiders, zinc) can cause hemolysis. Free
hemoglobin is toxic to the kidney. Hemoglobinuria can induce acute tubular necrosis through the formation of hemoglobin casts. IV
fluids should be started to combat hypovolemia and protect the kidneys. Toxin cause tremors/seizures can lead to myoglobinuria (see
Why so agitated? notes). Myoglobin, a monomer containing a heme molecule similar to hemoglobin, when excreted in the urine can
precipitate, causing tubular obstruction and acute kidney injury.
**Why So Yellow?**

**Liver Toxicants**

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**Household and Industrial**

*Xylitol* is a sugar alcohol. It is used in sugar-free products such as gums and candies as well as for baking. It doesn’t cause significant increases in blood glucose or insulin in humans. However, in dogs, xylitol causes a rapid, dose-dependent insulin release followed by potentially significant hypoglycemia. Signs can include vomiting, weakness, ataxia, depression, hypokalemia, seizures, and coma. Some dogs have developed liver dysfunction or failure following ingestion of xylitol although the mechanism has not been established.

**Pharmaceutical**

*Acetaminophen* (Tylenol®, non-aspirin pain reliever, APAP) is a synthetic non-opiate derivative of p-aminophenol. APAP acts primarily in the CNS to increase the pain threshold and may also inhibit chemical mediators that sensitize the pain receptors to mechanical or chemical stimulation. The antipyretic activity of APAP is achieved by blocking the effects of endogenous pyrogens by inhibiting prostaglandin synthesis. Two major conjugation pathways are used to metabolize APAP by most species (P-450 metabolism followed by glucuronidation or sulfation). Acetaminophen-induced hepatotoxicity and nephrotoxicity is due to the formation of the oxidative metabolite, N-acetyl-para-benzoquinoneimine (NAPQI). Glutathione can conjugate and neutralize NAPQI, but when glutathione stores are depleted, NAPQI binds to sulfhydryl groups on the hepatic cell membrane and damages the lipid layer. NAPQI causes severe oxidative stress to RBCs leading to methemoglobinemia and Heinz body formation. Methemoglobin values increase within 2-4 hours, followed by Heinz body formation. Clinical signs include depression, weakness, hyperventilation, icterus, vomiting, methemoglobinemia, hypothermia, facial or paw edema, death, cyanosis, dyspnea, and hepatic necrosis. Liver necrosis is less common in cats than in dogs. Clinical signs of methemoglobinemia may last 3-4 days. Hepatic injury may not resolve for several weeks.

*Aspirin* (acetylsalicylic acid, ASA) is the salicylate ester of acetic acid and is a weak acid derived from phenol. Aspirin reduces pain and inflammation by reducing prostaglandin and thromboxane synthesis through inhibition of cyclooxygenase. At very high doses, aspirin and other salicylates uncouple oxidative phosphorylation leading to decreased ATP production. Salicylates also affect platelet aggregation. Signs may include vomiting (+/- blood), hyperpnea, respiratory alkalosis, metabolic acidosis, gastric hemorrhage, central lobular liver necrosis, and bleeding diathesis. Fever and seizures may be seen due to the uncoupling of oxidative phosphorylation.

*Ketoconazole* is a broad-spectrum imidazole antifungal agent that alters the permeability of the cell membrane and inhibits intracellular enzymes of susceptible fungi. Adverse events following therapeutic doses may include hypertension, nausea and vomiting, liver toxicity, skin eruptions, and adrenal suppression. Hepatitis, hepatocellular necrosis, icterus, and fatal hepatic failure have been reported.

*NSAID* hepatotoxicity can be seen with any NSAID and is thought to be an immune mediated reaction. Most cases are reversible with supportive care.

**Metals**

*Iron* is an essential mineral that is important in oxygen delivery to tissues, enzymatic processes, and oxidative metabolism within the body. Accidental overdosing of iron supplements may cause acute iron toxicosis, characterized by corrosive gastroenteritis and hepatic injury. Iron absorption from the gastrointestinal tract is highly regulated by the body. Iron is carried in the blood by a protein called transferrin, which conveys the iron to the liver where it is transferred to ferritin. In the liver, iron is either utilized or stored in small amounts as ferritin or as in larger amounts as hemosiderin. When the level of iron exceeds the amount of protein available to bind it, free iron causes oxidative injury to hepatocytes. This hepatic injury may be massive and severe, as in the case of an acute massive iron overdose, or it may be chronic and fulminating, resulting in cirrhosis of the liver over time. Clinical signs of acute iron toxicosis include bloody vomiting and/or diarrhea, abdominal pain, weakness, shock, collapse and death due to shock or anemia. Animals that survive may subsequently develop signs of acute hepatic failure such as lethargy, anorexia icterus, fatty stools, and coagulopathy.

*Copper* is an essential dietary mineral in mammals and a variety of copper compounds are used in manufacturing, medicine, agriculture, and welding (anthelmintics, algaecides, fungicides, wood preservatives, livestock footbaths). Copper accumulates in hepatocytes, where it damages lysosomal membranes, resulting in release of copper and lysosomal hydrolases into the cytoplasm. In Bedlington terriers, an autosomal recessive genetic defect is responsible for the sequestration of copper within the liver. The result is chronic-active hepatocellular necrosis, ultimately resulting in fibrosis and macronodular regeneration. Young dogs may show...
episodic lethargy, anorexia, and vomiting indicative of active liver disease. Older dogs (> 6 y) may show icterus, weight loss, anorexia, vomiting/diarrhea, hepatomegaly, ascites, and coagulopathy (i.e. end-stage liver disease). West Highland white terriers, Skye terriers, Doberman pinschers, and keeshonds are breeds in which high hepatic copper levels have been found in both animals with normal livers and those with significant hepatic injury. It is not unknown whether the sequestration of copper is the cause of hepatic injury, or whether the copper levels are a consequence of some other disease process.

Plants

**Amatoxins** are found in some of the Amanita, Galerina and Lepiota sp. of mushrooms. These mushrooms have a wide distribution throughout the U.S.. Amanitins inhibit nuclear RNA polymerase II, interfering with DNA and RNA transcription, thus inhibiting ribosomal protein synthesis. Cells with high metabolic rates (hepatocytes, intestinal crypt cells) are most sensitive. There are three phases in amatoxicosis. A latent phase of approximately 6-24 hours is followed by a gastrointestinal phase with abdominal pain, vomiting and diarrhea. This phase usually lasts 2-3 days. The hepatic phase begins 36-48 hours after ingestion. Jaundice progresses to fulminant hepatitis with hepatic coma, coagulopathies and anuria. Many humans have required liver transplantation following ingestion of amatoxin-containing mushrooms.

**Blue green algae** (cyanobacteria) can be found in many lakes, ponds and rivers throughout the world. Toxic blooms are seen following warm, sunny weather. Toxic blooms are seen more frequently in ponds that get runoff from heavily fertilized fields or from feed lots or pastures bearing significant numbers of animals. Steady winds often propel toxic algae to the shoreline where animals are exposed. The most important toxin-producing genera of fresh and brackish water blue-green algae are *Microcystis, Anabaena, Oscillatoria, Aphanizomenon, Nodularia*, and *Nostoc*. The primary toxic effects of blue-green algae in animals include acute hepatotoxicoses, peracute neurotoxicoses, and gastrointestinal disturbances. *Microcystis, Oscillatoria, Nodularia*, and less often *Anabaena* may produce hepatotoxins called microcystins. Microcystins cause disorganization of the actin filaments of the hepatic cytoskeleton, leading to cellular collapse. Clinical signs in animals with hepatotoxicoses include weakness, stupor, prolonged capillary refill time, pallor of mucous membranes, bloody diarrhea, and cardiovascular collapse. Clinical signs are usually observed within 12 hours after exposure. Death may occur within a few hours to a few days, depending upon the amount ingested and the toxicity of the bloom. Death often is preceded by coma, muscle tremors, and seizures. Death usually results from intrahepatic hemorrhage and hypovolemic shock and/or acute liver failure.

**Sago palms** (*Cycas* and *Macrozamia* sp.) are ornamental plants found in tropical to subtropical climates, but they can also be grown as houseplants. There are three toxins in cycads. Cycasin is thought to be responsible for the hepatic and gastrointestinal signs. Sago palms also contain two neurotoxins (B-methlamino-L-alanine and an unidentified toxin). The seeds contain the highest amount of cycasin, but the entire plant is toxic. Cycasin causes centrolobular and midzonal coagulative hepatic necrosis along with GI hemorrhage. GI signs begin within a day and laboratory values (ALT, bilirubin, Alk Phos) become abnormal in 24-48 hrs. The most common signs are vomiting (+/- blood), depression, diarrhea, anorexia, and seizures. Mortality rate is about 30%.

Mycotoxins

**Aflatoxins** are mycotoxins produced by *Aspergillus flavus, Penicillium* spp. and possibly other fungi. The most commonly affected crops are corn, peanuts, and cottonseed, but rice, sweet potatoes, wheat, oats, barley, millet, sesame, sorghum, cacao beans, almonds and other nuts can be affected. Aflatoxin metabolites bind with cellular components including nucleic acids, organelles and regulatory proteins thereby disrupting normal cellular processes. Signs of acute toxicity include anorexia, lethargy, vomiting, bloody diarrhea, weakness, and seizures. Liver failure, oliguria, and DIC often result in death within a few days.

**Fumonisin** is most noted for causing equine leukoencephalomalacia (blind staggers, moldy corn poisoning), but it can affect all species. Fumonisin is produced by *Fusarium verticillioides* which grows primarily on corn. Fumonisins are structurally similar to sphingosine a constituent of sphingolipids. Fumonisins inhibit sphingolipid biosynthesis and liver damage may be a consequence of derangements in cell membranes and disruption of normal regulatory mechanisms within cells due to the accumulation of sphinganine. Treatment is symptomatic and supportive.
Normal micturition consists of a urine storage phase (bladder relaxed and filling with closed urethra), and urine voiding phase (bladder contracting and emptying with relaxed urethra). Appropriate storage and voiding depend on intricate and coordinated interaction of the nervous system, urinary bladder and urethra. Storage disorders manifest as urinary incontinence, voiding disorders manifest as urine retention (usually). Successful treatment depends foremost on accurate problem localization/description, and neurophysiologic understanding.

Lower urinary tract anatomy and neurophysiology of micturition

Key anatomic components of the lower urinary tract (LUT): 1) detrusor muscle: smooth muscle of bladder body and neck; 2) internal urethral sphincter (IUS): formed from the smooth muscle of the urethropvesicular junction; 3) external urethral sphincter (EUS): includes striated muscle encircling portions of the urethra distal to the IUS; and 4) ureterovesical junction: proximal to IUS, at junction of bladder body and neck. The urethral closure mechanism = bladder neck + smooth and striated urethral musculature = the “outflow tract” or “outlet.”

Sympathetic (adrenergic) input controls the storage phase via hypogastric stimulation of detrusor beta receptors (smooth muscular relaxation and bladder filling) and alpha-1 bladder neck/urethral (IUS) receptors (outlet closure maintaining continence). During storage, pudendal (voluntary) nerve input stimulates nicotinic cholinergic EUS receptors, causing contraction of striated (EUS) muscle, and additional outlet closure when needed (e.g., when coughing/sneezing; to temporarily override urge to void when inappropriate).

Parasympathetics (cholinergics) rule voiding. Detrusor stretch receptors transmit filling sensation/voiding urge via pelvic nerve and pain/distension via hypogastric nerve to higher centers. The pelvic nerve stimulates contraction via detrusor muscarinics, raising bladder pressure. Simultaneously, sympathetic input to the outlet is inhibited at the level of the brainstem, allowing IUS and EUS relaxation. When bladder pressure exceeds closure pressure voiding occurs, then the system is “reset” for filling.

Urinary incontinence is involuntary urethral urine leakage, and occurs most in spayed female dogs, less in male dogs, and rarely in cats. The culprit is usually urethral sphincter(s) failure.

Causes of incontinence

Neurogenic

In neurogenic incontinence, leakage is rarely the sole apparent abnormality; usually pelvic limb deficits or a sacrococcygeal (“tail-pull”) presentation is present. UMN lesions generally produce sphincter hypertonus and retention; LMN lesions cause sphincter hypotonus and incontinence, as can brain diseases and some generalized neurologic disease (e.g., dysautonomia). Treatment and prognosis for control of leakage depend primarily on resolution of underlying cause (e.g., surgical management of intervertebral disk herniation; fixation of sacral fracture).

Non-neurogenic

Urethral sphincter mechanism incompetence (USMI)

USMI is the most common cause of canine incontinence. It’s seen most often in spayed, medium to large-breed adult bitches, but can occur congenitally, and the development and degree of USMI in a given patient may be multifactorial. Castrated and intact male dogs can (uncommonly) develop USMI, as can cats (very rarely). A recent report suggests OHE prior to 6-7 months as a predisposing factor in bitches weighing >20 kg at maturity. In otherwise healthy, neurologically normal dogs with concentrated urine and without LUT inflammation, diagnosis of USMI is confirmed by positive response to drugs that increase outlet resistance. USMI causes intermittent incontinence, seen most often during recumbency, and particularly during sleep.

Alpha-1 agonists (e.g., phenylpropanolamine[PPA]) and estrogens (e.g., diethylstilbestrol, estriol) effectively treat USMI. Most (80-95%) USMI bitches respond very well to one or a combination of these. If inadequate response to a single agent, combination therapy may be more effective. PPA is first-line therapy for USMI cats and male dogs. Male dogs can also be treated with injectable testosterone, alone or with PPA. Unfortunately, only ~45% of male dogs with USMI are medically well-controlled.

Depot gonadotropin releasing hormone (GnRH) analogs may help USMI refractory to PPA and estrogens. GnRH analogs down-regulate production/secretion of FSH and LH, and multifactorially improve continence; bladder capacity is improved but urethral pressures don’t increase. In a recent report GnRH analogs +/- PPA controlled incontinence within 5-10 days in 12/13 dogs with either USMI refractory to PPA and estrogens, or an inability to take PPA.
Endoscopic submucosal bulking injections (crosslinked collagen or extracellular matrix) into the proximal urethra can help refractory USMI. These injections narrow the urethral lumen and improve outflow resistance. Effects may last up to several years, but then may need to be repeated, and the mucosa may not tolerate more than one or two repeated injections.

Surgical procedures including colposuspension, cystourethropexy, and seromuscular urethral slinging have also been used for refractory USMI. Singly, durability of effect has been limited, but a recent report on colposuspension/urethropexy combination surgery shows significantly increased length of efficacy. Artificial hydraulic sphincters have also been used, and transobturator vaginal tapping procedures may (with practice) prove a less invasive, economical, effective procedure for USMI bitches.

**Detrusor instability/overactive bladder/urge incontinence**

Detrusor instability is failure of bladder relaxation during storage, and causes leakage due to involuntary, uninhibited detrusor contraction. Less common than USMI, it’s characterized by intermittent incontinence, often with activity or excitement, and may resemble pollakiuria or inappropriate urination since dogs will often posture when leaking. Detrusor instability may occur alone or in combination with USMI in male or female dogs, and is treated with antimuscarinic (anticholinergic) agents (e.g., imipramine, oxybutynin, dicyclomine) that increase bladder capacity and decrease spasticity.

**Ectopic ureter**

Ureters implanting anywhere but the trigone are termed ectopic, and ureteral termination distal to the trigone conducts urine to the proximal urethra or vagina and usually causes continuous dribbling from birth. Cystoscopic laser ablation (ideal), surgical reimplantation into bladder, or nephrectomy if kidney is severely hydronephrotic is required for correction. Ectopia may be unilateral or bilateral, and other urinary abnormalities are often also present (e.g., short, wide urethra; USMI). The presence of multiple anomalies may necessitate medications to achieve continence even after correction of ectopia; thorough pre-op urinary tract assessment refines prognosis and helps predict need for ongoing medical management.

**Prostatic disease**

Incontinent males should be screened for bacterial prostatitis, prostatic neoplasia, and other prostatic disease (rectal palpation, urine/prostatic fluid analysis, ultrasonography) since these diseases can engender urine leakage.

**Cats**

Cats are rarely incontinent, but common causes for feline incontinence include congenital anomalies, neurologic injury/malformation, viral disease, and USMI. Juveniles should be screened for ectopic ureters and vaginoureteral anomalies, and tested for FeLV, once LUT inflammation and polyuria are excluded. PPA may be effective in non-neurogenic adult USMI. Urinary/fecal incontinence occur with tail-pull injury and in Manx with sacral malformation.

**Diagnostic approach to incontinence**

History and physical (including observed voiding) inform lesion localization; signalment alone will narrow differential list. Minimum data base in incontinence includes CBC/chemistry (rule out polyuric diseases) urinalysis and culture. Polyuria may precipitate or exacerbate incontinence by overwhelming bladder capacity, so poorly concentrated urine should be confirmed and investigated, if persistent. Most incontinent dogs don’t need imaging at initial workup, but is recommended for incontinent pets: <1 year old; with onset following a surgical procedure; with continuous leakage; who are male; with leakage from anatomically abnormal sites; with recurrent UTI, recurrent vaginitis, hematuria, crystalluria, or azotemia; and when considering surgical correction.

Survey radiography detect radio-opaque uroliths and gross bladder, vertebral, or pelvic malformations. Cystoscopy is the gold standard for detecting abnormalities of the ureters and urethra; excretory urography (with radiographs or CT) is helpful when cystoscopy is not available. Ultrasound helps assess UUT/LUT anatomy, and detects prostatic or trigonal masses interfering with normal outlet closure. Digital vaginal exam, vaginoscopy and/or contrast vaginogram help evaluate presence, position and severity of vestibulovaginal stenosis, or the possible presence of vaginal urine pooling.

**Relapsing or refractory incontinence**

When relapse or failure of presumed USMI response occurs, re-assessing dose, selection and compliance with medications, re-screening for infection or polyuric disease, ruling out an underlying anatomic anomaly/lesion or mixed disorder, behavioral component, or senility may be helpful. If these possibilities are ruled out, surgical interventions may be appropriate.
Postrenal azotemia occurs when any process distal to the renal tubules interferes with urine collection, containment, or excretion, and the resulting retention of wastes can rapidly cause life-threatening fluid, electrolyte, and acid–base derangement. Acute postrenal azotemia (usually urethral obstruction, ureteral obstruction, or traumatic urinary tract rupture) is common, particularly in cats, and may be fatal without rapid correction. Because postrenal azotemia results from urinary tract rupture or obstruction, rather than intrinsic damage to the kidneys, it has the inherent potential for reversibility; thus, the possibility of a primary postrenal disease or component should be initially investigated in every azotemic patient.

Lower urinary tract obstruction (LUTO)
LUTO is usually diagnosed by history and by palpation of a turgid, painful bladder. Owners may report unproductive attempts to urinate, which are sometimes mistaken for constipation. Urolithiasis and neoplasia are the most common causes of canine LUTO. Urethral stones obstruct more male than female dogs due to the smaller relative urethral diameter and longer length in males; the urethral curving around the ischium; and, most restrictive, the presence of the os penis. Calcium oxalate and ammonium urate stones are the stone types that most frequently cause urethral obstruction due to their small size, tendency to occur multiply, and increased incidence of both types in males.

Urolithiasis or mucocystalline urethral plugs cause most feline LUTO. Mucocystalline plugs are generally associated with feline idiopathic cystitis (FIC) and are seen in both males and females, but they cause obstructive disease almost exclusively in males because of comparative urethral anatomy. Survival rate is 90-95% with treatment, and recurrence rate of FIC-associated obstruction is 15-40%. Predisposed cats tend to be one or more of the following: young adults, overweight, dry food eaters, and indoor-only. The following factors seem to decrease risk of re-obstruction: use of a 3.5 Fr (rather than 5 Fr) indwelling urethral catheter following relief because of comparative urethral anatomy. Survival rate is 90-95% with treatment, and recurrence rate of FIC-associated obstruction is 15-40%. Predisposed cats tend to be one or more of the following: young adults, overweight, dry food eaters, and indoor-only. The following factors seem to decrease risk of re-obstruction: empirical antibiotics, glucocorticoids, anxiolytics, and NSAIDs. Older cats are more likely to re-obstruct than are younger cats.

Upper urinary tract obstruction (UUTO)
Acute UUTO may occur due to intraluminal, extraluminal, and intramural causes, such as uroliths, nonmineralized material, trauma, neoplasia, proliferative disease, ureteroceles, inflammation, fibrosis, stricture, and inadvertent surgical trauma or ligation. For UUTO to cause azotemia, bilateral disease—either bilateral ureteral obstruction, or unilateral ureteral obstruction with dysfunction or absence of the contralateral kidney—must be present.

The most common cause of UUTO is calcium oxalate urolithiasis (CaOx), usually in cats and small dogs. Incidence of CaOx has increased dramatically in the past 20 years (now >90% of analyzed nephroliths and ureteroliths). CaOx stones form in renal parenchyma, and may stay there or pass into the ureters and bladder. Formation risk is multifactorial, including degree of urine saturation with calculogenic minerals, urinary crystallization/ aggregation/growth inhibitors, and urinary crystal aggregation/ growth promoters. UUTO can be simultaneous, bilateral obstruction; however, acute unilateral obstruction with prior contralateral obstruction (causing dysfunction) is much more common. These pets frequently have one firm, atrophied, nonpainful smaller kidney, and one larger, painful, turgid-feeling kidney. Some astute owners may detect subtle clinical signs during an initial unilateral obstruction (e.g., antisocial behavior, flank licking, or back or abdominal pain), leading to earlier detection of the presence of kidney/ureteral stones.

Trauma, renal biopsy, or renal hematuria can cause UUTO with clotted blood. Accumulated debris (e.g., from inflammation or ureteral trauma, fungal granuloma, sloughed renal papilla) may also result in a nonmineralized UUT obstruction. In our experience, such obstruction may occur due to acute renal tubular necrosis from toxin, acute pyelonephritis, or luminal trauma from presence or passage of stones. Experientially, these obstructions often occur proximally in the UUT, may be acutely bilateral, and can be milked to a dilated region the ureter or the ureteropelvic junction for removal. Ureteral damage (inflammation, fibrosis, mineralization, stricture) can cause complete or partial UUTO. Iatrogenic ureteral ligation, transection, crushing, or devascularization occurs most often during uterine body ligation during OVH, particularly when the bladder is distended and ureters are slackened. Postoperative progressive ascites, azotemia, and/or ureteral/renal pelvic dilation mandate prompt, aggressive assessment (ascites cytology/chemistry, excretory urography/antegrade pyelography, retrograde cystography, exploratory laparotomy).

Treatment for acute UUTO depends on etiology. If metabolic derangement is not life-threatening, medical management with fluids, analgesics, and ureteral relaxant(s) may permit passage of an intraluminal obstruction in 1-3 days. Severe derangements require...
medical stabilization and/or hemodialysis, and/or surgical relief of obstruction (ureteral stent, ureterotomy/re-implantation, subcutaneous ureteral bypass).

**Urinary tract rupture**
Rupture of UUT or LUT results in urine collection in the retroperitoneum, peritoneum, or subcutaneous tissues. Presenting signs may be associated with trauma, uremia, chemical irritation (cellulitis, urine peritonitis), or, if urine is infected, septic issues. Causes of ureteral rupture include surgical damage, calculi, neoplasia, infection, and blunt or penetrating trauma (uncommon). Bladder or urethral rupture occurs most often secondary to caudal abdominal trauma (dogs), urethral catheterization (cats), or excessive manual pressure during bladder expression. Preexisting LUT pathology (e.g., urethral or bladder tumors, bladder wall damage from prolonged obstruction) can cause or predispose to rupture from instrumentation or compression.

**Common laboratory abnormalities**

**Azotemia**
LUTO pets may or may not be azotemic, but UUTO cats are some of the most azotemic pets we see. Some will have uremic oral ulcers. Very high BUN often correlates with marked post-obstructive diuresis.

**Metabolic acidosis**
IV crystalloids and relieving obstruction usually corrects acidosis (which can be severe); if not, adding sodium bicarbonate is appropriate.

**Hyperphosphatemia**
Hyperphosphatemia can drive hypocalcemia, but is corrected by relief of obstruction and fluid therapy. Phosphate binders are not usually used/needed in acute settings.

**Hyperkalemia**
Hyperkalemia can be marked (>10 mmol/L), can stop normal cardiac function, and is usually the direct cause of death when obstructed animals are left untreated. IV crystalloids and relieving obstruction usually suffice to correct potassium, but if cardiotoxicity is present, IV calcium, IV sodium bicarbonate, and/or insulin/dextrose may be needed to buy time to relieve obstruction.

**Hypocalcemia**
May be present and may be moderate to severe, but not treated unless the pet is clinical.
Anatomy and physiology
The prostate, a glandular organ surrounding the proximal urethra, is the only canine accessory sex gland, and results in disease in ~75% of intact male dogs over their lifetimes. Secretory epithelial and stromal tissue are admixed within a capsule containing alpha-1 adrenergically-innervated smooth muscle. Growth and function are androgen-mediated (chiefly dihydrotestosterone); castration induces quick prostatic atrophy (~50% at 3 weeks, and ~70% within 9 weeks).

Diagnostic testing
Physical examination
The prostate is usually palpable per rectum at the pubic brim. Manually tipping the bladder caudally and/or elevating the dog’s front end can move the prostate caudally into reach. The normal prostate is bilobed, soft-firm, smooth, symmetrical, mobile, and nonpainful. Prostates of intact Scotties may be up to 4 times larger than prostates of equally-sized other breeds’.

US and urethrography
The prostate surrounds the urethra distal to the trigone and should be smoothly rounded. The lobes are easily distinguished, with diffuse fine mottling or whorls, but without cysts. Contrast urethrography may show reflux into the prostatic ducts, and urethral narrowing through the prostatic region.

Cytology
Prostatic cytology may be performed on prostatic fluid, massage samples, and aspirates. Most commonly, prostatic fluid is collected from intact dogs by manual ejaculation. Most dogs will ejaculate with manual stimulation, but ill or painful dogs may resist stimulation. Prostatic massage is preferred for fluid collection when cancer is suspected (usually more cellular), or when illness, pain, or disposition complicates ejaculation. Prostatic massage is done post-voiding, in lateral recumbency. The urethra is catheterized and the bladder is emptied, flushed with sterile saline, and again emptied. The catheter tip is withdrawn to just distal to prostate. Massage is performed per rectum, then 5-10 ml of saline flushed through the catheter. The catheter tip is advanced collect fluid and cells from bladder. Submit ejaculate and massage samples for cytology and culture.

FNA also yields culture/cytology samples. Take care if cystic disease, abscessation, or neoplasia is suspected. Inadvertent abscess rupture can be a surgical emergency, and slight risk of needle tract seeding accompanies certain neoplasia (e.g., TCC).

Histopathology
Traumatic catheterization (TC), endoscopy, US-guided core biopsy, or surgery can all provide biopsy samples. TC resembles prostatic massage sampling, except negative pressure is applied to the catheter, and the catheter is moved back and forth in the urethra to shear and collect tissue drawn in the side-holes. Endoscopic samples are often quite small due to size-limited urethroscopy in males (often TC samples are larger!), but US-guided cores provide excellent samples (don’t biopsy urethra!).

Prostatic diseases
Benign prostatic hyperplasia/hypertrophy
BPH comprises both hypertrophy and hyperplasia of stromal and secretory tissues, is a spontaneous, normal, aging change, occurs only in intact males (unless other androgen source is present), and is not pre-neoplastic. Many BPH dogs show no signs, but it may be found due to bloody discharge (from increased parenchymal vascularity and cystic change). In men BPH causes dysuria; this is rare in dogs due to minimal prostatic smooth muscle, and greater gland mobility in the dog. Dogs thus are more often presented for tenesmus or ribbon-stools, because prostatic enlargement impedes normal defecation before functionally or mechanically interfering with normal urination. Diagnosis is of exclusion: an intact dog with an enlarged but otherwise normal prostate, negative urine/prostatic-fluid/aspirate culture, and normal cytology. Castrates don’t get BPH without a non-gonadal androgen source. Secretory markers (e.g., PSA, prostate-specific esterase) aren’t useful in dogs.

Castration is preventative, curative, and the quickest, safest, most effective treatment. 5-alpha-reductase inhibitors, progestogens, and estrogens have also been used for management. Estrogens predispose to squamous metaplasia and prostatitis. Progestagens may resolve BPH without affecting semen quality, but increase appetite and predispose to diabetes mellitus and hypothyroidism. Finasteride (5-alpha-reductase inhibitor), decreases gland size without reducing spermatogenesis or libido (inhibits conversion of testosterone to DHT but doesn’t affect testosterone production). Dogs respond in 2-4 weeks with few side effects. Clinical signs recur off medication, so therapy is considered temporary treatment for owners who wish to breed, cryopreserve semen, or achieve show titles prior to castration. Saw palmetto doesn’t treat BPH.

Bacterial prostatic diseases
Prostatitis is diffuse bacterial infection/inflammation, and a prostatic abscess is a cystic collection of suppurative fluid in the parenchyma. Prostatitis can be acute or chronic. Acute prostatitis usually causes pain and/or fever, lethargy, and hyporexia, and may
cause dyschezia, abdominal pain, or pelvic gait abnormalities. Dysuria, pyuria, hematuria, and/or urethral discharge may be present. Chronic prostatitis may be silent (just pyuria and bacteriuria). Prostatic abscesses may present like acute prostatitis, or rupture, causing sepsis/acute abdomen. Prostatitis is assumed in any intact male dog with UTI.

Septic inflammation in prostatic fluid, massage sample, prostatic aspirate, or biopsy sample is diagnostic. With ejaculate or massage sample, >10,000 cfu/ml significant; any aspirate or biopsy sample growth is significant. *E. coli, Staph, Klebsiella*, and *Proteus* are most common. US usually shows parenchymal mottling. Abscesses appear as cystic structures, with anechoic, echogenic, or mixed contents.

Culture/sensitivity on aspirate, massage, or urine directs antibiotic choice. Normal prostate/blood barrier prevents diffusion of many medications, but acute inflammation disrupts the barrier so most antibiotics initially penetrate. In general, though, best antibiotics for prostatitis are trimethoprim-sulfas, fluoroquinolones (especially enrofloxacin), and chloramphenicol. Initially 4-6 weeks of therapy is recommended, with culture 1 week and 1 month afterward. Removing predisposing factors (e.g., castration for BPH) greatly increases odds of cure. Re-culture should be performed at 3-6 month intervals. Abscesses require drainage and appropriate antibiotics. Traditionally, open surgery and marsupialization/omentialization were used, but recent reports of US-guided drainage/culture with reassessment q 1-6 weeks and re-drainage as needed is promising. In two small studies, median number of drainage treatments needed was 2, with abscess resolution in all dogs.

Castration speeds resolution of both prostatitis and prostatic abscess, and also helps prevent recurrence. Antibiotics for several weeks before castration facilitates drug delivery to deep prostatic tissues prior to inducing atrophy and involution.

**Prostatic cancers**
The only primary prostatic disease of castrates is cancer. Prostatic cancer occurs in < 0.6% of dogs, but 2-4x more in castrates. Beagles, Bouviers, Dobermans, English springers, German shorthair pointers, Scotties, Westies, and Shelties are predisposed (last three are also predisposed to TCC). Though castration has been shown to increase risk of prostatic ACA, the over-all risk in castrated males is still very low, and the benefits of castration likely outweigh this low risk in most cases. This assessment should be made for each pet individually.

Prostatic cancer is usually adenocarcinoma (ACA), with TCC next most common. Prostatic carcinomas are not androgen-dependent in dogs, nor mediated by hormonal changes once extant. In general, any castrated dog with prostatomegaly has cancer until proven otherwise. Clinical signs may be due to the primary tumor (dysuria/stranguria, urine retention, tenesmus, ribbon stools, altered gait) or to metastasis (liver, lung, pelvic/sublumbar nodes, spine/pelvis, colon, rectum, spleen). Fever, lethargy, weight loss, and hyporexia occur, especially with metastasis.

Neutrophilia, ALP elevation, hematuria and pyuria are common. Radiographic may include prostatomegaly and a periosteal reaction along pelvic bones, sometimes femurs. US may show hyperchoic parenchymal stippling (focal mineralizations), and blotchy, whorled parenchymal mottling. TC, US-guided FNA, or core biopsy are reliable, non-invasive diagnostics.

Prostatectomy and chemotherapy for ACA can be helpful, but no therapy is curative, and most prostatic cancer has metastasized at diagnosis. Palliative therapy with piroxicam or carprofen can increase comfort and decrease inflammation, may have tumor anti-angiogenic effect, and can prolong survival. Chemotherapy is also used, but overall prognosis is poor. Urethral stenting, laser debulking, and percutaneous cystostomy tubes/buttons are options for dedicated owners.

**Paraprostatic cysts**
In intact males, remnants of the embryonic Wolffian ducts can fill with fluid, forming paraprostatic cysts, easily diagnosed with US. Clinical signs result from space-occupying intrapelvic cysts, with possible stranguria, bloody urethral discharge, and tenesmus, but many dogs show lethargy or decreased appetite, presumably from discomfort. Prostatic parenchyma is usually normal or shows BPH. Therapy is surgical resection; castration prevents further cyst formation.
Obtaining productive, actionable results from practice data can be difficult at best if not approached deliberately and methodically. The most common trap is recording data and producing reports without a clear basis for doing so. This discussion will help you to categorize and prioritize value for your practice, identify common (and uncommon) assessments of practice data, and create measurable value from the results.

Getting started is the hardest part
The most important steps to deriving productive insight from your practice data are also the most difficult: Identifying what is important to you and your business, as well as why it is important.

- Consider obstacles that interfere with these objectives and overcome them
- Focus first on value, not inputs/outputs

Characterizing and prioritizing value

Characterizing

- Needs of the business
- Needs of the people in the business
- Needs of the clients
- Needs of the pets

Prioritizing

- Current impact
- Future impact
- Include all stakeholders

Assessing needs

- First articulate needs in natural language, then translate the needs into data outputs
- Consider actions that could be taken based on results
- Determine what inputs are required to produce the outputs
- Scope it with basis (time, sample size, etc.)

Creating value

- Evaluate the results as pre-planned
- Story-telling with data
- Implement actions as appropriate
- Assessing and re-assessing value
Rapid Fire:
Informatics and Veterinary Medicine
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We are going to go over both clinical and practice management specifics that every practice should be looking at and how to use the data. Want to know at least 5 things that will improve your medicine and 5 things that will improve the profitability and operations of your practice? Come listen and participate in rapid fire veterinary informatics.
Avoid the Most Common Veterinary State Board Complaints with Informatics
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Drawing on experience as a practitioner working full-time in private practice, a member of my state’s board of veterinary medicine, and president of the Association for Veterinary Informatics, I will provide insights, examples and solutions to avoid many of the most common complaints made against a veterinarian to the state board.

Complaints from animal owners to state boards of veterinary medicine have been increasing in recent years owing to a number of factors. Some complaints are warranted and a result of true negligence or malpractice. Many complaints are due to misunderstandings between the client and the practitioner. Some complaints are completely unjustified. Even unwarranted complaints cause stress for the veterinarian, loss of work to address the concern, more work for a (usually volunteer) board, and loss of faith in and weakening of The Bond.

Most client complaints fit into a few categories: consent to diagnostics, procedures and costs; incomplete or improper documentation; explanation of negative results and outcomes; poor client education and take home instructions; and a lack of understanding by the veterinarian of how the laws work and what is expected and required of them.

We can use informatics, and, more specifically, the electronic medical record, to reduced the number of client complaints. Informatics includes collecting, storing, retrieving, analyzing, and presenting data to achieve better health outcomes. Veterinary informatics is a rapidly evolving science that blends information technology, communications, social and behavioral science, and veterinary medicine, to improve the quality and safety of patient care.
Checking Out Veterinary Informatics: From the Library to Your Practice
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It’s a phrase often uttered by students and adult learners, “I’m not techy.” Rarely does anyone say “I am techy,” because what that really means is “I’m willing to try and figure it out.” Few realize that one advantage of today’s rapidly changing technology is that an informatics culture requires multiple skill sets for success. There is a place for everyone – systems thinkers, terminologists, indexers, artists, spell checkers, interface designers and testers, surgeons, pathologists, data analysts, and yes, programmers. If you cannot see yourself jumping into programming head first, start by checking out a library database.

Embracing technology and overcoming perfectionism
Informatics can be a hard sell to veterinarians due to our nature as perfectionists. Much has been written about the perfectionist tendencies of those entering veterinary and other health care professions. Some have implied that perfectionism in veterinary medicine may be growing as it becomes increasingly feminized.¹ Add to this mindset an environment where errors can lead to life threatening consequences.

Now contrast health care professions with information technology, where perfectionism is frequently frowned upon in favor of agility and exploration. While there is little incentive to test the technology readiness of veterinary students, it is worth asking if health care professional schools are failing to attract, or even selecting against, applicants who are technology oriented. A 2006 Canadian study showed nursing and medical students are not technologically prepared, particularly those who are female, from rural areas, and over the age of twenty-five,² all factors present in veterinary student demographics. It is important to note that the study looks at technology readiness (Technology Readiness Index TRI), embracing technology to accomplish a goal, not technology competence which is simply one’s ability to use technology.³,⁴ While doctors in practice continue to decry electronic health record (EHR) systems,⁵ EHR training and EHR-mediated communication are now routinely incorporated into many pre-clinical nursing and medical training programs.⁶

Motivating a veterinary informatics initiative
How can veterinary medicine be motivated to adopt a culture of informatics? It has long been known that veterinary motivation to learn aligns most closely with a professional identity of providing quality patient care, and less with practice management aspects of the job.⁷,⁸ As more institutions embrace technology “for the social good,”⁹ this phrase is likely to be a large motivator in veterinary medicine as well. Diagnostic and treatment technology receives much more hype than EHR’s due to the immediate and visible impact on patient care.

Veterinary medicine in the United States lacks the financial incentive to convert from paper based to electronic records that Centers for Medicare and Medicaid (CMS) payouts provided in human medicine. Veterinary incentives to convert to EHR’s have been more pragmatic in terms of replacing poor handwriting, enabling remote entry, billing and inventory control.

Role of the library
There is very little about paper record systems or a scanned paper-dependent system that prepares veterinarians to document in electronic health record systems in a way that enables future extraction of data. This is where using library electronic resources can have an impact. Not only is the library a comfortable and forgiving environment for the non-techy, but active learning of literature database searching provides an understanding of how and why to document in EHR’s. In addition, it reveals how we know what we know and do not know as a profession. The impact and potential of information gathering and data collection in non-academic practice settings has been difficult to measure and visualize to the broader veterinary community. That is changing due to several factors.

1. Veterinarians have greater access to scholarly literature than they have ever had, with the onset of PubMed in 1997 and Google Scholar in 2004. More recently public policies have mandated free open access to research funded by the NIH, NSF and USDA.
2. There is growing awareness of the decentralized nature of veterinary literature. Veterinarians have never had a system of hospital library access; they have always cobbled information together from a variety of biomedical, agriculture and basic science resources. The One Health movement has brought to light the veterinarian’s role in public health and their need for information access.
3. More private and corporate practices are adopting EHR systems, and more ancillary services are becoming interoperable with EHR’s.
4. Large practice-based studies are popping up in the veterinary literature. VetCompass, a system gathering data through hundreds of UK private practices, has demonstrated that big data studies are possible in veterinary medicine.

**Role of evidence based veterinary medicine**

Attendees should refer to the EBVM Network [http://www.ebvmlearning.org/](http://www.ebvmlearning.org/) for step-wise tutorials on how to do evidence-base veterinary medicine (EBVM). The components of evidence based medicine (ask, acquire, appraise, apply and assess) are addressed here only in terms of their application to an informatics initiative.

**Ask**

Asking questions seems basic and yet it is the number one reason why evidence takes so long to be implemented in practice. Estimated average time lag from research to clinical practice is 17 years. EBVM instruction uses the PICO format to construct questions, where P is patient, population, problem, I is intervention/exposure, C is comparison (to a standard treatment or diagnostic test), and O is outcome. Questions can be about diagnosis, etiology, prognosis or treatment. Herein lies one of the difficulties in teaching evidence based medicine to students, as many are still asking known background questions about pathophysiology, not novel foreground questions. In reality, 85% of the clinical questions veterinary practitioners ask are about treatment.

**Acquire**

This aspect of EBVM has the greatest application to understanding electronic health records and electronic documentation. Students often lose the forest for the trees. They get caught up in identifying study types without understanding why they need to consider study types for their level of evidence in clinical decision making. Or they narrow in on a specific geographic location without considering its larger context. Controlled vocabulary in databases, such as Medical Subject Headings (MeSH) terms in PubMed, allow for narrowing or broadening a search in ways that other search engines do not. Electronic databases and reference management systems also provide an introduction to why metadata standards are important for future discovery. Many veterinarians assumed that we would make the leap to artificial intelligence (AI) enabled search of free text by now, and yet AI has failed thus far to provide the narrower and broader context and facet search that use of metadata and controlled vocabulary provide.

**Appraise**

Evaluating the literature is the most difficult aspect of EBVM that health care practitioners face due to time constraints. Initially they stop searching at the point of discovery, rather than contrasting and comparing all of the available evidence, and later, they lack time to read or interpret studies. This is where newer resources that pre-appraise evidence save the time of practitioners. These resources have been lacking in veterinary medicine, but VetCompanion is attempting to fill this role, with topically organized synopses of the best available evidence. Systems like this that bring EBVM to the point-of-care are more likely to have impact on clinical decisions.

**Apply**

Applying population studies to individual cases with co-morbidities, confounders and information gaps is not intuitive. This needs to be taught with low-stakes opportunities to practice. Guidelines and clinical bottom lines derived from critically appraised topics are few and far between in veterinary medicine.

**Assess**

Reflective practice systems in the form of clinical audits, checklists, development of internal guidelines and morbidity and mortality reports, while commonplace in human medicine, have yet to be widely adapted in veterinary medicine.

**How can we foster a culture of informatics in the veterinary education system?**

As a discipline:

1. Support evidence based veterinary medicine. Only by struggling with our current imperfect EBVM system will we have a workforce prepared to understand the issues and unique contexts of veterinary medicine. Artificial intelligence systems are developing, but not quickly enough to interpret context at a granular enough level to be reliable in a clinical setting; AI systems still rely on the annotation skills of subject experts.
2. Recruit veterinarians with IT experience, perhaps by offering scholarships, and foster informatics interest in veterinary schools. Many graduate programs in other disciplines establish prerequisite technology skills for entering students and set an expectation of self-learning.
3. Collaborate with evidence based veterinary medicine groups, precision medicine groups, epidemiologists and local health departments.
4. Teach small animal epidemiology. With 2/3 of US veterinary graduates entering companion animal practice now, small animal epidemiologists are needed to work with shelters, breed organizations and public health offices.
5. Establish informatics learning modules for incorporation into DVM teaching. These might be disciplinary specific related to LOINC, radiology, or business management, to help students understand the breadth of veterinary informatics.
As an individual:
1. Expect less of ourselves and one another. Set goals but realize that everyone is learning.
2. Start by searching literature databases. An understanding of the struggle between controlled vocabulary and natural language, and wrestling with data standards and extracting data, will inform how future systems are used and developed.
3. Find out what we as a discipline don’t know, and use that to inform practice based studies.
4. Use informatics to motivate and maintain a passion for life-long learning.

References
Veterinary medicine is quickly evolving in this digital age to a profession that doesn't just want good evidence on which to base their medical decisions, but in fact demands it. As frustration with the lack of traditional research studies builds, many have begun to focus on building their own practice-based evidence. Every practice creates lots of data every day and should be able to use that data. Unfortunately, most veterinary electronic medical record systems focus more on the billing side of practice and have not put emphasis on the medical record side of the system. It is now widely accepted that standardizing the way we store information, such as problems and diagnoses, is important to improving the quality of the data stored, however, our EMR systems are either not implementing the desired standards or they are not focused on implementing them fully. This lecture will highlight problems in one area: implementation of SNOMED CT© as exemplified by the AAHA Problem and Diagnosis Terms (AAHA PDT). We will review the areas in which vendors need to improve their implementations to improve ease of use in practice and help veterinary personnel understand these problems so that they can communicate effectively with their own vendors.

The biggest source of frustration for users in systems that have implemented the AAHA PDT is the limited implementation of descriptions. One of the strengths of SNOMED CT© and the Veterinary Extension of SNOMED CT (VetSCT©) is the fact that it is a concept-based terminology that provides multiple terms (called descriptions in SNOMED) for each medical concept. This is important to accommodate the variations in linguistic preferences among veterinary professionals. It is not uncommon to find many different terms used to describe the same disorder or finding. For example, consider this disorder definition taken from an associate information page on the Veterinary Information Network, “a primary myocardial disease wherein fibro-fatty replacement of the right ventricular (and less commonly left ventricular) myocardium occurs, which results in ventricular arrhythmias (i.e. arrhythmogenic) and syncope. Less commonly, myocardial dysfunction and congestive heart failure can occur. Sudden death is relatively uncommon. ARVC most commonly affects adult boxer dogs. It has been rarely reported in the cat”1. This disorder used to be taught to veterinarians as Boxer Cardiomyopathy. Now it is recognized as Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC). Either of these phrases are commonly used to describe this medical disorder. SNOMED CT allows this disorder to be identified with a single concept identifier, but has both descriptions associated with that same concept. This concept and both of its descriptions are included in the AAHA PDT. Unfortunately, systems that have implemented the AAHA PDT are not structured to allow multiple descriptions to point to the same concept, so they must choose only 1 description to include for users. ARVC is currently the preferred term for this concept in AAHA PDT and it is likely the one being used in these systems. Imagine how frustrating that would be if the only term you could remember was Boxer Cardiomyopathy! Users need to encourage vendors to allow searching of all available descriptions in the AAHA PDT, even if they only choose to display the preferred term.

Many system developers have been operating under the misconception that once the ‘right list’ is found for standardizing a data element, their job is done. However, the reality is that no list can ever be complete. These lists need to be able to change and adapt over time as medicine changes, as user preferences change and as improvements are made to the lists themselves. Systems need to be able to recognize when a user needs the lists to adapt. The AAHA Diagnostic Terms Editorial Board (AAHA DTEB) has done its best to ensure that the most common findings and disorders are included in the AAHA PDT, but no one can guarantee 100% coverage as many diseases are rare or isolated to specific regions, etc. This means that inevitably there will be times when users are unable to find the findings/disorder that they need. Systems must provide a way for users to record the information, without altering the standardized list, and then trigger a submission or request for review by the terminologists at VTSL and/or the AAHA DTEB for inclusion in the AAHA PDT. Oftentimes this will result in a response that indicates the proper term already exists and guides the user towards the appropriate code (due to the limitation discussed above about limited description implementation). It may also result in the addition of a term or concept to the AAHA PDT that will be released in the next update distributed on a semiannual basis (April & October of each year). With either result, ideally the system would also have a way to relay the information back either to the user directly or in some way updating the stored data where appropriate. Adequately handling user needs and requests is an important part of helping the terminology to grow and improve so that users will have what they need to record their day to day cases.

As the AAHA PDT grows and adapts, systems must, of course, update with these changes. New concepts and descriptions are added. Concepts and descriptions are retired. All of these changes must be made in the systems using the AAHA PDT. Systems must plan for these updates and apply them in a timely fashion. It’s a simple idea, but does require significant system programming to

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Fix Your Master Problem List, Fix Your Practice
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achieve this in a smooth, seamless fashion that doesn’t overburden users. Each system developer will have to determine how best to manage this task, but it must be at the top of the priority list for the systems to keep up with user demands on the terminology.

Another aspect of terminology use that is often overlooked is the handling of legacy data and historical changes to the terminology. Ignoring legacy data effectively makes unusable any data stored either prior to the implementation of AAHA PDT or with AAHA PDT codes that have since been retired. Legacy data is any piece of data that is stored using a code that has been inactivated or retired. The most common instance of legacy data is practice data stored in a medical record using system interface lists prior to implementing a standard terminology such as AAHA PDT. The codes used previously, called legacy codes, have no formal relationship to the AAHA PDT concepts so the computer system has no way of relating records stored with the legacy codes to records stored with equivalent codes in the AAHA PDT. When analysis of data is performed using the AAHA PDT, the cases stored with the legacy codes will be left out, potentially losing a large chunk of data for analysis. This could be overcome by creating a formal connection between the legacy codes and the AAHA PDT codes, called a mapping. By creating one to one “matches” for all (or as many as possible) of the legacy codes to equivalent concepts in the AAHA PDT, when searches or aggregations are performed using AAHA PDT, the data coded with the equivalent legacy codes (via the mapping) can also be included. A similar issue happens when codes in the AAHA PDT are retired. Historical changes to SNOMED CT are maintained within the terminology itself creating a formal link between retired concepts and their best replacement concepts. This historical link can be used in the same way that a mapping to legacy codes is used. Unfortunately, we have two major obstacles to overcome. First, the creation of mappings between legacy data and AAHA PDT is a labor intensive, manual job. This is compounded by the fact that vendors can’t do this work for us since every practice is going to have a slightly different list thanks to the highly customizable nature of interface lists. This means that each practice that wants to maintain the usefulness of its legacy data will need to create their own mapping. Our second obstacle is the fact that systems aren’t set up to use mappings or the historical SNOMED relationships when querying or analyzing data. We’d like to see a process that allows a user to input their legacy mapping into their system so that the system could then be programmed to use those linkages each time data searches are done. A similar process (though we would expect this to be automated by the vendor maintaining the AAHA PDT in the system) should use the historical SNOMED relationships as well to further complete the legacy data inclusion.

As you can see there are several major improvements that need to be made by vendors to fully implement standardized terminology such as the AAHA PDT in a way that maximizes benefit while minimizing user frustration. Vendors are unlikely to recognize the need for these improvements without requests and demands from their users. It’s up to us to work with our vendors and help them understand that better data is important to our practice and therefore important to their business as vendors of medical record systems.
Do Better Medicine With Your Untapped Goldmine- Your Data
Jonathan Lustgarten, MS, PhD, VMD
Red Bank Veterinary Hospital
New York, NY

How many of you have seen unique cases in your clinic, even though the literature says they are rare? How many times did you wish you knew the disease or resistance prevalence at your hospital? What other possible things do you always wish you knew about the medicine you practice? During this talk, we will go over both basic and advanced concepts and highlight what you can use to improve the medicine you practice. We will also have a brief session that will allow opportunity to discuss new investigations that might be of interest to you.
Putting Information into Practice: Better Management and Operations Decisions
Jonathan Lustgarten, MS, PhD, VMD
Red Bank Veterinary Hospital
New York, NY

Running a hospital is a demanding effort that requires not only understanding of what is going right, but also what you can be doing better. Knowing where to look for this information can even be more challenging. Once you have this information, you can make decisions that help your practice become more efficient, provide marketing opportunities, help with inventory stocking, determine appropriate service prices, and help manage some of the harder things in your practice such as labor.

During this talk, we will go over the various types of information you should have at your fingertips, various key performance indicators that you can generate from your systems, and how using what your practice generates every day can help catch problems before they develop.
Easy Steps to Success with Informatics without Widespread Standards Adoption

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With the widespread adoption of standardized coding, human health has long had access to insights and efficiencies that continue to elude the veterinary services industry. Insurance and regulation were the driving forces behind standardization in human health, but with only 1% of the 86 million cats and 78 million dogs in the United States being insured and with regulation being slow to come, numerous initiatives to define and implement such standards have fallen prey to seemingly insurmountable adoption challenges. Simply put, there just isn’t a clear reason in the veterinary market to make the necessary investments to achieve standardization. Or, is there?

What could be achieved through coding standardization?
Maybe there actually are good reasons to invest in standardization. Consider that there are 68,000 veterinarians across roughly 25,000 practices in the United States. Conservatively, that amounts to over 1 million patient visits every day, yielding hundreds of millions of transactions and medical records each year. Right now, this information exists in electronic form in databases all over the country! Unfortunately, it’s all apples and oranges as there is no standardization process allowing the meaningful correlation of all these data points.

Veterinarians hunger for accurate epidemiological data. The ability to recognize diagnostic and treatment patterns to improve treatment protocols, demonstrate real and ongoing value to pet owners, predict potential disease outbreaks, and ultimately improve health outcomes is wildly valuable. Transactional and medical record data sourced from disparate systems and correlated to a single standard would provide the inputs for successful epidemiological research and evidence-based medicine.

Stellar health outcomes are the ultimate goal of all veterinarians, which is why so many of us who aren’t veterinarians ourselves love serving this industry; the passion for the mission is a joy to witness and to be a part of. Unfortunately, most veterinarians must also be entrepreneurs and business people. Whether primary responsibility for a practice’s financial success is solely in the hands of a practicing veterinarian or if delegated to a practice manager, these individuals find themselves in a constant chase for increased efficiency and profitability. Again, this is a case where standardized data can help. Access to regional pricing benchmarks, recognition of treatment compliance gaps, and consistent business intelligence relative to practice financial health would all be great wins for the entire industry and for each individual practice.

While still relatively low, especially compared to many European markets, pet insurance penetration is a fast-growing service in the United States. Insurance providers not only need to differentiate themselves in an increasingly crowded market, but they also need to ensure that using their service is a pleasant experience from the perspectives of both the pet owners and the practices. As such, these companies are always looking for innovative solutions to ease the claims submission and adjudication processes and bring many more patients into the protective fold of insurance. The more efficient the claims submission and adjudication process becomes, the lower the insurance rates will be which is a win for everyone. The ability to interpret disparately sourced transactional and medical data in a standardized way addresses this goal.

Pharmaceutical companies and other product manufacturers also seek market differentiation and increased penetration. Aggregated and de-identified usage and revenue metrics can help them understand how their products are being received and how they can improve their efforts to increase product distribution.

Solutions and answers to these challenges and questions live within data that exists today. We just need to solve the challenges associated with unlocking the data’s potential.

Signal vs. source
The value of standardized data is real and significant. The hard part is figuring out how to make this standardization a reality. Critical to formulating a successful plan for accomplishing meaningful standardization is understanding whether all data contributors in the veterinary community must store records using a single well-defined standard, or if it’s possible to convey the data, however stored, in readily comparable terms. In recent years, numerous attempts to suggest or even impose certain coding standards within veterinary practices have realized only limited success. The goals of these initiatives have been noble, and the standards themselves are often well thought out. The challenge, however, has been with adoption.

A suggestion for addressing this concern is to shift responsibility for this standardization away from where the data is stored and on to a data interpretation layer. This is achievable through a number of different approaches, depending on the level of accuracy and specificity needed from the output:

1. **Human-Generated**: Through careful review of each transaction or medical record from a source system, a correlation can be established between the source record and a standard record. Assuming the resources applied to this effort
include experienced in-practice professionals who understand the market and common veterinary PIMS usage behavior, this method would produce by far the most robust output. However, it’s also the most expensive approach from both financial and time perspectives and does not scale to service the entire industry.

2. **Automated via Rules**: By applying a well-defined set of rules to source transaction or medical record data, consistent output can be achieved very quickly. This is an attractive option for its speed, but it must be noted that the results are only as good as the rules and that the rules must be created and maintained by humans.

3. **Automated via Machine Learning**: This approach is often seen as the panacea for data standardization, and in fact it could be just that. One aspect that’s often overlooked however is that in order for machine learning to be successful, there must be a wealth of existing data for it to learn from and it would only ever produce results as accurate as the learning data. In other words, human-generated data must exist and be continually updated and maintained to ensure the ongoing efficacy of a machine learning approach.

One clear takeaway from examining the available approaches to leveraging a data interpretation layer to deliver standardization is that a hybrid approach is likely the most effective.

**Finding the carrot**

Nearly every veterinary practice uses a completely distinct set of inventory and service codes. This means different codes, different descriptions, as well as different approaches to coding structure and organization. These codes define the historical and ongoing financial and medical records within a practice’s system. The validity of any standardization effort relies on the quality of the source data, and so it’s logical to consider whether it might be possible to incentivize practices to improve the digestibility of their data, and how that might be done.

So, what can practices do to ensure their data can be accurately standardized by an interpretation layer? It really comes down to consistent and thorough articulation of concepts. Some key points to consider are:

- Ensure the services, products, and diagnostics used within a practice each have their own code and that the code’s description is clear, thorough, and non-duplicative.
- Ensure that each code is properly configured within the service, product, and diagnostic portions of your PIMS system.
- Minimize changing the meaning of codes over time wherever possible.
- If supported within the PIMS, inactivate unused codes to reduce the possibility of their inaccurate use.
- Avoid combining multiple services or inventory items into a single code. Many PIMS support the idea of ‘group codes’ or ‘linked items’; these are fine to use as they still ensure the integrity of the individual components.
- Many PIMS support ‘miscellaneous’ codes to allow for transacting non-standard or unconfigured items. Avoid using this functionality wherever possible.
- Avoid modifying code descriptions or adding text to them at the time of transaction. This is especially important if the modifications alter the underlying meaning of the code description.
- Adopt a logical but not overly complex code categorization structure.

Making adjustments to meet this brief set of best practices certainly represents a lesser investment than adopting an entirely new standard coding structure within a PIMS. Nevertheless, it still requires some effort and so understanding why a practice would invest resource in doing so is important.

- Thorough and meaningful coding structure within a practice allows for much more accurate and consistent reporting from within the PIMS. This is especially true over time with trending, seasonality, and period-over-period metrics.
- A clear and comprehensive coding structure improves proper charge capture as practice associates will not be burdened by having to navigate an unwieldy or ambiguous system.
- The clearer and more organized a practice’s coding structure is, the greater the standardization results with an interpretation layer will be.

**Low hanging fruit**

While previous initiatives have focused on wholesale adoption of standards, giving practices the option to participate to varying degrees could provide broader samples where participation is easiest and deeper data where practices have the greatest interest.

An incentivized participation-based solution is one where through its ongoing use, a practice effectively ends up specifying the correlations between their own data and an external standard. Consider an integrated reference lab solution or an integrated online pharmacy solution. In each case, there is tangible value to both the practice and the service provider in understanding how the in-practice codes relate to the service provider’s codes. Performing all of this mapping, or standardization, up-front is tedious and is a disincentive to service adoption, but if the service allowed this to be done in real-time, on-the-fly, that would yield a pleasant user experience all while accomplishing the requirement of correlating the disparate code sets.
Another solution is to apply an automated standardization approach and provide the practice with an interface to review and modify the results, especially those results with low confidence levels. The investment from the practice in this case may be higher, but if the service(s) provided with the standardized data is valuable enough it would work. Improved business intelligence, especially for corporate groups without a standardized practice management system, would be one highly valuable application for this approach.

The big fundamental
No, this is not referring to Tim Duncan of San Antonio Spurs basketball fame. Rather, this is acknowledging that any level of interoperability between disparate systems, whether in the veterinary community or otherwise, fundamentally requires a dedication and commitment to data access. Practices must be free to utilize their data however they choose and be afforded unfettered access to data-driven services that increase operational efficiencies, expand market reach, and ultimately and most importantly, improve patient health outcomes. Systems and service providers must be wholly committed to thoroughly protecting the integrity, security, and privacy of practice and pet owner data at all times. The promises of standardization depend on our collective commitment to these fundamental principles.

By reframing the initiative towards standards interpretation, the promises of standardization become more pragmatically and cost effectively attainable. Combining dedicated teams of industry data experts with targeted tooling, machine learning technology, and innovative approaches to data capture and interpretation, we can bridge the gap between where we are now and where we want to be.
Diagnosing and Treating Frustration-Yours (with Seemingly Uninformed, Skeptical, or Unappreciative Pet Owners)
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Working in a Veterinary environment we are presented with unique situations that require knowledge and skill that we have each spent years working to “master”. I put master in quotes because in the end we are always learning and never really become the “master” of it all. We have years built behind this knowledge base and yet we have only a few minutes to try to share this level of understanding to another human in working to help them care for their pet. Sometimes it is literally seconds and the person is an emotional fire-storm due to the situation, which only makes it that much more challenging. In the past the frustration I felt in not being able to help people through these difficult moments, led me to a place of name, blame, judge. At first it was the client- Clients are stupid and your cant fix stupid. Then it was my support staff- they just don’t care and are useless. Then my superiors (boss)- How can you connect with people when you see so many people. Then it became all of the profession which then included all of society. Finally ending in me, I sucked because I could not do it all.

Over time I have come to understand that this path to name, blame, judge did not work in my favor. I thought it was helping to off set the sinker that pulled me into my ocean of shame. Now I realize that feeding that “Cynical Serpent” as I was drowning in my ocean of shame, only led me to become disconnected and fell even more isolation. I started to fight, and began the path away from name, blame, judge and towards recognize, embrace, connect. During that path I recognized that I had to embrace this place of working with clients who are uninformed, skeptical, and appear unappreciative. I had to learn to not allow their space to become my reality. This is the story of that journey, a journey I know a number of us are on.

People often truly don’t realize the risk involved.
We have the basic knowledge of what is going on with cases and we have the responsibility to protect all those involved. We have the knowledge and capability to often “dissolve” a situation, yet we frequently don’t have the time to explain why we are short or direct during the process. People will then not understand why they are being excluded from the “facts”. They will not have the knowledge of the risk and we as a veterinary team are trying to keep them safe.

In this section of the lecture we will walk through a story which helps to show the value of awareness in understanding the emotions that come related to both the clients and staff in working through clients often not recognizing the risks involved. Taking a path to remembering that it is not about us, but in fact the clients are scared and confused. When they are in this place of fear and confusion, we then become the focus of the source of those emotions, but we are not the cause. It is not that we are to be a door-mat going forward, more about helping us each prepare for the reaction we will receive from people and find a way for ourselves to recover from it. I found that was the first step in working through this space and not jumping to name, blame, judge. Self-forgiveness is the foundation to a sustainable career in this industry and these situations require a large level of both self-awareness and forgiveness.

I didn’t get to say good-bye
I, as suspect many of us, value the human animal bond in many ways. I know that I entered into veterinary medicine related to the concept of zooeyia, the positive influence that animals have on humans. I wanted to support zooeyia and I think a number of you joined the profession with the same thought and drive. I accept that at times I will not like the outcomes of a case. I also accept the fact that sometimes I have to cause pain to the lives I work on to help them. There was one case that shook me to the core and taught me how to understand helping myself recover from those cases. I will walk the audience through this case and how I came to embrace the emotions of fear of the unknown related to a client’s perspective. This led me to begin finding the path to where we can help people through these difficult situations, by allowing us to recognize the impact it has on us as well as the client. Sometimes it really sucks being stuck outside the treatment room watching your loved one being cared for and not to be there to hold their hand. It sucks for the caretakers as much as the clients and pets. We go into emergency mode during those times and put our emotions on the self, what is our recovery for ourselves when that moment has passed? I don’t know about you but I used the suck it up philosophy, and well it didn’t work out so great in the end. Now from this case I learned a new path driven away from name, blame, judge and towards recognize, embrace, connect.

When we are emotional we often do not think straight
The vast majority of us in society are socially capable of interacting in a respectful and successful manner with each other on a daily basis. We read verbal and non verbal cues and respond to work to connect with each other as we go about our daily interactions. Then something traumatic happens and all of sudden we fall into flight/flight/freeze sympathetic dominance and our logic is out the
window. A state which was well received in the past to help us manage the risk and dangers of the world to survive. Without it we
would have died off as a species years ago. Although this response has served us well, it can be difficult when we require our brain to
be “on line” to work through those situations. At that point the emotions do not tend to serve us well. Both our clients and ourselves
can fall into this space and when we both do, it can be a path leading to much sorrow and anger.

Often when in this place of emotions, I found myself being driven from a place of shame and when I felt shame I needed someone
to blame. We will again walk through a situation where this very emotional challenge presented itself for myself. We will break
down the path and then the emotional acceptance that needed to be present to allow moving back to logic and toward the path of
connection. We are all humans and have emotions, learning to embrace them and recognize that we cannot be composed and under
control all the time is a giant step forward to finding sustainability in this profession.

**We do this everyday**
The concept that we do this everyday is nothing new. We can speak in “doctor talk” or “client talk”. Often when fresh out of school
we have to relearn how to speak as we will talk over a client’s head with all the “-ologies” and “-itises”. In starting 1 Life Connected I
began to recognize the many things that I do not do everyday that others do and I developed a new awareness of how the other side
feels. I hated it. NO, let me make it clear, I loathed it. The anger I would disperse to the other individual working in their “we do this
everyday” space was shocking. It was truly eye opening.

Yet the frustration I saw from them, when really they were just trying to help me and really wanted to help me, was just as
emotional. We will walk through this space looking at the perspective from both the client and our own in learning how to navigate
the frustration of coming from different “we do this everyday” spaces. This is not just frustrating to the client, this is equally
emotionally draining to us and our teams. Finding what is our path to ensure staying out of name, blame, judge when we enter into
this emotion and instead find presence with recognize, embrace, connect.

**Time**
Everyone doesn’t want to wait and let’s just be real, the concept of not having enough time in the day sucks! Our interactions with the
support of technology are causing life to be getting faster and faster, and people don’t have “time” to sit still. This is the culture that
we live in not just within our industry but our society as a whole. We might not like it, but it is the path we are going as we become
more and more reliant on technology. Think about it, when was the last time you used an actual physical map to get somewhere, not
one on your phone or the map provide by your GPS, but a paper map. Exactly.

This could be a lecture in itself when diving into the emotions related to feeling we never have enough time. Time is such a large
sinker in our daily tasks within the hospital. It can feel like the “to do” list is never ending and always growing. I honestly don’t have
an answer to this, it is one I fight with daily myself. What I have begun to understand is that the lack of time can drive strong
tendencies towards the path of name, blame, judge. Almost like a direct IV line to feeding the cynical serpent. We are normal in
going there, and recognizing we are there is the first step. In the end I have found that self-forgiveness is at the core in working to find
a path towards recognize, embrace and connect with the emotions related to time. For each of us that is unique. We will discuss some
key pieces that help along the way, and in the end the largest piece in creating the space is to recognize that we are not inadequate
from what we don’t get done, instead we are adequate in what we are able to achieve each day.

**The beast of burden of the profession**
Finally there is one large piece to cover in helping to embrace the space of our personal frustration in this industry. I call it the beast
of burden for the profession. The human animal bond drives much of our conviction to help animal and commonly a foundation in the
desire to be in this industry. That same human animal bond driving us is what drives clients and society to put us as veterinarians up
on an unrealistic, unsustainable pedestal. I don’t like it, and it angers me that this pressure is placed on me, you know the that I must
love all things about animals all the time. That I must constantly want to talk about animals. That I would want to hear a strangers
most horrible loss of a pet in first meeting them. That is not what I want. However I need that bond to be present, for if society does
not honor my profession, I cannot do what I desperately want to do, help animals.

That is our beast of burden, and I have no easy answer on how to embrace it. All I am trying to do is create the space to recognize
it. Each of us then can start the path to our unique journey in finding how to “see” the beast and embrace it. Without it owning us.
Without it defining our value. We can honor that beast of burden without losing ourselves in the profession and the first step is
moving away from name, blame, judge. I want to yell at society too, for putting so much pressure on us, for making us feel like we
are never are giving enough. When I did take that path, it almost ended in the loss of myself both mentally and physical.

Today to embrace the beast we each elect to connect with the sinkers and see them as the situation that they are, a situation and not
a representation of our value or worthiness. Connecting with these emotions of frustration and anger and disappointment, is a hard
journey to take. Let’s start the process one step at a time, because we are each unique and deserve to have our lives spoken for.
Top 10 Tips: How to Become a Recovering Perfectionist
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As most veterinarians, I struggle with hyper-achievement syndrome which is a form of perfectionism. Wikipedia defines perfectionism as “a personality trait characterized by a person's striving for flawlessness and setting excessively high performance standards, accompanied by overly critical self-evaluations and concerns regarding others' evaluations.”[1][2] It is best conceptualized as a multidimensional characteristic, as psychologists agree that there are many positive and negative aspects.[3] In its maladaptive form, perfectionism drives people to attempt to achieve an unattainable ideal, and their adaptive perfectionism can sometimes motivate them to reach their goals. In the end, they derive pleasure from doing so. When perfectionists do not reach their goals, they often fall into depression.”

In the end I found that having this drive for unattainable results was great in getting me into and through veterinary school, but it has not been so great in supporting a sustainable career in the veterinary profession. When you add in other core characteristics in which I, and most veterinarians have, such as being an introvert, having a type A personality, being more analytical in approaches to problems, have a strong ability for compassion, and being a people pleaser with workaholic tendencies, I have found it to be a recipe for disaster. Therefore I wanted to share my story in hopes to help others along their personal journey.

The backdrop for wanting to share comes from the concern that suicide and burn out is high within our profession. The AVMA released information about a survey they conducted looking into the mental health of the US veterinary professionals’ wellbeing and one finding from this review was that 1 in 6 veterinarians have considered suicide post veterinary school.[4] At this point honestly I could spend the remainder of this presentation referencing suicide cases and depression/burn out statistics in our profession, but in the end, I very easily could have been one of those statistics. I clearly remember the moment I had serious thoughts of suicide at one point in my life and I made the conscious decision that I wanted to live and recognized the need for help. The biggest part I want to share is that you are not alone in this journey. It is hard and continues to be hard, that is why the title to this presentation is “I Am a RecoverING perfectionist” Not “I Am a RecoverED Perfectionist”. This is an active decision I have to make the majority of most days and I know that this will probably be the case for the rest of my life.

The core basics I have learned through my journey I broke down into a few areas in which I cover in this presentation. At the center of it all being SHAME and the struggle I have in managing my personal shaming I do to myself each day. In helping me to understand shame I have studied the work of Brene Brown Ph.D., LMSW. She is a research professor at the University of Houston Graduate College of Social Work. She has been featured on multiple TED talks, including one that went viral which covered the topic of shame and vulnerability. She also has a number of books on the subject and was a focus of a PBS special. In the end shame is defined by Brene as “the intensely painful feeling or experience of believing we are flawed and therefore unworthy of acceptance and belonging”. No one likes to talk about shame, but in order for me, as a veterinarian, to start recovering from my perfectionism; I needed to confront my personal shaming head on.

I feel that the primary feelings which almost led me to a drastic decision lived in the shame I felt in my hatred of being a veterinarian and thinking I made a mistake. How could I feel that way? I worked so hard to get here and yet I hate what I do? What is wrong with me, I must be a horrible person. That was the thought process I followed in shaming myself and so producing my own feelings of unworthy of acceptance and belonging. In addition the thought of all the people I would have disappointed and the pressure to support my debt and bills added to the spiral down. I still work on this every day, and I want to share the tools I have found to help me along the way.

Below are the 10 key points I have recognized in my personal journey and I will share in this presentation. They are not presented in the order in which I recognized them in my journey, in addition none of them I feel I have truly conquered.

I would like to note that with that last statement, as a recovering perfectionist, I am OKAY with that.

1. Accepting that I will disappoint someone every day, and that someone may include myself.
2. Becoming comfortable with the unknown and learning the great things in life will come my way when I don’t “Plan” everything.
3. Showing vulnerability, especially in front of others.
4. Learning and accepting my limitations.
5. Accepting my mistakes as learning moments and understanding that they do not define me.
7. Shifting from things to experiences, and people not social media.
8. Accepting that emotions are okay and never to be judged.
9. My judgment of others is only me attempting to hide my personal shame.
10. Recognizing that I am not alone.

The final piece I would share that I have learned in this journey is that I am nothing if I don’t take care of myself. If I do not put my health and wellbeing first in my life, I cannot be there for others.

In conclusion; I truly believe that self-forgiveness is the foundation to a sustainable career in veterinary medicine. I strive for that each and every day. I am a work in progress and learning to love and enjoy every moment and honestly some days are easier than others.

Life is beautiful and I hope you work to find your own connection to its beauty.

Suicide and wellness resources
https://www.avma.org/ProfessionalDevelopment/Personal/PeerAndWellness/Pages/default.aspx
http://www.suicidepreventionlifeline.org/

References
We enter into this profession making a large investment of time and money into our education. This education covers the space related to medical knowledge, in both learning the physical skills and the science behind the practice of medicine. Hours and hours are dedicated to these areas as we start on this career. In fact years are committed to for all that enter into the profession to have not only the license to work as a veterinarian or technician, but to truly grasp the ideas and skill sets to perform the job. Along the way emotions develop with a variety of situations, we are expected to inherently learn how to cope through these. We stumble through in the process some of us falling into a resiliency, yet many left to flounder and struggle with staying connected to our passion and life.

Just as in training for a marathon, we cannot just focus on the medical knowledge and technical skills. If we want to look at sustainability in this industry, we have to start working to train for the mental piece as well. Marathoners will work to find ways to help with the emotions and mental struggles that will come along during that 26 mile running event. Now is the time for us to also find that path in veterinary medicine. In going through my CCFP certification, the program shared 5 key points that can be utilized to help manage the risk of compassion fatigue. Although an individual’s journey may not be “exactly” compassion fatigue, these tools are supportive in helping to find the emotions and mental struggles that will present themselves in this career for a number of reasons.

Before we dig into the 5 areas recognized for resiliency in our marathon of a career, one concept should be addressed and that is the idea that this is an individualized journey for each person. Therefore what brings each person to the place of struggle and discontentment can be different from another. In that same thought process, what works for one person may be different than for another. There is no “one fix it recipe” for the overall struggle. The overall theme of moving from Name, Blame, Judge and towards Recognize, Embrace, Connect is where resiliency lies for all aspects of the struggle within the veterinary industry.

**Self regulation**

This is simply the ability to shift from the sympathetic to parasympathetic state while staying fully engaged in the activities of daily living. Perceived threats are often triggered from an attachment trauma or the feeling of not living up to a certain idealistic vision. Self regulation takes us to the place of learning to recognize when these triggers have occurred and move from the reactive state of Name, Blame, Judge and towards the acceptance place of Recognize, Embrace, Connect. The sympathetic system is important to us, it is not about turning it off and walking around as perfectly run status stable machines. Learning to find the tools to move from the fight/flight/freeze mode first requires to acceptance that we are not flawed in being there.

This is shared in an earlier lecture through the brown gauze moment with the group. That time in practice where you have that day where so many things don’t go right and you never get a break. Where you are on constant “emergency mode” and work a full 12 hour day without, drinking, eating or even going pee. This can lead to a state of chronic sympathetic dominance.

Lets stop here and talk a bit about chronic sympathetic dominance. This can be looked at as pain versus suffering, which can be seen as acute versus chronic sympathetic response. Suffering is when we perceive the pain signal as a threat. In our world we cannot avoid the “pain” negativity of work, what we strive to do is work to prevent suffering. This is not a quick fix, but there are many ideas of how to break the cycle of chronic sympathetic dominance (suffering). Without accepting that we are normal for feeling the “pain” it can make it hard to leave the Name, Blame, Judge game. The Blame/Judge game can be towards ourselves or others. Breaking the cycle starts with recognizing that we are normal for feeling the “pain” and falling into the fight/flight/freeze response.

Once we recognize we are in the sympathetic response, the next step is to embrace the space we are in. For a number of individuals this can be brought on in how in learning how to bring ourselves into the physical moment and relax our body. Thereby allowing the pre frontal cortex to come back on line. This can be achieved in learning to put energy onto things that are within our control. This is not the space to dig deeply into the techniques, but a few will be discussed at a high level to provide insight. The goal will be to find what resource works best for the individual on their unique journey.

**Intentionality**

Choice versus demand. This is truly the place of where intentionality shows up and can be very difficult to find the path to moving from fear to choice is a true challenge. We will dive into a scenario where we walk through one such situation, where the individual moves from demand to choice. This is learning to see that there are real demands and perceived demands. Recognizing the fact that demands are present is a reality we cannot ignore. We cannot just put on a pair of rosy glasses and look at the world as all unicorns and rainbows, the reality is that there are real demands and risks. Not recognizing those can have consequences and that is a fact. Learning to embrace the demand and then recognize the perceived demands we are placing in addition to the reality is where intentionality comes in. Is it a demand or a choice. Do you have to go to work today? Or do you choose to?
Intentionality does not let us be the victim. Living in chronic sympathetic stimulation can lead us to feeling that the environment has control over us and defines our options. This does not mean we go the path of entitlement and avoid responsibility, that is not intentionality. It is moving from Naming, Blaming, Judging our environment as the only source to the pain. Recognizing the pain and embracing our choices related to it, fully understanding the consequences will lead to the journey of allowing intentionality back into our career. The 1 Life Connected message helps to make this connection to intentionality by encouraging individuals to find their why, and then connect that why with what they are choosing to support each day.

Viktor Frankl, Holocaust survivor, captures this concept beautiful in his book *Man’s Search for Meaning* when we stated: “Between stimulus and response there is a space. In that space is our power to choose our response. In our response lies our growth and our freedom.”

**Perceptual maturation**

This can be referred to as moving from burn out to burn through, in that we correct our perception on what we are truly experiencing in our career. Let’s walk through this progression as a caregiver in the world of the veterinary profession. First we start at the stage of fact that the outcomes of our work are all of who we are, and we aim for positive outcomes and goals only to realize that they are out of our control. The flaw being we place our sole worth based on those outcomes. We begin to embrace our powerless state and do the best that we can and begin to fall into the belief that others evaluation of our outcomes, doesn’t mean anything about our worth. Next as we begin to try to come to grips with the powerless place we feel within our profession, we start to find that our workplace is just a place to practice our internal conviction and vision through our code of honor and commitment to life. Nothing more, nothing less.

This then leads into the final stage of recognizing that our career is always going to ask more of us than we can give. The demands will never be satisfied with what we offer. We then move into a place were our value is not solely focused on outcomes, we become resilient to judgment of others and find a balance in our humility. In the end our worth is intrinsic not extrinsic. We begin to except that what we are capable of doing is enough and find peace in our actions and our level of commitment. Sounds super easy right?

As you can imagine this is not as easy as it sounds. This is where the concept of filling balloons that the 1 Life Connected message shares in helping to offset the sinkers that come into play in the struggle to stay content in our veterinary career. We cannot stop the sinkers, they do not however define us, and we have the peace to recognize and embrace the place to allow us to stay connected to our values and dedication to the profession. When the outcome of our cases no longer solely defines our value to the profession or this world, that is perceptual maturation. This often can fall into the space of filling a spiritual wellbeing balloons, however that may appear for each of us.

**Connection**

This simply means having a community in which we feel safe. A group of individuals that we can go to provide us inclusion and acceptance, but that which will also hold us accountable and challenge us in a way that we feel continually accepted. They help to identify shame within us and then show up with empathy to help diminish the shame and move out of the Name, Blame, Judge space and begin to travel towards the Recognize, Embrace, Connect journey to our authentic sustainable careers. These individuals are available to us within 48 hours and are a small group of people who may or may not know each other. These are the individuals that we know will answer that text or call late at night, but that are also not the ones to feed the “drama” or “pain” but in fact embrace us and then help us move forward instead of swimming in the ocean of shame with us.

We empower these individuals to tell on ourselves and to keep us true to our authentic self. They help us resolve that attachment trauma that is taking us to the place of perceived threats and so our sympathetic response. They listen and don’t interrupt, however don’t agree with all our points and thoughts and will push back with our best interest at heart. The goal is to define 5 of these individuals and approach them. Let them know you would like for them to be a part of the lifeline team you have developed for your self and what being in that space would look like. If they accept that commitment to you, then you set up check in and calls as you feel are appropriate. In the end you may actually be that same person for them.

These individuals help to fill a number of emotional and mental balloons. The goal is not to develop a group of people that commiserate the pain, but instead help us each move towards Recognize, Embrace, and then Connect!

**Self care**

Finally self care. Self care is probably the most discussed area we hear related to “combating” Compassion Fatigue and the struggles of this career. What the lecturer has found is that it is easy to say, we should exercise, meditate, eat healthy, have decent sleep, enjoy yoga, enjoy a “hobby” etc. etc. etc. But actually starting on this path to truly including self care really starts at that step of moving from Name, Blame, Judge in relationship to ourselves. There is always going to be someone that needs something from us, or a task that needs to be completed. Stopping and allowing the self care acts to occur starts with forgiving ourselves in that we cannot do it all. These self care acts fill all 4 types of balloons, mental, spiritual, physical, and emotional. It gets us out and being active instead of passive.
Yet we will continue to find every reason to not move forward with our self care. Justifying every reason why everything else is more important. Remember Recognize, Embrace, Connect. It isn’t easy, it is the path to sustainability in this profession. How do you find the time and space to develop self care in your routine? The answer to that question is – YES! This lecture will not provide that answer as it is already inside of you. What you do have now is permission to find your unique balloons and then fill them. What ever it is. Running, yoga, meditation, playing with your kids at the playground, taking a walk for 20 minutes in the middle of the day around the clinic, coloring, dancing, singing, painting, spending time in nature, snuggling with your pets at the end of a day, again what ever that is. These are the “fuel” that keep us going and stop us from sinking into that ocean of shame and falling into the fear and demand space. Finding any kind of aerobic activity for 15-20 minutes 3 times a week alone can have profound positive effects.

There are so many resources out there to help you find a way to stay on course with these self care dedications. We each have permission to go find them and allow them to be present. What balloons do you commit to filling?
Fear is imaginary, where as danger is a reality. Some where along the way we start to switch in seeing fear as the reality, when in fact it is something our mind produces. Perceived threats are something common in the profession, and can feel as real as a true threat. These perceived threats can trigger the fear response and move us away from a place of choice and towards a feeling of demand.

We enter this profession with a passion that drives us to sustain many sacrifices and struggles, and at some point the resiliency to move forward becomes extremely challenging. The fear of not doing enough, not being enough, not knowing enough, not working enough, not caring enough becomes our reality and the feeling of “appearing to be” a failure is no longer our fear of failing, but replaced with our danger of the truth that we in fact failing. This lecture takes a look at fear and danger and how it presents in the profession and then teaches the audience how to recognize the difference and direct our minds towards the reality of fear versus danger. Learning a path to embrace the risk and provide a road map in managing the ever-present unknown in this industry.

There are number of decisions that are required to be made day in and day out for a veterinarian team. In addition there are a number of technical skills that must be utilized in executing many of the treatment plans needed for the patients we work with. The other piece to consider for the environment of the veterinary team is the constant space of unknown. We never know when that patient will not respond well to a certain medication, have a reaction to a vaccine, fight compliance of care, or with no reason just not follow the books in their development and progression of disease and/or medical condition. It is no wonder that every day “mistakes”, “judgment errors” and “failures” occur. It is really the space that all teams live within daily. Some of these situations as many of us have learned, can lead to devastating consequences. These repeated consequences tell our brain that the risks are a reality and then the path to the feeling of true danger takes the place of risk.

Therefore it is safe to say that dealing with “perceived threats” and “failure” is a constant environment we live in as we enter into this profession. When you add in that many of us have perfectionist tendencies and are high achievers, this can be a recipe for much pain and suffering. In working to “cope” with this constant state of unknown and feeling of powerlessness, we can start to fall into the pattern of looking for who to blame. The shame that we can feel with these situations, often will drive us to a path of name, blame, judge. This path does not make as bad people, we are in fact normal that we go there. Often this is driven by the fear of being seen as weak or unworthy. We then look to vilify others to justify the outcome and then the real danger can come when we become the villain within our own minds.

Sustainability in this industry comes from learning how to adjust that path from name, blame, judge, and move towards recognize, embrace, connect. To help illustrate this concept, we are going to go on a journey related to a rather public event. Many of us heard of this event where a commercial airliner lost both engines on take off due to bird strike and the Captain and his co pilot landed the plane on the Hudson River. My sister’s dog (now my dog) actually had a front row seat to watch the plane land on the river. Using this story let’s take a look at the various aspect of dealing with risk and danger.

**When there are no risks, intentionality**

How easy is it to receive feedback and direction when learning how to play a new instrument, or play a new sport? Why is it we can laugh at ourselves as we fall over and over trying snowboarding for the first time, yet when it comes to a surgery we have never performed, it can elicit an anxiety attack? Often the risks that are involved with the situation at hand can influence how we embrace the idea of failure. This is not a bad place and in fact is a place that drives our success and continued pursuit of achievement. Recognizing that our intensions in both scenarios are the same can help provide comfort. We don’t walk into the “fun” new space with the idea of not wanting to succeed; we just place less value of influence on our self worth and value if we do not.

This is not an easy shift, recognizing our intensions can be the first step in this shift away from Name, Blame, Judge and towards Recognize, Embrace, Connect. We challenge ourselves to go and do things that many would never. For example, anesthesia is such an unknown every day, yet we walk into case knowing that the benefits outweigh the risks. It doesn’t mean we don’t critic our actions and learn from them, we should look at and hold ourselves and others accountable. The point is in remembering our intensions, especially when things go wrong. Lean on those intensions to help embrace the pieces that are difficult to review after the incident. Do you think that when Captain Sully and his co-pilot got on that plane that day they wanted to try something new and land the plane full of people, on the Hudson River? No, they were doing what they always did and making people lives better by helping them move around the country quickly. In addition, what were their intensions when they landed that plan on the river? To save lives! Even if it had not gone as well as it had, that didn’t change their intension of wanting to save lives.
Planning and being prepared feels really good
If I can just control everything, and I mean EVERYTHING, I can prevent all failures and remove all fear. This trap is such a common path we try. We throw ourselves into trying to control all variables and factors, to the point of it almost becoming an all consuming focus in our lives. Yes- check lists help prevent disasters and this could not be more evident then in the events that happened with the plane landing on the Hudson River.

Captain Sully and his co pilot leaned on checklists and training to help navigate through a potential disaster. They went into response mode and because of their training could keep the emotions in check through the events that unfolded. Captain Sully saved the lives of everyone on that plane that day, despite losing both engines at the lowest altitude then any other commercial airliner in history. Training and preparing along with checklist had their place that day. However this also bring up an important point, there will be times and situations we cannot train for. Like it was just noted, the plane lost both engines at the lowest altitude then any other commercial airliner. This is not an exact situation that either Captain Sully or his co-pilot had ever trained for. This leads us to the next point, the human factor.

The human factor-
There is one factor we cannot control and that is the human factor and the emotions that will present along with the brains perception and processing of the senses and the cues that present during each situation. There was a movie that was shared to tell the story of the events from that day Captain Sully landed the plane on the river and then the movie shared the events of the investigation that followed. Although all lives were saved, a multi million dollar plane was lost, in addition a question had to be examined, was the risk of landing on the Hudson the best course of action?

The movie is emotional and there is a point where other pilots are working through simulators to show that time and time again how they could get back to an airfield and land without taking the risk of landing on the Hudson. However, at that point Captain Sully shares one key factor- the human factor. In all the simulations, the pilots knew that they were going to have a bird strike and lose both engines. They calmly immediately turn the plane and head back. They were prepared and they were able to practice. Captain Sully was prepared as well, in that he followed a checklist as the incident happened, the key piece he did not have though was the knowledge of what he had left in his plane after the encounter with the birds. He and his co-pilot had never practiced for this situation. They needed at least 30-40 seconds to determine what they had and then make the decision with still limited information on how to minimize the consequences.

The pilots in the simulators were given multiple attempts to allow the positive results of landing at an airfield; Sully did not have that luxury as he had one shot. In addition when the pilot simulators where required to take the time for that critical evaluation and processing that occurred post fallout from the bird strike, which was essential in the success of that day for the crews and all the passengers on the plane, the simulator pilots were unable to make it back to any airfield. In fact it was clear that Captain Sully made the right decision.

Believe in your decision, yet learn from them
The instruments in the data collected post water landing did not confirm that both engines were destroyed, it actually suggested that one engine was still functional. However Captain Sully stated, he felt it go and was very confident that from his years of experience that he did not have either engine. For the outsiders it is easy to see options with having the benefit of time and data spread out clearly for them. For Captain Sully and his crew, all they had was the information in front of them. In addition, they have their body’s response to the apparent threat that absolutely can effect some logical processing, referring back to the human factor.

Captain Sully stood by his decision, however he also allowed the information to be reviewed and took the time to question his decision and ensure to learn from what they were sharing. He allowed the feedback to be shared, but did not stray from the fact that he knew what he felt. He did question his actions; he did not question his motivation and self-value with the decisions he did make. Later when the engines were both found it was confirmed indeed that the instruments were incorrect and in fact the Captain was correct, both engines were gone.

The emotional after effect-
In the time of critical decision-making we often go into response mode. Leaning on training and skills we practice for years, we put our emotions in check and dive in. In some situations that approach can be harder to do than others, but in the end in almost every situation our brain does what we need it to do. Put the emotions on hold and get through the situation. This also happened for Captain Sully and his co-pilot that day. You see them walking the plane after the evacuation to ensure that no one is left on the plane. Then you see Sully grab his pilot coat and flight clipboard as he is the last to leave the plane. During the next hours that followed, Sully and his co-pilot are determined to get a head count to ensure all lives were saved.

The issues that made this difficult is that people went out either side of the plane onto each wing, often splitting families and loved one up. As boats in the area scrambled to get to the plane to save the people, as this was in the dead of winter and now hypothermia was a large threat, people were taken to various places and no head count was made. People were then triaged and taken to a number
of hospitals spread throughout New York and New Jersey. It was not until late into the evening that they were able to confirm that all souls were accounted for and alive. In that moment you see in the movie, Tom Hanks, who played Captain Sully, relax and the emotion washes over him. I can almost feel the emotion that overcomes him, because I remember having those hit me post events too.

During the incident we put our emotions on hold, there will be a point sometime after that those emotions will come to us. This is not a weakness nor a place for name, blame, judge to show up. We want to embrace these emotions and work through them. It may be difficult, no let me re-phase that, it will be difficult. It is a step in working through embracing the situation as it is, a situation and not a representation of our worthiness or value. We have a right to those emotions, as our emotions are our internal passion connecting with our physical self. We deserve to let them happen and give them their space.

**Protect your time to recover**

During the Hudson incident, there was a hotel that worked hard to hide Captain Sully and the entire crew from the media and public to allow them the time to recoup. To give them the space to let go and allow the emotions and feelings from the day work through them before having to answer to anyone, or discuss the situation in which they too had just lived through. We don’t always have the ability to do this as the crew did that day; I mean I don’t think we could just lock ourselves away for 12 hours with every incident that presents itself. I will challenge us to each allow ourselves to take a moment, or a few to step away. Let the emotions hit. Step into a room alone to just be. Or take the time to call a close friend or loved one to reconnect and provide that acceptance we need to remind us that this situation does not define who we are. We often don’t feel we have the time for this as the day still has requirements. What I am saying is that it is not only normal to allow embracing our emotions, it keeps us connected and is required for our personal wellbeing and sustainability in this profession.

Often it will be implied that you just have to get back on that horse again after a traumatic incident so that you don’t let it take hold of you. Maybe we need to let it take hold of us, then move through it. It might mean that someone might have to wait, or people might be upset that you are not there for them. Well many of those people on that plane, missed their meetings and family events they were headed to. The crew was set to have a day full of travel and this caused delays and reschedules to occur. I don’t think anyone judged them for taking time to recover and if they did, well that is on those individuals that judged the crew.

You have a right to your emotions and have permission to work through them. “Mistakes”, “failures”, “judgment errors”, whatever you want to call them are going to happen, for our entire lifetime in this career. We cannot avoid them. You might be saying well Captain Sully did not “make a mistake” everyone lived. Yes this is true, but just because everyone lived does not mean his actions were the only option. Plus often after critical events, regardless of the outcome, we will start to evaluate if we could have avoided the situation with planning and preparing. Having our judgment of failure present on the front side of the event not the actual event itself.

In this profession we too will have cases that have unexpected outcomes, often these are not much different than that day on the Hudson. Sure some pieces could be preventable when we have all the facts. Sure we can learn from them when we review them after the fact. Sure there may have been other paths to take. But in the end our intensions were solid and from a positive place, we worked with what we had in front of us, we utilized our training and checklists where appropriate, and then we leaned on the human factor when appropriate. Finally we can start to recognize that each situation will have emotions that follow, and begin to allow us time to recognize those emotions, embrace them, and then stay connected with life. This path is imperative for our sustainability in this industry.

Our actions do not define our value and this is the underlying theme in learning to move from fear and stay within the place of risk. Moving away from name, blame, judge, and towards recognize, embrace, connect is a difficult path but it is one worth fighting for. You may have had opportunity to have a better approach to that client communication or surgery or whatever situation that presented itself during that day. The key point to take away is that YOU however are NOT a failure.
A ringing telephone in a veterinary clinic is a beautiful thing. I tend to laugh when I hear doctors and staff make a statement that they could get some work done if that damn phone would stop ringing. I wonder if they hear themselves speaking. What they fail to realize is that damn phone is the critical factor to them having an income and a job. We have to create a culture that wants the phone ringing off the hook. A culture where we train all of the staff to professionally answer the phone and take care of the clients efficiently and confidently.

99% of the time the ringing telephone is a client with a need or a want. This could be a phone shopper, a client wanting to schedule an appointment, a boarding reservation, checking on lab results, a perceived emergency, medication refill, patient status update and we could go on and on with the list. But again, the fact the phone is ringing is a great thing.

When answering the phone, don’t be fake. Let your staff’s personality come out in a professional and consistent manner. Assure they are welcoming and inviting but more importantly confident and efficient. We don’t need each phone call to last 10 minutes because we are unsure of ourselves or do not know how to control the phone conversations. The phone call should have some type of resolution. The phone voice should be pleasant and enthusiastic. It should sound focused and interested with switching of tones etc.

What we don’t want is a nasally monotone raspy voice that makes us want to hang up and call another veterinary practice.

There are service companies that give far more training to their staff for the phones than we ever do in the veterinary industry. I would encourage you to highlight 5-8 of the top categories on why clients call in and then set specific training for each. The categories could be Phone shoppers, Estimates, Pharmacy Refills, Euthanasia Requests, Complaints, Scheduling of Appointments, Boarding Reservations, and Lab Requests. Create a list of everything someone answering the phone might need to know about each category and then train the staff on them.

Continue the training with observation training. Have each staff member watching specific procedures in your hospital. Consider have them observe a spay, neuter, wellness exam, and dental. They are going to be working with clients or potential clients on the phone. Having observed first hand these procedure they will be able to more confidently describe and educate the clients on why they would want to choose your clinic. After the observation training is complete, move into role playing. Guess what? No one ever likes to role play but it can be extremely effective. Think of the person answering the phone as the face of the company. When you start to think the person talking on the phone is responsible for your entire image, you’ll agree to conduct a little more role playing to assure our message and dialogue sound professional and smooth will be well worth the time.

It is important to note it used to be if a client received poor phone service, they would shrug their shoulder and realize there is nothing they can do about it. Today, this has changed. We are seeing the use of social media as a way for clients to vent about their frustrations and describe their sometimes comical encounters with service employees. Don’t find your clinic on YouTube or Facebook with a ranting client playing an imitation of what your un-trained team member sounded like on the phone. Don’t be the next viral blog by a story of your staff being uncaring or insensitive to a client or potential client who appeared to of had an urgent matter. Work with your staff to understand they are on stage at all times when on the phone and you never know where the client will take the information and interaction or who they will tell. Err on the side of caution and assume each conversation is going to be blasted all over the web. It might not seem fair but this the technologically connected world we live in. After all, people post pictures of their dinner plates on Facebook. A juicy unprofessional phone interaction with your staff will really give them some substance for a post.

It is important to remember 99% of our phone calls are a client or potential client with a need or want. If they are making the effort to call, then they are wanting effort from the veterinary clinic to take some type of action. Many times, clients will call with a medical concern. It is important for the staff to realize their number one objective is to get the client into the clinic. Furthermore, the support staff needs to remember they are not doctors nor should they try to play the part. Their job is to alleviate fear from the client by confidently communicating to them they want to get the pet owner some answers. The best way to do that is to have the veterinarian do a nose to tail exam.

Plan for other scenarios that relate to the phones. Create some type of policy when family and friends call for doctors and staff. Create a system the staff understands and make sure it is not abused. We create a list for our doctors to identify when they want to be interrupted out of an exam room. For some it is when their spouse or children call for others it is when a specialist or referral doctor is calling. Communicate to the staff and it will alleviate a lot of missed opportunities on the phone. Make sure your messages are complete and accurate. This should include client name, phone number, client ID, date, time, message and initials.

Many of us think our phone call etiquette is above average. Let the clients decide. This should be part of your regular feedback from your clinic surveys. Ask the clients to rate how well your staff are on the phone. Ask specific questions such as “Do you feel
like all of your needs and wants are taken care of when you call into ABC Veterinary Clinic?” “Do you feel the staff to be knowledgeable and efficient when calling into ABC Veterinary Clinic?” “Would you agree you are taken care of in a timely manner when dealing with our staff on the phone?” “Do you feel your messages are returned in a timely manner when calling in?” Simply having the client rate your phone skills on a scale of 1-10 is going to do very little in giving you direction on where to improve.

Work with your staff and create a culture where a ringing telephone is viewed as a great thing and not a hindrance. Train the staff to be knowledgeable and professional. Create a culture where everyone jumps to answer the phone after they have been trained and checked off. Having untrained and unprepared staff answering the phone may do more harm than if the phone call was never answered. Now go turn up the volume on your clinic phones and wait to hear that beautiful sound.
Help! I Think I’m Spending Too Much on Staff Compensation
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Staff costs are one of the biggest expenses a practice incurs. What should the practice do if these costs are too high? First of all, it is important to drill deeper into the accounting and PIMS records to truly understand whether the costs are too high and WHY this is so. Secondly (and most importantly), changes need to be made in the practice to improve efficiency and costs.

Often times a practice will say “I can’t afford to pay more in salaries and benefits!” However, this issue of what a practice can “afford” to spend on compensation is a tricky one. There are a number of studies showing what a typical practice spends on compensation as a whole and what certain positions pay. These are excellent starting points in analyzing a practices’ compensation costs, but it is also necessary to delve deeper, not just into the components of compensation but into what that compensation “buys” such as efficiency, productivity, pet care and client service. Practices also need to be careful about how they deal with staff costs during challenging economic times; there is a fine line between a reasonable increase in the focus on productivity vs. reducing staff costs and hours to a point where client service and pet care suffer.

Getting started with the analysis
Establishing an effective compensation system is not an easy task. Any business owner or manager would like to believe that a simple formula is all that is necessary to establish initial compensation when hiring and to determine raises thereafter. However, the really smart ones know that it is more complicated than that. Remember too that the “compensation package” includes both the dollars paid to the employee for their work efforts as well as the benefits given them and should be established and reviewed in total.

The first step in controlling costs is to put an accounting system in place that allows the practice to carefully track expenditures. A chart of accounts listing all of the expenses normally seen in veterinary practices should be used to categorize costs in the accounting system. An excellent starting point is the AAHA Chart of Accounts, which was designed specifically for veterinary practices. All of the accounting systems commonly used by veterinary practices (QuickBooks, Peachtree, etc.) can be customized to fit these accounts. Just having the accounts set up correctly isn’t enough, however. The bookkeeper or other individual entering the bills (in this case payroll) into the system must do it carefully. In order to have the information needed to manage payroll effectively, the following categories should include:

- Owner compensation
- Associate veterinarian compensation
- Management compensation
- Support staff compensation (technicians, receptionists, kennel workers, etc.)
- Payroll taxes
- Health insurance
- Continuing education (primarily registration fees)
- Travel and lodging
- Dues
- Veterinary licenses
- Uniforms
- Other benefits—most practices will have other benefits as well; these categories should be set up individually

Some practices have used a catchall “Employee benefits” category but this makes it very difficult to analyze costs effectively and should not be used.

Staff compensation review
In the first stage of the staff compensation review, the following areas should be focused on:

- Review all transactions in the various accounts to make sure payments are in the right categories
- What are the dollar amounts of staff compensation listed in your profit & loss statement?
- How have these changed from the prior year?
- What is the percentage of gross revenue for staff compensation in the current year?
- How has this changed from last year?
- How do your percentages compare to the various published benchmarks?
- How do your salaries and benefits compare to the local market?
- What are the costs of various benefits (both in dollars and as a % of gross revenue) in the current year and in the prior year?
Calculate various staff efficiency metrics such as hours per transaction or number of full time equivalent staff per full time equivalent doctors

As with most of the important things in life, you get what you pay for with employees. In order to attract and keep quality people, a practice must pay the going wage for a certain job. Ideally, the practice’s pay scale is in the top 25% of the range for a certain position. In determining market pay, the practice owner or manager must not only look at national averages for compensation and benefits in veterinary clinics, but also must look at local pay scales and look at pay scales for jobs outside of veterinary medicine.

Once the above information has been gathered, it should be possible to determine if compensation is really higher than makes sense. The next step is to understand the drivers and which ones you can control.

- Are staff working too many hours?
- Is there too much overtime?
- Are the team members overpaid?
- Do the team members have the skills and knowledge necessary to work efficiently?
- Are the systems in the hospital well designed to lead to a productive workflow?
- Are team members there at the right times of the day and the right days of the week?
- How does staff compensation compare to doctor productivity? The problem may not be that staff costs are too high but that doctor productivity is too low.

### Staff efficiency metrics

One way to analyze staff numbers is to compare the number of FTE staff to the number of FTE doctors. It is important to remember, however, that increasing numbers of staff per FTE doctor may not make sense if doctor productivity is low. The "right" number of staff people per veterinarian will vary in financially successful practices and must be analyzed closely in conjunction with profitability. It is very easy to increase the number of staff per doctor to a level which allows for an easier work environment but seriously erodes profitability.

Another more specific measure of staff and veterinarian productivity and efficiency is the average number of staff and doctor hours per transaction. Average staff hours per transaction are calculated by dividing the total number of hours worked by all support staff members during a certain time period by the number of transactions incurred in that same time period. Average doctor hours per transaction are calculated by dividing the total number of hours worked by all doctors during the same period by the number of transactions incurred in that period. Average total hours per transaction are calculated by adding staff hours per transaction to doctor hours per transaction. As the name of the metric states, these results are averages for all transactions from the simplest examination to a long and complicated surgery. However, comparing these figures from period to period in the practice and to other practices can be very useful in determining areas for improvement.

### Too many staff hours

This is the most common reason for high staff costs and can have multiple causes—poor scheduling, lack of skills, poor workflow systems, etc. Improved staff utilization is a more critical element of successful practices now than ever before. Practices that want to become more profitable can no longer just count on fee increases to achieve this goal. Improved profitability and patient care must come from improved business practices. In addition, employees who are allowed to learn and grow and use more skills are generally happier in their jobs and more likely to stay with the practice than those who are only allowed to do less interesting tasks. Effective leveraging of employees is also critical to the productivity of the veterinarians and the practice as a whole. Veterinarians who delegate duties to appropriate staff members are able to see more clients and generate more gross revenue and profits. This increased profitability is essential to providing good quality medicine and surgery and to continual investment in team members in the form of increased salaries and benefits and increased continuing education.

In order to achieve optimal staff utilization, the practice must:

- Have a detailed, understanding of the skills staff members must have to provide outstanding patient care and client service
- Hire effectively—find and keep the employees with the right skills and attitudes necessary to achieve the practice’s goals
- Provide high quality, effective and ongoing training programs to both new team members as well as those who have been in the practice for awhile
- Have high levels of employee retention—reversing door employees aren’t around long enough to be efficient and effective
- Design and implement efficient policies, procedures and systems for getting things done
- Schedule staff, appointments, and surgeries effectively and in synch with each other
- Delegate effectively—tasks should be done by the lowest level person who can do the job properly
• Monitor staff activities frequently—in most practices staff are always busy doing something—what they are doing, however, is the key point—is it the most important activity that should be done?

• Regularly review staff utilization metrics

**Overtime**

Once a high level of overtime is noted, it is generally fairly easy to control via scheduling, hiring or individual performance management. Sometimes overtime is a temporary issue that occurs after one team member leaves and another is being hired. Other times it is due to poor scheduling. If overtime occurs regularly, staff members should generally be scheduled at 37-38 hours/week rather than at 40 to allow for this. Some employees actively seek overtime; if they are incurring it without permission; this has to be dealt with as would any other performance problem.

**Overpaid team members**

Most of the time, high compensation costs aren’t due to overpaid team members but it does occur when a practice has given regular raises to staff members who have been at the practice for a long time. Generally a cap has to be set on future raises; this can be difficult for employees to understand and must be handled with sensitivity if the practice wants to keep the employee. Sometimes this realization that an employee is grossly overpaid (especially compared to their contributions) can be a good start to increasing their responsibilities or moving them out of the practice.

Analysis such as that described above will take a little bit of time, but the results will be well worth the effort as it will allow the practice to better manage one of its largest cost
Selling a veterinary practice can be difficult logistically, financially and emotionally and the hardest part is often just knowing where to start.

The first thing a seller should do is think through why you want to sell and, more importantly, be sure that this action will accomplish your goals. Common reasons to sell include:

- Ready to fully retire
- Want to decrease workload
- Desire to change careers
- Desire to practice more medicine
- Health issues
- Divorce
- Location/demographics
- Management frustrations
- Financial issues
- Want to bond associate to practice

**Types of practice sales**

Outlined below are the most common types of practice sales and key characteristics of each.

**Full or partial sale to an associate**

- Common transaction type-easiest to complete
- Greatest opportunity to know if buyer/seller are right fit and have right skills
- Good method of seller succession planning
- Some financial investment from the purchaser is good, but seller shouldn’t count on it—associates often have limited financial resources
- Seller must be willing to provide full disclosure during the sales process and allow participation in management and financial decisions afterwards
- Usually lock in timeframe for rest of practice purchase (if applicable) as well as real estate purchase at time of initial transaction
- Usually best option for C corps because is generally a stock sale (assuming only selling part of the practice)
- Some financial investment from the purchaser is good, but seller shouldn’t count on it—associates often have limited financial resources
- Seller must be willing to provide full disclosure during due diligence period and should have reasonable expectations as to sales price, terms, etc.
- Usually lock in timeframe for real estate purchase at time of initial transaction (if applicable)
- Buyer will perform thorough due diligence

**Sell the entire practice to an unknown veterinarian**

- Harder for seller to find buyer—may use broker
- Seller should finance the deal only if absolutely must—outside financing is preferable
- Seller has less control over what will happen to practice after the sale
- Some financial investment from the purchaser is good, but seller shouldn’t count on it—associates often have limited financial resources
- Seller must be willing to provide full disclosure during due diligence period and should have reasonable expectations as to sales price, terms, etc.
- Usually lock in timeframe for real estate purchase at time of initial transaction (if applicable)
- Buyer will perform thorough due diligence

**Sale to a multi-practice corporate entity**

- Sellers may have this option if they meet the corporate buyers’ criteria—generally they want profitable practices with minimum of 2-3 doctors and over $1,300,000 in revenues in good locations
- If they want your practice, seller may get a great price and mostly in cash
- Corporate buyers are very sophisticated and knowledgeable; agreements are complex and seller must have a good attorney to help them through the process
• Seller needs to understand the employment provisions—usually can’t leave right away
• Corporate buyers often don’t purchase real estate-lease rates may be low
• Deals can be quick
• Generally asset purchases
• Will have to adapt to corporate expectations—this is not easy for all sellers

Gifting to a DVM child
• Uncommon transaction
• Tax issues must be addressed
• Community property issues may make the deal more complex
• A good lawyer and CPA familiar with both federal and state issues is critical in these transactions

Merger
• A great idea if seller/buyer are the right partners
• Can be very slow process but is still the fastest way to grow practice and increase potential associate buyers
• The quantity of practice mergers is increasing and that trend is likely to continue; however, absolute #s of mergers is still small

Finding a buyer/seller
If you are a seller, finding a buyer can be difficult and time-consuming. Use as many options as possible when looking for this person. Speak with your own associates, advertise in various publications, contact brokers and use networking wherever possible. You can also consider non-veterinarians if your state allows it. Determine what buyers in your area are looking for and then set about creating a plan for marketing your practice.

Key things buyers are typically looking for include:
• Location: primarily urban and suburban
• Type of practice: primarily SA
• High quality medicine and surgery
• Profitable practice
• Lifestyle--$$ and hours
• Attractive, freestanding facility

If all of these factors don’t apply to the practice being sold, the practice will need to be stronger in the remaining categories and the seller will need to work hard to emphasize the practice’s good points. Fix the problems that are fixable.

Advisors
Both sides will need advisors to help with the process; these are some of the common ones: practice consultant, practice appraiser, real estate appraiser, practice broker, CPA, and attorney. At a minimum, each side needs their own CPA and attorney—a good quality appraiser will prepare the valuation on a “non-advocacy” basis

Practice appraisal and feasibility analysis
A valuation of the practice is essential if you are selling to another veterinarian. This is typically done by the seller.

A feasibility analysis, sometimes called an ability-to-pay analysis, looks at the reasonableness of the appraiser’s value determination from the perspective of the buyer. The analysis attempts to answer the question, “Can the buyer successfully afford to purchase the practice at the value determined by the appraiser?” Buyers will typically have a feasibility analysis done to see if the earnings from the practice (salary and profits) will cover: repayment of debt incurred to purchase practice, increased income taxes related to practice purchase, and a cushion in addition to a fair market value salary for the work done in the practice.

Determining a FAIR value (versus one that favors either a buyer or seller) is important for several reasons:
• A value that is too high decreases the chances of finding a buyer for the practice and, should it be sold, increases the likelihood the buyer may be unable to meet the debt obligations resulting in business failure and the seller being forced to “take back” what is left of the practice.
• A value that is too low fails to reward the seller for efforts put into building the practice.
• An inaccurate value diminishes the chance of realistic personal or business planning.

Having a practice appraised is one of the single most important tasks a hospital owner will have done throughout his or her practice career, because, for most practitioners, the practice is the most valuable financial asset they own. Valuing a business properly takes skill, experience, knowledge and training. In order to determine a fair value, a competent appraiser is needed—one trained in accepted valuation techniques and experienced in valuing veterinary practices.

A valuation may not be necessary if you are selling to a corporate group but you will still need to have an understanding of the profitability of the practice and its financial strengths and weaknesses in order to assess corporate offers.
**Purchase/sale structure**
The seller will also need to determine exactly what will be included in the sale and how will the sale be structured. Is this a stock or an asset sale? Are you also selling the real estate where the clinic is located? In a corporate stock sale, the buyer is buying a % of the business as a whole, not part of the individual assets and liabilities and the buyer becomes responsible for known and unknown past liabilities of organization. In an asset sale, the buyer purchases individual assets—tangible or intangible and DOES NOT purchase liabilities. In general, sellers want to sell stock because of better tax advantages and buyers want to buy assets, both because of better tax advantages and the fact that they don’t inherit the liabilities of the old business. From a practical perspective, however, if the sale is a partial sale to an associate, it will be a stock sale. Almost all sales of 100% of a practice will be asset sales.

Obtaining the real estate associated with the practice is a critical issue for most buyers that must be resolved at the time of purchasing the practice. When and at what price will they get to buy it? If you’re going to lease the real estate for a while, you must deal with these issues: FMV rent, term, renewal options and type of lease.

**Legal documents**
An attorney can advise both the buyer and seller on the preparation of legal documents, from the pre-purchase agreement through the sales contract. Do not skimp on legal services. Common documents include: pre-purchase confidentiality agreement, letter of intent, letter of offer, memorandum of understanding, the purchase agreement, and a buy-sell agreement.

Planning is essential for a successful practice sale. Start early—5-8 years in advance if you need to do an S corporation election or need to significantly improve the practice value; 3-5 years in advance for fine-tuning value. A baseline practice appraiser 5-10 years prior to sale can be very useful in planning for the sale.
Making the decision to buy a practice is a scary proposition! Buying a business is the biggest financial transaction most veterinarians will ever engage in and it is critical to make a good decision. Many practice transactions go well but some don’t; fortunately, you can save yourself some heartache with better upfront analysis.

Three of the most important questions to ask in any practice acquisition are:

1. Do I really want to own a practice at all?
2. Do I want to own THIS practice?
3. Is this practice fairly priced?

Do I really want to own a practice at all?
This is the first major question to be answered; not everyone really wants to be an owner and it is better to figure this out sooner rather than later. Common reasons for owning include: increased earnings and equity in the practice, an increased ability to do things your own way, increased work schedule flexibility and work life balance and the personal satisfaction of having created and managed a successful business. The specific definition of each of these reasons will be different for each person—what does “increased earnings” mean, for example? It is important to nail down YOUR meaning because that will help you with decision making. If increased earnings is an additional $40,000/year, you might be able to do that as an associate. If it’s an additional $400,000/year, you are probably going to need to own a practice.

Of course, all of these benefits (no matter how they are defined) won’t occur immediately and the practice owner must be willing to put in the time and energy to get through the more challenging early years.

Any veterinarian considering starting a practice should give thought to these questions:

- Do you have strong organizational and managerial skills?
- Are you interested in the management side of the business as well as the medicine side?
- Is your physical health good?
- Are you willing to work the longer hours necessary to make a practice successful in the early years?
- Can you adapt to change?
  - How will owning a practice affect your family goals?
  - Does your spouse or significant other support this plan?
  - Will he/she be understanding of the time commitment?
  - If you’re a woman, will you want to take time off to have children? When? What will happen in the practice while you are out?
  - How do you feel about risk?
- Few veterinary practices go bankrupt, but many only achieve average financial success
- Are you willing to do what it takes to achieve your personal financial goals?
  - Do you have the personal financial resources to make practice purchase an option?
- What will be your spouse's contribution to the finances during this period?
  - Have you obtained the level of medical and surgical competency necessary to work on your own?

Do I want to own this practice?
When deciding on whether THIS practice is the right one, there are many factors to consider including:

- The type/quality of medicine practiced
- The facility and location
- The client base
- The practice culture

Practice culture is a bit of a murky concept; it’s one of those things everyone can recognize but is difficult to define. And while we tend to think of culture as something to worry about while you own a practice, it’s equally important to consider in a practice transition; i.e. when you are thinking of buying, selling or merging a practice. This can be difficult to ascertain if you don’t get to spend a lot of time in the practice before making a decision to buy it but it is important to do what you can.

So first of all, what is practice culture? Culture has to do with the values and beliefs important to the practice and the business style--how the company is run. My favorite way of defining culture is as described by Herb Kelleher, co-founder and former CEO and Chairman of the Board of Southwest Airlines: “Culture is what people do when no one is looking.”
Most people judge culture by things they see happening in the practice; for example, what behaviors are rewarded and condoned, how involved team members are in making decisions that involve them, and whether the practice really focuses on a good client experience or just gives it lip service.

Because culture is all about day to day values and actions, it is a critical component of a good transition fit. As a buyer, you need to know yourself, the kind of practice you want and how you plan to operate the practice before committing to a partnership or the 100% purchase of a practice.

Buying a practice with a culture that is not a good fit for you often ends badly. I once worked with a buyer who purchased a very profitable practice located in a blue-collar area. The practice focused on preventive care at an affordable price, had been in the area forever and had a great reputation. The buyer wanted this practice because of its location and purchased it with the idea that he could educate and motivate the clients to provide more advanced care for their pets (and pay for it.) It is generally possible to make some changes to a practice but a total makeover usually doesn’t work; in this case the clients left to find the kind of care they wanted (and used to have) and the practice owner filed for bankruptcy.

And of course, culture isn’t just about client care and relationships. It is also about the way a practice operates internally. If you are a buyer, think about your management style and how that will fit. For example, how do you handle conflict? Are you good at listening to both sides and facilitating a resolution? Or more the type to make a snap decision about the problem and expect people to buck up and shut up? If you are the snap decision type, you are going to work better in a more structured practice environment with a practice manager who can handle the fuzzier aspects of conflict resolution.

If you are buying a fixer-upper practice, a very critical question revolves around the kind of clients you want and how you will attract them. It used to be that a veterinarian could open a practice anywhere and it would be a success. However, many areas have become very saturated with veterinary practices. Pet owners in the area who want a veterinary practice already have many to choose from. If you are trying to grow a practice in an area that already has many options, you must have a clear vision of what is going to be unique about your practice either from a medical, a client service or a business model perspective. And, as importantly, how are you going to communicate that difference to potential pet owners? In most areas, there aren’t large numbers of pet owners who don’t already have a practice with which they are affiliated. This means your practice has to not only reach out to pet owners with the message that your practice is unique and better but also persuade them to leave their current practice. Not an easy task.

**Is this practice fairly priced?**

A valuation of the practice is essential. This is typically done by the seller. At a minimum, the seller needs to provide an asking price. Buyers typically don’t pay to have a practice appraisal done. They work with a veterinary financial advisor to determine the reasonableness of the price through the use of a feasibility analysis and other information review.

A feasibility analysis, sometimes called an ability-to-pay analysis, looks at the reasonableness of the value or asking price from the perspective of the buyer. The analysis attempts to answer the question, “Can the buyer successfully afford to purchase the practice at the value determined by the appraiser or the seller asking price?” Buyers will typically have a feasibility analysis done to see if the earnings from the practice (salary and profits) will cover:

- Repayment of debt incurred to purchase practice
- Increased income taxes related to practice purchase
- “Decent” living

The feasibility analysis is one of the most valuable tools available for negotiation with a seller.

There are, of course, many more steps to the purchase process but they are “easy” in comparison to answering the above questions. These questions are the critical starting point.
What You Don’t Know About Starting a Practice Can Kill You
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Many veterinarians are considering starting their own practices when they can't find one to purchase that fits their criteria. Practice startups can be very successful but they are hard work and harder now compared to 10 years ago. The owners who succeed with a startup have done their homework and planned extensively before moving forward.

Veterinarians are frequently ill prepared by training or experience for the major business analysis and decision-making associated with starting a practice. For most, these decisions are some of life’s most pivotal choices with long lasting lifestyle and financial implications. Such decisions should be based on sound information collected in the course of the due diligence and the qualified advice of experienced consultants and advisors.

Some of the critical issues to be considered include:

- What does the veterinarian want to get from owning a practice?
- Why a startup compared to a purchase?
- What is going to be different about your practice? Why will pet owners want to come here?
- Personal traits/factors needed: time, energy and money needed to get the practice off the ground, support and understanding from family and friends
- Where will the money come from?
- Demographic analysis of the proposed area
- Cash flow projections—can you live without a salary in the early days? Do you have a solid understanding of how much things will cost?
- What advisors do you need? Practice consultant, CPA, attorney, real estate broker, other? How do you select good quality people?
- What are other planning steps necessary for a successful startup?

What do you want to get from owning a practice?
This is the first major question to be answered; not everyone really wants to be an owner and it is better to figure this out sooner rather than later. Common reasons for owning include: increased earnings and equity in the practice, an increased ability to do things your own way, increased work schedule flexibility and work life balance and the personal satisfaction of having created and managed a successful business. Of course, all of these benefits won’t occur immediately and the practice owner must be willing to put in the time and energy to get through the more challenging early years.

Why a startup instead of purchasing a practice?
This is the second major question to be answered. In my experience, startups are generally a default option for those who do them. The new owner would rather have purchased a practice but there weren’t any suitable hospitals for sale in the area they wanted to be in. Pursuing a startup as a default option isn’t a bad thing to do as long as the new owner has the skills, financial resources and commitment to make the practice a success. It generally takes more time and effort to make a startup a success as compared to a practice purchase and it takes longer for the owner to be able to take home a salary and receive a return on his/her investment.

What is going to be different about your practice? Why will pet owners want to come here?
These are the next critical questions and perhaps even more important than the first two. It used to be that a veterinarian could open a practice anywhere and it would be a success. However, many areas have become very saturated with veterinary practices. Pet owners in the area who want a veterinary practice already have many to choose from. If you are going to start a new practice in an area that already has many options, you must have a clear vision of what is going to be unique about your practice either from a medical, a client service or a business model perspective. And, as importantly, how are you going to communicate that difference to potential pet owners? In most areas in which startups take place, there aren’t large numbers of pet owners who don’t already have a practice with which they are affiliated. This means the new practice has to not only reach out to pet owners with the message that their new practice is unique and better but also persuade them to leave their current practice. Not an easy task.

Personal traits necessary for success
Any veterinarian considering starting a practice should give thought to these questions:

- Do you have strong organizational and managerial skills?
- Are you interested in the management side of the business as well as the medicine side?
- Is your physical health good?
• Are you willing to work the longer hours necessary to make a practice successful in the early years?
• Can you adapt to change?
• How will owning a practice affect your family goals?
  o Does your spouse support this plan?
  o Will he/she be understanding of the time commitment?
  o If you’re a woman, will you want to take time off to have children? When?
• How do you feel about risk?
  o Few veterinary practices go bankrupt, but many only achieve average financial success
  o Are you willing to do what it takes to achieve your personal financial goals?
• Do you have the personal financial resources to make practice startup an option?
  o What will be your spouse’s contribution to the finances during this period?
• Have you obtained the level of medical and surgical competency necessary to work on your own?

Financing
There are a number of lenders who regularly finance veterinary practice startups. Terms can vary; it is worthwhile to talk to several. It is important that the borrower have a good credit history, nothing in their background to indicate they will be a poor owner/manager and a well-thought out plan for the new practice.

Demographic analysis of the proposed area
A demographic analysis of the proposed area can be very helpful; although it may show that the area doesn’t need any more veterinarians. The quality of the analyses is totally dependent on the quality of the input data.

Cash flow projections
Cash flow projections for the first 3-5 years are a critical component of the startup planning process. Most veterinarians hire a veterinary specific financial advisor to put these together. How much revenue will be generated in the first few years is often an unknown but estimates are still possible. It is better to be conservative with revenue estimates and happily surprised than vice versa. Expenses are easier to estimate than revenue, but it is important to include ALL expenses in the projections. The rougher the estimate, the less likely they are to be accurate. The projections serve as a reality check for the project and a way for a potential owner to gauge the risk of the project.

As a part of preparing the projections, a detailed list of the total funds needed for the project is critical; this should include: leasehold renovation, equipment and supplies, startup costs, and working capital. Compiling this list in detail insures important items aren’t missed and, if the amounts start to go over what a lender will be willing finance, serves as a framework for prioritizing the costs.

What advisors do you need?
Most veterinarians starting a practice utilize the services of a veterinary financial advisor to prepare the cash flow projections, an attorney to draw up the legal entity papers and review the facility lease and other documents and a real estate broker to help locate the property and negotiate the lease. Often times these advisors can also help with other aspects of project planning. The working capital borrowed from the bank can be used to pay for these costs.

What are other planning steps necessary for a successful startup?
Preparing a business plan even if your lender doesn’t require it is a great way to think through many of the aspects of starting a practice. This advanced planning helps assess the feasibility of the project as well as reduces the number of tasks that have to be done close to or after opening. Common components of a business plan include:

• Executive Summary
• Description of the Veterinary Profession
  o Challenges and opportunities
  o Plans for capitalizing on opportunities and overcoming challenges
• Drawings of Practice Layout/Building Blueprints/Site Drawings
• Products and Services to Be Offered
  o Medical & surgical, boarding, grooming, retail, other
  o What will be unique about your practice
  o What will you offer when opening
  o What are plans for future
• Demographic Analysis
• Competition
  o Description of nearby colleagues
  o Services offered
  o Hours
  o Proximity
  o Size and type of facility
  o Nature of practice
  o Primary clientele
  o Strengths and weaknesses
  o Other potential competitors
• Marketing Plan
  o Trade area boundaries
  o Marketing programs
  o Costs of marketing plan
• Operations
  o Staffing
  o Hours
  o Management strengths and weaknesses
  o Plans to overcome weaknesses
  o Responsibilities of owners
  o How will basic business administration be handled--reception, accts receivable, accts payable, payroll, inventory control, kennel, etc.
  o Cash Flow Projections

Successful startups are very possible but they involve a lot of work. Planning is essential for a successful practice startup--start early and be thorough in your research and preparation. Get help in the areas in which you are not an expert.
Essentials in compensation
A checklist on what you need to know

- Explain what drives wages
  - Overhead
  - Skills
  - Specialization
  - Productivity
  - Team communication
  - Longevity?
  - Job Market

- Have an Offer Letter
- Have a Compensation Statement (page 6)
- Wage and Hour Division - DOL (FLSA)
  - Exempt:
    - Outside Sales
    - Must Be Paid Over $23,600/year * - law pending
    - Guaranteed Regardless of Hours Worked
    - Some Exceptions (leave, suspension, business closed) - Non-exempt:
    - Entitled to Overtime Pay
    - Most Employees Covered by FLSA Assess Duties

Understand how to calculate work hours

Commuting time must be paid if the employee is required to another work site other than their normal work-site. They must also be paid when requested to go back and forth from the work-site for emergency situations at their normal place of employment.

On Call – If you require the employee stays on work-site while waiting for work assignments, they must be paid for the time. If the employee can use the time for their own enjoyment off the premises, it is not payable. If the employee has little control over the time, that is payable.

Sleep Time – If you require an employee to be at work for over 24 hours or more you must generally count sleep time as payable.

Lectures, meetings, and training – if you require the employee to attend you pay for the time, travel time included if it is not at the normal work-site. The only time you don’t have to pay for the time if ALL these points are met:

- The employee attends a meeting outside of regular work hours.
- Attendance is voluntary.
- The instruction is not directly related to the employee’s job.
- The employee doesn’t perform any productive work during the instruction session.

Meal and rest breaks – You don’t have to pay for an ACTUAL meal period. The employee must be completely relieved of duty so the employee can enjoy a regularly scheduled meal.

This counts for scheduled breaks as well. Some states have specific rules over the federal law, be sure to check. Many employees expect a paid meal break during an 8 hour or longer shift. Such breaks may increase productivity and team satisfaction.

Record keeping requirements

1. Name, Address, Occupation and gender
2. Birth date if under 19 years old
3. Hour and day when workweek began
4. Total hours worked each work day
5. Total daily or weekly earnings
6. Regular pay rate for any week when overtime was worked
7. Total overtime pay for work week
8. Deductions and additions to wages
9. Date of payment and pay period covered

Garnishments

1. Wage attachment
2. Know what your state requires
3. Payment Agreement (page 6)

Health care coverage
- Do shop this out yearly
- HSA’s – a trend of the future
- Increase coverage with longevity
- COBRA – Consolidated Omnibus Budget Reconciliation Act –
  - 20 or more employees and you offer health insurance
    - Termination under all cases except gross misconduct.
    - Their hours have been reduced.
    - They are eligible for Medicare
    - Allows for continued coverage by the worker up to 18 months on average.

Vacation/personal time
- Migration to PTO – build up a bank of time (page 68 in Employee Manual section)
- Increase with time
- Caps
- Watch your STATE LAWS
  - Defined more by State than Federal Law
- Explicit Policy

Other benefits
- Consider your package carefully
- Cafeteria Style
- Discounts
- Bonuses
  - Attendance
  - Training
  - Performance
- Employee Assistance Program (EAP)
  - Emotional
  - Family
  - Substance Abuse
  - Work or Financial Concerns
  - Relationship Issues
  - Simple
  - Unexpected
  - Reinforcement
  - Fair

What about employment contracts
- Separate arrangement
- Wage – production – ProSal
- All benefits outlined
- Dues/licenses
- Continuing Education
- Restrictions
- Standards of conduct
- Termination or non-renewal
Trends in benefits & compensation

• Same Sex Partner Benefits
• Sports
• Community Activity
• Tuition Reimbursement
• Choice Models on Health Insurance
• Discount Arrangements
• Service Awards
Position: [insert title of position]
Preferred Start Date: [insert preferred start date]

NAME, I’m pleased to offer you a full-time position as our JOB POSITION at [Practice Name].

As we move forward, my goal is to ensure that you have the tools and resources to be successful in our thriving veterinary practice. We are excited about having you start at the hospital, particularly with the skills and creativity you will bring to our team.

In becoming a [insert title of position], your ultimate goal will be to assure quality care to patients, exemplary service to our clients and to be a valued member of our team. You will be key to the growth of our services for patients and clients, and critical in the development of our practice.

Your compensation package will include a starting wage of [wage], as well as the benefits included are attached with this letter. Or, we have agreed upon compensation of twenty-three percent (23% of production). Compensation is subject to all taxes and withholdings are required by law or the policies of [Practice Name]. Look this over carefully, we trust that you agree this is a very fair offer and you pleased with the total compensation being provided to you.

You will receive your full benefits package after a 90-day introductory period. Afterwards, medical, dental, and vision insurance plans are provided as well as paid time off (PTO) and paid sick time as defined in the hospital employee manual. On your first day, we are scheduling you to speak with our Human Resources Team Leader who will guide you through all the benefits available as a valued team member here at the practice.

In accepting our offer of employment, you certify your understanding that your employment will be on an at-will basis, and that neither you nor any employee of the [Practice Name] have entered into a contract regarding the terms of the duration of your employment.

Your start date is [insert preferred start date], and you should ask at the reception desk for me. Please come dressed in [describe dress] with the appropriate footwear. Everyone at the practice is very excited to meet you and there will be a reception in your honor at [time].

Please sign and reply to this email to let us know that you have accepted the position. If you have any questions, then please let me know through [email address] or by contacting me by phone at [phone number].
Year end total compensation statement  
Example after formatting

Prepared for: Dr. Hope Smith  

As a valued team member, Brown Veterinary Hospital is pleased to present you with your Total Compensation Statement. This statement shows the pay and benefits that were afforded to you as part of your total compensation package over the past year. Please contact Dr. Brown or the Hospital Administrator if you require additional information about your compensation package.

<table>
<thead>
<tr>
<th>Hospital's Annual Cost</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Prepaid for: Dr. Hope Smith</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Salary/Wages</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Your gross earnings for the year were $72,658. As stipulated in your employment contract, you were afforded a guaranteed base salary of $65,000 for the year. Your actual earnings based on production exceeded this amount by $7,658 (or 12%).</td>
<td>$ 72,658</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pension Plan</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>The practice matched contributions of 3% to your SIMPLE retirement plan.</td>
<td>$ 2,180</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Health Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>The practice paid 50% of the premium for health insurance coverage</td>
<td>$ 6,870</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dental Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Dental insurance through our group policy was provided through ABC Dental Insurance Co. You paid 100% of the premium for this coverage.</td>
<td>$ -</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>AFLAC Supplemental Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Supplemental condition specific coverage was provided through AFLAC. You paid 100% of the premium for this coverage</td>
<td>$ -</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Life Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>The practice paid 30% toward the premium for a term life insurance policy</td>
<td>$ 3,700</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Disability Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Disability coverage was provided through AFLAC. You paid 100% of the premium for this coverage</td>
<td>$ -</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Continuing Education</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>You used $923 of the $1,500 provided for your continuing education</td>
<td>$ 923</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Professional Dues, Memberships and Licenses</th>
<th>Annual Cost</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>License renewal</td>
<td>$ 125.00</td>
<td></td>
</tr>
<tr>
<td>Professional medical association dues</td>
<td>$ 385.00</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Additional Benefits</th>
<th>Annual Cost</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Travel/meal reimbursement</td>
<td>$ 285.00</td>
<td></td>
</tr>
<tr>
<td>Uniform allowance</td>
<td>$ 138.00</td>
<td></td>
</tr>
</tbody>
</table>

| Total Professional Dues, Memberships and Licenses Paid by the Hospital | $ 510.00 |  |
| Total Additional Benefits Paid by the Hospital | $ 423.00 |  |

<table>
<thead>
<tr>
<th>Total Compensation</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$ 87,344</td>
<td></td>
</tr>
</tbody>
</table>

Brown Veterinary Hospital also paid the following expenses based on your wages:

<table>
<thead>
<tr>
<th>Hospital's Annual Cost</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Social Security/Medicare</td>
<td></td>
</tr>
<tr>
<td>This is the government-required portion paid by the hospital for your social security/medicare</td>
<td>$ 5,558</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Workers’ Compensation</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>The amount paid by the hospital for workers’ compensation</td>
<td>$ 1,875</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>State Unemployment Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>The amount paid by the hospital for state unemployment insurance</td>
<td>$ 240</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Federal Unemployment Insurance</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>The amount paid by the hospital for federal unemployment insurance</td>
<td>$ 36</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total Paid by Hospital for Workers Compensation and Federal/State Taxes</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$ 7,729</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hospital's Annual Cost</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cash Compensation</td>
<td>$ 72,658</td>
</tr>
<tr>
<td>Total Retirement Contributions</td>
<td>$ 2,180</td>
</tr>
<tr>
<td>Total Paid for Health Benefits</td>
<td>$ 6,870</td>
</tr>
<tr>
<td>Total Income Protection</td>
<td>$ 3,780</td>
</tr>
<tr>
<td>Total Miscellaneous Benefits</td>
<td>$ 1,875</td>
</tr>
<tr>
<td>Total FICA, Workers Comp., &amp; State/Fed. Unemployment</td>
<td>$ 2,729</td>
</tr>
</tbody>
</table>

| Total Cost to the Hospital | $ 95,073 |  |
The compensation shown above includes time off from work during which the hospital paid your wages. The cash value of paid time off afforded to you during the year is shown below.

<table>
<thead>
<tr>
<th>Paid Holidays</th>
<th>Hospital's Annual Cost</th>
<th>$1,956</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paid Time for Continuing Education</td>
<td></td>
<td>$838</td>
</tr>
<tr>
<td>Paid Time Off (PTO)</td>
<td></td>
<td>$3,960</td>
</tr>
<tr>
<td>Estimated Benefit Included in Totals Above</td>
<td></td>
<td>$6,754</td>
</tr>
</tbody>
</table>
Employee Manual
Mark Opperman, BS, CVPM and Sheila Grosdidier, BS, RVT
Veterinary Management Consultants
Evergreen, CO

Employee manual
Why?
• Introduces
• Provides
• Shares
• Informs
• Explains
• Contributes

Information provided in the Employee Policy and Procedures Manual draft is for informational purposes for [Practice Name] to use as a guide to prepare its own, personalized employee manual. Every effort has been made to offer veterinary practices the most current, correct, clear, and accurate information possible. State and federal laws are constantly changing and it is the veterinary practice's responsibility to review the manual for accuracy, completeness, timeliness, and validity of any information that will be used. In view of the possibility of human error or changes in laws and regulations, neither VMC, Inc., nor any other party that has been involved in the preparation or publication of this employee manual warrants that the information contained herein is in every respect accurate or complete, nor shall they be responsible for any errors or omissions or for the results obtained from the use of such information. All information is subject to change without notice. VMC, Inc., excludes and expressly disclaims all express and implied warranties not stated herein. VMC, Inc., assumes no liability for the use of any of these policies and procedures.

VMC, Inc., does not provide legal advice. [Practice name] should have the finalized employee manual reviewed by legal counsel to ensure that all information contained in the final employee manual is in compliance with current state and federal laws directly relating to the size of the practice.

Employee manual checklist
Components to include in your employee manual
1) **FOREWORD** ..........................................................................................................................
   1) Welcome .............................................................................................................................

2) **GENERAL CLINIC INFORMATION** ................................................................................
   2.1 Organizational Structure .................................................................................................
   2.2 Our Motto ........................................................................................................................
   2.3 Mission Statement ............................................................................................................
   2.4 Hospital Philosophy .........................................................................................................
   2.5 Hospital History ..............................................................................................................

1) **DIVERSITY** .........................................................................................................................
   1) Equal Employment Opportunity Commitment to Diversity ............................................
   2) Anti-harassment Policy and Complaint Procedure ...........................................................
      1) Individuals and Conduct Covered ...................................................................................
      2) Non-retaliation ..................................................................................................................
      3) Complaint Process ..........................................................................................................
3) American with Disabilities Act .........................................................................................
4) Immigration Law Compliance ..........................................................................................

2) EMPLOYMENT ..........................................................................................................

4.1 Employee Classification Categories ..............................................................................
   4.1.1 Nonexempt employees ............................................................................................
   4.1.2 Exempt employees ................................................................................................
   4.1.3 Introductory Period Employee .............................................................................
   4.1.4 Full-time Employee ...............................................................................................}
   4.1.5 Part-time Employee ................................................................................................
   4.1.6 Volunteer (optionally, Observer) ...........................................................................

4.2 Internal Transfers/Promotions ....................................................................................

4.3 Nepotism, Employment of Relatives and Personal Relationships .................................

4.4 Misconduct ..................................................................................................................

4.5 Counseling, Discipline and Performance Correction ....................................................
   4.5.1 Progressive Disciplinary Process ..........................................................................}
   4.5.2 Disciplinary Process ...............................................................................................}
   4.5.3 Appeals Process .......................................................................................................
   4.5.4 Performance and Conduct Issues Not Subject to Progressive Discipline ..............

4.6 Separation of Employment ..........................................................................................
   4.6.1 Separation of Employment ....................................................................................
   4.6.2 Return of [Practice Name] Property .................................................................

5.1 WORKPLACE SAFETY .................................................................................................

5.2 Drug-Free Workplace ...................................................................................................
   5.1.1 Introduction ............................................................................................................
   5.1.2 Consequences of Alcohol/Drug Abuse ...................................................................
   5.1.3 Required Testing ....................................................................................................
   5.1.4 Follow-up ...............................................................................................................}
   5.1.5 Consequences: .......................................................................................................}
   5.1.6 Crimes Involving Drugs: ......................................................................................
   5.1.7 Inspections .............................................................................................................

5.3 Workplace Bullying ......................................................................................................

5.4 Violence in the Workplace ..........................................................................................
   5.3.1 Prohibited Conduct ...............................................................................................}
   5.3.2 Reporting Procedures .........................................................................................
   5.3.3 Risk-Reduction Measures ....................................................................................
   5.3.4 Investigation ...........................................................................................................
   5.3.5 Corrective Action and Discipline ...........................................................................

5.4 Weapons-Free Workplace (optional) ............................................................................

5.5 Fire ..............................................................................................................................

5.6 Tornado or Other Severe Weather Conditions ...............................................................

5.7 Accidents .....................................................................................................................
5.8 Hazardous Materials ..............................................................................................................................................
5.9 Dosimeter Badges ................................................................................................................................................
5.10 Safety Equipment ................................................................................................................................................
5.11 Safety ....................................................................................................................................................................
5.12 Smoke-Free Workplace ......................................................................................................................................
5.13 Injury and Illness Prevention Program (optional) ............................................................................................
5.14 Internal Investigations and Searches ................................................................................................................

6 WORKPLACE EXPECTATIONS..............................................................................................................................................

   6.1 Confidentiality and Hospital Records ................................................................................................................
   6.2 Conflict of Interest ..............................................................................................................................................
   6.3 Outside Employment ..........................................................................................................................................
   6.4 Solicitations, Distributions and Posting Materials ..............................................................................................
   6.5 Conflict Resolution ............................................................................................................................................
   6.6 Attendance and Punctuality .................................................................................................................................
       6.6.1 Punctuality .................................................................................................................................................
       6.6.2 Disciplinary Action .................................................................................................................................
       6.6.3 No-Call/No-Show ....................................................................................................................................

   6.7 Attire and Grooming ..........................................................................................................................................
       6.7.1 Jewelry and Tattoos (alternative) ............................................................................................................
       6.7.2 Uniform Allowance .................................................................................................................................

   6.8 Electronic Communication and Internet Use ......................................................................................................
       6.8.1 Cellular Phones .......................................................................................................................................
       6.8.2 Computer, E-mail, and Internet Usage ....................................................................................................
       6.8.3 Blogging and Social Networking Policies and Guidelines ........................................................................
       6.8.4 State of Social Media ..............................................................................................................................
       6.8.5 Responsibility ..........................................................................................................................................
       6.8.6 Reporting Violations ...............................................................................................................................
       6.8.7 Discipline for Violations ..........................................................................................................................
       6.8.8 Topic matter guidelines ............................................................................................................................
       6.8.9 Inaccurate or Defamatory Content ........................................................................................................
       6.8.10 Off-Limits Material ................................................................................................................................
       6.8.11 Social Media – Acceptable Use ..............................................................................................................
       6.8.12 Right to Monitor ....................................................................................................................................
       6.8.13 Restrictions on Employee Camera Use ................................................................................................
       6.8.14 Hospital Monitoring ................................................................................................................................

   6.9 Employee Personnel Files ..................................................................................................................................
       6.9.1 Employee File Access (verify state requirements) ..................................................................................
       6.9.2 Release of Employee Information ...........................................................................................................
       6.9.3 Genetic Information Nondiscrimination Act (required if 15 or more employees) ..................................

   6.10 Meetings ..............................................................................................................................................................

   6.11 Personal Notebook ...........................................................................................................................................
6.15 Hospital Property (Optional) .............................................................................................................
6.16 Personal Property (Optional) .............................................................................................................
6.17 Personal Work Area ............................................................................................................................
6.18 Media ..................................................................................................................................................
6.19 Ethics ....................................................................................................................................................
6.20 Political or Public Activities and Contributions ................................................................................
6.21 Treatment of Patients ..........................................................................................................................
6.22 Honesty ................................................................................................................................................
6.23 Client Management ............................................................................................................................
   6.23.1 Professional Knowledge .....................................................................................................................
   6.23.2 Professional Courtesy .......................................................................................................................
   6.23.3 Advising Clients ................................................................................................................................
6.24 Team Requirements and Expectations ..............................................................................................

7 COMPENSATION ........................................................................................................................................
   7.1 Performance and Salary Review .........................................................................................................
   7.2 Payment of Wages ...............................................................................................................................
   7.3 Time Reporting .....................................................................................................................................
7.4 Overtime ..................................................................................................................................................
7.5 On-Call Pay (non-exempt employees) .................................................................................................
7.6 Employee Travel and Reimbursement .................................................................................................
7.7 Work Schedules .....................................................................................................................................
7.8 Forced Closing and Severe Weather Conditions ..............................................................................

8 TIME OFF/LEAVES OF ABSENCE ...........................................................................................................
   8.1 Holiday Pay .........................................................................................................................................
   8.2 Vacation (Option to PTO) ......................................................................................................................
   8.3 Sick Leave (Option to PTO) ...................................................................................................................
   8.4 Personal Leave (option to sick leave) ..................................................................................................
   8.5 Paid Time Off (option to vacation/sick/personal leave) ......................................................................
   8.6 Family and Medical Leave (FMLA)(over 50 employees) ...................................................................
      8.6.1 General Provisions ..........................................................................................................................
      8.6.2 Eligibility .........................................................................................................................................
      8.6.3 Type of Leave Covered .....................................................................................................................
      8.6.4 Amount of Leave ............................................................................................................................
      8.6.5 Employee Status and Benefits during Leave ................................................................................
      8.6.6 Employee Status after Leave ........................................................................................................
      8.6.7 Use of Paid and Unpaid Leave ......................................................................................................
      8.6.8 Intermittent Leave or a Reduced Work Schedule ........................................................................
      8.6.9 Certification for the Employee’s Serious Health Condition ........................................................
8.6.10 Certification for the Family Member’s Serious Health Condition

8.6.11 Certification of Qualifying Exigency for Military Family Leave

8.6.12 Certification for Serious Injury or Illness of Covered Service Member for Military Family Leave

8.6.13 Recertification

8.6.14 Procedure for Requesting FMLA Leave

8.6.15 Designation of FMLA Leave

8.6.16 Intent to Return to Work from FMLA Leave

8.7 Personal Leave of Absence

8.8 Maternity/Adoption Leave (check # of employees and state requirements)

8.9 Child Care Leave (optional, identify state law)

8.10 Bereavement Leave

8.11 Jury Duty

8.12 Voting Leave

8.13 Military Leave of Absence

8.14 Lactation/Breastfeeding

9 BENEFITS

9.1 Health Insurance

9.1.1 Medical and Dental Insurance

9.2 HIPAA

9.3 Civil Unions and Domestic Partners

9.4 Life Insurance (Optional)

9.5 Disability Insurance (Optional)

9.6 Retirement Plan (401K) (Optional)

9.7 Workers’ Compensation Benefits

9.8 Veterinary Services at Reduced Rate

9.8.1 Veterinary Services at Reduced Rate (Option 1)

9.8.2 Pet Health Insurance (Option 2)

9.8.3 IRS Guideline

9.8.4 Employee-Owned Pets

9.9 Employee Incentive Program (Optional)

9.10 Continuing Education

9.11 Team Entertainment and Education Functions

9.12 Employee Benefits Program/Package

10 SUMMARY

11 Acknowledgements

11.1 Acknowledgement of At-Will Status

11.2 Acknowledgement of Practice Policies
11.3 New Employee Orientation Checklist .................................................................
How to develop a team of “10” health care team members

Creating a resource pool of applicants

- In house posting
- Okay, steal them
- Print (not much)
- Internet
- Employment agencies???
- Employee Referral Program
- Other
  - Veterinary or Technician schools, Associations
  - Internships
  - Scholarships
  - Marketing – Who are you?

Three step interview process

- Initial Interview - pre-screening checklist - page 2
  - Overall impression
  - Match candidate to position – qualifications - How?
  - First impressions

Creating a resource pool of applicants

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Three step interview process

- Initial Interview - pre-screening checklist - page 2
  - Overall impression
  - Match candidate to position – qualifications - How?
  - First impressions
# Screening Interview Checklist

## Applicant's Name: __________________________ Date: __________

<table>
<thead>
<tr>
<th>Application Component to review</th>
<th>Notes:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cover letter rating:</td>
<td></td>
</tr>
<tr>
<td>Resume rating:</td>
<td></td>
</tr>
<tr>
<td>Best method to contact applicant</td>
<td>1st call: 2nd</td>
</tr>
<tr>
<td>Review the resume. Note any gaps or inconsistencies. Ask for any additional skills the candidate may have that are not listed.</td>
<td></td>
</tr>
<tr>
<td>Does the applicant have general work experience?</td>
<td>Overall: YES NO Animal Oriented: YES NO</td>
</tr>
<tr>
<td>- Veterinary practice</td>
<td>- Personal Pet Care</td>
</tr>
<tr>
<td>- Boarding</td>
<td>- Pet Store</td>
</tr>
<tr>
<td>&lt;1 year relevant experience</td>
<td>&gt;1 year relevant experience</td>
</tr>
<tr>
<td>Little or no experience</td>
<td></td>
</tr>
<tr>
<td>Employment stability</td>
<td>One job 2-3 yrs. Steady job in last 1-2 yrs.</td>
</tr>
<tr>
<td>Questionable explanation of gaps</td>
<td>Current student</td>
</tr>
<tr>
<td>Unexplained gaps, many jobs in last 5 years</td>
<td></td>
</tr>
<tr>
<td>Certifications or Degrees or Level of education required for this position</td>
<td></td>
</tr>
<tr>
<td>Advise the candidate will have to produce proper verification later in the interview process</td>
<td></td>
</tr>
<tr>
<td>Phone Interview Assessment</td>
<td>Date: __________ Time: __________</td>
</tr>
<tr>
<td>Discuss the philosophy of the practice and ask the applicant to share information about the philosophy at their last place of employment.</td>
<td></td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>--------</td>
</tr>
<tr>
<td>Confirm employment record point by point. Review reasons for leaving each position. Make sure all gaps have explanations.</td>
<td></td>
</tr>
<tr>
<td>Position desired and schedule (discuss position requirements and give them a copy of the position's schedule). Do they have any restrictions in being able to work this schedule?</td>
<td></td>
</tr>
<tr>
<td>What are the most important qualities to have for this position?</td>
<td>Get their answer and then explain your expectations.</td>
</tr>
<tr>
<td>What did you like best about your past position?</td>
<td>Best:</td>
</tr>
<tr>
<td>What did you like least?</td>
<td>Least:</td>
</tr>
<tr>
<td>Relevant classes taken (list them). Are you able to provide proof of the highest education level completed?</td>
<td></td>
</tr>
<tr>
<td>Not to limit you to a certain wage, but what is the minimum salary you would accept for the position?</td>
<td></td>
</tr>
<tr>
<td>Describe your perfect job.</td>
<td></td>
</tr>
<tr>
<td>Why should we hire you?</td>
<td></td>
</tr>
<tr>
<td>What are the pros and cons of working with a team?</td>
<td>Pros:</td>
</tr>
<tr>
<td></td>
<td>Cons:</td>
</tr>
<tr>
<td>Are you willing to agree to have a drug test, a criminal background check and a reference check appropriate for the position?</td>
<td>Ok to check references: ___ Yes ___ No If no, why?</td>
</tr>
<tr>
<td>If applicant was referred by another employee, get that employee's name</td>
<td></td>
</tr>
<tr>
<td>Explain the interview process and ask them what questions would you like to ask me?</td>
<td></td>
</tr>
<tr>
<td>Personal traits and interaction</td>
<td>Attentive</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Timeliness</td>
<td></td>
</tr>
<tr>
<td>Understanding of position</td>
<td>5 - Superior</td>
</tr>
</tbody>
</table>

**Overall rating**

- Set up for next interview
- Send Regret letter

**Recommendation**

**Additional Notes**

**Screeners' signature**

---

**Second Interview**
- In-depth
- Interview Report form - page 7
- Tour of practice - observe responses

**Observational Interview**
- Purpose
- Observational Interview Release form - page 8
- Train team on objectives

**Reference checking**
- Pre-Employment Questions to Avoid
- Issues with standard references
- Electronic background screening
- Options
  - Employee background investigations
  - [www.ebline.com](http://www.ebline.com) - background check whitepapers
  - Accredited
- Pre-employment testing
  - Job knowledge
  - Math
  - Language
  - Integrity?
Second interview
- In-depth
- Interview Report form - page 7
- Tour of practice – observe responses

Observational interview
- Purpose
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Reference checking
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  - www.ebiinc.com - background check whitepapers
  - Accredited
- Pre-employment testing
  - Job knowledge
  - Math
  - Language
  - Integrity?
  - Cognitive ability?
  - Emotional intelligence?

Why electronic background
- Accuracy
- Detail
- Verification
- Required Information
INTERVIEW - FIRST IMPRESSION

Name of Applicant: ________________________________

Date: ________________  Evaluated by: ________________

Rate each category: 1=Poor, 3=Average, 5=Great!
Circle rating and check appropriate boxes.

1. APPEARANCE  1  2  3  4  5
   □ Neat  □ Sloppy
   □ Well groomed  □ Lack of Grooming
   □ Appropriate Attire  □ Dressed for: ____

2. ATTITUDE  1  2  3  4  5
   □ Enthusiastic  □ Lack of Interest
   □ Interested  □ Dull
   □ Inquisitive  □ Monotonous

3. PERSONALITY  1  2  3  4  5
   □ Assertive  □ Arrogant
   □ Confident  □ Presumptuous
   □ Reserved  □ Shy, Timid
   □ Good eye contact  □ Poor eye contact

Should we schedule an interview?
   □ Yes  □ No
### INTERVIEW REPORT

Name: ___________________________  Date: ___________________________

Position Desired: ___________________________

*Check the appropriate box in each category, then make additional comments below.*

<table>
<thead>
<tr>
<th>APPEARANCE</th>
<th>BEARING</th>
<th>EXPRESSION</th>
<th>JOB KNOWLEDGE</th>
<th>MOTIVATION</th>
<th>PERSONALITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indifference to attire &amp; grooming</td>
<td>No bearing, lacks confidence</td>
<td>Uncommunicative, confused thoughts, poor vocabulary</td>
<td>None as pertains to this position</td>
<td>None, apathetic, indifferent</td>
<td>Unpleasant</td>
</tr>
<tr>
<td>Careless in attire, poor grooming</td>
<td>Often appears uncertain, poor posture</td>
<td>Poor speaker, has difficulty with ideas</td>
<td>Will need considerable training</td>
<td>Doubtful interest in position</td>
<td>Slightly objectionable</td>
</tr>
<tr>
<td>Functional attire, neatly groomed</td>
<td>Holds self well, room confidence</td>
<td>Speaks well, expresses ideas adequately</td>
<td>Basic, but will learn on the job</td>
<td>Sincere desire to work</td>
<td>Likeable</td>
</tr>
<tr>
<td>Well groomed</td>
<td>Looks of self reflects confidence</td>
<td>Speaks, thinks clearly, with confidence</td>
<td>Well versed in position, little training needed</td>
<td>Strong interest in position</td>
<td>Pleasing</td>
</tr>
<tr>
<td>Immaculate attire and grooming</td>
<td>Highly confident, inspires others with presence</td>
<td>Exceptional speaker clearly articulates confidence, idea well thought out</td>
<td>Extremely well versed, able to work without further training</td>
<td>Highly motivated, eager to work, asks many questions</td>
<td>Extremely pleasant, charming individual</td>
</tr>
</tbody>
</table>

**Overall Impression:**

- [ ] Unsatisfactory
- [ ] Marginal
- [ ] Satisfactory
- [ ] Very Good
- [ ] Excellent

**ADDITIONAL COMMENTS:**

Should we interview further?

- [ ] Yes
- [ ] No

**Interviewer:** ___________________________

**Date:** ___________________________
OBSERVATIONAL INTERVIEW
RELEASE FORM

(Date) 

Observer: ____________________________

TO: [Practice Name]
Address
Address

It is understood and agreed that my presence at (Practice Name) is strictly on an observation basis with no remuneration associated with the activity. I am neither an employee nor an agent nor am I associated with the (Practice Name) in any capacity other than as an observer for potential employment and therefore, hold the (Practice Name) and its associates harmless and free of any and all claims which may arise as a result of my observational interview.

Witness

(Applicant)

(Signature)

(Print Name)

(Signature)

(Print Name)

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How to recognize potentially great health care team members before you hire them…

- They put family first, before work.
- Money is important, but job satisfaction is their number one concern.
- They tend to work long hours and take pride in their work.
- They have a well-defined sense of their role. This can be detected in the manner in which they dress for the interview. They have a lot of energy that can be directed in a positive way.
- They are emotionally mature. This is evidenced in their concern for others, financial judgement, and length of time previously employed.
- They are compatible. Remember, you’ll have to work long hours with these individuals, so trust your feelings about getting along.
- They are motivated based on family destiny. Ask about father’s and mother’s occupation, type of education planned for family, and how the candidate relates to other members of the family.
- They can channel hostilities. Hire someone with enthusiasm, not a rebel willing to jump on any bandwagon.
- They want a boss they can respect as being competent.
- They consider accuracy a crucial element of the job.
- They have a need to finish a task once it’s started.
— JOB DESCRIPTION —

RECEPTIONIST

Classification: This is a nonexempt position under the Fair Labor Standards Act (FLSA)

Reporting Structure: The receptionist works under the direct supervision of the receptionist team leader/office manager and/or veterinary practice manager, who will indicate general assignments, limitations and priorities. Recurring assignments are performed independently. Deviations or unfamiliar situations are referred to the supervisor. Completed work is reviewed for technical accuracy and compliance with established procedures.

Revision Date: 6/28/2016

Receptionist Objectives: The purpose of this position is to serve as receptionist at [Practice Name], to perform record keeping duties, to perform clerical duties related to patient care and treatment, and to provide miscellaneous support to the veterinary practice manager and health care team. These service functions include, but are not limited to, reception (visitor and telephone), maintenance of veterinary medical records, accounts maintenance, cash processing, data entry, word processing and mail service. This position requires a practical knowledge of hospital organization and services, the basic rules and regulations governing visitors and animal patient treatment, data transcribing, word processing, and a practical knowledge of the standard procedures, veterinary records and terminology used in the hospital. Regular attendance and timeliness are an essential function in order to fulfill the requirements of this position.

Position type and expected hours of work:

Full or Part-Time
8 hour Shifts Monday – Friday for Full-Time Weekend shifts required
Overtime may be required

Education and Experience:
High school diploma or equivalent Veterinary experience preferred

Essential functions

Clerical
• Schedule appointments, obtaining all necessary data concerning the patient and owner. Prepare all required forms in advance when possible.
• Prepare to receive appointments by retrieving client records, preparing needed forms in advance of clients’ arrival. Complete required forms such as new client form, patient visit form, client report, consent forms, estimates, payment agreements, etc. and obtain all necessary information.
• Assure that all financial obligations are met by owners. Collect client fees, make change, process credit card transactions and assist in making count of cash drawer, run end of day transactions.
• Assist in the updating of client files; prepare and mail thank you cards and “welcome aboard” cards, reminders. Follow-up with clients when clinic records indicate no recent visits.
• Perform a variety of clerical duties, receiving, sorting, distributing mail, sending out mailings, cleaning, organizing reception area, type memos, correspondence, reports and other documents. Assist in the ordering, receiving, stocking and distribution of supplies.
• Collect client fees, post and record payments, make change, process credit card transactions and run end of day transactions.
• Ensure the cleanliness and organization of the reception area. Open and close practice

Customer and personal service
• Provide friendly, quality client care to the patients and clients of [Practice Name].
• Professionally administer all phone calls - answering client inquiries in a prompt and friendly manner, scheduling appointments, recording messages.
- Requires strong communication and client service skills. Considerable tact and diplomacy is required. Ability to greet clients in a professional, friendly, hospitable manner - check clients in, discharge patients.
- Receive incoming calls, screen those that are handled by other health care team members and take care of routine calls. The routine calls include those seeking information about veterinary services (“telephone shoppers”). Provide knowledgeable sub-professional advice concerning the care and treatment of animals.
- Check clients in - Greet clients in a professional, friendly, hospitable manner.

Veterinary policies/Procedures
- Follow established hospital policies and procedures in referring clients for immediate treatment of their pets when requests are accompanied by complaints of acute symptoms. Determine nature of injury/illness and attempt to reassure distressed pet owners. Determine whether immunizations and/or tests are current. Recommend update of necessary immunizations and/or tests to clients when applicable.
- Discharge patients. Review charts of patients being discharged from the clinic for completeness of information, make new appointments or note changes in patient status as necessary. Enter charges and set up future reminders in system. Present clients with medications, instruction.
- Perform over-the-counter selling of specialty merchandise comprised of pet grooming aids and sundry veterinary items. Exercise technical knowledge of products sold and demonstrate salesmanship abilities. Explain and demonstrate products, answer questions concerning products purchase/use.
- Fill veterinary prescriptions with appropriate medication; provide routine instructions to owners concerning prescriptions for medications.
- Collect lab specimens from pet owners, match patient record to the sample and submit samples to veterinary technician or nurse.

Computer
As required, enter data into the computer system, retrieve and modify computerized records. The practice management software includes, but is not limited to, such areas as reminder list of patients for periodic notifications, receipt and/or invoicing to update medical/financial records; accounting to include the general ledger, accounts payable, accounts receivable, billing and aging of accounts, income distribution, inventory control, client records, pet records, medical records, payroll; word processing to produce letters for general correspondence and special mailings to clients, etc.

Competency
Basic skills
- Possession of strong organizational skills.
- Knowledge of hospital procedures and operating instructions for making appointments, assembling patient medical records, recording test results, relaying information regarding patient’s condition, and compiling and submitting data on patients treated.
- Knowledge of the spelling and meaning of commonly used terminology of veterinary medicine to accurately record results of tests and file veterinary medical reports according to alpha, numeric or subject matter headings.
- Understanding the implications of new information for both current and future problem-solving and decision-making.
- Performs other duties as assigned. Ability to multi-task

Communication skills
- Excellent verbal and written communication skills. Possess exceptional interpersonal communication skills.
- Requires strong client service skills. Personal contacts are with pet owners affected by a variety of problems, visitors and other healthcare team members. Considerable tact and diplomacy is required. Must accurately relay owner’s account of the medical complaint(s) of the pet(s) involved to the healthcare team member who will be involved in treating the patient(s).
- Knowledge of the structure and content of the English language including the meaning and spelling of words, rules of composition, and grammar.
- Requires active listening skills, giving full attention to what other people are saying, taking time to understand the points be made, asking questions as appropriate and not interrupting at inappropriate times.
- Requires telephone conversations Requires use of electronic mail
- Requires writing letters and memos
- Requires face-to-face discussions with individuals or team members

Social skills
- Work well with all employees and ensure that your actions support the hospital, the doctors, and the practice philosophy.
- Ability to work independently on assigned tasks as well as to accept direction on given assignments.
• Monitoring/Assessing performance of yourself, other individuals or the practice to make improvements or take corrective action.
• Ability to adjust actions in relation to other’s actions
• Teaching others how to perform a task Actively looking for ways to help others
• Being aware of others’ reactions and understanding why they react as they do.
• Requires dealing with unpleasant, angry or discourteous people

Technical skills
• Knowledge of computers and relevant software applications including MS Office (Word).
• Perform routine maintenance on equipment and determine when and what kind of maintenance is needed.

Physical demands
The physical demands described here are representative of those that must be met by an employee to successfully perform the essential functions of this position. Reasonable accommodations may be made to enable individuals with disabilities to perform the essential functions.

<table>
<thead>
<tr>
<th>Task</th>
<th>None</th>
<th>Less than 1/3</th>
<th>1/3 to 2/3</th>
<th>More than 2/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stand</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Walk</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Sit</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Use hands to finger, handle, or feel</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Climb or balance</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stoop, kneel, crouch, or crawl</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Talk or hear</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Taste or smell</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The job requires the following lifting requirements and/or exerted force be performed on the job.

<table>
<thead>
<tr>
<th>Lifting Amount</th>
<th>None</th>
<th>Less than 1/3</th>
<th>1/3 to 2/3</th>
<th>More than 2/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Up to 10 pounds</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Up to 25 pounds</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Up to 50 pounds</td>
<td></td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Up to 100 pounds</td>
<td>X</td>
<td></td>
<td></td>
<td>(with assistance)</td>
</tr>
<tr>
<td>More than 100 pounds</td>
<td>X</td>
<td></td>
<td></td>
<td>(with assistance)</td>
</tr>
</tbody>
</table>

Specific vision abilities required by this job include close vision, distance vision, color vision, peripheral vision, depth perception and ability to adjust to focus.

Work environment
• While performing the duties of this job, the employee is exposed to hazards associated with aggressive patients; hazards associated with infected animals and controlled substances; exposure to unpleasant odors and noises; exposure to bites, scratches and animal wastes; possible exposure to contagious diseases.
• Follow federal and state animal health laws and regulations including OSHA and DEA.
• This job description is not designed to cover or contain a comprehensive listing of activities, duties or responsibilities that are required of the employee.
• Other duties, responsibilities and activities may change or be assigned at any time with or without notice.
[Practice Name] is an equal opportunity employer. It is the policy of the practice to prohibit discrimination and harassment of any type and to afford equal employment opportunities to employees and applicants without regard to race, color, religion, sex, national origin, age, disability, genetic information, gender identity or expression, or veteran status. The practice will conform to the spirit as well as the letter of all applicable laws and regulations. The practice will take action to employ, advance in employment and treat qualified Vietnam era veterans and disabled veterans without discrimination in all employment practices.

I have read and understand the Receptionist Job Description.

Employee Signature: ________________________________ Date: __________________________
Motivation and Retention
Mark Opperman, BS, CVPM and Sheila Grosdidier, BS, RVT
Veterinary Management Consultants
Evergreen, CO

What is motivation?
- Motivation vs. “Fear”

Factors that must be present in order for a person to be motivated
- Fulfillment of one’s basic needs
- Healthy work environment
- Security in one’s employment
- Knowledge and ability to do the job required
  - Effective hiring and training procedures
- Knowledge of the practice’s employee policies and procedures
  - Employee manual

Is money a motivator?
- Can money be used as a motivator?
- Six Factors That Can Outweigh Salary (page 4)

Motivational techniques
- Basic tenet: “Treat employees in the same manner as you would wish to be treated.”
- Positive reinforcement
  - The strongest motivator you have at your disposal
  - Verbal
  - Non-verbal
  - Frequency
- Negative reinforcement
  - Is this a motivational technique?
- Job enrichment
- Continuing education
  - In-service meetings
  - Educational seminars
- Involve staff in the decision-making process
  - Incorporation of new projects into the practice
- Open door policy
  - Effective communication
  - Understanding an employee’s position
- Twenty Low-Cost Activities to Boost Staff Morale (page 5)
- Six Commonly Ignored “Secrets” for Keeping Employees Motivated (page 6)

Delegation: A key to success
- Why is delegation so important?
  - Must we delegate to be effective managers?
- Delegate, don’t abdicate
  - Delegate for efficiency
  - Selectively delegate
    - Tasks That Should Not Be Delegated (page 6)
- How to delegate effectively
  - Make sure individual has knowledge and ability to do the job
  - Individual must have interest in doing the job
  - Explain task to be accomplished
  - Make the project their project
  - Provide support and reference material
• Define and state authority necessary to do the job
• Establish time table
• Provide adequate time to perform the job

• Institute automatic feedback controls
  o Follow-through
  o Never undermine a delegated responsibility
  o Use positive reinforcement

• Why do some staff members resist having responsibilities delegated to them?
  o They do not wish to make the necessary decisions involved
  o They are not sure how much authority they have
  o They do not feel equipped to handle the work and believe that they do not have enough information or direction
  o They are not prepared to accept responsibility
  o They do not see what is in it for them
  o They have made mistakes in the past that have embarrassed them or made you angry.
  o They are not aware that you have actually delegated something to them.
  o They feel that they already have too much to do.
  o They think the task is inappropriate for their job category or temperament.

• Some Common Reasons Why We, As Managers, Fail to Delegate
  o You feel that you must do everything and think that no one else can do it as well.
  o You may lack confidence in your staff.
  o You might be a perfectionist.
  o You might not like change.
  o You may have an inferiority complex.

• Must We Delegate For Success?
  o Positive results for effective delegators
  o Negative effects

• How Do You Rate As a Delegator?
  Answer the questions with a yes or no
  1. [ ] I am very effective at delegating
  2. [ ] I am not delegating as much work as I should
  3. [ ] I am an ineffective delegator and must work on delegating many unnecessary chores that are slowing me down.

Job features
  Do you know what your employees rank #1?
Here’s an interesting test you and your employees can take to determine if you share the same values regarding work. Ask staff members to rank the following items according to the importance they attach to them. Then, rank the items according to the importance you think your staff attaches to them. Compare and discuss.
(1 = most important; 8 = least important)

<table>
<thead>
<tr>
<th>STAFF RANKING</th>
<th>JOB FEATURE</th>
<th>DOCTOR RANKING</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Interesting Work</td>
<td></td>
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<tr>
<td></td>
<td>Credit for Work Done</td>
<td></td>
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<tr>
<td></td>
<td>______________________</td>
<td>______________________</td>
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<tr>
<td></td>
<td>Interesting Work</td>
<td></td>
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<td></td>
<td>______________________</td>
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<tr>
<td></td>
<td>Fair Pay</td>
<td></td>
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<td></td>
<td>______________________</td>
<td></td>
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<tr>
<td></td>
<td>Understand/Appreciate</td>
<td></td>
</tr>
<tr>
<td></td>
<td>______________________</td>
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<tr>
<td></td>
<td>Counsel Problems</td>
<td></td>
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<td></td>
<td>______________________</td>
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<tr>
<td></td>
<td>Promotion on Merit</td>
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<td></td>
<td>______________________</td>
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<td></td>
<td>Good Work Conditions</td>
<td></td>
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<td></td>
<td>______________________</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Job Security</td>
<td></td>
</tr>
<tr>
<td></td>
<td>______________________</td>
<td></td>
</tr>
</tbody>
</table>

**Six factors that can outweigh salary**
- Agreeable Working Conditions
- Recognition
- Fringe Benefits
- Lifestyle
- Challenge
- Fulfillment

**Twenty low cost activities to boost team morale**
1. Send a Letter of Commendation to a Staff Member for Performance Above and Beyond Normal Expectations
2. Provide Lunch for All Employees on the Last Working Day Before a Holiday
3. Provide Staff with Fresh Fruit or Other Foods for Snacking During Breaks
4. Buy Corsages for Staff on Special Occasions
5. Send a Card to All Staff Members on Their Birthdays
6. Invite Part-time Employees to All Staff Social Events
7. Write Personal Messages such as “Thank You” or “Happy Birthday” on Payroll checks
8. Work Side-By-Side with Staff Once a Year (Or More Often) on a Community Help Project
9. Personally Introduce New Hires to Each Staff Member
10. Greet Each Staff Member at the Start and End of Each Day
11. Send a Card to Each Staff Member on His/Her Anniversary
12. Hold Morale Building Meetings to Inform Staff of Your Practice’s Successes
13. Reward Staff Who Miss One Day or Less During the Year Due to Illness or Injury
14. Buy a Vase for Your Business Assistant’s Desk and Provide Fresh Flowers on a Surprise Basis
15. In Each Month That New Patients Exceed an Established Figure, Take All Employees Out for Dinner
16. Hold an Annual Staff Appreciation Party
17. Give a Small Gift to an Employee Each Time a Patient Makes a Positive Comment about Him/Her
18. Plan a Staff Social Event at Which You Do the Cooking and Serving (Picnic, Barbecue, Etc.)
19. Give a Reception for Every Employee Who Retires
20. Design an “Employee of the Month” Plaque for your Reception Area

Six commonly ignored “secrets” for keeping employees motivated
- Never Oversell A Job
- Keep Assistants Informed
- Keep Vertical Channels of Communication Open and Clear
- Keep Jobs Challenging
- Encourage Self-improvement and Create Opportunities for Advancement
- Finally, Make Sure Employees Know Exactly What is Expected of Them

Tasks that should not be delegated
- The Signing of Business Checks
- The Final Word On Collection
- Spot Checking On Financial Or Personnel Records
- All Patient Related Duties
- Making The Final Decision On Major Management Policy

The essentials in retention
- Know What the Employee Wants
- Know What The Practice Needs
- Long Term and Short Term Results
- Variation
- It Starts With What You Say
- It Continues With What You Do

Reasons for turnover
- Poor communication
- Unskilled supervisors/managers
- Unsatisfying job responsibilities
- Problems with working conditions
- Pay below market levels
- Inadequate benefits
- Insufficient recognition
- Lack of advancement opportunities
- Interpersonal conflicts
- Personal problems (family, health, etc.)
- Wealth of external opportunities

Checklist of employee retention tools
- Employee Relations Tools - non-compete agreements, focus groups, exit interviews, etc.
- Compensation Tools - sign-on and retention bonuses, merit and incentive pay, etc.
- Benefits Tools - expanded benefits, unique services, annual benefit statements, etc.
- Scheduling Tools - flexible scheduling, sabbaticals, telecommuting, etc.
- Workplace Enhancement Tools - casual dress, career pathing, timely performance appraisals, etc.

25 ways to increase team retention today
1. It starts with 4 quarters – Put 4 quarters in your left pocket and each time you make a genuine compliment to a team member, move a quarter over to your right pocket. Your goal is to have all the quarters, every night in your right pocket.
2. Involve the team in decisions that affect their position.
3. Celebrate it – Celebrate each team member’s anniversary with the practice, not just their birthday.
4. Ask them – Make it a point to ask team members what they like best and then do more of it!
5. Set clear expectations – Satisfied team members know what is expected of them every day at work. Very few people are mind readers. You have to share the information to expect results.
6. Hold a quick 2 minute meeting in the morning and share with the team the goal for the day and any special issues that they need to know about to make everyone successful.
7. Set practice traditions, like every morning, each team member has to tell one other person something good that happened the day before.
8. Encourage feedback from your team – Ask for ideas and then talk about how that helps patients, client service, overall benefit to the team and the practice. Show respect and appreciation that your team member took the time to share with you what they believe will help the practice.
9. Make sure your wage is based on performance, not just longevity.
10. Give out a surprise incentive. If you see a team member pick up the dirty paper cup when they were walking through the parking lot into work, how about coming up and asking them to trade that cup for the two movie tickets you have in your hand?
11. Make sure wages are comparable with other jobs in your area and tell your employees what they need to do to get a raise.
12. Have new employee training mapped out so that it helps the employee succeed. Don’t just throw them into the practice and hope for the best.
13. Treat everyone with respect and do not allow individuals to not observe this point.
14. It’s okay to have fun. Let team members know you appreciate their sense of humor and look to add a little humor to the day. Create a smile board with funny pictures, silly stories and happy faces. Put it in the treatment area so everyone can enjoy it. How about taking digital pictures of people in the clinic, posting them and then have a contest for the funniest caption?
15. Praise for attempts to do it well. Let team members know you appreciate the effort.
17. Share information promptly and clearly. Don’t let it leak out to the team. If they need to know it, tell them right away.
18. Actively listen to what your team tells you. They want to be heard.
19. Provide regular training; get the team involved in deciding what they want to learn.
20. Promote from within whenever you can.
21. Train your supervisors well; they can be the leading element in keeping good team members.
22. Consider offering retention bonuses or increasing vacation for team members who stay long term with the practice.
23. Make sure team members have the equipment and supplies they need to get the job done.
24. Know why team members stay and know why they leave. Your problem may not be the employee; it may be your hiring process.
25. Change your mindset – The most important thing that you do is support the team that serves your clients. Without them, you have no practice.
Disciplinary process

Disciplinary actions

- Employee Performance Agreement (page 6)
- Employee Performance Agreement Review (page 7)

Firing

- Make a decision and act upon it—do not procrastinate—indecision is detrimental to you and to your practice
- Document your employees actions
- Atmosphere in which to conduct discharge - Termination Meeting Checklist (page 9)
- State practice’s position and factual evidence
- Approaches:
  o Try to present so employee realizes he/she would be better suited in another position
  o Direct approach
- Do not keep a discharged employee in the practice
- Exit interview (page 10)
- Questions to be asked during an exit interview
- Termination Checklist and Letter (page 13 & 12)
- Fear of Failure: How to recognize it and what to do about it
- How to Become an Effective Leader

Receptionist performance evaluation

Name: ___________________________ Date: ___________________________

Date of last review: __________________ Date of Employment: ______________

Person preparing review: __________________________________________

Rating guide

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>Almost always exceeds expectations; quality of work is highest caliber;</td>
</tr>
<tr>
<td></td>
<td>greatly exceeds required job criteria.</td>
</tr>
<tr>
<td>Very Good</td>
<td>Performance almost always meets expectations and exceeds expectations on</td>
</tr>
<tr>
<td></td>
<td>occasion; always above average work; fulfills job criteria very well;</td>
</tr>
<tr>
<td></td>
<td>requires minimal supervision.</td>
</tr>
<tr>
<td>Adequate</td>
<td>Performs to expectations most of the time; satisfactory most of the time;</td>
</tr>
<tr>
<td></td>
<td>fulfills job criteria adequately.</td>
</tr>
<tr>
<td>Needs Improvement</td>
<td>Does not perform to expectations; satisfactory only on occasion;</td>
</tr>
<tr>
<td></td>
<td>significant improvement should be achieved to fulfill job criteria;</td>
</tr>
<tr>
<td></td>
<td>requires more than normal amount of supervision.</td>
</tr>
<tr>
<td>Unsatisfactory</td>
<td>Almost never performs as expected; lacks any proficiency; major improvement is</td>
</tr>
<tr>
<td></td>
<td>required in order to fulfill job criteria.</td>
</tr>
</tbody>
</table>

If improvement is not met in specified time period, job termination may result.
1. **Punctuality**: Demonstrates punctuality and dependability. Is consistently on time for all work periods and returning from breaks. Works scheduled hours and is rarely absent. Clocks in and out appropriately.

   Rating _______     Points _______

2. **Judgement**: Able to discern when a pet should be seen and when verbal information will solve the problem.

   Rating _______     Points _______

3. **Client Communication**: Personable. Greets team members and clients with an upbeat, positive tone and a smile. Maintains a positive, friendly attitude. Conveys warmth and caring. Treats all clients and patients with the utmost respect and care.

   Rating _______     Points _______

4. **Housekeeping**: Work area is always presentable and neat. Watches waiting area to assure that it is neat, clean and odor-free.

   Rating _______     Points _______

5. **Scheduling**: Schedules appointments, following practice guidelines, after obtaining all necessary data. Prepares any necessary forms, sends out new client information.

   Rating _______     Points _______

6. **Initiative**: Assists in identifying and solving various problems related to how the hospital is run. Brings ideas to the supervisor. The employee searches out new tasks and expands his/her abilities professionally and personally.

   Rating _______     Points _______

7. **Telephone Skills**: Professionally answers the phone in three rings or less. Uses the appropriate greeting. Has a pleasant and cooperative phone voice. Never places a call on hold without the caller’s permission. Returns to calls holding with updated information on how much longer the wait will be. Skillfully answer telephone shopper inquiries following the hospital procedures for phone shoppers.

   Rating _______     Points _______

8. **Computer Literacy**: Shows great proficiency in handling the various computer operations required on a daily basis. Enters information accurately. Runs reports, retrieves information, and updates client records timely and accurately.

   Rating _______     Points _______

9. **Accounting Skills**: Is accurate in handling monetary transactions and making change. Is able to complete the daily sheet, deposit slips and records accurately in the computer.

   Rating _______     Points _______

10. **Knowledge**: Possesses the necessary veterinary medical knowledge to be able to answer most client questions. Knows and understands hospital policies and protocol.

    Rating _______     Points _______

11. **Attention to Detail**: Employee is a detailed individual in all aspects of his/her job. Small details do not escape his/her attention. Is able to remember low priority items and do them during slack times. Can prioritize job duties well.

    Rating _______     Points _______

12. **Professional Attitude**: Possesses strong client service skills. Is able to handle irate clients with relative ease. Remains calm in crisis situations.

    Rating _______     Points _______
13. **ACCURACY:** Demonstrates accuracy, thoroughness, neatness and dependability. Writes legibly. Records correct information on records as well as controlled substance log. Files records accurately.

   Rating _________ Points _________

14. **DRIVE:** Shows a real desire to achieve excellence in every aspect of job area. Desires to make the job exciting and fulfilling. Is eager to learn new procedures/techniques and is open to change.

   Rating _________ Points _________

15. **TEAMWORK:** Works well with all team members and ensure that your actions support the hospital, the doctors, and the practice philosophy.

   Rating _________ Points _________

16. **AUTONOMY:** Employee works independently on assigned tasks as well as accepts direction on given assignments.

   Rating _________ Points _________

17. **HOSPITAL PROCEDURES:** Follows hospital policies regarding patient admittance, immunizations, discharges, etc. Provides proper instructions, medications and enters reminders into system.

   Rating _________ Points _________

18. **CLERICAL DUTIES:** Employee is able to accurately and proficiently perform a variety of clerical duties, mailings, cleaning, organizing reception area, run reports, type correspondence and other documents.

   Rating _________ Points _________

19. **MARKETING:** The employee is effective in marketing to and educating the client about vaccinations, parasite control, and other services and products that we provide. Exercises a technical knowledge of products sold.

   Rating _________ Points _________

20. **APPEARANCE/GROOMING:** Presents self as professional and dress reflects that presentation to clients. Is always clean and well groomed and wears appropriate uniform/clothing in accordance with job requirements.

   Rating _________ Points _________

**OVERALL RATING______ TOTAL POINTS_____**

Comments/Recommendations/Goals to attain

This individual has demonstrated positive performance of growth and development in the following areas:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Areas where improvement in performance and effectiveness can be shown by this individual:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
Future Goals for Employee:

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

My employer and/or supervisor has reviewed this evaluation with me. I understand the criteria on which I have been judged and my reviewer has discussed my goals for the future.

Employee Signature: ___________________________ Date: ________________

I have completely reviewed this evaluation with my employee.

Supervisor Signature: ___________________________ Date: ________________

Employer Signature: ___________________________ Date: ________________

**Employee performance agreement**

___ Counseling  ___ Warning  ___ Final Written Warning

Name/Title of employee: ___________________________

Name/Title of direct supervisor: ___________________________

Summarize the situation and its implications. Define performance objectives for the employee, outlining what the employee is responsible for doing to correct the situation. Check the appropriate availability of supporting documentation. Review job description to clarify expectations.

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

___ Supporting documentation is available and attached  ___ Supporting documentation is not available

Outline the supervisor's responsibilities; what the supervisor will do to assist the employee in meeting the objective(s).

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

*If this is a formal warning and this is marked, the employee's employment status is considered probationary, making the employee ineligible for pay increases, vacation usage, or promotions.*
Describe how the supervisor will determine if objectives are met. Outline the consequences if objectives are not met.

If the above stated objectives are not met, further disciplinary action up to and including termination may result.

This performance agreement will be reviewed on _____/____/____ on page 2 of this form (Employee Performance Agreement Review).

Employee comments*:

__________________________________________________________  Date:___________________

Employee's Signature: ___________________________  Date:___________________

Supervisor's Signature: ___________________________  Date:___________________

Witness' Signature: ___________________________  Date:___________________

Employee performance agreement review

Follow-up to:  ___Counseling  or  ___Warning, dated: ___/___/____

Name/Title of employee: ____________________________________________________________

Name/Title of direct supervisor: _____________________________________________________

Progress Review—summarize the employee's actual performance compared to the objectives outlined on the Performance Agreement. Highlight areas of improvement and/or areas of continued concern. Check the appropriate box regarding availability of documentation. Review job description to clarify expectations.

___ Supporting documentation is available and attached  ___ Supporting documentation is not available

Overall evaluation of the performance:

____ The employee has met all performance objectives.
____ The employee has made progress toward the completion of performance objectives or has met some objectives.
____ The employee has not made any progress toward completion of performance objectives.
____ The employee's performance in noted areas has declined.

Recommendation:

____ The objectives were met and the performance agreement has been fulfilled.
____ The employee has made some progress, but improvement is still needed. Performance agreement will be revised or time frame to meet objective(s) extended to ___/___/___ (date). Attach new/revised agreement.
____ Performance did not improve to stated performance objective(s). Further action is warranted (see attached documentation):

___Warning (new review date ___/___/___)  ___Suspension  ___Demotion  ___Transfer
___Termination
___Other

________________________

750
Supervisor Comments (include consequences the employee may face if the situation precipitating the Performance Agreement should reoccur):

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

Employee Comments* (please use the back of this form for further comments):

__________________________________________________________________________

__________________________________________________________________________

__________________________________________________________________________

Employee's Signature: _______________________________________________________

Date: _____________________________________________________________________

Supervisor's Signature: _____________________________________________________

Date: _____________________________________________________________________

Witness’ Signature: _________________________________________________________

Date: _____________________________________________________________________

Termination meeting checklist

Conduct the meeting in sequence as follows:

1. All documentation or performance/disciplinary issues have been completed.
2. Tell the employee the purpose of the meeting. Although the reason for termination should be communicated, there is no need to go through a step-by-step analysis of the documentation supporting the reason for discharge.
3. Advise that the decision is final and cannot be reversed.
4. Where appropriate; advise that alternative in-house positions were explored.
5. Emphasize that all relevant factors were reviewed.
6. If applicable, stress that everyone involved in management activities agreed to the decision.
7. Tell the employee the effective date of the termination.
8. Review with the employee a written summary of benefits. Where applicable, this summary should include severance pay, compensation for vacation and sick time, continuation of health and life insurance benefits, other benefits and re-employment assistance.
9. Have final paychecks ready. If the employee is to leave immediately, have any final checks, benefits or vacation payments prepared and inform the employee how to collect his or her personal belongings and leave the premises.
10. End the interview by saying that the employee will be notified of any other matters that must be dealt with, such as COBRA continued health coverage.
11. Wish the employee good luck and express confidence in his or her future.
12. Stand, extend your hand and remain standing until the employee has left the meeting site.
13. Check your state laws regarding the requirement of providing a standard service letter. In these states the employee must request verbally or in writing a letter verifying the employee’s dates of employment, type of services provided and reason for conclusion of employment.

Employee exit interview

TO: ________________________________________________________________

FROM: ____________________________________________________________

Practice Name
I would appreciate it if you would take a few minutes to respond to the questions below. All Answers will be held in strict confidence. Thank you.

How long were you employed with our practice?

Job classification?

Why are you leaving?

Would you describe your working relationship (with respect to both your particular job and your relationship with fellow team members) as pleasant or unpleasant? Please rate on a scale from 1 to 10.

1 2 3 4 5 6 7 8 9 10

Poor        Excellent

Comment:

Do you feel that your particular job was important and significant in the overall operation of the practice?

Are there any particular practices or working conditions that either led to your decision to resign or that you feel are detrimental to a satisfactory working relationship? If so, have you any suggestions on how to eliminate them?

Are there any particular practices or working conditions that you feel are particularly beneficial to an effective working relationship and that should be maintained?
Would you care to make any other comments?

The above information is true to the best of my knowledge, information and belief

Signed: ________________________________ Date: ________________________________

Termination letter example
Dear Mr/Mrs/Ms,

Further to our meeting of (date), I confirm that your employment with (Company) is terminated with effect from (date).

As stated at our meeting on (date), the reason for termination your employment is as follows;

• Reason 1 - e.g. summary of redundancy reasons.
• Reason 2 - summary of gross misconduct or poor performance and what steps had been taken, and when, to enable the employee to rectify the situation.
• Reason 3 - etc

Clearly state individual requirements such as return of company car, equipment, submission of expense claims etc and any other administrative details.

Clearly state actual leaving date, and details of notice period, holiday pay, general pay and pension or other benefits, plus redundancy settlement if appropriate.

Clearly state how the employee can appeal to the decision - the employee’s rights, the appeal process and appeal time frames.

Please sign, date and return this letter as confirmation of receipt of this letter and any attachments/enclosures.

Yours truly,

Name
Position

Termination checklist

<table>
<thead>
<tr>
<th>Employee Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Employee Name</td>
</tr>
<tr>
<td>Employee position</td>
</tr>
<tr>
<td>Term Date</td>
</tr>
</tbody>
</table>

If termination is involuntary
[] Documentation of performance issues and disciplinary action is in employee file.

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### Before employee’s last day of employment

<table>
<thead>
<tr>
<th>Human Resources</th>
<th>Accounting/Finance</th>
</tr>
</thead>
<tbody>
<tr>
<td>[] Prepare COBRA Letter</td>
<td>[] Final paycheck is being prepared.</td>
</tr>
<tr>
<td>[] Schedule exit interview</td>
<td>[] Check for final balances on employee account, make arrangements for payment.</td>
</tr>
</tbody>
</table>

### Office Coordinator/Facilities

| [] Cancel voicemail account effective employee’s last day. |  |
| [] Request to have employee’s network access closed effective employee’s last day. |  |
| [] Cancel password for security access and collect any keys |  |

### Last day of employment

| [] Provide COBRA letter & explain |  |
| - 60 days to elect coverage | - 45 days to send in premium for all months since coverage ceased |
| - Premium due 1st of the month | -  |
| [] Non-Disclosure Agreement | - Provide copy |
| - Explain Non-compete | - Retrieve any confidential information |
| [] Last paycheck (please check one) | -  |
| - Provided at exit interview OR | - Mailed after termination date |
| - [] Provided at exit interview OR | - [] Mailed after termination date |
| - Provide 401(k) Withdrawal Form | -  |
| [] Address Changes Verified | -  |
| [] Collect or verify computer system(s) or equipment | -  |
| [] Collect security card | [] Collect cell phone |
| [] Collect phone card | -  |
| [] Collect corporate credit card | [] Exit Interview Questionnaire |
| [] Eligible for rehire? Yes_____ No | -  |
| [] Terminate status in the HR system | -  |

### After the employee’s last day

| [] Check for any additional amounts owed |  |
| [ ] Mail final pay stub to former employee if necessary. |  |
| [ ] Complete and submit benefit forms to stop coverage with Insurance. |  |
| [ ] If former employee submits a request for COBRA coverage, re-enroll using Insurance enrollment forms. (Refer to COBRA process document.) |  |

### Reason for leaving

_____________________________________________________________

_____________________________________________________________

Employee Signature ____________________________ Date ____________
New employee orientation checklist

- can vary from state to state
- Provide employee with personal storage space. Discuss protection of personal property at work
- Tour the hospital. Provide a detailed hospital tour which points out emergency exits, eye wash stations, employee restrooms, employee break room, bulletin board and work schedule. Identify the exam rooms, kennel, surgery/treatment area, pharmacy, radiology, etc. and what each area is used for.
- Show Employee designated parking area
- Introduce employee to doctors and other team members. Identify trainee’s immediate supervisor
- Complete the following required forms for your personnel record: New Hire Reporting for state requirements: http://www.sba.gov/content/new-hire-reporting-your-state

Verify Completion of Application

- W-2
  - I-9 or E-Verify (Complete in entirety within 3 days of employment)
  - Verify Social Security card & driver’s license as required by I-9
  - Complete all required new hire forms
- Direct Deposit Approval
  - Proof of current vehicle insurance (if driving personal auto for hospital)
- Give employee an empty notebook for training notes
- Present employee with [Practice Name]’s employee manual
  - Review the hospital’s hierarchy chart (management structure)
    - 1) Present At-Will Employment acknowledgement and have employee sign and place in their personnel file
    - 2) Review benefits and effective dates
    - 3) Discuss practice dress code and present employee with uniform
    - Review hospital schedule for meals and breaks
    - Review the payday procedures and overtime policy
- Employee to sign review and understanding of employee manual and place in their personnel file.
  - Present employee with [position] job description
    - Review general expectations for the position, as well as protocol for annual review
    - Present employee with a blank performance evaluation form
    - Review the veterinary technician duties to be completed daily
    - Learn the location and operation of time clock software.
      - Discuss timeliness and attendance expectation
      - Show employee the proper protocol for submitting a request for days off form and how work schedules are presented and posted.
- Present employee with copy of phase training document. Explain protocol (trainee to sign off on each phase, if trainee has questions – ask, etc.) The phase training document should be carried on their person until training is completed in its entirety.

Employee Signature: __________________________________________

Management phase training checklist and instructions

- The phase training program is to assist your new or existing team member with success in their position and requirements of the hospital.
- All team members should have an up-to-date job description which closely matches the phase training program.
- The phased training program should be provided to the team member after completion of the New Employee Orientation Checklist.
- The team member should be instructed that the goal over the next 90 days is to make sure that they are properly trained by their supervisor or their designated trainer and signed off on the lists of skills and knowledge tasks.
- The phased training program keeps the trainer, trainee and practice manager abreast of the training status and gives them an open line of communication.
- The phased training programs are excellent risk management tools to alleviate training oversights.
- The phased training programs can be useful during performance reviews or if performance issues should arise.
Practice Managers should complete the checklist below for each team member using the phased training program. This checklist will ensure that the Practice Manager is tracking the compliance of the team members’ training progress.

Team leaders should complete and sign off on the respective position before advancing to the Team leader phased training program. For example: the Receptionist Team Leader will complete and sign the Receptionist phased training program before moving to the Receptionist Team Leader phased training program.

Management phase training checklist [Practice Name]

Date: __________________________

Employee Name: ______________________________

Position: ____________________________________

Employee’s Immediate Supervisor: __________________

☐ Complete New Employee Orientation checklist before presenting phase training to new employee. Employee to sign completion of checklist.

☐ Verify that all required documents from the New Employee Orientation checklist are completed and signed.

☐ Assign Trainer to each task before presenting to new employee.

☐ Inform trainer(s) that they will be assisting the new employee with their training. Ensure that the current schedule will accommodate the training times and needs.

☐ Assign Due Dates to probable duration.

☐ Set up personal reminders to check progress of new employee with trainers.

☐ Set up personal reminders to verify with new employee status of phase training and that tasks are being completed in a timely manner.

☐ Inform the team member to write down something new they learned or any changes, updates or addition that need to be made to the phase training document. Set up a time to discuss with the team member if needed.

☐ Once new employee has completed a Phase, review with employee and sign off completion.

Receptionist phased training program

Employee (Trainee) Name __________________________ Hire Date __________________________

Purpose: The purpose of this program is to introduce the Receptionist to the practice and bring them into the hospital’s philosophy of care and service. Through this program, the new Technician will become familiar with the day-to-day operations, management, and standards of care within our hospital.

The first column of the table below is the Skill/Knowledge and the Trainer that is assigned to the task for training the employee. The second column are the tasks that need to be trained and the third column is where the trainer initials that the training is complete and the date of completion.

Although a probable duration is stated for each phase of training, these are meant only as a guide and neither the trainer nor the trainee should sign off on a phase until they feel that they fully understand and are comfortable performing all the job tasks listed.
# Phase I - Welcome to our practice!

Probable Duration: One Day - Two Days (Due Date ________)

<table>
<thead>
<tr>
<th>New Employee Orientation Checklist</th>
<th>The Employee has signed and completed the New Employee Orientation Checklist</th>
<th>Initials/Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>OSHA Training</td>
<td>Conduct OSHA training. Explain OSHA standards, MSDS sheets, etc. Give employee handout regarding safety and complete OSHA test. Inform team member what they are to do if an OSHA officer shows up and ask for a tour of the practice. Make sure they know the practice OSHA safety officer’s/coordinator’s name</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Observation</td>
<td>Trainee to observe (senior) receptionist. (1 hour)</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Telephone Procedures</td>
<td>Show proper way to: Answer phone, Take messages Place callers on hold Route messages to doctors and other team members</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Watch LifeLearn training CD “Enhancing your Telephone Skills.” Note to practice: This CD can be purchased at <a href="http://www.lifelearn.com">www.lifelearn.com</a></td>
<td></td>
</tr>
<tr>
<td>Basic Animal Handling</td>
<td>Learn basic animal handling principles. Before signing off, trainee must demonstrate proper animal handling with at least two patients.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conclusion of Phase 1</td>
<td>Review of Phase I of training program. Trainee is asked if he/she has any questions or needs further training on any part of Phase I.</td>
<td>Initials/Date</td>
</tr>
</tbody>
</table>

**Phase I of training complete**

*My signature below signifies that I have completed Phase I of the Receptionist Phased Training Program and that I fully understand all concepts covered and I am comfortable in my knowledge and ability to perform the procedures introduced in Phase I of this program. Supervisor signature below signifies that employee has successfully completed Phase I and has answered all pertinent questions.*

---

**Employee (Trainee)**

**Date**

**Supervisor**

**Date**

---
<table>
<thead>
<tr>
<th>Reference Materials</th>
<th>Present trainee with materials to review.</th>
<th>Initials/Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present Trainee with the Common Medical Terminology handout.</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Other client education materials</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Other __________</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Review:</td>
<td>Review above presented materials with trainee</td>
<td></td>
</tr>
<tr>
<td>Other __________</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Scheduling</td>
<td>Explain:</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Basic appointment scheduling procedures.</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Scheduling guidelines and special circumstances (heartworm season, etc.).</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Fecal test</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Logging On/Off</td>
<td>Demonstrate how to log on and off the computer properly.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Review company policy regarding computer use and password maintenance/usage.</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Software</td>
<td>Complete veterinary software training module</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Greeting Clients</td>
<td>Explain the proper way clients and their pets are to be greeted and treated when they come to the practice.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Obtain client information</td>
<td>Review obtaining all necessary data from clients to prepare forms i.e. new clients, consent forms, medical care plans (ie, estimates).</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Obtaining a weight</td>
<td>Demonstrate how to obtain a weight on a pet.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Wait Time</td>
<td>Demonstrate how to handle situations where there is an extended wait</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Alert Assistant About Visit</td>
<td>Explain outpatient protocol -- the assistant is to be alerted that the client and patient are ready.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Controlling Odors</td>
<td>Explain procedure for controlling odors and maintaining a neat and tidy front desk. Discuss danger in using bleach and that bleach should NEVER be mixed with ammonia.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Noise Pollution</td>
<td>Explain procedure for minimizing noise pollution. (e.g. barking dogs are escorted to a private area or an exam room) Explain proper use of ear plugs.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Pulling Forms</td>
<td>Show how to retrieve forms &amp; the filing/computer system.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Before signing off, trainee must demonstrate the ability to properly handle.</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Checklist</td>
<td>Demonstrate how to use and/or create a checklist.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td>Messages</td>
<td>Review the proper way to answer the phone and take messages.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>Trainer:</td>
<td>Initials/Date</td>
<td></td>
</tr>
<tr>
<td><strong>Confirmation Calls</strong></td>
<td>Explain procedure of calling clients the day before their appointments to confirm their appointment.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td>-------------------------</td>
<td>--------------------------------------------------------------------------------------------------</td>
<td>--------------</td>
</tr>
<tr>
<td><strong>Surgery Quotes</strong></td>
<td>Explain the proper procedure for quoting surgery prices</td>
<td>Initials/Date</td>
</tr>
<tr>
<td><strong>Medical Care Plan Book</strong></td>
<td>Demonstrate how to use the Medical Care Plan Book and the appropriate way to go over a medical care plan.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td><strong>Fax, Copier, Phone System</strong></td>
<td>Demonstrate the use of necessary office equipment.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td><strong>Mail</strong></td>
<td>Explain how to take out and pick up the mail.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td><strong>Vaccination Due Dates</strong></td>
<td>Explain how to check vaccination due dates. Before signing off, trainee must demonstrate the ability to handle this task properly.</td>
<td>Initials/Date</td>
</tr>
<tr>
<td><strong>Conclusion of Phase II</strong></td>
<td>Review of Phase II of training program. Trainee is asked if he or she has any questions or needs further training on any part of Phase II. Trainee signs off on Phase II.</td>
<td>Initials/Date</td>
</tr>
</tbody>
</table>

**Trainee Comments - Phase II**

*Use this area for any comments you have concerning this phase of your training. This will help us to improve our training systems and ensure that adequate training is provided to you. Your comments will be read by the management of the practice and kept in your confidential employee file.*

**Phase II of Training Complete**

*Math signature below signifies that I have completed Phase II of the Receptionist Phased Training Program and that I fully understand all concepts covered and I am comfortable in my knowledge and ability to perform the procedures introduced in Phase II of this program. Supervisor signature below signifies that employee has successfully completed Phase II and has answered all pertinent questions.*

<table>
<thead>
<tr>
<th><strong>Employee (Trainee)</strong></th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Supervisor</strong></td>
<td>Date</td>
</tr>
</tbody>
</table>

**Phase III**

*Probable Duration: One Week (Due Date ___)*

<table>
<thead>
<tr>
<th><strong>Adding New Client</strong></th>
<th>Demonstrate how to add a new client.</th>
<th>Initials/Date</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Entering Charges</strong></td>
<td>Demonstrate the correct procedure for entering charges into the computer. Before signing off, trainee must demonstrate the ability to correctly enter charges.</td>
<td>Initials/Date</td>
</tr>
</tbody>
</table>
| Payments from Clients | Explain the process of accepting payment from clients  
Credit cards  
Cash  
Check  
Care Credit | Initials/Date |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Fee schedule</td>
<td>Demonstrate how to print a list of charges.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Team Meeting</td>
<td>Review recent team meeting minutes and the protocol for reviewing minutes if employee is unable to attend a meeting.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Hospital Tours</td>
<td>Explain protocol for client tours or when clients are allowed to visit patients in boarding or the hospital.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Treatment Board</td>
<td>Demonstrate how to properly use the treatment board.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Contagious Soak</td>
<td>Demonstrate the procedures followed for a contagious soak.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Vaccine Protocol</td>
<td>Demonstrate a working knowledge of vaccine protocol.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Appointment Scheduling</td>
<td>Demonstrate basic appointment scheduling.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Surgery Appointment Scheduling</td>
<td>Demonstrate the ability to schedule surgery appointments.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Hospital Organization</td>
<td>Explain the organization of the hospital and workflow.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
<tr>
<td>Surgery Forms</td>
<td>Demonstrate how to correctly fill out surgery forms.</td>
</tr>
<tr>
<td>Trainer:</td>
<td></td>
</tr>
</tbody>
</table>
| Collect Laboratory Specimen | Collect laboratory specimens from pet owners:  
Match patient record to the sample  
Submit the samples to veterinary technician or nurse  
Present clients with medications and routine instructions | Initials/Date |
| Trainer: | |
| Assign Bloodwork (In-Hospital) | Demonstrate the proper way to assign bloodwork within the practice. Before signing off, trainee must demonstrate the ability to handle this task properly. | Initials/Date |
| Trainer: | |
| Outside Labs | Explain the procedure for calling outside laboratories.  
Explain the procedure for outside laboratories results | Initials/Date |
<p>| Trainer: | |
| Communication with Clients | Learn hospital guidelines for communicating with clients in different types of situations such as general queries, scheduling appointments, routine and non-routine medical questions, patient emergencies, prescription refills | Initials/Date |
| Trainer: | |
| Medical Recalls | Demonstrate the procedure to follow when recalling clients. Before signing off, trainee must demonstrate the ability to handle this task properly. | Initials/Date |
| Trainer: | |</p>
<table>
<thead>
<tr>
<th>Task</th>
<th>Description</th>
<th>Initials/Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleaning Exam Rooms</td>
<td>Explain how to properly clean and disinfect an examination room.</td>
<td></td>
</tr>
<tr>
<td>Boarding Forms</td>
<td>Explain how to complete boarding forms</td>
<td></td>
</tr>
<tr>
<td>Boarding Reservations</td>
<td>Explain how to make a boarding reservation.</td>
<td></td>
</tr>
<tr>
<td>Cancel Boarding Reservation</td>
<td>Demonstrate the ability to properly cancel a boarding reservation.</td>
<td></td>
</tr>
<tr>
<td>Admitting Boarders</td>
<td>Demonstrate the correct procedure to follow when admitting boarders. Before signing off, trainee must demonstrate the ability to handle this task properly.</td>
<td></td>
</tr>
<tr>
<td>End of Life Appointments</td>
<td>Explain how end of life appointments are scheduled and how greeter should anticipate and prepare for these types of appointments.</td>
<td></td>
</tr>
<tr>
<td>Checking out the Client</td>
<td>Demonstrate how to check-out a client</td>
<td></td>
</tr>
<tr>
<td>Marketing</td>
<td>Discuss marketing to clients</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Discuss how to promote the practices products, programs and services.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Explain the use of passive marketing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ensure that employee gains a technical knowledge of products sold</td>
<td></td>
</tr>
<tr>
<td>Conclusion of Phase III</td>
<td>Review of Phase III of training program. Trainee is asked if he or she has any questions or needs further training on any part of Phase III. Trainee signs off on Phase III.</td>
<td></td>
</tr>
</tbody>
</table>

**Trainee comments - Phase III**

*Use this area for any comments you have concerning this phase of your training. This will help us to improve our training systems and ensure that adequate training is provided to you. Your comments will be read by the management of the practice and kept in your confidential employee file.*

---

**Phase III of training complete**

*My signature below signifies that I have completed Phase III of the Receptionist Phased Training Program and that I fully understand all concepts covered and I am comfortable in my knowledge and ability to perform the procedures introduced in Phase III of this program. Supervisor signature below signifies that employee has successfully completed Phase III and has answered all pertinent questions.*

<table>
<thead>
<tr>
<th>Employee (Trainee)</th>
<th>Date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supervisor</td>
<td>Date</td>
</tr>
<tr>
<td>Task</td>
<td>Description</td>
</tr>
<tr>
<td>----------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Opening and Closing</td>
<td>Demonstrate the procedure for opening the hospital</td>
</tr>
<tr>
<td></td>
<td>Demonstrate the procedure for closing the hospital</td>
</tr>
<tr>
<td>Surgical Charges</td>
<td>Demonstrate how to check surgical charges. Review the travel sheet. All services rendered should be highlighted.</td>
</tr>
<tr>
<td>Price Quotes</td>
<td>Explain how and when the Trainee is to quote prices.</td>
</tr>
<tr>
<td>Client Transaction Reports</td>
<td>Demonstrate how to prepare a Client Transaction Report.</td>
</tr>
<tr>
<td>Vaccination Protocol Handout</td>
<td>Present trainee with vaccination protocol handout and explain how to use.</td>
</tr>
<tr>
<td>Correspondence</td>
<td>Demonstrate how to communicate with clients regarding medical status, medical instruction, itemize and review the client statement, inform clients about hospital policies, payment and credit policies</td>
</tr>
<tr>
<td></td>
<td>Demonstrate how to print client correspondence i.e. reminders, thank you notes, new client letters</td>
</tr>
<tr>
<td>Bank Deposits</td>
<td>Explain how to prepare the bank deposit and complete the deposit slip.</td>
</tr>
<tr>
<td>Credit Cards</td>
<td>Explain the correct procedure to follow when batching credit cards.</td>
</tr>
<tr>
<td>End of Day</td>
<td>Explain the End of Day procedures. Before signing off, trainee must demonstrate the understanding of this task.</td>
</tr>
<tr>
<td>Returning Products</td>
<td>Explain which products can be returned. Demonstrate the correct procedure to handle returns.</td>
</tr>
<tr>
<td>Coupons</td>
<td>Explain how to handle coupons.</td>
</tr>
<tr>
<td>Bounced Checks</td>
<td>Explain the procedure to follow when a check bounces.</td>
</tr>
<tr>
<td>Accounts Receivable</td>
<td>Explain the procedure for handling accounts receivable issues.</td>
</tr>
<tr>
<td>After Hours ER Fee</td>
<td>Explain the after hour’s emergency fees.</td>
</tr>
<tr>
<td>Prescription Filing</td>
<td>Demonstrate how to correctly fill a prescription and the expectation that all prescriptions should be proofed.</td>
</tr>
<tr>
<td>Controlled Substances</td>
<td>Demonstrate the correct procedure used when dispensing controlled substances.</td>
</tr>
<tr>
<td>Recognizing an Emergency</td>
<td>Discuss referring clients for immediate treatment of their pets when the requests are accompanied by complaints of</td>
</tr>
</tbody>
</table>
### Trainer:  
**Heartworm Testing & Prevention**  
Initials/Date

#### Trainer:
**Flea Prevention 101**  
Initials/Date

#### Trainer:
**Client Complaints**  
Initials/Date

#### Trainer:
**Displays and Retail**  
Initials/Date

#### Trainer:
**Refreshment Area**  
Initials/Date

#### Trainer:
**Office Supplies**  
Initials/Date

#### Trainer:
**Clean Front Area**  
Initials/Date

#### Trainer:
**When in Doubt**  
Initials/Date

#### Trainer:
**Conclusion of Phase IV**  
Initials/Date

---

**Trainee Comments - Phase IV**  
Use this area for any comments you have concerning this phase of your training. This will help us to improve our training systems and ensure that adequate training is provided to you. Your comments will be read by the management of the practice and kept in your confidential employee file.

---

**Phase IV of Training Complete**  
My signature below signifies that I have completed Phase IV of the Receptionist Training Program. I believe that I fully understand the concepts covered and I am comfortable in my knowledge and ability to perform the procedures introduced in Phase IV. Supervisor signature below signifies that employee has successfully completed Phase IV and has answered all pertinent questions.

---

**Employee (Trainee)**

**Date**

**Supervisor**

**Date**
Clear! Shock Your Team to Life with These Energizing Strategies
Bash Halow, LVT, CVPM
Halow Tassava Consulting
New York, NY

Start with a sense of mission
Everyone wants to feel as though they are part of something great. Paint that picture for them. ‘See’ the great business that you want to build. Help others to see it. Show your existing and prospective employees how they can be a unique and important person in your vision of the future.

Dove Lewis, an emergency referral center in Portland Oregon, has already established itself as an exemplary center for care, but they continue to bait their employees to push past previous goals and grasp for new heights. Their current vision statement, ‘To have a global influence on the emergency and specialty veterinary profession…’ challenges everyone from the CEO down to revisit everything that they do, to improve themselves and to improve their organization. Dove is embarking on the equivalent of a moon launch: dangerous, but with an exhilarating prize, the first steps on a new world.

Take a look at your Mission statement. If it puts you to sleep, put it to sleep. Write what matters most to you. Say what you mean, not what you think you should sound like. Kick its tires. Make sure it can handle whatever kind of terrain lies ahead.

Partner don’t parent
Stop holding the hands of your adult workers. If you have employees still in the toddling stage, then send them back to nursery school where they belong. Your clients, patients and coworkers don’t feel like working with them and neither should you.

You are not your employees’ daddy or mommy. If you feel like being a daddy or mommy, don’t open a veterinary clinic to serve your needs; invite a friend over for dinner and wine and do it the old fashioned way.

Everyone wants to feel special and everyone wants to win. Figure out a way for every member of your team to uniquely contribute to the goals of the practice and then partner with them to make sure that it happens.

Assist your employees’ development
Getting in the way of your team members success can be issues with self doubt, jealousy, negativity and so forth…. probably a lot of the same things you’ve faced (and hopefully overcome) in your professional journey. Help team members clear these hurdles. Think of yourself as a personal coach watching someone workout. Give them a tip to lift the weight or do the squat in a way that leads to the best outcome. Teaching people how to be better, without shame, captures loyalty.

Nurture effective forums
Throttle up your meetings. Try standing and brainstorming for 10 minutes instead of sitting and chatting for 60. Break your practice up into small working groups where small issues are discussed, thought through, and solved. Keep the sessions energized and moving. Cue the exit music for anyone who talks more than 3 minutes. Sometimes people have to talk to hear themselves think, but more than 3 minutes of thinking out loud kills the energy of the group.

Abbreviate your agendas to one or two items. Energy, interest and meeting effectiveness drops off substantially after 20 minutes or more. Pick a problem, engage the group in thought and discussion, brainstorm solutions, and leave with an action plan.

Wake up people’s heads with a drawing exercise at the start of the meeting. Research ‘ice breakers’ that push employees to do something physically or mentally that turns on the creative (and hopefully fun) side of their minds.

Remember that all of the chitchat that meetings eventually devolve into is a natural need for all groups. We require social time. So let’s find a way for team members to socialize without shutting the practice down, feeding everyone pizza and wasting valuable meeting time. Sending people on breaks in groups, if the schedule allows, gives people a chance to talk, get to know one another and enjoy each other’s company. Have a budget for ‘drinks on us’ that’s available to any group of employees that wants to spend off-work hours gathering for lunch, dinner, drinking, bowling or whatever. Encourage them to invite team newbies along for the ride and include them in the fun. Some of our world’s most innovative business ideas (think Apple, Zappos, etc.) were cooked up over drinks in some bar long after everyone punched out.

Terminate poor performers
Get rid of the deadwood. Fire poor performers and affirm that the additional effort and energy that star performers give your organization matters.
Feedback and celebration
Remember how we keep calling our employees team members? So name a team where the coach sits in the field house and weighs in on the team member’s performance once a year. Managers should get out of the office and on the floor where they can witness and applaud your team members’ success.

Collect objective data on your business and share it with your team, and then use it as a jumping off point for a discussion on how everyone can improve. Take your most positive reviews (not your negative ones) and drill down into the particulars of each. Closely explore how you won, not how you lost, as a way to underline the behavior and actions that work and as a way to teach other team members how to better succeed. Consider celebrating team members on social media (with the proper permissions, of course). A public celebration of employees (provided it feels warranted and sincere) feels great for everyone involved.

You
If you’re not jazzed, your team members aren’t going to be jazzed. You are and will always be instrumental to your team’s sense of excitement and accomplishment at work. Hire all the practice managers or sub managers that you want, your role as chief visionary and figurehead can’t be outsourced. I’m not asking you to spend 7 days a week in the building, but if you can only punch in for one, make sure it’s a whopping good one.

I’ll leave you with a story
Picture it. I’m at a practice that’s undergoing complete reconstruction. The interior is a disaster. It’s 9pm and the only veterinarian working, the owner, is quietly washing his hands after treating a patient. I watch him rub the soap on his hands and mark how peaceful he appears.

What makes him happy?
What makes him happy is this: he has created and continues to create a place where he can grow, self-actualize and win.

Find a way to help others find themselves in the same position. Help them find themselves contentedly washing their hands, amidst a practice strewn with hurdles and caution signs, feeling happy and thinking that they’ve finally discovered just how great they really are.
Creating Better Expectations for Interpersonal Behavior in Practice
Bash Halow, LVT, CVPM
Halow Tassava Consulting
New York, NY

Healthy workplace interaction isn’t achieved by more policing on your part (and anyway…can you imagine how onerous such a job would be?), but achieved by individuals who take responsibility for how they think and behave with their coworkers.

The best way to understand how to help others to become more self-aware is to work on your own self-awareness and to study how you repeatedly error when it comes to healthy interaction.

Journaling is an excellent tool to explore one’s strengths and weaknesses. Quiet time allows for effective time with one’s thoughts. Take a walk with the dog and take one question with you, ‘why do I react the way that I do?’ Understanding how you tick will help you coach others off their hamster wheel of misguided thinking and interacting. Begin to teach others how to interact by working on your own interactions and understanding what helped you to change your ways.

**Underline the value of positive workplace interactions**

Unhealthy workplace interaction is so demoralizing, so deflating, and so destructive that management teams should have systems in place to avoid it in their business.

**Use civility rules**
These are written tenets by which everyone is expected to behave (greet everyone when you arrive to work, don’t ostracize team members, respectfully share your concerns with others, etc.) It seems silly: written rules that can be found on the bulletin board of any grade school, but our busy lives and the filter of social media have inured us to the feelings and thoughts of others. Major companies throughout the U.S. have added civility rules to their employee manual because they understand the major impact that rudeness (or perceived rudeness) can have on productivity and culture.

**Give equal weight to fairness**
Leaders that show favoritism create fertile ground for animosity between employees.

Provided you have respect and fairness as a foundation in your business and that you are sufficiently self-aware, you can use the following techniques to help your team improve the way that they interact with coworkers.

**Create an environment of accountability and responsibility**

It’s hard for coworkers to swallow the importance of kindly interacting with team members that don’t follow through with what they say they will do or who do slip shod work. Don’t avoid difficult conversations. Make it clear to all employees that if they say they’re going to do something, they should do it or communicate otherwise. Part of respect is staying true to your word.

**Stimulate thought**

You don’t have to always be the guy or gal brave enough to share your thoughts on how others can improve. Help team members wake up to who they are and how they behave with online emotional intelligence tests (just Google them). Allow them to take a Myers Briggs test. Invite your Zoetis rep to your practice to conduct a DISC training class. These are excellent tools to stimulate self-reflection and hopefully self-awareness.

**Create opportunities to get acquainted**

The more time your team has to learn about one another and find trust, the more likely they’ll behave better with one another. I’m not a big fan of bowling nights or after work get-togethers simply because so many team members are parents and need to spend time with their children, but why not organize some continuing education off campus with a select group of people? Time in the car or eating lunch with one another is a great way for everyone to get better acquainted, and because it’s CE related, it won’t feel like a superficial sacrifice of time.

**Inculcate a sense of team**

Employees that feel part of a team are more likely to interact better than those that feel as though it’s every man or woman for him or herself. When hiring, allow your employees to participate in the selection process. Remember that a big factor in working ‘as a team’ is enjoying the people that you are working with. Expecting employees to work well with people that they don’t enjoy being around isn’t wholly realistic. During the hiring process, allow team members a chance to sit and talk with candidates (OFF the floor) so they can determine if they are a good match.
Why are you angry?
My business partner is a serene employer. She can weather employee and business set backs with a level head and calming demeanor. I don’t notice. Boy, she ticks me off.

For whatever reason, things that don’t bother others infuriate me. A lifetime of ranting and anger outbursts have characterized me as entertaining, obnoxious, a know-it-all, unbearable, destructive, brilliant, and bullying. I think it’s safe to say that my often-negative outlook on things has undermined nearly all of my efforts to be as successful as I could have been.

After years of cycling on this hamster wheel of outward and inward destruction, I decided to stop and take a moment to examine why I was angry. What I discovered was the key to curtailing the behavior, improving my success as a leader, and most importantly, increasing my personal happiness.

Why do you get angry when you or your team falters or fails at goals? If you are like me and the many other veterinary professionals with whom I work, it’s because you take your responsibility of caring for animals and their owners very seriously and to fail at it feels like a reflection on your character and your commitment.

Not always bad, anger can be a sign from a perceptive and invested individual that something is off. Directed constructively, anger can lead to enormous positive change. The appropriately named not-for-profit MADD, which stands for Mothers Against Drunk Driving, has an annual budget of 45 million and has done enormous good in the world simply because one woman found a positive way to direct her anger into a venue for change.

Planning
One reason why goals don’t often go as planned is because we leaders have spent an insufficient amount of time planning how they should be achieved. It’s okay to enter a meeting with a vague idea of what you would like accomplish, but it is your job as a leader to make sure that the details of the idea unfold as the group and you work forward.

One strategy that I have found enormously helpful is to play with the idea myself before I introduce it to others. Tinkering around with a new concept or protocol…beta testing it if you will…gives me a chance to figure out what I actually want. With a clearer understanding of what I’m trying to accomplish and build, I am much better at directing others to replicate the process and more likely to get a good outcome.

Training
I have hired, coached and supervised many kinds of people in my life: all ages, all socio-economic backgrounds, all intelligence levels. I believe that communicating a vision of what you want accomplished to others is fraught with crossed wires and static. A clear idea of what you want to accomplish and a systematic approach to uploading others on how you want things done improves your chances of success. It’s time consuming; it’s a drag…especially when you’re already swamped with work… but methodical training is essential.

Feedback is important to team members, but it can be overwhelming, especially for new hires. There’s an art to correcting employees in their first weeks of employment or their work on the latest project. Look for signs that you may be overwhelming your employee. If today isn’t the best day to hone their behavior, another time with present somewhere down the line. Rome wasn’t built in a day.

Let it go
After you are dead, the world of veterinary medicine will go on. Even now, veterinary medicine gets accomplished all around without a syllable of input from you. Take a leap of faith. Give a little elbowroom to those that are also working on whatever project is at
hand. It might not get done to your satisfaction today, but with time, you and your team can sort through additional details and ultimately shape a product that meets or even exceeds your expectations. Additionally, because you’ve allowed others to own the project and work on it more autonomously, you are more likely to capture their interest and discretionary effort.

**Bullets for managing your anger**

I once interviewed a PHD and renowned expert on the topic of workplace anger. I asked, ‘What advice do you have for those of us that tend to blow up at work?’ His answer, after a lifetime of education, research and work on the topic was, ‘Try counting to 10’.

**I could have killed him.**

But he’s right. Taking 10 seconds before you respond (well in my case, I need 24 to 48 hours to cool off) can improve your chances of keeping your cool. In my case, a night’s rest is all I require to emerge with a whole other perspective. ‘What was I so upset about?’ is often my morning-after thought.

We require time to center, an increasingly challenging prospect in an age that insists on drowning out every thought in our head with a cellphone alert, with banal background music, insipid public alert messages, talk show blather, Internet pop ups, and so forth (huh-oh, who is getting angry again?)

I figure, we already have a dog; we should walk it. We have a cat. We should plop it in our lap and love it and spend some time to remembering that when we’re not ticked off, we’re kind of remarkable. When we’re not intent on kicking over sand castles, we’re capable of creating a lot of joy for others. The fact that we get angry is a sign of intelligence, perception, and a passion to pursue what’s right, but let’s agree that letting it get the best of us only worsens the very thing we were upset about to begin with.

**Express it**

Find a way to express your anger constructively. Make sure you’re calm (for me this means taking a waiting period to cool off), give the person that you are talking to the benefit of the doubt (in my experience, they are rarely clued into how a particular thing is making me feel), and share your thoughts. In such interactions I regularly self-check. I ask myself, ‘Am I building a relationship here or am I just being right?’

We avoid delicate situations because we’re concerned that conflict will spoil our relationship (i.e., the employee will quit!). I suppose the employee may quit, but most times, people appreciate that you care enough to be honest.

Relationships aren’t always happy. Occasional conflict and the resolution of that conflict create a sense of trust that the relationship is tested and true. Just try to share your feelings without being nasty or disrespectful.

I’ve been in anger recovery now for about 7 years with numerous relapses. In retrospect, the 4/5ths of my life I’ve spent shouting hasn’t demonstrated my power or my strength, it has underlined my mediocrity, my weakness and my fear. In the past, a project went south, and I got mad because I thought the failure was a reflection of who I was. In fact, it wasn’t the failure, but my reaction to the failure, that demonstrated to others how inferior I really was.

Anger has never helped me get a goal accomplished (unless I used the anger to fuel constructive action on my part) and has never provided the lasting catharsis that I thought it would (blow ups only feel good for about 20 seconds before leaving you feeling gross and toxic). Take time to think about why you get mad. Reflect on whether or not such a feeling has helped you to succeed or to move your goals forward. And enjoy the walk with the dog. Exercise is good for the soul.
Teach Your Team How to Relate to Clients and Build Compliance
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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.

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**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.
Consolidation, technology and an eroding middle class (Pew Research Center, 2015) are reshaping the future of veterinary medicine. The animal health landscape of 2020 and beyond includes very successful, well-managed, private practices; a significant number of publicly-held and corporate practices that focus on affordability; and a dwindling, beleaguered, collection of low margin practices that have failed to plan in the face of obvious warning signs. Veterinary healthcare leaders interested in a long, lucrative and successful career should plan for change now and take a proactive position in leading their hospitals and the profession.

In this paper, we’ll look at the most significant factors that will define our future marketplace; what the career of a veterinary manager will look like in such a world; and changes that practice managers can undertake now for themselves and for their practice that will ensure optimum success.

The future of veterinary medicine will be influenced by the following trends:

- Better informed, more devoted pet owners with less money than the pet owners of today
- Consolidated groups of practices, both publicly and privately owned, and an increase in low margin, bare-bones practices
- An increase in fixed and non-fixed business expenses that drives down margin and that most significantly impacts non-consolidated practices
- Increased veterinary management acumen and better leadership in general
- Credentialed technician and talent shortages
- A widening technology gap between large companies and small businesses that puts the latter at a note-worthy disadvantage

More devoted pet owners with less money

More pet owners consider their pets to be members of their family than ever before. In a 2011 Psychology Today article Do We Treat Dogs the Same Way As Children In Our Modern Families? Author Stanley Coren PH.D, F.R.S.C. writes:

A new online survey by Kelton Research, involving about 1000 people, shows that the status of dogs as family members is changing. It appears that in the minds of the Americans who responded to the survey, dogs are becoming more important as family members, particularly as children. Most recognize that this represents a change in attitude since nearly 60% believe that their dogs are currently more important in their lives than were the dogs that they had during their childhood days (Coren, 2011) (Coren, 2011)

According to the AVMA, 51% of pet owners allow their pets to sleep in the same bed, another 31% buy their pets holiday presents, and an overall 63% of pet owners think of their pet as family (AVMA, 2012). According to a study that was conducted by the Pew Research Center in 2010, 85% of respondents said that they considered their dog to be a part of their family and 78% considered their cat to be one (Pew 2015). Wall Street, taking note of the exploding popularity of sites like LOLcats, Corgi tumbblrs and social media outlets alive with pet photos also recognizes an American populace that loves, if not adores their household companions (Or, 2014).

However according to the AVMA Pet Ownership and Demographics Sourcebook the number of pet owners taking their pet to the veterinarian decreased by 8% for dogs and 24% for cats in 2012. Between 2007 and 2012 there was a nationwide 13.5% decrease in veterinary cat visits. One reason respondents cited for not going to the veterinarian? Money (AVMA, 2012).

Today’s American is worth far less than they were in 2003. According to The Atlantic:

Median net worth has declined steeply in the past generation—down 85.3 percent from 1983 to 2013 for the bottom income quintile, down 63.5 percent for the second-lowest quintile, and down 25.8 percent for the third, or middle, quintile. According to research funded by the Russell Sage Foundation, the inflation-adjusted net worth of the typical household, one at the median point of wealth distribution, was $87,992 in 2003. By 2013, it had declined to $54,500, a 38 percent drop. And though the bursting of the housing bubble in 2008 certainly contributed to the drop, the decline for the lower quintiles began long before the recession—as early as the mid-1980s." (The Atlantic, 2016)

According to the Social Security Administration, as of 2014, 67.2 percent of wage earners had net compensation less than or equal to the $44,569.20 raw average wage. By definition, 50 percent of wage earners had net compensation less than or equal to the median wage, which is estimated to be $28,851.21 for 2014. In 2014, 75 percent of Americans earned $55K a year or less (SSA, 2014) To put all of that in perspective, the U.S. Department of Health and Human Services draws the poverty line for a family of four at $23,850.00 (Univ. of Wisconsin Institute for Research on Poverty).

If the American public continues to humanize pets in the future, there will be a solid demand for veterinary care, but it must be affordable to a growing population with less money than it has today.
The opportunity in consolidation is dramatically changing our industry
Veterinary consolidation is trending up. According to the 2013 AAHA Pulsepoints, 6.9% of respondents identified themselves as being a part of a multipractice group, three times the percentage reported in 2011 (AAHA, 2014). In 2014, Summit Partner’s NVA, with a run-rate earnings of 69 million dollars, was purchased by Ares Management for an estimated 920 million dollars or 13.3 times EBITDA (Or, 2014). That’s more than double the 4-6 times multiple seen in most sole practice sales. In March of 2016, VCA agreed to buy an 80% share in CAPNA (Companion Animal Practices of North America, a group of 56 free standing veterinary practices) for 344 million dollars or 10.7 times a 2016-projected EBITDA (The Fly, 2016).

This is significant. How does a 60-something veterinarian turn away from an offer that’s double what he or she would otherwise be offered? Alternatively, how do younger veterinarians, potentially saddled with debt, but eager for a chance at ownership, compete with the prices that buying groups can and are offering? How does acquisition change a practice’s culture and its management’s autonomy?

Since the sale of NVA and CAPNA, Vetcor Inc. and PetVet Care Centers could be next. With only 5.5% of veterinary revenue being produced by VCA, Wall Street views consolidation of veterinary practices as a big opportunity for private equity investment (Or, 2014). Consolidation will likely reshape a significant part of the veterinary landscape of the future and while that’s not necessarily bad, it’s certainly very different from what exists today.

The rise of on demand veterinary services
In May of 2016, a congress of professionals met at the University of Michigan to discuss an alarming fact: today’s recently-graduated veterinarians have a 2-to-1 debt to income ratio (Williamson, 2016). Some of these veterinarians, looking for ways to take control of their debt and gain autonomy over their lives, are taking a fast track into business ownership and/or independence. They’re hiring a single, on-demand employee and taking their practice mobile (Cummings School of Veterinary Medicine, 2014). In some cases, a central office dispatches the veterinarians, lowering the veterinarian’s time invested in managing client load and scheduling. Central offices, or the young veterinarians themselves, leverage facile digital marketing skills to outcompete brick and mortar vets for premium online visibility. These practices’ low-cut pricing structure puts additional competitive pressure on stand-alone practices.

Sites like Petcoach pay veterinarians on a case-by-case basis to answer online veterinary questions. Their aggressive marketing means that Dr. Google just added one more associate to a list of veterinary resources that will outcompete your practice for top search engine results and the next new client. It means that sole practitioners can completely eliminate the need to own a brick and mortar facilities or even the infrastructure to book appointments.

An increase in fixed and non-fixed expenses
Comparing practices in the AAHA Pulsepoints 4th and 8th editions (2006 and 2014 respectively), 25% of veterinary practices saw an increase of expense-to-gross ratio by 5.1% or greater (AAHA, 2014) with decreases in surgery, sedation and anesthesia, hospitalization, euthanasia and other medical income. Additionally average transactions, active clients per veterinarian, and new client numbers were all lower than the 2011 averages (AAHA, 2014). Surprisingly Pulsepoints reports that expenses for web presence, online reputation, and digital communication and marketing account for 1 to 1.5% of a practice’s annual gross revenue, roughly the same amount of money we used to spend in the old Yellow Pages days (AAHA, 2014), but I can’t imagine that that is true and I expect as we get better at calculating the money we're investing for visibility and marketing, including payroll hours, we’ll determine that our expense in this area is much higher.

Decreases in revenue may have something to do with the increased expense-to-gross ratio. Online price shopping forces practices to lower margins on what used-to-be more profitable pharmacy and over-the-counter products. The rise of low-cost, spay-neuter clinics and other not-for-profit (and some times publicly funded!) facilities providing low cost veterinary care have impacted our service sales. Additionally, there is the aforementioned decline in veterinary visits.

It’s important to point out that when one looks at overall net profit as a percent-to-gross revenue, AAHA Pulsepoints indicates that 25% of practices earned 5% more (or greater) in 2014 than they did in 2006, while 25% of all practices in the same study had a 5% decrease (or greater) in net return between 2006 and 2014. But there is also an indication that overall non-veterinarian staff per FTE veterinarian is down. If practices have reduced the size of the staff in response to the recession or if practices are swopping out hard-to-fill credentialed nurse positions for non-credentialed personnel, (Wu, 2015) that could indicate that higher net returns are due to austerity (AAHA, 2016).

Credentialed technician shortages
DoveLewis Emergency Animal Hospital, a provider of veterinary professional education (On the Floor @Dove), recently discussed their decision to include non-credentialed employees as members of their professional nursing team as a response to an acute, ongoing shortage of credentialed technicians.

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“The shortage for credentialed and licensed techs is widespread and while some states have not been affected, most have been. The BIG issue – the industry is simply not retaining technicians. Their career span is a short 5 years. We lose them to other careers, commonly they go to human healthcare nursing where they can make significantly more.” (Maxwell, 2016)

Anecdotally, Kenichiro Yagi, BS, RVT, VTS (ECC, SAIM), a familiar face on the speaking circuit, says he frequently hears audience members expressing concern about technician shortages. According to the Bureau of Labor Statistics, veterinary technicians or technologists with a 2 or 4-year degree respectively, earn a mean pay of $15.30/hour or 32K a year. That’s at least 10K dollars per year less than the starting pay of a registered nurse (BLS, 2016)

Tech shortages mean higher payroll costs to retain credentialed staff, retention issues as valuable team members migrate to practices with better benefits and opportunities (consolidated groups), and/or increased training responsibilities for the practices as they struggle to cobble together nursing teams from non-credentialed team members.

**Increased veterinary management acumen**

Our future’s successful veterinary practices won’t owe their staying power to dumb luck. Though some practices with strong leaders will fail, none will succeed that don’t embrace effective, informed leadership as part of their business model. Leaders in these practices can be former client care representatives, assistants or technicians, but they won’t be without a passion for leadership or a solid education in veterinary management through organizations like the VHMA.

The days of winging it are through (indeed have been for some time). Tomorrow’s practice managers won’t be in training wheels. They will be informed, experienced business leaders proactively responding to market pressures.

**A widening technology gap**

Today’s small business must manage an increasingly large web presence that includes online reputation management (reviews), SERP (Search Engine Rank Position) and an ongoing dialogue with clients on social media. Additionally Google, the web’s most popular search engine, primarily ranks websites by original, popular content forcing businesses to regularly update their websites. I think most would agree that those online responsibilities alone are a formidable obligation, yet a new responsibility looms on the horizon: leveraging online user data.

In a NY Times article titled, Facebook Is Using You, the author states:

> Facebook made $3.2 billion in advertising revenue last year, 85 percent of its total revenue. Yet Facebook’s inventory of data and its revenue from advertising are small potatoes compared to some others. Google took in more than 10 times as much, with an estimated $36.5 billion in advertising revenue in 2011, by analyzing what people sent over Gmail and what they searched on the Web, and then using that data to sell ads. Hundreds of other companies have also staked claims on people’s online data by depositing software called cookies or other tracking mechanisms on people’s computers and in their browsers. If you’ve mentioned anxiety in an e-mail, done a Google search for “stress” or started using an online medical diary that lets you monitor your mood, expect ads for medications and services to treat your anxiety” (Andrews, 2012)

As online users, we have a profile we create, but data-aggregation companies like Google and Facebook are creating one for us based on what we search for, what we write in emails, who are online friends are, what we buy, what we search for, and literally every key stroke we make on the computer, tablet or smart device. This information is used to create the world’s most effective marketing tools to be sold to the highest bidder. Not a bad world to live in... unless you can’t be the highest bidder.

If you thought it was hard to keep up with posting on Facebook, look out. The next wave of marketing options available to you will be the most effective selling tools in the history of the world, but they will cost money and small veterinary practices, already cash-strapped and challenged for time and expertise in this area, will find it hard to both keep up and pay up.

**The veterinary practice manager in 2020 and beyond**

**Scenario 1: Success**

Five years from now you will be directly responsible for a practice that is consistently growing and financially healthy. You will have achieved this by reviewing the internal and external forces at work on your business and on the market and made successful, confident, proactive business decisions. You will have grown the business to keep pace with your growing talent and your increased salary demands. You will have built a vertical pathway for yourself and many members of your team.

Yes, your clients will have less money, but you will have put together some payment strategies for them that put great care within reach. After all isn’t that what these people who think of their pets as ‘family members’ want?

Yes, you will have higher expenses, but your leadership skills will have honed a team that puts forth its best effort. Clients will pay the additional money; they’ll wait the additional time; they will elude the online ads of your corporate competitors because you matter when it matters most: face-to-face, in the lobby, in the exam room, and on social media (Wu, 2014). As one veterinarian recently said to me at a conference, “No one can compete with what I do for my clients in the room”. You will be successful because you will have actualized your team to be a walking, talking billboard of your mission statement. Technology is all well and good, but no future, however bleak, will dethrone client connection as reigning supreme.

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You’ll be known in the community through your presence online, through your practice owner’s (and your own!) charismatic leadership. You’ll be thought about, talked about, and connected with as a personality, not a practice.

You’ll be happy. So will your team members. In general, happy people are a magnet. Happy people in a business are a business magnet. As a leader, you’ll pay attention to your workplace culture.

In a highly competitive world, the need to constantly inspire employees will change the perception of what inspires. Leaders will realize that fresh, changing environments with powerful, authentic stories on the walls is more inspiring than expensive art collections. In the future, more attention will be paid to marketing and branding internally to connect employees to the company’s mission and the impact employees have on their customers. Including individual employees in the brand story and allowing them to become a part of history in the making will be an effective employee engagement strategy. (Roby, 2015)

You’ll be on a constant look out for opportunity. You’ll have hooks in the stream to catch the best employees, the best new clients, the best deals, the best opportunities for your team to learn. When people ask you what an entrepreneur is, you’ll tell them that it’s a business person that trusts their gut and takes big risks and you’ll reference a few stories from your own practice and your own career as a way to expand upon that definition.

It is likely that your practice will be part of a loose union of similar practices that share proprietary management strategies and pricing information, broker buying deals, and potentially collude on exit strategies for some if not all of the members of the group. A portion, if not all, of your future practice management continuing education will come from within the group or from outside parties that structure education (and its cost) to specifically meet your group’s demands. Your group will scratch the backs of preferred vendors and they’ll scratch yours, but because of your group’s size, your ‘scratch’ will cover more ‘back’. The future education for you and your team will be more effective, specific to your needs, and in part bank rolled by vendors eager to do business with you and your group.

Scenario 2: Your practice will have been purchased and you’ll come with the package
You may be one practice in a group of a dozen or so, or part of a larger company that includes 50 or more practices. Your practice’s owner may or may not have shared his intent to sell with you and on the day of the transition you may have been completely caught off guard. Don’t hold a grudge against your former boss. This was his or her chance at retirement and a way to ease him or herself out of the veterinary work schedule over a few years.

Your salary was sustainable so the new owners kept you in place. Besides, the new owners wanted to make sure that the transition went smoothly and there was as little disruption to business as possible.

You may be asked to focus more closely on things like payroll and inventory costs and asked to take more aggressive measures to reduce both. In this new world, decision-making will likely be more collaborative and time consuming.

You may have easy access to hard-to-come-by resources like HR and legal advice, marketing materials, and the support of other managers of your caliber and experience level. As a part of a larger group of practices, you will attend regional or district meetings where you’ll find inspiration and reaffirmation of the company’s goals. Such meetings will provide you a chance to shine and grow in entirely new ways and be the first step to a larger, more influential position within the company.

Scenario 3: Fading into the sunset
Another scenario is that we wake up in 2020 and find you growing transparent. Waiting for the future to happen to you, then reacting to it, will put you and your company in a constant position of catch up. Choosing to focus on the day-to-day without ever proactively planning and leading towards a better future is an abdication of your real role and a PTS pathway for your practice. Choose to coast along as though it is business as usual means that both you and your practice will fade off into the sunset, will mean that hundreds of thousands of dollars worth of business value will spill through your fingers, means that you will have donated your market share to your competitors.

Scenario 4: New horizons
There’s also a chance that you will be pressured to retire or that you will work yourself out of a job. Practices of the future will be pinched between a universe of online price shopping that stymies price increases and growing expenses. Businesses are trending towards the lean in mean. In an article published in the Seattle Times, the author writes, “A decade ago, (new businesses) in Washington (State) employed five or more people. Now, they’re hiring only about three workers, according to Bureau of Labor Statistics data.” Martinez, 2014) Your future employer may love you, but they may not be able to afford you. With your tremendous experience, knowledge base, and education, a position with a larger private or corporately-owned veterinary practice may be possible, but competition for the salary that comes along with such a job is likely to be stiff, not only from within the ranks of organizations like the VHMA, but from outside our industry, as young talented MBA graduates scour the job scene for work.
During the conference, we’ll have a panel of former and existing veterinary practice managers, who have tested the job-market waters outside of the practice walls, and who will report back their thoughts on the switch. You’ll hear stories of those that have gone onto veterinary business ownership, financial management, veterinary business acquisition, ‘consulting’, bookkeeping, website design, online support, team training, and sales for veterinary supply, communication, equipment, and pharmacy companies.

But graduating, or perhaps better put, shifting into fields adjacent to veterinary management will not be an option for everyone. If the salary for such jobs is high, it’s likely that the applicant pool will be very competitive and the screening process extensive (Wolfe, 2016). In America, those with advanced educational degrees earn more than Americans without advanced degrees. Older men and women without advanced degrees are more likely to be challenged to find well paying work and are more likely to withdraw from the employment pool altogether (Strause, 2012). It’s not entirely necessary to know what line of work you will be in five years from now should your practice management position go away, but you can still prepare for a potential new career by completing your college degree, earning a more advanced one, or finding ways to assist practices with their online presence, marketing, online security, recruiting, training and work culture needs (Uzialko, 2016).

There’s very good chance that you will cobbled together a workweek as an on-demand laborer. Successful businesses of the future, small and large, will adopt the ‘Hollywood model’ of getting work done. They’ll assemble smart, expert teams around a short list of goals, and then when the work is finished, disband the group (The Economist, 2015).

Businesses of the future will need to take advantage of the wealth of competitive advantages that lie in analytics, collaboration, marketing and online presence (including cyber security, cloud-based services, mobile adaptiveness), but their ‘lean and mean’ models will look to on-demand outside resources for help in this area; a boon for the skilled manager that not only understands how to make adaptations and updates in these areas, but who can do so in the context of the specific needs of the veterinary practice (Uzialko, 2016).

You may be part of America’s vast number of sole proprietors numbering in the millions. You’ll be your own boss, helping practices here there and everywhere with things like bookkeeping, financial oversight, software support, marketing, training, inventory and so forth all by way of the Internet. You’ll work from home with a load of laundry turning in a nearby room or a chicken roasting in the oven for when the kids get back from soccer. You’ll also be responsible for constantly looking for work, paying all of your education and business expenses out of pocket, and forced to face the same small business pressures you once had as a practice manager.

The impact on professional organizations
Organizations like the AVMA, State VMAs, the American Animal Hospital Association, and even our beloved VHMA itself exist to bring lobbying efforts, consistency, camaraderie, and high standards to the profession. Up to this point, these organizations have been clear leaders of the industry.

However, if the future includes more consolidated groups of practices that are self-determining, with their own thoughts on standards, their own lobbying efforts, and their own tracks of continuing education pointed specifically at internal goals, do the membership demographics of professional organizations change? If consolidated groups of practices leverage their size to capture hard-to-come-by sponsorship dollars for their own purposes, does that impact how professional groups are funded? Today’s professional organizations have tight budgets and are staffed with small crews. Will these organizations be able to meet the demands of their future members or will their efforts be eclipsed by the support and training that comes from within consolidated practice groups?

Will large vendors, eager to hold the attention and loyalty of their clients, develop their own manager groups, continuing education, and on-demand medical and management resources? Will there be other certificate programs for practice management, leadership and so forth that come, not from our Industry’s standby organizations, but from veterinary, for-profit companies?

The way forward
It’s not all Sturm and Drang. Nimble, smart, privately owned, well-managed practices will always be a part of our future veterinary landscape. Why? Because great service and genuine care will never go out of style and people who consider their pets to be family will do whatever it takes to make sure that they are well. These gifted practices have already begun to structure tomorrow’s success. Here is a list of what they are doing today.

Identity and goals
Success begins with a clear idea of what you want to be and how you’re going to be it. Future, privately owned practices won’t be run-of-the-mill; they’ll stand out. They will exemplify great service, overt caring, and expertise. The teams of these practices will be smart, engaged and engaging. Leaders of these practices will ask one question and answer it thoughtfully, ‘What are the defining and distinguishing elements of our practice?’ This question is often explored, but too-often incompletely answered in the mission statements we have written for our businesses.
Plan
Once you have a short list of clear goals, work with all members of your team to develop a written plan for how you will achieve it. Practice owners and leaders shortchange themselves when they assume that strategic planning is onerous or unnecessary. Emphatically, it is neither. A short list of what is vitally important to work towards is freeing for a taxed management staff and liberating for a team that can often feel pulled in too many directions. Use the strategic planning tools at www.halowtassava.com for more help in undertaking the straightforward and enjoyable task of planning.

Build a team
As a child, I was scrawny and picked upon. One summer’s eve our neighborhood’s favorite dad called together a game of flag football and much to my complete delight, he not only singled me out to be on his team, but called me into a private huddle where he uploaded me on a strategy of how we were going to win. Before each scrimmage, he directed me to go to a particular place in the field where he threw the football to me and I, to my sheer amazement, caught it. Subsequent to that, I was tackled merciless, but I got up from the dirt smiling more broadly than I had in my entire life. That man had given me a chance to noticeably and successfully contribute to the efforts of a winning team.

That’s a dynamic that exists in all teams in which members work with their heart and soul and it’s one that will exist, on some level, in your successful practice of the future.

Team building is straightforward. Hire people you want to see succeed and then provide them training, individual attention, and caring oversight to help them shine. The problem is that that responsibility is time consuming, emotionally draining and sometimes a trial of one’s patience. Nonetheless, there is no way around it. Build into your plan for the future a methodology by which you accomplish the above, not by which you search for an easier, but ultimately unsuccessful, workaround.

Learn and leverage the online world
The online world provides small businesses an unprecedented, cheap, extremely effective way of engaging existing clients and capturing new ones (Dugas, 2012), but it requires an investment of time and skill. It’s very likely that leaders will have to outsource this responsibility to someone in the future, but that will not absolve them of understanding enough about how effective online marketing works to oversee the process. Future practices will have tighter budgets, so it’s essential that their online marketing dollars be efficiently spent. To that end, all practice managers should hold their breath and take a plunge into learning how to build an effective online presence.

Practices should shift the way they search for new team members to best connect with affordable, young talented people almost all of whom can be found online. Find ways to celebrate and highlight your team’s efforts through social media. Consider buying help-wanted ad space on social media sites. Film short videos of your practice team in action as a way to inspire young people to join your force.

Clear patient and client care standards
Your team members’ job isn’t client education; it’s client connection. Education is secondary to stopping, listening, empathetically reacting, and then making a straightforward recommendation to the client based on your standards of care. Your future practice will distinguish itself because each of your team members, in their own, individual way, will be a caring mouthpiece of your practice’s expertise, organization and experience; but most importantly each member of your team will be a source of connection, an extension of the kind of relationship every practice owner wants to cultivate with his or her clients.

Boots on the ground
It is said that George Washington returned from some battles with more than 17 bullet holes in his waistcoat. One of our greatest leaders in American history didn’t bark out orders from his office in Mount Vernon, but mounted his horse, rode amidst the troops, and fought alongside all of his men. Get out of your manager’s chair and onto your feet. There is work to be done at your desk, but the more important work is happening in the lobby, and exam, treatment, surgery and kennel rooms. Get to it.

Look in the mirror
On some level, you’re a contributing reason why your practice growth efforts stall. Insight into how you may be holding your team (and yourself) back are critical to your practice’s (and your) long term success. Find a way to gain insight into who you are and why you do what you do. In all seriousness, you might try a therapist. They’re trained to help people ‘see’ themselves and to figure out why they do what they do. That kind of knowledge is essential if you are going to improve.

Face facts
You may have problems as a leader, but you’re not the only reason why your team’s efforts stall. Many of you know of one, two or several members of your hospital that drag the entire place down. You’re not crazy. Those individuals really are a wrong fit. Terminate them.
Act

Your story and the story of the business that you run is an unfinished manuscript the last page of which has today’s date on it. Take up your pen and decide where this story is going to go, which characters you’ll introduce and which ones you’ll kill off; how you’ll behave and where you’ll be in this brave new world of tomorrow.

Success in the movies follows a familiar pathway: the main character loses everything; a defining moment when the same character commits to change, a montage of images showing the character’s short-term triumphs, and finally the last five minutes of the film where the character lives happily ever after. Real life seems much different.

Your future success is inevitable if you stop gauging it by material accomplishments. Think of adversity and failure, not as setbacks, but as part of a path forward and upward, because, indeed, they really are. You can be the weakest person on the team and still catch the football; you can eat a mouthful of dirt and still stand and smile. Even stumbles launch you forward.

Real life is better than the movies. In real life, we have the lead role, we understand everything that the main character thinks and feels, the photography is amazing and the audio is in Surround. Best of all, the popcorn and soda are much, much cheaper.

Just do the part where you choose to change. Commit. Choose a path. Act. The future doesn’t have to happen to you. You can happen to the future.

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Until 10-15 years ago, veterinary clinics could pay little mind to their pharmacy management and yet still rely on it for a steady stream of revenue. Pricing products was simple as all clinics used the same standard mark-up method. As management software developed, this became even easier with built-in pricing and inventory strategies. However, multiple outside influences have recently begun changing this paradigm:

• Existing pharmacies and retail entities have begun tapping into the veterinary profession with profit goals of less than one fifth of historical veterinary profit margins

• Online entities continue to spring up every day offering extremely low prices and convenience of home delivery

• Human generics lure people away from safe, FDA-approved veterinary products

• Generics are being dispensed for as little as $0 at large stores to bring customers into their stores for other purchases

• Generic forms of older products are being developed by large retail companies to be sold at significantly lower prices at their stores

Since 2011, the annual number of veterinary prescriptions being filled outside of the clinic has almost doubled with revenue loss to these entities approaching $500 million each year. With most practices drawing 17% of their revenue from their pharmacy, the effects of this trend can be significant. Clinics have tried to compete by carrying the same human generics or price matching outside companies. However, this has a huge impact on the clinic's profit. When that loss is calculated into the clinic's overall value, a retiring veterinarian could lose hundreds of thousands of dollars at the time their practice is sold.

While increasing service-based revenue can help to alleviate these losses, there are still changes that can be made in the veterinary pharmacy to help as well. The most influential is to choose innovative, non-shop-able products when determining your patients’ treatment. Injectables are the best example of these products. They require an establish Veterinary-Patient-Client Relationship to administer and, therefore, cannot be obtained at an outside source. Unfortunately, current pricing models make using these products in larger animals a cost-prohibitive venture.

Veterinary pricing has historically involved the simple mark-up method. It is easy to implement and it was assumed that all competitor clinics were using the same standard 2.5x mark-up. Before clients realized that veterinary medications could be obtained elsewhere, it allowed clinics to gain a 150% profit on medications dispensed from the clinics. Unfortunately, most retail entities are only seeking 16-24% profit margins. When these companies realized the potential revenue to be had by tapping into the veterinary field, the pricing difference they offered our clients was huge. Any clinic that did not start taking protective measures began seeing a negative impact on their clinic's revenue and reputation in the community.

The mark-up model is also unfair to the owners of larger patients. While these owners should expect to be paying more for larger doses of medication, the mark-up model of pricing creates a situation where client cost increases at a much steeper rate than the cost of the medication. When comparing a 10-pound dog to a 100-pound dog, the clinic is collecting over 17 times the profit from the owner of the larger dog for making the same diagnosis, dispensing the same medication, and certainly not doing 17 times the work.

The solution to this dilemma is the margin model of pricing. It is a simple method that allows the clinic to cover cost of the medication, overhead, and associate production, yet still take in a steady profit from patient to patient. It lowers the client cost of medications in larger patients and will eliminate the sticker shock associated with using newer, more innovative products. As a result, clinics can provide better care to their patients. They can be heroes to their owners for both helping their pets and keeping the cost of care more affordable. All the time, they are still protecting their revenue and profit.
Being an associate veterinarian (especially for a recent graduate) can be stressful at times. As a new graduate, you may be concerned that you are not efficient enough, spend too much time on surgeries, or that you do not communicate well enough with clients, and so on. There is also the feeling of being on your own and practicing veterinary medicine for the first time. This feeling can be overwhelming; however, with good communication skills and a desire to learn, the practice of veterinary medicine can be very rewarding. Another concern is that of paying off school loans after graduation. The loan repayments that our new graduates are faced with are at an all time high, and these numbers are most likely only going to increase in the future. So, despite the anxious and scary feelings of being a new associate veterinarian, these new graduates must also seek for positions that will compensate them well enough to survive. Young veterinarians are always looking for ways to increase their gross production so that they may reap the benefits of a possible raise or even a bonus at the end of the year. There are numerous "small" ways to increase an associate's gross production. It is as simple as practicing great medicine. The following are a few suggestions for helping to increase your gross production:

**Ear cytologies**

An ear cytology should be performed on every patient that is shaking his or her head, has red and inflamed ears, or just a large amount of dirt in the ears. This test is very simple to perform and only requires a few minutes. They allow us to determine if we are dealing with a yeast or bacterial infection and also guide us to the best treatment for the current condition. For example, the average price for an ear cytology is $18.00, and one of the most common issues we see in veterinary medicine is diseases of the ear. Therefore, if you see just 5 ear cases per day and perform 5 ear cytologies, this will create $20,700.00 in gross production if working 4.5 days per week. Ear cytologies serve to practice good medicine and, at the same time, are great income builders for the practitioner and the practice.

**Fecal examinations**

A fecal examination should be performed on every newly acquired pet and should be recommended annually as a good preventive measure. This examination should also be performed on every patient with vomiting, diarrhea, or weight loss. It only requires a few minutes and supplies us with a large amount of information concerning the pet's current gastrointestinal health. If intestinal parasitism is diagnosed, the fecal examination will guide us to the type of deworming agent to use. Fecal examinations are not only great diagnostic tools, but also great income producers. For example, on average, I see 15 cases per day in which I perform a fecal examination, and the cost for this test is $16.00. Therefore, this will create $55,200.00 in gross production if working 4.5 days per week. It is much more satisfying to be able to explain to a client exactly what we are treating (intestinal parasites, bacteria, etc.) after performing a fecal examination than just placing the patient on an antibiotic without knowing what exactly is being treated. It is money well spent and the client's respect us much more if we can give them a diagnosis.

**Annual wellness blood work**

Annual wellness blood work should be performed in all of our middle aged to geriatric patients, and should be recommended in all patients. It is so much easier to diagnose a chronic disease (liver/kidney) if we closely monitor the numbers. This close monitoring allows us to initiate the proper treatment at the ideal time. Also, if blood work is performed at a young age, we will obtain a good "base line" to compare to in the future. It is so nice to have the opportunity to catch acute or chronic conditions before it becomes too late. Also, clients appreciate it if we diagnose their pet's condition(s) before it is too late. From an income standpoint, I see an average of 5 senior pets per day and the wellness blood work costs $75.00. By doing this every work day, once again with a 4.5 day work week, you will create $87,750.00 in gross production for the fiscal year. Annual wellness blood work is a great practice builder, very good income producer, and, once again, it is great medicine.

**Chronic medication blood work**

Any patient that is on a chronic medication (NSAID's, steroids, immunotherapy, seizure medications, etc.) should have blood work performed every 6-12 months.

**Supplements**

Another great therapy we have in practice today is that of supplements. There has been some controversy on how well these supplements work and if they are improving our patient's health. These include joint supplements (Glucosamine/Chondroitin Sulfate), fatty acid supplements (Omega 3 Fatty Acids), liver supplements (Denosyl, Denamarin), and many others. I personally
believe greatly in these supplements and that they have the potential to increase the health and longevity of our furry friends. We should all be discussing the use of them in our exam rooms. When presenting the research of these supplements to clients, coupled with the long term results, they are very appreciative. As far as joint supplements are concerned from an income standpoint, my clinic sells the glucosamine/chondroitin supplement by the bottle. A bottle lasts, for most patients, 5 months and we sell it for $61.00 per bottle. I recommend this supplement for all of my patients, but especially large breed dogs, small breed dogs with orthopedic abnormalities, and middle aged cats that hide their arthritic pains so well. I do not have an exact number of patients for you, but if you have 1,000 patients on this supplement daily, you will be selling 2.5 bottles per year at $61.00 per bottle. This creates $152,500.00 in gross production for the fiscal year. Having said this, I do not recommend marking up supplements very much since they are a long term product and not a medication. My clinic routinely increases the client cost by 30% over clinic cost. These supplements are perfect for our patients' health, great practice builders, and, most importantly, serve to build the doctor-client-patient relationship and the human-animal bond.

Weight loss plans
If you are like me, about 60-70% of the patients you see are either overweight or obese. This is becoming a huge (no pun intended) trend and it does not seem to be slowing down much. The pet will be fed the same foods the humans eat, over fed the treats and regular food, and not allowed enough exercise. I have begun formulating personal weight loss plans for my overweight patients. In these weight loss plans, I discuss the pet's current weight, ideal weight, and the amount of daily calories to feed in order to obtain the ideal weight. I break the plan down on a monthly basis and set weight loss goals for each month. I have the clients bring the pet in every month for a weight check. During the initial visit, I will discuss how to determine the current body conditioning score. I will first ask the client how he or she feels about the pet's weight (underweight, ideal, overweight, or obese), and then give my professional opinion. This is the best way to spark the conversation. It is wonderful to see the smiles on the client's faces when their pet comes in wagging his or her tail, happy as can be, and has lost down to the ideal weight. One of the best ways to increase your gross production, and your success as a veterinarian, is to find your own niche in daily practice. I have found this through focusing a good bit on weight loss and formulating the weight loss plans. I have found that if I just go one extra step to discuss the weight issue and formulate a plan, my clients are so much more satisfied and they become a client for life.

Healthy treats
A healthy treats list is something that all veterinarians should have available. Our clients come to us for diet and treat recommendations and we should all be educated on what diet and treats are going to be best for our patients. My healthy treats list consists of many fresh fruits, vegetables, and yogurt (list compiled from Chow Hounds by Ernie Ward, DVM) and they are low calorie and fun for clients to feed their furry friends. All of my clients are amazed when I present them with the list. The best time to present and discuss this list is during puppy or kitten visits. It is always a good idea to get our clients and patients started out on the right path as far as nutrition is concerned. It is much easier to start a good, healthy habit than to change a bad habit.

Educational handouts
Our clients are presented with good news and bad news every day, and hopefully the good outweighs the bad. We see many different types of issues on a daily basis. One of the most important and valuable pieces of information a veterinarian or staff member can present the owner with is an educational handout. These handouts can be originals formulated by yourself or you may find many through veterinary software companies. They should give a brief description of the condition that has been diagnosed and the treatment plan involved. Handouts mean so much more to the client than we could ever imagine and they also allow us to save time and remain efficient in the work place.

Client callbacks
The most important lesson for a veterinarian to learn and master is that of client communication. This is what makes or breaks a practice. If you are not a good communicator, you will never be seen as a good veterinarian in the client's eyes. This especially applies to our surgery patients. On a routine day, most of us perform a number of surgeries that we consider routine. However, in the client's eyes, no surgery is routine because their baby is being put under sedation or general anesthesia for the procedure. Most clients are usually on pins and needles while awaiting the results. One of the best clientele builders for a young veterinarian is client callbacks following a surgical procedure. All young veterinarians should take a few minutes to call their patient's parents following surgeries. This allows us the opportunity to communicate with the client, put them at ease by letting them know that all went well prior to, during, and after surgery, and shows the client that you are concerned with their pet's well being and safety.

Update your clients
We have hospitalized patients and drop-off patients every day. This is routine for us, however, we must always remember that it is not routine for our clients. We need to update our client's on their pet's status at least 1-2 times daily. The easiest way to remember this is to treat every patient as if he or she was your own pet and that you would like to constantly know how that pet is doing. This
involves a simple, short phone call for us, but means so much more to the client. A great, successful veterinarian is one who communicates very well with his or her clients.

**Euthanasia**

Euthanasia is the toughest part of our job. We are faced with many situations in which the pet is very ill and the clients are emotional wrecks. The most important lesson for all veterinarians to learn is that of preparing ourselves for these situations and never allowing ourselves to get into a rush during this trying time in the client’s life. The way we handle euthanasia deeply impacts our client's view of us as veterinarians. I recommend offering services such as cremation, clay paw prints, and even something as simple as shaving some of the pet's fur off to send home with the client. These small services, in conjunction with a compassionate attitude from the veterinarian and staff, mean so much in our client's eyes that most of them will never want to leave your practice.

So, you are probably catching onto a trend here with all of the previous tips. These are great tips to help any veterinarian increase his or her gross production (the numbers speak for themselves) and loyal clientele. If you just remember that practicing great medicine and possessing good communication skills equals good income production, you will be a successful veterinarian. Once again, it is as simple as practicing great medicine.
To be a great veterinarian and to offer the best medicine possible, it all boils down to YOU......Not Really!  You may be a great practitioner and practice great quality medicine, and you may own a beautiful, fancy practice, but, if you don’t know how to treat and motivate your staff, what’s the point?  You will never be successful unless your entire team is successful.  It is very important to teach your staff all the technical aspects of the job and how to communicate with clients, but these are just tiny little bits and pieces of what brings success in a practice.  In order to rock n’ roll with our staff, we must get to know them and learn what makes them tick.

Be a great CEO
We would all love to be the CEO of our own company, right?  But what does this truly mean?  To me, CEO stands for “Chief Energy Officer.”  In the book, The Energy Bus, Jon Gordon describes how our emotional energy defines our own lives as well as those of us who work around us.  If you bring dull, lifeless energy to work everyday, that is exactly what your company will be...... dull and lifeless.  If you bring excited and enthusiastic energy to work, then your company and those around you will most likely share the same outlook towards their daily jobs and tasks.  Your energy sets the tone, so make it a good one.

Define the roles for your staff
We all want to have a purpose in our life and in our jobs.  It is easy to call each staff member either a technician or a receptionist, but that is SO BORING!!  We need to be on the lookout to see which of our employees are leaders and which are followers.  Who are the ones that are always stepping in to lend a helping hand, the ones that are always bringing new ideas to the table, and the ones that learn a task and then teach it?  These are our leaders and we need to reward them.  These priceless employees need to be given special roles such as head technician and head receptionist.  They need to be given some authority over the other staff members.  This allows them to know that their hard work has not gone unnoticed.  This also takes a huge weight off of the veterinarians.

Social butterfly
In today’s world, everything is about technology.  Our businesses are all over the internet and social media sites and there are often times numerous client reviews that, in a way, “define” our practices.  Social media can be great for your practice but you have to keep up with it.  When it comes to our staff, we usually have rules against cell phone use and computer use during work.  However, some of our staff members are so attached to their phones and social media sites that this can become very tough to monitor.  Instead of reprimanding this staff member, you should place them in charge of managing your clinic’s social media presence.  They are already good at it and, by doing this, they will receive more satisfaction out of their job and most likely become a more enthusiastic employee.

Find a common Ground
One way that I Rock n’ Roll with my staff is by, literally, rocking n’ rolling with them!  We constantly play music in our clinics.  Music has been proven to provide a more calm and soothing working environment.  It also allows for employees to sing along and, on many occasions, forget where they are and begin dancing in very embarrassing ways.  This constantly provides for a comical work environment for all of our staff.  Our staff constantly remains focused, but we also enjoy a fun and mostly stress free work environment.  Certain types of music have also been proven to lessen the anxiety level in pets while in the hospital.

Make it personal
We are always looking for ways to celebrate in our clinics.  Whether it be a birthday, engagement, the birth of a child, or a staff promotion, we always make it a personal celebration for that particular staff member.  It does not cost a lot of money to show that you care about the lives of your staff.  It can be as simple as providing a cake or a small gift certificate.  The lasting effect of doing this for your staff far outweighs the cost.

What’s the incentive?
We all offer special events in our practices such as dental month, senior wellness month, and so on.  During these events, we tend to be a little bit busier and our staff must work that much harder to make them a success.  Once these events are over, how do we show appreciation to our staff for all the hard work, or do we?  Of course we could all say, “wow, we had some great success during this promotion and we would just like to say thank you for the hard work,” but is this really enough.  I am a firm believer that a true “thank you” goes a long way but there are many ways to show our appreciation even more.  The best way to do this is to offer incentives.  During dental health month, you could set different goals to achieve and offer incentives once each level is passed.  If you staff performs 50 dentals, then you could provide lunch or a gift card and if they perform 100 dentals, you could take them all out
to eat. There are so many ways to do this and it just makes your staff even more enthusiastic about the event or promotion that is being offered.

**Make time for and listen to your employees**

You may be in the clinic all day with your employees but are you actually spending quality time with them and listening to them? Many of us require our staff to obtain a history and vital information from our patients prior to our exams but do we really do a great job of providing quality listening time to all of their hard work. This is so easy to do but is also hard to do at times, especially if we are busy with multiple rooms. However, if we are going to ask our staff to work hard at communicating with the clients and performing a brief exam on the pet, we need to make sure to provide a listening ear to all their hard work. We also need to make time for them and experience how successful they are becoming in our practices. It is not enough to just be present in the building, we need to provide quality interaction with our staff throughout the day.

Yes you are a veterinarian and you may even be the owner or the one in charge, but you must realize, you do not always know what is best. Your team interacts with the clients and pets just as much as, if not more than, you do. A great staff always listens to clients and they look for ways to improve a client or pet’s experience in the clinic. They also listen to what the clients like about our clinics and what originally brought them there. By doing this, our staff are able to come up with unique ways to reach to existing and potential clients. These ideas often become some of the most successful practice builders in our clinics.

**Servant leadership**

Our staff are not our servants, they are our team. We need to serve them more than they serve us. We need to help each staff member discover his personal strengths and then provide opportunities for them to implement these strengths for the better of the practice. We need to help our staff do what they do best, but first, we must figure out what that is.

**Sometimes you just have to let go**

No matter how hard you try, you will never be able to please everyone. We are all going to experience times in our practices during which we have a “toxic” employee. These employees will ruin your practice and staff morale faster than you could ever imagine, and the majority of the time it is not on purpose. These “toxic” employees become this way because they were never a great fit for your team. It may hurt to let someone go but it is better for you, your team, and for the “toxic” employee.

There are many ways to rock n’ roll with your team and make sure that you are all on the path to practice success. First off, you must remember that you, as the veterinarian, set the energy of your staff and if this energy is constantly one of enthusiasm and success, your team will thrive. We need to treat our teams like family and celebrate all successes, no matter how great or small. Let’s all get ready to Rock n’ Roll!!
We have potential clients hunting for us everyday. They probably are not dressing up in camouflage and loading up their rifles, but they do go to internet sources and “word of mouth” sources. These potential clients use their five senses to decide where to take their beloved pet for the best care. So get ready, these pet owners have you in their crosshairs and are using the following senses to decide if your practice is worth the shot:

Sight
This day in age, the first source these clients will use is the internet. It has never better served a business more to have a website than it does today. However, just because you have a website, does not mean it is going to bring in the business. When searching for a veterinarian, a potential client wants to get the quick overview. Our websites must be informative but also clean and professional. It is great to have a clinic tour or pictures of your patients on your site, but not on the home page. When a person visits your site, there are just a few very important pieces of information for which they are looking. The primary aspects of the site that need to be seen are the clinic’s logo, the contact information and address, and your business hours. Secondly, they will be looking for an “About” page that will discuss our practice philosophy, our doctors (pictures and bios), and possibly a clinic tour and client reviews.

Upon winning over the potential client with our website or by client “word of mouth,” the next step is the “meet and greet” as I like to call it. The client and pet(s) will come to visit. The first eye-catcher should be a professional appearance, which should include a clean facility, nicely dressed and well-groomed staff and doctors, and educational exam rooms. By this I mean educational posters and models in the exam rooms or, at best, interactive screens to keep the clients entertained and to help them learn more concerning the health of their pet.

Hearing
Those exam room doors are wonderful to have while preparing for the patient on the other side. You and your team can get everything together without ever being spotted by the patient or client; however, what many people do not realize is that the sense of “hearing” is at its strongest during this part of the clinic visit. This sense is 100% alert for the client and the patient. As far as the clients go, they can hear everything the staff may be discussing whether it pertains to the client’s pet or to random subjects. Many staff members do not think before they talk when it comes to this scenario. We must strive to keep our staff aware of this and to keep a positive attitude at all times in the clinic. Of course there will be days when we have team members that are “in the dumps,” and when this occurs, we must have a plan set in place. I recommend having an “in the dumps” protocol. What I mean by this is having a plan of action or an escape route for these particular situations. This escape route should include a “quiet place” for these team members to escape to, whether it is an office, a quiet break room, or a quiet, outdoor sitting area. This will also help to keep the team morale at its highest.

As far as the pet goes, each and every one is different. We see some pets that are as happy as they can be at our practice, however, many of our patients are shaking in their fur boots. The sense of hearing is off the charts during this time. Every drawer that is slammed, every bark and purr that occurs, and every word that is uttered, sends these pets into a frenzy. We all need to strive to make every effort in keeping these pets calm. We have 2 options by which we can do this. We could install all padded, sound proof walls in our exam rooms, or we could just always keep the mentality of putting ourselves in their furry boots to serve as a reminder of the pets feelings at the time. I think the latter option would be the easiest and most economical.

Touch
How does your clinic touch its clients and patients, especially first time visitors? Upon entering, do they receive a warm welcome or a bitter hello? It all starts with our receptionists. Every receptionist needs to introduce themselves by name and, if at all possible, welcome the client and pet by name. At the same time, if an appointment has been scheduled, the receptionist needs to reiterate the reason for the visit or the client’s concern. We also need to take an up close and personal visit to our own waiting rooms at least twice a year. During this time, we need to sit in the chairs and feel how comfortable they are. We need to look around at the scenery and pictures/ads that may be visible, and we need to scan the floors and walls for any surprise stains that may have been left behind. I also recommend having an area where a client can receive a cold bottle of water and maybe even a snack.

The next step for the client is the exam room. What is the overall feeling in the exam room? Does it provide comfortable seating and plenty of room for the patient and client to interact with us? We also need to strive to have educational exam rooms. Upon completion of the exam, we need to have a knowledgeable technician discuss all aspects of the exam and make sure the client fully understands any treatments that are given and that he or she leaves with no unanswered questions. Upon check out, the receptionist
needs to go over an itemized list of services rendered and medications given and then ask if the client has any questions before checking out. Having said all of this, in my opinion, one of the best ways to satisfy the sense of touch, is to offer a clinic tour. This allows the client to get an inside look at where and how their pet will be treated.

**Smell**

Of course we are always trying to make sure our clinics have a good smelling environment, however, after working in the clinic for so long, we can sometimes become immune to smells and they may no longer offend us, but they may blow up the noses of our clients. Nothing is more offensive to a client or visitor than a malodorous clinic area. Also, I guarantee if someone asks a client about your practice, he or she may say you are the best veterinarian in the world, but the subject of the bad smell will definitely surface. This factor will drive more clients away than you could ever imagine. On a more personal note, when I was interviewing for my first position out of veterinary school, one of the first aspects of the clinic that I noticed was the smell when I walked in the door. If it was a malodorous clinic, it did not matter how the interview went from there, I was guaranteed to turn any offer down. So believe me, when it comes to this area of concern, this doctor NOSE what he is talking about.

**Taste**

You are currently saying to yourself, how in the world can a client taste my practice. Well, figuratively speaking, they can. The sense of taste refers to our clients leaving with either a good taste or a bad taste in their mouths. In order to successfully pass this sense of clinic hunting, you must combine all of the other four senses together. If we successfully meet the senses of sight, hearing, touch, and smelling, I guarantee your clients will always leave with a good taste in their mouths, figuratively speaking.

So, how does your clinic rate according to the 5 senses of clinic hunting? I recommend you write the 5 senses down somewhere that can be seen by all of your team members and have each of them give opinions in each category. If you realize that your clinic passes all 5 senses with flying colors, then I guarantee that every new client will keep you in their crosshairs and never hunt elsewhere again. If we strive for excellence when it comes to the five senses, we will have more clients than we can handle in no time.
5 Things You Must Ask Before Your Boss Retires
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Life is inevitable. We all grow older and we retire. We know the day will come that we will retire, but it is more of a concept than a reality. But many veterinary professional work for practice owners who are closer to retirement than they are. If you have a boss that is older than you, there are things you need to know before his/her last day!

1. What does the practice owner have in mind for the practice?
Odds are there is no official plan in place. Your boss may have just seen his/her exit from the practice as an ambiguous idea that is now looming as a reality. Very few veterinarians that own a practice already have a plan in place. At the very minimum, it is a great idea to have an estate plan to allow the practice to continue in the event of the owner’s death.

It is reasonable and important for an associate vet or practice manager to find out this type of information. Ask if you can plan a regular meeting to discuss planning and health of the practice, just like any relationship, this one requires communication.

It is wise to discuss if the owner’s departure and plan for it if possible. According to David McCormick, MS, CVA, most practice sales take 3-4 months to complete once terms are agreed to. Many times the staff will not be aware of the transfer until the final days.

No matter what you decide to do, you will need to know a rough idea of what the owner plans so that you can plan for yourself. Departures can be sudden, slow transitions, or even have the previous owner stay on as an associate. Sometimes departures are due to death or injury and if the conversation has been opened, everyone at least has a starting point to try to honor the owner’s wishes.

2. Do I want to be a practice owner?
Think about your own personality and situation. Practice owners shoulder a lot of responsibility above and beyond those associated with being a vet or practice manager.

An owner is a veterinarian, an HR person, an accountant, a marketer and more. He/she decides wages and staff and ultimately, all profitability (or lack thereof) can be attributed to the owner’s choices. Owning a business is an excellent investment since the owner gains not only his/her salary, but also builds equity in the practice, increasing net worth.

Business leaders possess certain traits, according to Forbes. Consider each of these honestly and make sure possess or are willing to grow these traits.

- Passion- a strong sense of purpose and the idea that you are on a mission for a reason
- Resilience- Bad things will happen, but good leaders can shake them off and never stray from the purpose
- Strong Sense of Self- successful business owners have the confidence that if they work hard, their mission will succeed.
- Flexibility- Being able to make changes on the fly and come up with ideas that are “out of the box” is a very valuable trait for practice owners.
- Vision- Successful business leaders see opportunity everywhere.

I would add persistence to this list. The ability to try innovative ideas (and know how to monitor and test the success or failure abandoning them when they fail or doubling effort when they succeed) over and over and never stopping is a trait that is essential to a successful practice owner.

3. How do I figure out if I want to buy this practice?
Perhaps the owner thinks that his/her practice is worth more than it truly is. Most owners do. The old adage goes that something is worth only what you can sell it for. The practice you are considering might not be the best investment for you. There are professionals that can help you know, taking into account growth and cash flow. If words like “growth, revenue and cash flow” make you feel sick, you might want to reconsider practice ownership.

Once you have decided that the practice is something that you want to consider for purchase, you should look into financing. This should not be frightening for you because a solid practice pays for itself and allows you as the owner to continue to have income and grow equity.

Financing is easier than you might think. If you are a veterinarian with at least 3 years experience and good credit, you will need only a relatively small down payment to finance a practice purchase. Again, a good practice pays for itself.

4. What will happen to me if I do not buy this practice?
If you are not interested in practice ownership, you should understand that buyers are mostly interested in maintaining the current staff as much as possible. They hope for a seamless transition. On the day that the loan closes, all contracts are terminated and must be renegotiated. You will have a new boss and eventually, a new practice culture and structure. New is not always bad (but is not always good either) and the new owner will be interested in making the investment work.
Many large practices are purchased by corporations. Again, the new owners will be interested in keeping the staff and veterinarians that are currently working in the practice. They have defined it as successful enough to purchase and you are a part of the success.

Some practice owners decide to close their practice. In this case, the practice was likely not profitable anyway or the present owner would want to cash out on the investment. If your owner plans to shut down at his/her retirement, this is something that you surely want to know.

5. What is stopping me from acting, either to pursue purchase or to solidify my future with the practice in another way?
Fear- If you want to buy a practice, seek information. You will find that there are more resources and opportunities out there than you think. A profitable practice is a good investment for someone who wants to be an owner. If not your current practice, then there is another. Embrace your vision and your persistence. You can find a way to make it happen for you. Do not be afraid of the unknown and remember all the resources available to help you.

Ignorance- There is a difference between ignorance and stupidity. Ignorance is a lack of awareness and stupidity is lack of intelligence. Make yourself aware. Explore your own traits and your goals for the future. You never have to fly blind with all the resources out there.

Don’t be afraid to ask questions. You have every right to know. If your answers reveal to you that you might excel at practice ownership, take advantage of the resources provided here. No one wants to see colleagues fail and odds are the owner wants to feel good riding off into the sunset knowing that the practice is in good hands and everyone is happy.
Finding New Clients isn’t Match.com, but You Still Want a Good Fit
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As a veterinary professional, you may not be looking for Mr. or Miss Right, but you are looking for Rights-Right clients. Just like every person that you ever dated, not all clients are right for your practice. Relationships take work and even some planning. Dating apps and websites have capitalized on compatibility. Guess what? You need compatibility too!

Incompatible people are not a good match. You don’t want clients that are destined to be unhappy with you. You want clients whose basic ideas align with yours so you can find happily ever after.

So start with your own questionnaire but for yourself
What do you want?
Some people who are dating aren’t really interested in a long-term, committed relationship. Some people truly seek their soul mate and have specific goals for the relationship. Neither of these people is wrong. But if you tried to match a “player” with someone seeking marriage, it would be a recipe for disaster. They see the future in different ways and their life goals are different. That match would end in a parting and it might even get ugly. An ill fated match like that for a veterinary facility could end ugly too, with bad reviews and bad word of mouth.

It’s important that you understand yourself and know which kind of relationship you seek before you ever fill out that online dating survey or go on a date. Expecting a good outcome from a poorly matched couple is like expecting oil and water to blend.

What kind of practice do you have—or what kind of practice do you want to have—and how do you tailor your search for the clients that are right for you?

Wham bam, thank you ma’am
Some veterinary practices aren’t seeking committed clients. Their goal is to see the maximum number of patients in a given time and perform the minimum care. These practices provide a valuable service to owners matched to that model. Many pet owners want the minimum care that will meet state laws and that will cover the most basic preventable diseases and parasites. These practices cover their overhead with sheer volume. This is a valid business model, especially if you’re the type of person who thrives in a fast-paced, “low touch” kind of workplace.

The staff and veterinarians at these practices focus less on making clients feel “warm and fuzzy” because they know the attraction for the relationship was based on the “basic needs only” plan. Doctors, managers and team members can get a warm and fuzzy feeling themselves because they know they’re helping a large number of animals get crucial care that they would have missed. In this type of practice, it must very clear that it is basic minimum care, so that owners do not misconstrue what they received and expect unreasonable outcomes. Communication is your best friend (again) in managing expectations.

As veterinary students and staff members, we’ve been told to think we must provide state-of-the-art care to every client and patient no matter what. But if the entire profession follows this practice culture (and charges appropriately for it), there is an entire segment of the population of pet owners that will be underserved and some pets will get no care at all. There is a place in this world for minimum, quick care. Just be sure that the owners know that minimum quick care is what they are getting.

Put a ring on it?
Practices that strive for a committed relationship for the life of a pet are the ones whose focus is on building a strong healthcare partnership with their pet owners. These doctors and team members want to ensure a long and healthy life for the pets and a happy family with a strong human-animal bond.

This type of practice is harder logistically, requiring longer appointment times and highly trained and highly motivated staff members who are aligned with these big goals for pets and clients. Time is needed with each client to address every facet of pet healthcare. These are the practices making extra effort to minimize patient stress and are more likely to need a large, well-appointed facility. Not every client wants or is willing to pay for this level of care. This type of practice covers its overhead by charging more per transaction because the overhead is high. Again, you must communicate with pet owners at every opportunity to make sure that you are on the same page with your expectations. A treatment plan should be presented prior to treatments and staff must be trained to explain every item with pros and cons.

A passing fancy
Some people in the dating scene have very specific quirks or visions for what they want in a relationship above all other factors. Maybe they like only brunettes or restrict their candidates to certain religious groups or political parties. These clients might prefer you if you fit the bill for their unique needs, maybe a cat only clinic or a specialty clinic. If your “getting to know your practice” survey revealed that you have something that might be attractive to niche clients, promote it. Make sure the world knows that you fill the bill where this area of interest is. Do you love to treat reptiles? Advertise it. Are you a behavior specialty? Your dating pool must
include vets, trainers and other pet professionals as well as owners, but dating success may mean the pet responds to your treatment and is released.

**Settling down**

Many practices are a hybrid of the extremes above. Most of them have business models that work, and when properly managed, all can show profit. But just like dating, you have to ask yourself: What makes you happy? Does playing the field make you feel empty because what you really long for is a committed partnership? Is all the effort to build a relationship tedious because you just want to help lots of pets and then go home?

Think about who you are and who you want to be in your practice. Make the necessary changes to your mindset and that of your staff to reflect your practice culture. If yours is a “Playing the field” practice, make sure everyone knows it by advertising discounts and specials. People may not want to admit it, but they know deep down that they get what they pay for and they should not be expecting comprehensive care from a low-cost spay and neuter practice, for example.

If you’re a “Committed relationship” practice, highlight special, long-term pets and clients on your website. Make sure your staff is customer-focused. Practice and recommend the best medicine. Manage your schedule so that appointment times are never rushed. Spare no expense in making your facility look and smell perfect. Offer tours any time. Plan events and open houses. You’re looking for a long-term commitment from these clients—show them what you have to offer in every way. Lavish them with attention!

And don’t worry too much about which practice model you decide is right for you right now. As life happens, you might change your mind. The bottom line is, for your happiness and success, you need to know what type of practice you have and what you’re really looking for from the entire client interaction. With some thinking, some planning and some intention to find the right client partners for you, maybe you can fall in love with veterinary medicine all over again.
How to Grow- Not Fight- with Local Competitors
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Whether we like it or not, veterinary medicine is a business and business means competition. We are all in this wide world together and ethics dictate that we not succeed by virtue of the failure of others, but by our own merit. Knowledge is power and you can find your own way in this capitalist world by carving out your place and finding your niche.

Learn all you can. Not because you want to "one up", but because you want to establish your own niche or way to stand out. Gone are the days where one vet makes every client with every species from all walks of life and demographic happy. We are no longer alone in our markets in most part of the world. James Herriot's world is past and we can't be everything to all people and animals. It makes us wistful, but comes with advances in all things. Dr. Herriot was the only vet in his community. He had no competition and he saw everything in the community, all sorts of animals and people. But he worked grueling hours and traveled far. He didn't need to make his niche because his niche made him. Our world is different.

Who is your competition?
Define a radius around your practice that you feel most of your clients come from. From your software, print your top accounts for the previous fiscal year.

You can use your cell phone from your office. Select your top clients (because these are the ones that are happy with you and you want more like them) and Google map the distance from their address to your facility. How far does the farthest travel to see you? Print out a page and draw dots of your furthest drivers. Grab your protractor and starting at their address make a circle around them that surrounds them with the distance they drive all the way around their home. Are there other vets in these circles? If so, these are your competition. Sometimes people will drive further to see you if they love you, but in reality, things have to be convenient for modern consumers. If there are other vets within the circles you have made, they are your local competition for your best clients.

Now you know who they are. Get to know their practices. Make a list of those practices that are in the circles and sit down with your computer. Google search each one and find their websites. If they do not have a website, they have removed themselves from your competition group since many new clients come from internet searches and in today’s world, not having a website is a sign that they are behind in marketing. Make sure you have a website for your practice as well or you are the one behind!

Check each clinic’s website for their mission and focus. In today’s online world, there are no secrets. You can easily find out all you need to know about the competition with a click.

Try to meet the vets at the competing practices. They are friends and not enemies. You are all trying to make a living doing what you love and most vets are decent and hardworking. Developing a rapport can be helpful if you need to borrow items sometime or even discuss clients you have both seen. Attend local VMA meetings. These groups are designed to promote the entire profession and you will likely find the opinions and discussion of other veterinary staff is valuable to you. You can get to know what has worked in areas outside your coverage area too. Networking with other professionals is invaluable.

Species?
For example, if your competitor spends time treating exotic animals, do you want to? Depending on the population in your area, there may not be enough exotic pets to justify your expense in tailored products and pharmaceuticals for more than one practice. Initiate a conversation with him/her about the ROI he sees on these patients. If he has a specific desire to treat them, refer calls at your practice to him and discontinue your treatment of them. If you are the one who loves them, make your case for why he should send calls your way. If your competitor tells you he has more than he can see, perhaps you can redefine which species you each prioritize.

Services?
No one can be everything to every client. Does your neighbor offer all the services that you do? Is there overlap? One of my close neighbors does not offer boarding at all. I have an entire building for kennels. He is not interested in boarding at all and finds it a hassle. I adore having my boarding (for known clients only) as an added value service for my clients.

We are both happy with our offerings on this front.

Grooming?

Surgery?
Maybe your neighbor loves orthopedic surgery, but you do not. Could you work something out where he could do the procedure and return the pet to you? Remember, if your clients are happy with you, they will not stray to a competitor just because they spent time there.
Dentistry?
Do you have any equipment you could offer access to? We have digital dental radiology, but some of our neighboring practices do not. We have communicated that we have this technology on our front sign and occasionally my competitors will send a patient in for a dental radiology consult. I am happy to act as an imaging center in these cases and return the pet to their care with diagnostics in hand.

Hours?
My area is not typical. We have learned that our clients do not take advantage of our Saturday hours very often, so we tracked them for a year and based on that data decided to only offer Saturday hours one day per month by appt. The two geographically closest vet clinics have discovered things about their own clients which ironically are very different from mine. One offers only Sunday hours and is closed on Saturday. The other swears that he does more business on Saturday than any other day. So we all coexist. Occasionally my clients will see one of them on weekends, but they seem to always return to me and we all feel good about our hours. My clients have gotten used to my hours and I believe it is a quality of life issue for my staff and since most Saturday hours failed to cover overhead, it was an easy choice.

Culture?
You have probably heard mention of the practices around you. You should never take what disgruntled clients say as the whole truth, but it might give you a hint about their practice culture. Do clients complain about wait time or feeling hurried? Maybe they are a low touch, high volume practice (which could indicate you should be different) Many times the website description will give a hint to the mission and culture of a practice. For example, if the text says, "We make extra time at each appointment to be sure that all your questions are answered." Then you may surmise that (at least in theory), the practice wants to be higher touch and lower quantity. Sometimes the answer lies in what they don't say or even show. Remember, if a clinic does not have a web presence at all, you might think that they have not kept up with current trends.

Let’s examine some examples of web sites and see if we can surmise the practice culture.

Any special initiatives?
Is your neighbor promoting some initiative, like being Cat Friendly or is she trying to grow her dental practice? If so, it doesn't mean that you can't pursue similar initiatives, but you might highlight differently. Play to your strengths and let them play to theirs. Are their initiatives something that redefines the standard of care for your area? Things like Fear Free and your lack of participation could make you look like you do not care about fear. Seeing how hard your neighbor promotes their enterprises can help you decide how you will handle yours.

What about products?
Some of my local competitors offer products that I have elected not to. Some clinics have no inventory and some find that they move a lot of products. In the world of PetMeds, you have to decide for yourself what you will carry and how much. Find out from your competition what they offer. Do they offer an online store? It should be obvious and readily available through their website, so you can find out.

Reputation
Read the reviews for their practice and your own. Can you make sure that you are meeting the needs of those that are not satisfied with them? Find themes among the reviews and make sure that your practice does not have similar complaints. Don’t forget that not all reviews are honest and some are disgruntled through no fault of the clinic, but you can learn from the review and learn. See if they respond to their reviews. This is something that should be happening. If they are not, be sure that you are. Be kind and non reactive. Try to direct the complainer to contact you personally for a resolution.

Do your homework
Maybe communication and cooperation could benefit you both. At the very least, knowing what you are up against will benefit you in the long run and help you be the best you can be. Even if your competition is formidable, no one can be everything to every client. You can find ways to balance offerings and fill gaps so that you can both survive and thrive in your local jungle.
You cannot manage what you do not measure
We’ve all heard the adage, but how many of us actually practice the principles of tracking and managing the numbers that should drive our efforts. My experience says not many. This session will explore how successful practices leverage the digital metrics available to manage their online marketing efforts.

Set reasonable expectations
Being reasonable about your goals and expectations could, in fact, be one of the most important aspects of setting up a digital marketing program. All too often practices set upon a marketing journey with lofty expectations that are impossible to track or achieve. Practice employees jump on the program band wagon but are left feeling as if they failed when the program does not meet the expectations that were set and eventually the practice abandons the marketing medium because it “does not work.” Try communicating with industry thought leaders about their experience in the space, reach out to peers to determine their experience, and perhaps begin a very small program with the goal of actually learning what your practice can expect from the medium. Then, with the experience in hand, future program development can be planned as you are familiar with the results and can develop reasonable (responsible) expectations.

Understand what the goal is before you develop any marketing medium
Perhaps the first step in developing the “goal” for any marketing medium is to understand a bit about who you are. Because many of your goals will (or should be) financial based, it would be helpful if you had a good understanding of your practice profit margins. It would be great if you had the time and resources to dive deep into your financials and your marketing efforts to try to track the impact that every marketing dollar has on each service’s bottom line, but that is probably not reasonable (see above). Basic information about your current gross revenue, profitability, target profitability, and minimum profit margins would be very important here as these numbers become the starting point and ultimately the foundation for a well-managed marketing plan.

How would it be possible for a practice to set a goal of increasing the profitability of a service, raising weekly gross revenue numbers, or development of a special discount/loyalty program (although I deeply despise discounting) without understanding the actual financials. If you do not know these numbers, do not be afraid, you are not alone. I frequently find myself in conversations with veterinary professionals that are in the same space. Ask your accountant for some help here.

Great goal examples are:
- Increase the profitability of a service
- Increase the frequency you are performing a service
- Increase the number of new clients
- Decrease the number of clients who lapse more than 13 months
- Increase the frequency of client visits
- Decrease the number of “open” appointment slots
- Increase the average client transaction
- Increase monthly gross revenue

What is analytics?
Analytics are bits of information that tell the story of how your digital marketing efforts are actually reaching their intended target. When used properly, they are one of the driving forces behind the success of the digital space as now we can track the performance of our efforts much more efficiently. Back in the “old days” we had to rely upon surveys and general data to demonstrate how well our marketing or advertising efforts were performing. Those days, for the most part, are gone.

The most common form of analytics are called “Google Analytics”. This is a free service available through Google, installed within the code of your website, and is used to monitor/report information about your web traffic. In most cases, your practice will need to reach out to your web development folks and have them assist with the installation of analytics. Most website projects should already have analytics installed, in fact, if you do not…you should be concerned as analytics provide the important information necessary to determine the success of the project.
A word of caution
While the code for tracking and ultimately the information developed through the analytics code is free, you should expect that a web marketing firm will charge you (or build into your monthly service fee) for a professional breakdown of your website analytics. Look at analytics much like any blood test you would run on a patient. The data from the test means much less without your professional opinion on how to proceed with that information in hand.

Examples of content that comes in analytics
Over the years I have seen thousands of reports that have been developed using Google analytics information. While a practice can get lost in the data and “push back” because of head spins, it will help if you remember the goals that were set at the beginning of the program. Analytics allows you to look at how your website performs, how users interact with the site, and ultimately how well the website “converts”.

Conversion is the act of causing a web user to perform the desired task. Good examples of conversions are requesting an appointment, clicking to call the practice from a mobile device, filling out a form, watching a video, or submitting their contact information. So, if your marketing goals were to increase the number of calls to the practice through the website, you could determine the baseline for calls after a month of performance, then monitor that metric over time. Adjustments could be made regarding the call to action, color of the “call” button, positioning of the “call” button, meta description used to describe the web page in the search engine results, and overall page layout so that optimum results are achieved.

During the lecture we will take a much deeper look at an analytics report and will explain what many of the data points mean, but to give a sneak peek at what analytics can deliver, you should know that you can determine how many people visit your site and leave immediately, how many people browse many pages of your site, where they leave, how long they stay on your site, how many people visit via desktop, how many visit on their mobile devices (even phone model numbers are available), which pages receive the most (and least) traffic, how many people click on a particular button, which keywords are most beneficial, and much more information than the format of these notes will allow.

Value of automating reminders
Thus far we have only discussed the analytic information that can be developed through Google in relation to your practice website. Please understand that additional information is available through other services or products used in the course of your practice operation.

Take, for instance, automated reminder services. Many successful practices recognize the value of automatically extracting information from their practice management software and sending text, email, or even postcard reminders for upcoming appointments, services, and those that are overdue. But few reminder services and/or practices take the information to the “next level” by sorting the clients by the average amount spent on the pet to be reminded, tracking revenue generated by reminded clients on a weekly basis, understanding the impact a missed appointment has on your bottom line, realizing how many pets are overdue and the amount of dollars that are potentially “missing” from your revenue, the dollars gained by reactivating lapsed clients by connecting the reminder with real revenue for that particular client, and much more!

We will take a deeper dive into this topic during the talk, but the key take away here is to look for opportunities for your practice to utilize the services and processes you already have in place to marry your marketing and communications efforts with actual invoices.

How to use data from analytical data
As I mentioned earlier, simply having the data is not enough. At very least you must understand what each of the metrics mean and at best, you have identified the metrics that are most beneficial to the growth of your practice and are using those metrics to guide you through future marketing efforts. With this information in hand you can expand your efforts into new and perhaps more beneficial mediums.

For example, through your website analytics you might find that most of your “organic” website traffic comes from a certain group of key words or phrases. This information can be used to develop a more robust pay-per-click campaign which will provide more data for you to track and segment. By looking deeper at the pages which get the most traffic, you might see who gave their information to you on that page. Then, by comparing and tracking reminders and client reactivation revenue, you can begin to assign values to the actual keywords and phrases used to promote your practice.

The bottom line here is that the digital space provides a huge amount of data that, if collected and understood, can provide practices like yours an extremely valuable tool for practice growth!
When the Internet was first developed it was wide open space much like the wild west. In those days when a website made claims about being the best or the most convenient business, the search engines often responded by positioning that website high when web users requested that information. As the web became more congested and competing websites made similar claims, the search engines needed to develop algorithms that sorted authentic content from that which was less than genuine.

I like to describe this situation to be very similar to a piece of electrical conduit. When empty, it is very easy for an electrician to push a piece of wire through the metal pipe. As more wires are introduced to the pipe it becomes incrementally more difficult to introduce additional wires. As a result, wire lubrication, additional force, and/or alternative conduit needs to be installed in order to deliver the wire. Do you see the connection? Often, special effort needs to be paid towards getting your practice’s message on top of other practices that are ranking organically.

When you combine this crowded factor with the fact that the search engines need to monetize their efforts through paid advertisements, it should be quite easy for you to realize how paid search was born. Over the years web users have grown accustomed to seeing a combination of organic and paid search results when using search engines like Google, Yahoo, and Bing.

As social media became more popular, those channels found themselves in exactly the same position as they are mainly free services that need to monetize and have particular usefulness for businesses like your veterinary practice. Social channels also have a unique interest in limiting the amount of advertisements that a personal user will be exposed to at any given time so as to limit the possibility that the user will abandon the channel because of very frequent advertisements.

From the very beginning of social channels like Facebook, YouTube, and Twitter, users have been sharing information about their personal habits, likes, dislikes, locations, age, gender, relationship status, profession, and many other aspects of their personal lives. This information has been tracked and stored as a part of each user’s profile. Advertisers benefit by being able to properly target those and channel users who best fit their target market.

This situation presents a very unique opportunity because ads can be presented to those social media users without the user searching for keywords or phrases that traditionally drive organic and paid search within the search engines. It is a true “push” environment that serves up relevant information to users who fit the advertiser’s demographic. Social channels have been known to only serve ads that get the most traffic and ultimately convert well for the advertiser. This prevents advertisers from blanket marketing across the network and flooding the space with irritating ads.

In order to be effective in social advertising, you must realize that those who are exposed to your ads are probably not in the mindset of your product while they are browsing their social channels. For that reason, your ad needs to be “disruptive” and hard-hitting. By disruptive I mean that it needs to stop the social user in his or her tracks and immediately shift their attention from being a casual social user to one who is now interested in your practice’s offering. During the lecture we will take a deeper dive into the actual creation of disruptive ads and provide some understanding of exactly what it means to stop hey social user and cause them to change direction towards your practice.

Before you embark on any paid search plan through the search engines or social media, it is extremely important that you conduct great keyword research. Many successful practices engage the services of SEO firms at this point. Professional analysts are capable of conducting research into your practice’s market and determining which keywords and phrases are most valuable. With this information in hand you are more prepared to create copy that will effectively engage social users.

High-quality imagery, videos, and motion overall have all been known to be extremely important when developing effective social media advertising. Take a moment and scroll through your favorite social media channel and notice the advertisements that cause you to stop what you're doing and pay attention to their message. More than likely they are advertisements from firms that are within your field of interest and provide a different view or look than the typical post within your feed. This is disruption. My experience is that you have less than one second to stop a user from passing your ad. During the lecture we will take a look at several good and bad social media ads so that you are most comfortable with effective techniques.

After you have created your ad and have launched it on your social channel, it is important that you understand the value of properly receiving someone who clicks the ad. I am referring to the specific treatment of the landing page designed to receive this user. It is extremely important that the landing page created is customized for this particular user and immediately addresses the subject matter at hand.

Imagine a situation where you are shopping for a television. Through a Google search you are directed to a television manufacturer’s website. In the first scenario you are directed to the television manufacturer’s homepage and then exposed to their entire brand. In the second scenario you are directed to the section of their website that deals exclusively with televisions. On this
page you are allowed to select which features apply to your current need. I ask you…which scenario is the most useful? I suggest the latter is more beneficial as you will spend less time searching for the information you need and more time actually drilling down into the feature set that this manufacturer offers.

Your website and practice is no different. When utilizing social pay per click advertising, you must create dedicated and custom landing pages that are focused on the subject matter of the ad and provide information for the web user that directly addresses the problem and remedy demonstrated in the social ad. During the lecture we will take a close look at the journey a social media user takes and demonstrate the different feeling created by a custom landing page as opposed to directing a social user to your practice’s homepage.

Advanced pay per click users understand the benefit of retargeting their website visitors so that they have additional chances to have impact upon them. This strategy involves identifying the people who have visited your website, noting the reason they visited, documenting their actions while they were on your page, and creating additional ads that are custom developed around those facts. This technique is known as retargeting and can benefit practices by circling back and re-engaging with prospective clients who have not converted during their initial visit. This is a true second, third, or fourth chance at influencing the prospective client with enticing ads well placed within social channels and internet search.

I am certain that you have been exposed to retargeting yourself. Just the other day I was shopping for a tent for my daughter’s birthday present. I did not make a purchase the first time I was on the sporting goods store website however I spent a considerable amount of time reviewing the features of one particular tent. In the days that followed that experience I was served up advertisements offering additional incentives if I clicked that ad and continued my journey down that website’s consumer funnel.

The ad contained a picture of the tent I was looking for, a list of the main features, and the call to action that included a 5% discount if I utilized that link at that time. Well, the end of the story is that my daughter now owns a tent.

During the lecture we will examine how retargeting works, discuss some best practices for utilizing retargeting in a veterinary practice’s plan, and allow you to set up a plan for successful retargeting your previous website visitors.
Most successful business owners understand the value of creating great policies for they become a guiding light that allow the business, its employees, and even its customers to operate efficiently under known and predictable circumstances. If done correctly these policies can help form procedures and ultimately the flavor and brand that pet owners become accustomed to when relating to your practice. With the rise and huge popularity of social media, I am forever amazed by practices who fail to realize and implement effective policies and procedures for social media behaviors.

Please understand that I am not an attorney, nor should any portion of this content be taken as legal advice. Instead, utilize this information as inspiration towards creating social media policies and procedures that best fit your practice climate. I strongly suggest that you involve your legal counsel and/or human resources advisors when creating your social media plan as they will have specific understanding of your local laws as well as previously developed Human Resources policies that are already underway at your practice.

The purpose of these proceedings and lecture are to facilitate a conversation about social media policy, highlight some situations that should be addressed or considered within your practice, and ultimately provide you with the understanding of how important it is to have a social media policies and procedures handbook within your practice.

Anytime I discuss creating policies or procedures for my business I feel that it's necessary to mention how important it is to utilize metrics that are easy to track, contain reasonable goals, and are ultimately enforced on a unilateral basis. For example, creating a policy whereby employees need to respond to all social media posts within 30 minutes of the post being made may be unreasonable and unenforceable. Your legal counsel or human resources advisor can help you craft language around effectively addressing time frames for social media response.

When speaking of enforcing the policy and applying consequences towards failing to follow the established policy, you must be careful utilizing language that you are not willing to enforce in all instances. Meaning, if you say that an action will result in an employee's termination, you must be willing to enforce the policy with your most valuable staff member as well as the person who makes the same mistake but is much less valuable to your practice's success. While this is very basic human resources guidebook creation advice, I felt it was necessary to mention considering my recent exposure to several social media policies and procedures manuals that were created at the practice level. It appears as though many practices have allowed social media managers without proper human resources and policy creation experience/training to develop social media policies that are applied inappropriately to the practice environment. This is obviously a very serious situation that should be guided by conversations and documents like this but ultimately developed and approved by professional human resources and legal advisors.

My experience within this space and lecturing on this topic has allowed me to understand the value of group discussion. For that reason, I have created a list of items that many practices should consider when developing a social media policies and procedures manual. You will be in a lecture hall with many of your peers. I will facilitate a conversation that will include experience based dialogue involving audience members, utilizing my experience running a small business within the veterinary space, and working with many practices across the country. This approach will highlight situations that other practices have experienced and allow you to create a series of questions or "what if" scenarios that can be organized and properly presented to your legal and human resources advisors. In doing so, you may experience great savings by being efficient with time and effort attempting to imagine obvious and less than obvious scenarios with an attorney who charges by the hour.

While I plan on addressing all of the below questions during the lecture, it should be understood that this guided, yet free flowing conversation may lead the conversation along the path of the room’s most important issues. I will guide the conversation through the experience and assure that many needs are addressed and met as possible.

Topics for our lecture conversation

- Will you allow your employees and practice owners to be “friends” on social media?
- How will you handle employees who want to befriend clients on social media?
- Who will post on your practice social pages? How often will they post?
- What content will be posted on your social pages? Where will it come from?
- What are the consequences for using content that the practice does not own?
- Who will approve the content? Will there be a process for review?
- How will your practice respond to positive and negative reviews left online?
- When will you seek legal advice?
- How will you handle extreme circumstances created by social media.
• Has anyone in the lecture been bullied online? How did you handle the situation?
• Is it possible to create a policy that prevents employees from commenting about your practice?
• Can you insist that an employee make their personal social profiles public or private?
You need SEO
Most practices understand that in order to be successful they need a solid marketing plan. However, very few practices “get” that this plan must include search engine optimization. Search engine optimization is the art of properly developing, maintaining, and positioning content and code on your practice's website and throughout the internet so that when pet owners are searching for services like yours, they are provided your website as a resource. This talk will walk you through the basics of search engine optimization and begin to discuss how to convert clients from those who have searched for services like yours, into longtime clients.

Custom designs allow you to stand out
First, it must be mentioned that your practice's website should be custom. This is extremely important as the average pet owner does not know the difference between good or bad veterinary medicine and we will be looking for your personality when browsing the internet. Besides why would you utilize a template that many other practices could use when developing your most valuable marketing tool? Also when speaking about your practice website and its development, you should pay great attention to make certain that your website passes the Google+ mobile friendly test. During the lecture we will discuss how to evaluate your website and how to run your side through google's tool. This is extremely important as Google has been very forward about the fact that websites that are not mobile friendly will not be considered for search.

Content is still king
With that said, we should now focus on the value of content. Since Google's spiders are only capable of reading code and content, it is extremely important that you spend time creating very high value content. By “high value” I am mean content that has been created to attract the search engines and is extremely inviting to those website users who visit your page. Care should be taken to develop a list of keywords and phrases that are most often used by those in your community searching for services like yours. Often times practices engage the services of SEO certified copywriters who are professionally trained to create the content that will best position there practice's website. During the lecture we will take a deeper dive into content by looking at a few websites from the perspective of a Google spider. This exercise will highlight the importance of keywords and the appropriate positioning of those keywords on a practice website.

Calls to action, value propositions, and conversion points
When developing your website content you should focus on creating great calls to action, value propositions, and trackable conversion points. A call to action is simply a statement that asks a web user to perform a desired action. Great examples of calls to action are call the practice now, book your appointment today, register for our newsletter, and learn more about dentistry. Value propositions are the give and take of the transaction. They are the “if you do this”, “we will give you that”. Examples of value propositions are "call the practice now, and receive VIP priority treatment" and "Book an appointment today and receive 10% off your next boarding stay". When created properly, these calls to action and value propositions are married with clickable and trackable buttons that become conversion points.

If, while laying out your website and the content which will populate your site, you take the time and ask yourself exactly what you would like the user to do on that page, you very well could determine your call to action, value proposition, and trackable conversion point. By properly installing google analytics on your website, you should be able to monitor the web user traffic and determine how effective your call to action, value proposition, and trackable conversion points are by reviewing reports that show how many people actually visited that page and ultimately clicked on that button. If done properly, this data should be the most valuable analytics and SEO report information. Too many practices spend time focusing only on keywords and keyword positioning. In the end, it is quite possible that you can have great keyword ranking without any conversions. This would mean that you have spent a tremendous amount of effort getting your website to be seen but once pet owners arrive, they are not performing the desired action. The bottom line here is that keyword rankings do not necessarily equal an increase in new pet owners or revenue.

During the lecture we will examine some great calls to action and effective placement of keywords and key phrases so they are attractive to both the search engines and pet owners. We will also take a deeper dive into the back side of a website so that you can return to your practice and determine if effective SEO techniques are being applied.
Sales funnels
Once you have attracted the attention of local pet owners it is important that your website is designed to route them through the journey of converting from someone who is interested in services like yours to becoming a client who will stay with you for many years. This process is most often referred to as a sales funnel. A sales funnel is wide at the top and narrow at the bottom much like a traditional kitchen funnel. The reason it is wide at the top is that the message being delivered at that level, or at the entry point of your website, is acceptable for all who would land on your site. As the pet owner’s journey throughout the site becomes more specific, the funnel narrows as the message is only appropriate to a select group of people. By the time the prospective customer reaches the bottom of the funnel, they are receiving extremely specific information that is geared towards asking them to convert. During the lecture we will take a look at a few well-developed funnels and discuss how a prospective customer may walk through the funnel and what information is most appropriate at the different stages.

It is impossible for me to deliver a paper or lecture about search engine optimization without discussing the two sides of positioning your website to be found by those who are utilizing search engines like Google. These two sides are "organic" and "paid" search.

Organic search
This type of search happens most naturally. It involves creating great content, developing effective website code, using effective design strategies, positioning well written content in valuable on-site and off-site places, and utilizing keyword strategies that attract users through the search engine results pages and retain those who ultimately land on your site. Organic search does not involve a payment to a search engine like Google but may require a payment to a professional search engine optimization firm that is experienced at managing projects like yours. This process can often be very cost-effective but will more than likely take longer for results to be obvious when compared to paid search.

Paid search
Paid search or pay per click, has a much more immediate effect. This strategy involves utilizing great keyword and key phrase research along with a keen understanding of the demographics of those who may be looking for services like yours. Paid search results appear in the search engine rankings at the top and bottom of the page. Practices that use this technique are most often interested in immediate search results and are very often competing with very well established websites that rank for the desired keywords.

A word of caution: paid search can be a very slippery slope as it is quite easy to mistakenly create ads in a fashion that results in the ad being displayed to and ultimately clicked on by those who are outside of your target market. I strongly suggest that if you are interested in a paid search campaign, that you seek the guidance or services of a professional pay per click agency.

During the session we will take a deeper dive and examine the most appropriate instances where organic or paid search can be applied.
People like stories
Recently, a trend has developed within the small business marketing world that has cast a light on a technique long used by very sophisticated brands. This technique, known as storytelling, appeals to a consumer's desire to be most connected with a brand or the people that operate within a business.

Why is this important?
In the veterinary world it is easy for us to understand that the average pet owner does not know the difference between good and bad veterinary medicine. People can most easily understand emotions and feelings created by familiar situations. As a result, my experience says that pet owners respond very well to emotional-based messages that are authentically created by the people and personalities that have created and support the veterinary practice.

Storytelling is an opportunity for practices to demonstrate who they are, the experience a current client or prospective client can expect when utilizing the services of your practice, and provides a very unique canvas for practices to relate a message and provide clearer understanding of a need for desired action.

Figure out which stories should be told
Before you start crafting your story, it is important that your practice understands what types of stories need to be told. To do this, I suggest you refer to your practices marketing plan. Identify the products, services, or themes that you would like to share with your current and prospective clients, and make a list of these instances. Then, conduct some research within your client and employee base by sending out surveys and having live conversations with those who are important to your practice about the things that are important to them. From this research you should develop a list of qualities and components of your practice that are most popular. Listen for common keywords like compassion, care, customer service, attention to detail, and other distinguishable and brand worthy attributes that should become the theme of many of your stories. Then, with this information in hand, you should call upon your life experience and the experiences of those who are close to you to frame up stories that are easy to understand and demonstrate the topics discovered during your research.

Be authentic
It should be noted that the term "stories" often suggests that the message may have a fairytale or fabricated component. While some businesses and professional storytellers have been successful utilizing a bit of elaboration or fiction in their stories in order to demonstrate a point, I suggest that your stories be as authentic as possible. By doing so, they will be easy for you to tell and will parallel the natural experience your clients have when interacting with you or your staff.

Allow your clients to experience you
We had a Schroeder family St. Patrick's Day tradition that involved our family dressing up in our finest Irish wear and making an annual visit to our local McDonald's for a Shamrock shake treat. My children looked forward to it as it often became a conversation that began months before St. Patrick's Day. Several years ago, my youngest daughter Lilly was in the third grade. On that particular St. Patrick's Day we, as usual, visited our local McDonald's for dessert. Immediately upon entering the restaurant Lilly noticed her third-grade teacher was sitting with her family in the corner doing the exact same thing our family had planned. As soon as Lilly recognized Mrs. Carr, her focus shifted from getting that Shamrock shake to wanting to interact with her teacher and her teacher's family. Reluctantly, I allowed Lilly to visit her teacher’s table but instructed her to return immediately to our table so that her teacher could enjoy some privacy. After Lilly did so, we began enjoying our family tradition but as our McDonald's visit continued I found myself watching Lilly and realized that she was no longer interested in the Shamrock shake but was, in fact, completely amazed by the fact that her teacher was at McDonald's.

Lilly’s amazement caused me to contemplate the situation for a great deal of time before I realized why Lilly was so amazed. It turns out that Lilly never was able to picture Mrs. Carr outside of the classroom. The fact is that when Lilly arrived at school each day Mrs. Carr was standing at the door waiting to greet her. Then at the end of the day, Mrs. Carr waved goodbye as Lilly boarded the bus. As far as Lilly was concerned, Mrs. Carr curled up in a ball and slept in the corner of the classroom. In Lilly’s mind Mrs. Carr did not have a life outside of the third-grade classroom. After our McDonald's encounter, Lilly began to look at Mrs. Carr as a "real" person.
I suggest to you that many of your clients are unable to realize that you have a life outside of your practice. You are Mrs. Carr. Storytelling can be an excellent way for you to demonstrate the fact that you have a life outside the practice. You are dealing with life issues just like your current and prospective clients. You are a pet owner and are often faced with the same decisions they are facing. By doing so, you will experience increased compliance, decreased missed appointments, a higher level of interaction, and an overall increased amount of respect paid to the time you share with them during your appointments.

**How you can use storytelling**
Storytelling does not have to be literal. I am not suggesting that you create an environment whereby your employees and clients are sitting cross legged at your feet hanging on your every word. But rather, I suggest you come up with easy to understand and transfer bits of information that help clients and employees understand the subject matter at hand. This technique can be used during your appointments, when creating copy for your website, during daily social media posts, writing blogs, and when developing videos that demonstrate your practice’s offering.

**Meet Kindra Hall**
As a part of my introduction to and continued study of storytelling, I have followed a thought leader within the space named Kindra Hall. If you are interested in learning more about storytelling and applying it to your practice, I highly recommend you find her website and begin following her on social media. Recently, I attended a handful of Kindra's lectures. She mentioned a project that stopped me in my tracks and forever validated storytelling and the value of sharing story-like information.

**Not so worthless objects**
This study is known as the "significant objects" project and can be found online at significantobjects.com. In order to demonstrate the value of storytelling and the ultimate impact it can have on a consumer, a group of writers visited garage sales, thrift stores, and other similar so that they could purchase items that are both random and worthless. Examples of these items included a piece of wire, and old salt shaker, a ceramic apple, and a one eyed stuffed animal. With these items in hand the group of writers created fictional stories about the origin of each item. Most times, these stories demonstrated the owner's emotional bond to the item.

These items were then posted on auction sites like eBay and sold for values that often paralleled 100 times their purchase price. Items that were purchased for under $.50 were being sold for more than $50. This demonstrated the fact that the purchaser was more interested in relating to and owning the story then they were the random, otherwise worthless item.

**We will practice storytelling**
During the lecture we will spend time telling some stories. I will demonstrate how revealing portions of my personal life can help you better understand who I am and why you should pay attention to my message. Then, we will take a deeper dive into some of the situations whereby you can apply storytelling in your practice’s big picture plan and day to day routine.
Why Should Corporations Have All the Fun?
Use Logo and Colors to Create a Memorable Experience
Bill Schroeder
InTouch Practice Communications
Schererville, IN

I'm sure that most veterinary professionals will agree with me that the average pet owner does not know the difference between good or bad veterinary medicine. With that said, I often struggle with the common approach or message that many veterinary practices utilize to market their businesses. All too many of our peers make statements like "we practice the highest quality medicine in the tri-state area" and expect pet owners to be able to understand what that means and somehow differentiate that message from the exact same message that practice down the street is utilizing. While pet owners are not veterinarians they are consumers who are accustomed to experiencing other brands and businesses that they don't understand. Often times, they do so by making decisions confidently out of emotion and overall comfort with the way a brand makes them feel.

Many of us have had the opportunity to share the experience of Disney theme parks with our children. While there we were greeted with smiles, happy thoughts, pleasant music, tasty treats that might not be readily available back at home, and overall fantasy like experiences set in a very pleasant climate. As we walked down Main Street, heard the songs play over the speakers, and watched the characters interact with us, we were being subjected to their branding plan that had much greater impact than the immediate space we were within.

These pleasant feelings were tucked away and saved once we got on the airplane and headed back home. Then, when our children popped in the Disney videos or when commercials are shown for products Disney offered, those familiar sounds and images brought back pleasant memories that made us feel good by remembering the experience we had in their theme park. Making a purchase outside of the park supports that positive feeling and ultimately keeps us closer to the Disney brand.

There have been many studies done about the massive world brands that are Disney, McDonald's, and Nike. Countless numbers of marketing students have dissected those brands and have identified overt and covert techniques utilized by their marketing agencies to affect their consumer base. In the end, the game of influencing their target market becomes a much more psychological game than expected.

Please understand that this talk is not about attempting to "Disney up" your practice. The resources required to do so would never be covered by the revenue experienced. Besides it's not necessary for you to do so because the market you are looking to affect is much smaller. With that said, it is important that you understand the value of creating a brand, doing so consistently, and relating that branch to your current and prospective pet owners so that they can develop feelings about your practice that are share worthy and memorable. In doing so, you will build a relationship with your client base that is less likely to be penetrated by less sophisticated competing marketing messages.

Often times when I begin a conversation with a current or prospective client about branding they lean towards color palette and logo. This is a great place to start, however at the very beginning of this conversation, I feel it necessary to pause and establish the understanding that it goes much deeper than appropriate selection of color and making certain that the logo demonstrates that you're a veterinary practice. It involves creating a culture and feeling that parallels the experience a pet owner will have when visiting your practice and creates a Disney like memory. Simply put, when they are reminded about your practice by exposure to your logo, veterinary related issues, or their pets, you have supplied them with enough historical and emotional information that they can immediately recall the experience they had while in your practice.

This is far from a quick fix approach. It is a long term culture shifting process that requires you to perform a great deal of self-examination and process building so that your brand becomes unique and recognizable. During the lecture we will discuss some very common branding techniques, ways that you can learn about who you are and who you want to be, and how your staff can help you develop and support those branding feelings that have a great impact on your current and prospective customers.

For the purposes of this talk we will spend a considerable amount of time focusing in on one of the most obvious, easy to understand branding techniques: logo design and color selection. I should caution you though that while this makes an extremely good visual presentation, it is very difficult to explain using the written word. I find myself creating these notes and being in a position that is very much like describing the color blue to a blind person who has never had the opportunity to experience color. So, with that said we will discuss some logo best practices here and will save visual demonstrations for the actual lecture.

When creating a logo for your practice you must focus on customization. It is extremely important that you not utilize stock clip art images or those images found on Google when creating your logo. Issues revolving around copyright infringement and other brands being able to utilize the same image or design for their business can cause legal issues or market confusion. This is an example where you should always seek professional assistance when developing your logo. Unless you are a graphic artist, you are not a graphic artist. The adage of not knowing what you don't know certainly applies here and can cause great additional expense if your logo is not done properly.

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I recently was exposed to a practice that had a friend develop their logo. This friend utilized imagery that was readily available through Google search and actually developed a pretty good design. The practice took this logo, created business cards, brochures, lobby signage, and a very large lit sign for the street in front of the practice. All of these pieces contained their new logo. Part of their redesign was to launch a new website. After a few months they received a letter from a very well-known graphic artist that found her artwork as being a part of that practice’s the logo and insisted upon having the logo removed or a fee of $25,000 paid for use of her artwork. The practice elected not to pay the fee, removed the logo from all print work, signage, and their website and took a loss in that they had to re-create a new logo and all of the supporting materials I previously mentioned.

Being as though I have first-hand experience with this case I am extremely confident that the friend of the practice meant no harm. In fact, she had no knowledge that she had done anything wrong for the images she used were readily available on Google search. Her mistake, and the mistake of the practice, was purely a lack of knowledge and experience.

Over the years I have been honored with the experience of helping many practices envision, develop, and produce affective logo design. Very often these ideas stem from simple conversation or epiphany like moments that demand “pen to napkin” design. While this grass roots, very raw approach is extremely appealing to me, it is very important that you understand the impact of converting those napkin or sketchpad designs over to a digital format. Many practices do not have a digital copy of their logo and are restricted buy the quality and flexibility of a flat, unchangeable logo design.

While working with a professional designer you should have the ability to receive all art components in a digital format so that they can be manipulated for future use. If done properly, it is very easy for adjustments to be made to a logo’s colors or size because many components are saved as individual layers that can be separated and manipulated independently. If you are currently in the position where you do not have a digital copy of your logo, please seek the services of a professional design firm that more than likely will have the ability to convert your logo over to a digital format and provide you a crisp, very flexible version for future use.

Responsible logo design involves looking at your logo project from many different angles. At the time of the logo creation you should always consider how your logo will be used and seen on different mediums. One great place to start is with color. I highly recommend that you insist that the graphic artist deliver the first versions of your design without any color being applied. This will allow you to experience the logo without any influence of color and demonstrate that it will be able to stand on its own when utilized in a black and white fashion. Failing to do so can easily put your project in jeopardy as similar or blending colors used in a logo can cause images to be unrecognizable when only “grey colors” (black and white) are available. After proving that your logo can stand on its own without the assistance of color, I suggest that colors are applied and tested.

When speaking of color, it is important that when you establish a color palette for your practice, that the artist or agency you work with provides you with the color codes that match the colors you selected for your brand. These codes lead future designers and printers to the exact color match so that brand consistency is possible. In the design and print space, these colors are referred to RGB and CMYK color codes.

Next, focus on size. Choose designs and fonts for text that are as easily readable on a business card as they are on the sign in front of your practice. All too often I come across logos that are developed and approved by practices in a large format but when shrunk down to the size of a business card, they become unrecognizable globs of ink that actually make the practice look less than professional.

During the lecture we will take a deep dive into logo design. I will run through some logo best practices, give examples of logos that clearly demonstrate a business’ purpose, and will demonstrate common mistakes made when creating a practice logo.
“Many online pet pharmacies are reputable. Some, however, may be businesses breaking Federal, State, and sometimes, International laws. These illegal online pharmacies may sell medicines that are counterfeit, outdated, mislabeled, or incorrectly formulated. The medicines may not contain the actual drug or may contain incorrect amounts of drug. Some may not work as well if the product is old/expired or has been stored in conditions that were too hot, cold, or humid. Others may not have the proper directions for use. If you are unhappy with the products you ordered, you may not be able to get your money back from an illegal online pharmacy”.

Protect Yourself and Your Pet, Be A.W.A.R.E US Food & Drug Administration, Animal and Veterinary, Online Pharmacies

Medical products are a key part of patient care and client compliance. They may not realize it, but clients receive amazing benefits if they purchase medications like heartworm, flea, and tick products from you rather than a pet specialty store or Internet pharmacy. Your job is to educate them about these benefits. Emphasize your quality control for handling and storing drugs. Share information about manufacturer guarantees. Highlight the convenience of purchasing necessary medications during client visits or ordering from your practice’s on-line pharmacy.

Plus – and this is very important – be sure clients know that your products are comparably priced. Many national retailers claim that they’re less expensive, but often it’s simply not true. Clients, however, don’t realize this unless you point it out.

The potential impact on revenue from lost product sales is significant, and many Well-Managed Practices® are feeling the pinch. See Figure 1 for a year-to-year comparison of self-reported changes in pharmacy revenue.

In a 2015 study conducted by Vets First Choice of 2.3 million transactions for 263,000 dogs analyzed over a 12-month period across 99 practices, the study found that heartworm compliance was just 25% and flea/tick compliance was just 16%. The financial impact of clients walking out without a recommended prescription is huge – the opportunity cost per practice in the study was $400,000 a year.

A 2003 study by AAHA showed that compliance rates for therapeutic diets was even worse – canine compliance was just 19% and feline compliance was just 18%.

Dr. Bob Beede, co-owner of Intermountain Animal Hospital in Meridian, Idaho, refuses to let that income go without a fight. “We must take action or lose market share,” he says. “We could keep our heads in the sand until we have no product sales, or compete by offering our own online pharmacy. We’ve chosen to compete. We tell our clients that, in this economy, we’re trying to help them,” Dr. Beede says. “We can sell the products online from our trusted sources at a lower cost, since we’ve eliminated our handling costs. The clients understand this and give us kudos for caring about them.”

According to Marsha Heinke, DVM, EA, CPA, CVPM, inventory ordering costs alone can amount to 8% to 15% of the product cost. These costs include all the tasks to determine the inventory needed, meeting with representatives, placing the order, receiving, documenting and putting the order on the shelf, and then documenting the order for bookkeeping purposes. An additional 25% to 40% of the product cost must be added to include the cost of the capital tied up, property taxes, insurance to guard against damage, theft, or other loss, spoilage, and time and paperwork to meet OSHA standards. “The holding and order costs, combined, comprise 25% to 40% of the total unit cost depending on the amount of time the inventory is on the shelf, the success of discouraging pilferage, and the efficiency of purchasing,” says Heinke.

Key objectives and observations from Benchmarks 2016 Well-Managed Practices® related to their online pharmacy include:

- Use an online pharmacy to improve practice operations and internal functions
- Leverage the use of technology to increase efficiency and productivity by better using the online pharmacy and helping clients set up auto-refills
- Use auto-prescription refill reminders and auto-ship refills from the online pharmacy to increase efficiency
- The online pharmacy is one technology “thing” that has provided the biggest ROI

Using a digital prescription management platform that includes a way to pre-authorize, and/or recommend, approve, and renew prescriptions, a way for clients to purchase the recommended products online and receive automatic shipments where applicable, and a dashboard to prompt action and document and track performance can significantly improve client compliance.

A January 2017 white paper published by Vets First Choice reported results from a 10-practice Pharmacy Study Group (PSG). The practices identified the following major benefits they’ve received as a direct result of their digital prescription management platform:

1. Improved compliance including for diets.
2. Gives the practice the ability to offer more products without the capital investment required to stock them in house.
3. Allows the practice to have more visibility and control over compounded medications.
4. Enhanced client convenience making their lives easier.
5. Allows the practice to shift in-practice product inventory to higher margin products.
6. Frees up cash to invest elsewhere in the practice.
7. Allows staff time to focus on caring for clients more vs. handling inventory.
9. Did not cannibalize in-house sales or profits.
10. Allowed the veterinarians to focus on medicine while being more productive.

The PSG reported the following aggregate revenue performance results:
- Total practice revenue increase of 8% ($1.8M)
- Service revenue increase of 8% ($1.3M)
- Lab revenue increase of 15% ($756K – part of the service revenue)
- In-house product revenue decline of 4.5% (-$257K)
- Online product revenue increase of 59% (734K)
- The per veterinarian growth results in the PSG were:
  - Total practice revenue increase of 14% ($88K)
  - Service revenue increase of 14% ($65K)
  - Lab service revenue increase of 21% ($30K – part of the service revenue)
  - In-house product revenue increase of .5% ($800)
  - Online product revenue increase of 67% ($60K)

\[\text{Net gain $477K}\]

Get started with your own practice on-line pharmacy

Start small
As with anything new, start small and start easy. Make a few select products or product categories available online. Preventive medications, medications for chronic conditions and therapeutic diets are great opportunities to establish success. Look for situations where a client maintained your recommendations for a short period and then stopped. Help the client set up the pet’s prescription in your on-line pharmacy.

Get staff buy-in
Hospitals that are using digital prescription management platforms successfully have the support of the entire practice team. Take time to discuss with your staff how your online pharmacy enables you to practice better medicine and provide better service. Motivate your employees to promote your online pharmacy by creating contests for capturing walkouts (clients who leave without their prescribed medication), establishing team targets or goals, or by simply asking team members to fill prescriptions for their own pets through your online pharmacy. Once your staff has experienced the convenience themselves, they’ll be your biggest advocates!

Talk it up
Discuss the practice’s online pharmacy capabilities everywhere: in the exam room, during discharge, and on the phone. Let clients know your clinic’s on-line pharmacy means they can purchase their pet’s medications from you online, at competitive prices, and be assured that the drugs have been handled and maintained properly and are labeled for use in the United States with the manufacturers’ guarantees in place. And, their money stays local. Most clients using direct-to-consumer pharmacies do so out of convenience and may not know you offer an online pharmacy too.

Get peer input
There are clinics all around the country that have embraced digital prescription management and can help you understand the process and benefits of the online approach. Ask how they implemented their practice online pharmacy.

Ask staff to keep their ears open
What prescriptions do clients ask you to send to outside pharmacies? Add those items to your online pharmacy for client convenience and to retain the revenue for the practice. Create a script for staff members to follow when they receive requests to forward prescriptions to outside pharmacies. If you pay attention to what clients want, you’re more likely to keep their business.

And don’t forget to educate your clients about the benefits of supporting locally-owned businesses. Multiple studies show that buying from locally-owned businesses keeps more money in the community.
- A 1995 landmark study of new Wal-Mart stores conducted by Iowa State University professor Kenneth Stone found that 84% of Wal-Mart’s sales simply shifted dollars away from existing local merchants versus creating new sales in the community.
- A study conducted by Civic Economics found that approximately 73% more money stays in the community when consumers choose locally-owned and independent businesses.
- And The New Economics Foundation, an independent economic think tank based in London, England, compared what happens when people buy produce at a supermarket versus a local farmer’s market or a community-support agriculture program and found that twice the money stayed in the community when folks bought locally.
Here are five reasons to patronize a locally-owned veterinary practice.

1. **Employment opportunities** - small, locally-owned businesses are the largest employers in the U.S. Veterinary practices employ your family, friends, neighbors, and family and friends of friends. While sales in the 500 largest corporations grew 700% in the past 20 years, they are now net dis-employers – firing more people than they hire despite record profits.

2. **Client service and patient care** – locally-owned veterinary practices hire people with specific skills and continue to develop their expertise, thus strengthening employees’ abilities and providing better service to clients. Locally-owned veterinary practices focus on developing healthy, long-term relationships with their patients and clients.

3. **Investment in the community** – the owners of locally-owned veterinary practices live in the community, are less likely to leave, and are more invested in the future of the community. Veterinarians and their staff volunteer, serve on boards of non-profit organizations, support local schools, support and participate in community activities, and donate to worthy local causes.

4. **More money stays in the community** – local veterinary practices purchase from other local businesses. Buying local goods and services helps grow other area businesses and speeds the circulation of money in the community. If money circulates more quickly and passes through more hands, more people have the benefit of the money and what it purchases for them.

5. **Maintain a unique community character** – people are attracted to communities that preserve their one-of-a-kind businesses and distinctive character, but often forget their survival depends on our patronage. A marketplace of small businesses is the best way to ensure innovation over the long-term.

Veterinarians are painfully aware of the cost of inventory, the importance of having the necessary inventory items when they’re needed, and the importance of preventing loss from shrinkage and out-of-date drugs. However, many practices lack the time, manpower, or processes to effectively manage inventory. If you haven’t yet developed a solid inventory management program, it’s time to get started. Don’t be shy about asking for help. Many management consultants, veterinary distributors, pharmaceutical manufacturers, online pharmacy platforms, and practice management software companies offer guidance to help practices establish an effective inventory management program. It’s never too late (or too soon) to get started!

Take steps today to protect your pharmacy. Communicate the benefit of buying from you and supporting locally-owned businesses. Pay attention to your medical standards of care and monitor consistency between the doctors. Track and monitor compliance to identify opportunities for additional, necessary care. Establish your own online pharmacy, and stock products in house from your trusted sources. Proactively market your practice to educate clients and your community about the services your offer. Follow up on unsatisfied reminders for appointments and prescription refills and take responsibility for getting necessary appointments scheduled and the prescription refilled. The results will be worth the effort!

**Figure 1 – Slipping product revenue**

How much has your volume of medication dispensed declined in the past two years because clients are using other sources to purchase their products?

<table>
<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>No change</td>
<td>19%</td>
<td>15%</td>
<td>13%</td>
<td>13%</td>
<td>23%</td>
<td>9%</td>
</tr>
<tr>
<td>Less than or equal to 5%</td>
<td>65%</td>
<td>63%</td>
<td>52%</td>
<td>48%</td>
<td>34%</td>
<td>41%</td>
</tr>
<tr>
<td>6% to 10%</td>
<td>12%</td>
<td>27%</td>
<td>28%</td>
<td>32%</td>
<td>38%</td>
<td>25%</td>
</tr>
<tr>
<td>11% to 20%</td>
<td>4%</td>
<td>3%</td>
<td>5%</td>
<td>7%</td>
<td>5%</td>
<td>5%</td>
</tr>
<tr>
<td>More than 20%</td>
<td>0%</td>
<td>1%</td>
<td>2%</td>
<td>0%</td>
<td>1%</td>
<td>3%</td>
</tr>
<tr>
<td>Our volume has increased</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>17%</td>
</tr>
</tbody>
</table>

*Source: Benchmarks Well-Managed Practice Studies by WTA Veterinary Consultants and Veterinary Economics*
The Elusive 20% ROI:
Can it be Done?
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 20% 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 and large bags of diets 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.greencyr.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 20% ROI

Gross Revenue 100%

Variable Expenses 23%
Fixed Expenses 7%
Staff Compensation 22%
Facility Expenses 8%
Total operating expenses (60%)

Amount Available for Associates and Owner 40%

Doctor Compensation 17%
Owner Management Compensation 3%
Total Doctor Compensation (20%)

Owner Return on Investment 20%

Reinvestment – Equipment 3%

Remaining Amount Available to Owner 17%

Compare your expenses to these benchmarks

Variable Expenses (as a % of total revenue)

<table>
<thead>
<tr>
<th>Item</th>
<th>Top 10%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs and medical supplies (includes radiology, surgery and hospital supplies but excludes food, shampoos, etc.)</td>
<td>9.9%</td>
</tr>
<tr>
<td>Heartworm, flea, and tick products</td>
<td>4.3%</td>
</tr>
<tr>
<td>Laboratory</td>
<td>4.1%</td>
</tr>
<tr>
<td>Diets (therapeutic and retail)</td>
<td>3.0%</td>
</tr>
<tr>
<td>Over-the-counter retail products (e.g. toys, collars, shampoo)</td>
<td>0.4%</td>
</tr>
<tr>
<td>Credit card fees</td>
<td>1.6%</td>
</tr>
<tr>
<td>Bad debt, collection fees</td>
<td>0.1%</td>
</tr>
<tr>
<td>Cremation, care of remains</td>
<td>0.6%</td>
</tr>
<tr>
<td>Sales and use tax</td>
<td>0.7%</td>
</tr>
<tr>
<td>Medical waste disposal/radiation badge monitoring</td>
<td>0.1%</td>
</tr>
<tr>
<td>Practice vehicle expense</td>
<td>0.1%</td>
</tr>
<tr>
<td>Total</td>
<td>24.9%</td>
</tr>
</tbody>
</table>

Fixed Expenses (as a % of total revenue)

<table>
<thead>
<tr>
<th>Item</th>
<th>Top 10%</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>Category</td>
<td>2022 %</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
<td>--------</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>9.1%</td>
</tr>
<tr>
<td>Non-doctor Staff Compensation (W2 wages as a % of total revenue)</td>
<td></td>
</tr>
<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>24.7%</td>
</tr>
<tr>
<td>Facility Expenses (as a % of total revenue)</td>
<td></td>
</tr>
<tr>
<td>Annual rent or mortgage payments</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>8.0%</td>
</tr>
<tr>
<td>Reinvestment</td>
<td></td>
</tr>
<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>2.0%</td>
</tr>
</tbody>
</table>
Making your practice more profitable and positioning it for continued success isn’t about luck. You don’t roll the dice and hope to meet your medical and financial goals for the year. Being profitable is about playing the right hand at the right moment and making changes in your practice that maximize your strengths. Try these tips to ensure your practice will be flush with success.

**Tip #1 – Make strategic fee changes**

Instead of annual, across-the-board increases, review fees quarterly and determine the ones you’ll increase, the fees that won’t change, and in some rare situations the 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.

- **Competitively price shopped 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.

- **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>In-house pharmacy cost</th>
<th>Markup – 150%</th>
<th>Handling fee</th>
<th>Final price to client</th>
</tr>
</thead>
<tbody>
<tr>
<td>$10</td>
<td>$15</td>
<td>$10</td>
<td>$35</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 select 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 February, senior citizen discounts, 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 price concessions under 15%.

- **Price doctors’ non-shopped services based on value.** Published resources like WTA’s *Benchmarks Well-Managed Practice Study* and AAHA’s *The Veterinary Fee Reference* provide insights about pricing value-based services. A client can see the tangible benefits of your services in a happy, healthy pet. Continue to look for other opportunities to influence clients’ perception of value. What’s most important to them and keeps them coming back? What’s a turn-off? If you don’t know, ask them. Does your external and internal “visual” 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.

**Tip #2 – Re-energize your practice’s compliance efforts**

If your team has run out of steam on compliance, perhaps it’s time to change the word we use to describe “patients receive the healthcare necessary to ensure a long, healthy life in harmony with our medical protocols.” I like the word harmony, but feel free to choose your own, favorite word. In the book *If Disney Ran Your Hospital*, author Fred Lee describes four levels of motivation – the first, compliance, is defined as “doing what someone makes me do,” which isn’t very motivating! Mr. Lee 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). So let’s change what we call “compliance” to move clients to the “habit” level of motivation.
Not sure where your practice stands when it comes to client compliance? Here’s a quick and easy project to find out. Choose three critical preventive wellness components of care your patients need, but not all are getting. Meet with your team and set daily, weekly, monthly, and annual goals to increase the number of times you provide this necessary care. Monitor your progress, discuss the results, and make any adjustments necessary to hit your goals.

**Example:** a practice chose fecal testing, dentistry, and senior wellness testing as the areas they wanted to improve patient compliance with. This practice has 6,000 active patients with the following age distribution: less than 3 years of age – 25%, or 1,500 patients; 3 to 6 years of age – 25% or 1,500 patients; more than 6 years of age – 50% or 3,000 patients.

The doctors and staff set the following goals:

<table>
<thead>
<tr>
<th>Percentage Goals</th>
<th>Goal</th>
<th>Actual</th>
<th>Opportunity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fecal testing</td>
<td>90%</td>
<td>50%</td>
<td>40%</td>
</tr>
<tr>
<td>Senior wellness testing</td>
<td>90%</td>
<td>40%</td>
<td>50%</td>
</tr>
<tr>
<td>Dentistry - pets 3+ years of age</td>
<td>50%</td>
<td>20%</td>
<td>30%</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Number of occurrences Goals</th>
<th>Goal</th>
<th>Actual</th>
<th>Opportunity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fecal testing</td>
<td>5,400</td>
<td>3,000</td>
<td>2,400</td>
</tr>
<tr>
<td>Senior wellness testing</td>
<td>2,700</td>
<td>1,200</td>
<td>1,500</td>
</tr>
<tr>
<td>Dentistry</td>
<td>2,250</td>
<td>900</td>
<td>1,350</td>
</tr>
</tbody>
</table>

They set a weekly goal of doing 46 more fecal tests, 29 more senior wellness tests, and 26 more dental procedures. They will track and monitor their progress and make any necessary adjustments to hit their targets.

**Tip #3 – Spark growth with a new service**

Adding new services to your repertoire creates fresh energy and enthusiasm for the entire team. New services are another opportunity to differentiate your practice, expand the offerings for existing patients, and attract new clients. Rehabilitative therapy topped the list for Benchmarks participants in the past 2 years, followed closely by laser therapy. Behavior counseling made the list of future additions in the next 2 years. Dr. Richard Vesper, owner of Avery Animal Hospital in Hilliard, OH has been providing behavior counseling for 10 plus years. Yet it never quite took off in the way he had envisioned. With the assistance of his technician, Gretchen Latham, they’ve reworked their approach and are seeing good results.

If you’re looking for a new growth center, or a new campaign to improve client compliance with previous recommendations, check out dvm360.com/toolkit. The packages there on dentistry, parasitology, dermatology and more feature client handouts, team member handouts, staff meeting ideas, Facebook posts and tweets, audio and video clips and more to help you educate and inspire.

**Tip #4 – Use the marketing technology you pay for**

Boosting revenue requires an effective marketing plan. And there are lots of companies that offer lots of options to help craft your marketing plan. Do you know how many marketing tools your company has purchased, or which technology software does what? Do they overlap? Have you kept up on the latest technological innovations for use in and for veterinary hospitals? Is your hospital even using all of the “stuff” you’ve bought? Does it work? Do your clients like it? Does the team like it?

If your hospital’s like most, you probably don’t know the answer to all of those questions. To start, gather what you need for an overview. Think of this as a treasure hunt. Pull current invoices for the marketing and technology services you subscribe to, including your website, digital marketing, print ads, Yellow Pages (or other directories) and any other marketing you engage in. Make sure you know what each service does; call the provider to ask if you’re not completely sure. Are new services being added? Can the price be lowered now?

Create a spreadsheet so you can compare the services you’ve subscribed to side-by-side. The goal of this exercise is to narrow the list to services and technology you’re actually using and find the value amongst all of those must-haves you purchased through the years. Kelly Baltzell, President and CEO of Beyond Indigo Pets recommends including the following information in your spreadsheet:

<table>
<thead>
<tr>
<th>Product or service – what is it?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do you use it? Is it duplicated elsewhere?</td>
</tr>
</tbody>
</table>

When attending a convention or inspiring meeting, you hear about the latest, greatest, next best thing and sometimes buy it or sign up for it on the spot. The practical need for the service may not be clear. Plus even with the best of intentions and some utility at the time, as the years pass, technology changes. For example, some practice management software now offers text and e-mail reminders, so a secondary service with the same services may now be redundant.
Is it meeting your goal?
If you don’t know the answer, or the service is sitting unused, that’s a red flag. Marketing services should make happy clients and keep the door swinging. Technology needs to be useful to the staff, the pet owners, or both. Measurement should be in ROI – both financial and in happy clients!

How much does it cost?
What’s the monthly cost of the service and technology? Can the price be lowered? What’s included in the cost?

Is it a “want” (this is so cool!), a “need” (our clients expect this), or a “must-have” (we can’t get by without this)?
We’ve all made impulse purchases based on emotion in our personal and professional lives. This spreadsheet is the perfect opportunity to step back and see if that e-mail reminder system or website optimization company is something your business really needs. Over time, a need may change to a must-have and a want into a need. But it goes the other way too. Some must-haves slowly fade away (Yellow Pages, anyone?).

Once every product and service is in the spreadsheet, the decision-makers in your hospital should be able to see how useful they are. Talking to everyone in the team will help too. Are there any needs or must-haves missing from the list? If there are, those items would be the next ones to research and purchase.

In Benchmarks 2016: A Study of Well-Managed Practices, Bill Schroeder, owner of InTouch Practice Communications, explains how to dig through online reviews and SEO to manage your on-line reputation – an important part of your marketing plan. He recommends:

- **Step 1** – set up Google Alerts at google.com/alerts to receive notifications when certain words or phrases are indexed by Google (like about your name and your practice name).
- **Step 2** – establish accounts with Yelp, Google, and Facebook, and then let clients know that you appreciate review comments on those channels.
- **Step 3** – set up a service to automatically send electronic surveys after each appointment so that reviews can be gathered, organized and positioned properly on the practice website.

Tip #5 – Implement patient-calming, patient-friendly techniques
Is it worth it to calm and quiet the practice, to gently handle patients, to treat and distract for exams, and to teach clients about stress in their pets and how to teach them another way? Yes! Practices using low-stress techniques are seeing revenue growth and believe these techniques lead to better medical care. To enhance your team’s knowledge (or if you haven’t yet begun), get certified through AAHA’s Fear Free certification program (fearfreepets.com). Visit [www.dvm360.com/lowstress](http://www.dvm360.com/lowstress) for free handouts, videos, galleries, how-tos and general advice about low-stress veterinary visits. Attend specialized tracks on behavioral health of pets and low-stress veterinary visits when attending conferences like NAVC. And for extra help with your feline patients, the American Association of Feline Practitioners provides fantastic guidelines about how to ensure your feline patients leave your practice calmer, and visit [catvets.com/cfp/veterinary-professionals](http://catvets.com/cfp/veterinary-professionals).

Tip #6 – Create a sales culture
The word “sales” can conjure up negative stereotypes of the used car salesperson who says whatever is necessary to get you into that old clunker. It’s time to change that misconception and define the true meaning of selling: education that allows clients to make informed decisions.

A sales culture benefits all stakeholders. Clients receive the peace of mind that comes with making the best decisions for their pet’s well-being. Pets receive complete care. Staff members take pride in knowing their expertise is directly benefitting pets. Practice owners feel that same pride and see the increase revenue that leads to better team pay, a healthier bottom line, and a stronger business to sell down the road. Resolve this year to make your practice the definitive source of pet well-being in your area by building a practice where all staff members provide the education and recommendations necessary for optimal patient care.

Dr. Michael Watts, owner of Clevengers Corner Veterinary Care in Amissville, Virginia, says he, his associates, and his staff got into veterinary medicine to help pets, not for the money. This is also very likely for you and your practice. However, Dr. Watts notes that the money serves as a good measure of how well you’re doing in reaching the goal of best serving patients and clients. In fact, he chooses to share sales figures with his staff. He creates a culture where sales are a measure of compliance, and compliance is a measure of how well pets are getting the care they deserve.

Dr. Brent Cook, owner of Kingsbrook Animal Hospital in Frederick, Maryland agrees. He says they’ve gone through phases where they talked about sales and revenue with all the staff, but most of the time that didn’t work well for their practice. “Most of our staff members are not in it for the money – they would give up the clothes on their back to help an animal in need. As a result, if we bring up too much about sales and revenue, they start to think that all we care about is the bottom line and not our patients. But that’s easy to fix from both sides. Good medicine is profitable. So we educate our staff and our clients about what is best for their pet, and the sales and revenue take care of themselves.”
Dr. Cooks says he does talk about revenue with the doctors and managers, but it’s always in conjunction with a discussion about making sure consistently good medicine is offered to patients no matter which doctor they see. Rather than look at sales of professional services, they look at a specific item, like tonometry, and see how many tonometries each doctor completed relative to the number of patients they saw. If they are all consistently practicing the same quality medicine, then this value should be about the same for all of the doctors. Sometimes it is, sometimes it’s not. “If it’s not,” says Dr. Cook, “we discuss why it’s different. Sometimes we find that a doctor just wasn’t educated on the service, sometimes the other doctors offer a better way for them to word it to clients. The key is the discussion that gets everyone on the same page. Nobody gets defensive because their numbers aren’t as good. After our discussion, they want to increase “sales” because it’s better medicine, not because it’s more profitable. But, better medicine is more profitable, so it’s a win-win.”

Tip #7 – Join a WMP management group
These groups of 20 high-quality practices meet twice a year and discuss anything and everything related to managing a veterinary practice. From financials and fees to marketing, staff development and transition planning – strategies and insights are willingly shared by the members.

Group member Dr. Dean Tyson says, “We always return from our WMP Group Meeting energized and excited about bringing the information back and implementing it. We are thankful to have partnered with the WMP Groups, are grateful for our achievements thus far, and look forward to continuing the journey.”

Member Dr. Peter Fisher adds, “Membership in our WMP Group has transformed the way I manage our hospital and how it functions on a day-to-day basis. I feel better about how my practice operates and have a new understanding about our finances.” Contact Wutchiett Tumblin and Associates or visit wellmp.com to learn more about our WMP Management Groups.
Cost is a proven obstacle to veterinary care for many pet owners. Often, it’s not the price of services itself, but the fear of the “big bill,” according to the Bayer Veterinary Care Usage Study conducted by Brakke Consulting. Financial tools and communication can help minimize this barrier, improving client satisfaction while at the same time improving compliance and revenue for the veterinary practice. The tools include pet health insurance, preventative care plans, medical credit cards and written financial policies.

Pet health insurance
Research conducted by the North American Pet Health Insurance Association (NAPHIA) showed that the majority of veterinarians wish all clients had pet health insurance. The NAPHIA research also showed that significantly more pet owners would purchase pet health insurance if their veterinary practice actively recommended it. NAPHIA is a trade association representing virtually all of the companies that offer pet health insurance in the US and Canada.

Veterinarians responding to the NAPHIA survey identified three key areas in which pet health insurance contributes to patient health and practice revenues. Insurance increases:

- Compliance on recommendations
- Purchases of veterinary services
- Overall health expenditures on pets

In addition, the research demonstrates that clients with insured pets visit the veterinarian more often. Clients spent 29% more per year on veterinary care if their dog was covered by pet health insurance, and a whopping 81% more on cats. The increased lifetime value of patients covered by pet health insurance is dramatic.

Most pet insurance is purchased within the first year of ownership and/or after the pet’s first visit to a veterinarian. In addition, the people most likely to take a strong interest in pet insurance are first-time pet owners, and experienced pet owners with a new pet.

Targeting new pets, new pet owners and new clients helps the practice team focus on those clients most likely to be receptive to the information. It’s also the ideal time for clients to benefit most from the coverage.

Again, educating clients about pet health insurance and recommending one or two specific companies can pay big dividend in increased visits, revenue and patient health. Veterinarians recommend many things they don’t sell: Regular exercise. Safe environments. Behavior training. All benefit the pet’s health and wellbeing, and make pet ownership more enjoyable and rewarding. Pet insurance fits in that same category.

Preventive care plans
Preventive care plans, also called wellness plans, also help clients manage their pet care expenses. The Bayer Veterinary Care Usage Study published in 2011 showed that pet owners would visit the veterinarian more often if: (1) veterinarians provided a health plan for their pet, and (2) pet owners could pay for routine veterinary services in monthly installments. Preventive care plans satisfy both objectives.

To determine the impact of preventive care plans on practices, Brakke interviewed corporate and independent practices that offer wellness plans. The independent practices included Bigger Road Veterinary Clinic in Kettering and Springboro, OH; Doral Centre Veterinary Clinic in Doral, FL; and Lansing Veterinary Clinic, Lansing, IL. National Veterinary Associates, a privately held corporate owner of veterinarian practices, has installed Pet Annual Wellness (PAW) Plans in 129 of its practices, with more than 40,000 active plans.

All practices found that wellness plans increased visits and revenue, expanded and improved patient care, helped attract new clients, and strengthened the practice-client bond. “Wellness plans make preventive care more affordable” was a common theme among all practices interviewed.

NVA found that professional service visits of those purchasing plans increased 69% compared to the period before purchasing plans, from a mean of 3.3 visits per year to a mean of 5.5 visits per year.

Lansing compared the number of visits before and after purchasing plans, as well as the number of visits by plan participants compared to non-plan patients. Clients with wellness plans visited nearly twice as often as those without plans, and much more frequently than they did prior to purchasing plans (Fig. 1).
Both NVA and Lansing found that annual revenue per patient increased significantly for patients with wellness plans compared to those with no plan. In Lansing’s case, clients with wellness plans spent much more on veterinarian services than they did in the year prior to starting the plan. They also spent substantially more than those clients without plans (Fig. 2).

Similarly, NVA found that the mean amount spent annually per patient for medical services increased 57%, from $389 to $613. NVA found that expenditures on non-medical products and services outside of the plans increased 29% as well, from a mean of $223 to $286. So the total “lift” from wellness plans was $287 per patient per year.

Bigger Road Veterinary Clinic’s experience was similar. Its records showed that clients spent an average of 65% more in the year after purchasing wellness plans than they did the prior year.

The practices typically offer 10 to 15 plans, including plans for both dogs and cats. Prices varied. Many “standard” plans were in the $25-$35 per month range, but some were as high as $45/mo. Premium plans, which typically include spay/neuter for juvenile pets and dental prophylaxis for adult pets, typically cost $10-$15 per month more. Some practices offer a third level, or “advanced” plan, with added services typically recommended for senior pets. Plans sold for dogs far outweigh plans for cats. $30 per month seems to be the sweet spot for standard plans, especially for dogs. At that price most pet owners find preventive care very affordable.

Both NVA and Lansing discount services 35% to 40%. Part of the rationale is that wellness plans provide for a much more comprehensive package of services than clients typically buy, including such things as twice-a-year wellness exams and fecal tests. In addition, clients rarely utilize all the services included in the plan, so some of the discount is “recouped” in un-used services. The combination of a more robust services, services paid for but not used, plus additional outside-the-plan purchases, add up to substantially increased revenues per patient – as well as a higher level of veterinary care.

Bigger Road Veterinary Clinic does almost no discounting from regular prices, but it does include at no cost a small number of inexpensive services such as nail trims. Yet it has sold more than 500 plans in a little more than a year.

All practices referenced in this presentation charge a one-time enrollment fee, generally $45-$50. The enrollment fee helps gain a commitment to the plan from clients, and helps offset the deferred cash flow due to receiving revenue in monthly installments.

Most plans include free visits. NVA PAW plans include up to four visits per year. Bigger Road plans include three visits and Doral plans five. Lansing offers unlimited free visits.

Medical credit cards
Having emergency credit available can make a huge difference both to pet owners and the veterinary practices that serve them. The most widely used credit service is CareCredit, a financial services firm serving several medical professions.

In 2011, Brakke compared data for the years 2007 through 2010 from the practice management systems of more than 500 veterinary hospitals, including approximately 100 practices that use CareCredit and 400 that don’t. In addition Brakke analyzed data from more than 350,000 pet owning-clients of approximately 120 veterinary practices that use CareCredit and compared the behaviors of clients that use CareCredit to pay for veterinary expenses to those who were non-users.

The analysis showed that the use of CareCredit had a substantial impact on client spending, and a significant, sustained and positive impact on veterinary practice income.

Specifically:
- Clients that used CareCredit spent 93% more per year and 47% more per pet at their veterinary practices than clients without CareCredit.
- Veterinary practices that offered CareCredit generated 19% more medical revenue and 17% more total revenue than practices that did not offer CareCredit.
- Practices that were more active users, defined as practices with at least 25 client applications or $50,000 in total CareCredit charges per year of CareCredit generated 31% more medical revenue and 26% more total revenue annually than non-users.
The impact of CareCredit was immediate. Veterinary practices that enrolled with CareCredit generated 11% more medical revenue within the first 12 months following enrollment than in the 12 months prior to enrollment.

Written financial policies
Perhaps the most important financial tool is communication. That is, making sure that clients know and understand the practice’s financial policies, and the tools available to clients. It is important to have those policies in written form. Share them with new clients, and periodically with established clients.
When talking about pet owners, it’s easy to picture in our head the typical pet-owning family: Dad, mom – white of course, two children, a dog, maybe a cat or two. While in many cases that might be a reasonably accurate description of some clients, it’s really a stereotype. Or maybe wishful thinking. The fact is, there are important but subtle changes taking place in the pet owner population, and those changes can easily impact whether they purchase services from you, and if so under what circumstances.

As we talk about these changes, it’s important to remember that all retail business is local. So some may potentially impact your practice more than others. The key is to think about which ones may be happening in your neighborhood, and how you can address them among your clients.

**Single-parent families**
The percentage of households with two parents is declining. In fact, by 2014 only 69% of household had two parents, down from 87% in 1960. Not only are 25% of households headed by a single mother, in an additional 15% of households, the mother is the sole or primary provider. In most, but not all of those households, mean income is lower than those in which men are the primary breadwinners.

Implications: In single-parent households, time – and often money – are challenges. Offering convenient hours, such as evening or weekends, can make it easier for them to fit in veterinary care. Also, be sure your clients are aware of drop-off services offered by your practice.

**Reduced home ownership**
Home ownership in the US is declining. Adults living with parents is the highest it has been since the 1940s. In 2015, 43% of men 18-34 years of age were living with parents or relatives. In addition to adult children living at home, there has been a shift from owning to renting, such that home ownership is at the lowest level it has been in years. Pet population tends to follow household formation, and household formation took a big hit during and immediately following the Great Recession. Another, unrelated trend that is bearish for pet population is the aging population. Pet ownership tends to start declining when adults reach 50 to 55 years of age and declines to very low levels by the time people reach 70 or older. And guess what? The 55 and older cohort is the fastest growing population group in the US.

Implications: If you’re not seeing a lot of new clients, the stagnation of the pet population may be a factor. That means you have to work all the harder to attract new clients. More people are sourcing pets from shelters and rescue groups than ever before. Work with these organizations, as well as breeders and welcome organizations to identify people that may have a new need for veterinary services.

**More millennials**
Now I’ve got better news. Currently those in the 25 to 29 and 30 to 34 age cohorts are the third and fourth largest groups in the US. However, by 2020, those two groups will be the largest population groups. This age range is when people are most likely to purchase a home, get married, have children and acquire pets. In fact, many of them are deferring having children and acquiring pets instead. They are partly responsible for an uptick in the number of households owning pets. While millennials are favorable to owning pets, keep in mind that they aren’t exactly like their parents’ generation when it comes to pet care. Brakke Consulting conducted a study recently and learned that millennials are less dependent on veterinarians for pet care advice, and are more open to purchasing healthcare products and services from non-conventional sources. They are also more likely to outsource services than their parents, so they are heavier users of daycare, boarding, and grooming services (Fig 1, 2, 3).

**Figure 1. Loyalty to veterinarian by generation**

<table>
<thead>
<tr>
<th></th>
<th>Millennials</th>
<th>GenX</th>
<th>Boomers</th>
</tr>
</thead>
<tbody>
<tr>
<td>I completely trust my veterinarian's recommendations</td>
<td>64%</td>
<td>70%</td>
<td>78%</td>
</tr>
<tr>
<td>I generally comply with my veterinarian's recommendations</td>
<td>67%</td>
<td>70%</td>
<td>79%</td>
</tr>
<tr>
<td>I feel a sense of loyalty to my vet</td>
<td>55%</td>
<td>59%</td>
<td>63%</td>
</tr>
</tbody>
</table>
**Figure 2. Product Sourcing by Generation**

<table>
<thead>
<tr>
<th></th>
<th>Millennials</th>
<th>GenX</th>
<th>Boomers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Want choice in where to fill scripts</td>
<td>62%</td>
<td>54%</td>
<td>45%</td>
</tr>
<tr>
<td>Visiting vet less often</td>
<td>17%</td>
<td>12%</td>
<td>12%</td>
</tr>
</tbody>
</table>

**Figure 3. Pet Care Outsourcing by Generation**

<table>
<thead>
<tr>
<th>% Having Used</th>
<th>Millennials</th>
<th>GenX</th>
<th>Boomers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daycare</td>
<td>32%</td>
<td>20%</td>
<td>7%</td>
</tr>
<tr>
<td>Boarding</td>
<td>34%</td>
<td>23%</td>
<td>15%</td>
</tr>
<tr>
<td>Grooming</td>
<td>65%</td>
<td>48%</td>
<td>41%</td>
</tr>
<tr>
<td>Dog walking</td>
<td>30%</td>
<td>17%</td>
<td>4%</td>
</tr>
<tr>
<td>In-home pet sitting</td>
<td>33%</td>
<td>24%</td>
<td>13%</td>
</tr>
</tbody>
</table>

Implications: If you offer daycare, boarding and/or grooming, this is an excellent target for your services. In addition, keep in mind that many of these are first-time pet owners, so it is worth your while to spend extra time educating them about proper pet care, including proper veterinary care.

**Changing Ethnicity**

Every year, the US becomes less white. In fact, within 50 years all of us, regardless of race or national origin, will be minorities. In recent years, the greatest influx of immigrants has been Hispanic. The tide is turning, and increasingly Asians will be the predominant immigrant group. Today 87% of pet-owning households are white. By 2025 – less than 10 years from now – only 60 percent of pet-owning households will be white. Keep in mind that different races and ethnic group have different approaches to pet ownership and pet care, as well as the different levels of resources to fund them.

Implications. Make sure you are reaching out to all the potential constituencies in your trade area. If you have a large Hispanic or Asian population in your area, reach out to them through their community organizations and religious institutions. The schools are often a great place to reach various ethnic groups as well.

**Financial Stress**

One of the things the Great Recession taught us is that financial pressures impact veterinary care. But while the recovery from the recession seems pretty much over in most areas, that can be an illusion. It’s important to recognize that financial pressures haven’t gone away. Median earnings for men in the US haven’t changed significantly since the 1970s. Women’s earnings have improved about 25% over that time, so that has helped in two-parent households. We’re still a long way from getting back to where we were before veterinary visits started declining in the early 2000s.

But wait, you say. Business was rocking and rolling in the early 2000’s. Maybe visits weren’t increasing rapidly for many of us, but business was good. Here’s why. Remember the housing bubble? Home values were increasing rapidly. It was easy to sell your house and get a mortgage on a bigger one. From 2001 to 2006, American’s pulled $5 trillion – yes trillion – dollars out of home values and spent it on things like big screen TVs, cars, restaurant meals, vacations and, yes, veterinary care. That’s about $17,500 for every man, woman and child in the US. That money is gone, and it won’t return any time soon. For one thing, the housing bubble is over. For another, people are saving more than they’ve saved in a long time. That’s a good thing for their financial security. But it does mean that they are spending less on veterinary care and other discretionary items.

The recovery hasn’t occurred evenly across the economy. It has heavily favored the well educated. In Chicago, for example, median earnings are still way below 2007 levels in all categories except those with graduate degrees. Underemployment is still rampant, especially among those with less than a college education.

The result of all this is a declining middle class. Since 2000, the middle class has declined from 54% to 50% of the population. And exactly what is the “middle class”? A family of four with annual household income of $44,000 to $132,000 is considered middle class. The lower socio-economic levels have increased, from 27% of the population to 29%. There has been a similar gain, from 18% to 21% in the upper groups. If you practice in an upscale neighborhood, those trends may well be favorable to you. In the last 30 years, the only demographic group that has experienced an increase in net worth is the upper class! If you service the 80% not in the upper class, recognize that there’s still a lot of financial pressure on your clients.
Implications. This continued financial stress has a lot of implications for veterinarians. First, it will make it more difficult to continue to raise prices. There’s evidence that inflation in the cost of veterinary services is driving pet owners away and causing continuing declines in veterinary visits. That’s a lose-lose proposition for you, your clients, and the patients you care for. So look for ways to managing pricing, especially for shopped and routine services. Try to improve value, not price. Also look for ways to make it easier for clients to pay for your services. Things like monthly paid wellness plans, pet insurance and medical credit cards like CareCredit. We’ll talk more about those in the next two hours.

Again, what’s going on in your neighborhood is what matters most. But changing demographics can sneak up on you. Be alert to financial pressures of your clients, or to shifting racial or ethnic cohorts in your trade area. Don’t just target the same group of clients you’ve always targeted. Those new groups need care for their pets, too. But they may buy differently than the clients you’ve traditionally served.
Research demonstrates that veterinarians are highly supportive of clients having pet health insurance. In fact, 56% of veterinarians in the US (85% of Canadian veterinarians) wished all their clients had pet health insurance. In related research, 50% more pet owners said they would purchase pet health insurance if their veterinarians actively recommend it.

What the studies show is that having brochures from a few insurance companies available in the waiting area isn’t enough to communicate “recommendation” to pet owners. There are many other, easy and more proactive steps that practices can take to increase client interest in pet health insurance.

Two research studies were conducted by the North American Pet Health Insurance Association (NAPHIA). One survey, in October 2015, was among a sample of 626 US and Canadian adults who had primary or shared responsibility for pet care. The second, in January 2016, was among 505 US and Canadian companion animal veterinarians in private practice. NAPHIA is comprised of pet health insurance (PHI) organizations from across Canada and the United States. NAPHIA’s membership makes up over 99% of all pet health insurance coverage in effect in North America.

It’s easy to see why veterinarians prefer patients to be covered by pet health insurance. When asked their expectations of pet health insurance, as well as how well those expectations were met, veterinarian list the following as high priorities on which pet health insurance delivered the most:

- Produces increased compliance on recommendations by current clients
- Increase purchases of veterinary medical services
- Produces increased expenditures on pets by current clients.

There is tremendous medical and financial value to increased use of pet health insurance. According to the studies, dog owners with pet health insurance spent 29% more annually for veterinary care; cat owners 81% more. Mean annual expenditure on veterinary services for insured dogs was $324 v. $251 for non-insured dogs. Mean annual expenditure for insured cats was $264 v. only $146 for non-insured cats!

Contrary to an opinion sometimes voiced in veterinary circles, the vast majority of veterinarians do not fear that increased use of pet health insurance would lead to managed care. Ninety-five percent of veterinarians surveyed did not agree with the statement, “Pet health insurance companies will have too much influence in the veterinary profession if it becomes commonplace.”

For pet owners, especially those who were less familiar with pet health insurance, a recommendation from either their veterinarian or a veterinary practice staff member was highly influential in whether or not they would purchase a pet health insurance policy. According to the research, pet owners don’t necessarily expect their veterinarian to be an expert on policy costs and coverage. Rather, what they are looking for is reassurance that the veterinarian believes pet insurance is a valuable component of responsible pet ownership.

Interestingly, most pet owners who buy pet health insurance aren’t doing it for strictly economic reasons, according to the research. Rather, they are buying it for peace of mind, and a sense of security that they are doing the best for their pet. Among those pet owners who had pet health insurance, the most common reasons were:

- Is helpful to pet owners (59%)
- Shows you love your pet (55%)
- Shows you are a responsible owner (53%)
- Provides peace of mind (49%)
- Is a good investment (48%)
- Helps avoid the need to make painful choices about care (42%).

So how can veterinary practices foster more use of pet health insurance by their clients? Here are 10 ways.

Select one, or at most two, pet insurance companies to support

Pet owners are looking for a recommendation, not a whole catalog of choices. If there are brochures from four or five companies in the waiting room, the client won’t pick up any.

Designate one or two key staff members as the pet insurance liaisons, or go-to persons

Typically these would be full-time receptionists, hospital administrators and/or practice managers. These individuals could be the ones that research the various companies and pick one or two for the practice to recommend. Regardless, the designated pet health insurance liaison should be familiar with the recommended companies and able to answer basic questions.
Provide pet health insurance policies for one or more pets of the staff liaisons
This will insure that they are familiar with the company’s policies and claims submission forms. It also provides a perfect answer when the client asks, “Which company do you use?” Many pet health insurance companies provide discounted policies for veterinary staff members, and many practices insure at least one pet for each staff member.

Provide a link on the practice website to the pet insurance company or companies recommended by the practice.
This makes it easy for the client to obtain more information, and makes the practice the gateway to more information about pet health insurance.

Determine which clients already have pet health insurance
Each time a client schedules an appointment or visits the practice, ask if the pet has pet health insurance. Note in the patient’s record that it insured. Over time, this will provide a census of how many patients are insured. It will also help measure if the practice’s efforts to increase use of pet health insurance are gaining traction.

For uninsured pets, hand the client a brochure and encourage them to find out more
Tell them that the practice recommends that the owners of all healthy pets at least explore pet health insurance, and which company(ies) the practice recommends. Offer to answer any questions once the client has read the brochure and perhaps visited the website. Sick or injured pets may or may not qualify for pet health insurance until they are fully recovered and healthy once again due to preexisting conditions.

Note pet health insurance company and claim number in patient record
Recording that a patient is covered by insurance informs veterinarians and staff that treatment costs may qualify for reimbursement. This can facilitate decision-making when discussing options with the client. It’s also convenient to keep copies of claims forms with the pet’s policy number in the patient file. Your clients will appreciate the personal touch.

Submit claims
Practices that have the best success in fostering the use of pet health insurance volunteer to submit claims on behalf of clients. This is an added level of service deeply appreciated by client, and its makes it much more likely that claims will be filed in a timely manner. Further, when claims are submitted at time of service, its highly likely that the client will be paid promptly, in many cases before a credit card bill comes due. By working closely with just one or two pet health insurance companies, submitting claims can become routine and takes only a minute or two.

Recommend 30-day non-cost trial policies
Where allowed by state Departments of Insurance, some pet health insurance companies offer 30-day no-cost introductory policies. Clients like them. Veterinarians clearly like them too. If the pet insurance company you recommend offers no-cost trial policies, be sure to inform clients, especially new clients or existing clients with new pets. There may be time sensitivity, too. Generally, no-cost trial policies must be activated within one or two days of a veterinary visit to get full benefits. Consider having clients activate the introductory policy from their smartphone before they even leave the practice.

Engage the whole practice
Every employee from owner and associate veterinarians to kennel attendants should be enlisted in the campaign to increase use of pet health insurance. They need to be educated about the value of pet health insurance to their work and income, as well as the practice’s reasons for supporting pet health insurance. All should be encouraged to recommend pet insurance to clients when opportunities arise. Veterinarians and their staff members are the information resources most valued by pet owners. By actively recommending pet health insurance and providing a high level of service, every practice has the opportunity to increase the number of insured patients. While it may not be possible to get every client to purchase pet health insurance, each and every policy makes a positive impact on the pet’s health, the client’s compliance and peace of mind, and the practice’s success.
Level up your confidence: Identify the imposter voice and own your successes

Women professionals often exhibit lower confidence levels and the selection, training, and working environment of clinical veterinary medicine can encourage a sense of being an imposter and sabotage confidence. There is always someone close by who is more accomplished and successful than you (at least on paper). This lack of confidence leads to behaviors that further undermine achievement, and the behaviors that follow are significant barriers to women achieving “work-life balance” and leadership positions within veterinary medicine. More women veterinarians need to step up to the leadership table and recognizing the way in which the imposter syndrome may be affecting you—and how dealing with it can be one big step towards realizing your full potential.

Develop emotional resilience and combat compassion fatigue: Learn the difference between using your heart and your head

8th century Scottish philosopher Adam Smith and 19th century British anthropologist and sociologist Herbert Spencer wrote about perspective-taking as a "cognitive, intellectual reaction" and empathy as a "visceral, emotional reaction". It is important to understand that perspective-taking is exclusively the process of taking an alternate point-of-view, and has nothing to do with taking on the feelings of others.

Because this differentiation is commonly overlooked, perspective-taking is frequently confused with empathy. For this reason, the use of perspective-taking and empathy as synonyms is frequent within the scientific literature. The two are not the same, however.

You can have empathy but be lousy at perspective taking. If you find your perspective taking skills are lacking, you may find clients argumentative, uncooperative, and uncompliant.

For example, one can perspective-take a fellow individual’s thoughts and feelings. However, the perspective-taking process does not necessarily lead to feelings of empathy. Rather, that determination may be made after the perspective-taking process has finished. When veterinarians deal with so many emotions from so many people on a daily basis, it is critical to decide which client emotions you decide to get involved with, as you only have so much room in your psyche for all your own challenges and emotions plus that of others.

By reducing your power within an interaction, you can sharpen your perspective. When you’re in an encounter, assume you’re the one without power — because the research shows an inverse relationship between power and accurate perspective taking.

Second, don’t confuse perspective-taking with empathy. Both are important. But perspective-taking is very much a cognitive skill. You do it better by thinking about another person’s interests, not only understanding what emotions they’re feeling. Furthermore, too much empathy can submerge your self interests and that of the pet. Perspective taking is understand the needs, wants, desires, and core values of the client. Empathy is feeling those feelings. Learn the difference.

Eradicate procrastination and take control of your life

Most of the time, it’s not lack of experience that is holding us back, but rather the lack of determination to do what we need to do to be successful.

We can put so much energy into coming up with distractions or excuses as to why we can’t be, do, or have the things we want - what if we used that energy to go for what we want instead?

When we say we are unqualified for something, or don’t have time, what we are really saying is that we are too scared to try or it isn’t a priority. Procrastination is one of the easiest and sneakiest forms of self-sabotage.

Tips to live by

1. You know more than you give yourself credit for (no more imposter voice!)
2. You are drawn to things you are naturally good at.
3. Necessity is the best teacher.
4. Done is better than perfect.
5. Passion is stronger than fear.
6. Notice where you stop - and unblock!

- Mindfulness isn’t for Sissies: how meditation changed my life, and tips for developing and practicing meditation
- Mental Toughness: Tips from the Navy Seals and Long Distance Triathlon

Bite by bite, the elephant is gone

How do you eat an elephant? One bite at a time. Faced with a daunting task we feel overwhellmed, fight, flight, or freeze sets in, and we stop before we have even started.
Navy SEALs and triathletes break down any large task: divide the elephant into neatly digestible parts and eventually it will be gone. Triathletes focus on the next immediate objective—the next point in the horizon—and prevent their minds from passing to the entire race. Veterinarians can learn to use this technique to take on any large challenge - take it one tiny step at a time. Break down any daunting ask into immediate, bite-sized objectives. Ideally, these tasks should be able to be accomplished in a day - if you can’t finish it, make the task smaller, focus only on completing one at a time. To avoid the overwhelmed feeling, do not think about the whole project or task.

**Visualize success**
In a certain study, basketball players improved their free throw accuracy by 23% from just visualizing the free throws. Players who practiced actual free throws only improved by 24%. That’s only a 1% difference!

Helpful visualizations have the following qualities:

- Vivid and detailed. Imagine the particulars. Make it as real as possible. Imagine it so it feels good.
- Replay the image or scenario repeatedly, and try to go longer than a minute.
- Make sure you are focused on the positive aspects. For example, if you want to have a successful surgery, don’t say ‘I don’t want there to be complications.’ Instead, go for positive: ‘I want to control bleeding in this 10 year old pyometra and have the patient wake up routinely. Do not envision yourself failing. Instead, repeatedly envision yourself in a state of effortless success.

Application: The next time you have a big, stressful event coming up, use visualizations to imagine yourself succeeding.

**Emotional intelligence**
In times of great stress, stress hormones - adrenaline, cortisol, and norepinephrine - can boost focus, energy and potentially save our lives. However, when these hormones stay elevated for long periods when we are stressed all the time, it affects immune function, motivation, mood, and sleeping. We basically turn ourselves into cushingnoid dogs.

The SEALs simple solution is something called the 4 by 4 for 4 to turn off stress hormones:

- Breathe in for 4 seconds
- Breathe out for 4 seconds
- Repeat for 4 minutes

Application: This helps if you do meditation already, but the next time you catch yourself feeling stressed, stop and take several deep breaths. Bestselling author Tim Ferriss recommends stopping everything and taking a simple 3 breaths before going on with your day.

**Nonreactivity**
We have more control than we think. We can’t control what happens in our clinics, but we can control our interpretation of it. This is called ‘reframing’. Take one possible belief or worldview, discard it, and reach for a more positive one. What could have been interpreted as a negative situation could now be interpreted as positive.

Application: Take an active look at how you are interpreting external events. Once you recognize it, challenge that view. Try to reframe any negative views into more positive ones. Reach for any thought that gives you a sense of relief or positivity.
No matter how brilliant of a veterinarian you are, if you cannot understand the perspective of your clients, what motivates them, their core beliefs and fears, their generational values, then you can’t attain compliance, client loyalty, build your business, or practice successfully.

So how do we know who we are dealing with in the exam room? How do we find out what their needs, wants, and desires are? How do we deal when we have a totally different perspective of what the pet needs than the client?

The first step is to gain and respect the client’s perspective, and the best way to do that is develop a multi-dimensional perspective, which is the purpose of this session. In the session, we will play the ‘E Test’, which is a fun way to determine an individual’s ability to take perspective in a complex social situation - like the exam room, right?

Perspective taking should be pretty simple - take the opinions, ideas, and thoughts of others to be more important than your own, and you will automatically find it necessary to listen more carefully and take their point of view into consideration. It’s easier said than done, however, especially when you are very smart and have a ton of knowledge, and even harder when you are considered to be the expert. When it comes to perspective taking, smarts actually becomes a curse, because it becomes more difficult to take the perspective of the other when you already know the answer.

In this session, we will learn strategies on how to develop a multi-dimensional perspective to get inside your client’s head, and see the situation from his/her eyes. Understanding the client’s perspective better will help you understand why they are in your office, what their goals for the visit are, and most importantly, how to move them to action.

Furthermore, we will be using three video case studies to demonstrate our new found perspective taking skills. To learn more about the skill of perspective taking, I recommend reading ‘To Sell is Human’ by Daniel Pink. Also visit www.drsarahwooten.com for free handouts and exercises to try back at your hospital.
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 barriers to compliance, and simple communication tweaks that will strengthen your confidence, reduce confusion in the client, and instill a sense of goodwill between client and veterinarian.

The goals of the session: Review the AAHA compliance studies

- Teach small communication changes that will have big impact, for example:
  - The client and veterinarian need to be on the same page. Make sure the client understands what you are saying. Finish with ‘does this make sense?’
  - Learn to say ‘your pet needs’ vs. ‘I recommend’.
  - Draw parallels to human health issues to make the issue relevant and better understandable for the client.
  - Acknowledge the difficulty of the situation right away – establishes empathy and instills loyalty.
  - Learn to recognize fight, flight, and freeze in your clients and deal appropriately with each reaction.
  - Learn client needs for autonomy, certainty, and empathy.
  - Learn 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.
  - Stop using big words.
  - Use discharge instructions and handouts.
  - Give explicit and easy to understand reasons for follow-up.
  - Be sympathetic to client concerns for affordability, and learn to work with the client.
  - Develop your best euthanasia best-side manner. A good end inspires incredible client loyalty, and for good reason. This is hard.
  - Don’t assume that every client wants an in depth explanation.
  - Don’t assume that the client is not doing his/her best.
  - Give praise and positive affirmation to the client.
As veterinary practices have become more modern, sophisticated and technologically advanced, so has our ability to perform veterinary dentistry to a much higher level than was ever thought possible. Through specialization of the profession and a wider availability of these specialists, we are able to offer our clients’ referrals for more advanced care to board certified veterinary dentists.

As veterinary technicians and veterinarians we need to be completely aware of what kinds of dental care and treatments are available, and when to offer a referral instead of opting for more basic dental care in hospital.

The primary concern that we often see in dogs and cats is periodontal disease; however if teeth can be salvaged instead of extracted through periodontal surgical techniques and home care, then through these treatments we could benefit the patient over the long term, to retain important teeth for their function.

**Dental radiographs**
Radiographs must be obtained to fully assess the extent of any suspected bone loss. Evaluation of a full set of intraoral dental radiographs will help determine the success of any proposed advanced dental procedure, as well as give the veterinarian a baseline to monitor the progress of treatment. If your veterinary practice does not have the ability to obtain those dental radiographs and the client is interested in advanced dental care and saving teeth rather than extraction, then considering referral from the onset may be in the best interest of the patient.

**Advanced periodontal therapy**
Larger and more important teeth can be difficult to extract even with significant periodontal disease, which can result in horizontal or vertical bone loss, furcation bone loss and tooth mobility due to loss of attachment. When we look at teeth through clinical observations and measurements as well as radiographically, we must assess the true extent of the pathology. A tooth can be evaluated on a root by root basis as well as an individual side of each tooth root. A tooth with significant bone loss (>50%) on a tooth root’s surface may have a very poor prognosis even with advanced periodontal surgery, especially if the bone loss is all the way around the root or what is called a four-walled defect.1, 3 The area in between a multi-rooted tooth’s roots is called a furcation and if the bone is lost from this area it reduces the success of an advanced procedure even further.1, 3

**Total attachment loss**
This is the sum of the measurement of any gingival recession on the root’s surface, as well as any pocket depth beyond that gingival recession. If gingival recession is not present then it is just the measurement of any periodontal pocket depth beyond what may be considered to be a normal sulcular depth for that specific tooth, in that specific pet’s mouth. This differs depending on the size of the animal, size of the tooth and length of the tooth root specifically.

In order to measure total attachment loss you must use a periodontal probe with clearly marked 1mm increments and measure from the marginal gingival edge to the bottom of the sulcus or periodontal pocket if there is attachment lost.1, 3 The bottom of the sulcus is normally attached to the tooth’s surface at or very near to the cementoenamel junction (CEJ).1 When attachment is lost at this point a periodontal pocket is created and a pathological process begins. The periodontal probe should be used with a gentle hand, in line with the vertical axis of the tooth and walked around the tooth’s structure recording measurements in at least four places around each tooth root. Whenever these measurements are greater than what would be considered a normal sulcular depth around that particular tooth, the measurement should be recorded on the patient’s dental chart.1

Conditions such as gingival enlargements further diagnosed by histopathology as gingival hyperplasia, can create a false pocket depth and not true attachment loss so careful measurement of the excess gingival tissue and noting if the bottom of the sulcus is at the CEJ is important to determining the extent of attachment on these teeth.1

If the bone loss or total attachment loss is <50% and there is not significant furcation involvement, or less than a four walled defect, it may be possible for advanced periodontal surgical techniques, frequent follow up care (possibly under anesthesia) and daily homecare which is a commitment that the client must make when attempting to “save” important or strategic teeth.

If a periodontal pocket depth exceeds 5mm, it is recommended that open root planing: RP/O (root planing-open) be performed with the use of flap surgery to facilitate the visualization of the bony defect and exposed root surface and allows the practitioner to treat the area to the best of their ability to get the best possible outcome from periodontal therapy.1

If the periodontal pocket depth is less than 5mm, root planing-closed can be performed (RP/C). This technique involves the use of a hand curette instrument below the gingival margin, adapted to the surface of the root that requires cleaning. Using the sharp blade of the curette, we want to carefully remove the bacterial laden debris from the cementum of the root surface. Thus improving the health of the local periodontal tissues and smoothing the rough root surfaces allowing the re-attachment of the periodontal ligament as possible.
The use of any curette involves four basic steps:

1. Holding the curette in a modified pen grasp, create a fulcrum by placing your ring finger near the tooth area to be instrumented but not in the “line of fire” to avoid the blade cutting your finger.
2. Insert the curette with the face of the blade in the “closed position” face towards the tooth root, this allows for adaption of the curette beyond the calculus below the gingival margin.
3. Rock blade handle so as to bring the terminal shank into a parallel position to the root, thus engaging the sharp edge of the blade into the root surface.
4. Working stroke: pull the instrument in either a vertical direction towards the crown tip, oblique direction across the crown or horizontal direction.
5. Readapt and repeat the motions in overlapping strokes to ensure the cementum of the root is free from bacterial laden debris and smooth to the touch of the instrument.

**Periodontal bactericidal ultrasonic debridement**

The final step in ultrasonic cleaning. A specially made periodontal tip insert is required for this procedure or some dental ultrasonic units are already equipped with a tip that can be safely inserted sub-gingivally. Please consult your ultrasonic equipment manual regarding which tips are safe to insert under the gum line into the sulcus, and at what setting the machine should be turned down to, reducing the frequency of vibrations to a safe level for this purpose.

Periodontal bactericidal ultrasonic debridement occurs due to the ultrasonic sound waves causing microscopic bubbles to form and then implode in the gingival sulcus, cavitation. These implosions can cause the bacterial cell walls to be disrupted and along with the water rinsing through the area at a certain pressure further reduces the concentration of bacteria within the space.1

**Advanced periodontal flap surgeries**

Techniques to perform flap surgeries are fully described in several dental text books and can be learned by veterinarians at wet labs taught by veterinary dentists on the subject, however if surgical procedures are indicated that are beyond the practitioner’s skill level then referral may be the preferred option.

**Apically repositioned flap**

This technique can be used to help attached gingiva lay over any remaining alveolar bone, it requires that there is at least 2mm of gingiva to extend towards the crown.1 This surgery moves the gingiva down onto the root surface after the area is cleaned of unhealthy bone, granulation tissue and debris; and then the area is allowed to heal.2 This procedure can be performed on mandibular incisors to allow for a reduction in periodontal pocket depths, allow for daily cleaning by the client and to allow easier cleaning of areas of furcation exposure on multi-rooted teeth.2

Contraindications for this procedure would be >50% bone loss especially on a four-walled defect, grade three (3) tooth mobility and the presence of less than 2mm of attached gingiva before surgery.1

**Laterally positioned (pedicle) flap**

**Indications**

When the root surface of a single tooth is exposed significantly due to a cleft that extends to or near the mucogingival line.1

**Contraindications**

Tooth mobility due to loss of bone on more than one wall of the alveolar socket, furcation bone loss or lack of commitment on the client’s part for daily homecare and more frequent follow up professional dental care.1

Carefully created and planned vertical releasing incisions, and the creation of a donor flap which is moved laterally over the area and sutured, is required for this technique.1 The goal is to partially cover this exposed root surface and allow for at least 2mm of attached gingiva to help preserve the health of this particular tooth, the area of tissue that is exposed from the donor site will heal in by second intention.1, 3

**Free gingival graft**

Indicated in specific individual teeth with a cleft like defect that are free of endodontic disease and tooth mobility is not present.3

Contraindications if endodontic disease is the cause and endodontic disease is not treated first.3 Concurrent periodontal disease must be treated and controlled, if there is tooth mobility the success of this technique will be poor.3 Success will also depend on the client’s willingness to perform daily recommended homecare and follow up treatment with the veterinarian.3

In this procedure a gingival graft is obtained from a donor site separate from the site to be treated and often on the buccal surface of attached gingiva over the maxillary canine, this site offers the largest expanse of tissue.3 The donor graft is carefully harvested using a template and careful technique is used to avoid damage to the periosteum under this split thickness of tissue.3 The donor tissue is then used to graft over the recipient site with careful surgical techniques that are fully described in dental surgical texts.3

**Guided tissue regeneration**

The goal of this type of advanced periodontal therapy is to help facilitate the development of cementum on the root’s surfaces and the regeneration of healthy periodontal attachments.1 Barrier membranes that are either absorbable or non-absorbable are specifically
positioned to prevent granulation tissues from invading the area and to allow bone and periodontal ligament cells to develop in the area where they have been destroyed by periodontal disease.¹

The use of bone inductive materials can assist in such procedures where significant bone has been lost in two and three walled bony defects, and areas of class two (F2) furcation bone loss in multi-rooted teeth.

**In summary**

Basic uses of dental hand curettes, as well as knowledge of dental, periodontal anatomy and treatment techniques that will help regain attachments and healthy periodontium, are very important to the improvement of oral health in our companion animal patients. Further techniques for periodontal therapy using these hand instruments, should be pursued in a dental lab format as well as further reading on the subject.

**References**


Dental instruments range from power instrumentation such as that of the dental delivery unit which may house many different handpieces such as the high-speed, low-speed, air-water syringe, the ultrasonic scaler and maybe even suction. A stand alone motor pack can be used for low-speed work and you can also use a separate ultrasonic scaler unit instead. Fine tipped sharp hand instruments are also an important part of thorough and complete veterinary dentistry.

These items will all need care and maintenance to ensure their proper function, safe use and longevity. The veterinary dental technician should perform daily safety checks on the power equipment, provide the maintenance that is recommended by the manufacturer’s guidelines and clean the equipment in between patients to prevent cross-contamination and ensure infection control.

Handpieces
High and low-speed dental handpieces need care and maintenance. These are the hand held air or motor-driven pieces to which we attach the prophy angle, dental burs for cutting and drilling or contra-angles or reduction gear angles to do more advanced dental procedures. The use of an approved lubricant is recommended by most manufacturers; it may be in the form of a spray or liquid. This lubricant is placed in the smaller of the two holes in the bottom of the handpiece, and then the handpiece is reinserted and screwed on to the cord and then operated for 20-30 seconds to distribute the lubricant into the working parts in the hand piece.

This should be done as often as recommended by that particular handpiece’s manufacturer.

The dental delivery system may require that dilute bleach and water solution or a manufacturer approved solution be run through the water lines in the system without the handpieces attached to remove the bio-film or bacteria that builds up in the water containers and the water lines. Check with the manufacturer before you perform this kind of maintenance for the unit’s specific requirements.

Clean the outside of the hand pieces with 70% isopropyl alcohol to remove debris, then the hand pieces may be autoclaved if desired.

Scaler tips
The scaler tip should also be cleaned to remove debris and then can be autoclaved to ensure sterility in between patients. Each scaler tip comes with a guide that will help you determine when the tip needs to be replaced due to wear down. If a tip loses even 2-3 millimeters of length, it is much less effective than it should be. Piezoelectric type scaler tips are less expensive than the magnetostrictive stack type inserts, however due to the higher frequency that the piezoelectric tip operates at, wear down will occur and it will need to be replaced much more often.

Ferrite rod inserts are easily broken if accidentally dropped so the hospital should always have an extra replacement rod on hand if you are using this type of scaler.

The leaves of nickel alloy in the magnetostrictive type insert should be inspected daily for any fractures in the stack or separations at the base of the insert. If these are detected, the insert will need to be replaced. It is a good idea to have an extra insert on-hand to replace a broken or damaged insert without interrupting the quality of patient care during a procedure.

Fine hand instruments
Scalers and curettes require daily care and sharpening to maintain a useful instrument. The fine metal blade edges of these instruments should be kept sharp at the proper angulations and undamaged. Damage can occur due to dropping the instrument, improper use of the instrument or incorrect sharpening techniques.

It can be very helpful to have a new instrument kit handy for a quick comparison when you are discerning if an instrument is damaged, incorrectly shaped or angled at the working end. You can quickly replace an instrument that has been damaged or worn beyond repair with these extra instruments without having to wait for an ordered instrument to arrive.

The parts of the scaler and the curette
When holding an instrument to locate the parts of the instrument, first locate the terminal shank and orient it so that the terminal shank is perpendicular to the floor, the face of the instrument is up towards the ceiling and the toe is pointing towards your nose when you look at the instrument. When holding a curette this way, the handle will often be angled out to one side or the other when the terminal shank is perpendicular to the floor.

- **Handle:** This is the part of the instrument on which we place our thumb and fingers for the main grip to hold the instrument. Different sizes, weights and textures are available. Dental scalers and curettes are double ended so the handle is in the center of the instrument extending on each side to the instrument’s working ends.
- **Shank**: This is the next portion of the instrument on each end. It is made of metal and depending on the type and use of the instrument.
- **Terminal/Distal Shank**: Farther down toward the working end, the terminal shank is where the actual working end of the instrument attaches on each end of the double-ended instrument.
- **Blade**: The working end or tip of the instrument. It is a mirror image of the opposite end.
- **Face**: The top surface of the blade end of the instrument.

**Gracey Curettes** are angled at 70 degrees from the terminal shank and have only one cutting edge on the lower side.

**Graceys have an offset attachment at 70 degrees and only have one cutting edge on the lower side.**

**Dental Scalers** have a triangular shape to the working end and a pointed sharp tip.

**Graceys have an offset attachment at 70 degrees and only have one cutting edge on the lower side.**
Instrument sharpening: Goals

1. Keeping the instrument’s cutting edges sharp enough so that the operator can be effective with each working stroke at removal of calculus without a tremendous effort and to avoid burnishing the calculus on to the dental surfaces.
2. Maintaining the instruments integrity, original shape and function as much as possible.

Scalers that are to be used on the coronal surfaces above the gingival margin have two blade cutting edges, one on each side of the working end. The tip of the scaler is not blunted but kept at a sharp point; just the sides of the instrument’s working end are to be sharpened.

Curettes are to be sharpened in a similar manner, however care should be taken to make sure that each cutting surface is sharpened at the proper angle and that the tip or “toe” of the instrument is “blunted” or rounded off at each sharpening to prevent obtaining a sharpened tip that would not be appropriate to insert subgingivally because it would lacerate sulcular attachments.

There are two main methods used for sharpening instruments with a sharpening stone:

1. Moving stone-stationary instrument method
2. Stationary stone-moving instrument method

The choice of method depends on the operator and what they find easiest to use.

- Required items for instrument sharpening: Coarse and fine stones
- Arkansas stone (fine grit): This is a fine grit stone that is used for instrument maintenance and finishing or after re-contouring with a course grit stone if that is necessary. This stone does not remove as much metal when used so it is not appropriate for use when an instrument is very dull or needs to be re-contoured.
- India stone (medium grit): These stones are more coarse in grit and helpful when more metal removal is required to re-contour or sharpen an excessively dull instrument.
- Honing stone oil: Both types of stones require that special honing stone oil be placed on the face of the stone before sharpening and gently wiped into the crevices of the stone. Be careful not to remove all of the oil when spreading it across the stone with a finger or paper towel. When the oil is on the stone it protects the stone from the metal shards embedding in the stone and shortening the life of the stone, and the oil reduces friction.
- Ceramic stone: (Not required): Ceramic stones are not as commonly used in sharpening veterinary dental instruments, however if this stone is what you have available to use, it is a fine grit stone (not as useful for reworking or reshaping a dull instrument) and requires water as a lubricant not honing oil.
- Care and disinfection of the sharpening stones: Stones can be autoclaved if desired, however instrument sharpening should take place after the instrument has been cleaned with water and an approved cleaning solution, dried and autoclaved. The heat from the autoclave will dull metal instruments so sharpening after autoclaving is recommended. Autoclaved instruments used on the sharpening dental stones should not contaminate the dental stones which should eliminate the need for autoclaving the stones unless they are used to sharpen instruments during dental procedures.
- Stone shapes and sizes: The most common size for the flat Arkansas stone is one inch wide by four inches long. This is a good size for most people to hold in their hands when sharpening. The India stone should also be a flat type of stone, larger in size; approximately 1 ½” X 4 ½” long with a rounded side and a contoured sloping edge on the other long side. Using a conically shaped Arkansas stone on the face of the instrument will be the last quick step in the sharpening procedures. All stones are easily broken if dropped so care should be taken to ensure a firm grip on the stone and the stones should be kept in a cushioned container when not in use to prevent damage.
- Acrylic test stick: This round short piece of plastic about the size of a straw is used to “test” the hand instrument’s cutting edges for sharpness both before and after the sharpening procedure. The cutting edge of the instrument should “bite” into the acrylic stick when engaged slightly. It should not bounce off the test stick.

Moving stone-stationary instrument method

1. Hold the instrument very steadily on the edge of the counter top with the terminal shank perpendicular to the floor at 12 o'clock and the tip or toe pointing towards the operator.
2. Immobilize the instrument by bracing the instrument on the edge of the counter and resting your lower arm on the counter top.
3. Hold the oiled stone face towards the side of the working tip of the instrument at approximately 70 degrees or at 1 o'clock or 11 o'clock. The stone is then moved using an up and down motion, ending on a down stroke to sharpen the cutting edge of the instrument. Sharpening should be done by moving from the heel of the instrument at first and then more towards the tip or toe of the instrument last.
4. When sharpening a curette with a blunted toe the operator must take the stone and bring it forward in an up and down motion around the toe at a 70 degree angle to make sure it is blunted and not sharpened into a point. Always end on a down stroke.
Universal curettes have a cutting edge on both sides of the instrument so you can keep the instrument in the exact same position and just move the stone to the opposite edge and repeat the process on that cutting edge.

1. The conical stone is then oiled and rolled over the face of the instrument a couple of quick times to remove any metal filings off of the cutting edges or face of the instrument.

2. The instrument is then turned over, repositioned, stabilized and the stone is repositioned in the other hand so that the cutting surface(s) on the other end may be sharpened in a similar way.

If you do not have a guide to help you position the instrument, then the terminal shank should be as close to lined up with 12 o’clock (90 degrees) on an imaginary clock face as possible. The angle off of that for the stone should be at or near 70-80 degrees for universal curettes and scalers but not for Gracey curettes. Gracey curettes are off-set already at 70 degrees so you will need to line the stone up with the one cutting surface at or near 50-55 degrees and only sharpen the lower side not both sides.

A protractor or a paper guide can be tremendously helpful in visualizing the above angles.

Methods of sharpening dental elevators and luxators

Usually a stationary stone-moving instrument technique is employed to sharpen these tools.

It is very important for the instrument's working end to maintain its shape and integrity. Using a method that sharpens the back side of the edge of the instrument will avoid removal of the wings on a winged elevator and prevent thinning of the instruments cutting surface which could weaken the end of the instrument over time.

1. Place stone on table and hold stone with one hand to keep it stationary. Drop a couple of drops of sharpening oil on the stone and smooth this over the surface.

2. Place side of working end on stone, pointer finger on top. Handle at table top. Holding instrument firmly in hand.

3. Raise handle to 45 degrees off of the table.

4. While rotating the working end under in a fashion on the stone that draws a "smile" on the stone. Rotate palm up and sharpen all the way around the back side edge of the elevator or luxator, then go back over the "smile" to the beginning position.

5. Use care not to press down too hard when you start the movement, when you get to the palm up position with the pointer finger under the instrument you will automatically not be putting as much force on the stone with the instrument.

6. Maintain the 45 degree angle off of the stone's surface through-out the whole movement.

7. After several passes across the stone in the "smile" format make sure to take the conical stone and run it through the face or concave side of the working end a couple of times to remove the "wire-edge" that will accumulate on the instrument.

8. Use your magnification to visually inspect the convex surface to insure even sharpening.

9. Use the Arkansas stone for daily sharpening and the India Stone for more aggressive sharpening and fixing metal spicules that may be on the instruments edge from misuse.

Mechanical sharpening devices

There are a few very nice mechanical sharpening devices available. Though the mechanical sharpeners are more expensive initially, they have the advantage of taking the positioning guess work out of the technique to produce consistently sharp and correctly angled instruments.

- Rx Honing Machine® (This machine can even sharpen scissors!)
- http://www.rxhoning.com/sharpening-sets/
- Side-Kick® by Hu-Friedy® www.hu-friedy.com

References
Oral Anatomy, Pathology and Charting for Veterinary Technicians
Benita Altier, LVT, VTS (Dentistry)
Pawsitive Dental Education
Prosser, WA

Oral anatomy and pathology
Before we can recognize what may be abnormal we need to have a full understanding of what is considered normal anatomy in the canine and feline patient’s mouth. Dental disease can affect the patient throughout the entire body due to bacterial shed from oral infection spreading through the bloodstream and affecting vital body systems and organs over the long term. A general physical exam and pre-anesthetic blood and urine testing and any other testing recommended by the veterinarian should ideally be performed prior to anesthesia to further assess the overall health and anesthetic risk for the veterinary dental patient.

Oral anatomy can be divided into soft tissues and bony or hard tissues. As we assess the veterinary patient we will assess both types of structures for any abnormalities. It is a normal tendency to focus on looking inside the patient’s mouth and focusing on the teeth when we are looking for oral disease; however we need to start by examining the patient’s head, skull and facial areas first and then moving on to the inner oral cavity.

Patient examination
On the conscious patient we want to begin by visually observing the head and face of the patient.
- Do we see any areas of swelling, inconsistencies or imbalances?
- We should notice the eyes of the patient, are they protruding out of the orbital sockets, and is there any ocular discharge, masses or drainages around the eye margins? Are the pupils dilated or constricted in a bi-lateral manner?
- Does the patient tilt the head to one side or the other?

Once a visual inspection of the head and neck has been performed then we can move on to palpate the structures of the skull and face.
- Palpate the bones of the skull, above and below the eye areas, over the cheeks and zygomatic arches.
- Palpate the mandibular bones from the mandibular symphysis to the caudal edge of the mandible and continuing on back to the temporamandibular joint on each side.
- Palpate the lips on all sides assessing for any swellings or signs of pain or discomfort.
- Palpate the mandibular lymph nodes and any other regional lymph nodes in the area.
- A visual inspection of the ear canals with an otoscope is an important part of the overall skull area exam.
- Does the patient respond appropriately to simple neurological tests done on the face and head?
- Assess the patient’s occlusion. This must be documented while the patient is either awake or just after induction of anesthesia prior to the placement of an endotracheal tube so that the mouth can be closed completely without any obstructions.

These same parameters should be re-examined once the patient is properly anesthetized, intubated and at a safe plane of anesthesia for examination. A much more thorough oral examination will take place at that time.

Inside the patient’s mouth we can further divide this area into two regions; the first region encompasses all of the soft tissues of the lips, which inside the mouth are covered by oral mucosa, called the buccal mucosa, which consists of stratified squamous epithelium.1 This area, called the vestibule, continues until it reaches the area demarcating the beginning of attached gingiva known as the mucogingival margin.1 The second region begins at the attached gingiva and extends medially towards the hard palate on the maxilla and from the mucogingival margin on the mandibles medial towards the tongue, this is called the oral cavity proper.

Anatomy of the teeth and surrounding structures
- Alveolar Bone: This is not the name of a particular bone but more of a descriptor of the bone that surrounds and supports the tooth structures in the mouth.
- Alveolar Jugum: This is the palpable alveolar bone that overlays a large tooth root such as the maxillary canine tooth.
- Alveolar Margin: This is the most coronal edge of the alveolar bone that surrounds the teeth; it consists of very dense cortical bone.2
- Attached Gingiva: Covering the alveolar process this attached gingiva which is highly keratinized can withstand the forces of mastication.2
- Cementum: Serving as a protective covering for the tooth root and a surface for the periodontal ligament fibers to attach, this calcified mesenchymal tissue is avascular in nature.2
- Cementoenamel Junction (CEJ): This is the junction where the enamel from the crown surface of the tooth and the cementum that covers the root surface of the tooth meets.
- Crown: This is the portion of the tooth structure that is supragingival or above the gingival margin when erupted. It is covered with a very hard substance called enamel.
• **Dentin:** This substance consists of hard calcified tissue containing tubules and makes up the greater volume of the inside of the tooth. Overtime the dentin becomes thicker as the patient ages. Dentin surrounds the pulp chamber and root canal’s blood and nerve supply and fuses with the cementum on the inside of the tooth root and the enamel on the inside of the tooth crown.

• **Enamel:** This is the smooth and shiny surface that covers the coronal aspect of the tooth and serves to protect the dentin from environment of the oral cavity. It consists of hydroxyapatite crystalline compounds and is the hardest substance in the body. Once the enamel is destroyed or lost it cannot be regenerated.

• **Free Gingival Margin:** The most coronal edge of gingival tissue and circumnavigates the tooth crown and forms the gingival sulcus. It is not attached to the tooth’s surface.

• **Gingival Sulcus:** This is the area that lies between the marginal gingiva that is resting against the crown of the tooth but is not attached and the tooth crown itself. We use our periodontal probe to explore the gingival sulcus for loss of attachment at the bottom of this sulcus where the normal attachment should be.²

• **Junctional Epithelium:** At the base of the gingival sulcus this tissue is the beginning of the attachment of the attached gingiva to the alveolar bone. This should be located at or near the cementoenamel junction. Care should be taken not to destroy this attachment when cleaning the patient’s sulcus.

• **Mucogingival Junction (MGJ):** The line that demarcates the transition from the attached gingiva to the alveolar mucosa.²

• **Odontoblasts:** These cells line the pulp cavity and produce dentin throughout the tooth’s life which causes the dentinal walls to thicken as the tooth ages.² This decreases the size of the pulp canal over time.²

• **Occlusion:** The positional relationship between the maxillary teeth and the mandibular teeth when the mouth is in a fully closed position.

• **Periodontal Ligament (PDL):** These ligaments attach the tooth via the cementum on the outside of the tooth root in many directions to the bone that lines the tooth’s socket. Consisting of cells, collagen fibers and nearly 70 percent water these ligament fibers play a significant role in the tooth’s capability to withstand the daily forces of chewing.

• **Periodontium:** The periodontium are the structures that support and surround the teeth themselves. It consists of the alveolar bone, attached gingiva to the mucogingival junction, the periodontal ligament fibers which attach the tooth root to the tooth socket and also the cementum which is the outside covering of the tooth root.

• **Pulp Cavity:** Containing the highly vascular and nerve tissues; this cavity can be further divided into the root canal in the root section of the tooth and the pulp chamber in the crown section of the tooth.

• **Root:** This is the portion of the tooth structure that is below the gum line or subgingival, the root is covered with a substance called cementum and should be firmly attached to the alveolar socket by periodontal fibers. These periodontal fibers run from the alveolar bone to the cementum on the root surface acting like shock absorbers when the pet masticates and the teeth need to move in the socket slightly as this process of chewing occurs.

• **Dental Directional terms:**

  • **Mesial:** This refers to the tooth surface that is directed toward that of the first incisor in the quadrant.

  • **Distal:** This surface is the surface that is opposite to the mesial surface.

  • **Lingual:** This is the tooth surface that is closest to the tongue.

  • **Palatal:** This is the surface that is closest to the palate. Usually used with maxillary teeth only.

  • **Caudal:** Refers to structures or moving in the direction towards the back of the mouth.

  • **Rostral:** Refers to structures or moving in the direction towards the front of the mouth.

  • **Apical:** In the direction of the apex of the tooth root.

  • **Coronal:** In the direction of the tooth crown.

  • **Labial:** Surface towards the lips of the patient.

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Note:

On a specific tooth if we are discussing the direction towards the central incisor then we are referring to the mesial direction. If we are referring to the direction on a specific tooth moving away from the central incisor in that quadrant then we are moving in a distal direction. Caudal and rostral refer to general directions of movement within the oral cavity not directions specific to a single tooth.
Charting the patient’s oral structures
Now that we have a better grasp on how to properly do an orofacial exam on the conscious and also anesthetized patient we need to understand the techniques involved and methods of proper documentation of the findings on a dental chart using the American Veterinary Dental College’s (AVDC) www.avdc.org, method of abbreviations and notations.

It is important to have and use a complete dental chart that allows us to document findings in a three dimensional way to create an overall picture of all sides of the tooth and root structures below and above the gum line. Having a pre and post treatment image of the mouth structures is also an important step to help follow treatment over time and to ease the documentation and readability of the chart.

The chart should also have a place to document the patient’s signalment, hospital and practitioner information, date of charting and treatment, skull type, occlusion, periodontal disease, plaque, calculus, gingivitis and notations regarding regional anesthesia.

Instrument required for charting
Periodontal probe and explorer
This instrument on one end is used for measuring periodontal pocket depths, loss of gingival attachments, extent of gingival hyperplasia and furcation bone loss. A shepherd hook explorer is used as a tactile instrument to further assess for any defects in the enamel or exposed dentin surfaces, the cementoenamel junction and/or exposed root surfaces if any. A probe and shepherd hook explorer is often a double ended instrument.

Charting should be performed in a systematic and consistent fashion each time. The practitioner should start at the midline and working in quadrants document all findings as thoroughly as possible, noting all abnormal findings on the chart at their location using the buccal, coronal or palatal/lingual view to illustrate. Using the AVDC abbreviations as necessary to document certain conditions and findings, further defines our charting.

Once the dental chart has been completed for the pre-treatment pathology then a plan can be made for further diagnostics and treatment of the patient’s oral cavity. Further diagnostics such as radiographs, will assist in the proper documentation of structures that we cannot visualize below the gingival margin.

If a tooth appears to be missing because the crown is not present in the mouth, a radiograph should be obtained and if the root structure is missing then the whole tooth can be circled as missing on the chart. If there is root structure still present then only the crown of the tooth should be circled on the chart. If a tooth is unerupted then it should be documented on the chart within the bone in which it resides.

The patient’s dental chart is a part of its permanent medical record and should be further documented with a written account of all pathology, diagnosis, radiographic interpretation, treatment or procedures performed, prognosis, client discussions, homecare recommendations, follow up care recommended and a long term plan for management and prevention of oral disease for that particular patient.

During the procedure a proper anesthesia record should be maintained including all drugs, doses and methods used for the general anesthesia, analgesia and any other drug therapy. A record of all anesthesia monitoring as it is documented should be kept during the procedure as well.

AVDC abbreviations
These abbreviations help us to document specific findings easily and allows us to communicate within the veterinary profession about these findings through a defined understanding of what each abbreviation means.

It is important to have an understanding of the differences in using the words: stage, index and grade. Each of these has a different meaning when we are discussing disease.

- Stage: This is used when we are discussing the extent of pathological lesions in a course of a disease that is likely to progress.
- Grade: The quantitative assessment of the degree of severity of a disease or abnormal condition at the time of diagnosis, irrespective of whether or not the disease is progressive.
- Index: The quantitative expression of predefined diagnostic criteria whereby the presence and/or severity of pathological conditions are recorded by assessing a numerical value.

Below is a list of the most commonly used abbreviations in the general practice of veterinary dentistry.

Triadan numbering system
100=maxillary right  200=maxillary left  300=mandibular left
400=mandibular right

*Deciduous teeth are noted 500-800 starting at the animal’s upper right quadrant and moving clockwise.

Dogs have 28 deciduous teeth and 42 adult teeth
Cats have 26 deciduous teeth and 30 adult teeth

Remember the rule of 4’s and 9’s. 4’s=canines and 9’s =first molars
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Diagnostic</th>
<th>Treatment</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>AB</td>
<td>Abrasion</td>
<td>Alveolectomy/Alveoloplasty</td>
<td>Object caused tooth wear</td>
</tr>
<tr>
<td>ALV</td>
<td>Alveoloplasty</td>
<td>Bone removal or contouring alveolus</td>
<td></td>
</tr>
<tr>
<td>AT</td>
<td>Attrition</td>
<td>Tooth on tooth wear</td>
<td></td>
</tr>
<tr>
<td>ATE</td>
<td>Extrusion</td>
<td>Abnormal tooth extrusion</td>
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<tr>
<td>B</td>
<td>Biopsy (see specific types)</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
<td></td>
</tr>
<tr>
<td>CA</td>
<td>Caries</td>
<td>Carious lesions</td>
<td></td>
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<tr>
<td>CB/C</td>
<td>Crossbite caudal</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
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<tr>
<td>CB/R</td>
<td>Crossbite rostral</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
<td></td>
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<tr>
<td>CEJ</td>
<td>Biopsy see specific types</td>
<td>Cementoenamel junction</td>
<td></td>
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<tr>
<td>CL/B/L/T/P</td>
<td>Chewing lesion</td>
<td>Buccal/labial/sublingual-tongue/palatal</td>
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<tr>
<td>CR/A</td>
<td>Crown Amputation</td>
<td>crown amputation/intentional root retention</td>
<td></td>
</tr>
<tr>
<td>CU</td>
<td>Contact Mucositis</td>
<td>contact mucosal ulceration</td>
<td></td>
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<tr>
<td>D</td>
<td>Diastema</td>
<td>open space between teeth</td>
<td></td>
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<tr>
<td>DT/P</td>
<td>Deciduous Tooth</td>
<td>Deciduous tooth/persistent</td>
<td></td>
</tr>
<tr>
<td>DTC/R</td>
<td>Dentigerous cyst</td>
<td>cyst defect around unerupted tooth</td>
<td></td>
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<td>E/D</td>
<td>Enamel Defect</td>
<td>involves only the enamel</td>
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<td>E/H</td>
<td>Enamel Hypoplasia</td>
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<td>E/HM</td>
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<tr>
<td>GC</td>
<td>Gingival Curettage</td>
<td>Curettage of gingival lining only</td>
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<tr>
<td>GE</td>
<td>Gingival Enlargement</td>
<td>in the absence of a histopathology dx</td>
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<tr>
<td>GR</td>
<td>Gingival Recession</td>
<td>measured in millimeters</td>
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<td>IO/R-repair</td>
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<tr>
<td>OAF</td>
<td>Oroantral fistula</td>
<td>OAF/R-repair</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
</tr>
<tr>
<td>OM</td>
<td>Oral Mass</td>
<td>Oral or maxillofacial mass: see <a href="http://www.avdc.org">www.avdc.org</a></td>
<td></td>
</tr>
<tr>
<td>ONF</td>
<td>Oronasal fistula</td>
<td>ONF/R-repair</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
</tr>
<tr>
<td>PD 1/2/3/4</td>
<td>Periodontal disease</td>
<td>Stages of Periodontal disease</td>
<td></td>
</tr>
<tr>
<td>PRO</td>
<td>Professional dental cleaning</td>
<td>Scaling/polishing/irrigation</td>
<td></td>
</tr>
<tr>
<td>RCT</td>
<td>Root canal therapy-standard</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
<td></td>
</tr>
<tr>
<td>RP/C, RP/O</td>
<td>Root planing Closed/Open</td>
<td>Root planing without or with visualization</td>
<td></td>
</tr>
<tr>
<td>RTR</td>
<td>Retained tooth root</td>
<td>tooth root remains</td>
<td></td>
</tr>
<tr>
<td>ST</td>
<td>Stomatitis</td>
<td>ST/CS: Caudal stomatitis</td>
<td>See <a href="http://www.avdc.org">www.avdc.org</a></td>
</tr>
<tr>
<td>T/A, T/LUX</td>
<td>Fractured tooth</td>
<td>Avulsed or Luxated tooth</td>
<td></td>
</tr>
<tr>
<td>T/FX</td>
<td>Fractured tooth</td>
<td>See additional handout</td>
<td></td>
</tr>
<tr>
<td>T/NE</td>
<td>Near pulp exposure</td>
<td>See tooth fracture classifications</td>
<td></td>
</tr>
<tr>
<td>T/NV</td>
<td>Non-vital tooth</td>
<td>Tooth ceased to mature-dead tooth</td>
<td></td>
</tr>
<tr>
<td>T/SN</td>
<td>Supernumerary tooth</td>
<td>Extra tooth in the quadrant</td>
<td></td>
</tr>
<tr>
<td>T/SR</td>
<td>Supernumerary root</td>
<td>Tooth has atypical extra root</td>
<td></td>
</tr>
<tr>
<td>T/U</td>
<td>unerupted tooth</td>
<td>Tooth did not erupt-remains in bone</td>
<td></td>
</tr>
</tbody>
</table>
Classifications of tooth fractures

www.avdc.org

EI: Enamel Infraction (Craze lines)
EF: Enamel Fracture (enamel only)
UCF: Uncomplicated Crown Fracture (Does not involve the pulp/crown only)
UCRF: Uncomplicated Crown/Root Fracture (Does not involve pulp)
CCF: Complicated Crown Fracture (Pulp is open)
CCRF: Complicated Crown and Root Fracture (Open pulp both root and crown)
RF: Root Fracture

Tooth resorption

Type 1: Traceable periodontal ligament space, roots not resoring
Type 2: Periodontal ligament space mostly absent, roots are resorbing (root by root basis)
Type 3: Combination of type 2 and type 1 in same tooth.
TR1: Mild loss of hard tissue * Cementum or cementum and enamel only.
TR2: Moderate loss of hard tissue that does NOT extend into the pulp.
TR3: Deep dental hard tissue loss that does extend into the pulp; most of the tooth remains intact and retains its integrity.
TR4: Deep hard tissue loss extending into the pulp: Most of the tooth has lost its integrity.
   TR4a: Both crown and root(s) are equally involved
   TR4b: Crown is more severely affected than the root(s)
   TR4c: Root(s) are more severely affected than the crown
TR5: Remnants of dental hard tissues are only visible as irregular radiopacities and the gingival covering is complete. (End stage tooth resorption).

References

3. www.AVDC.org/nomenclature
Pathophysiology

Pain, by definition, is a localized suffering associated with bodily disorder, a basic perceived sensation induced by a noxious stimulus, received by naked nerve endings, characterized by physical discomfort and typically leading to evasive action. Pain can facilitate self-preservation and is a deep-seated natural defense mechanism to prevent bodily harm. This kind of response by the body to a potentially harmful sensation such as burning, pressure or a forceful jolt allows us time to respond quickly and move away from the danger of a harmful object causing the above sensation. This type of pain can be referred to as physiologic pain, and is the result of Adelta (fast) and unmyelinated C fibers which transmit a slower, less intense pain sensation being stimulated through nociceptors, or bare nerve endings at the site of potential injury on the body.3

Somatic pain, a type of peripheral pain involving the joints, muscles or periosteum, can best describe our veterinary patient’s oral pain due to the localized sensation of pain in the mouth and skull structures.3 It can be further described as a sudden sharp jolt, pulsating discomfort or a dull constant pain. When these raw nerve endings or nociceptors are continually activated due to the ongoing insult to the oral tissues through periodontal treatment, root canal therapy, extractions, excision or incision of oral masses, gingivectomy, fracture repair and other invasive dental procedures, clinical pain will be perceived as a result.3 This stimulation causes a response called “wind up” which can produce alterations in the way that the patient responds to even the gentlest touch during recovery from a procedure eliciting this over-stimulation of the Central Nervous System (CNS).4 This response is termed alldynia and is something that we can and should prevent in our patients through the use of a multimodal approach to anesthesia/analgesia.5 This article does not go into a full discussion of multimodal drug protocols used in pre-anesthetic pain relief and prevention plans, these should be referred to in other texts.

The prudent use of local or infiltrative anesthetic drugs and regional, or area specific, nociceptive obtunding techniques should be used to prevent transduction or the conversion of unpleasant stimuli at the site of insult into impulses in the trigeminal afferent nerves, transmission or conveyance of these electrical impulses towards the CNS, modulation which in the case of oral stimulation happens between the neurons from the A-delta and C fibers synapsing with specific neurons in the nucleus caudalis within the medulla of the brain.5 Finally the perception of pain takes place in the cerebral cortex of the brain, and is the true manifestation of conscious pain perception in our patients.3 If this perceived pain is not treated or prevented then hyperalgesia ensues and the pain response increases even into nearby uninjured tissues resulting in further central sensitization or the “wind-up” phenomenon of pain.5

Why use regional anesthesia for dentistry?
The use of regional anesthetics in dental patients has many advantages. These include:

- Extensive analgesia to the targeted tissues thus reducing the quantity of inhalant anesthetic required and the elimination of the “roller coaster” up and down effect if the anesthetized patient perceives painful stimuli.2
- Reduction in the amount of inhaled anesthetic by the patient will lower the incidence of adverse events due to low blood pressure and bradycardia.3
- Prevention of pain can also reduce the time of convalescence through a decrease in the harmful catabolic process, which can hasten tissue healing and prevent a depressed immune response, which decreases the likelihood of infection overall improving patient recovery.3

Drugs used in regional anesthesia such as bupivacaine hydrochloride 0.5% and lidocaine hydrochloride 2% act to affect all three areas of the nociceptive pathway: transduction, transmission and modulation.5 Due to its length of action (3-10 hours) bupivacaine hydrochloride 0.5%, with or without epinephrine, is the most routinely used regional dental anesthetics today in veterinary dentistry.2

Disadvantages of regional anesthesia include

- Potential nerve damage
- Accidental intravenous injection, which can cause cardio- toxicity or arrhythmias with the use of bupivacaine 0.5%. Or as in the case of lidocaine hydrochloride 2% which can be given IV, excessive doses given intravenously can cause CNS or cardiac toxicity.6
- Feline patients have a much lower threshold for drug toxicities. Lidocaine hydrochloride should be only used very cautiously in cats.

Expediting the use of the appropriate regional anesthetic technique will allow adequate time for the onset of drug action prior to any noxious stimulation.
A thorough knowledge of cranial anatomy and foramina used in these techniques is required. The use of a canine and a feline skull can be very helpful as an example to visualize and palpate the target areas on the anesthetized patient. **Regional Anesthetic Drugs and Dosages**

**The maximum toxic dose should be calculated every time, for each patient.**

*For Bupivicaine the maximum dose should be calculated at 2mg/kg of patient body weight.*

Each regional block to be performed should have its own pre-determined quantity of drug not to exceed the total maximum volume of anesthetic. Common quantities used are 0.10mL per 10 pounds of body weight. Not to exceed the maximum quantity calculated. Bupivacaine has a delayed onset of action of at least 6-10 minutes and some authorities report even up to 30 minutes, once given in a foramen, but it has duration of 3-10 hours. Regional anesthetics cause vasodilatation, which hastens the reversal of the pain preventative effects due to the added blood supply to the area injected, aiding in the removal of the drug. The addition of epinephrine to bupivacaine will cause vasoconstriction and increases the drugs' duration of action.

- Products containing epinephrine should not be used in patients with hyperthyroidism or cardiac disease or in conjunction with the use of halothane as and inhalant anesthetic.

**Glossary**

- **Allodynia:** When a normally non-painful stimulus causes a painful response.
- **Analgesia:** Absence of a pain perception.
- **Buccal Mucosa:** Lines the oral cavity facing the cheeks.
- **Central Sensitization:** Hyper-excitability of pain perception. Also referred to as “wind up”.
- **Clinical Pain:** A painful response that is noted by the clinician after ongoing nociceptors stimulation has changed the way the afferent nerve system responds to stimuli from injury.
- **Hyperalgesia:** An increase in pain response caused by local inflammation as a result of noxious stimulation to the area.
- **Mandible:** Lower jawbones separated into a right and left mandible joined by a symphysis.
- **Maxilla:** The bones of the upper jaw structure.
- **Multimodal analgesia:** The use of analgesic drugs from different classes that act in synergism to promote a more complete pain prevention protocol.
- **Nociceptors:** Nerve endings that can be activated by noxious stimuli to transmit electrochemical impulses through the afferent nerve pathways for perception of pain in the brain.
- **Regional Anesthesia:** The use of an anesthetic solution injected into an area at or near a foramen containing a major nerve branch to effectively “block” the pain pathway to a specific region of the mouth.

**Table I: Infraorbital regional nerve block**

This block affects the bone and soft tissues of the anterior maxilla in that quadrant.

The bony margin of the foramen is palpated just dorsal to the distal root of the maxillary third pre-molar. The needle is then inserted just into the opening using caution to aspirate the syringe in 4 different planes to confirm that a blood vessel has not been punctured, only then should the anesthetic be injected very slowly into the space. Digital pressure over the injected area for 30-60 seconds will further allow the agent to disperse into the canal.
Table II: Maxillary regional nerve block
This blocking technique affects the bone, palatal aspects, soft tissue and all maxillary teeth of the dental quadrant injected.

Site for maxillary nerve block in a dog.
The injection site is located dorsal to the last molar at the ventral junction of the zygomatic bone on the maxilla, by injecting the anesthetic agent slowly into this area after careful aspiration to avoid any accidental injection into a vessel you will effectively block pain sensation to all of the maxillary teeth in this quadrant. Great care should be taken not to advance the needle past the intended area due to the location of large vessels and the ocular globe in this area.

Table IV: Inferior alveolar (mandibular) regional nerve block
The tongue, mandibular teeth and soft tissue are affected on the infiltrated quadrant to the symphysis.
The mandibular foramen is easily palpated intra-orally in the dog. It is located just rostral and dorsal to the prominent angular process on the lingual side of the mandible. Careful intra-oral palpation along with extra-oral injection technique to place the tip of the needle over the foramen will accurately deposit the anesthetic agent to effectively block all ipsilateral mandibular teeth and adjacent soft tissues and tongue.
References

When we think of the words “a dental” which we have commonly come to know as really any dental care that is done under general anesthesia for our veterinary patients, we are really over simplifying what is, or should be, occurring when we undertake professional dentistry for animals.

Dentistry is a large discipline that requires its own specific set of skills, knowledge, correct instrumentation and equipment to accomplish prophylactic and therapeutic treatment. Extensive training and practice by the practitioner are required to ensure the correct methods are used to reduce or eliminate infection and pain in our patients’ mouths.

A thorough knowledge of anatomy and pathology as previously discussed is a starting point for the professional dental treatment of veterinary patients. Only after diagnostic methods have been employed by an oral exam, complete charting of the oral cavity and obtaining dental radiographs, can we determine the extent of the disease and create a treatment plan for the patient. When the diagnostics have been completed then the actual treatment can begin.

If there are no indications of periodontal disease, fractured teeth, tooth resorption, missing or unerupted teeth, supernumerary, crowding, mobile teeth or other situations that will need more advanced treatment, the dental prophylaxis can begin. Prophylaxis, which means the prevention of disease, really can only apply to healthy mouths that need to be thoroughly cleaned of any plaque and tartar to help prevent pathology that may develop if the plaque and bacteria that it contains is allowed to stay in contact with the oral soft and hard tissue structures.1

If the patient’s mouth is currently in a state of active disease such as that of periodontitis, where there is destruction of the periodontal ligament and alveolar bone occurring, this requires more extensive treatment which should correctly be called “Periodontal Therapy.”1

Periodontal therapy requires more involved and invasive treatment of pathology to bring their mouth back to a healthy state. This should be discussed with the client to help inform and increase understanding of why disease has already occurred and why treatment of this disease will require longer anesthetic procedures, possible tooth extractions and dedication to homecare procedures if the client desires to try and save teeth instead of having teeth extracted. Sometimes the periodontium has undergone such destruction that saving teeth even through more advanced methods is simply not a feasible option due to a poor prognosis for the tooth, lack of commitment to homecare or future anesthetic visits, to allow follow-up therapy to be performed.

General anesthesia is required for all veterinary dental patients and should be undertaken with all of the same precautions that would be allowed for any surgical candidate. Pre-operative blood/urine or other diagnostic testing should be performed and interpreted. An estimate for treatment should be presented to the client with a contingency plan for further authorization once the patient is fully evaluated under anesthesia and radiographs have been obtained. Prevention of hypothermia, hypotension or other anesthetic complications should be carefully assessed and steps should be in place to manage the patient as closely as possible to prevent any situations that could be avoided.

A multimodal pain management protocol should be taken into consideration as well, especially if periodontal surgery or extractions are part of the dental treatment plan. The use of regional dental anesthetic blocks is fast becoming a standard of care in oral and dental pain management when painful procedures are to be performed.

Use of a cuffed endotracheal tube is a must to prevent accidental aspiration of fluids or debris from the oral cavity into the airways. This tube should be monitored closely for any obstructions and the cuff should be checked again several minutes into the procedures to ensure a secure seal without causing any trauma to the trachea by over-inflation.

There are several steps involved in the Complete Professional Dental Prophylaxis, these are outlined below and will be discussed in further detail. If the patient is not in the prophylactic category then periodontal therapy and possible exodontics or extraction of diseased teeth will take place. We will discuss the dental prophylaxis first.

**15 step oral evaluation and treatment**

1. Oral exam on “conscious” patient: Evaluate occlusion as well as all soft and hard oral and facial tissues as much as possible.
2. Take “before” treatment photographs at this time.
3. Oral exam on anesthetized patient: rinse mouth with a 0.12% chlorhexidine oral rinse prior to this evaluation. Gross Calculus may need to be removed to allow for access to soft tissues.
4. Thorough charting and documentation of all oral structures. Attempt to locate any areas of concern or pathology: missing teeth, fractured teeth, periodontal pockets, mobile teeth, extra teeth etc.
7. Perform regional anesthetic blocks at this time if necessary.
8. +/- Periodontal Therapy and Exodontics.
9. Obtain post-extraction or treatment radiographs as indicated prior to suturing extraction sites.
10. Re-evaluate occlusion as necessary after extractions. Extubate and then re-intubate.
11. Clean all dental surfaces both supragingival and subgingival by using the ultra-sonic power scaler and then hand instrumentation to ensure coronal and root surfaces are completely clean.
12. Polish all crown surfaces with fine grit prophy paste.
13. Irrigate all tissues and the periodontal sulcus with water and gently dry tooth surfaces with air to further evaluate that all areas are clean and free of debris, calculus and polish.
14. Take “after” treatment photographs
15. Optional treatments: Apply sealants such as Oravet™ or Sanos® to dry clean crown surfaces.

**Basic dental instrumentation**

<table>
<thead>
<tr>
<th>Item</th>
<th>Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>Explorer/probe:</td>
<td>Probe/explorer combination (UNC15/23) General use</td>
</tr>
<tr>
<td>Scalers-dog:</td>
<td>Towner/Jacquette Sickle Scaler</td>
</tr>
<tr>
<td>Scalers-cat:</td>
<td>Morse 0-00</td>
</tr>
<tr>
<td>Curettes-dog:</td>
<td>Gracey 1/2</td>
</tr>
<tr>
<td></td>
<td>Barnhart 1/2 Universal curette</td>
</tr>
<tr>
<td></td>
<td>Columbia 13/14 Universal curette</td>
</tr>
<tr>
<td></td>
<td>Columbia 4R/4L Universal curette</td>
</tr>
<tr>
<td>Curettes-cat:</td>
<td>Double ended NV series feline curette (Shorter working end is ideal for cats)</td>
</tr>
<tr>
<td>Sharpening kit:</td>
<td>India slip stone (for reworking instruments), Arkansas stone kit (daily sharpening), stone oil and plastic test sticks.</td>
</tr>
<tr>
<td>Power Instruments:</td>
<td>Ultrasonic scaler of either a piezoelectric, magnetostrictive stack insert or a magnetostrictive type with a ferrite rod insert. This scaler must have a water source to cool and irrigate the working end while in operation. It can be a stand-alone unit that is attached to a pressurized water bottle or integrated into a dental delivery unit that houses a compressor, which uses compressed air to drive the slow-speed handpiece, ultrasonic scaler, high-speed handpiece and air/water syringe. Low-speed handpiece: The handpiece is either driven by an electric motor pack or integrated into an air driven dental delivery unit.</td>
</tr>
<tr>
<td>Prophy Angle:</td>
<td>This attaches to the low-speed handpiece and allows a prophy cup to be placed on the working end. This is used to polish the teeth after the cleaning. Disposable angles are important to prevent patient cross-contamination.</td>
</tr>
<tr>
<td>Prophy paste:</td>
<td>These should be single use individual cups of prophy paste, to prevent patient cross-contamination, which when used on the teeth following the ultrasonic and hand instrumentation of the tooth will reduce any microscopic grooves created in the enamel to eliminate a rough, plaque-retentive surface that can be created by scaling and curettage. Fine, medium or coarse grits are available, however it is usually recommended to choose a fine or medium grit paste to prevent removal of too much enamel when polishing and to create a smooth surface. Some prophy pastes contain fluoride so be aware that fluoride can interfere with some restoration procedures and should not be used in these cases on a tooth that will have a restoration performed. Flour pumice is a good choice for those needs. Do not use a fluoride type of prophy paste if you plan to use Sanos® Sealant on the patient.</td>
</tr>
</tbody>
</table>

**Chlorhexidine Oral Solution:** This 0.12% chlorhexidine solution is used to irrigate the oral structures to help reduce bacterial aerosolization exposure to the operator and bacteremia to the pet’s bloodstream.

**The fifteen step dental cleaning procedure with/without advanced care**

**Step 1: The basic oral/facial/skull examination**
This exam should occur in the awake patient. This helps us to create a treatment plan for the client prior to anesthesia. This is just the initial exam however. Please see the notes in the pathology and anatomy section.

**Step 2: Photographs**
With the use of a digital camera we can take before photos of the oral cavity and hard and soft tissues of interest so that we may offer the client a visual comparison.

**Step 3: Oral exam on the anesthetized patient**
This is a more thorough evaluation of the oral and facial structures including a complete occlusal evaluation. (Evaluate occlusion prior to the placement of the endotracheal tube on the sedated patient.)
Step 4: Charting
Thorough documentation as previously discussed is a must in locating any areas of concern or pathology and working toward creating a plan for further diagnostics and treatment. The use of the instrument called the explorer/probe is necessary during this step. Also a dental mirror can further enhance our ability to visualize the areas of concern. Proper lighting and magnification will greatly increase our ease of recognition of oral structure abnormalities. A complete chart that allows full documentation of pre and post treatment with multi-directional views of each tooth will assist in complete and accurate documentation as well. Decontamination of the gross calculus may be necessary at this time to help facilitate correct charting.

Step 5: Radiology
As fully discussed in the radiology notes, this is an important step in further diagnostics and documentation of oral structures and should be obtained prior to commencing treatment. Some prophy pastes can be seen on radiographs so post-treatment radiographs should be obtained prior to the polishing step.

Step 6: Treatment plan
The creation of a treatment plan for each and every tooth. Paring the clinical findings from the oral examination, documented by charting and the radiographic findings to create this plan. If the plan is to truly perform only prophylactic procedures such as supra and subgingival cleaning and polishing then regional anesthesia and a more complicated treatment plan will not be necessary.

Step 7: Regional anesthetic blocks
These should be performed now and given a few minutes to take effect before more painful stimulus is caused by extractions or subgingival curetage.

Step 8: Advanced periodontal therapy, exodontics, endodontics or other procedures
Prioritize these procedures as directed by the veterinarian.

Step 9: Post extraction radiographs
As indicated by changes to tooth or bone structure these radiographs should be obtained prior to suturing the extraction sites to avoid the need to undo sutures to retrieve root or tooth structure that was inadvertently left behind during the extractions.

Step 10: Re-evaluate occlusion as necessary
This can be an important step if extractions of major teeth were performed to insure that there are no complications from the patients jaws closing more fully or if we are extracting teeth to alleviate any current malocclusion issues.

Step 11: Dental cleaning procedure
If the patient does not require any other procedures as outlined above then step 7 will be omitted. The dental prophylaxis consists of removal of gross calculus and then removal of all dental calculus from the crown surface or supragingival. The use of the ultrasonic scaler to do most of the major work and then the hand scalers to perfect the work are helpful for this.

The ultrasonic scaler, which operates at a vibration range of 18,000 to 45,000 cycles per second, is utilized to break up the calculus or tartar deposits on the coronal surface of the teeth. The handpiece should be carefully held in the hand with a comfortable grip only using the active sides of the instrument’s tip with the handpiece held parallel to the long axis of the tooth.

The instrument tip should be continuously in motion over the surface of the enamel in a cross-hatching pattern. Care should be taken to keep moving on to the next tooth and allowing each tooth to cool down in between sessions of cleaning. Ultrasonic vibrations can generate enough heat that if persistent can cause thermal damage and possible necrosis to a tooth’s vital inner blood and nerve supply. Water spray, in a fine mist, further reduces the heat that is created and should be sufficient and always in use. The water also helps rinse the debris off of the tooth as it is being scaled clean.

Subgingival calculus and plaque must be removed if there are periodontal pockets below the gingival margin. This is a very important step if truly effective dental cleaning is desired. If there is any debris left below the gum line in the gingival sulcus or in a periodontal pocket then the prevention of oral infection and disease will not be accomplished.

The use of curettes which can safely be inserted under the gingival tissues and into periodontal pockets is recommended. Due to the rounded back and toe of the curette, this instrument, which will reduce the likelihood of damage to the attachments at the bottom of the sulcus if used properly, should be used instead of a sharper pointed scaler.

Curettes must be held in a modified pen grasp, a fulcrum should be established, the instrument should be adapted to the surface to be cleaned, the blade of the instrument should be engaged and then the down or cleaning stroke performed. Overlapping strokes in different planes will ensure that the surface that needs to be cleaned will be completely cleaned.

Different variations of dental curettes are available and help with instrument adaption whether working in the most rostral portion of the mouth on incisors or adapting the instrument to caudal pre-molars and molars. Choosing the correct instrument for the area to be curetted is important to successful adaption, effective plaque and tartar removal and prevention of operator injury due to inappropriate handling of the instruments at awkward angles.

Periodontal bactericidal ultrasonic debridement is the final step in ultrasonic cleaning. A specially made periodontal tip insert is required for this procedure or some dental ultrasonic units are already equipped with a tip that can be safely inserted sub-gingivally.
Please consult your ultrasonic equipment manual regarding which tips are safe to insert under the gum line into the sulcus, and at what setting the machine should be turned down to, reducing the frequency of vibrations to a safe level for this purpose.

Periodontal bactericidal ultrasonic debridement occurs due to the ultrasonic sound waves causing microscopic bubbles to form and then implode in the gingival sulcus, cavitation. These implosions can cause the bacterial cell walls to be disrupted and along with the water rinsing through the area at a certain pressure further reduces the concentration of bacteria within the space.1

**Step 12: Polish tooth crowns**

This important step helps to create a smooth, non-plaque retentive surface so that the teeth will remain free of plaque.3 Polishing can remove any plaque that was missed during the scaling and curettage phase of cleaning and helps to remove stains from the enamel.1 This requires that a **prophy cup**, usually a fairly soft cup, be attached to the working end of a **disposable oscillating prophy angle**. This is attached to the slow-speed handpiece either on the motor pack dental unit or the air-driven dental delivery unit which rotates at between 1,000 to 3,000 rpm.3 The oscillating disposable prophy angle produces excessive heat from being generated by rotational forces on the tooth surface and also prevents the patient’s hair from winding around the angle when in use near the patient’s hair on the lip or cheek areas.1 Disposable, one-use prophy paste cups further prevent patient cross-contamination from the alternative of a multi-use large container of paste.1 Metal non-disposable prophy angles can be used but generally are not oscillating and require cleaning and maintenance to keep them functioning correctly.

Ample prophy paste should be applied or rubbed onto the tooth surface via the non-spinning prophy cup prior to commencing polishing. It is the prophy paste and not the cup that does the actual smoothing of any enamel defects so this is an important step. By smoothing the paste on the teeth in quadrants prior to polishing you avoid spraying as much prophy paste around the mouth and onto the operator. The use of prophy paste helps to reduce the friction on the surface and minimizes the heat that is generated as well. The choice of prophy paste will depend upon whether fluoride is desired and the grit of the paste required.1,2,3 Standard paste is either fine or medium grit and usually contains fluoride.3 Course prophy paste can be used to remove stains from the enamel, however it will remove more enamel and also should be followed up by a fine paste as the ending step to polishing.1,2

All coronal surfaces, buccal, palatal or lingual, mesial and distal should be polished in a systematic fashion by starting at the most caudal teeth and working towards the midline or central incisor in each quadrant. The prophy cup should be applied to each surface with only enough pressure to slightly flare the cup out onto the surface and into the gingival sulcus area.1 Thermal damage to the tooth pulp can occur if the oscillating prophy cup is kept on the tooth for more than a few seconds.

**Step 13: Irrigation of sulcus and teeth**

The gingival sulcus is a prime place for left over debris to accumulate after a thorough dental prophylaxis or periodontal therapy has been performed. If this debris is allowed to stay in the sulcus it will act as an irritant and source of further inflammation or possibly even a periodontal abscess in the future.1,3

Potential debris is dental calculus, cellular debris, prophy paste and plaque containing harmful bacteria. We must gently lavage this debris out of the sulcus either by using the three-way syringe on our dental machines to use air and water together to rinse all of the prophy paste and debris from the crowns and sulcus. In addition to this, we may choose to utilize a 6-12 cc syringe filled with a 0.12% chlorhexidine gluconate oral solution and rinse the sulcus around each tooth completely with a 22-28g blunt tip needle or cannula.1,2

**Step 14: Post treatment/cleaning photographs**

These digital images along with images of the dental radiographs if applicable can be shared with the client along with the pre-treatment photos to further illustrate the remarkable difference that dental prophylaxis, periodontal therapy or more advanced dental treatment can make for their pet. These can be printed out in color for the client to take home, shared with the client via e-mail or sent to a specialist for their evaluation if necessary to help facilitate future treatment and care. Photos and radiographs also help us to follow visual changes in the patient’s dental health over time. These serial photographs can really show a client the progression of disease if we neglect homecare or professional cleanings.

**Step 15: Application of dental sealant products or fluoride if indicated: Fluoride foam**

Application of fluoride foam is controversial but may have some benefits to patients, such as decreased tooth sensitivity especially if dentin is exposed on the coronal or root surfaces because it acts to seal exposed dentinal tubules, an anti-plaque and antibacterial effect because it inhibits bacterial metabolism; and it can help the enamel resist decay.1 Cavities in dogs are rare and extremely rare in cats so this may not be a viable reason to apply fluoride in our veterinary patients.1 The downsides to using fluoride are possible toxicity if chronically used in higher than recommended doses and the interference of fluoride with certain restorative, bonding or sealing agents.1,3

If fluoride is applied it should be applied to cleaned, polished, lavaged and dried tooth surfaces. The fluoride foam should be allowed to remain in contact with the enamel or dentin for 3-5 minutes, after that it should be carefully wiped off with dry gauze, do not rinse fluoride foam off with water because it will inactive the fluoride.3
Oravet™ Sealant
Merial Oravet™ is a non-toxic waxy polymer that is applied to the clean and dry tooth surfaces of both cats and dogs. The professional application is of higher viscosity than the thinner homecare kit. It is the base application for the prevention of dental plaque adherence to the enamel surfaces of the tooth crowns. It should be applied up under the gingival margin on the surfaces of the crown and in the sulcus to prevent plaque from accumulating under the gum line. The manufacturer recommends that the client begin the homecare kit applications two weeks after the initial professional application to keep the thickness of sealant in place on the enamel surfaces.

Sanos®: AllAccem, Inc
Sanos® is a product that was developed to help improve gingival health and prevent periodontal disease by providing a liquid bandage like barrier that when applied to the gingival sulcus stays in place for up to 6 months. It has a V.O.H.C.(Veterinary Oral Healthy Council) label for prevention of plaque and tartar accumulation and it prevents gingival inflammation that may be caused by the plaque bacteria invading under the gingival margin. It is easily applied to clean and dry teeth and gingival sulcular surfaces with the brushes in the kit. It dries quickly and is clear to slightly opaque in color. It is non-toxic and approved for use in both cats and dogs.

Note: If the practitioner plans to use Sanos® on the teeth and gingiva then a fluoride application is not recommended.

References
4. www.vohc.org
5. www.fda.gov/ForConsumers/ConsumerUpdates
How to obtain veterinary dental radiographs
When you are first learning how to obtain a radiograph of diagnostic quality it is important to understand the basics of patient, film or
digital sensor and primary x-ray beam positioning.

By creating a standard for patient position, sensor position and then the resultant x-ray beam position will become almost standard
as well to minimize all of the variables that come into play when getting the angles correct. This creates a method for obtaining dental
radiographs that is easy to repeat with each patient.

Remember this formula:

\[ A + B + C = D \]

Animal position (A) + X-ray beam (B) + Sensor(C) = (D) Diagnostic image

It is also important to understand how the basic settings on your x-ray unit functions and how to manipulate these settings to obtain the
best exposure for that particular patient and teeth to be radiographed.

Each x-ray unit may be a little different however, commonly used modern dental radiographic machines take a lot of the guess
work or the necessity of developing a technique chart, out of the process by having a computer aided control panel that has easily set
parameters based on the species and tooth to be exposed.

Digital sensors require much less radiation exposure than dental film packets do, so make sure if you are using a digital sensor that
you set the exposure time at the appropriate place for a starting point. Slight changes in the exposure time will be made during the
whole mouth series when we are x-raying areas with more or less bone and soft tissues since the machine is pre-set at a KVP and MA
for dental uses already.\(^3\)

Note
Canine Patients at minimum will need a series of 10 to 23 exposures for a full mouth series, more if the patient is larger or you are
using a size 2 digital sensor for large teeth.

Feline Patients at minimum will need a series of eight (8-10) exposures for a full mouth series.

Step 1: Patient positioning: (A)
Positioning of the patient is very important when you are first learning to radiograph oral structures. If you always position the patient
the same way when you are obtaining maxillary teeth or conversely mandibular teeth, then the angles that you will use with the
primary beam (PID) and the analog dental film, PSP or digital sensor in the mouth, will usually coincide with the patient position.

Placing the patient in ventral recumbency is the best position for all maxillary exposures in the canine and feline patient. Have the
patient lie on its chest with the head resting about 3-6 inches above the table top either placed on a 3 x 5 plastic container filled with
sand and sealed shut (I recommend a 500mL plastic irrigation saline bottle that has squared sides), this works very well to rest the chin
or head of most patients on it to elevate the head up off of the table top surface. The goal here is to make sure that the hard palate is as
parallel to the table top surface as possible. Make sure the head is resting as flat as possible on the “container”, the chin should neither
be tipped up nor down.

Dorsal recumbency is the preferred position for all mandibular exposures. When you are ready to move the patient to this dorsal
position, carefully turn the patient on its back ensuring that you do not twist the endotracheal tube. Try to make sure that the most
ventral edges of the mandibles are as close to parallel to the table top surface as possible. This may require that you roll up a small
towel and place it comfortably under the patient’s neck to make sure again, that the chin is neither tipped up towards the ceiling or
down towards the table top.

Step two: Placement of the digital sensor in the patient’s mouth (C)
The placement of the digital sensor in the mouth is done in a specific way. The flat surface of the sensor is towards the primary beam.
The sensor should be placed fully in the mouth with the edge of the sensor at the edge of the crowns in many cases; the cord from the
sensor should always be directed toward the front of the mouth and should come out of the mouth towards the nose of the patient in
between the mandibular and maxillary incisors.

Step three: X-ray beam angles (B)
When we are exposing the film, phosphor plate or digital sensor to the x-ray beam we want the resultant image to be a true
representation of the root and crown structures size and dimensions. Incorrect angulations of the primary beam will distort the image
much as our shadow is distorted on the ground when the sun “primary beam” is too high overhead or too low towards the horizon. When the primary beam is bisecting the vertical axis of the tooth root or as in the case of the sun beam and our upright body at just the right angle the resultant “shadow” that is cast on the ground (from our body) or on the film, PSP plate or sensor from the tooth structures is just the same height as the tooth (person’s body) in question.

Place the digital sensor in the patient’s mouth at the area to be radiographed with the edge of the sensor at the edge of the crown(s); most of the sensor will be inside the patient’s mouth. We are trying to project the roots onto the sensor so we need plenty of room in which to do that. The sensor will lay from palate to crown naturally, no need to make the sensor flat or parallel to the palate. Obtaining dental radiographs in the most efficient and quickest way possible is both in the best interest of the patient and the dental provider. This author prefers to start with the maxillary incisors, move on to both maxillary canine views, and obtain all of the pre-molars and molars on one maxillary quadrant and then move on to the opposite maxillary quadrant. Then the patient is rotated to dorsal recumbency; the mandibular canines and incisors are obtained together if possible and then the side views of the mandibular canines and the first and second pre-molars on each side of the rostral mandible in the dog, then the remaining mandibular pre-molars and molars are obtained on each side respectively to finish the whole mouth series on the canine patient. This technique minimizes the changing of the vertical angle of the x-ray beam as much as possible as each exposure is obtained. The feline patient whole mouth series follows the same progression with fewer exposures due to the smaller mouth and fewer teeth to x-ray.

Vertical angle: The angle that can be dialed in on most machines, this controls the length of the tooth as it appears on the resulting digital image.

Horizontal Rotation: This is the movement of the x-ray tube head in a horizontal direction around the patients head. 0 degrees is pointing at the midline of the head from the nose, 90 degrees is pointing at the midline of the head exactly perpendicular to the midline of the head. All other angles are more or less than those two horizontal rotations. Example: 30 degrees, 70 degrees, 110 degrees horizontal rotation.

When exposing the maxillary incisors you will need to adjust the vertical beam to 70 degrees, the cone (PID) of the x-ray unit will fit down right over the patient’s nose in many cases for this exposure. Horizontally the beam will be rotated to point at the midline of the patient’s head at 0 degrees.

Always keep in mind that when you get to the maxillary canine tooth roots that they are at a more horizontal angle and very far back in the patient’s mouth compared to the maxillary incisors. You will need to change the angle of the vertical beam to avoid foreshortening of these roots to 60 degrees. You must take two separate exposures at a horizontal rotation to each side of the face at 70 degrees, this will result in an image of each maxillary canine tooth because you cannot obtain one view and see both root apices clearly due to the probable superimposition of the pre-molar teeth on the roots of the maxillary canine teeth.

All maxillary pre-molars and molars can be obtained with a vertical angle of 45 degrees and horizontal rotation of the beam from 90 degrees for the ‘05’06’07, to 110 degrees horizontal for the ‘08’09’10. The mandibular canines and incisors can both be viewed in one exposure unless the crowns are desired and then two individual exposures may be required. A nearly parallel technique can be used with the primary beam oriented directly above the tooth roots and the sensor parallel to the roots and the ventral edges of the mandibles and the PID is at a vertical angle of 75-80 degrees and horizontal rotation of 0 degrees.

When exposing only the mandibular incisors, pull the sensor more rostral in the patients’ mouth with the edge of the sensor at the edge of the most coronal aspect of the crowns, again the primary beam will be at 70 degrees vertical and 0 degrees horizontal.

In the feline patient, the zygomatic arch of the maxilla can be superimposed over the maxillary pre-molars, by decreasing the vertical angle to 40 degrees and rotating the beam horizontally to about 80 degrees, just slightly less than the 90 degree mark you will find that the zygomatic arch appears to be more like a ghost over the pre-molar teeth and much easier to interpret.

**Parallel technique**

This is the simplest technique in veterinary dental radiology and is commonly used for the mandibular pre-molar and molartooth307, 308,309,407,408,409 in the cat and the 407, 408,409,410,411,308,309,310,311 teeth in the dog. The digital sensor is placed in the oral cavity with the patient in dorsal recumbency. The sensor is as close to parallel to the tooth/teeth roots as possible. This allows us to focus the primary beam at a more perpendicular angle directed right through the bone/teeth at the sensor and get an image that closely represents the true size and dimensions of the structures without elongation or foreshortening.

By adjusting the angle of the primary beam slightly up (the patient is in dorsal recumbency) over the mandibular bones at 40 degrees and slightly caudally or distally in a modified oblique position you will have much success in obtaining all of the roots on your mandibular exposures in the feline patients. You should have teeth 307,308,309 in one exposure and teeth 407,408,409 in the other exposure for each mandibular quadrant.

Some more difficult exposures to obtain such as the 310,311 or 410,411 molars in dogs require a similar adjustment to the primary beam at 10 degrees with a distal tube shift to 100 degrees horizontal rotation, when you can only push the sensor so far back in the patients’ mouth due to limitations caused by bony structures or soft tissues. It is very important that the sensor follow the mandibular bones caudally so that the resulting image is strait and the last molar tooth is visible.
S.L.O.B. rule
Whenever we are obtaining exposures of teeth with multiple root apices we need to be able to differentiate one root from another root. Superimposition of one root on top of another root hinders our ability to make diagnostic decisions for that tooth.

There is a rule that helps us to obtain a radiograph of a tooth such as the maxillary fourth pre-molars #108 and 208 in the cat and dog and to shift the image such that the mesial-buccal root and the palatal root are no longer superimposed upon one another.

*Same Lingual Opposite Buccal* means that when we shift the primary beam from being at a direct angle focused on one tooth to an oblique position going through the tooth/roots to the sensor, the palatal tooth root will follow the shift of the primary beam. The palatal tooth root can be described as lingual since it is more toward the tongue, so that is why the word “lingual” is used in this rule.

The most common method is to take the primary beam and shift it to 110 degrees caudally or distally. The resultant image will show the distal root where it is in a caudal location, the palatal root will now be in the middle and the mesial/buccal root will be the most rostral on the sensor’s resulting image. Conversely if the beam is shifted horizontally to 70 degrees rostrally then the resultant image will show the palatal root in the most rostral position, the mesial/buccal root in the middle and the distal root will be superimposed over the first molar position.

Keeping the patient in a constant position while obtaining all of the maxillary views with a standard setting for the vertical x-ray beam and ensuring that the sensor is in the correct position in the patient's mouth to accept the image that we are projecting toward it is a very simple and repeatable concept. Check to make sure the patient is in the same position with every exposure, and then setting the patient into a standard position in dorsal recumbency for the mandibular views will greatly speed up the efficiency and accuracy of obtaining diagnostic dental radiographs.

**Diagnostic image (D)**
What is a diagnostic image?

There are some basic requirements for our images of dental structures to be diagnostic. For every image obtained ask yourself these questions.

1. Did I get the tooth or teeth desired on this image?
2. Did I get 100% of the root structures of these teeth on the image?
3. Did I get at least 3mm of bone visible around each apex of each root on the image?
4. Did I get at least 3mm of crown structures beyond the horizontal margin of alveolar bone?
5. Is the image the correct length? Did I elongate or foreshorten the tooth structures?
6. Is the exposure correct? Is the image too dark or too light?
It is important to understand that ear disease is only a symptom (no more specific than “pruritus”). As Dr Flemming Kristensen stated “A patient showing ear problems is a dermatology case until proven otherwise”. As Dr James Noxon brilliantly observed- the challenge is not only to find the “WHAT” is causing the otitis but the “WHY” do they have otitis. The technician can help in many ways determine both the WHAT but also the WHY, beginning with a detailed history. An

Specific questions that should be asked include:

1. When did the symptoms first occur? This is an important question, because many owners will only tell you when this current episode of symptoms occurred, not the very first time it occurred;
2. Other than the problem the owner presents the patient for, you must ask all owners if the dog has EVER had problems with excessive licking, scratching, chewing, biting or rubbing. Has the dog ever had ear problems before this episode? If so, when, with what medication and what was the response to treatment;
3. Where does the dog live- indoor, outdoors, both? Describe the environment, especially the outdoor environment;
4. Is the dog on heartworm and flea preventative? If so, what product, how often is it administered and is it year round or seasonal?
5. Are there any other pets in the household? If so, what kind and are they symptomatic. If they are cats, do they go outside?;
6. Are any of the humans in the household showing “new” skin problems? If so, what kind;
7. Do they board the dog, take him to obedience school, training or to the groomers? If so, when was the last time?;
8. Do they know if the parents of the dog or any siblings have ear or pruritic skin problems? If so, what was done and what was the response?;
9. What does the dog eat?
10. How do the ears seem today- is today’s presentation the best, worse or average since the problem began?
11. Do you notice if the symptoms were better, worse or no different or not sure between the different seasons.

In addition to obtaining the history, the technician should be sure that other information is readily available to the veterinarian. This would include having the age, breed and sex information in the record and if the dog has had previous ear or skin problems- GET A COPY of medical records from the previous veterinarian. Many times treatment can be expedited by reviewing what previous treatments and tests were prescribed and the response.

The veterinarian will then do complete physical examination since some times there are systemic signs associated with otitis externa (fever associated with pemphigus, lethargy associated with vasculitis, etc) This is followed by a complete dermatologic examination. Because ears are really just skin attached to the skull many diseases that affect the ears frequently will affect the rest of the skin and vice versa. Therefore even when a dog is presented only for otic disease the veterinarian will still need to examine the rest of the body. And the opposite also holds true, when a dog is presented for skin disease the veterinarian will also do an otic examination.

Now diagnostics and treatment needs to be pursued. As mentioned previously when dealing with a cause of otitis we want to know WHAT is causing the otitis and WHY did it occur. To understand the differences it is helpful to divide factors that are involved in otitis into predisposing, primary and secondary factors.

Primary (underlying) cause(s) of the ear disease (the WHY) 1,2,3,4,5 These would include:
- Parasitic (including Demodex, Otodectes, Sarcoptes);
- Foreign bodies;
- Hypersensitivities- 80% or more of the dogs with atopic dermatitis (food or environmentally triggered) have otitis externa6,7 (NOTE OE may be the ONLY symptom in 3-5% of the environmentally triggered atopic dermatitis cases and

2 Griffin CE. Otitis externa and media. In: Griffin CE, Kwochka KW, MacDonald JM, eds. Current Veterinary Dermatology. St. Louis:Mosby-Year Book; 1993:244-262
it may be UNILATERAL!!; it may be seen in cutaneous adverse food reactions where it too may be the ONLY symptom in up to 20% of the cases and also may be unilateral or flea allergy dermatitis. In cases of FAD there should be involvement of the posterior 1/3 of the body in addition to the OE;

- Allergic or irritant contact dermatitis;
- Endocrinopathies, keratinization or sebaceous gland disorders leading to an altered lipid layer in the epidermis, alteration in normal keratinization or glandular function; idiopathic seborrhea (is there such a disease?)- Hypothyroidism is commonly cited as a cause of otitis externa. It has been stated that "Recurrent bacterial and yeast infections of the skin and ears often occur secondary to hypothyroidism and may be the only presenting signs." The author refers the reader to a textbook that makes this statement. If you look at that reference you see that this is the 6th edition of a dermatology textbook and in that textbook the reference is the 5th edition of the same textbook. The 5th edition references the 4th and the 4th edition references the 3rd edition. When you further investigate the reference you find that the original reference is NOT about hypothyroidism but about hyperthyroidism in mice and the effect of administering sheep RBC to mice. So the bottom line is there NO EVIDENCE that otitis externa will be the only clinical sign associated with hypothyroidism.
- Autoimmune or immune mediated diseases (eg pemphigus complex, vasculitis- note these diseases involve the pinna canals);
- Zinc responsive dermatosis (will involve more than the pinna);
- Juvenile cellulitis;
- Immunosuppressive diseases (distemper, FeLV, FIV, parvo virus);
- Neoplasia (adenoma, adenocarcinoma);
- Dermatophytosis (affects the pinna rather than the ear canal).

In addition to identifying the primary cause, secondary factors must be addressed if possible. Secondary factors don’t cause ear disease but increases the risk of developing ear disease and may make successful treatment more difficult. Secondary factors are: anatomical factors (eg- long pendulous ears in the Basset Hound or stenotic ear canals in Shar Peis); excessive moisture in ears (swimming); and iatrogenic trauma (plucking hairs from the ear canals, cleaning ear canals with cotton tip applicators).

Lastly perpetuating factors must be identified and treated (the WHAT). These factors don’t initiate the problem, but will cause the disease to continue, even with the elimination of the primary factor, once it has been established until these factors have also been addressed. Perpetuating factors include:

- Bacteria (coci most commonly *Staphylococcus intermedius* (acute infections), beta hemolytic streptococci and rods most commonly *E. coli, Pseudomonas* spp (chronic infections); *Proteus* spp, *Klebsiella* spp and *Corynebacterium* spp);
- Fungi (*Malassezia pachydermatis* (which may cause a hypersensitivity reaction so that small numbers may be significant));
- Progressive pathological changes;
- Otitis media;
- Contact hypersensitivity/irritant;
- Treatment errors (most commonly due to under treating the infection).

Laboratory tests are a necessary component to the proper workup of a case of canine ear disease and once again the technician is invaluable in performing these tests. CBC, serum chemistry profile, urinalysis, skin scrapings, fungal culture, endocrine testing and skin biopsies may be necessary depending on what the differential diagnoses are for that patient. Cytologic examination of a roll swab sample should be performed on any discharge. The numbers & type of bacteria, yeast and inflammatory cells should be quantitated. In cases of OE the question of what is an abnormal number of organisms, per oil field, has not been settled. Depending on the study, cutoff numbers, per oil immersion field, that differentiates between normal and abnormal ears ranges from >1 Malassezia to >4

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853
Malassezia and from >1 bacteria to >10 bacteria\textsuperscript{13,14} It is the author’s opinion that the number of organisms present to be considered significant is not just a “number”. The author doesn’t perform cytology on normal ears – it is only done if the ears that are inflamed or have exudate. Therefore ANY organism seen will be considered significant and will be treated as part of the therapy regardless of the number present. Since many times we will select our favorite otic product for the initial treatment of otitis externa, the question may be raised, why do the cytology if you are going to use your first line product in most cases anyways?. The key reason is if there are WBC or rods on the initial cytology I will ALWAYS do a follow up cytology regardless of the appearance of the ear canal since these never should be present in a normal ear. Otherwise the only time cytology is performed during therapy is when the ear is not clinically normal. If there is a single population or a mixed population of organisms (coccoi, \textit{Malassezia}) present at the initial examination without rods or WBC’s and the ear is clinically normal at the recheck examination, follow-up cytology is not performed.

In a report evaluating otitis clinical score, it was concluded that cytology was unable to differentiate between normal and affected ears and also that cytology failed to identify clinical success in otitis treatment.\textsuperscript{15} With the information gathered above, the treatment is directed toward the primary cause(s) (eg parasiticidal treatment, food trial, intradermal testing and allergen specific immunotherapy, etc) and perpetuating factors. Ear cleaning is performed in the clinic with a bulb syringe or by retrograde tube flushing with a red rubber tube (under anesthesia). If on the initial examination the ear canals are swollen and painful, ear cleaning may not be performed on the first visit, preferring to use topical glucocorticoids (GC) and systemic GC for 10-14 days to decrease the swelling. Once the swelling has decreased it will be much easier to examine the ear canals and visualize the TM.

Cleaning agents contain substances that soften and emulsify wax and lipids. This initial cleaning is necessary in order to remove debris that may interfere with the effectiveness of topical agents and to reduce inflammatory debris (bacterial toxins). The author doesn’t usually have the owner do cleaning after the initial exam since it seems that many owners have trouble with just medicating the ear, let alone cleaning too. Many of the cleaners have a low pH leading to discomfort if used in an inflamed ear. A study comparing 2 ear cleaners (original formulation and then a new formulation) noted that in 38% of the cases with the old formulation and 37.5\% of the cases with the new formulation dogs had a moderate to marked avoidance to having the cleaner instilled.\textsuperscript{16} This behavior was believed to be due to either a reaction to the ear cleaner or just overall animal irritability. Also the base in the otic ointments/suspensions (mineral oil, liquid paraffin) acts as a ceruminolytic agent. In addition, a recent study calls into question whether any of the ear cleaners have any ceruminolytic activity.\textsuperscript{17} In this study the ceruminolytic activity of 13 ear cleansers was evaluated using a standardized synthetic cerumen (SSC) that mimics the composition and texture of canine cerumen. Of the tested products only Cerumenex\textsuperscript{®}, Epiotic\textsuperscript{®} and Vet Solutions Ear Cleaner\textsuperscript{®} are available in the US. The test products were incubated with mild agitation for 20 min with 500 mg of SSC previously compacted at the bottom of a test tube. Ceruminolytic activity was then assessed by quantifying the SSC removed by decantation. Overall, Otoclean\textsuperscript{(R)} (OT) was most efficacious, reaching an activity of 86–90\% followed by Netural\textsuperscript{®} (NET) with a 39\%, Specicare\textsuperscript{®} (SP) with a 23\% and Cerumene\textsuperscript{®} (CE) with an 8\% ceruminolytic activity. None of the other products displayed any ceruminolytic activity. It was concluded that, in the experimental conditions used in this study, only 1/13 products had significant ceruminolytic activity. Please note that the company that manufactures OT funded this study A follow up study by Robson, et al using Australian and US products revealed that 15/24 cleaners had <5% efficacy while only 6/24 ear cleaners had >80% efficacy-none of which are available in the US.\textsuperscript{18,19} Lastly a study was performed to test the efficacy of the 4 ear cleaner products compared to distilled water. Only the product manufactured by the study’s sponsor performed better than distilled water.\textsuperscript{20}

\textsuperscript{15} Nuttall, T. and Bensignor, E. (2014), A pilot study to develop an objective clinical score for canine otitis externa. Veterinary Dermatology, 25: 530–e92
\textsuperscript{17} Sánchez-Leal, J., Mayós, I., Homedes, J. and Ferrer, L. \textit{In vitro} investigation of ceruminolytic activity of various otic cleaners for veterinary use. \textit{Veterinary Dermatology} 2006, 17: 121–127
\textsuperscript{18} Robson DC, Morton D, Burton GG, Basset RJ. \textit{In vitro} ceruminolytic activity of 24 ear cleaners against standardised synthetic canine cerumen. (Abst.) 24\textsuperscript{th} Proceedings of the North American Veterinary Dermatology Forum, Savannah 2009, Georgia: 217
\textsuperscript{19} Robson D, Morton D, Burton G In vitro ceruminolytic activity of 23 ear cleaners against standardised synthetic canine cerumen: preliminary results Australian College of Veterinary Scientists Dermatology Chapter Science Week Proceedings 2008: Neoplasia, Oncology and Otitis
To the author’s knowledge there are no studies showing that ear cleaning has any impact on the treatment or prevention of otitis externa. However, there is a study that evaluated the efficacy of a dermocorticoid administered twice weekly in the ear canal as a long term maintenance measure to prevent recurrence of OE in dogs with atopic dermatitis. Twenty atopic dogs with relapsing (>3 episodes/year) bilateral OE were included in the study. After successful treatment of otitis externa with a topical antibiotic-antifungal-corticoid combination, dogs’ left and right ears were each randomly allocated to either an ear cleansing maintenance regimen once weekly or the same regimen followed by application of three drops of 0.0584% hydrocortisone aceponate in the ear canal two consecutive days per week. The dogs were examined on day 30 the q 60 days. At the end of the 6 month study it was determined that the probability of remaining free of relapse was 95% in group B (cleaner plus 2 consecutive days/week of a dermocorticoid) compared to 50% in group A (median time to relapse: 90 days) (P < 0.01) after 6 month. The authors concluded that twice weekly hydrocortisone aceponate application in the ear canal provides an effective maintenance regimen to control canine allergic otitis. My comment is that cleaning was not effective when compared to cleaning PLUS the steroid. I suspect if they only did the steroid the outcome who have been the same but this was not evaluated in this study.

There is frequently a discussion of the ototoxicity of agents put into ears. Remember that it is inner ear damage, specifically vestibular and/or cochlear damage that occurs with ototoxic agents, not middle ear damage. In order for a drug to cause damage to the inner ear it must either get to the inner ear hematogenously or by traveling thru the middle ear and entering the inner ear thru the vestibular (oval) or cochlear (round) window(s).

In humans because ofloxacin otic solution (Floxin Otic®) is the only topical agent to be labeled by the U.S. Food and Drug Administration (FDA) for use when the tympanic membrane is perforated, oral antibiotics have traditionally been used in this situation. However, according to otolaryngologists because the risk of cochlear damage with the use of other topical medications seems quite small, perforation alone is not an indication for oral antibiotics.

The opinion of this author is that the concern for ototoxicity due to topical medications is overstated. This position is supported by a consensus panel on reviewing the use of ototopical antibiotics. In their report they stated “There have been very few irrefutable cases of ototoxicity reported (after proper use of a topical otic preparation). Under many circumstances, it is difficult to separate the underlying disease process, which is also known to cause ototoxicity, from ototopical drug use.” They go on to state “For more than 40 years, the most common treatment has been aminoglycocide combination drops. A longstanding debate over the safety of these millions of doses given”.

The author has only seen one ototoxic reaction that was suspected to be due to a topical agent and in that case the TM was intact! Therefore, agents are chosen more for their effectiveness than the concern about ototoxicity, especially since there are very few agents that have been proven to be safe in cases of a ruptured TM. It is more important to get rid of the infection than to avoid (effective) drugs because of ototoxicity concerns. Also, just because the TM is intact doesn’t mean that the barrier function is complete, therefore, even in the presence of an intact TM it is possible to get drugs into the middle/inner ear.

In the author’s practice, technicians clean the ears and then will examine the ear to note if the ear canal is adequately cleaned – if so then the technician would have the veterinarian finish their otoscopic examination. In order to know if the ear canal is adequately cleaned the technician needs to know how to perform an otoscopic examination.

When performing an otoscopic exam, the first thing is to remember that the ear canal is “L” shaped. Due to this curve in the external ear canal, the ear canal must be straightened in order to see the horizontal canal and the tympanic membrane. This is accomplished by placing the tip of the cone of the otoscope in the opening of the external ear canal. As you advance the cone is proximally you need to pull the pinna laterally (outward). By “stretching” the pinna laterally into a straight line horizontally the ear canal becomes straight and allows examination of the horizontal canal and the tympanic membrane.

After the technician has verified the ear is as clean as he/she can get it, the veterinarian needs to evaluate the appearance of the “cleaned” ear canal to note changes as mentioned earlier in the lecture. It is also important to note if the tympanic membrane is visual and intact. If it is not visible, why is it not visible- is the ear canal too swollen? Is there a mass or a ceruminolith obstructing the ear canal? Is the dog too painful to allow thorough evaluation?

After ear cleaning topical agents are dispensed. The author prefers ointments over drops because of the impression that ointments get the drugs to the region of the tympanic membrane better than drops do (this may be a volume issue more than the formulation- it has been reported that it takes 1.0 cc of medication to get down to the TM in a medium sized (40 pound) sized dog - personal communication). The other advantage of ointments is that the base vehicle in the otic ointments (mineral oil/liquid paraffin) acts as a ceruminolytic agent.

Most topical products contain a combination of glucocorticoids, antibacterial and antifungal agents.

Before discussing a couple specific scenarios I want to address an under treated aspect of otitis- that is PAIN. If you have or someone you know has had an ear infection, you know how PAINFUL it is. We should not be examining or cleaning ears on dogs if there ears are painful without adequate sedation/analgesia!! The author has seen many dogs that were initially unmanageable when dealing with their ears that subsequently allowed otic examinations and treatments once the PAIN was gone.

Specific scenarios- note for any of the treatments the key to success is filling the ear canal with whichever product you choose to use. The recommended low volume (5-8 drops) of the otic product is a frequent cause for failure to respond to treatment.

Acute otitis (and/or infrequent) externa treatment overview. It is important to differentiate whether this is a first time occurrence, a recurrence or an unresolved infection. The only way to know this is to do follow-up examinations on ALL cases of OE. Remember that the absence of symptoms is not synonymous with resolution of the disease. This means that owners are unable to determine whether the infection is resolved and the dog must be rechecked. THIS IS WHERE THE TECHNICIAN CAN HELP RE-ENFORCE THE IMPORTANCE OF RECHECK EXAMINATIONSIn cases of chronic (recurrent and/or Recurrent/unresolved/chronic otitis externa. These are impossible to differentiate without follow up examinations and will dictate the long term management of the disease. If it is unresolved is it because of owner compliance? If it is poor compliance then this problem must be resolved! If it is recurrent (or unresolved with good owner compliance) in addition to the above, a very aggressive search is performed to identify and treat the primary, perpetuating and secondary factors. Treatment should be for a minimum of 30 days.

As you can see the technician is an important team member in the management of otitis externa.
Marijuana is formed from the dried leaves and tops of the hemp plant (Cannibus sativa) (Svienska 2008). Marijuana has been a part of recreational, religious and medical activities of a variety of cultures for over 5000 years (Krietzer 2009;Burns 2006). Indeed, it was among the most commonly prescribed medications in the United States Pharmacopeia until declared illegal in the 1930s. It subsequently has been declared a controlled substance, Class I: No currently accepted medical use and a high potential for abuse. However, particularly with the legalization of medical marijuana in several states, this classification clearly no longer applies and Schedule II status is being promoted by advocates (High potential for abuse, but less than Schedule 1, with sever psychological or physical dependence; considered dangerous, but significant clinical indication). The potential efficacy of marijuana for control of pain has led to a passage of laws allowing medical use in several states. This, in turn, has stimulated a flurry of scientific activity in an attempt to provide evidence for medical use of marijuana. Not surprisingly, pet owners have also been engaged in the conversation, with a potentially legitimate reason for administering marijuana. Not surprisingly, however, most of the scientific information generated as evidence is intended to support human, rather than veterinary use. To understand the allure of marijuana, and specifically if its promise of relief from what ails our pets is hype or hope, one must understand the plant.

Marijuana ingredients
Marijuana is a pharmacologically (and toxicologically) diverse herb. Cannibus contains at least 480 unique compounds, with their presence varying with the plant product. Plant products include, in addition to marijuana, hashish and hashish oil, formed from the resin secreted by the plant. It is important to note that the amount of any one compound in the hemp plant can vary markedly, depending on the plant part.

The most well known of the compounds in the hemp plant are the cannabinoids, a term used to refer to a terpenephenolic compounds. The discovery of cannabinoids led to the recognition of the endocannabinoid system with endogenous cannabinoids. Since their discovery, both by pharmaceutical companies and substance abusers have synthesized synthetic compounds. “Phytocannabinoids” is thus used to refer to those occurring in the plant whereas “endocannabinoids” refers to endogenous and synthetic chemicals. Endocannabinoids also appear to be important as neuroprotectants (e.g, antioxidants, inhibition of calcium influx and excessive glutamate production), for example, that associated with CNS ischemia or hypoxia, or the presence of neurotoxicants. These effects appear to be mediated predominantly by CB1 (located particularly in the dorsal horn of the spinal cord) although CB2 also plays a role, depending on the tissue (Svienska 2008). Cannibinoids also inhibit neuroinflammation (see therapeutic indications).

Close to 70 phytocannabinoids, divided into 10 classes, have been identified in the hemp plant, and particularly marijuana. Table 1 lists those associated with presumed therapeutic use (Brenneisen Ch. 2). Among the cannabinoid compounds found in marijuana, THC is the most pharmacologically and toxicologically relevant, and the most understood. It is THC that is responsible for most of the natural effects of the Cannabis plant. It acts by binding to the CB-1 receptor. CBD is the next “best” phytocannabinoid. In addition to its anxiolytic effects, it also reduces unpleasant side effects, primary due to potent inhibition of cytochrome P450 3A11 which otherwise would metabolize THC to much more potent psychoactive compounds.

In addition to the cannabinoids, marijuana contains approximately 140 different terpenoids which are responsible for its scent. The terpenoids yielded from a marijuana plant depend on the type of Cannibis (based on drug or fiber content), the part of the plant, its sex and age, whether or not it is cultivated in or outdoors, when it is harvested and the conditions at harvest, and how it is dried and stored. The serotinergic effects of marijuana (5-HT1A and 2A) may reflect the impact of these essential oils, contributing to analgesia and mood modification. Other components in the plant include nitrogen containing compounds (n = 70: alkaloids, amines); carbohydrates, including common monosacharides (n=13: fructose, glucose, mannose), selected disaccharides (sucrose, maltose), and several polysaccharides (eg, cellulose, pectin) as well as several sugar alcohols (n = 12; mannitol, sorbitol, glycerol). A number of flavonoids also are present (n=23); among them, apigenin has a wide variety of effects, including interaction with benzodiazepine receptors, resulting in an anxiolytic effect. Other ingredients include fatty acids (n=33) and others.

The target: Cannabinoid receptors
The endocannabinoid system is comprised of cicosanoid cannabinoid [CB; protein g coupled, negative to adenylyl cyclase] receptors [CBr], their endogenous ligands and the enzymes responsible for their synthesis and degradation. This system as a known contributor to physiology has been recognized for only about 25 years old. In general, the system contributes to homeostasis (Relax, Eat, Sleep, Forget and Protect; McParland 2014 At least two CBr have been identified in many species, including the dog. CB1r occurs in the brain but also occurs in some peripheral tissues (cardiovascular, reproductive, gastrointestinal). They are responsible, in part, for central and peripheral regulation of food intake, fat accumulation and lipid and glucose metabolism. The dopaminergic reward pathway is stimulated by CB1 receptors, motivating eating, smoking and substance abuse. CB2r are located principally on immune
cells, but this includes microglia. CB2 is also located on neurons where it may be associated with cell differentiation (Svizenska 2008). In the CNS, CB receptors are suggested to influence neurotransmitter release. At least 5 endogenous cannabinoids have been described, with anandamide (CB1 and 2 agonist, but higher affinity for CB1) being the most thoroughly studied. It is synthesized by post-synaptic neurons, acting as a retrograde messenger to influence neurotransmitter, and particularly GABA, release. It is extremely unstable, being rapidly hydrolyzed to ethanolamine (an antimistamine) and arachidonic acid. Cannabinoids are able to disrupt short-term memory, impair cognition and time perception, alter mood while enhancing body awareness, discoordination, sleepiness, and reduce attention focus and the ability to “filter” irrelevant information.

As with many CNS active drugs, marijuana is associated with both tolerance (higher concentration needed to impart a similar pharmacologic effect) and withdrawal (a clinical syndrome of nervousness, tension, restlessness, sleep disturbance and anxiety). However, the long elimination half-life of the most active ingredient, THC (and others) appears to preclude a clear cut abstinence syndrome (Svizenska 2008). As with other addictive agents, laboratory rodents have been demonstrated to self medicate, suggesting an addictive component. It is important to note that although interaction with cannabinoid receptors is unique among plants to hemp, other receptors are also targeted (as noted above: benzodiazepines, serotonin, others). Cannabinoid deficiency has been linked as an etiology of a variety of illnesses: ("eCB deficiency syndrome") as an etiology in migraine, fibromyalgia, irritable bowel syndrome, psychological disorders, and others (McParland 2014).

Cannabinoid receptors have been studied in a limited fashion in dogs. Initial studies focused on relevance to humans and provide evidence that dogs may react with unique behaviors.

<table>
<thead>
<tr>
<th>Compound</th>
<th>Proposed Therapeutic Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBGA (cannabigerolic acid)</td>
<td>Antibiotic</td>
</tr>
<tr>
<td>CBG (cannabigerol)</td>
<td>Antibiotic, antifungal, Antiinflammatory, Analgesic</td>
</tr>
<tr>
<td>CBC (cannabichromene)</td>
<td>Antibiotic, antifungal, Antiinflammatory, Analgesic</td>
</tr>
<tr>
<td>CBDA (Cannabidiolic acid)</td>
<td>Antibiotic</td>
</tr>
<tr>
<td>CBD (Cannabidiol)</td>
<td>Anxiolytic, antipsychotic, analgesic, anti-inflammator, anti-toxicant, antispasmodic</td>
</tr>
<tr>
<td>CBN (Cannabinol)</td>
<td>Sedative, antibiotic, anticonvulsant, anti-inflammatory</td>
</tr>
<tr>
<td>THC (delta-9-tetrahydrocannabinol; delta-8 to a lesser degree)</td>
<td>Euphoriart, Analgesic, Anti-inflammator, Antioxidant, Antiemetic</td>
</tr>
<tr>
<td>THCV</td>
<td>Analgesic, euphoriant</td>
</tr>
</tbody>
</table>

Medicinal marijuana (?)
The proposed indications for medical marijuana have included, but are not limited to behavioral, sleep and gastrointestinal disorders, neuroprotection, antispasmodic but prokinetic, anorexia, nausea, glaucoma, diabetes, immunosuppression, malaria, anti-inflammatory and, of course, pain (Table 1, Izzo 2009). However, other potential indications have included A proposed advantage of medical marijuana compared to a single drug (e.g., dronabinol, a synthetic THC [Marinol®]), is the multiple compounds contained in the plant. Two advantages are offered: 1. The compounds might act synergistically (a “synergistic” shotgun) to provide an enhanced desired pharmacologic effect while 2. at the same time, mitigating (one compound acting on another) undesirable effects. However, evidence for a synergistic benefit is lacking based on the lack of differences when THC is consumed as marijuana, versus Marinol® (humans). (Brenneisen 200X). Presumably, because marijuana contains so much THC, it may not be the most effective portion of the plant and it may contribute to more side effects (hence the question mark for this section; see also Marijuana and pets). Note that some plants may be designed to contain more or less of a specific target compound.

Pain and inflammation/Immunomodulation
The capacity for cannabinoids to control pain is among the most studied responses. They are effective in both acute (phasic) and chronic (tonic) pain. They peripherally and centrally modulate processing of nociceptive signals. They act as antihyperalgesics. CBD has demonstrated efficacy in experimental models; the effects also appear to involve transient receptor potentials (Izzo 2009). CBD also influences T-cells, causing a generalized immunosuppressive effect. A number of mechanisms of immunomodulation have been proposed, including altered interleukin or tumor necrosis factor production or release, neutrophil migration, production of specific antibodies, etc. Arthritis and psoriasis are among the chronic inflammatory diseases in humans for which CBD has demonstrated or is suggested to have some potential efficacy.
Epilepsy
Experimentally, CBD attenuates experimentally-induced seizures in animals; this may reflect reduced calcium fluxes (Izzo 2009). THCV also has been associated with some anticonvulsant effects by virtue of its inhibitory effects on CB1.

Anxiolytic
These effects have been demonstrated in healthy human volunteers (Izzo 2009). CBD exerts benzodiazepine independent effects, possibly by activating post synaptic 5-HT1A receptors.

Neuroprotection
CBD is an antioxidant and as such has been proposed for treatment of Alzheimer’s disease, Parkinson’s disease and Huntington’s disease. Restoration of calcium homeostasis may prevent apoptosis (Izzo 2009). In rodents, CBD reverses brain damage associated with ischemia.

Anti-Emeris/ appetite suppression
Again, CBD has been demonstrated in animal models to be effective for the control of vomiting otherwise not responsive to 5-HT-3 antagonists. THCV and synthetic CB1 antagonists decrease food intake.

Diabetes mellitus
CBD inhibits development of diabetes in non-obese diabetic mice, including ameliorating clinical signs of disease. This appears to reflect, in part, control of pancreatic inflammation, but also reduction of oxidative stress in target tissues (eg, retina).

Bone formation
A number of cannabinoids (essentially all in Table 1) stimulate mesenchymal stem cells responsible for bone formation and fracture healing. CBD also controls bone resorption, reducing bone loss (Izzo, 2009).

Cancer
A number of the cannabinoids (all in Table 1) have antiproliferative-ant apoptotic effects in a number of tumor cell lines. The National Cancer Institute has a link describing ongoing activities. http://www.cancer.gov/about-cancer/treatment/cam/patient/cannabis-pdq

Antimicrobial: CBC and CBG have demonstrated potent antibacterial effects towards selected microbes, including methicillin resistance staphylococci (MIC of 0.5 to 2 mcg/ml).

Finding evidence to support either the negative or positive effects of cannabis can be difficult because such information is often tainted with emotionally-mediated opinion. PRO-CON (http://medicalmarijuana.procon.org/view.resource.php?resourceID=000881) is a useful site that provides links to evidence using a categorical approach, as well as information on approval status among the states.

Designer cannabinoids
Several approaches are currently underway to bring cannabinoids to the medical arena. Hybrids typically of C. indica or C. sativa are developed with the intent of generating specific combination of properties designed for a specific purpose. http://www.leafly.com/hybrida is a website that delineates over 550 different strains, each with a differing combination of attributes.

Approved products: Several products are undergoing regulatory approval either in the Unites States or other countries. In general, these products are either concentrated forms of a single cannabinoid, or a synthetic variation of one with THC and CBD the primary compounds. In the US, Dronabinol (THC) is undergoing approval as an appetite stimulant for AIDS or cancer patients and Nabilone (THC-like) for control of vomiting in patients undergoing chemotherapy. It is used extralabel for control of pain. In the UK, nabixomol is a combination of THC and CBP (1:1) undergoing approval for treatment of spasticity associated with multiple sclerosis and epidiox is a synthetic CBD analogue undergoing phase 1 clinical trials for treatment of pediatric epilepsy.

Synthetic Cannabinoids. An increasing problematic issue is the synthesis of cannabinoids. Modification of an R group on the cannabinoids that does not alter the psychotropic effects results in a compound that is not in the list of illegal drugs. Such products are sold in a variety of stores as “non-illegal substances” under names such as “spice” and other names. The DEA has passed emergency laws that are intended to make illegal the sale of any compound that is based on modification of cannabinoids. However, testing of such products is difficult because of the ease with which chemical modifications are made.

Regulatory considerations
In 1937, as the prohibition on alcohol ended, rather than focus on the potential benefits of the cannabis plants, the US government chose to prohibit it. In 1996, California ended this prohibition with the Compassionate Use Act which provided for the use of medical marijuana. Many states have followed suit, with two states legalizing marijuana for recreational use. (http://www.governing.com/gov-data/state-marijuana-laws-map-medical-recreational.html) Currently, at least 24 (PRO-CON). States have approved marijuana in some form. According to NORML (http://norml.org/states), a site dedicated to law reformation, 34 states have some type of conditional use, 15 states of decriminalized use, 14 states of medical marijuana laws. However, throughout the US cannabis is a controlled substance meaning it has a high risk of abuse potential and no recognized medical benefit. Cannabidiol, the major cannabinoid other than THC often cited for medical use also falls under the same category; potentially, sale of products containing less than 0.3% CBD is allowed, although this was not confirmed by the DEA. 10 different pharmaceutical cannabis products (including synthetic) are undergoing some level of approval (http://medicalmarijuana.procon.org/view.resource.php?resourceID=000883) which begs the question regarding medical benefit.
Marijuana and pets
Regulatory issues regarding the use of medical marijuana in pets are unclear and likely to remain as such for some time. Legalization in states has yet to include veterinary medicine. As such, most of the information surrounding marijuana and pets is from a toxicologic standpoint. THC is among the compounds cited as a toxicologic hazard in detection (police) dogs (Llera 2008). It is the most common drug to which detection ogs are exposed. Both dogs and cats may become intoxicated with smoke inhalation as well as ingestion of food containing marijuana (or hashish). It is absorbed rapidly following either oral or inhalant administration with clinical signs evident within 30 to 60 minutes of ingestion, although one reference (Osweiler 2008) indicates onset as long as 12 hours after exposure. Cannabinoids of medical significance appear to undergo first pass metabolism and as such, the risk of toxicity with inhalant products is much greater. The implication for medical use is that oral administration may not be cost effective. The drug is eliminated by hepatic metabolism and biliary excretion with elimination being complete in 5 days in dogs; duration of toxicity ranges from 30 minutes to 3 days, but 18-24 is the average. Enterohepatic circulation contributes to the prolonged half-life. The most common signs of toxicity following ingestion in dogs include tachycardia, hypotension, depression, ataxia, vomiting (inducing emesis is not recommended in clinically depressed dogs because of the risk of aspiration), altered behavior, bradycardia, hypersalivation, weakness, hypothermia and seizures. Legalization of medical or recreation marijuana among the states is likely to be associated with an increased incidence of toxicity, with a 4 fold increase cited in one study (Meola 2012). Treatment is largely supportive, with sedation with benzodiazepines or phenothiazines as needed. antiemetic therapy may be indicated.

Among the approaches that veterinarians can take regarding the use of medical marijuana in animals is the use of “legal” (“non-illegal”) hemp-based products. For example, commercially available dietary supplement purportedly consisting primarily of hemp stem are being marketed for dogs. Because the stem contains very little THC but a high proportion of CBD and other non-psycotropic cannabinoids, as well as flavonoids, terpenoids and other potentially beneficial compounds, product claims include effective analgesia without psychotropic effects. While such a product is more appealing than marijuana as an analgesic, neither data supporting efficacy nor safety nor quality assurance data is thus far available. No data exists regarding the efficacy of any portion of the hemp plant as an analgesic, or for other therapeutic indications in companion animals.

References
Tips to Technicians from a Soft Tissue Surgeon
Harry Boothe, DVM, MS, DACVS
Auburn University
Auburn, AL

The pre- and postoperative care of the patient is often a critical determinant of outcome in the surgical patient. The veterinary technician is an integral part of the team that provides perioperative care to the soft tissue surgical patient.

Pre-surgical considerations
Consider the following factors when assessing operative risk: patient age, urgency of the situation, nature of the clinical problem, benefits of surgical intervention vs. outcome without surgery, anticipated duration of surgery, and presence of concurrent medical problems. While age itself is not a disease, older animals may have compromised function of various organ systems that may adversely affect anesthesia or surgery. Decisions regarding risk vs. benefit of surgical procedures are impacted by personal clinical experience; however, the veterinary literature also provides a useful perspective. In general, emergency surgical procedures have a greater risk than elective procedures. Procedures lasting longer than 90 minutes have a greater potential for insult to the patient, including wound infection, compared to shorter duration procedures. Patients with concurrent medical problems may pose greater risks for surgical and anesthetic complications.

Effective client communication is a prerequisite for a successful surgical procedure. The surgeon and veterinary technician are integral components in discussions with clients regarding surgery, including benefits, risks, potential complications, prognosis, possible alternative courses of action, pre-surgical work-up, operative and postoperative courses, and financial responsibilities. The more effective the communication, the lower will be the opportunity for misunderstanding or disappointment.

Surgical planning has three components: 1) pre-surgical course of action - patient evaluation, imaging appropriate to the procedure, pre-surgical treatment, and patient preparation; 2) intra-operative plan - support personnel needs, surgical approach, equipment needs, and a plan for provision of postoperative enteral nutrition; and 3) postoperative needs - imaging, oxygen administration, nutritional and fluid support, analgesic protocol, medication needs, and bandage application.

Pre-surgical patient evaluation should include a complete physical examination that focuses on the affected body system(s) and cardiovascular and respiratory systems. Laboratory work-up should be appropriate for the surgical procedure, the presumed diagnosis, and patient status. Younger patients may be effectively evaluated by using quick assessment tests (qats: PCV, total solids, glucose, and BUN). Older patients or those with concurrent medical problems often have a more extensive pre-surgical evaluation, including a CBC, biochemical panel, and electrolyte assessment. Imaging appropriate to the procedure (e.g., radiographs, ultrasound, CT scan) should be performed, with consideration given to timing of diagnostic procedures that require anesthesia (e.g., CT scan).

Pre-surgical treatment and patient preparation
Proper pre-surgical treatment has the goal of minimizing patient morbidity and maximizing success. Considerations include correction of electrolyte and acid-base abnormalities, replenishment of blood volume and red cells, and medication administration that is specific and appropriate for the procedure (e.g., phenoxybenzamine in patients with presumed adrenal neoplasia).

Correction of electrolyte and acid-base disturbances ideally should be based on serial determinations. Maintenance of electrolytes within the reference range should help minimize patient morbidity and mortality. Replacement of red blood cells may be necessary when the PCV drops below 20% or when the hemoglobin concentration is < 7 gm/dl. Ideally, one should verify the patient’s post-transfusion PCV prior to surgery. Timely administration of medication appropriate for the surgical procedure or surgical condition may effectively prevent select postoperative problems.

Appropriate surgical site preparation includes proper clipping and scrubbing of the surgical area. One important consideration is the size of the surgical area to be prepared. Anticipation of the need to manipulate surrounding tissue (e.g., a limb) or to remove normal margins around a mass should be factored into how large the surgically prepared area should be. Use clipper blades that are clean, sharp, and well lubricated. Keep the clipper blades parallel to the skin, and begin the clip in the same direction as the hair growth. Finish the clip by directing the clippers in the opposite direction of hair growth. Disinfect clipper blades for 20 minutes in either 70% isopropyl alcohol or 2% chlorhexidine solution. Scrub the clipped area using either a chlorhexidine- or povidone iodine-based scrub. Patient skin irritation occurs more commonly with povidone-iodine vs. chlorhexidine-based products. Follow manufacturer’s recommendations when using specific scrub products. Consider using sterile gloves to provide a final sterile prep of the clipped area with the patient positioned on the surgery table. This final prep follows an initial prep that is performed prior to transporting the patient to the operating room.

Perioperative antimicrobials
The most frequently administered perioperative medication is an antimicrobial agent. The use of perioperative antimicrobials is based on the potential for bacterial contamination to occur during the operation and any additional contributing patient factors (e.g.,
underlying disease). Cefazolin (22 mg/kg IV) is the most commonly used perioperative antimicrobial agent in veterinary patients. Perioperative antimicrobials are designed to prevent wound infection; thus, they are administered before any bacterial contamination occurs. Duration of antimicrobial use is short (i.e., < 24 hours). Generally, 2 or 3 doses of cefazolin are administered: dose #1 immediately preoperatively, dose #2 90 minutes later, or at the conclusion of the procedure if surgical duration is < 90 minutes, and dose #3 immediately postoperatively. Patients undergoing clean-contaminated or contaminated surgical procedures usually receive a perioperative antimicrobial agent. Patients undergoing a dirty surgical procedure usually require therapeutic antimicrobial agents; such agents are started pre-surgically. Patient factors may warrant use of perioperative antimicrobials in clean surgical procedures (e.g., orthopedic procedures with implants).

Perioperative analgesics
Pain management is appropriate in the patient slated to undergo a surgical procedure. Use of perioperative analgesics results in a quicker return to normal behavior by the patient. Intervention prior to the onset of painful stimuli (pre-emptive analgesia) decreases the analgesic requirements. Providing analgesia also reduces the incidence of adverse sequelae (e.g., sepsis and hypoventilation). Opioids are employed commonly in the perioperative period, including those administered by constant rate infusion. Injectable analgesic agents are replaced by oral analgesic agents prior to dismissal of the patient.

Nonsteroidal anti-inflammatory drugs produce analgesia and reduce inflammation. While they are approved for perioperative use, the author prefers to delay administration until the early postoperative period. Monitoring blood pressure and observing for evidence of gastrointestinal ulceration, bleeding disorders, or renal compromise should be performed.

Fluid therapy in the surgical patient is often continued for at least 24 hours after surgery. Such therapy helps maintain tissue perfusion and circulating blood volume in the immediate postoperative period. Once oral intake of food and water commences, intravenous fluid supplementation is usually stopped.

Operative considerations
A good working knowledge of aseptic surgery and its associated principles is essential for the technician functioning as an intra-operative assistant. Ability to properly open packs and set up and maintain a sterile field also are essential skills. Whether the technician scrubs or serves as a non-scrubbed OR assistant, their participation can help assure both asepsis and enhanced intra-operative efficiency.

Postoperative considerations
In general, take care to assure that complications associated with surgery and anesthesia are minimized. Monitor the patient closely and continuously, particularly in the early postoperative period. Keep the patient warm and dry. Apply and change bandages appropriate for the surgical procedure. Change the recumbent patient’s position to prevent hypostatic pulmonary congestion, atelectasis, and decubital ulcers. Administer supplemental oxygen via nasal catheter or other means, as appropriate. Continue analgesics and fluid support until they are no longer needed. Promptly provide oral nutrition appropriate for the patient’s condition and surgical procedure.

References and suggested reading
Anesthesia for Surgical emergencies covers a wide variety of situations and patients can range from stable to severely debilitated. The sympathetic nervous system is stimulated in the emergency situation in an attempt to maintain blood flow and oxygenation to tissues. Almost all anesthetic drugs blunt the response to this stimulation which can change a patient from compensatory shock to decompensatory shock quickly. Therefore the anesthetist has to have a strong grasp on assessing patient stability before the procedure, monitoring the patient critically and identifying problems swiftly. Pain control is also critical as unchecked pain becomes extremely difficult to address and can also lead to untoward sympathetic stimulation.

Monitoring
Before sedation/anesthesia it is necessary to obtain a minimum database including: PCV, TP, BG, BUN, electrolytes, lactate, acid-base status, urine specific gravity, ECG, blood pressure and pulse oximetry. Intravenous access should be attempted before sedation if at all possible unless the patient is severely distressed or fractious and in that case, IM or SQ premedication can be performed. Regardless of the type of emergency, there should be one dedicated technician monitoring the patient once sedation/anesthesia is initiated. These patients are dynamic and their status can change dramatically over a matter of minutes.

Specifics on monitoring CV (HR, EKG), Respiratory (SpO2, capnography), and blood pressure will be discussed but follow the American College of veterinary Anesthesiologists recommendations.

Specifics pro’s and con’s of the following drug types will be discussed in detail
- Sedatives/Tranquilizers (Diazepam, midazolam, Acepromazine, etc)
- Anticholinergics
- General Anesthetics
- Opiods
- NSAID’s

This lecture will also include case examples for some of the most common surgical emergencies

GDV- Patients are often hypovolemic and require aggressive fluid resuscitation (endpoints will be discussed). Gastric decompression should be done as soon as possible and before anesthesia is initiated. Electrolyte abnormalities and acid-base imbalances should be corrected and cardiac arrhythmias should be treated. IV access (two preferably) is necessary and all emergency drugs should be calculated before anesthesia.

Opiods +/- a benzodiazepine can be used for sedation for decompression/premedications. As opioids can cause vomiting, the best choices are fentanyl, methadone, buprenorphine IM or IV.

Induction protocols include Ketamine + benzodiazepine OR fentanyl-midazolam-ketamine combination for patients in severe shock. Propofol should be avoided due to hypotension following IV administration.

Dystocia- Anesthetic requirements are reduced due to decreased MAC/FRC and increased progesterone levels. IV catheter, clipping and prepping can call be done before anesthesia in calm patients. Premedication should be kept to a low dose opioid or none at all. Alpha 2-adrenergic agonists (Xylazine, Dexmedetomidine) should be avoided as they have been associated with a decreased survival rate in neonates. Local blocks along the linea can provide intra-postoperative pain relief

Propofol is the induction agent of choice even though it crosses the placenta, because it is rapidly cleared and minimal fetal depression occurs. Postoperatively Buprenorphine can be administered with few side effects and acceptable analgesia OR a postoperative epidural with morphine.

Hemoaodbenem- These patients commonly present in hypovolemic shock and although definitive treatment to stop the bleeding is important, the patient must be stabilized first. Endpoints for resuscitation include heart rates below 150, systolic blood pressure above 80mmHg, and if possible a PCV at or above 24%. Unfortunately, many patients will need surgery to stop the bleeding (bleeding masses). Colloids +/- blood products are often necessary during surgery and the same premedication/induction protocols for a GDV apply.

Urethral Obstruction- Cats are often overlooked when discussing emergency surgical procedures but UO is a very common disease that requires emergency sedation/anesthesia in often times very sick patients. Before any drugs are administered, the patients electrolyte and acid-base status must be evaluated and hydration assessed. IV fluids (preferably 0.9% NaCl) should be initiated rapidly, even before decompression of the urinary bladder to help resolve hyperkalemia. Severe hyperkalemia should be addressed with calcium gluconate (stabilizes cell membranes to minimize cardiotoxic effects) and dextrose (promotes intracellular translocation of potassium) and an EKG should be monitored closely for bradycardia.
After IV catheter placement, chemical restraint for urethral catheterization can include many different protocols

- For an otherwise healthy cat:
  - Ketamine + benzodiazepine + buprenorphine (slow onset ~30 min).
  - Kitty magic classic (Dexmed, torb, ketamine) OR can substitute an opioid for torb (hydro/buprenex/methadone/fentanyl).

  If there are periods of respiratory depression with a UO, acidosis gets worse so be careful with pure opioids (consider premixed with Buprenorphine) and dexmedetomidine.

  Induce with Ket/Val- not as much respiratory depression as Propofol- remember most animals not intubated. In dogs, most times intubated so Propofol ok as intermittent positive pressure ventilation performed to prevent respiratory acidosis.

  In cats with structural heart disease, not usually systolic (restrictive/unclassified or HCM) so a diastolic disease. There for the increased afterload of dexdom is ok. Ketamine increases work load of the heart- don’t use in dogs with heart disease because more likely to get arrhythmias than cats.

  Ocular Emergencies (Proptosis)- Ocular emergencies are often caused by trauma so a complete evaluation of any life-threatening injuries need to be made before any sedation/anesthetic event. Short-acting or reversible drugs are preferred and premedication with an opioid/benzo/anti-cholinergic is recommended. Opioids less likely to induce vomiting are ideal. Anticholinergics are included to reduce the risk that the surgeon may induce the oculocardiac reflex and many brachycephalic patients have a high resting vagal tone. Induction with Propofol allows for a rapid safe induction.
Feral Cats and Animal Welfare
Jenifer Chatfield, DVM, DACZM
Dade City, FL

Feral, or free-roaming, domestic cats are one of the top threats to biodiversity world-wide. In addition, feral cats present a persistent and nearly ubiquitous public health threat. However, these threats seem to go unrecognized by most people, including some veterinarians. Several management strategies are employed to mitigate the severe impact of feral cats on the environment and on the public health. Any discussion of management strategies and search for better methods of control for these cats typically results in very emotional exchanges. The incredible passion on both sides of these discussions makes arriving at a reasonable solution to mitigate the significant and on-going damage from these cats nearly impossible.

Feral cat populations continue to rise as an effective management strategy for reducing the population and mitigating the impact on sensitive ecology remains elusive. Veterinarians must find a way to continue constructive and professional discourse in search of a better strategy overall.

Summary
1. Feral cats are a source of infectious disease.
2. Feral cats have a significant negative impact on endangered and endemic populations
3. Currently employed management strategies are variably effective at reducing feral cat populations in a reasonable timeframe.
4. Veterinarians should encourage responsible pet ownership, including discouraging the feeding of feral cats.
5. The feeding and maintaining of feral cat colonies is incongruent with a common-sense approach to public health and animal welfare.

References/Suggested reading
Patrick Foley, PhD, Janet E. Foley, DVM, PhD, Julie K. Levy, DVM, PhD, DACVIM, Terry Paik, DVM. Analysis of the impact of trap-neuter-return programs on populations of feral cats. JAVMA December 1, 2005, Vol. 227, No. 11, Pages 1775-1781.
The Little Things to Make Euthanasia Better in Clinics
Mary Gardner, DVM
Lap of Love Veterinary Hospice
Bandon, OR

The euthanasia appointment is one of the most emotionally challenging appointments for the entire staff (and owner). This lecture will go over all aspects of the appointment including how to handle the initial phone call, discussing the processing, handling payment, technical aspects of euthanasia and body care.

Being good at death
We are not taught to be good at death. No one taught me how to walk into an exam room for a euthanasia, what to say to a crying teenager, or whether or not to hug the old man that just lost the last piece of his late wife. I received no direct guidance about the proper verbal and non verbal techniques that make this “most difficult appointment” just a bit easier on everyone, including myself. And from our numerous discussions with new grads, it’s a common theme; about 75% of veterinarians graduate without ever administering the life-ending medication. It’s no wonder why our lectures are packed at conferences and why our hospice practice has more requests for externs than we can handle. We simply weren’t taught the intricacies of death, and as the only medical profession licensed to euthanize, we have an incredible privilege and responsibility to handle this procedure properly.

Euthanasia
If there is one thing to think about when approaching the euthanasia appointment, it’s “What would I do for my own family’s pet?” This involves not only you, but your immediate nonveterinary family as well. What could you do to help the ones you love through the process? Now make sure that is the minimum standard of service and care you give each of your patients and their caregivers!

Here are some tips to put this into practice:
The entire euthanasia process can be broken down into 4 stages:

Setting up the euthanasia appointment

- **Be the first to say the “E” word.** Clients hate to be the first ones to bring up “euthanasia.” They think you will judge them for not caring about their pet or that you will be mad at them for giving up too early. Be the first to say it. And even if they’re upset at you for the suggestion, at 2:00 am when they’re stressed because their dog is pacing all night or their spouse is yelling at them because their elderly cat has peed outside the litter box for the third time that day, they will know that you gave them permission to think about the next step.

- **Making the appointment:** How your support team handles this initial contact with the client is crucial. It took the owner a lot of nerve and emotion to call; many feel that they are making the appointment to kill their best friend. Guilt, worry, anxiety, sorrow are just a few of the ingredients in their emotional cocktail. The receptionist should have nothing else on their mind but assisting that client. They should not be put on hold, the receptionist should not be checking out another client at the same time, and if at all possible, background noise should be kept to a minimum. Most importantly, empathy must be conveyed; *I’m so sorry you’re facing this.* Do not be scared to show them some emotion, they want to know that you care.

During the appointment

- **The Arrival:** When the time for the appointment comes, everyone in the clinic should be prepared. The paperwork should be ready, dated, and IN the room. The room itself should be set up properly and one person should be prepared to assist the client. Meet the family at their car prepared to help them into the clinic. Even holding the door open while the owner manages the cat carrier is a huge help to the client. And of course, shuttle them to the room immediately.

- **Paperwork is best completed at this time before reality sets in with the family.** Again, emotions will only get deeper from here, not lighter!

- **The Space.** The room itself is very important. Regardless if it’s a separate comfort room or a regular exam room, you must do your best to make it as warm and comfortable as possible (it should not be the ‘cold sterile’ environment owner’s dread).

- **The veterinarian should go into the room and preferably not leave again until the pet has passed unless the owner requests time alone.** Go in with sedation and euthanasia already pulled up in syringes in your pocket, or given to your technician. Speak to the client and make a visual assessment of the pet. Do not pass judgement or appear to be uncomfortable with the decision unless you are certain you will not euthanize. Your discomfort will leave a family with guilt for years.

- **When explaining the euthanasia process, it is important to give the owner peace of mind that it is a gentle process.** Explain that euthanasia means “good death” and that the medication is an overdose of anesthesia, in which they go to sleep and don’t wake back up
• Offer them some time alone with their pet. If they want time alone, hand them the ‘ringer’ portion of a wireless doorbell. Have the ‘bell’ portion in the treatment room or give it to the technician assigned to the case. That way the owner does not have to leave the pet to find someone when they’re ready. The human animal bond should never be broken. Generally people do request a few minutes alone, but it’s usually a very short amount of time.

• **The Procedure:** Intra-muscular or subcutaneous sedation is crucial for the client’s experience and we are always discouraged to learn how many do not sedate pets before euthanasia, or provide only IV sedation (in which their pet rapidly goes from consciousness to unconsciousness, appearing dead). Having 5 minutes for the pet to slowly relax gives the owner time to watch their pet get comfortable. Many times I hear “I haven’t seen him this calm and relaxed in months!” We call this “secondary sedation of the owner.”

• When it comes time for the final medication, ask the owner “Max is ready, are you?” Never proceed without them fully knowing what is about to happen. They should also know that their pet will pass in 30-60 seconds. All too often owners do not realize it occurs as fast as it does. Whether you use an indwelling catheter, butterfly catheter, or straight needle, do your best to stay out of the way of the owner. Let them hold their pet and instruct them to “keep talking to her, she can hear you.” Giving them something to do keeps their focus off you and this surreal moment for them.

• After administration, listen for the heart and remain silent unless the owner speaks. This is an important moment and must be honored.

• Stay present in the room for a few minutes as you gather the syringe and supplies. Watch for agonal breath(s), twitching, or any other movements, which generally happens within 1-5 minutes post mortem. Since we do not recommend warning about all these side-effects before, this is the time to explain them if/when they occur.

**Memorial items**

• The paw print is the most traditional and cherished memorial item, even more than cremains sometimes! Every pet owner should be given one at the time of the appointment and given to the owner to take home that day (at no charge!). With air dry clay like Crayola Model Magic, this is inexpensive and takes very little time. Many clinics make the paw print after the clients leave but you are missing a huge opportunity to make the owners feel a little bit of joy at such a devastating moment.

**Body care**

• Never allow the owner leave their deceased pet alone. If they need time alone after the euthanasia, allow them that time and hand them the wireless doorbell again. This way, a technician can come back into the room as they leave.

• Know your crematory well. Understand how they do things and be confident they are providing the level of service your client’s deserve.

If there’s one thing we can tell you to improve your end of life care for pets and their families, it’s to provide the best from the get-go. Provide the kind of care that exceeds the expectations of 95% of the population out there. Do not cater to the 5% of people that are irregular.

The euthanasia appointment should not be the end of the client relationship, it should be the beginning of the next relationship you have with them! And remember, if it were your own pet, what would you do?
Sample Collection and Diagnosis of Tritrichomonas in Cats and Cattle
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Collection of sample
Cattle: Collection of preputial scrapping or semen is placed in BioMed Bovine InPouch filled with Diamond’s media.
   Cats: Fecal material the size of a pea is placed into a Feline InPouch kit
   Media is forced into top chamber of the InPouch and the sample is inoculated into the media in top chamber. Media is forced into bottom chamber. Label pouch with required info.

Processing of sample
Either ship overnight to diagnostic lab or place in incubator at 37C. Examine each pouch daily for 7 days using compound or culture flask microscope. Scan at 10X in at least 6 different fields examining pouch for characteristic undulating movement of trichomonads. Any positive samples should be further tested using PCR to determine exact Trichomonad species.

PCR testing
Used to identify the DNA of parasites. Positive PCR products should be sequenced. PCR negative does not necessarily mean that the animal was not infected with particular parasite, it just means the DNA was not amplified. Should be interpreted with caution.
Degenerative Myelopathy
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Degenerative myelopathy (DM) is a progressive disease of the spinal cord that affects many dog breeds. There are not any evidence based medical approaches for the treatment of DM; therefore veterinary technicians can play an important role in the management of these cases.

Pathogenesis
DM was first described in the German Shepherd Dog, and has historically been considered a disease that results in upper motor neuron dysfunction of the pelvic limbs.1 It has recently been realized that DM affected dogs also develop lower motor neuron signs later in the progression of the disease.2 Dogs with upper motor neuron disease exhibit a spastic gate or paralysis; while dogs with lower motor neuron disease exhibit a flaccid paralysis.3 DM is now classified as a degenerative disease that affects the axons of the spinal cord and peripheral nerves.

Signalment
Age at onset of clinical signs is usually older than 8 years with a mean of 9 years in large breeds.1 A study of affected Pembroke Welsh Corgis reported a mean age at onset of 11 years.4 There is no sex predilection. The disease has been histopathologically confirmed in many breeds with some of the most common being the German Shepherd Dog, Pembroke Welsh Corgi, Rhodesian ridgeback, Chesapeake Bay retriever and the boxer.5

Clinical signs
The key clinical signs of DM are progressive asymmetric ataxia (incoordination), spastic paraparesis (hind end weakness), and lack of spinal hyperesthesia (pain on palpation of the spine). These signs will progress to paraplegia and eventually will involve the thoracic limbs as well. The disease course to paraplegia can vary but most large breed dogs will progress to nonambulatory paraparesis within 6 to 9 months after onset of signs.3 Owners of large breed dogs often elect to euthanize when their dog is no longer able to support weight in the hind limbs. Small breeds are more easily cared for by the owner for a longer period of time. The median disease duration in the Pembroke Welsh Corgi was 19 months.5

The earliest clinical signs are asymmetric general proprioceptive ataxia and mild paraparesis. Asymmetry of signs is commonly reported but as the disease progresses the signs become symmetric. The paraparesis will progress to paralysis and ascend to affect the thoracic limbs. Flaccid tetraplegia occurs in the late stage. Muscle atrophy becomes evident in the pelvic limbs as the signs progress. Atrophy of the appendicular muscles occurs in the late stage. Difficulty swallowing, inability to bark and urinary and fecal incontinence can also occur in the late stage of the disease.2,4

Diagnosis
A presumptive diagnosis is based on clinical signs along with a series of neurodiagnostic procedures to exclude other disease that can mimic DM. The neurologic examination is critical in order to develop a diagnostic approach. The lack of spinal hyperesthesia is a key sign that distinguishes DM from other compressive myelopathies.4 Cerebrospinal fluid (CSF) analysis is done to rule out inflammatory disorders, electrodiagnostic procedures can be done to rule out other peripheral nerve and muscle disorders, and myelography, CT and/or MRI of the spinal cord can be done to rule out a compressive disease such as intervertebral disk disease or neoplasia.3 If no signs of other disease are found on these tests a presumptive diagnosis of DM is made. A definitive diagnosis can only be made through postmortem histopathology.1

A mutation in the superoxide dismutase 1 (SOD1) was discovered as the underlying cause for most forms of DM.2 Mutations of this gene in humans are an underlying cause for some forms of amyotrophic lateral sclerosis (ALS- Lou Gehrig’s disease). A DNA test for the SOD1 mutation is now commercially available. Dogs that test homozygous for this mutation are at risk for developing DM and will contribute one chromosome with the mutation to their offspring. However, some dogs that test homozygous never develop clinical signs, which suggest an age-related incomplete penetrance. Dogs that test heterozygous are considered carriers and are less likely to develop clinical signs but could pass on a chromosome to their offspring. Dogs that test homozygous normal are unlikely to develop DM and will pass on a protective normal allele to their offspring.5

Management strategies
Pharmacotherapies for the treatment of DM have been empiric with a lack of evidence-based medical approaches for determining efficacy. Aminocaproic acid has been promoted for long-term management; however, a recent study evaluated a combined therapy of aminocaproic acid and N-acetylcysteine with vitamins B, C and E, found no beneficial effects.7 Kathmann et al. reported data from 22
affected dogs that received varying degrees of physical rehabilitation. Dogs that received intensive physical rehabilitation had longer survival times (mean of 255 days) compared to dogs that received moderate (mean of 130 days) or no physical rehabilitation (mean of 55 days). The rehabilitation regimen consisted of active and passive exercises. These results suggest that survival time was positively associated with the physical rehabilitation performed but other studies are needed to measure the efficacy of physical rehabilitation in DM-affected dogs.

The goals of a physical rehabilitation program for an affected dog should be to maintain joint range of motion, reduce spasticity, and to retard disuse muscle atrophy and loss of neuromuscular function. When developing and beginning a rehabilitation program it is important to avoid fatigue. If the dog shows persistent weakness or morning fatigue after exercise on the previous day the therapist should readjust the exercise program in order to prevent further muscle damage. Nonstrenuous activity is recommended in ALS patients and has been shown to increase quality of life. Activities should include periods of activity with rest in between. Ideally a rehabilitation plan will be implemented at the first signs of DM.

Active exercises are recommended as long as some voluntary movements remain intact. This includes standing and weight shifting exercises, as well as sit to stand exercises. Balance exercises such as walking on an uneven surface (air mattress or cushion) and other proprioceptive exercises such as weaving through obstacles or walking over obstacles (cavaletti poles) may also be performed. These exercises should generally be started slowly, beginning with about 5 minutes once a day. The frequency and duration of exercises can then be adjusted accordingly for each individual patient.

Hydrotherapy is a commonly used modality in canine rehabilitation. An underwater treadmill can facilitate active movements while supporting the dog’s body weight through buoyancy effects. For example, a dog bears 91% of its body weight with the water at the level of the hock. Increasing the water level to the stifle the dog bears 85%, and to the level of the hip the dog bears 38% of its body weight. The resistance of the water also provides resistance, which allows for an increase in muscle strength. Hydrostatic pressure of the water has been shown to reduce edema and swelling. Walking or swimming in water has also been shown to improve general circulation. The water temperature should be kept warm because of the beneficial effects of heat when applied to tissues. Heat provides an increase in elasticity and blood flow of the tissue.

Passive range of motion exercises are indicated to help prevent joint contracture, maintain mobility of soft tissue, enhance circulation and improve synovial fluid production. To perform this exercise the patient should be relaxed in either a lying or standing position. The therapist will gently flex the limb to the point of resistance then extend in the same manner. The limb is also manipulated in a “bicycling” manner to simulate a normal gate pattern. A repetition of 15-20 times is recommended at least 3 times daily.

As a dog begins to lose proprioception protective boots to protect the paws can be recommended to the owner. The protective footwear can vary but should be properly fitted and water resistant. As the dog becomes more paraparetic sling support is needed. There are many commercially available slings. It should be properly fitted in order to prevent chaffing. A wheel cart should be considered for dogs that become nonambulatory. Wheel carts provide independence for a DM affected dog but the owner needs to be aware of monitoring for fatigue. It is also important to ensure that the cart fits appropriately.

The secondary consequences of DM are decubitus ulcers, pneumonia, urinary tract infections and contracture. The veterinary technician has an important role in being aware of these disease complications. If the dog is recumbent and unable to get in to a sternal position rotation is performed every 4 hours. The recumbent dog will need to be kept on soft padded bedding and the skin over bony prominences should be assessed daily for redness or irritation. The bedding should be absorbent in order to wick away moisture. This is critical to prevent fecal or urine scalding.

As the disease progresses to paraplegia the patient will likely lose the ability to voluntarily urinate. The technician will need to teach the owner how to manually express the bladder by applying gentle pressure to the abdomen. This will need to be done 2-3 times daily. If manual expression is not possible, intermittent catheterization should be performed. A urinalysis will need to be performed routinely to evaluate for the presence of a urinary tract infection.

References
Intervertebral disc disease (IVDD) is a common debilitating and painful disease seen most commonly in dogs. It is rarely seen in cats, horses and food animals.1 Degenerative changes within the intervertebral disc may lead to disc herniation. The goals of this presentation are to 1) understand the pathophysiology of intervertebral disc disease, 2) be able to recognize the clinical signs associated with the disease, 3) know the treatment options available and 4) be familiar with the nursing care and physical rehabilitation options for patients with this disease.

Hansen first classified intervertebral disc disease as type I and type II in 1951. Type I IVDD is an extrusion of the nucleus pulposus while type II IVDD is a protrusion of the annulus fibrosis. Acute noncompressive nucleus pulposus extrusion (ANNPE) is less commonly seen but has been documented recently using magnetic resonance imaging (MRI). Management of IVDD by a combination of medical and surgical methods is now well established with high success rates (up to 95%).2 The veterinary technician can play an important role in the management of these cases both pre- and postoperatively.

Pathophysiology

The spinal cord has a segmental arrangement with each segment having a pair of spinal nerves. The spinal cord is divided into segments based on characteristic clinical signs that arise when these areas are damage. The spinal cord is divided into four segments for localization purposes. The first is C1-C5, which would cause upper motor neuron signs to all four limbs. The second is C6-T2 (brachial plexus), which would cause lower motor neuron signs to the thoracic limbs and upper motor neuron signs to the pelvic limbs. The third is T3-L3, which would not affect the thoracic limbs and would cause upper motor neuron signs to the pelvic limbs. The fourth is L4-S3 (lumbosacral plexus), which would not affect the thoracic limbs and would cause lower motor neuron signs to the pelvic limbs.

Classic upper motor neuron signs include paresis or paralysis, normal to increased muscle tone and normal to increased spinal reflexes. Lower motor neuron signs include paresis or paralysis, decreased to absent muscle tone and decreased to absent spinal reflexes. Lower motor neuron lesions will generally cause neurogenic muscle atrophy as well.

There is an intervertebral disc in between each pair of vertebrae underneath the spinal cord along the entire length of the spinal column. The atlanto-axial joint is the only one that does not have an interposing intervertebral disc.3 The intervertebral disc permits motion of the spine while providing support under movement. The annulus fibrosis is the ligament that makes up the periphery of the disc and attaches to the vertebral end plates. The nucleus pulposus is the highly hydrated central portion of the disc.1

Hansen type I IVDD is herniation of the nucleus pulposus through the annulus and extrusion of the nuclear material into the spinal canal. This extrusion of the nucleus results in an acute compressive myelopathy. This type of IVDD is most common in chondrodystrophic breeds such as the dachshund and beagle although it can also be seen in large nonchondrodystrophic breeds.1 Calcification of the disc occurs most commonly in younger dogs. This calcification leads to a degenerative nucleus and a weakened annulus. The annulus cannot restrain the nucleus which can lead to an acute disc extrusion. This disc extrusion leads to an acute compressive myelopathy.

Hansen type II IVDD is protrusion of the annulus fibrosis caused by shifting of the nucleus. The annulus protrudes into the spinal canal causing spinal cord compression. This type develops more slowly and is common in older, non-chondrodystrophic breeds such as the Labrador retrievers and German shepherds.2

ANNPE usually occurs during activity and is a traumatic injury. Excessive force on the disc results in expulsion of the nucleus pulposus through the annulus fibrosis toward the spinal cord. The nucleus then disperses along the floor of the spinal canal and does not cause compression. This causes a concussive injury to the spinal cord.1

The severity of clinical signs depends upon the extent of spinal cord compression and the rate of development. Acute extrusions generally produce more severe clinical signs as compared to chronic protrusions. Less severe clinical signs are usually seen with lesions in the cervical vertebral canal because the vertebral canal is large as compared to the diameter of the spinal cord at that site.1

Clinical signs

Onset of clinical signs with Hansen type I IVDD is commonly acute. The clinical signs are seen as a result of concussion and compression to the spinal cord. The degree of signs vary from spinal hyperesthesia only to paraplegia without pain sensation and do affect prognosis.2 Spinal reflexes may be decreased, normal or increased, depending on lesion localization. For example, a compressive lesion at L5-L6 would cause decreased reflexes in the pelvic limbs due to involvement of the cauda equina. Spinal hyperesthesia (back pain) is usually seen due to compression of the meninges or nerve roots. An animal with back pain may have a hunch-back appearance (kyphosis) and tense its abdominal muscles. Lateralization of the disc herniation may cause asymmetry of
neurologic signs. Clinical signs can progress in order of functional loss, 1) loss of proprioception, 2) loss of motor function and 3) loss of pain perception. Loss of bladder function will also occur along with paraplegia. Clinical signs of Hansen type II IVDD are commonly slowly progressive and include hind end weakness (paraparesis), reluctance to rise or jump and possibly spinal hyperesthesia. Hansen type II IVDD may also cause neurologic signs that are asymmetrical due to lateralization of the disc herniation.

Diagnosis
A tentative diagnosis of IVDD can be made based on clinical signs, signalment and neurologic examination. Spinal radiographs may show evidence of degenerate discs such as narrowing of the intervertebral disc space, or degenerative changes of the spinal column such as spondylosis. However, myelography combined with CT (computed tomography) or MRI are necessary to definitively locate the area of spinal cord compression.

Myelography is injection of a contrast medium into the subarachnoid space followed by lateral, ventrodorsal and oblique radiographs. Attenuation of the contrast medium is seen at sites of compression. CT can be used adjunctively with myelography. It can help to further delineate lateralization of the compression. CT can also be used alone. It is less invasive and faster than myelography. Calcified disc material may be seen in the vertebral canal with a non-contrast CT. MRI is the gold standard diagnostic modality for acute spinal cord injuries as it may provide better clarity than other modalities. The diagnostic modalities chosen are dependent upon the surgeon’s ability to identify the site and side of the lesion and to rule out other diseases.

Treatment
Conservative therapy is indicated for animals that have one episode with mild clinical signs, with owners that have financial constraints or for those that have other medical problems that preclude anesthesia and surgery. Conservative management consists of pain control and cage confinement, with the most important of these being the confinement. Strict cage rest is recommended for a 4-6 week period. The patient should be confined to pet crate in a quiet room. Playpens should be discouraged because the animal will still have room to jump. The animal can be taken on short five minute leash walks three times a day to eliminate. If improvement is seen, then exercise is restricted to a leash for another 3 weeks. Analgesics and anti-inflammatory drugs should only be used if the client agrees to cooperate with the confinement instructions. Prednisone can be used at 0.25-.5 mg/kg PO BID for 3 days, then taper. Nonsteroidal anti-inflammatory drugs (NSAIDs) can also be used, but not in conjunction with steroids. Tramadol is another option for pain control. Physical rehabilitation, weight control and prevention of jumping may help to reduce the risk of recurrence.

Indications for surgical management include clinical signs refractory to conservative management, recurrence or progression of signs or paraplegia. Dogs with lack of pain perception should ideally have immediate decompressive surgery. Prolonged loss of pain perception carries a poor prognosis. Decompressive procedures for IVDD include hemilaminectomy, dorsal laminectomy, ventral slot and pediculectomy. Hemilaminectomy improves retrieval of herniated disc with minimal spinal cord manipulation. This procedure involves removal of the articular processes, pedicles and lamina in order to gain access to the herniated disc. Pediculectomy can be used as an adjunct technique in cases of a bilateral approach. This approach involves removal of the pedicle of the vertebra with preservation of the articular processes.

Nursing care
Supportive care of nonambulatory dogs should include prevention of decubital ulcers, urinary tract infections and muscle atrophy. If nonambulatory, the animal should be rotated from side to side every 4-6 hours in order to prevent decubital ulcer formation. If not able to voluntarily urinate the bladder should be manually expressed by applying gentle pressure to the abdomen or the patient should be catheterized whether it be indwelling or intermittent catheterization.

Swelling of the incision site should be treated with cryotherapy. Cryotherapy can be applied to the incision for 10-15 minutes every 4-6 hours in the acute inflammatory phase. After the inflammatory phase of healing is over (about 48-72 hours post operatively), heat therapy can be instituted. A hot pack can be placed on the incision for 10-15 minutes before beginning rehabilitation exercises. Passive range of motion should be performed by moving the patient’s joints through a normal range of motion for 5 to 10 minutes at least 3 times daily. The patient should be placed in lateral recumbency and should be in a quiet relaxing environment for this exercise. Standing exercises should begin as soon as the patient is able to support some weight on the affected limbs. Support is provided as needed while the patient is placed in a standing position to ensure loading of the pelvic limbs and correct positioning of the feet. After the patient is able to maintain a standing position exercises to challenge balance such as weight shifting and wobble board exercises may be instituted.

Ambulation is allowed at slow paces in patients with voluntary motor function. Assisted sling walking or underwater treadmill hydrotherapy are used to unload weight while allowing for ambulation. Hydrotherapy can be a very effective strengthening exercise due to the resistance and viscosity of water. Strengthening exercises can be added when ambulation improves and the confinement period is over. These can include walking up and down inclines, weavc around obstacles, walking on varying textures (sand, tall grass), stepping over objects of varying size (for proprioceptive awareness) and sit-to-stand exercises.
If the patient does not have voluntary motor function electric stimulation may be used to induce stifle and hock extension and flexion and to help prevent muscle atrophy. Swimming may also be beneficial to aid in the patient’s overall stability but is not recommended until after the cage confinement period is over. Active movement of the limbs can be elicited by applying resistance to the paw, thus stimulating the withdrawal reflex. A minimum of 3 weeks of physical rehabilitation is recommended. This rehabilitation period varies depending on the patient and the extent of disease. The degree of success with physical rehabilitation varies greatly and may take several months.4

References
Neurologic Examination for the Veterinary Technician
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The neurologic examination is a skill that takes practice, but one that every technician can be proficient in. It is vital in localizing the lesion and therefore confirming a diagnosis in a neurologic patient. The goals of this presentation are to: 1) understand basic neuroanatomy, 2) become proficient in obtaining a complete neurologic history, 3) understand how to perform a neurologic examination and 4) understand the basics of localizing a neurologic lesion.

**Neuroanatomy**

The nervous system is divided into two divisions, the upper and lower motor neurons. The upper motor neuron system (UMN) is composed of the brain and spinal cord (CNS). These neurons are responsible for voluntary motor function and modulating the activity of the lower motor neurons. UMN lesions produce a set of clinical signs caudal to the level of the injury. The classic clinical signs of an UMN lesion are increased muscle tone, normal to hyper reflexes and no significant muscle atrophy.²

The lower motor neuron (LMN) system is composed of the peripheral nerves that connect the central nervous system with muscles and glands. The lower motor neurons are located in the brain stem and all spinal cord segments. Each spinal cord segment has a pair of spinal nerves originating from it. The axons extending from these segments form the spinal and cranial nerves and consists of a dorsal (sensory) and ventral (motor) root. The lower motor neuron unit consists of these cell bodies, axons, the neuromuscular junction and the muscle itself. The classic clinical signs associated with a LMN lesion are loss of muscle tone, decreased to absent reflexes and severe muscle atrophy.²

An acronym that may be used to remember how to distinguish between UMN and LMN disease while performing the neurologic examination is RAT. The three things that must be evaluated are the reflexes (R), atrophy (A) and tone (T).

**Neurologic history**

Obtaining a complete history from the owner is essential and can provide evidence that a neurologic problem is present. The history should begin with the patient’s signalment; species, breed, age and sex. Many diseases are more or less likely to occur in certain groups or breeds of animals. Young animals are more likely to have congenital disorders while older animals are more likely to have neoplastic conditions. The onset of clinical signs is the next important part of the history. It is important to distinguish if the signs are peracute (minutes-hours), acute (day-week) or chronic (week-months). A timeline is the next part of the history. It can sometimes be exact or may be difficult to determine, but it is important to distinguish if the progression of the disease has been slow, fast, static, improving or waxing and waning. Animals may also have episodic disorders in which they are normal between signs.

Some clinical presentations can be difficult to classify, such as syncope versus a seizure. If an animal is presenting with such signs it is important to first obtain a complete description of the episodes. Did the animal urinate or defecate during the episode? The duration of the episode is also important. A typical seizure is about 1-2 minutes long while a vestibular episode could last much longer. It is also important to determine whether or not the animal was conscious during the episode. Did the animal respond to the owner during the episode? Was there a post ictal phase or was the animal normal right after the episode occurred? These are the questions that can help distinguish if the animal truly experienced a seizure or if it was something else such as syncope or a vestibular episode.

**Neurologic examination**

The equipment needed for a neurologic examination is a bright light source such as a transilluminator, a reflex hammer (pleximeter), a hemostat and cotton balls. The observation portion of the exam can be done while taking the history from the owner. The dog should be allowed to move freely in the exam room or in an open area. The dog’s mental status, posture and gait can be evaluated at this time. The animal’s mental status can be documented as alert, depressed, stuporous or comatose. Behavioral changes such as aggression can also be documented at this time. When evaluating a patient’s posture it is important to evaluate the whole animal beginning with the head, the trunk and finally the limbs. A common abnormality with the head is a tilt suggestive of vestibular dysfunction or a turn suggestive of forebrain disease. Abnormal posture of the trunk can consist of scoliosis (lateral deviation), lordosis (swayback) or kyphosis (dorsal deviation). Abnormal posture of the limbs includes increased or decreased muscle tone and improper positioning.²

Gait should be evaluated by having an assistant walk, trot and turn the animal in order to observe. Abnormalities of gait may include paresis (weakness) or ataxia (uncoordinated gait). Paresis essentially means weakness while paralysis suggests loss of all voluntary motor function. The prefix para- means the hind limbs are involved. Tetra- means all four limbs are involved and hemi- means the limbs of one side are involved. For example, if a dog is paraparetic it is weak in the hind limbs. There are 3 types of ataxia; proprioceptive, vestibular and cerebellar. Signs of proprioceptive ataxia include an inability to know where the feet are in space. These animals will cross over and knuckle their feet when walking. Vestibular ataxia can cause circling, a head tilt and torticollis. Animals
with cerebellar ataxia will have a dysmetric gait, which is characterized by long movements (overreaching) and overflexion of the joints. This can look like a “goose-stepping” gait.2

The responses that maintain an animal’s upright position are the postural reactions. The hopping reaction is evaluated by lifting the three limbs that you are not testing and pushing the animal laterally to observe their ability to hop on each limb. Equal responses should be seen on all limbs. General proprioception (GP) can confirm that a neurologic disease is present. The animal is given support under the chest or between the pelvic limbs and the paw is turned over so that the dorsal surface of the paw is on the ground. The animal should immediately return the paw to the normal position. CP deficits indicate a neurologic lesion and are not cause by orthopedic disease.2

The cranial nerve examination is the next part of the neurologic exam. An abnormality of a cranial nerve indicates a problem in a localized area that is not provided by postural reactions. There are 12 cranial nerves. Cranial nerve (CN) I is the olfactory nerve, which is responsible for the conscious perception of smell. It may be inferred from the history or assessed by watching the animal walk around the room to see if they are sniffing. Irritating substances such as ammonia should not be used to assess CN I because they can also stimulate the trigeminal nerve in the nasal mucosa. CN II is the optic nerve and is responsible for sensory of vision and the pupillary light reflex (PLR). Vision can be assessed by watching the animal navigate around a room. It may also be assessed by using the menace response. To perform the menace response the examiner makes a threatening gesture with a hand or finger at one eye being careful not to touch whiskers or push air into the face. The normal response is a blink. The PLR is induced by shining a light in each eye and observing pupillary constriction in both eyes (testing both direct and consensual PLR).

CN III (oculomotor nerve) is responsible for pupillary constriction and also innervates the dorsal, medial and ventral recti, the ventral oblique and the levator palpebrae muscle of the upper eyelid. This nerve is assessed by the PLR, observing for abnormal eye movements (nystagmus) and by looking for a ptosis (drooping upper eyelid). A physiologic nystagmus, or doll’s eye, can be seen by moving the head laterally. The eyes should track towards the direction you are moving the head. This is a normal nystagmus. CN IV (trochlear nerve) is responsible for motor function to the dorsal oblique muscle of the eye and is difficult to assess. A lesion of this nerve may cause a lateral rotation of the eye.2

CN V (trigeminal nerve) is responsible for motor to the muscles of mastication and sensory to the face. There are 3 branches of the trigeminal nerve; mandibular, ophthalmic and maxillary. The mandibular nerve innervates the temporal and masseter muscles. A bilateral lesion could cause a dropped jaw. A unilateral lesion could cause decreased jaw tone. The palpebral reflex tests 2 branches of the trigeminal nerve. The medial canthi tests the ophthalmic branch and the lateral canthus tests the maxillary branch. Stimulation of the nasal mucosa will also test the ophthalmic and maxillary. The mandibular branch can be assessed by touching or pinching the jaw.2

CN VI (abducens nerve) innervates the retractor bulbii and the lateral rectus muscles. Lesions of this nerve can cause a medial strabismus and an inability to retract the globe. It is assessed using eye movements and the palpebral reflex. CN VII (facial nerve) is responsible for motor to the muscles of facial expression, sensory for taste and the inner surface of the pinna. A lesion can cause the lips, eyelids or ears to droop and will usually be asymmetric. CN VIII (vestibulocochlear nerve) has 2 branches; the cochlear branch and the vestibular branch. The cochlear branch is responsible for hearing and may be tested using a loud noise or by performing a BAER (brainstem auditory-evoked response). The vestibular branch is responsible for orientation of the head and may produce many clinical signs if a lesion is present. Unilateral disease can cause ataxia, nystagmus and a head tilt towards the side of the lesion.2

CN IX (glossopharyngeal nerve), CN X (vagus nerve) and CN XI (accessory nerve) are commonly considered together. The glossopharyngeal nerve innervates the pharynx along with the vagus nerve. It also supplies some motor fibers to the salivary glands and sensory to part of the tongue. The vagus nerve innervates the larynx, pharynx and the palate. These nerves can be assessed by eliciting a gag reflex by inserting a tongue depressor into the pharynx. The spinal root of the accessory nerve supplies the motor pathway to the trapezius muscle and can be assessed by palpating for atrophy. CN XII (hypoglossal nerve) provides motor to the muscles of the tongue and is assessed by observing the animal extend its tongue and looking for atrophy.2

Spinal cord reflexes test the integrity of specific peripheral nerves and spinal cord segments. Decreased or absent reflexes indicate a LMN lesion. Normal to increased reflexes indicate a UMN lesion. Reflexes should be done while the animal is in lateral recumbency. The forelimb reflexes include the biceps, triceps, extensor carpi radialis and the flexor (withdrawal) reflex. The biceps reflex tests the musculocutaneous nerve, which originates from C6-8. To do this reflex the examiner puts a finger on the tendon and taps it with the pleximeter. The response will be either a slight flexion of the elbow or movement of the skin that overlies the biceps brachii. The triceps reflex tests the radial nerve, which originates from C7-T1 and is done in the same fashion as the biceps. The response with the triceps should be an extension of the elbow or a contraction of the muscle. It is an unreliable reflex, therefore a decreased or absent response should not be considered abnormal. The extensor carpi radialis reflex tests the radial nerve as well (C7-T1). To do this reflex the carpi radialis muscle is tapped with a pleximeter and extension of the carpus is observed. Sensory input for the withdrawal reflex is through the radial, median and ulnar nerves. The motor output is through the axillary, musculocutaneous, median and ulnar nerves (C6-T2). To do this reflex the toe is pinched in order to elicit flexion of the shoulder, elbow and carpus.1
The pelvic limb reflexes include the patellar, gastrocnemius, cranial tibial and the flexor reflex. The patellar reflex tests the femoral nerve, which originates from L4-6. To do this reflex the limb is supported under the femur and the patellar tendon is tapped with the pleximeter in order to elicit extension of the stifle. The gastrocnemius reflex tests the tibial nerve. The tibial nerve is one branch of the sciatic nerve, which originates from L6-S1. In order to test this reflex the hock must be flexed in order to put some tension on the tendon. The tendon of the gastrocnemius muscle is then tapped with the pleximeter and extension of the hock or contraction of the caudal thigh muscles is observed. The cranial tibial reflex tests the fibular nerve, which is the other branch of the sciatic nerve (L6-S1). The belly of the cranial tibial muscle is tapped with the pleximeter and flexion of the hock is observed. The flexor reflex (withdrawal) of the pelvic limb tests the sciatic nerve and is performed in the same fashion as for the thoracic limb. Flexion of the entire limb (hip, stifle and hock) is observed. The flexor reflex is a reflex and therefore does not require involvement of the brain. An animal that has no perception of pain can still have an intact flexor reflex. The perineal reflex is tested by light stimulation of the perineum. The response observed will be contraction of the anal sphincter and flexion of the tail. This reflex tests the pudendal nerve which originates from S1-3. The cutaneous trunci (panniculus) response is done by pinching the skin on each side of the vertebrae between T2-L4 and a twitch of the skin is observed. The cutaneous trunci is innervated by the lateral thoracic nerve that originates from C8-T1.

The last portion of the examination is the sensory examination. These tests should be done at the end of the examination so that you do not lose cooperation from the patient at the beginning. Hyperesthesia is by definition an increased sensitivity of nociceptive fibers to normal stimulation. To test for spinal hyperesthesia the examiner should begin caudally around L7 and press on each spinous process firmly moving cranially. The severity of the stimulus is then increased gradually from touch to deep palpation. If there is a painful area you will observe a behavioral reaction. The order of loss of function with a neurologic lesion is conscious proprioception, motor function, superficial pain, and lastly deep pain. Therefore, if the animal does not have motor function it is necessary to check for pain sensation of the feet. To test for superficial pain sensation the skin in between the toes is pinched with a hemostat. A behavioral response such as aggression or vocalization should be observed. A withdrawal is not a behavioral response. To test for deep pain sensation a hemostat is used to squeeze a digit. Again, a behavioral response should be observed.

Lesion localization

After completing the neurologic examination an attempt is made to localize the lesion to one anatomic site of the nervous system. The nervous system is divided into five levels of the spinal cord and five sections of the brain (or peripheral vestibular). If it is not possible to localize to one region then it may be a multifocal or diffuse process. The levels of the spinal cord are divided as follows: C1-C5, C6-T2, T3-L3 and L4-S2. The brain is divided as follows: cerebrum, diencephalon, brainstem, vestibular system and the cerebellum.

When localizing a spinal cord lesion the first step is to determine if the signs present are consistent with a UMN or LMN lesion. Classic signs of a LMN lesion are decreased or absent reflexes, loss of muscle tone and muscle atrophy. Classic signs of a UMN lesion are normal to increased reflexes, no significant atrophy and increased muscle tone. Paresis or paralysis is a primary finding with both UMN and LMN disease.

Lesions that are localized to the brain are further localized to a certain area of the brain according to the clinical signs that are present. Lesions of the cerebrum will usually cause behavioral changes, seizures, vision loss with intact PLR, contralateral decreased facial sensation, contralateral hemiparesis and postural reaction deficits. Lesions of the diencephalon (thalamus or hypothalamus) may cause UMN signs in all limbs or a hemiparesis and postural reaction deficits. It may also cause circling, CN II, III IV and VI deficits depending on the extent of the lesion. The most common signs of a lesion here are related to the function of the hypothalamus. Brainstem lesions will also cause UMN signs in all limbs or on one side (hemiparesis). Cranial nerve deficits are more extensive with brainstem lesions than with other areas. Mental status may also be altered with a brainstem lesion. Signs of vestibular disease include falling, rolling, head tilt, circling, strabismus, nystagmus and ataxia. A vestibular lesion can be further localized as being central or peripheral. Distinguishing between these two is very important because of the differences in treatment and prognosis. A central lesion will cause postural reaction deficits while peripheral disease will not. Central disease may also cause an altered mental status or CN V and VII deficits.

References

Neuromuscular Disease
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There are many diseases that cause diffuse lower motor neuron signs by affecting the nerves, neuromuscular junction or the muscle itself. This lecture will focus on three of the more commonly seen neuromuscular diseases; myasthenia gravis, tick paralysis and polyradiculoneuritis. The goals of this presentation are to 1) recognize the clinical signs seen with neuromuscular diseases, 2) understand basic pathophysiology of myasthenia gravis, tick paralysis and polyradiculoneuritis, 3) be familiar with the nursing care and treatment options for patients with neuromuscular disease.

Tick paralysis
Tick paralysis is seen most commonly in the United States and Australia. It causes generalized lower motor neuron signs similar to botulism and polyradiculoneuritis, and can be devastating if not treated in a timely manner. It is thought that the toxin comes from the salivary glands of the engorged tick. The most common ticks of concern in the United States are the Dermacentor andersoni (Rocky Mountain wood tick) and Dermacentor variabilis (common wood tick), with D. variabilis being the most incriminated. Sixty four tick species have been shown to have potential to cause tick paralysis. Cats in the United States seem to be resistant, although they are affected in Australia.1

The toxin acts by either interfering with acetylcholine release at the neuromuscular junction, or by inhibiting depolarization at the terminal portion of motor nerves. It can affect both sensory and motor nerves by altering ionic influences that mediate production of the axon potential.2 Clinical signs are only present when the tick is fully engorged, therefore the incubation period is constant. The disease progression mirrors the feeding habit of the tick species involved. Clinical signs appear 5-9 days following attachment of the tick. The earliest clinical sign is acute onset ataxia, followed by rapidly progressing paresis, flaccid paralysis and decreased to absent spinal reflexes. These are signs of a generalized lower motor neuron disease. Tetraplegia will develop within 12-72 hours of onset of clinical signs. The pelvic limbs are affected first, with progression to the thoracic limbs. The tendon reflexes, such as the patellar reflex, are usually lost before the withdrawal reflex.3 Cranial nerve involvement is rare in the United States. Nystagmus may be occasionally observed.2 Some dogs may have a weak bark, which suggests laryngeal involvement. Nociception, or sensory function, along with urethral and anal sphincter function are not affected. Dogs and cats in Australia develop more severe clinical signs. Death may result from aspiration pneumonia or respiratory failure in severe cases.

Diagnosis is based on history, clinical signs and identification of the offending tick. However, in some cases the tick may fall off so you may not rule out the diagnosis based on absence of the tick. Only one tick may cause clinical signs so a thorough search of the animal is required. Electrophysiology changes include decreased amplitude of evoked motor potentials and possibly decreased nerve conduction velocity. There is no evidence of denervation on electromyography. The disease may also be diagnosed based on rapid improvement of clinical signs following removal of the tick.2 Rapid recovery is usually seen within hours following tick removal and may continue over the next few days. However, some cases may persist for weeks. Care should be taken to ensure that the mouth parts of the tick are removed. Forceps are recommended for tick removal. The tick may also be sprayed with 70% isopropyl alcohol prior to removal to make the process a little easier. Animals should also be treated with an acaricide, and thick coated animals may need to be shaved. Supportive treatment is the same as for other lower motor neuron diseases that cause flaccid paralysis. This includes rotation every 4-6 hours to prevent decubital ulcers, maintenance of hydration and nutrition, prevention of aspiration pneumonia and mechanical ventilation if respiratory compromise occurs.1 Prevention of tick paralysis is based on acaricide tick prevention, preferably one that has continuous protection such as a monthly spot on product.

Polyradiculoneuritis
Polyradiculoneuritis, or “coonhound paralysis,” is very similar to Landry-Guillain-Barre syndrome in people. It has been seen most commonly in hunting dogs that have raccoon exposure, but has also been seen in dogs with no exposure to raccoons.

The disease causes immune-mediated segmental demyelination and degeneration of axons primarily in the ventral roots and spinal nerves. It is thought that a substance in the raccoon saliva causes the disease. Clinical signs are present because transmission from the ventral horn of the spinal cord to the muscle is blocked.2 Signs usually develop 7-14 days after raccoon exposure. Neurologic signs start as paraparesis and hyporeflexia that progresses quickly as ascending paralysis. Dogs will become tetraparetic within 24-48 hours of onset of signs. The neurologic signs are consistent with a generalized lower motor neuron disease with decreased to absent spinal reflexes and decreased muscle tone. These dogs may also develop respiratory paralysis as the signs ascend. Cranial nerves are usually all intact, but dogs may have a weak bark.
Myasthenia gravis

Myasthenia gravis (MG) is a disease of the motor end plate that causes a fatigable weakness. There are 2 types of MG; acquired and congenital. Acquired MG is one of the most common neuromuscular diseases of the dog. It is less common in cats. It is seen in dogs less than 5 years or greater than 7 years of age and is more common in females. The German shepherd, Akita, golden retriever and Labrador retriever are predisposed for MG. Acquired MG is immune mediated. Autoantibodies against the nicotinic acetylcholine receptors of skeletal muscle affect the transmission of acetylcholine across the neuromuscular junction. Since there are not as many receptors available there is more likely to be failure of transmission of the acetylcholine. When the motor nerve repetitively fires, all of the available receptors are bound and the stores of acetylcholine are depleted. This causes muscle weakness and fatigue.2

There are 3 forms of MG in dogs and cats. The focal form is manifested as megaesophagus alone or facial weakness. The generalized form is manifested as tetraparesis, megaesophagus and/or facial weakness. The acute fulminating form is manifested as acute, rapidly progressive, severe muscle weakness, megaesophagus, frequent regurgitation and respiratory distress.3 MG may also be part of a paraneoplastic syndrome, with thymoma being the most commonly associated neoplasia. This is more commonly seen in cats than dogs.

The hallmark clinical sign on MG is episodic muscle weakness. Examination may be normal when the animal is rested. Muscle weakness becomes progressively worse with exercise. As the animal walks they develop a short strided gait, then lie down. The palpebral reflex may fatigue with repetitive testing. The most common clinical signs seen in dogs is generalized weakness with megaesophagus or megaesophagus alone.2 Clinical signs in cats include generalized weakness, megaesophagus and dysphagia. Megaesophagus is not as common in cats because they have more smooth muscle in their esophagus than dogs.

Definitive diagnosis of MG is made by testing for serum autoantibodies to acetylcholine receptors. A positive titer is above 0.6nmol/L in dogs and above 0.3nmol/L in cats. A Tensilon test may also help establish a diagnosis. Edrophonium chloride (Tensilon) is a short acting anticholinesterase. The drug allows more acetylcholine to be available in the neuromuscular junction to interact with the available receptors. Edrophonium is given IV at a dose of 0.1-0.2 mg/kg after induction of weakness with exercise. If the patient goes in to remission. The dosage of drugs is gradually reduced and discontinued if possible.

Summary

Neuromuscular diseases cause generalized lower motor neuron signs including decreased to absent muscle tone, decreased to absent spinal reflexes and rapid muscle atrophy. The secondary complications from these signs can be just as debilitating as the disease itself. The veterinary technician plays an integral role in the management of these cases as they require extensive nursing care.

References

Rehabilitation of the Neurologic Patient
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Neurologic disease can cause loss of motor and autonomic function as well as sensory abnormalities such as analgesia, paresthesia or hyperesthesia. The secondary complications from these problems such as muscle atrophy or contracture, decubital ulcers, urinary tract infections and respiratory infections can be debilitating. Typical rehabilitation goals for a patient with neurologic disease are to minimize pain, reestablish neural pathways, prevent secondary complications and return the animal to independent function. The rehabilitation program should be designed along with other appropriate treatments for the underlying problem.

Acute spinal cord disease
The most common conditions that cause acute spinal cord injury are Hansen type I intervertebral disc disease (IVDD), traumatic injuries and fibrocartilaginous emboli (FCE). The ability to distinguish what types of neural tissue have been damaged is critical in order to predict the expected recovery and to design a rehabilitation program. The goals of rehabilitation for acute spinal cord disease are to minimize postoperative pain, maintain joint range of motion, minimize muscle atrophy and restore neuromuscular function.

Before beginning any course of rehabilitation for a neurologic patient it is important to consider the disease process and the stage of healing for that particular patient. Many post-operative neurologic patients will need to be strictly rested for 6-8 weeks. If this is the case it is important to allow time for adequate healing before beginning many exercises. For example, with a post-operative intervertebral disc disease patient it would be appropriate to start passive range of motion and assisted standing exercises the day following surgery, as long as adequate pain control is obtained. Water therapy may be appropriate with the correct modalities and appropriate monitoring. Other exercises should be restricted until following the 6-8 week period of cage rest.

Postoperative pain from inflammation may be relieved by cryotherapy. A cold pack should be applied to the incision for 10-15 minutes every 4 hours for the first 48 hours after surgery. After the acute inflammatory period of healing is over heat therapy may be instituted. This can be accomplished using a commercially available gel pack or a homemade pack out of a sock filled with rice. The heat pack can be applied to the incision for 10-15 minutes every 4-6 hours before beginning other exercises. The patient should be closely monitored during these treatments.

Passive range of motion should be performed if the patient does not have voluntary motor function, or if proprioceptive deficits or decreased strength preclude a normal gait. Passive range of motion will help maintain joint health; however, it will not improve strength or prevent muscle atrophy. It is performed with the patient in lateral recumbency. The upper limbs are put through a comfortable flexion and extension for 15-20 cycles. The limb is then moved through a “bicycle” pattern another 15-20 times. This is repeated on each limb and is performed 3-4 times a day until the patient is ambulatory.

If the patient has upper motor neuron disease, the withdrawal reflex will cause active flexion of the elbow and carpus or stifle and tarsus, thereby improving muscle tone. This is done by putting the patient in lateral recumbency and pinching in between the toes to elicit a withdrawal reflex. When the patient pulls the leg back the therapist creates a gentle tug of war. This can be performed for 3-5 repetitions per limb, 3-4 times a day.

Therapeutic exercises are beneficial to help improve muscle strength, balance and coordination in patients that have voluntary motor function. Sit-to-stand exercises may be performed if the patient has enough strength to stand up without assistance. This exercise strengthens stifle and hip extensor muscles. It should be repeated 3-5 times and done 2-3 times a day.

Assisted walking is beneficial when the patient has some voluntary motor function. Several short walks a day will help improve strength and coordination. A sling may be used for support if needed. A land or underwater treadmill may also be used if available. The goal is to walk as slowly as possible to encourage weight bearing on all limbs for 2-5 minutes depending on the patient’s ability. Multiple short walks per day are more beneficial than 1-2 longer ones.

Once the patient is able to walk unassisted and has had adequate healing time, incline walks may be added to the program. Pelvic limb strength is required for a dog to walk uphill while thoracic limb strength is required for walking downhill. Walking downhill also requires the pelvic limbs to reach under the body, requiring flexion of the hock, stifle and hip. When beginning, start with gentle slopes and progress to steeper ones as the patient is able.

Aquatic therapy may be beneficial to improve strength and joint range of motion. Swimming may need to be delayed in postoperative patients for 4-6 weeks due to the forceful muscle contraction that is involved, however, underwater treadmill may be performed sooner. Underwater treadmill may be instituted as soon as the surgeon is comfortable with the incision going in the water. Petroleum jelly may be placed over the incision to help protect it from the water.

An underwater treadmill can facilitate active movements while supporting the dog’s body weight through buoyancy. For example, a dog bears 91% of its body weight with the water at the level of the hock. Increasing the water level to the stifle the dog bears 85%, and to the level of the hip the dog bears 38% of its body weight. The resistance of the water allows for an increase in muscle strength.
Water is much more resistant than air, making it a better strengthening exercise than land walks. Hydrostatic pressure of the water has been shown to reduce edema and swelling which may be of benefit in nonambulatory patients. Walking or swimming in water has also been shown to improve general circulation. The water temperature should be kept warm because of the beneficial effects of heat when applied to tissues. Heat provides an increase in elasticity and blood flow of the tissue. It also helps to relax the patient.

There are many exercises that may be performed to improve balance and coordination in patients that have motor function but decreased proprioception. Weight shifting may be performed by placing the patient in a standing position and gently rocking them side to side or by lifting a limb off of the ground. Treats may also be used to encourage the patient to move their head up, down and side to side to encourage shifting their center of gravity and in turn shifting their weight. Balance boards, wobble boards, balance balls, cauletli rails and air mattresses can be used to help improve balance and coordination. These exercises should be tailored to the patient and should continue until they have a normal gait.

Neuromuscular electrical stimulation (NMES) may be beneficial to increase tissue perfusion, decrease pain and delay the onset of muscle atrophy. It can be used to delay the onset of neurogenic muscle atrophy in patients with lower motor neuron disease. It is contraindicated over the incision following a hemilaminectomy. NMES should be applied to affected muscle groups once daily for 15 minutes until the patient is ambulatory.¹

**Chronic spinal cord disease**

Chronic neurologic conditions are common in geriatric large and small breed dogs. They are usually degenerative conditions such as Hansen type II IVDD, spinal malformations, degenerative myelopathy, neoplasia and degenerative lumbosacral disease. The expectations and goals for recovery are different for chronic disease versus acute disease. The spinal cord lesion in these cases often results from an underlying, poorly understood, structural abnormality. They can also be difficult to treat because of the insidious onset of signs. It is preferable to begin treatment and rehabilitation when the animal is still ambulatory. The goals of a rehabilitation program include decreasing pain, improving joint range of motion, correcting muscle atrophy and restoring neuromuscular function.¹

Passive range of motion exercises should be performed if the patient has proprioceptive deficits or decreased strength. Joint range of motion should be measured in these patients to determine a baseline and establish which joints are the most compromised in order to develop a plan. If range of motion is decreased, stretching exercises may be added to help restore function. The joint and adjacent muscles may be warmed packed, then passive range of motion is performed. When the endpoint of flexion or extension is reached the therapist exerts gentle traction. A “bouncing” motion may also be used to assist in breaking down fibrous tissue. If the patient is uncomfortable following treatment, cryotherapy may be applied.¹

Withdrawal reflex stimulation may be applied as with acute disease. It should be performed 20 times, 2-3 sessions a day.¹ Muscle atrophy may be as important as loss of neuromuscular function in patients with chronic disease, and the rehabilitation program must address this. Therapeutic exercises such as sit-to-stands, assisted walking, balance, and coordination exercises are similar to those described for patients with acute disease.

**Peripheral nerve injury**

Some of the common causes of peripheral nerve injury include brachial plexus avulsion, fractures and intramuscular injection. Peripheral nerves regenerate at 1mm per day if they are in a Schwann cell environment.¹ Peripheral neuropathies may cause abnormal sensations that lead to self-mutilation. Denervated muscle will atrophy, which may lead to contracture. The goals of a rehabilitation program are to restore joint range of motion, improve strength, restore neuromuscular function and prevent self-mutilation.¹

Passive range of motion and stretching should be performed in the same way as for acute or chronic disease to prevent contractures. NMES may be applied to affected muscles to delay the onset of neurogenic muscle atrophy. This is the modality of choice if a muscle is completely denervated.¹ Therapeutic exercises may also be performed as in acute and chronic disease. In some cases loss of neuromuscular function may preclude some of these exercises.

**Neuromuscular disease**

Neuromuscular diseases include disease of the nerve, neuromuscular junction or muscle. Some of the common neuropathies include polyradiculoneuritis (Coon Hound paralysis), infectious neuritis (Neospora) and compressive neuropathies (degenerative lumbosacral disease). Botulism and myasthenia gravis are examples of junctionopathies. There are many different myopathies, such as muscular dystrophy. There may be involvement of the esophagus or laryngeal muscles with some of these disease processes, which may cause dysphagia or aspiration pneumonia. The goals of a rehabilitation program vary depending on the pathophysiology of the disease process and the clinical signs that are present. In most cases it will include intensive nursing care, maintaining joint range of motion, preventing muscle atrophy and restoring neuromuscular function.¹

Passive range of motion and stretching exercises can be performed as previously described. Walking in an underwater treadmill is great for these patients because the buoyancy of the water makes up for their weakness. Swimming may also be used in short 1-3 minute sessions every 2-3 days.¹ Therapeutic exercises such as assisted walking and sit-to-stand exercises may also be beneficial.

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Summary
Neurologic rehabilitation involves a combination of nursing care, passive and active exercises and therapeutic modalities. Many of these exercises do not require special equipment and can even be taught to the client to be performed at home. This requires cooperation from the patient, client and therapist.

References
Cervical spondylomyelopathy, or wobbler syndrome, is a common disease of the cervical spine in large and giant breed dogs, as well as horses. There is still controversy regarding the proper terminology for this disease. There are 14 different names with the most common at this time being cervical spondylomyelopathy (CSM), wobbler syndrome, caudal cervical spondylomyelopathy, cervical spondylopathy and cervical vertebral stenotic myelopathy. Other aspects of this disease, including pathogenesis and treatment options are also controversial. There are at least 21 surgical techniques that have been proposed for the treatment of CSM. This reflects a lack of understanding regarding the pathogenesis of CSM. Recent studies have focused on understanding the mechanisms of CSM, which is the only way the treatment options for CSM will evolve. The goals of this presentation are to 1) Differentiate between disc-associated and osseous CSM, 2) Recognize the clinical signs of CSM and 3) Understand the treatment options and nursing care required for patients with CSM.

Pathophysiology

Even though many breeds of dog may be affected with CSM, 60-70% of cases seen are either Doberman Pinschers or Great Danes. There are two different types of CSM. Disc-associated CSM is usually seen in middle aged large breed dogs, most commonly Doberman Pinschers. The osseous form of CSM is usually seen in young adult giant breed dogs, most commonly Great Danes. There is current evidence that suggests that disc-associated CSM in Dobermans is hereditary.

Disc-associated CSM is primarily associated with ventral spinal cord compression caused by protrusion of the intervertebral disc. There may also be dorsal compression caused by vertebral canal stenosis or ligament hypertrophy (ligamentum flavum). Vertebral canal stenosis may be absolute canal stenosis, which then causes direct spinal cord compression and clinical signs, or relative canal stenosis, which by itself does not cause clinical signs but does predispose the patient to develop a myelopathy. There are 3 factors that work in combination to produce the clinical signs seen with disc-associated CSM. The first factor is relative vertebral canal stenosis. Affected Dobermans seem to be born with a relative vertebral canal stenosis. The second factor is torsion. The caudal cervical spine has been shown to experience more torsion than the cranial cervical spine, which may lead to intervertebral disc degeneration. The third factor is protrusion of a larger volume intervertebral disc in the caudal cervical spine. The majority of disc-associated CSM are located in the caudal cervical spine at C5-6 and C6-7. About 50% of large breed dogs have one site of compression, while 50% have more than one site of compression.

Osseus-associated CSM is seen in young adult giant breed dogs. A congenital cause seems likely due to the fact that the disease is seen at an early stage, but is yet to be proven. These dogs have absolute severe vertebral canal stenosis due to proliferation of the vertebral arch (dorsally), articular processes (dorsolaterally) or articular processes and pedicles (laterally). The cause of spinal cord compression is due to a combination of vertebral malformation and osteoarthritic changes of the articular processes. Occasionally the osseus compression may also be complicated by intervertebral disc protrusion. This is more common in older dogs. The most commonly affected sites are C4-5, C5-6 and C6-7 in giant breeds. About 20% of dogs have one site of compression, while 80% have multiple sites of compression.

The pathophysiology of both disc- and osseus-associated CSM involves both static and dynamic factors. The key static factor is vertebral canal stenosis, whether it be absolute or relative. A dynamic lesion is one that either worsen or improves with movement of the cervical spine. Neck flexion causes stretching of the caudal cervical spinal cord. Continuous flexion and extension of the neck may cause spinal cord elongation, which leads to stress and strain within the cord.

Clinical signs

The mean age of onset in Dobermans or other large breed dogs (Dalmations, Weimaraners) with disc-associated CSM is 7 years. The mean age of onset in Great Danes or other giant breed dogs (Mastiffs, Rottweilers) is 3.8 years, but may be seen at a few months old. Males and females are both affected. Neck pain is the most common historical finding. A chronic progressive history is typical.

Most dogs present with a proprioceptive ataxia. The pelvic limbs are often more affected than the thoracic limbs. A two-engine gait is commonly seen in dogs with caudal cervical spinal cord disease. This consists of a short-choppy gait in the thoracic limbs and a long-strided gait in the pelvic limbs. The thoracic limb gait may also appear spastic with a “floating” appearance that is termed pseudo-hypermetria. This is due to upper motor neuron release and is different from true hypermetria, where the limbs will over flex and cause a high stepping gait. Proprioceptive deficits are usually seen in all 4 limbs, but occasionally may not be present despite the proprioceptive ataxia. Spinal reflexes will often be decreased in the thoracic limbs and normal in the pelvic limbs, which is suggestive of a C6-T2 myelopathy. Weakness, or tetraparesis, may also be seen at varying degrees. Thoracic limb lameness is occasionally seen as a result of nerve root entrapment.
Diagnostics
Survey radiographs may be performed in order to rule out other potential diagnoses including osseus neoplasia, trauma or discospondylitis. Radiographs cannot confirm a diagnosis of CSM. Radiographic changes suggestive of disc-associated CSM include changes in the shape of the vertebral body, narrowing of the intervertebral disc space and vertebral canal stenosis. Radiographic changes associated with osseus-associated CSM include osteoarthritic changes of the articular facets and may be seen on lateral or ventrodorsal views. Stressed or traction views, where the neck is flexed or extended, should not be taken as this may exacerbate clinical signs.

Myelography used to be the modality of choice for diagnosing CSM. Myelography is performed by injecting a radiopaque contrast into the subarachnoid space by means of a cisternal or lumbar tap. Lumbar myelography is preferred because it is safer and results in better quality of images. Lateral and ventrodorsal radiographs are then taken. The contrast allows for visualization of the spinal cord and assists in the diagnosis of myelopathies. However, it is an invasive procedure and does have risks. Postmyelographic seizures occur in 3-20% of dogs, and is more likely to occur in large dogs. Transient myelitis may also occur secondary to the contrast, therefore worsening of neurologic signs is possible. If computed tomography (CT) or magnetic resonance imaging (MRI) are not available myelography may be used, but it is no longer the modality of choice. Both CT and MRI offer many advantages over myelography alone.

CT is a fast test that allows for visualization of transverse sections of the spine. In order to identify the location of spinal cord compression it needs to be combined with myelography. CT is better for visualizing the direction and severity of spinal cord compression. It is also better for identifying the most severely affected site as compared to myelography.

MRI is the gold standard diagnostic modality for the diagnosis of CSM. MRI is able to detect signal changes in the spinal cord which allows for assessment of the spinal cord parenchyma. The signal changes allows for precise determination of the most severely affected site. MRI is more precise in determining the site, severity and nature of spinal cord compression. Transverse and dorsal images may be utilized to assess for vertebral canal stenosis. Hyperintensities on T1W images combined with hypointensities on T2W images may be correlated with a worse prognosis.

Treatment
Conservative treatment for CSM consists of pain management and activity restriction. Improvement or stabilization of clinical signs is often seen with medical management and may be preferred by some owners due to financial constraints or concerns about anesthesia. The clinician may recommend medical management for dogs with multiple compressive lesions affecting the lateral aspect of the spinal cord. Some clinicians start all dogs on medical management in order to evaluate improvement seen with it and give the owners some time to make a decision about surgery. One study showed that the clinical signs are either improved or stable in up to 81% of dogs with medical management. The most important aspect of medical management is activity restriction. Dogs should be kept in a kennel except for when taken outside to eliminate. A harness should be used instead of a collar and the dog should be kept on a leash when outside with no running or jumping allowed. Offleash, unsupervised activity should not be allowed. Anti-inflammatory doses of corticosteroids are often administered with a tapering dose over a 2-3 week period. Dexamethasone may work better in some patients so may be used in severely affected patients or as a rescue in dogs that acutely deteriorate. Omeprazole or famotidine are commonly given along with corticosteroids in order to prevent GI complications. NSAIDS may be used instead of corticosteroids if neck pain is a predominate sign. NSAIDS and corticosteroids should never be given together. Reasons for successful medical management may include the slow progression of spinal changes with CSM or remyelination of surviving axons.

Surgical management is largely the treatment of choice for CSM. The decision to pursue surgery should be based on many factors including severity of clinical signs, response, or lack of response, to conservative management, type and severity of cord compression, the presence of other concurrent disease and the expectations of the owners. There are many surgical techniques that have been proposed to treat CSM. These can be categorized according to direct decompressive, indirect decompressive and motion preserving techniques. Direct decompressive surgeries would include dorsal laminectomy, ventral slot and hemilaminectomy. Indirect decompression would involve the use of pins, bone grafts, plates, spacers, screws and/or polymethyl methacrylate (PMMA).

Disc-associated CSM is the type that has been the most surgical techniques proposed to treat it. These techniques are chosen based on the compression being static or dynamic. A ventral static compression can be treated with a ventral slot. Dynamic compressions can be treated with distraction/stabilization techniques including PMMA plug or pins and screws. If there are multiple compressive lesions it can be treated with distraction/stabilization techniques, most commonly a PMMA plug. If there are multiple ventral compressions a dorsal laminectomy may be an option.

Osseus-associated CSM is generally thought to be static, therefore direct decompressive techniques are usually recommended. The dorsal laminectomy and cervical hemilaminectomy are the techniques most commonly used for these lesions. They may also be treated with distraction/stabilization of the affected segments ventrally. A PMMA plug is used for this technique.
**Prognosis**
Surgical treatment for disc-associated CSM has a good outcome about 80% of the time. Conservative management is successful about 50% of the time. Therefore, surgery leads to a successful outcome more consistently and should always be considered. However, surgery does not change the long term outcome. One study showed that the survival time of dogs treated surgically versus dogs treated conservatively was the same with a median survival time of 36 months. Studies on osseus-associated CSM have showed similar results.²

**Conclusion**
Wobbler’s disease is an all-encompassing term that is used when referring to two different disease processes. There are two different types of CSM; disc-associated and osseus-associated. The two most commonly seen dog breeds affected are the Doberman Pinscher and the Great Dane. Veterinary technicians play an important role in the treatment of these cases as many times they require extensive nursing care and physical rehabilitation.

**References**
A cat is not a dog. Period. Developing a program for a cat that needs physical rehabilitation is sometimes challenging, but can absolutely be performed. The misconception exists that cats will not cooperate when asking them to perform therapeutic exercises. If you ask them nicely, they will gladly be receptive. (You should be smiling after this statement). The success of physical rehabilitation with cats demands a good understanding of feline behavior, including excellent handling skills. This lecture will hopefully give some insights into dealing with cats in a potentially stressful situation.

Physical Rehabilitation or Physiotherapy is concerned with physical function, and considers the value of movement and the optimization of physical potential as being core to the health and wellbeing of individuals. Manual therapy (e.g. Massage, Passive Range of Motion, Stretching), Thermotherapy (e.g. Hot and cold), Electrotherapy (e.g. Laser therapy, Ultrasound therapy), Neuromuscular electrical nerve stimulation (NMES), Exercise Therapy (e.g. Basic exercises for the postoperative orthopedic and neurological patient, Hydrotherapy, Strengthening exercises, Flexibility exercises, Endurance exercises, Balance and proprioception exercises, Gait reeducation, Postural management for neurological patients, Positioning and chest care for intensive care patients and Maintenance exercises for recumbent patients) can all be utilized in feline patients.

The most common reasons to perform physiotherapy in cats are generally related to injuries sustained as a result of trauma or joint conditions. Cats often make willing patients but sessions should be kept short and interesting, and should be undertaken in a quiet, relaxed environment. Cats are most often referred for rehabilitation for osteoarthritis, fractures, neurological conditions, femoral head and neck excision (FHNE) and weight reduction. Cats appear to have fewer developmental orthopedic diseases and orthopedic injuries as a whole.

Helpful hints for handling stress, anxiety and pain
If cats are faced with something stressful, their most common method of alleviating the stress they feel is to create distance between themselves and the stressor, i.e. they use flight. If they can’t run, they may attempt to groom or “waste time” hoping the stress goes away. As a last resort, they use aggression.

When the cat arrives at the rehabilitation facility or even if the rehabilitation is held in the patient’s home, the kitty may already be stressed or painful from whatever condition has prompted the need for therapy. Transportation to the vets can be stressful causing them to toilet or even vomit, something that may be particularly unpleasant for these obsessively clean animals. Waiting rooms can be extremely stressful for cats. Use pheromone diffusers like Feliway® or Comfort Zone® in the hospital/rehabilitation facility and exam rooms. Feliway® is clinically proven to help reduce stress related to traveling and visiting the veterinarian. Keeping waiting times to a minimum, having separate areas for dogs and cats, or providing benches, so that the cat carrier can be placed off the ground, thus helping the cat feel less exposed. Examination areas should be quiet and secure, with little or no traffic to cause disruption. Gather any equipment required for the visit prior to getting the cat out of the carrier. Take time to allow the cat to become familiar with the surroundings. Allow it to explore the area and feel more comfortable. Remember “less is more” in terms of restraint for cats. Avoid sudden or rapid movements, as these can be threatening. Encouraging the owner to bring the cat’s own bedding and toys not only makes the owner feel useful, but can also help the patient feel more settled through the retention of the more familiar scent. This time spent with the patient helps to develop a rapport between cat and nurse. Cats can mask signs of pain. The rehabilitation nurse must be skilled at recognizing pain in feline patients. The therapy will have little to no benefit if the patient is painful. Immediately alert the rehabilitation veterinarian if you suspect pain in your patient. Do not proceed with any stressful therapy until the cat is no longer suffering from pain. The owner should be asked prior to the first visit what “treats” their cat enjoys. Having a variety of low-calorie, palatable treats on hand is helpful in bonding with the kitty and goes a long way to establishing trust for future rewards after therapeutic exercises.

Manual Therapy
The therapies that veterinary technicians/nurses can perform include Massage, Range of Motion Exercises, Stretching.

Massage
Massage is defined as the therapeutic manipulation of the soft tissues of the body and has mechanical, physiological, and psychological effects. When massaged, muscle is mechanically stretched, reducing its tone and increasing its pliability. Over time, this can lead to a reduction in muscle soreness and an increase in connective tissue strength. Scar tissue is also mobilized and softened, helping to maintain movement between tissues and restore function after injury or surgery. Physiologically, massage increases interstitial pressure, which in turn increases venous and lymphatic flow. Massaging in a distal to proximal direction is recommended to move fluid from the extremities back to the central circulatory system. As the hands move, squeeze, and stretch the tissues, pressure differences are created between one tissue and another. High pressure pushes old fluid and irritating metabolites into...
the vasculature and areas of low pressure draw in new fluid. This flushing effect may be responsible for decreasing inflammation, pain, and muscle fatigue.12 The body and mind are both linked to the skin via the nervous system. Different types of touch will elicit different types of mental responses. Psychologically, massage decreases stress and anxiety, produces relaxation, and improves emotional wellbeing.1,10,11,12 The types of techniques used are Stroking, Effleurage, Compression (kneading, wringing), Friction, Percussion.

Range of motion exercises
Passive ROM exercises manually exercise joints through their natural pain-free range without voluntary muscle contraction. They are typically performed in patients with stiffness secondary to surgery or in weak patients unable to walk on their own.13

Active ROM exercises put joints through active muscle contraction. Activities include using cavaletti rails (i.e., a system of rails placed at adjustable heights and widths); climbing stairs; swimming; and walking in water, sand, or tall grass.13

Stretching
Stretches are also passive movements that help to improve or restore full range to a joint or full length to a muscle. Stretches create plastic (permanent) deformation and an increased length/range.1 Long-term effects of stretching include adding sarcomeres to muscle mass.1 Stretching is generally more effective if preceded by light exercise, massage, heat or therapeutic ultrasound, all of which increase the extensibility of collagen.

Electrotherapy
Many electrotherapy modalities can be used on feline patients. All possess inherent dangers and should only be used by operators who have received specialist training.

Laser14
The mechanisms by which low-level laser therapy (LLLT) decreases pain includes release of endogenous opioids, changes in conduction latencies of nerves, increase of cellular metabolism, increase in circulation, promotion of neovascularization, decrease in fibrosis formation and reduction of inflammation. Feline conditions that respond well to LLLT include osteoarthritis, degenerative lumbosacral stenosis, fractures, chronic wounds and stomatitis. Most cats tolerate the treatment well as it is not in itself painful and requires a relatively short time to deliver the treatment.

Ultrasound5
For deep tissue heating in veterinary physical therapy, therapeutic ultrasound (ThUS) is the commonly used modality to improve the extensibility of connective tissues, to decrease pain and muscle spasms, and to promote tissue healing and improve the quality of scar tissue. The biological effects of ultrasound differ depending on the used mode: using a continuous mode, the thermal effects are maximized and it is therefore primarily used for tissue heating before stretching. If pulsed ThUS mode is used, the thermal effects are decreased but other effects occur based on the phase of tissue repair, including the acceleration of the inflammatory process, increased fibroblast proliferation, and increasing tensile strength of healing tissues.

Neuromuscular electrical nerve stimulation (NMES)5
Electrical Stimulation (ES) is a useful therapeutic modality and is often possible in cats. In fact, many cats enjoy this modality. Nevertheless, cats must be introduced carefully to ES in order for them to become familiar with ES. Principally, ES can be used for muscle strengthening and pain control. Neuromuscular electrical stimulation is a form of ES whereby current is used to stimulate a motor nerve and cause the contraction of a muscle or muscle group. To stimulate a denervated muscle (e.g., in patients with spinal cord injuries), the muscle fibers must be excited directly and the ES is then called electrical muscle stimulation. For pain control, analgesia occurs because of several mechanisms such as the gate control theory and the release of endogenous endorphins. The most commonly used type of ES for pain control is transcutaneous electrical nerve stimulation.

Therapeutic exercises
Therapeutic exercises are one of the most important parts of the rehabilitation process. The design of the therapy program depends strongly on the needs of the individual patient and should ensure that the exercises can be performed safely without the risk to worsen the symptoms. The exercises should be selected based on the stage of tissue repair, and therefore, the rehabilitation veterinarian should understand the underlying pathology, the expected recovery progress, and biomechanical considerations.15 Exercise represents the final element in the process of helping a cat achieve optimum function following injury, surgery or disease. If assistance is required for the animal to perform an exercise, this can be provided manually or with the aid of ‘physio-rolls’, slings, harnesses or carts.

Therapeutic exercise may be used to improve4
- Aerobic capacity and endurance
- Agility, coordination and balance (static and dynamic)
- Gait and locomotion
- Neuromuscular capability and movement patterning
- Postural stabilization
- Range of motion
- Strength and power
- Pain

Types of exercise
- **Strengthening** - the quality or state of being strong; bodily or muscular power; vigor. Strengthening exercises include such activities as running, slope work (uphill and downhill), use of leg or body weights, dancing, wheelbarrowing and swimming.
- **Flexibility (suppleness)** - the quality of bending easily without breaking. Flexibility is important for cats as it also helps to protect against injury. Flexibility exercises include activities that make the cat reach or stretch for something, or encourage crawling under, through or over obstacles.
- **Balance and proprioception** – Balance is an even distribution of weight enabling someone or something to remain upright and steady. Proprioception is the ability to sense stimuli arising within the body regarding position, motion, and equilibrium. Proprioception diminishes with age, and is also affected by injury or surgery, especially following neurological damage. All cats need good balance and proprioception to function normally. Balance exercises include activities requiring rapid responses to changes in supporting surface (e.g., wobble cushion, balance pad, trampoline) and changes of direction when moving, as well as playing with toys, dancing and standing on a gymn ball. Proprioception exercises include weight shifting, walking in circles or weaving, walking over obstacles of various shapes, height and spacing, and walking over different terrains.
- **Endurance (stamina)** - is the ability of an organism to exert itself and remain active for a long period of time. Endurance exercises are less relevant to cats, which rely more on stealth and rapid movements to catch prey.

Land-based exercises
Land-based exercises should form the major component of exercise programs designed for cats because, being land animals, they must obviously be able to cope with life on land. Examples of land-based exercises are Bicycling, Assisted Standing, Weight Shifting, playing with Laser Lights, Toys, and Treats, Crawling Under Cavaletti Poles, and Wheelbarrowing and Dancing.

Water-based exercise
Hydrotherapy is one of the most useful forms of rehabilitation therapy, and has become a very popular modality for dogs to help in the recovery of musculoskeletal and neurological conditions. Water provides an ideal environment for performing non-concussive active exercise, and through its natural properties (buoyancy and resistance) can help improve limb mobility, strength and joint ROM. There are several forms of hydrotherapy, including pools and water treadmills. The rehabilitation technician/nurse should accompany the cat into the water to provide assistance and reassurance until it is accustomed to the activity. Some cats may be more accepting of water if it is initially introduced to it in the home environment (bath or sink), as a gradual progression from being bathed to being rehabilitated is often more acceptable. The presence of the owner can often provide confidence and reassurance to nervous cats. At no time should any animal be left unattended during a hydrotherapy session, because water aspiration and drowning are real risks. Therefore, a lifesaving vest in a small size for cats should be utilized. Postoperatively, hydrotherapy may be employed as soon as the surgical incision has established a fibrin seal (generally 48–72 h post-surgery), although in practice most hydrotherapy with dogs is started 2–3 weeks following surgery.

Conclusion
Physical rehabilitation for cats is different than that for dogs. The plan must be creative, fun, easy to follow and basically have short intervals for cats. The attention span for cats is much less than that of dogs. Prior to beginning any rehabilitation, the cat must be examined by the rehabilitation veterinarian, checked to make sure pain is not an issue, observed to ensure that stress is not a huge factor for the patient and have the rehabilitation veterinarian draw up the therapeutic plan. The rehabilitation veterinary technician or nurse will most likely be interacting a great deal with the owner, carrying out parts of the therapeutic plan and monitoring comfort for the cat. Feline patients will benefit from a rehabilitation program just like any patient. It’s all a matter of learning to speak “cat”.

References
How Can I Be a Patient Advocate in Pain Management for Exotics and Zoo Animals?

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We will first look at how veterinary nurses became advocates for their patients. Naturally we turn to the published literature where human nursing has the most evidence.

Nursing leaders since the time of Florence Nightingale have envisioned advocacy for both patients and caregivers as an integral part of nursing’s mission. While advocacy on behalf of patients and caregivers has remained a central concern—the American Nurses Association (ANA) publication Nursing: Scope and Standards of Practice identifies advocacy for safe, effective practice environments as a responsibility of the professional nurse. Nurses can fulfill the role of patient advocate by determining the best interests of their patients and using their own voices to promote those interests.

The use of behavioral and physiologic indicators is recommended for pain assessment in nonverbal patients. In humans, this group includes pediatric patients, traumatic brain injury patients, mental disabilities patients, geriatric patients and Alzheimer’s patients.

Veterinary technicians and nurses as advocates

The quality of pain management in practices seems to be directly related to veterinary technicians and nurses. The role of advocate for a nonverbal patient can be daunting. Veterinary technicians and nurses are in the unique position of being responsible for most of and the quality of patient care without the freedom to prescribe or initiate therapy. Knowledge of the physiology of pain and pharmacology of analgesics is essential for good communication between veterinarians and veterinary technicians and nurses. The skilled technician is a source of vital information required to choose and administer appropriate analgesics.

Communication

Technicians use critical thinking, observation, and interpretation skills to make important pain management recommendations. Discussion about each case directly with the clinician might include the technician's concerns about a patient or a general approach to managing different types of pain. Based on his or her interaction with patients, the technician may offer suggestions for adjustments in analgesic regimens, changes or additions to drug protocols, or the possible addition of sedatives, if needed. Technicians and nurses must learn to include important criteria when reporting to the veterinarian. The skills of pain assessment and current physiologic state of the patient must be accurately presented. They need to indicate that they can accurately tell how painful the patient is while explaining why or why not additional analgesics could be beneficial. Veterinary technicians and nurses have the responsibility of continually monitoring their patients and often develop a sense of which analgesics seem to work best under various circumstances. Technicians should provide as much feedback as possible for which analgesic protocols are working well, and which need to be improved to increase patient comfort. Pain management issues, such as the appearance and behavior of the patient that prompted the administration of analgesics; the type, dose, and timing of previous analgesic administration; and the response and any adverse reactions after administration, should be described.

Allowing the technician or nurse additional freedoms

Giving technicians greater choice and control over pain management involves trusting their judgment and experience. After standing orders are established, the success of pain management relies on giving skilled technicians the freedom to give analgesics as needed, to adjust dosages when required, to administer adjunctive medications, and to potentially reverse drugs when severe adverse reactions occur. If trust has been established, then the responsibility and freedom to administer agreed-on analgesics is rewarding for all concerned. Giving technicians a voice in the pain management process creates a truly positive team environment in which their thoughts and skills are valued. Patients ultimately receive better care, and technicians are satisfied knowing that they are doing everything they can to ensure the well-being of patients in their charge.

How stress and pain intersect

Pain and distress can be thought of in terms of a continuum of emotional and experiential states that may occur in an animal. Comfort represents a state of well-being, where the animal is contented and comfortable. Stressors acting upon the animal in increasing severity cause the animal to progressively become uncomfortable (Discomfort), then stressed (Stress), and finally distressed (Distress). Distress represents the extreme point in this continuum, on the far right. Stressors acting upon the animal may move the animal’s experience along this continuum between the extremes of well-being and distress. Depending on the nature and severity of a stressor and on the animal’s current state of being, the animal may adapt successfully to a stress (Adaptive Behaviors) or it may become distressed in a way that threatens its well-being or health (Maladaptive Behaviors). Maladaptive behaviors include abnormal feeding, absence or decreased grooming, and changes in social interaction (aggression, withdrawal). A departure from an animal’s normal behavior is an important indicator that it is undergoing pain and distress.
Signs of pain and distress

There are numerous stereotypical responses to stress or pain stimuli in animals, particularly in mammals. Nevertheless, species differences do exist. Recognition of changes in behavior and physical appearance in the species under study will allow early identification of an animal experiencing pain or distress. As caregivers, humans may know that an event or situation is no threat, but the animal usually does not function with the same information base as humans.

Non-human primates (NHP)

Monkeys often show remarkably little reaction to surgical procedures or to traumatic injury. Obvious signs of pain are not readily seen. Loud and persistent vocalization, for example, commonly signifies only alarm or anger. The animal in pain may be huddled in a crouching posture with a "sad" facial expression and glassy eyes, or it may sit hunched with its head forward and its arms across its body. It may avoid its companions and may stop grooming itself. A monkey in pain may also attract increased attention from its cage mates, which can vary from social grooming to attack. Acute abdominal pain may be shown by facial contortions, clenching of the teeth, restlessness, and shaking accompanied by grunts and moans. Food and water intake is usually diminished or absent.

Key Signs: hunched position, failure to groom, refusal of food or water, dejected appearance.

Mice

After procedures, which cause pain, mice may increase their sleeping times. Reduced food and water intake, with resultant weight loss, dehydration and wasting of the muscles on the back may be observed. Piloerection (erection of hair) and a hunched appearance indicate pain or distress. The animal fails to groom, but scratches more frequently. Sick mice are often isolated from the remainder of the group. Aggressive vocalization is observed in the early stages, decreasing where pain or stress reduces the ability to move and respond. The eyes appear sunken, and ocular and nasal discharge may be noted as the animal's condition worsens. The respiration rate increases and breathing may be forced or labored. As its condition worsens, the animal becomes quiet and unresponsive, separates from the group and eventually becomes unaware of its surroundings. Hypothermia is observed with increasing deterioration in condition; the animal feels 'cold' to the touch.

Key Signs: withdrawal, biting response, piloerection, hunched back, sunken eyes and abdomen, dehydration, weight loss.

Rats

Rats are generally docile and less aggressive than mice towards members of their own species and humans. Acute pain or distress is usually accompanied by constant vocalization and struggling. Rats will often lick or guard a painful area. Increased scratching can indicate chronic pain. A rat in pain will often sit crouched with its head turned into its abdomen. Sleeping periods will be disturbed and increase if pain or distress is present. An elevated respiratory rate associated with sneezing occurs where the respiratory system is affected. Increasing piloerection (staring coat) is noted, along with an increasingly untidy appearance as the animal fails to groom itself. The eyes may appear sunken, and ocular discharge is common, often progressing to red-colored hematomorphyrin exudate which may encircle the eye. Nasal discharge, if present, may be red-colored as well.

Initially, the rat exhibits increased angry or aggressive vocalization, especially on handling. There is a gradual reduction in vocal response as the pain or stress continues, and movement ceases unless a sudden painful stimulus is experienced. Hypothermia indicates significant deterioration in the animal's condition. A pale appearance indicates anemia or blood loss.

Key Signs: vocalization, struggling, licking/guarding, weight loss, piloerection, hunched position, hypothermia.

Guinea pigs

Guinea pigs are alert, but timid and apprehensive animals which will try to avoid capture and restraint. Rarely is there any aggression towards humans. Any sign of acceptance indicates the animal is unwell. Loud vocalization will accompany even minor and transient pain. Guinea pigs often appear sleepy when in pain. The eyes may be sunken and dull. There may be pain associated with locomotion, lameness, and careful gait due to sore feet in older animals.

Key Signs: withdrawal, vocalization, failure to resist restraint, staring coat, unresponsive.

Mongolian gerbils

Gerbils are highly active, nervous animals and usually attempt to avoid restraint. Signs of pain and distress are difficult to assess, as gerbils apparently, object to any interference. There is an increased level of response under painful or stressful stimuli. Ocular discharge is common. Under stressful conditions, the eyelids may be half closed, with dry matting of the eyelids. Dehydration is rarely seen, since the gerbil's normal metabolism enables full utilization of the water content of the diet. Only small quantities of urine are voided under normal conditions. Feces are normally firm, dry pellets. A hunching up and arching of the back may be observed, especially with abdominal involvement. Abnormal gait is associated with locomotion or abdominal involvement.

Key Signs: hunched appearance, weight loss, shock syndrome.
Syrian (golden) hamsters
Under normal conditions, hamsters will sleep for long periods during the day, and little activity will be seen. They often appear aggressive towards their cage mates and emit loud screeching noises, disproportionate to the degree of interference, when handled. This response increases under painful or stressful stimuli. Ocular discharge is commonly associated with stress. Daytime sleep periods may be extended and increasing lassitude may be seen except when the animal is being handled. Exploratory behavior is reduced. A hunched appearance is noted, as is an unwillingness to move, especially where abdominal organs are involved. Lateral recumbency can indicate that the animal is moribund. Normal gait is affected when pain is associated with locomotion. Stilted movements are sometimes associated with abdominal involvement, e.g., ascites following cirrhosis of the liver.

Key Signs: weight loss, hunched appearance, increased aggression or depression, extended sleep periods.

Rabbits
The rabbit presents significant difficulties in recognition of pain and distress, as it often quietly accepts apparently painful or distressing procedures; this may relate to its feral behavior where concealment is important to survival. Even healthy rabbits may not move frequently or indulge in exploratory behavior. Pain is usually characterized by a reduction in food and water intake (and thus weight loss and dehydration) and limited movement. Ocular discharge is a common response to stress in the rabbit, with protrusion of the nictitating membrane.

Under continued pain or stress, rabbits assume a ‘sleepy’ appearance. The animal exhibits increased depression, progressive unawareness and lack of response. The animal will often face the back of cage, away from light. Where foot soreness is involved, weight may be thrown forward or backward to reduce discomfort. Body stretching and lying flat are common indications of abdominal discomfort. Pain may be associated with locomotion, especially with sore feet.

Key Signs: reduced eating and drinking, faces towards back of cage, limited movement, and apparent photosensitivity.

Ferrets
Pain tolerance likely varies greatly between individual ferrets just like in other species. There is a proof that solitary-living animals and prey animals are masters of disguising pain. Ferrets in pain may stay curled in a tight ball or have a hunched back. They may have an altered gait. They may have decreased or increased food and water intake. They may exhibit bruxism. They may hide in the back of a cage, vocalize, be aggressive, not groom and look unkempt. The most likely causes of pain in ferrets are arthritis, cancer, or dental problems.

Key Signs: Stiff posture, demented behavior, lack of grooming, hunched head and neck, and inappetence.

Birds
Many clinical signs may be associated with pain in birds including change in temperament to either aggressive or passive, restlessness, reluctance to stand, perch or even move, anorexia, lethargy, a hunched appearance, tachypnea, and lameness. Birds may avoid a painful area and reduce grooming, or they may over-groom, feather pick, or chew at the area. Behaviors such as vocalizing and writhing may only manifest during acute or severe pain or may not manifest at all in birds.

Few consistent physiologic indicators of pain have been identified in birds. In feather-plucked chickens, heart rate and respiratory rate varied between animals but hypertension was a reliable indicator. Fecal corticosterone concentrations have shown potential as a non-invasive method of determining stress and pain.

Key Signs: Escape reactions, atonic immobility, inappetence, and avoidance of use of pain site.

Reptiles
Acute pain in reptiles may be characterized by flinching and muscle contractions. There may be aversive movements away from the unpleasant stimulus, and attempts to bite. More chronic and persistent pain may be associated with anorexia, lethargy and weight loss, although it is difficult to associate any of these signs of lack of well-being specifically with pain.

Key Signs: flinching and muscle contractions, weight loss, anorexia.

Fishes and amphibians
It is difficult to determine the nature of the response to pain in fish and amphibians. Although they exhibit a pronounced response to injuries or to contact with irritants, their response to chronic stimuli may be small or absent. Fish and amphibians with severe wounds which would cause immobility in a mammal, will often appear to behave completely normal, even resuming feeding. Fish and amphibians will react to noxious stimuli, such as that administered by a hypodermic needle, by strong muscular movements. Fish when exposed to a noxious environment, such as a strong acid, they show abnormal swimming behavior with attempts to jump from the water, their coloring becomes darker and their opercular movements become more rapid. Such effects are indicative of some degree of distress; however, it is not possible to describe these unequivocally as signs of pain. However, we must assume that which causes pain in mammals, birds and reptiles will also cause pain in fish and amphibians.
Key Signs: Amphibians: Muscular movements, closed eyes, color changes, rapid respirations, immobility, and anorexia.

Fish: Abnormal swimming behavior, attempting to jump out of water, rapid opercular movements, clamped fins, pale or darkened color, and hiding. Anorexia is the first sign.

Invertebrates

Nociceptive cells have been found in invertebrates and opioid systems are functional in Invertebrate nociception. Opioids and local anesthetics provide good analgesia.

Key Signs: Invertebrates: Rapid withdrawal.

Zoo animals

Wild animals look different, have different social systems, and react to pain and pain medications differently than their domestic species counterparts. Prey and predator species mask issues from their conspecifics (belonging to the same species) and caretakers. Predators also mask their pain in front of conspecifics due to the competition for territories and prey. Despite the differences in patients, the role of a veterinary technician as a patient advocate in zoo and wildlife medicine is like that of a domestic animal technician/nurse. The technician/nurse is there to help ensure that every animal is well cared for and has an excellent quality of life. Husbandry staff is trained to read subtle changes in the behavior, gait, or appearance of an animal. They will sound the alarm as soon as an animal begins to act uncharacteristically. Utilizing their knowledge to understand the basics of that animal or species. The entire team needs to be part of any treatment plan, as the responsibility of the animal’s daily care does not rest on one person. Overall appearance of an animal can also be a good indicator that something is awry with an individual animal. An animal that appears unkempt is most likely unthrifty due to illness, but this effect is also seen in animals with chronic pain. Animals will keep their heads lower; their ears may droop or fall back towards their heads. Stance can change by arching the back or tucking in limbs or the abdomen. Some animals that are sick or injured will allow a threat, that is, veterinary staff, to get closer than if they were healthy. An observer could then use changes in flight distance as an indicator of the severity of the injury or illness. While veterinary technicians or nurses do not prescribe medications, they typically dispense and administer them. Currently used treatments and doses have been extrapolated from similar species, which works in some cases, but not in the others. Forums and networking between institutions and zoo or wildlife professionals are valuable tools when trying to determine what analgesic protocol to use. Veterinary technicians/nurses have a discussion forum at the Association of Zoo Veterinary Technicians (AZVT) website http://www.azvt.org/.

Key Signs: Behavioral changes, appetite changes, animal isolation from their group or pack, aggression, lameness, unkempt appearance, or lowered head.

References

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No longer is it acceptable to say that farm animals do not need analgesic care. An understanding of pain, physiology, signs of pain unique to livestock and conditions that may require analgesic care are necessary for any veterinary nurse working with livestock species. How can owners/farmers be educated about the benefits of pain management in a herd situation?

Introduction
Livestock patients for this lecture will include cattle, sheep, goats and pigs. Animal welfare has increased the importance of pain management in livestock. Multimodal analgesia is the preferred method of pain relief for even minor surgical procedures. Animal suffering is no longer tolerated nor is the unwillingness by owners to pay for medications.1 Dr. Temple Grandin’s work in the livestock industry on animal behavior has shown that animals (livestock) are not just things to be owned, but instead they can feel pain and suffer.1 Veterinary technicians and nurses have the ethical duty to advocate for these patients to the same standard as any others.

Recognition of pain in cattle
Cattle in pain often appear dull and depressed, with the head held low and showing little interest in their surroundings. There are inappetence, weight loss, and, in milking cows, a sudden drop in milk yield. On being handled, cows may react violently or adopt a rigid posture designed to immobilize the painful region. Grunting and grinding of the teeth may be heard. Acute pain may be associated with bellowing. Generally, signs of abdominal pain are like those seen in the horse but are less marked. Rigid posture may lead to a lack of grooming due to unwillingness to turn the neck. In acute abdominal conditions, such as intestinal strangulation, the animal adopts a characteristic stance with one hindfoot placed directly in front of the other. At a certain level of severity, pain in ruminants can be identified easily because an animal is unable to hide the experience of pain or the physical insult causing the pain may interfere with normal function (e.g., lameness).2

Recognition of pain in sheep and goats
Sheep often only show subtle signs of pain, while goats are intolerant of painful procedures.1 Goats will often bleat, while sheep may only exhibit tachypnea, inappetence, grinding of teeth, immobility, or abnormal gait. Following procedures such as castration and tail docking, lambs may show signs of discomfort such as standing up and lying down repeatedly, tail wagging, occasional bleating, neck extension, dorsal lip curling (Flehman), kicking, rolling and hyperventilation.

Sheep are a timid prey species and unlikely to advertise that they are compromised; thus, one would expect overt signs of pain only after seriously painful procedures or experiences. Moreover, unlike cattle and goats, sheep tend to remain silent rather than vocalizing during painful procedures.2 Goats are more vocal animals than sheep or cattle. Kids commonly hide and wait for their mother to return and feed them, unlike lambs, which follow their mother all the time. This may explain the difference in the behavior of kids and lambs after castration.

Recognition of pain in pigs
Pigs in pain may show changes in gait and posture. They normally squeal and attempt to escape when handled; however, these reactions may be accentuated when the animal is in pain. Adult pigs may become aggressive. Squealing is also characteristic when painful areas are palpated. Handling of chronic lesions may not elicit signs of pain. Pigs will often be unwilling to move and may hide in bedding if possible.1

Pigs are stoic animals, but are also a prey species inclined to mask signs of illness, weakness, pain, or vulnerability until they can no longer compensate. Decreased food intake, as well as less time spent eating, has been well documented in painful or ill swine of all ages. Not all ill or painful pigs stop eating, but all pigs that stop eating are likely to be suffering.2 Decreased activity is an important sign of pain or discomfort in an animal like the pig that normally spends a large part of its day foraging and exploring. Can be an important sign of pain or distress. Pigs with forelimb arthritis frequently stand in a hunched position with more weight on the pelvic limbs. Lying and rising may be more painful than simply standing in a position in which pressure on the sore limb or limbs can be relieved. Pigs in pain may also be stiff, tremble, or isolate themselves from other pigs and people. Pigs with visceral pain may kick at the abdomen or may shift repeatedly when lying down, suggesting that they cannot get comfortable. Potential causes of chronic pain, such as neoplasmia or severe infectious or degenerative processes, are often discovered late during the disease and typically lead to euthanasia. Arthritis is a primary example of a chronic pain condition in the pet pig.
Pain scales for livestock

Cattle
In surveys of veterinarians concerning the use of analgesics in cattle practice, lack of knowledge in recognizing pain\(^3\), the belief that farm animals feel less pain than smaller animals\(^4\), economic reasons\(^5,6\) and the lack of valid and reliable instruments to assess pain have been cited as the main reasons why analgesics are not used more frequently\(^7\). In cattle, a scoring system for gait has been validated and found to be reliable and sensitive for identifying cows with severe hoof lesions.\(^7\) The UNESP-Botucatu unidimensional pain scale for assessing acute postoperative pain in cattle is a valid, reliable and responsive instrument with excellent internal consistency and discriminatory ability. The cut-off point for rescue analgesia provides an additional tool for guiding analgesic therapy.\(^8\) The Cow Pain Scale included, ‘attention towards the surroundings’, ‘head position’, ‘ears position’, ‘facial expressions’, ‘response to approach’ and ‘back position’. Further-more, treatment with a systemic analgesic significantly reduced the pain score of the group, where clinical examination suggested pain but did not affect the cows in the control group. Taken together, these results suggest that the Cow Pain Scale (CPS) may be used to identify cows in pain.\(^9\)

Sheep
Sheep have been studied to assess their pain. A study revealed that sheep that underwent maxillofacial surgery displayed postoperative hypersensitivity lasting at least three days after surgery, despite intra and postoperative treatment with opioids, an NSAID and local anesthesia.\(^10\) Another scale looked at locomotion in sheep.\(^11\) The results indicate that the locomotion scoring scale using groups of defined observations for each point on the scale was reliable and may be a useful research tool to identify and monitor locomotion in individual sheep when used by trained observers. The use of facial expression scoring to assess pain is a well-utilized, practical tool in both humans and non-human animals. Facial expression as a pain scoring method offers the potential to start to under-stand this side of animal pain. The Sheep Pain Facial Expression Scale (SPFES) provides an accurate and reliable method to recognize and assess pain in sheep. It also doubles as a training tool for veterinarians and farmers to learn more about changes in the facial expression of sheep when they are likely to be suffering from pain. Such a tool is likely to improve an observer’s ability to quantify pain in animals and allow observers to discriminate between different pain states independent of disease status, as well as detect the effectiveness of pain relief. Prompt recognition of pain using the scale will enable farmers and veterinarians to treat and manage their flocks better, reducing the impact of pain on their sheep, thus improving welfare and production.\(^12\)

Goats
Stockpeople's ability to recognize pain in their livestock, and to respond appropriately, is of utmost importance for animal welfare. Assessment of pain is complex, and attitudes and empathy are thought to play a role in peoples' responses to the sight of pain. The Norwegian dairy goat has been utilized to assess the ability of stockpeople to evaluate pain.\(^13\) A Numeric Rating Scale (NRS) for assessment of post-operative pain for Goats has been developed.\(^14\)

Pigs
There are a couple of studies that have undertaken to assess a Grimace Scale for Pigs.\(^15\) The Piglet Grimace Scale requires considerable further development as a potential tool to detect post-procedural pain in neonatal pigs.\(^16\) Full characterization of the Piglet Grimace Scale would give the opportunity to implement it as a cost-effective tool for the on-farm assessment of painful and or distressing conditions induced by husbandry in piglets. Finally, it would provide the basis for the development of a scale specific to non-neonatal pigs, which would benefit the assessment of pain in relation to spontaneous health conditions (e.g., lameness induced by degenerative joint diseases or infection) that are normally observed in older animals.

A dissertation was done in Uppsala, Sweden.\(^17\) The findings in this study suggest that also in pigs, a pain face can be identified. This porcine pain face seems to comprise an angled appearance of the area above the eyes, wrinkling of the snout, and tension of muscles around the mouth and along the lateral side of the head, as well as lowered ears held in an asymmetrical manner or completely backwards. These features were sometimes rather subtle however, and the method may thus be difficult to apply in a clinical situation. Further studies are required to evaluate and develop the porcine pain face, which may then possibly constitute a valuable additional pain assessment tool in pigs.

Procedures that cause pain in cattle
- GI or colic surgery - Severe
- Abomasopexy - Moderate
- Claw removal - Moderate to severe
- Dehorning - Moderate to severe
- Teat surgery - Moderate to severe
- Castration - Moderate
- C-section - Moderate to severe

Procedures that cause pain in sheep and goats
- C-section - Moderate to severe
- Perineal Urethrostomy - Moderate to severe
• Castration - Moderate
• Dehorning - Moderate to severe
• Claw removal – Severe

Typical procedures that require analgesia in pigs
• Castration: baby pigs, pet pigs, and adult or larger pigs
• Aural hematoma
• Caesarean section
• Amputation: digital
• Amputation: uterus
• Atresia ani
• Entropion: frequent in pot-bellied pigs
• Exploratory laparotomy
• Fractures
• Hernia: inguinal and umbilical
• Hoof trimming
• Joint lavage
• Mastectomy
• Ovariohysterectomy
• Prolapse: rectal, vaginal, uterine
• Tail docking
• Tooth clipping
• Tusk removal
• Cryptorchid
• Vasectomy

Medications used in livestock
When considering pain management strategy, one should contemplate prevention (preemptive) and control of those pain pathways already activated in nociception.¹

Most commonly used medications in Cattle are NSAIDS, opioids, local techniques such as epidural anesthesia, intravenous regional anesthesia (IVRA), and the use of local nerve blocks provide analgesia to specific areas, Alpha-2-Adrenergic agonists, Ketamine, and Analgesic Adjuvants. Constant Rate Infusions (CRIs) have been described.¹

Most commonly used medications in Sheep and Goats are NSAIDS, Opioids, Alpha-2-Adrenergic Agonists, NMDA Receptor antagonists (Ketamine), Local Anesthetic blocks and infusions and Analgesic Adjuvants. CRIs have been utilized in small ruminants.¹

Most commonly used medications in Pigs are Opioids, NSAIDS, Alpha-2-Adrenergic Agonists, NMDA Receptor antagonists (Ketamine), Local Anesthetic blocks and infusions and Analgesic Adjuvants. CRIs have been commonly utilized in pigs.¹

Withdrawal period
The withdrawal period is defined as the interval required after dosing for tissue concentrations of a drug or its metabolite to deplete to less than a specific concentration that has been established to be safe for human consumption. The withdrawal period MUST be emphasized in any animal that will enter the food chain.

Conclusion
Pain Management for Livestock patients is doable. The veterinary nurse must learn about recognition and assessment of pain in these patients. They need to learn how to score pain in cattle, sheep, goats and pigs. They can teach veterinary staff and owners about this pain. They can even help the owners learn how to score pain in their livestock. If the owner does not have a pain scale that they like, then the veterinary nurse can develop a pain scale that the owner/farmer can use. It is best to keep it simple and straight forward.

If you need pointers or papers about pain in livestock, you can email me whitester@gmail.com.

References
Inappropriate elimination has been reported as one of the most common reasons owners relinquish their pet. Veterinary practices should take a proactive approach to assist pet owners with the prevention and treatment of inappropriate elimination problems. It is imperative to inquire about elimination training success at each preventive care visit (from puppy/kitten to senior visits). Owners may not be forthcoming regarding an inappropriate elimination issue because they might be embarrassed or not know there is anything that can be done to help. Routine inquiry regarding the status of elimination training, results in early identification of an elimination issue. Not only can this help prevent damage to the human-animal relationship, but it can also prevent a lost client due to pet relinquishment.

Medical concerns and history taking
Inappropriate elimination can be caused by medical factors, behavioral factors, a lack of training, or a combination of factors. Medical and behavioral factors need to be ruled out and addressed by a veterinarian.

Whenever faced with a house-soiling issue, it is important first and foremost to rule out any possible medical factors which could contribute to or cause the inappropriate elimination. This is especially true when a previously house trained dog or litter box trained cat starts to eliminate inappropriately. However, young puppies and kittens can also have medical factors such as congenital disorders, vaginitis, infection of the prepuce, and urinary tract infections that can limit the success of elimination training.

Consequently, it is important to get a thorough history once elimination issues have been identified. Examples of some questions you might ask: How often does your pet eliminate in a day? Where do the majority of accidents occur? When do they occur (is the owner home and accessible to the pet)? How long has the pet been eliminating in the undesirable locations? Where are the desired elimination areas located (litterbox or outdoor elimination areas)? How often does your pet have access to those areas? What is your feeding and water access routine? Any changes in the household during the time that the behavior started? Are there other pets in the house? If so what is the patient’s relationship with them? These questions, although not all inclusive, are designed to help identify either possible medical and/or environmental factors associated with the inappropriate elimination.

Examples of behavior factors that may cause or contribute to inappropriate elimination include: generalized anxiety, sound sensitivities, separation anxiety disorder, aggression between pets in the household, and fear. Urine marking or spraying can be an indicator of stress or anxiety in the pet. The focus of this presentation will be on preventing and treating inappropriate elimination due to a lack of training.

Canine inappropriate elimination due to a lack of training
“Inappropriate elimination” (house-soiling) is only inappropriate in terms of location from the owner’s perspective. Dogs prefer to eliminate on a porous surface, away from their eating and sleeping areas, and where elimination has previously occurred. Although humans find it undesirable for the dog to urinate or defecate in the house, there are often many locations within the house that meet the above criteria.

The antecedent is usually a full bladder or colon and access to a “canine-approved” elimination location. To the dog, it is a self-reinforcing behavior; a full colon or bladder is relieved by eliminating. The motivation for elimination is normal voiding behavior. Medical and other behavioral factors should be ruled out.

Prevent the dog from developing a preference for indoor areas through supervision and confinement. Anticipate when the dog will need to eliminate, and supervise or confine the dog to prevent accidents. Meal feed a highly digestible diet at specific times. Allow free choice water consumption. Limiting access to water can result in a dog that ingests a large amount of water at one time. Provide opportunities for the dog to eliminate in appropriate areas. Reinforce the dog within a half second of the end of the void.

A dog does not develop full bladder control until at least 16 weeks of age. Accidents can often occur periodically during the first year of a dog’s life. They should become less and less frequent as the dog begins to learn the human-preferred location for elimination.

Develop a routine for taking the dog outside or to the preferred elimination location. Use a phrase such as, “Let’s go outside!” Exit out the same door. Teaching the dog to eliminate on leash can be advantageous because it allows for elimination opportunities in public places when traveling, it allows you to reinforce the dog immediately after elimination, and it also allows you to teach a specific area in the yard to eliminate. Owners might also consider teaching a “Hurry up” cue as well.

What should you do if the dog has an accident? If you catch the dog in the process of eliminating, you might use response substitution. In an upbeat tone, interrupt the behavior by clapping your hands or calling the dog’s name (remember upbeat tone). The goal is to get the dog’s attention but not to frighten or punish the dog. Use your special phrase such as, “Let’s go outside.” Take the dog to the desired location and reinforce elimination in that area. The other option is to let the dog complete the void. Then re-evaluate your prevention and management strategies to assess why the accident occurred.
Avoid verbal or physical reprimands for inappropriate elimination. This only teaches the dog to not eliminate in front of people and that makes it difficult to reinforce the dog in the appropriate location. If you find an accident after the fact, simply clean it up and assess your management strategy. Soiled areas should be cleaned with a good enzymatic cleaner. After allowing the enzymatic cleaner to dry for 24 hours, then make the area locally aversive. The area may be made less attractive as a future elimination area by applying an odor to the area that the dog finds undesirable.

**Feline inappropriate elimination**

After medical factors are ruled out, feline inappropriate elimination often falls into 1 of 2 general categories: house soiling or urine marking. Although it can also be a combination of house soiling and urine marking. House soiling will be the focus in this session. House soiling refers to a normal void that is being performed in an inappropriate location. Litterbox use is generally self-taught. However, preferences for substrate will depend on exposure to the substrate during the first few weeks and months of life.

Generally house soiling occurs due to either an aversion or dislike for currently available options or preference for a different substrate or surface.

Just as with dogs, elimination in cats is often performed in response to a full bladder or colon. The act of elimination is self-reinforcing as it provides relief. In this section the motivation of elimination is focused on a normal void.

Prevent and manage accidents from happening through the use of confinement and supervision. Although litterbox use is usually self-taught, confinement early on can help the cat be successful. Cats prefer larger (1.5 times the length of their body is suggested), uncovered boxes. A fine, unscented, clumpable litter is preferred. The boxes should be located in convenient locations with at least one litter box on each level of the home. The ideal number of boxes is the number of cats plus 1. If there are 3 cats, there should be 4 boxes. Being fastidious creatures, cats will be more willing to use a litter box that is kept clean. Scooping at least once daily is recommended.

The cat could be reinforced with a food treat after leaving the litterbox. It is best to provide the cat with privacy and not disturb the cat (even with a treat) while in the box. Trying to reinforce the cat while in the box could inadvertently create an aversion for the box.

Soiled areas should be cleaned with an enzymatic cleaner. Then, the area can be made locally aversive with an odor undesirable to cats. Avoid punishing the cat for the inappropriate elimination. Instead redirect to the litter box.

If after implementing the management and prevention suggestions above, the cat is still preferring a specific substrate (ie carpet), it might be necessary to try to re-train the cat to litter. The majority of house soiling situations can be resolved by providing preferable and adequate access to litter boxes.

Changing the cat’s preference can be more challenging. To implement this, access to the current substrate must be avoided. The cat might need to be confined initially or all carpets must be picked up temporarily. Appropriate litter boxes should be made available. If the cat will only eliminate on carpet, then provide a carpet remnant in the box with some litter by it. Gradually decrease the size of the carpet and increase the amount of the litter until the cat is reliably eliminating in the litter box.

**Conclusion**

Inappropriate elimination in dogs and cats is a common reason pets lose their homes. By inquiring about the status of elimination training at each and every preventive care visit, preventive and early intervention methods can be implemented. By providing clients with appropriate information regarding training the pet to a desired location, you will not only save lives but also enhance the human-animal bond.

**References**


Prevention of problem behaviors is easier than treatment. Problem behaviors in cats are often associated with the stress of other cats in the house or outside the home. In order to decrease social tension in multi cat households, provide core areas for each cat, increase vertical living space, and prevent exposure to outside cats. When introducing a new cat to a resident cat, it should be a gradual process.

There are pros and cons to confining the cat indoors versus allowing access to outside. The indoor environment is not as enriching or stimulating. However, the indoor environment is safer. Through management and environmental enrichment, indoor cats can be given alternative activities to allow for mental and physical stimulation.

**Environmental enrichment**

Implementing routine play sessions with the owner in the morning and evening provides for routine interactions. Cats enjoy playing with toys that encourage pouncing and stalking (predatory sequence). Feather toys on the end of a pole, small stuffed toys and balls encourage exercise and positive interactions with the owners. Any type of direct play with human hands should be avoided as it can promote inappropriate play and result in potential injury to humans through accidental scratches and bites.

Cats are natural grazers and they are designed to eat small amounts of food at frequent intervals. Food puzzle toys can help to prevent obesity in cats as there is more activity involved in the feeding process and they provide great mental stimulation. Food puzzle toys also allow for exploration and discovery.

Hiding food and treats around the house in small dishes or cups promotes exploratory behavior and appeases the cat’s natural desire to search for food. Although not a toy, some cats enjoy the option of having indoor grass available. Most are easily grown indoors and are made up of either wheat or oat grasses. Offering fresh grass can prevent cats from eating house plants while providing roughage to their diet.

Providing exploratory outlets through a variety of interactive toys will help to appease the cat’s curiosity. Interactive homemade toys can provide cats with hours of entertainment. A cardboard box with holes for the cat’s paws and toys dangling from sisal rope is an easy to make homemade toy that most cats will appreciate. Sisal rope is a durable rope made from plant fiber. It is often used for scratching posts for cats. These toys can be inexpensively made to entertain boarding felines and sent home with the owners at pick up.

Vertical space is important not only for management but also environmental enrichment. Vertical space creates areas for exploration and additional hiding places. Cat trees, perches, shelves and elevated hiding places are great options for creating vertical space for cats. Tunnels, boxes and paper bags can be used at lower levels. In the veterinary setting, a cardboard box or paper bag placed in a cage with a feline patient can allow the cat a safe place to hide in the novel environment.

Secure outdoor enclosures and fences are available to allow cats to have exposure to the outdoor environment within a confined area. Most are designed to not only keep the pet cat in the yard but also prevent other feline intruders from entering the yard. For safety supervision by the owner is recommended during controlled outdoor exposure. Acclimatizing a kitten to wearing a cat harness can also allow the owner to manage the cat outside; thus allowing for more exploration. Adult cats that have not had previous exposure to outside, may never be comfortable or enjoy being outside regardless of the owners’ best effort. Instead the owner can focus on making indoors as enriching as possible.

**The first veterinary visit**

One of the first items that should be addressed during the first veterinary visit is teaching the kitten to become comfortable with the type of handling needed to keep it healthy and well-groomed throughout its life. This will also make procedures run smoothly and save staff time by creating a relaxed and cooperative patient in the clinic. The first kitten appointment is the ideal time to prepare kittens for physical exams, venipuncture, teeth cleaning, ear cleaning and pedicures. Not only in kitten hood but even in adult cats, it is imperative that technicians avoid mishandling patients regardless of their behavior. The goal always should be to create a positive association with the examination process that can follow the pet through the rest of his life. Rough or forceful handling methods teach fear and mistrust and often result in a difficult to handle cat. It also sets a poor example for owners to follow and could be considered malpractice.

By taking a few extra minutes during the first appointments, the kitten will be able to acclimate to the environment. Utilizing treats or canned kitten food during the examination and vaccination process, the kitten will likely be so distracted she does not even notice. This is also creating a positive memory for the kitten.
Integrating a new cat to a multi-cat household can be stressful for the new cat as well as the resident cats. In order to provide for the most harmonious integration possible, it is best to take a proactive approach and systematically provide for a gradual introduction. Although the process may seem tedious, it often can progress quickly. However, if owners decide to “just see what happens,” a negative initial introduction could result in a much longer acclimation process or even worse, an inability for the cats to cohabitate.

The kitten or new cat should be set up in one room. The room should have all necessary resources, including a litter box, scratching station, food, water, bedding and toys. It is also a good idea to include a large multilevel cat cage. The kitten should be provided with numerous opportunities to interact and play with the owner throughout the day. For the first few days the new cat should be kept confined in a room. This will give the resident cats the opportunity to become accustomed to the new cat’s scent through a closed door. The procedure can be helped along by exchanging bedding between the animals. The scents of the cats can also be mixed by allowing the new cat to explore other parts of the house while confining the resident cats to a room. Another method to mix the scents of the cats is called artificial allomarking; a small towel can be rubbed on one cat, then the other, then again the first one, then the second one, etc. This helps to “mix” the odors and makes a communal scent between the cats.

Ideally the resident cats and the new cat should become acclimated to their own individual multilevel cat cage. This will aid in the visual introductions of the cats. All cats should be managed in their cage for a special meal time twice a day. Alternatives to the multilevel cat cage are either a travel carrier or a harness and leash. However, all the cats must be comfortable with the confinement method or harness prior to starting the introduction process.

Start at a distance that the cats can see each other but are not dissuaded from eating their special meal (the furthest distance possible for the layout of the house is best). Each day move the cages a foot closer, until they can be next to each other while eating. Once this has been accomplished, if the resident cats are not overly interested in the new cat, the owner may consider keeping the new cat in the cat cage for supervised periods of time (if using a travel carrier, place the carrier up on a table or elevated surface) while allowing the resident cats to be loose in the room. This will help to facilitate habituation to the presence of each other. The next step would be to allow the new cat to be loose in the room and the resident cats to be confined to their cat cage. Once it has been determined that amicable interactions are occurring between the cats and they are relaxed in each other’s presence, supervised periods of time loose together can be allowed.
If any direct staring, hissing, growling, or other threats are observed, a towel should be placed over the cage to interrupt the threat and the cats should be separated. Aggressive responses should be dealt with by ending the session and waiting 24-48 hours before attempting again. With the next session increase the distance between the cats and progress more gradually. Ideally, avoid all negative experiences while introducing the cats. Slower is actually faster because negative experiences will be remembered and will take time to overcome. Pheromone products such as Feliway® may also be useful if the resident cats or the new cat are stressed during the process. The entire process may take 2-6 weeks to accomplish new introductions depending on the individual cats. A quick progression of the same techniques can be used when re-introducing cats after one of the household cats has had a visit to the veterinary clinic. The cat that remained home will often reject the returning cat. Simply mixing their scents and doing a controlled special meal time and gradual introduction over a few hours can be effective in preventing a long lasting negative re-introduction.

**Resources**
Prevention is Easier than Treatment!
The Importance of Preventive Behavioral Service
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Undesirable behavior in pets results in a weakened human-animal bond. Behavior concerns are the number one cause for pet relinquishment. Forty percent of pets were relinquished to shelters for behavior issues.1 The number one behavior reason in one study for relinquishment was house soiling in both dogs and cats.2 Through preventive behavior services, we can educate clients on proper techniques for addressing normal behavior challenges in their pets. Thus, keeping pets in their homes, saving lives, and retaining a patient. The prevention of behavior issues is easier than the treatment of them.

An overview of several preventive services that can be offered within the hospital and implemented by or with the assistance of the veterinary technician or other trained professional will be discussed. These services include: pet selection counseling, puppy socialization classes, kitten classes, fun visits, private training or behavior modification sessions, (behavioral) wellness visits.

Pet selection counseling
Pet selection counseling is the first defense against preventing behavior problems and the first offense in influencing a strong human animal bond. Educating and preparing the prospective pet owner are the primary goals of this service. Misconceptions can be discovered and addressed. Not only will the new pet owner be better prepared but they can also set their new companion up for success.

Puppy socialization classes
In a true puppy socialization class, puppies should be in their socialization period. A good puppy socialization class will provide a safe environment for exploration and exposure to a variety of stimuli in a controlled and positive manner. The main focus at this age is not on manners training but on creating positive experiences for the puppy and teaching puppy owners appropriate and humane techniques for addressing normal puppy behavior. The benefits of puppy socialization classes include:

- Preventing behavior problems
- Decreasing pet relinquishment
- Bonding the client to the puppy and your facility
- Educating puppy parents on normal canine development and humane training techniques
- Acclimating puppies to handling and routine veterinary procedures
- Providing a controlled and safe environment for exploration
- Allowing for early intervention for high risk puppies
- Facilitating all puppies reaching their full potential

A good resource regarding the importance of puppy classes is available at www.avsabonline.org under position statements.

Kitten classes
Kittens attending a class should be under 14 weeks of age. The benefits of offering Kitten Classes in your hospital include:

- Creating a strong bond between the owner and the kitten as well as your hospital

- Educating owners regarding normal feline development and behavior
- Coaching owners on responsible cat ownership and management
- Providing a safe and controlled environment for exposure and desensitization to veterinary procedures
- Identifying and preventing behavior problems

Fun and victory visits
A fun visit refers to the pet visiting your hospital just for fun. No procedures are performed. Kitten and puppy classes in your hospital are fun visits. However, once the dog/cat has graduated from the class, it is equally as important that they continue to return to your hospital for good experiences. This helps build a positive emotional response and memories with your facility. This is generally considered a complimentary service, when it is a preventive. Victory visits involve a veterinary team member assisting with desensitization to gentle control and medical treatments or with changing an already established fear of the veterinary office. Victory visits are a service that should be charged and is considered a private training/behavior modification session.

Private training and behavior modification sessions
Private training and behavior modification sessions are scheduled appointments with a qualified team member to address manners and preventive training. Generally, behavior modification implies the treatment of an already existing fear or anxiety. Pending the situation, this may require a veterinary behavioral diagnosis and treatment plan prior to addressing. However, if preventively
addressing a mild apprehension, such as avoidance of the nail trimmers, a behavior modification session to coach owners on how to appropriately condition a dog or cat to nail trimming would be considered preventive.

**Behavioral preventive care visits**

You are already doing preventive care visits! Incorporating behavior questions into the history taking is imperative. Clients are not always forthcoming with behavioral concerns. They may be embarrassed that their cat is peeing on the carpet or that their 1-year-old dog is chewing the couch when home only. By screening for common behavior concerns, we can identify situations early before irreparable damage to the human animal bond has been done. Consider adding a specific preventive care visit for dogs and cats that are between 9-12 months of age, to address behavior concerns that have developed since the routine puppy and kitten examination series. The majority of pets are relinquished to shelters between 5 months and 3 years of age (dogs 47.4% and cats 40.3%) and have been owned between 7 months and 1 year (dogs 37.1% and cats 30.2%). This is a time we often do not see them in the veterinary hospital. By reaching out and suggesting a behavioral checkup during this time, early intervention can be provided.

**References**


**Resources**

American Veterinary Society of Animal Behavior (AVSAB) [www.avsabonline.com](http://www.avsabonline.com)

Society of Veterinary Behavior Technicians (SVBT) [www.svbt.org](http://www.svbt.org)

Puppy Start Right Instructors Course www.puppystartright.com

[Fear Free Foundations for Kittens and Puppies](http://www.vetfolio.com/fear-free/kittens-and-puppies)

Canine and Feline Behavior for Veterinary Technicians and Nurses. Co-editors Julie Shaw and Debbie Martin
Importance of early socialization

The socialization period can be defined as a sensitive period of development whereby a dog learns to communicate and relate to conspecifics, humans, and the environment. It is likely the most influential learning period of a dog’s life. This period of development lasts from 3 weeks to 12 weeks of age. Some fluctuation exists in the age range and variation between breeds and individuals will be seen. Between 12 and 16 weeks of age there is a swift decline in the dog’s acceptance to novelty, especially if there has not been appropriate exposure prior to this time.

Early positive proactive socialization is so important that every veterinary hospital should be providing their clients with access to an appropriate puppy socialization class. Poor socialization or deprivation of environmental exposure often leads to lifelong deficits and dysfunctional behaviors. Lack of exposure can be just as detrimental as a bad experience. The socialization period is a finite period of development in which the dog is genetically programmed to be more accepting of novelty. Consider puppy socialization classes as vaccinating against behavior problems.

The benefits of offering puppy socialization classes in the veterinary hospital include: bonding the client to their new puppy and the veterinary hospital, educating the client on normal canine behavior, addressing common puppy training issues, and providing a controlled and safe environment for puppy play. Puppy socialization classes may also help prevent behavior problems such as: inter-dog aggression, separation anxiety, and fear disorders related to a lack of socialization. Teaching puppies to enjoy restraint and handling will make the veterinary staff’s job easier. Puppy classes can also help identify problem puppies or high risk puppies for the development of future behavior problems. Thus, allowing for early intervention and maintenance of the human-animal bond.

Class format and logistics

Offering classes with open enrollment is the most effective way to reach most puppies. Because the class is limited to puppies between 7-12 weeks of age when starting, offering class orientation every one or two weeks is the most successful method for maximizing enrollment. The client attends an hour orientation prior to attending class with their puppy. Once the client has attended orientation, four 1 hour puppy socialization classes are offered. Classes meet on a weekly basis. Puppy classes should be staffed appropriately with 3-4 puppies per staff member ideally.

Orientation

Orientation prepares the owner for what to expect and what to bring to puppy class. The owner should complete a registration form and submit vaccine records. Puppy owners attend the orientation without the puppy. This allows for the client to learn about canine development, body language, problem solving, and develop some training skills without the distraction of their puppy.

Class outline

Classes should be well organized and the instructor should keep the class on track. The use of any physical or verbal reprimands would NOT be recommended in a good puppy socialization class. Typically, class format for each socialization class would include:

<table>
<thead>
<tr>
<th>Class Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Off leash controlled play</td>
<td>5 minutes</td>
</tr>
<tr>
<td>Introduction to training</td>
<td>2 minutes</td>
</tr>
<tr>
<td>Problem prevention topic 1</td>
<td>5 minutes</td>
</tr>
<tr>
<td>Exploration</td>
<td>25-30 minutes</td>
</tr>
<tr>
<td>Off leash controlled play</td>
<td>5 minutes</td>
</tr>
<tr>
<td>Problem prevention topic 2</td>
<td>5 minutes</td>
</tr>
<tr>
<td>Pass the puppy</td>
<td>5 minutes</td>
</tr>
<tr>
<td>Announcements and homework assignment</td>
<td>2 minutes</td>
</tr>
</tbody>
</table>

Off leash play should be controlled. The owners need to remain seated. Treats and belongings should be out of reach from the puppies. Instructors will interrupt inappropriate play. Play should be interrupted when the target puppy is fearful and does not recover or hides, when the instigator puppy does not reciprocate the message, if you are unsure if the puppy is enjoying the play (no role reversal in a while), if the puppies are having troubles self-regulating, or if other puppies are showing signs of fear. Do not verbally or physically reprimand the puppy. Instead use a treat or squeaky toy to get the instigators attention and redirect him. Practice redirections and recalls at other times during play as well. An over exuberant puppy may need to be managed separately to help facilitate redirection.
Training topics covered over a 4-week period include: attention, targeting, sit, come, leave it/drop it, and introduction to leash manners. It is important to educate clients on the benefits of positive reinforcement training and problems associated with positive punishment. Although some basic training is introduced in puppy class, the goals of class are socialization and problem prevention rather than training. Problem prevention topics covered over the 4-week period include: crate training, restraint and handling, elimination training, socialization, isolation training, play biting, prevention of food and resource guarding, solving problem behaviors, family expectations and consistency, and chewing.

The exploration component of each class revolves around weekly themes. The goal of exploration is to expose the puppies to novel environments, people, objects, surfaces, and sounds in a fun and positive manner. Treats should be used in abundance during this time. The owners should be coached on how to read their dog’s body language, let the puppy choose to interact or not, and how to make exposure fun and enjoyable. The focus with exploration is to provide positive proactive socialization and exposure.

Pass the puppy involves the puppy being handled by other participants in class. The instructor should intervene if a puppy is overly fearful or not taking treats. It should always be the puppy’s choice to decide if they want to leave their owners and visit with others in the class. Exposure is only beneficial if it is fun and enjoyable for the puppy.

Promotion
The entire veterinary staff needs to promote puppy socialization classes. The client should hear about it from the veterinarian, technician, receptionist, kennel assistant, and groomer. Place permanent signs in the waiting room and/or each exam room detailing the importance of puppy class. Advertise puppy classes in the local newspaper or magazines. Provide a link on the hospital website with information regarding puppy socialization classes. Promote your classes on your hospital’s social media pages.

Location criteria
The instructor should be able to visualize all clients and puppies during off leash play. With a large group, the room may need to be divided. The area should be puppy proof and escape proof. The size of the class will be dependent on the location. The area should be large enough to accommodate owner seating.

Disease prevention
Because the puppies attending class will not be fully vaccinated, precautions need to be taken. Each hospital should establish a health and wellness protocol for acceptance into puppy class. This will include things such as:

- Puppies should have a clean bill of health after a recent physical examination by a veterinarian.
- Puppies should be vaccinated by a veterinarian with at least a combination modified live DAP (distemper, adenovirus 2, and parvo) vaccine at least 10 days prior to attending class.
- Ideally, all puppies should receive a bordetella (Bb) vaccine (+/- parainfluenza and/or +/− adenovirus 2) at least seven days prior to attending class. Oral administration is preferred.
- Prior to attending classes, puppies should be fecal screened for intestinal parasites and treated for roundworm/hookworm infection (usually with Pyrantel pamoate) regardless of the fecal results.
- All puppies should be started on appropriate heartworm/intestinal parasite preventive (based on regional protocols).

Other recommendations to prevent disease include:

- Change clothes and shoes prior to class (especially important with shelter and veterinary personnel)
- Disinfect surface areas before and after each class: accelerated hydrogen peroxide solutions
- Wash any food storage items after each use (dishwasher safe items should be run through a dishwasher cycle if possible). Better yet, use disposable food storage toys (such as yogurt cups, cottage cheese containers, or empty syringe casings) and throw them away after use.
- Properly clean up puppy accidents (stool or urine).
  - Remove solid waste and spray with disinfectant and wipe up with disposable or washable towels.
  - Soak up urine with disposable or washable towels, spray with disinfectant, and wipe up with clean disposable or washable towels.
  - Avoid using a wet mop to clean up urine; this will only spread it across the floor.
  - Wash your hands or use hand sanitizer after cleaning up an accident.

Conclusion
Puppy socialization classes are the number one service a veterinary hospital can offer that bonds the client to their facility and to their new puppy. Puppy classes can be started with minimal work and capital investment. They are extremely rewarding and enjoyable for the staff members.
Resources
Being able to assist clients by providing appropriate recommendations for species specific behaviors, is a vital skill for all technicians to possess. Providing outdated or inappropriate recommendations could result in further damage to the human-animal bond, relinquishment, and even euthanasia.

After reviewing a brief explanation of learning theory, a step by step behavior solution model will be analyzed. We will put problem prevention and behavior solutions into action with some common normal canine behaviors. A training problem can be defined as a normal behavior of the pet/species that humans find undesirable. In contrast, a behavior disorder occurs when the pet suffers from an underlying emotional disorder, unrelated to training. Behavior disorders, as well as training problems, can be detrimental to the human-animal bond and compromise the welfare of the pet and/or the owner. The focus on this presentation will be to provide attendees with the knowledge and skills to provide behavior solutions to normal training issues and behaviors of puppies and dogs.

Learning theory
Thorndike’s Law of Effect states: behaviors that have a pleasant consequence will increase in frequency and behaviors that have an unpleasant consequence will decrease in frequency. Anything that increases the likelihood of a behavior occurring again is considered a reinforcement. If it decreases the frequency it is considered a punishment. Reinforcement and punishment can be further broken down to positive (adding something) and negative (removal of something). Positive punishment can be defined as adding something aversive to decrease future behavior. Positive reinforcement is adding something desired to increase future behavior. Negative punishment is withdrawing something desired to decrease future behavior. Negative reinforcement is withdrawing something aversive to increase future behavior. With training, any species of animal, but especially companion animals, focusing on rewarding desirable behaviors (Positive Reinforcement) and ignoring (extinction) or removing reinforcement (negative punishment) if necessary, are generally the most humane training techniques. Our training focus is heavy on positive reinforcement. Negative punishment (removal of reinforcement) may be utilized but rarely is it necessary if the environment is managed well to help the animal be successful.

Problems with aversives
Although aversives can be used to inhibit behavior or change behavior, it is riddled with problems. Examples of positive punishment (and negative reinforcement depending how they are used) are correction based training collars (pinch collars, choke chains, electric collars) and verbal or physical reprimands. Positive punishment inhibits learning, reduces creativity, and induces fear, anxiety and conflict. It is difficult to apply consistently and is inappropriate for puppies/kittens, pets with behavior problems, teaching new behaviors, or appeasing the pet’s underlying motivation.

In order to use positive punishment effectively, it should be applied every time the behavior occurs, within a half second of the behavior beginning, at a proper intensity, and not be associated with the owner. It is difficult to meet all these criteria. Reasons to avoid positive punishment are that it does not teach the pet what to do, the trainer is focused on bad behaviors, it does not appease the pet’s underlying motivation for the behavior, and it often damages the human-animal bond. It has also been associated with owner-directed aggression.

Characteristics of dogs and successful “pet parents” or trainers
The two most influential books for me that changed my perspective on dogs and training are The Culture Clash, by Jean Donaldson and Don’t Shoot the Dog, by Karen Pryor. In The Culture Clash Ms. Donaldson discusses the characteristics of dogs. The list below of general dog characteristics has been modified from The Culture Clash (James and Kenneth Publishers, 1996).

- Dogs are amoral. They do not know right from wrong. They know safe and unsafe. For example, it is safe to get into the trash when people are absent but unsafe when they are present.
- Dogs are opportunistic and self-centered. It is about what is in it for them.
- Dogs are social. Therefore they make good companions.
- Dogs are constantly learning from their actions. Learned behaviors may be appropriate or inappropriate for human counterparts. So even when we are not actively training they are still learning. Evaluate their behavior from a learning perspective.
- Dogs explore the world with their mouths. They lack thumbs. Everything is a potential chew toy.

To be a successful ‘pet parent’ there are some simple rules to follow:
1. Be fair; understand the pet’s perspective. Pets are amoral, opportunistic, self-centered, highly social (dogs), constantly learning, and everything is a chew toy!
2. Be a good teacher. Control what the pet learns through management and supervision. Guide them into making the right decisions. Don’t waste time telling the pet what not to do. Instead, teach him the correct behavior. Set the pet up to succeed.
3. Clearly communicate to the pet when he is performing the correct behavior. Catch him doing things right and reward him for it. A high occurrence of positive reinforcement will help your pet learn quickly.
4. Be consistent. Inconsistency and unpredictability cause fear and anxiety which can be a precursor to behavioral problems. Set the rules of the house and make interactions predictable and consistent.
5. Be your pet’s advocate. He can’t speak for himself.

Meeting the social, physical, and exploratory needs of the dog
Maintaining a schedule and routine with puppies and adult dogs makes their lives more consistent and predictable. Ideally, all dogs should be meal fed twice a day (very young or small puppies may require 3 times a day initially), walked off the property twice a day for 10-20 minutes, and trained using positive reinforcement twice a day for 5-10 minutes. The pet owner should incorporate play with training. A variety of toys should be available to the dog and the toys should be rotated, so it appears that there are always “new” toys. Providing an appropriate outlet for exploratory behavior, such as “sniff” walks and food exploratory activities, provides for mental stimulation. If dogs are not provided scheduled/routine outlets to meet their social, physical, and exploratory needs, they will often find less desirable ways, from the owner’s point of view, to meet those needs.

Behavior solutions model
By following these steps, you can learn to prevent, modify, or decrease unwanted behaviors. The first step in changing behavior is to decide whether this is a normal behavior for the age of the pet and species? Puppies chew things and usually do not come completely house trained. If the undesirable behaviors have a fear, aggression, or anxiety component, then the issue may be a behavior disorder rather than a training issue.

ABC’s
Once it is determined that this is a normal behavior, just one the pet parent finds undesirable, then the ABC’s (Antecedent, Behavior, Consequence) of the behavior should be identified. The antecedent sets the occasion for the behavior to occur. Examples might be environmental stimuli (the mail man approaches the house, the owner returns home) or internal (bladder is full, dog is hungry). The behavior is what the pet does in response to the antecedent. The consequence is what happens during or immediately after the behavior, which will then affect whether the behavior will be more or less likely to occur again in the same situation.

Motivation
By looking at the ABC’s of a behavior, we can often determine the motivation, which is step 2. What is the pet getting by performing the behavior? Is the behavior self-reinforcing? In an attempt to simplify things, when using the term self-reinforcing, I actually mean reinforcement by something other than the owner’s attention. The consequence is not under the control of the owner. Am I rewarding the behavior in some way? To simplify motivation think of it in terms of it is either self-reinforcing (reinforced by something other than human attention) or reinforced by human interaction (or socially motivated).

Management/Prevention
Once the ABC’s and motivation have been determined, the ability to prevent or manage the behavior should be explored. Step 3 is can you prevent or manage the behavior in a humane way? For example: can you supervise your puppy or confine him so the majority of eliminations are only on a preferred substrate (outside)? Can you prevent the dog’s access to the front door, where he sees and hears the mailperson every day? Can we control or avoid the antecedents?

Management and controlling what the dog learns is important to prevent the learning of undesirable behaviors. Puppy owners should puppy proof the house, use baby gates, an exercise pen, and/or a crate to help manage the new puppy and set him up for success.

Also, proactively reinforcing the dog for desired behavior rather than being reactive to undesirable behavior, helps the dog learn quickly what behaviors are desired by the owner. This takes some training of the person, because in general we all tend to be more reactive than proactive. Management and prevention include being proactive and reinforcing desired behaviors, controlling the antecedents, and supervision.
Solve it!
If the behavior cannot be prevented or managed (or our management system has failed), then we have to proceed to step 4: Solve it!
There are two options depending on the motivation; Ignore the behavior or Response substitution. If the motivation and consequence has been socially motivated for human attention, the behavior should be ignored if possible. Behaviors that are not self-reinforcing or rewarding to the pet will cease to occur if ignored (i.e. no reinforcement is provided). If the reward for the behavior is human attention, i.e. he jumps on you and you push him away, he nudges you and you pet him, he barks and you toss a toy, it is likely that ignoring the pet in these situations will cause the learned behavior to cease. Ignoring means; not looking at, talking to, or touching the pet at these times. Initially, the attention getting behavior will worsen because in the past it has resulted in a desired consequence, but if you continue to ignore the behavior it theoretically should extinguish. However, the implementation of extinction can be difficult for owners to implement and can produce stress or frustration for the animal. Ignoring (avoiding reinforcing) generally works best if it is not a long-established behavior. It is also important to recognize that the emergence of numerous undesirable behaviors, may reflect a lack of adequate social, physical, and exploratory outlets for the dog. Rather than using extinction, the owner can instead proactively (before the dog does the undesirable behavior) direct the dog to a desired behavior that can be reinforced.
Behaviors that are self-reinforcing cannot be ignored. For example: to the dog, barking at the mailperson at the door makes the mailperson leave and it works every time. Some socially motivated behaviors may also not be able to be ignored and allowed to extinguish. For these behaviors, it may be necessary to use response substitution:

1. **Interrupt the behavior** by getting the pet’s attention. Clap your hands or call the pet’s name in an upbeat tone of voice. The interrupter should not be frightening, an indicator of impending punishment, or be given in a negative tone. The use of “Ah-Ah” or “No” should be avoided.

2. **Give your pet a cue for an alternate appropriate behavior** that has been previously taught to the dog with positive reinforcement training. The alternate behavior should be incompatible with the undesirable one. For example, if your dog is barking at the mail person, clap your hands or call the dog’s name (upbeat, calm tone) and ask him to come and sit. You may gently prompt the appropriate behavior with a flat collar and leash if necessary. Ideally, be proactive, get the dog’s attention before he is already in full swing of the undesirable behavior. If you hear the mailman approaching, proactively call your dog to you and reinforce with a treat. Keep him busy with play or training until the mailman leaves.

3. **Reinforce** your pet for the appropriate behavior with a food treat or other high value reinforcer. You may keep the pet busy with a food stuffed toy or several different behaviors (sits and downs) or redirect him to an appropriate activity.

**Problem solving in action**
Even after setting up an ideal environment for learning with appropriate management and a consistent routine, there will still be normal behaviors that a canine will exhibit that may be problematic for canine parents. Through interactive discussion with the audience, some common canine behaviors will be examined and the prevention and problem solving model will be applied.

**Conclusion**
By understanding normal canine characteristics and recommending to our clients appropriate humane techniques for addressing undesirable behaviors, we can enrich the relationships canine parents have with their dogs and enhance the human-animal bond.

**References**

**Resources**
Over the past decade, the importance of early environmental exposure for puppies has been recognized. However, all exposure is not the same. Positive proactive exposure is an active process that takes preparation and planning. When most people think about socialization, they think about habituation and exposure: taking the puppy or even adult dog to various places and allowing it to habituate to new environments. The problem with this is that without positive proactive socialization, a dog may become sensitized to environmental stimuli, resulting in negative associations. Socialization is not simply about habituation, it is about making exposure fun and positive.

Rather than providing a neutral experience or, even worse, an overwhelming experience, new experiences should be made positive with the use of desensitization and classical conditioning or counterconditioning:

- Classical conditioning is the process of replacing a neutral emotional response to a stimulus with, in this case, a positive emotional response. With dogs, this can often be accomplished by using treats.
- Classical counterconditioning is the process of replacing a negative emotional response to a stimulus with a positive emotional response. With dogs, this can often be accomplished by using treats.
- Desensitization is the process of reducing sensitivity or reactivity toward stimuli through gradual and controlled exposure.

Although desensitization and counterconditioning techniques are often used retroactively to treat existing fears or aversions, it is important to also use these techniques proactively to help prevent fearful associations. During positive proactive exposure, treats are used extensively. In this approach, a dog is not placed in a situation that is overwhelming or that produces a fear response. Instead, novel experiences are made positive from the start through desensitization and classical conditioning. Prevention is the key: it is much better to be proactive rather than reactive.

Some key concepts when implementing positive proactive exposure:

- Let the dog acclimate to a new environment by staying clear of the "action" when first arriving (utilize distance)
- Let the dog first observe crowds at a non-stressful distance (desensitization)
- Be proactive; assume the dog could potentially be afraid of a new person, object, or environment and use treats liberally to prevent a fear response and make a positive association from the start (classical conditioning/classical counterconditioning)
- Control what the dog learns and keep experiences positive
- Be aware of the dog’s body language at all times; be the dog’s advocate
- Do not wait for the dog to show signs of fear before initiating treats (classical conditioning)

Puppies should be positively exposed to novelty daily during the socialization period. Repeated exposure is important because dogs do not generalize well. Dogs see ‘in pictures’. Learning is situational and context specific. If the ‘picture’ changes in some way, it is new to the dog.

Socialization and exposure is an active and lifelong process.

- If a puppy parent were to stop socializing a puppy at 3 months of age, the puppy would be more likely to become fearful as an adolescent dog.
- It is much more difficult to socialize a puppy after 3 months of age without early positive foundation memories.
- As the puppy developmentally becomes more suspicious of novelty, positive exposure is probably equally important.
- Even the well socialized young puppy might regress if positive exposure does not continue into adolescence.
- After the socialization period is over, strive for at least 3 to 4 novel and/or repeated positive experiences a week. This can easily occur out on a daily walk in the neighborhood.

Tips for taking socialization and exposure on the road…

- Take a variety of pea-sized, soft, tasty, treats that have been “pre-approved” by the dog
- Bring the dog’s favorite toy
- A hungry dog will be more interested in food treats
- Remember water and a bowl, a proper fitting collar or harness, and a 6 foot leash
- Be aware of the dog’s body language and be their advocate
- Be proactive with the use of treats
- Allow the dog to explore at his own pace
- Situations should be controlled; manipulate distance and avoid overwhelming the dog
Socialization can and should be short; 5 to 10 minutes is often enough. Try to avoid going over 30 minutes unless the dog is able to have a reprieve. Measure out the amount of treats the dog will be fed and end your session before you run out.

Have Fun!

What to do if the dog becomes frightened
When addressing fear reactions, utilize the behavior modification techniques, classical counterconditioning and desensitization. These techniques previously were utilized proactively with keeping exposure positive. Here, the techniques are implemented in reaction to fearful stimuli.

Food treats are paired with the sight of or reaction to fear evoking stimuli. In an attempt to make the food change the emotional response from an unpleasant to a pleasant emotion (classical counterconditioning).

Using desensitization, gradual exposure (without fear) is accomplished by controlling the distance and intensity of the stimulus. In general, the further the dog is away from the object, the less frightened he will be.

If the dog suddenly stops taking treats or starts grabbing roughly at the treats or is no longer able to respond to known cues, the exposure has progressed too quickly. It is difficult to change the emotional response if the dog is refusing treats. Introduce the dog to the stimuli from a distance and let him approach at his own pace. Reinforce the dog for showing interest and moving towards the stimuli by giving a treat and then encouraging him to follow you away from the stimulus; thus, giving him a break.

If desensitization is implemented improperly such that fear of the stimulus is induced, the dog is likely to become more reactive (sensitized) or afraid of the stimulus with future encounters.

If a dog becomes frightened:

- Use treats liberally
- Get the dog to a non-stressful starting point
- Allow the dog to investigate at his own pace
  - Reinforce with treats and possibly movement away from the stimulus
- Avoid coddling or reprimanding the dog—try to keep your behavior the same as with positive proactive exposure

Summary
Have a plan and be prepared. Socialization is only good if it is enjoyable for the dog. Taking a proactive approach can help prevent needing to do “rehabilitation” later. Encourage pet owners to make it a fun outing for themselves and their canine companion.

Resources
Choosing the right rebreathing bag and tubes for your patient

Tidal volume is the volume of air inhaled and exhaled during each breath. Tidal volume is often estimated at 10-15mL/kg of lean body weight. Rebreathing bag size should be 3 to 5 times tidal volume. Remember to round up on the size of your rebreathing bag. For example a 20kg dog would have a tidal volume of aprox. 300mL. so if we calculated 5 x 300= 1500mL which we would round up to 2L.

When to use the rebreather vs. non-rebreather

Non-rebreathing circuits depend on high oxygen flow to remove exhaled carbon dioxide from the circuit between breaths. The decision to select a non-rebreathing circuit is often made by the weight of the animal with many clinics using a non-rebreather system for any patient weighing less than 10kg, but it is actually a decision that the patient is too small to overcome the resistance of a rebreathing circuit.

Calculating O2 flow rates

There is no universal agreement as to the proper flow rate for the various anesthesia breathing systems. The AAHA recommended flow rate of 200ml/kg/min for non-rebreathing systems is generally accepted as appropriate. That flow rate is 33 times more oxygen than is needed to meet a patient’s metabolic oxygen consumption each minute, but that high flow rate assures the patient will not rebreathe any of its exhaled carbon dioxide. The flow rate for rebreathing systems traditionally falls within 20 – 40ml/kg/min, most often settling at 30ml/kg/min.

Leak test the anesthesia machine

Before any anesthetic event it is important for you (the awesome technician anesthetist!) to do a leak check to ensure the system can properly deliver anesthetic gas and oxygen as well as properly remove CO2 and anesthetic waste gases.

- With the correct anesthesia hoses and reservoir bag attached to the anesthesia machine, ensure that the machine is correctly connected to your oxygen source and waste gas scavenging system.
- Close the pop-off valve or occlude the quick release valve. The pop off valve prevents the inadvertent buildup of pressure in the system, and should remain open except during positive pressure ventilation.
- Occlude the end of the anesthetic delivery hose with your thumb or palm of your hand.
- Fill the system by using the oxygen flush valve, fill the reservoir bag until the pressure manometer reads 20cmH2O, then stop. You can also turn on the flow of oxygen to fill the reservoir bag until the pressure reaches 20cmH2O.
- Hold pressure in the bag by continuing to occlude the end of the anesthetic delivery hose.
- Watch the pressure manometer—it should remain steady at 20cmH2O for at least five seconds.
- Open the pop-off valve to relieve the pressure in the system.

If the anesthesia machine failed the leak test, check the anesthesia delivery hose and reservoir bag for holes, and the scavenging/CO2 scavenging system for leaks. Another common location for leaks is the connection and housing for the absorber assembly, which contains the absorbent for CO2. Soda lime granules on the gaskets can sometimes prevent a tight seal. Repair or replace components as necessary, then try again until the machine passes the leak test before connecting the patient to the anesthesia machine.

What opioids do and why we love them

Opioids are considered by many to be the prototype analgesic. They have a wide range of analgesic action from ultra-short acting agents such as remifentanyl to longer acting agents such as hydromorphone. Their general reversibility makes them especially attractive in higher risk cases. And in some cases they are relatively inexpensive. They are also extremely versatile in that they can be administered via many different routes. Opioids can be given as oral tablets, intermittent injection, constant rate infusion, transdermally, or epidurally.

The effects of opioid analgesics are dependent upon the receptors at which they act. Currently, there are three major classes of opioid receptors recognized within the CNS. They are as follows mu, delta, and kappa. All three classes of opioid receptors produce some level of analgesia.

Drugs acting on opioid receptors are also classified as being agonists, partial agonists, mixed agonist/antagonists, and antagonists.
Opioid agonists
These drugs have high affinity for the mu opioid receptors responsible for analgesia and sedation. Opioid Agonists include: Morphine, hydromorphone, oxymorphone, fentanyl, methadone, etc.

Partial Agonists
These drugs by definition are only partially as effective as agonists. This is because its binding with the mu opioid receptor produces an effect that is less pronounced than that of an opioid agonist such as fentanyl. An example of a partial agonist would be buprenorphine.

Mixed agonist/Antagonists
These opioids work by exerting an agonist effect at the kappa receptors being responsible for sedation and some analgesic properties. They also act as an opioid antagonist at mu receptor sites. Agonist/antagonist opioids can include butorphanol and nalbuphine. These drugs can also be used to reverse some of the unwanted side effects of full agonist opioids such as excessive sedation. (Wagner, 2009)

Antagonists
These drugs work to fully antagonize and reverse the effects of opioids at the mu and kappa receptors. Drugs in this category include naltrexone and naloxone. These drugs will cause increased alertness. They will also reverse the analgesic effects of opioids so opioid antagonists should be used with caution in the painful patient.

Local blocks are your friend!
Local blocks are a cheap and easy way to add additional analgesia. Local blocks can be considered for all procedures from dentistry to surgery. Specific local blocks will be discussed in depth during the lecture.

HR & ECG
CATS: under anesthesia HR 120bpm- 250bpm. Small Dogs under anesthesia: 80-140bpm Large Dogs under anesthesia: 50-80bpm. It is important to keep in mind what the heart rate, respiration and/or ECG were on the on PRE-OP exam. Also keep in mind what anesthetic drugs were given, as will they effect HR. The ECG is simply a recording of the electrical activity in the heart. The following are a few important ECG waveforms you should know:

- **Sinus Arrhythmia**: Variation in sinus rhythm related to respiration and resulting from vagal tone inhibition. Heart rate increases with inspiration and decreases with expiration.
- **Sinus Tachycardia**: A regular sinus rhythm with a heart rate above 160 bpm in adult dogs (220 bpm in puppies, 180 bpm in toy breeds, and 140bpm in giant breeds) and above 240 bpm in cats.
- **Atrial Tachycardia**: A supraventricular tachycardia where the P wave configuration differs from sinus P waves. The rate is rapid, but the rhythm may be irregular.
- **Atrial Fibrillation**: Numerous unorganized ectopic foci in the atria discharge impulses at very high rates causing uncoordinated activity of the atria and loss of effective muscular propulsive movement. Atrial complexes appear as erratic fibrillatory waves.
- **Ventricular Premature Complexes** (VPC’s): An ectopic beat originating in the ventricles. You will see no P-wave associated with the QRS complex. This can be a problem because it decreases cardiac output because of decreased filling time for the ventricles. VPC patterns that require special attention are; Bigeminy (when every other beat is a VPC) and pairs or triples of VPC’s.

Blood pressure management
Blood pressure is typically recorded as two numbers, written as a ratio like this: 110/80 mmHg. Systolic: The top number, which is also the higher of the two numbers, measures the pressure in the arteries when the heart beats (when the heart muscle contracts). **Diastolic** is the bottom number, which is also the lower of the two numbers, measures the pressure in the arteries between heartbeats (when the heart muscle is resting between beats and refilling with blood)

Hypotension is one of the most common anesthetic complications; Hypotension is usually defined as mean arterial blood pressure less than 60mmHg or systolic pressure less than 90 mmHg. If blood pressures are too low (hypotension) you can start by decreasing your inhalant anesthetic level if possible. Often when lowering your inhalant, you will need to provide additional sedatives or analgesics to maintain the patient at an acceptable level of anesthesia. Discuss with your doctor an IV bolus of your pre-medicant opioid or benzodiazepine. A second step should be to increase the fluid rate if possible. If the patient has no underlying cardiac issues, consider a quick bolus of 10 ml/kg (5 ml/lb) over 5 minutes. Also, verify proper cuff selection, a cuff that is too large will result in falsely low readings. If the pet is somewhat bradycardic, consider a dose of an anticholinergic such as glycopyrrolate. A next step may involve adding a colloid. Vetstarch (hydroxyethyl starch) is made from natural sources of starch. Vetstarch increases the volume of blood plasma. You can also consider discussing with your attending clinician administering dobutamine or dopamine infusion.

ETCO2 considerations
Capnography indicates how much CO2 is being eliminated from the lungs by measuring exhaled CO2 with a device that senses the CO2 level. It is a sensitive indicator of lung function and may help guide the doctor, nurse, or respiratory therapist to adjust the breathing machine or it may provide an early warning that the lungs are not functioning properly.
Post op patient management
There are many ways that post-op pain can be treated. The most important aspect of managing chronic 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 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.

References
Bednarski, R. et al. 2011 AAHA Anesthesia Guidelines for Dogs and Cats
Low stress handling is an invaluable component of veterinary examinations and care for all patients. Although most of our practice focuses on pet dogs, we do need to also be aware of the unique aspects of low stress handling for working dogs. Low stress handling is important for both dogs and cats, although this presentation will focus primarily on dogs. As the first person to interact with the patient, the veterinary technician sets the tone of the visit and can create the low stress environment that allows the rest of the visit to go smoothly for the pet, the client and the whole veterinary team.

Training your clients’ pets for veterinary visits
Very few clients train their pets to incorporate behaviors that would facilitate veterinary care or utilize prevention strategies like fitness and conditioning which may help reduce the incidence of injuries. Providing owners with the tools and motivation to incorporate low stress handling as well as conditioning exercises will help establish a strong working relationship and make your job easier! The best time to introduce low stress handling and positive veterinary visits is when the animal is young. An important focus should be making the veterinary visit fun and positive. This can be done by having the owner bring their dog or cat into the hospital for a social visit. Have them stop by and get a treat or play a game or just get a nice massage. This helps reduce the association of hospital visits with pain and stress. For practices that offer a rehabilitation facility, many pets will physically benefit from the conditioning but will also associate visits with fun and feeling better. At home, the owner should practice restraining the pet. This is important not only for veterinary visits but if the pet were ever injured. The owner may want to have a special mat (yoga mats work well and can be brought with the pet for every visit) that the pet learns to lie down and “settle”. The client should practice touching the pet’s feet and head and ears and moving its legs and tail. To associate this with a positive event, food rewards are typically most effective. Important pointers for this type of training is that the food should not be overly exciting and the owner should talk in a calm quiet voice and use slow gentle body movements. Utilizing a table or elevated platform for the examination (as long as there is a nonslip mat) can actually facilitate the calming response. The owner can use this training to provide preventive care, like brushing the teeth, cleaning the ears and trimming the nails. Dr. Sophia Yin’s book and videos on Low Stress Handling provide the nuances of this type of training. In addition, the owner can muzzle train their pet, even if the dog is the gentlest dog in the world. If the pet becomes injured, the ability to place a muzzle safely and without creating stress will make care of the pet much easier. An easy way to muzzle train a dog is to use a little peanut butter or squeeze cheese on the inside of the muzzle (basket muzzles are preferred). This gets the dog to willingly put its face in the muzzle. Once they are doing that without fear, the food can be delivered after the dog puts its face in the muzzle, thus conditioning the dog that the muzzle signals good things to come.

Techniques and strategies for those unanticipated visits
Despite all of the planning and training, there will be times that a pet comes to your hospital and is not relaxed! This will be more evident in the case of an emergency since the owner will also likely be upset and the pet will respond to the emotion of the owner. There is a balance between minimizing stress and providing timely care. Obviously in any life-threatening emergency, immediate stabilization is paramount. The attending veterinarian will need to make the decision of whether the owner’s presence is beneficial or detrimental for the stress and care of the pet. If they are to be separated, regular and clear communication is essential. If the situation is not emergent, then taking the extra time to minimize the stress of the examination will pay back in a better client relationship and a more cooperative patient. A thorough knowledge of body language is a valuable tool when aiming for low stress interactions. The ability to recognize the subtle signs of stress, particularly in dogs that maintain a high level of self-control – until they absolutely lose it. Be alert to ear and tail carriage, displacement behaviors, appeasement behaviors and avoidance behaviors. If the condition does not preclude the use of food during the exam, peanut butter (as long as there are no peanut allergies in the handler or staff), squeeze cheese or small tasty treats may be enough to gain confidence and cooperation of the dog. If the stress level is high, then the dog will probably not be willing to take food. Cats are a whole different animal, and low stress handling techniques are well described by Dr. Sophia Yin, in this lecture we will focus on dogs. Ideally, a stressed pet should be examined in a quiet(er) room with low lighting and good flooring. Yoga mats can provide a safe non-slip surface on the floor or exam table and may help reduce the stress. Ideally, owners should bring their own yoga mats to the hospital. The use of classical music has some potential calming effects. For many dogs the use of massage may be successful in creating a state of relaxation. There are several massage techniques that have been described and even certifications in animal massage. For relaxation massage, circular motions around the neck and ears can be effective. Acupuncture or acupressure on GV20 (Bai Hui) located at the top of the head on the dorsal midline in a notch at the rostral end of the external sagittal crest, may also provide some calming. There are several products or techniques that aim to reduce stimulation. A cloth cap that covers the dog’s eyes has been advocated for dogs that are visually stimulated by their environment. A snug fitting body wrap has also been recommended for dogs that have thunderphobia or body sensitivity. An ace bandage can be
wrapped around the neck, body and legs to apply pressure to similar points and may have some calming effect. There are also pheromone products available for both dogs and cats in atomizer or collars that may help calm the pet if the level of anxiety is not too high. If these approaches don’t work then moving to chemical restraint is an option.

**The value of low stress handling**

It is much easier to work with animals that are not clouded by the effects of stress. The clinical signs observed will be reflective of the underlying problem rather than masked by anxiety or fear. This will lead to a more rapid assessment and ability to provide appropriate care. It is also less draining for the staff to work with animals that are not fearful. Fear in all species can interfere with clear thought and performance! Staff can feel that the animals are getting the best care if they are not being forcibly restrained or wrestled and everyone’s safety is more secure. Finally, clients that participate in this low stress approach will be more connected to your practice.

**Low stress handling of the working dog team**

As a rule, canine handlers, regardless of their discipline (police, scent detection, assistance) are committed to the care of their working partner. Canine handlers often spend more of their time with their canine partner than with any one else (including their spouse) and often the handler relies on their canine partner for life saving service. The ongoing training builds an understanding and bond that puts the handler and dog in a unique synchronicity. It is commonly considered that emotions run up and down the leash through this very strong bond. It is also possible that the dog may manifest physical or behavioral changes that are a reflection of the handler’s mental or physical state. It is invaluable to develop a relationship with working canine teams before a crisis happens. Working together with the handler and their dog will build confidence and reduce stress to both the handler and the dog. The liability issues of having a handler restrain their dog may be different than those associated with having a pet owner restrain their dog, but this has not been tested in the courts. Surprisingly, despite the commitment and investment, many canine handlers are not well educated about the medical needs of their partner. One of the first components of establishing a relationship with the handler is to involve them in the active care of their dog. Handlers should be encouraged to attend canine first aid courses, whether they are offered by the Red Cross, other organizations or by your practice. Handlers will recognize changes in behavior that may underlie a more serious condition, long before any veterinarian can identify a physiologic disturbance, but they often don’t know how to take a pulse or even recognize the signs of gastric dilatation and volvulus! It is vital to involve the handler in all decisions related to the care of their partner. The ability for the dog to work may be influenced by the condition which brought them to your practice (which may be a result of their occupational hazards), by the procedures or the medications deemed necessary. The attending veterinarian should understand the type of work that the dog is expected to perform and whether the dog must function in the immediate future. For a detection dog or police dog, an acute illness will result in the dog having time off work; however, for a guide dog or a medical detection dog, time that they are not working could be a potential risk for the handler. While the effects of sedation are obvious, it is not clear how long a dog’s performance will be impaired after anesthesia. For dogs that rely on olfaction, drugs and diseases can impair their performance. There is very little information about the impact of drugs on canine olfaction. To be safe, medications should be considered carefully prior to prescribing for working dogs.

**Resources**


Ohio State University Behavior Dept

https://indoorpet.osu.edu/veterinarians/implementinglowstress

Penn Vet Working Dog Calm Husbandry

https://www.youtube.com/watch?v=kgwAzyKeILU

Body wraps for anxiety


Fear Free

https://fearfreepets.com/

Penn Vet Working Dog Center – Certified Working Dog Practitioner Program

www.PennVetWDC.org
At their most basic, fluids are aqueous solutions with different solutes in different concentrations formulated for different purposes. The types of solutes that are contained in fluids can include: electrolytes, dextrose, synthetic colloidal particles, proteins (albumin and clotting factors) and red blood cells. Approximately 60% of body weight consists of water that is distributed into different body compartments. 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 called extracellular water is distributed between the intravascular (25%) and extravascular (75%) spaces.

The electrolyte composition of water in the intracellular space and extracellular space is quite different. Within the cells the most ubiquitous cation is potassium with relatively small contributions from sodium, calcium and magnesium. The major anions in intracellular fluid are phosphates, proteins and bicarbonate. These gradients are maintained through an energy dependent process with the sodium-potassium ATPase pump being the most important contributor.

Contrast that with the electrolyte composition of extracellular water in which sodium is the predominant cation with negligible contributions from potassium, calcium and magnesium and chloride is the major anion with relatively minor contributions from bicarbonate, phosphates and proteins. The kidneys maintain the concentrations of electrolytes within the extracellular fluid.

The balance of three forces determines water movement within the body: tonicity (osmolality), oncotic pressure and hydrostatic pressure. Small molecules such as electrolytes or sugars within the water determine tonicity. These molecules can move freely between the intracellular and extracellular fluid space according to concentration gradient although they are unable to cross cell membranes without a specific transport system. Oncotic pressure or colloid osmotic pressure is determined by the concentration of larger molecules such as proteins that are able to move freely between the intravascular and extravascular spaces. Hydrostatic pressure is the mechanical pressure exerted by water against a membrane and is not related to any particles present in solution.

Patients may require fluid therapy for a variety of reasons including increased fluid losses such as diarrhea or vomiting, decreased fluid intake or fluid shifting between compartments. The fluid types available can be broken down into crystalloid type fluids (isotonic, hypotonic and hypertonic) or colloid type fluids (synthetic, natural, blood products and blood substitutes).

Isotonic crystalloid fluids contain water-soluble molecules such as electrolytes and sugars; they do not exert oncotic pressure and can be used for the replacement of isotonic fluid losses such as dehydration or shock. These fluids are often referred to as replacement fluids because the electrolytes contained in the fluids are similar to the electrolytes in plasma (i.e. the fluids replace the plasma). Replacement fluids rapidly expand the plasma volume but for only a short amount of time with 75% of the fluid administered leaking into the interstitial space within 30 minutes. Common additives to replacement fluids include potassium chloride and dextrose. The most common replacement fluid options are 0.9% sodium chloride, Normosol R/ Plasmalyte 148, and lactated Ringers solution.

Maintenance fluids are used to replace the amount of fluid and electrolytes that are consumed by the body on a daily basis. Since normal daily fluid loss is hypotonic, these fluids are also hypotonic and typically contain less sodium and chloride but more potassium than replacement fluids. Examples of maintenance fluids include Normosol-M / Plasmalyte 56 and 0.45% NaCl. Occasionally, it is necessary to replace pure water without the presence of any electrolytes; in this case the fluid of choice is D5W which is water with 5% dextrose added to prevent thrombophlebitis.

The last category of crystalloid fluids is hypertonic fluids. These are typically used to shift fluid from one compartment to another and they may or may not contain electrolytes. The most commonly used hypertonic fluids are hypertonic saline (3, 5, 7.2%) or mannitol.

Colloids are aqueous solutions that contain large molecules incapable of readily crossing the vascular membrane. These molecules may be synthetic (starch compounds) or natural (proteins) and most use a base solution of 0.9% sodium chloride. Colloids are generally used to shift fluid between compartments and then hold the water within the plasma compartment. The increase in plasma volume following colloid administration is usually slightly greater than the volume of fluid infused. The degree of fluid shifting that occurs is secondary to the number of molecules present whereas the duration of the effect is related to the size of the molecules. The most common formulations available are hetastarches (eg. Hespan) and tetrastarches (Vetstarch). Natural colloids include human and canine albumin preparations and plasma products (fresh frozen or frozen plasma).

Devising a fluid treatment plan begins with calculating a fluid rate. This calculation must include the patients daily maintenance needs, (fluid lost through metabolism), any dehydration that was present and any ongoing losses that are occurring including respiratory losses, urinary losses and fecal losses. Many formulas are available to calculate maintenance rates. For ease of use the following formula is most frequently used: fluid rate in mL = [Body weight (kg) x 30] + 70. The second step is determining a route of administration, the most common options include: intravenous, intraosseous, subcutaneous and oral. If the intravenous route is selected a catheter can be inserted into a cephalic vein, saphenous vein (medial or lateral) or jugular vein following surgical...
preparation of the site. Ideally the largest gauge catheter that fits comfortably with the vein is selected and once placed is secured with tape.

Fluids can be delivered via infusion pump or gravity. If using gravity it is necessary to determine how many drops per mL the selected administration set requires. The hourly fluid rate is converted to mL/min by dividing the hourly rate by 60. This mL/min rate is then multiplied by the drops per mL to determine how many drops per minute are needed. After instituting a fluid plan it is important to monitor the patient’s response to therapy by following daily body weights and clinical changes.

Several complications can arise with fluid therapy. Possible complications include electrolyte disturbances, fluid overload (colloids), blood clotting disorders (colloids), allergic reactions and thrombophlebitis, and may be due to fluid type selection, fluid administration rate or catheter management. To prevent complications from occurring electrolytes should be checked daily, respiratory rate should be monitored and the catheter insertion site and limb should be evaluated daily.
It is not a secret that diabetes mellitus is a common endocrinopathy seen in the canine and feline patient. Studies have shown that the frequency of this disease has increased over the years. Defining the type of diabetes, proper treatment and obtaining remission are now reachable goals with feline diabetes mellitus and the veterinary team.

There are two types of diabetes mellitus. Type I is typically seen in dogs and is caused by insulin deficiency due to either destruction or reduced to secretion of insulin by the beta cells in the pancreas. Type II diabetes is typically seen in a cat. It is caused by either insulin resistance or the inability of the beta cells to function normally. It can also be some degree of both dysfunctions in a cat.

Insulin is a hormone is produced in the inslets of Langerhans in the pancreas and is essential for life. Its functions in the body include, to lower the level of glucose in the body, it causes blood glucose, amino acids, and fatty acids that are in the blood to be absorbed into body cells and used for energy. The pancreas is primarily an exocrine gland responsible for the secretion of pancreatic fluid into the digestive tract following a meal. The islets of Langerhans, scattered throughout the pancreas, contains hundreds of thousands of cluster cells. The islets are endocrine tissue which contains four different cell types. The most numerous are beta cells which are responsible for secreting insulin and amylin, followed by the alpha cells which produce glucagon, the delta cells which produce somatostatin and lastly, the gamma cells which produce pancreatic polypeptide. Housed within the cell membrane of the beta cells are channels that detect the presence of glucose. The normal response of the beta cell, based on the channels ability to detect glucose, is to produce/release insulin when an increase in circulating blood glucose is detected to maintain a normal circulating level. So the pancreas is made up of both exocrine and endocrine tissue.

Glucagon aids in metabolism and the use of glucose in the pancreas. It stimulates the liver to convert glycogen, the storage form of glucose, to glucose also stimulating glucogenesis. The end goal of glucagon is to raise the level of blood glucose in the body. Glucagon, produced by the alpha cells of the pancreas, may be thought of as a “counter-signal” to insulin. Simply stated, glucagon is secreted by the pancreas in response to insulin falling below normal levels.

Insulin sensitivity and/or dysfunction beta cells can be the cause. Insulin sensitivity is the inability of insulin to lower blood glucose levels. If insulin sensitivity is lowered the body will need more insulin to meet the same goal as a normal body. Insulin sensitivity is also known as insulin resistance. So, insulin resistance and beta cell dysfunction is type II diabetes.

Risk factors for diabetes mellitus include; obesity, breed, sex, age, medication and lifestyle. Multiple studies have shown that obesity is a huge risk factor due to abnormal hormonal release and inflammatory mechanisms. Male cats are more predisposed but females can also develop diabetes mellitus. If patients are on medications such as glucocorticoids it will put them at risk of developing diabetes mellitus.

So if the body is not functioning correctly, constant hyperglycemia occurs in becomes toxic to the beta cells. This is glucose toxicity and causes further dysfunction. Of glucose levels are closely regulated and glucose toxicity is resolved, diabetic remission can occur.

A CBC, chemistry, urinalysis, and urine culture are recommended to diagnose diabetes mellitus in the canine and feline patient. Hyperglycemia and glucosuria in patient with clinical signs of diabetes mellitus is confirmation of the diagnosis. A blood glucose concentration above 250 to 300 mg/dl is diagnostic and clinical signs are present.

A high-protein, low carbohydrate diet, such as Purina DM, can be helpful in treating diabetes mellitus and can minimize postprandial increases in blood glucose and reduce insulin requirement in some cases. Avoiding high, simple carbohydrate foods such as semi moist foods can make treatment successful. Some cats may not need insulin therapy at the time of diagnosis and only require diet therapy. Those patients should be monitored closely because most will progress to needing insulin. Feeding and insulin administration should always be done at the same time to help eliminate a hypoglycemic episode. Correction of body weight can help reduce insulin resistance and in type II diabetes mellitus, it will improve insulin secretion. In some cases weight control and feeding a proper diet can properly control the blood glucose levels.

In the canine patient nutrition is important as well since almost all canine diabetics are insulin dependent their dietary management will not remove the need for insulin like their feline counterpart. However, nutrition in the canine diabetic can help improve glycemic regulation. A high carbohydrate and fiber diet will minimize postprandial fluctuations of glucose after meals.

**Interesting facts about insulin dosing**

Insulin will be absorbed more quickly in a warmer environmental temperature due to increased peripheral blood supply. Insulin is broken up into categories, they include: short acting insulin, intermediate acting insulin, and long acting insulin. In veterinary medicine, studies show that each patient is different when it comes to duration of action and peak times. This is why it is so important to do in clinic and at home monitoring.
**Intermediate acting insulin**
Protamine zinc insulin, PZ I, is composed of a mixture of beef and pork insulin. In the feline patient PZI has previously been indicated as long-acting insulin but a study showed that they can be used every 12 hours at 0.25-0.5 U/kg for adequate control of diabetes mellitus. (7)

**Long acting insulin**
Glargine insulin, Lantus, is a human insulin that is marketed a long acting, peak less insulin. The duration of action is typically 18 to 24 hours with a peak at around 7 hours. The dose used is 0.25-0.5 units/kg every 12 hours. This dose can be changed on an individual basis by the veterinarian based on home and in clinic monitoring. Some studies have shown that newly diagnosed cats on Glargine insulin have a higher probability of remission and better glycemic control than patients on PZI insulin by day 17 of treatment.

Detemir or Levemir is a long acting human insulin that can be used in cats with diabetes mellitus. Detemir can be used in cats at the same recommendations as glargine.

**Remission**
Diabetic remission is achievable in the feline patient. The individual’s therapy should be tailored with permission as the therapeutic goal. It has been shown that remission rates can be as high as 90%. If remission for the individual is not achievable, control of clinical signs (polyuria, polydipsia and weight loss) along with glycemic control should be obtained. Close monitoring at home and at the clinic, insulin therapy and proper diet will achieve the goal of remission. All patients can come out of remission at any point. Concurrent infection and other systemic illnesses will make remission harder to achieve.

Frequent visits to the clinic for glucose curves or home monitoring is important. After starting therapy, glucose levels may need to be checked daily with weekly complete glucose curves at home or at the hospital. Glucose curves should last 12 hours and be checked and recorded every 3 to 4 hours.

**Monitoring**
Complete glucose curves when adjusting insulin doses can be as often as every seven days. Order compliance and attention to detail is important to help monitor the progress or decline of the patient. Frequent exams to monitor progress and ensure no concurrence illnesses will help keep the patient in good health. Lab work such as, CBC, biochemistry panel, urinalysis and urine culture may need to be performed with exams if indicated. Fructosamine can be used if the patient is stressed or if the glucose curve results do not match the client’s history. The fructosamine is a blood glucose average over the past 2 to 3 weeks. This number is only an average and does not indicate the severity of daily fluctuations so therefore it should not be used to adjust insulin doses.

**Home care**
Home care for the feline diabetic patient is very important. The technician will play a huge role in client education and helping the clients become accustomed to home care for their diabetic cat. Insulin administration, dosing of insulin, buying insulin syringes, buying a glucometer, clinical signs of hyperglycemia and hypoglycemia, proper diet and home monitoring are the main topics of client education. The clients keeping a journal of eating and drinking habits, glucose readings and dosage administration will help them communicate with the veterinary staff on recheck visits. The technician will need to teach clients how to give insulin injections and choose proper locations for blood glucose checks. If the patient is in need of a new diet or a weight loss program, the technician will need to educate them of proper feeding protocols.

**References are available upon request.**
The definition of “Triage” is the following, to the art and practice of being able to assess patients rapidly and sort them accordingly to
the urgency of treatment required. Management of multiple emergencies is always a challenge to the complete staff of a veterinary
clinic. It is very important to be able to assess or “triage” a patient accurately and quickly in these situations. Developing a process in
the clinic is a must. All support staff should know their roles with emergencies and be comfortable with them. Role playing and drills
are important before this arises so each person can practice their skills. A receptionist should know how to give clear directions to the
clinic and ask appropriate questions when the owner calls the clinic to pass on pertaining information to the doctor and technician.

Taking a quick scan of the room when you enter will help direct you to which patient needs assessing first. Immediate recognition
of a life threatening emergency is the key to successful treatment. Always treat the patient with the most life threatening condition
first. As a technician taking a brief but direct history is essential as you assess the patient. Asking the owner the following questions
are a good start for the history and a complete history can be taken after the patient is stable, what happened to your pet? How long
ago did this happen? Does your pet have any known allergies? Has the pet had any past medical problems? Is your pet on any
medications? If yes, what dose and when was the last dose given?

A patient that is having a seizure is more critical than a patient that hasn’t been eating for 5 days. A patient that has been hit by a
car and is standing in the lobby with abducted elbows needs medical attention before the cat that is straining in the litter box. As you
can see all of these conditions need medical attention and it is your duty as a technician to triage the patients appropriately.

Patient assessment
(The following section is adapted from the author’s contribution in writing from the VSPN Notebook ®, A CRASH PLAN)

A - Airway
- Evaluate if the patient has a patent airway
- Is there any type of foreign body or obstruction?
  - Use the “finger sweep” method and/or suction to evaluate an obstruction of the airway
  - Use caution if patient is conscious
  - When an upper airway foreign body is present, it is necessary to perform an emergency tracheotomy

B – Breathing
Is the patient breathing?
- No- intubate immediately and start life saving measures
  - First, breathe 2 large breaths for the patient with 100% oxygen
  - Then, breathe 8-10 bpm for the patient with 100% oxygen
- Yes- evaluate the patient for dyspnea
  - What are the patient’s mucous membrane colors?
  - Refer to C-Circulation for descriptions
  - What is the patient’s pulse oximetry status?
  - If below 90%, provide oxygen supplementation.
  - What is the patient’s Partial Pressure of Oxygen (PaO2) in the arterial blood?
  - An arterial/venous blood gas will need to be drawn and evaluated
  - 80-110 mmHg = normal
  - >80 mmHg = hypoxic
  - >/= 60 = initiate oxygen therapy
  - When oxygen concentration is above 21% (room air) the PaO2 values are different. The expected PaO2
    should be 5 times the fraction of inspired oxygen (FiO2). For example if the FiO2 is 40% then a Pa O2 of 200
    mm Hg would be considered normal.
  - Venous levels of oxygen will always be lower than arterial.

What is the respiratory rate and pattern?
- Normal- dog 15-30 and cat 20-30
- Rapid and shallow- also known as choppy or “dys-synchronous” respiratory pattern- pleural space disease
- Slow and deep- also known as “Kussmaul” respiration- may indicate metabolic acidosis in patients with diabetic
  ketoacidosis or renal failure
Postures and patterns that indicate dyspnea
- High pitched stridor on inspiration - may indicate an upper airway obstruction, i.e. laryngeal paralysis/edema, foreign body aspiration
- Head Extension- trying to elongate the airway to maximize each breath.
- Abducted Elbows- allowing more movement from the chest cavity to maximize each breath.
- Abdominal Breathing- on expiration abdominal muscles will push the remainder of each breath out if the chest wall is not functioning correctly.
- Cheyne Stokes- normal or hyperventilation followed by periods of apnea or hypoventilation, indicative of a disorder of the central respiratory center.

Auscultation
- Crackles- suggestive of pneumonia, pulmonary edema, pulmonary contusions or fluid overload
- Muffled- suggestive of pleural effusion, pneumothorax or hemothorax
- Wheezes- suggestive of feline bronchitis, obstruction, lower airway disease or feline asthma

C- Circulation/Cardiovascular
What is the patient’s mucous membrane color?
- Pink- normal
- Cyanotic (blue) - lack of oxygen
- Icteric (yellow) - liver disease
- Red- toxins, shock
- Pale Pink- hemorrhage or anemia
- Brown- intravascular hemorrhage or acetaminophen toxicity

What is the patient’s circulation status?
What percent is the patient dehydrated?
- Less than 5% - history of fluid loss but no significant findings on physical exam
- 5%-7% - oral mucous membranes are dry without panting or tachycardia
- 7%-10% - mild to moderate degree of decreased skin turgor, dry oral mucous membranes, tachycardia with normal pulses.
- 10%-12% - moderate to severe degree of decreased skin turgor, dry oral mucous membranes, tachycardia and decreased pulse pressure.
- 12% or greater- severe degree of decreased skin turgor, dry and pale mucous membranes, tachycardia, severely decreased pulse pressure.

What is the patient’s heart rate and rhythm?
- Palpate pulses
  - What is the patient’s pulse quality, and are they synchronous with the heart rate?
    - Pulses should be synchronous with the heart rate
    - Non-synchronous pulses with heart rate can suggest an arrhythmia or obstruction in circulation
- Perform non-invasive blood pressure
  - Feline normal (mm Hg) - Systolic 100-160, Diastolic 60-90, MAP 80-120.
  - Canine normal (mm Hg) - Systolic 100-160, Diastolic 60-90, MAP 80-120.
- Perform an electrocardiogram; if any abnormalities are found notify the veterinarian on duty immediately.

Is the patient presenting with a form of shock?
- Hypovolemic Shock- most common form of shock - due to fluid loss of any type (hemorrhage, volume loss or third spacing of fluids)
  - Clinical signs of canine shock- 1st stage (Compensatory Shock) - tachycardia, hyperthermia, hypertension, injected mucous membranes, rapid capillary refill time and normal pulse quality. Second stage (Early Decompensatory Shock)-pale mucous membranes, tachycardia, prolonged capillary refill time, hypotension, hypothermia and dull mentation. Third stage (Late Decompensatory Shock)-pale to cyanotic mucous membranes, bradycardia, severe hypotension, pulses weak or absent, hypothermia, stuporous mentation, organ failure and cardiac arrest.
  - Clinical signs of feline shock- Clinical signs of the 1st stage not generally seen. 2nd stage (Early Decompensatory Stage)-bradycardia, hypothermia and hypotension, weak peripheral pulses, pale mucous membranes, weakness and general collapse. The 3rd stage (Late Decompensatory Shock)-same as canine.
- Cardiogenic Shock- seen in any heart failure that impedes cardiac output, characterized by pump failure and increased central venous pressure
  - Pump failure- due to cardiomyopathy arrhythmias and valvular dysfunction
Clinical signs include heart murmurs, jugular distention, collapse, rails or crackles noted on thoracic auscultation, systemic hypotension, tachycardia, increased central venous pressure, increased oxygen needs and decreased cardiac output.

- Distributive Shock- seen in sepsis, anaphylaxis, neurologic diseases and pharmacologic or toxic reactions
  - Normal phases of hypovolemic shock occur.
- Traumatic Shock- seen with extensive tissue trauma
  - Can be seen in conjunction with hypovolemic shock

Is there any arterial bleeding?

- Note any external wounds
- Place pressure bandages to any hemorrhaging wounds

Place a large bore intravenous catheter to administer fluids and necessary medications

Institute treatment if hypovolemic or traumatic shock is present

- Shock doses for crystalloid fluids
  - Canine- 90 ml/kg/hr
  - Feline- 45 ml/kg/hr
- Administration of shock fluids
  - Start with ¼ shock dose over 15 minutes
  - Reassess the patient’s heart rate, respiratory rate, mucous membranes, capillary refill time and non-invasive blood pressure
  - If patient is still dehydrated, start the 2nd, ¼ dose over 15 minutes and reassess
  - Repeat until patient is rehydrated or until “shock dose” is complete

CPR

Recognition of a patient in cardio-pulmonary arrest is very important. After recognizing that the patient is not breathing, the first thing to do is to capture an airway. After establishing an airway either by endotracheal intubation or emergency tracheostomy it is important to ventilate for the patient correctly. Ventilate the patient at a rate of 10 breaths/minutes with a tidal volume of 10 ml/kg.

The oxygen flow rate should be 150 ml/kg/min

External chest compressions should be started next by placing your hands over the fourth and fifth rib space. Compressions should displace the chest wall by 25-50%. They should be done at a rate of 80-120 times/min. Most dogs and cats can be in left or right lateral recumbency, if the dog is barrel chested, they should be in dorsal recumbency. If only one team member is present CPCR can still be done, breathing twice then doing 30 chest compressions and repeat cycle until further help arrives. Internal chest compressions should be done in specific situations only, such as with a penetrating thoracic trauma or if the patient is in the operating room.

Monitoring the effectiveness of chest compressions during CPR is essential. This can be done by palpation of pulses in the femoral artery or by applying a Doppler monitor to the eye of the patient and listening for blood flow. If femoral pulses are not palpated or noise heard on the Doppler the technique must be adjusted. Repositioning the patient or changing the person doing compressions are the first things to do with inadequate compressions. Remember maintain blood flow and oxygen to the brain and vital organs is the goal in CPR. The most accurate way to monitor the effectiveness of CPR is end tidal carbon dioxide (ETCO2). The capnograph, which monitors the ETCO2, fits between the end of the endotracheal tube and oxygen source. The ETCO2 will be slightly elevated with effective compressions.

Indications for the use of drugs in CPR are, to control life threatening emergencies, increase heart rate, and to improve myocardial oxygenation. Routes of administration vary with each drug. Common routes include, intratracheal (IT), intracardiac (IC), intravenous (IV), and intraosseous (IO).

There are several cardiac rhythms that are common with CPR. They are the following, ventricular asystole, pulseless electrical activity and ventricular fibrillation. Ventricular asystole is characterized by the absence of both mechanical and electrical activity. Treatment is to use epinephrine and atropine. Pulseless electrical activity is without adequate mechanical activity to cause sufficient cardiac output (pulses). It can be caused by insufficient myocardial oxygenation. Treatment includes Narxone, epinephrine and atropine. Ventricular fibrillation is when chaotic, disorganized ventricular activity is seen.

No perfusion to the body takes place when this arrhythmia occurs. Treatment includes external defibrillation at a dose of 2 joules/kg. If that dose does not convert the rhythm, it can be increased. If fibrillation does not convert the rhythm, then epinephrine is administered.

Defibrillation is more successful when used early in CPR. It eliminates the arrhythmia by sending an electrical current through the heart. This allows the cardiac cells to depolarize and then repolarize all at the same time then ideally the heart will return to normal function. To defibrillate a patient paddles are used and one paddle is placed on each side of the patient’s chest over the heart. Gel is placed on the paddles before placing them on the patient. The person holding the paddles must yell “clear” to inform all the other team members of what is happening, then making sure no one is touching the patient, they can discharge the defibrillator. If someone
is touching the patient when it is discharged, they WILL be shocked as well. Remember isopropyl alcohol is flammable and metal
tables will carry the electrical charge. If the patient is on a metal surface they must be removed before defibrillation occurs. Prolonged
life support includes any complications after successful resuscitation. In most cases reoccurrence of cardiopulmonary or respiratory
arrest is high with in the first four hours. Cerebral resuscitation is a huge concern due to the lack of blood flow to the brain during
CPR. During CPR, hypoxia and ischemia occur which leads to cerebral edema.

Monitoring the patient is critical following CPR. Using an EKG to monitor electrical activity of the heart, SPO2 monitor the
oxygen status of the patient and supplying oxygen if necessary. Monitor either invasive or non-invasive blood pressures, and regular
physical exams including pupillary light responses, motor function and breathing patterns are done frequently to monitor the patient’s
cerebral function. Almost always these patients will need oxygen supplementation via an oxygen cage, flow by, or nasal insufflations.
The heart will almost always need support in the first 4 hours following successful CPR.

Practicing with a case scenario is a good way to get you ready for that day when more than one emergency comes through the door
at the same time. Use the following questions to help guide you through those situations:

- What is most likely wrong with the patients?
- Does one or both of these emergencies have a life threatening condition?
- Which emergency needs medical attention first?
- What do we do with the other emergency for the time being?
- What should we be concerned with for the top priority emergency?
- What should we be concerned with for the other emergency?

References available upon request.
Infectious diseases

Infectious diseases are commonly seen in the ICU. Infectious disease is a disease that can be transmitted by a specific kind of contact. There are many infectious diseases that the feline patient can have. They include; parasite, virus, fungal and bacterial. Written protocols should be in place for infectious disease. Proper personal protective equipment (PPE) should be worn with these patients. It should be mandatory for all personnel to follow that plan. The plan should include what PPE to wear, where to house the patient, how to deal with their wounds (if they have any) and how to clean up after them.

Proper cleaning protocols and adhering to them is a must. The author’s place of employment uses bleach to wipe everything down and then use a steam cleaner and allow surfaces to air dry each time after treating a known multi drug resistant (MDR) patient. Everything that the infected patient comes into contact with must be cleaned properly.

If the patient has open wounds, transporting patients around the hospital in a designated carrier will help eliminate contamination. Also don’t forget to protect patients from nosocomial infections by keeping all wounds and incisions clean, dry and covered at all times when in the hospital.

The veterinary staff wearing gowns, gloves and booties at all times when in contact with the MDR patients and keeping them in a separate ward are common standard protocols for MDR patients. If the patient is considered critical and needs to be in ICU or a fluid ward, proper precautions are made. Proper PPE is worn at all times, they are kept in a cage that is considered a low traffic area, so at our hospital they are kept in the back of the room with an empty cage between them and another patient, just to help establish a barrier. Separate laundry and trash cans are used with MDR labels on them. The laundry is washed separately and the use of laundry detergent with bleach is necessary to properly disinfect the laundry.

A large draped area is placed on the floor in front of their cage so when they need to come out of the cage for exams, treatments they are placed on the draped area and not the floor. That drape is changed at least every 24 hours. If they have open wounds, a designated area should be used to perform examinations and treatments to not contaminate multiple areas of the hospital. Separate instruments, stethoscopes and thermometers are used and kept for these patients. In the author’s place of employment, an infectious patient receives a set of instruments while hospitalized that is used on them and when they leave they are disinfected and sterilized. Then you are not using your stuff to monitor the patient and infecting all of your other patients while on your shift.

Keep visitations with owners to a minimum and the owners have to wear proper PPE when visiting. Separate exam rooms are used for these patients. And doing any procedures with a MDR patient should be done at the end of the day so there is time for proper cleaning protocols to take place and to limit the number of patients being exposed.

These are very serious infections and should not be taken lightly, not only are you protecting the other patients in the hospital but you are protecting yourself. Usually veterinary personnel seek medical advice if they know or think they have been infected by the patient. If you think you have been infected by a MDR patient, seek medical attention, do not hide it. If you are immunosuppressed, it may be a good idea to remove yourself from any high risk situations.

Patient assessment

Assessment of the airway, breathing and circulation when triaging a patient is important. Evaluate if the patient has a patent airway. Obvious foreign body or obstruction can sometimes be seen while approaching the patient. Use the “finger sweep” method and/or suction to evaluate an obstruction of the airway. Use caution if patient is conscious. When an upper airway foreign body is present, it is necessary to perform an emergency tracheotomy.

Assessment of breathing can also be done while approaching the patient. A technician should immediately note if the patient is breathing. If they are not, intubate immediately and start life saving measures and breathe 8-10 bpm for the patient with 100% oxygen. If the patient is breathing, things to consider are “What are the patient’s mucous membrane colors?” (Refer to C-Circulation for descriptions) “What is the patient’s pulse oximetry status?” If below 90%, provide oxygen supplementation. “What is the patient’s Partial Pressure of Oxygen (PaO2) in the arterial blood?”

An arterial/venous blood gas will need to be drawn and evaluated. Values below reflect normal on an arterial blood gas; Hg = normal, >80 mmHg = hypoxic, >/= 60 = initiate oxygen therapy. (When oxygen concentration is above 21% (room air) the PaO2 values are different.) The expected PaO2 should be 5 times the fraction of inspired oxygen (FiO2). For example if the FiO2 is 40% then a PaO2 of 200 mm Hg would be considered normal. “What is the respiratory rate and pattern?” Normal- cat 20-30 rpm. In the hospital it can elevate to 40 rpm. Rapid and shallow- also known as choppy or “dys-synchronous” respiratory pattern- pleural space disease
Postures and patterns that indicate dyspnea in a patient include high pitched stridor on inspiration. This may indicate an upper airway obstruction, i.e. laryngeal paralysis/edema, foreign body aspiration. Head Extension an indicate trying to elongate the airway to maximize each breath. Abducted elbows can indicate the patient is trying to allow more movement from the chest cavity to maximize each breath. Abdominal breathing on expiration abdominal muscles will push the remainder of each breath out if the chest wall is not functioning correctly. Cheyne Stokes is a breathing pattern that is identified by normal or hyperventilation followed by periods of apnea or hypopventilation, indicative of a disorder of the central respiratory center.

When auscultating a patient, abnormalities will include crackles, wheezes or muffled heart sounds. Crackles are suggestive of pneumonia, pulmonary edema, pulmonary contusions or fluid overload. Muffled cardiac sounds are suggestive of pleural effusion, pneumothorax or hemothorax. Wheezes are suggestive of bronchitis, obstruction, lower airway disease or feline asthma. When assessing circulation, it is important to note the mucous membrane color, heart rate, pulse quality and dehydration status. Pulses should be synchronous with the heart rate. If the pulses are not synchronous it is suggestive of an arrhythmia or obstruction in circulation.

Lastly to assess circulation status it is important to determine if the patient presenting with a form of shock? Hypovolemic Shock is the most common form of shock and is due to fluid loss of any type (hemorrhage, volume loss or third spacing of fluids) Clinical signs of feline shock- Clinical signs of the 1st stage not generally seen. 2nd stage (Early Decompensatory Stage)-bradycardia, hypothermia and hypotension, weak peripheral pulses, pale mucous membranes, weakness and general collapse. The 3rd stage (Late Decompensatory Shock)-pale to cyanotic mucous membranes, bradycardia, severe hypotension, weak pulses, stuporous mentation, organ failure or cardiac arrest. Clinical signs of canine shock include; 1st stage (Compensatory Shock) - tachycardia, hyperthermia, hypertension, injected mucous membranes, rapid capillary refill time and normal pulse quality. Second stage (Early Decompensatory Shock)-pale mucous membranes, tachycardia, prolonged capillary refill time, hypotension, hypothermia and dull mentation. Third stage (Late Decompensatory Shock)-pale to cyanotic mucous membranes, bradycardia, severe hypotension, pulses weak or absent, hypothermia, stuporous mentation, organ failure and cardiac arrest.

Cardiogenic Shock is seen in any heart failure that impedes cardiac output, characterized by pump failure and increased central venous pressure. Clinical signs include-heat murmurs, jugular distention, collapse, rails or crackles noted on thoracic auscultation, systemic hypotension, tachycardia, increased central venous pressure, increased oxygen needs and decreased cardiac output. Distributive Shock- seen in sepsis, anaphylaxis, neurologic diseases and pharmacologic or toxic reactions and the normal phases of hypovolemic shock occur. Traumatic Shock is seen with extensive tissue trauma and can be seen in conjunction with hypovolemic shock.

Immediately placing a large bore intravenous catheter to administer fluids and necessary medications will be indicated in these patients. Patients with heart murmurs should be assessed carefully and fluids are administered at a lower rate to not further their condition. Under the guidance of the veterinarian starting crystalloid fluid therapy is first line treatment for hypovolemic shock. The canine shock dose of crystalloid fluids is 90 ml/kg/hr. The feline shock dose of crystalloid fluids is 45 ml/kg/hr. Administration of shock fluids will include starting with ¼ shock dose over 15 minutes, at the end of the first 15 minutes reassess the patient’s heart rate, respiratory rate, mucous membranes, capillary refill time and non-invasive blood pressure. If patient is still dehydrated, start the second, quarter dose over 15 minutes and reassess. Repeat until patient is rehydrated or until “shock dose” is complete.

CPR
The following recommendations for CPR are adapted from the RECOVER Initiative from AVECC and VECCS. Recognition of a patient in cardio-pulmonary arrest is very important. After recognizing that the patient is not breathing, the first thing to do is to capture an airway. After establishing an airway either by endotracheal intubation or emergency tracheostomy it is important to ventilate for the patient correctly. Ventilate the patient at a rate of 10 breaths/minutes with a tidal volume of 10 ml/kg. The oxygen flow rate should be 150 ml/kg/min.

External chest compressions should be started next by placing your hands over the fourth and fifth rib space. Compressions should displace the chest wall by 25-50 %. They should be done at a rate of 80-120 compressions/minute. Most cats can be in left or right lateral recumbency. If only one team member is present CPR can still be done, breathing twice then doing 30 chest compressions and repeat cycle until further help arrives. Internal chest compressions should be done in specific situations only, such as with a penetrating thoracic trauma or if the patient is in the operating room.

Monitoring the effectiveness of chest compressions during CPR is essential. This can be done by palpation of pulses in the femoral artery or by applying a Doppler monitor to the eye of the patient and listening for blood flow. If femoral pulses are not palpated or noise heard on the Doppler the technique must be adjusted. Repositioning the patient or changing the person doing compressions is the first things to do with inadequate compressions. Remember maintain blood flow and oxygen to the brain and vital organs is the goal in CPR. The most accurate way to monitor the effectiveness of CPR is end tidal carbon dioxide (ETCO2). The capnograph, which monitors the ETCO2, fits between the end of the endotracheal tube and oxygen source. The ETCO2 will be slightly elevated with effective compressions.
Exhaustion one is feeling. This theory is based on not only exhaustion but cynicism and inefficacy as well. It is a nice thorough guideline that can be used to help assess burnout. In the past burnout has been solely regulated by the amount of depression. Although a topic like burnout is highly controversial, the Maslach Burnout Inventory is one proven approach to burnout. It has been shown that most people exhibiting signs of burnout also meet the criteria for clinical "standard" has been reached in the psychology world to determine if someone is exhibiting signs of burnout. This is called the Maslach Burnout Inventory. There have been several studies done on burnout, first being studied in the 1970’s. Since then several studies have been done and a “standard” has been reached in the psychology world to determine if someone is exhibiting signs of burnout. This is called the Maslach Burnout Inventory. It has been shown that most people exhibiting signs of burnout also meet the criteria for clinical depression. Although a topic like burnout is highly controversial, the Maslach Burnout Inventory is one proven approach to burnout. It is a nice thorough guideline that can be used to help assess burnout. In the past burnout has been solely regulated by the amount of exhaustion one is feeling. This theory is based on not only exhaustion but cynicism and inefficacy as well.

Signs of burnout include exhaustion, getting 8 hours of sleep in important to keep yourself rested and relaxed. If you work an off shift, such as nights or weekends, it is important to still get appropriate amounts of sleep. Black out curtains, sleeping masks or other sleep aids will help you feel rested and ready for your day at work. Relaxation is important in preventing burn out. Taking time off periodically to rest and restore will prevent burn out. Lack of motivation, if you are dragging yourself into work and thinking you hate your job the entire way there, which is a sign of burn out. Complications at home or work, relationships with the people around you, either at home or work can cause stress in your life. Find someone to talk to, friend, family member, supervisor, that can help you with interpersonal relationships. Not taking care of yourself, each day it is important to eat properly, exercise, rest and take time for yourself. Health problems, health problems can contribute to burn out if you do not feel you are getting enough rest or the job you are performing causes anxiety or pain in your every day routine. Don’t be afraid to ask for help. Take time for doctor’s appointments so you can be healthy and enjoy your life.

Tips to help with burn out include starting the day with a relaxing ritual, do something you love every day, adopt healthy eating, exercising, and sleeping habits, set boundaries, take a daily break from technology, nourish your creative side and learn how to manage stress. Everyone’s stress level is different, you have to learn how to manage yours and figure out what is best for you.
Common calculations commonly used by the ICU technician include the following:

- **Drug calculations:**
  - Units needed = weight (kg) x dose
  - Amount needed = dose/concentration of drug
- **Whole blood transfusion mL needed**
  \[ \text{CAT} = \text{patient weight (kg)} \times 70 \times (\text{desired PCV} - \text{current PCV}) \]
  \[ \text{PCV of donor blood} \]
- **RER (Resting Energy Requirement)** = 70 x weight (kg) to the 0.75 power
- **MER (Maintenance Energy Requirement)** = activity or illness factor x RER
- **Food dosage** = kcal required/caloric density of food
- **Fluid deficit (L)** = % dehydration (decimal) x weight (kg) x 1,000 mL
- **Drip rate** = \( \frac{\text{volume of solution mL x drops/mL}}{\text{volume in drops/minute (or ggt/min)}} \times \text{Time (in minutes)} \)
- **mL/hr** = \( \frac{\text{volume of solution mL}}{\text{time in hours}} \)
- **CRI Calculations** - \( \frac{\text{drug mL/hr x amount of fluid (ml)}}{\text{fluid mL/hr}} \)
  - Need to know the dose rate of the drug
  - Need to know the patient's body weight
  - Need to know the fluid administration rate
  - Need to know the drug concentration

References available upon request.
Administration of blood products can be beneficial to critical patients. The blood products should come from a trustworthy program, either commercial or private. The benefits of the transfusion should outweigh the risk for the recipient. Potential risks to the recipients would include transmission of infectious diseases from the donor if not properly screened or a transfusion reaction. Even though you may in the trenches there are a few key tools to know about transfusion medicine, all of those will be discussed here.

Component therapy has become important in veterinary medicine and is used today instead of transfusing whole blood to every patient in need. Fresh Whole Blood (FWB) is a unit of blood that has been obtained less than 8 hours prior to administration. It contains all the cellular and plasma components of the blood. It is to be administered to patients who are in need of red blood cells, plasma and platelets. Stored whole blood still contains all the cellular and plasma components of the blood except platelets. It is to be stored at 4 ºC and has an expiration date of 28 days. It is used in patients that are anemic and hypoproteinemic.

Packed Red Blood Cells (pRBC) is a unit of blood that has had the red blood cells separated from the plasma content within 8 hours of collection. This blood component should be stored at 4º C and has an expiration date of 42 days. The most common indication for a pRBC transfusion is anemia.

Fresh Frozen Plasma (FFP) is a unit of blood that has been separated from red blood cells and the plasma components are remaining. FFP is viable for up to 1 year when it is stored in temperatures of -20 to -40 ºC. All coagulation factors, albumin and protein are present in this component. A common indication for use is primary or secondary coagulopathies. FFP transfusions have also been proven to benefit patients with acute pancreatitis, disseminated intravascular coagulation (DIC), liver failure, rodenticide toxicity and parvo virus. Frozen or Stored Plasma (SP) is frozen plasma that has been stored at temperatures of -20 to -40 ºC for greater than 1 year. It is viable for up to 2 years. Stored plasma no longer contains clotting factors. Indications for this component include hypoproteinemia and hypoaalbuminemia.

Other blood products such as cryoprecipitate, cryoprecipitate-poor plasma and platelet rich plasma are also available as component therapy. Commonly those types of components are obtained from commercial blood banks due to the infrequency of use and cost associated with preparation of the product. Cryoprecipitate contains high levels of fibrinogen, fibronectin, factor VIII and von Willibrán’s factor. It is indicated in patients with coagulopathies due to any of the above plasma protein deficiencies. Cryoprecipitate-poor plasma is indicated in patients that are hypoproteinemic but the risk of synthetic plasma expanders outweighs the benefit. Platelet rich plasma is only viable for 5 days at a constant agitation. Most commercial blood banks make platelet rich plasma available.

Blood typing can be an important step in the transfusion process. A canine blood donor that has never received a blood transfusion is considered to be a universal donor if they are DEA 1 negative. It is thought that the most important canine blood type is DEA 1 because it has a strong alloantibody response after sensitization. Other canine blood types include DEA 1.1, 1.2, 1.3, 3, 4 and 7.

Feline blood types include A, B and AB. Feline blood type A is the most common and feline blood type AB is the rarest. Feline blood types A and B have naturally occurring alloantibodies that can cause severe, life threatening transfusion reactions. The blood type AB does not have naturally occurring alloantibodies but should receive type A blood if they need a transfusion. Simple blood typing cards can be purchased to be used in the clinic to determine the blood type of the recipient. Even though a blood type has been performed on the recipient and donor, it should never take the place of performing a crossmatch before the transfusion to ensure the two are compatible. Bedside agglutination cards are available from DMS laboratories, The Rapid Vet Company©, Rapid Vet-H Canine and Feline. This is a rapid cross matching system that can be done reliably when performed properly.

Cross matching can be a fast and useful tool to help determine if the patient will have a transfusion reaction. A crossmatch will look for the presence of alloantibodies of the recipient’s blood or plasma against the donor’s blood or plasma. A major crossmatch will look for alloantibodies in the recipient’s plasma against the donor’s red blood cells. A minor crossmatch will look for alloantibodies in the donor’s plasma against the recipient’s red blood cells. The presence of agglutination will determine an incompatible crossmatch. If the patient is already exhibiting auto agglutination or hemoglobinemia, some cross matching methods may be undiagnostic.

Administration of blood products should be through a commercially made filtered administration set. The rate should start out at approximately 25% of the calculated dose for the first 30 minutes to one hour of the transfusion. The patient should have a temperature, pulse and respiratory rate (TPR), blood pressure, mucous membrane color and capillary refill time (CRT) performed before and then every 10 minutes for the first hour. At the end of the introductory period the rate can be increased to the calculated rate and vitals should be performed every 30 minutes to 1 hour for the entirety of the transfusion. To reduce the risk of bacterial contamination of the transfusion, it should be administered over a four hour period.
Transfusion reactions can be divided into immunologic and non-immunologic reactions. The febrile nonhemolytic transfusion reaction (FNHTR) is a common immunologic reaction noted. Non-immunologic reactions include; transmission of infectious diseases from the donor to the recipient and sepsis induced bacterial contamination from the unit or volume overload.

If the reaction is mild the treatment therapy can consist of stopping the transfusion and monitoring the patient’s vitals. Restarting the transfusion in approximately thirty minutes at a reduced rate can usually be handled by the patient. Other clinical signs seen in a mild transfusion reaction include; fever, urticarial and facial edema.

Moderate transfusion reactions can include clinical signs of fever, tachycardia, tachypnea or vomiting. The transfusion should be stopped and glucocorticoids can be administered. Supportive care such as fluid therapy should be assessed individually in each patient. The patient’s vitals should be monitored closely and the transfusion can be restarted if necessary. Severe transfusion reactions can be life threatening but are rare. Clinical signs will include tachypnea, hypotension, collapse, fever, bradycardia or even sudden death. The transfusion should be stopped immediately and administration of epinephrine intravenously. Supportive care for the patient can include intravenous fluids, oxygen therapy or CPR. If bacterial infection or sepsis is suspected in the patient, blood cultures of the patient and the product should be performed.

Transfusion medicine is an important part of critical care medicine. Even though risks are present with administration of blood components, lives can be saved.

References available upon request.
So how does the patient get from the uncomplicated diabetic patient to the complicated diabetic? There are many different reasons why a patient could suddenly have a complication of diabetes. The common complications are diabetic ketoacidosis (DKA), insulin resistance, hyperglycemic hyperosmolar syndrome and hypoglycemia.

Getting to the bottom of it will take good history taking skills and a little detective work. Some things to consider are insulin ineffectiveness due to the following:

- Inactive insulin: Be sure to ask the owners how the insulin was stored. There are some general guidelines for insulin storage and handling. Insulin should never be frozen, used beyond the expiration date or exposed to direct heat or light. Each insulin formulation has specific guidelines and should be included on the product insert.
- Diluted insulin: Insulin dilution is a popular practice with the very small patients because their dose is tiny and hard to accurately pull up in a syringe do to the volume of the dose. This should not be done unless absolutely necessary and dilution should only be done by a licensed pharmacist.
- Improper administration technique: ask the owner to show you where on the pet they are administering the insulin dose
- Improper dose: ask the owner to show you on the appropriate syringe how much insulin they are pulling up
- Incorrect frequency of dose: always ask the owner what time(s) the insulin is administered, not just how many times daily. There can be a large variance between “twice a day” and 6am and 6pm
- Impaired insulin absorption: dehydrated patients do not have adequate tissue uptake of drugs injected by the subcutaneous route

Once insulin ineffectiveness is ruled out, possible insulin resistance should be considered in the patient.

Insulin resistance is a condition when a normal dose of insulin produces a less than ideal clinical response. Many diseases can cause insulin resistance, some common causes in dogs may include:

- Hyperadrenocorticism – Cushing’s syndrome is a result of too much circulating cortisol. The effects of cortisol on the metabolism of carbohydrates will decrease the cellular utilization of glucose and increases glucose output from the liver.
- Exogenous steroids – administration of corticosteroids for the treatment of another disease can result in the same physiological response as a patient with Cushing’s syndrome.
- Concurrent systemic infections – Diabetics may have underlying renal compromise due to the increase in protein in the urine brought about by elevated blood glucose which can cause urinary tract infections.
- Hyperthyroidism - The thyroid gland affects the metabolic rate as well as the rate of energy use, and the absorption of nutrients. Hyperthyroidism causing insulin resistance is actually rare in the feline patient. (1)
- Acromegaly-There has been documentation of elevated growth hormone secretion as well causing insulin resistance in the feline patient.
- Concurrent systemic illness- It has been proven that pancreatitis, renal disease, liver disease or cardiac disease will cause insulin resistance in the feline patient.

Common diseases that cause insulin resistance in cats include:

- Acromegaly
- Exogenous steroids
- Concurrent infections
- Hyperthyroidism
- Concurrent renal, liver or cardiac disease

**Diabetic ketoacidosis**

DKA is a result of an improper balance of concentrations of all the hormones insulin, catecholamines, glucagon, cortisol and growth hormone. An insulin deficiency in the body is counter regulated by an excess of the catabolic hormones, especially glucagon. Now there is hyperglycemia present in the body, when the concentration of glucose exceeds 260-310 mg/dl in cats it exceeds the renal threshold, spilling into the urine. Osmotic diuresis is present with significant calorie loss, polyuria and polydipsia. Lipase is activated by the improper insulin: glucose ratio in the body so it then mobilizes adipose. Adipose is stimulated for the primary energy source because of the loss of calories and unavailability of glucose and insulin to the body.

Long chain free fatty acids then transport the fat to the liver. Liver ketone formation is preferred over transformation into triglycerides due to the increase of glucagon. Ketone bodies produced by oxidation of free fatty acids change into acetone and acetoacetate becoming an acid. In a normal body, ketone are then metabolized by tissue to form carbon dioxide and water then used
to form bicarbonate. The bicarbonate is then used to help buffer another ketone in the extracellular fluid. In a diabetic body the ketone formation in the liver will exceed the muscle’s ability to metabolize the ketone which will cause accumulation in the blood. So then the excessive production of ketone combined with the reduced production of bicarbonate will result in ketonuria and eventually metabolic.

During osmotic diuresis the body will lose not only glucose but sodium, potassium and water in the urine. The body will compensate for all the negatively charged ketone loss in the urine by excreting additional positive charged electrolytes, those include sodium and potassium. More sodium will be lost through the kidney due to lack of insulin in the body. Sodium is the primary extracellular electrolyte that holds water within that space. The regulation of sodium balance in the kidneys and the maintenance of effective circulating volume are closely related. The changes in effective circulating volume are triggered by specific volume receptors in the cardiopulmonary circulation, the carotid sinuses, aortic arch and the kidneys. This activates a series of effectors throughout the body to correct the volume depletion. Most of the receptors will then sense a change in pressure and dilate or constrict to compensate for the change in circulating volume. The receptors that are located in the renal afferent arterioles then activate the renin-angiotensin-aldosterone system (RAAS). The non-renal receptors will help govern the activity of the sympathetic nervous system.

Now total body water is significantly decreased and the patient is hypovolemic and if left long enough untreated in a state of hypovolemic shock. This will lead to prerenal azotemia and a decreased glomerular filtration rate increasing the amount of ketones and glucose in the blood even more and finally resulting in metabolic acidosis. With circulating cortisol and epinephrine in the blood because the body is in a “stressed” state, this will increase the level of glucose in the blood even more, exacerbating the patient’s condition. Metabolic acidosis is the result of the exchange of a hydrogen ion for intracellular potassium. Insulin is required to drive potassium back into the cell so with the decreased amount of insulin, potassium will then become extracellular. Most serum chemistry profiles only measure extracellular levels and the total body concentration of potassium is not considered to be decreased.

The most common acid/base abnormality in DKA is metabolic acidosis. It develops because of several different reasons but almost always causes an elevated anion gap. Anion gap is the mathematical difference in measured cations and anions and represents the unmeasured anions. The anion gap is increased in DKA because the concentration of unmeasured anion in the blood is increased due to the production ketoacids and the decrease of bicarbonate concentration. The most important cause for metabolic acidosis in DKA is the production of acidic ketones. Fatty acids that are released can be used for energy in most tissues including the liver but without insulin free fatty acid conversion to triglycerides is impaired. When this process is impaired triglycerides are converted to ketones instead of being oxidized to carbon dioxide. So the liver is then reset to metabolize free fatty acids due to the lack of insulin and increased glucagon to favor ketone production instead of oxidation of fatty acids to carbon dioxide. The other reason for acidosis is the overproduction of lactic acid due to the impaired tissue perfusion from dehydration, shock and reduced renal excretion of hydrogen ions. If the disease has progressed far enough, mixed acid/base disturbances will be seen in the patient. Neurological compromise will lead to depressed respiration (respiratory acidosis) or metabolic alkalosis can be seen with vomiting and diarrhea.

Some clinical signs that the owner may report are, polyuria, polydipsia, lethargy, weakness, hyperventilation, anorexia, vomiting, diarrhea, weight loss, depressed, or coma. Many of these patients have some type of underlying or secondary disease. Other clinical signs include, abdominal pain, neurological abnormalities ranging from depressed mentation to abnormal gait to a coma. Weight loss, muscle wasting, and cataracts can be seen. Some people report a “fruity” odor in the patient’s breath due to the overwhelming amount of ketones in the patient. This is not a reliable clinical sign to use for diagnosis.

Initial database for an emergency patient should include blood glucose, PCV/TS, urinalysis, venous or arterial blood gas and a biochemistry panel. Quick analyzers can be purchased to run some of these tests while waiting on full panels. A glucometer will have results in a matter of seconds if your clinic does not own bed side analyzers. Urine glucose and ketone reagent strips are available for fast results while waiting on full urinalysis. A PCV/TS will give information on your patient’s dehydration status and can be read in just a few minutes. Some bed side analyzers now have the capabilities to run venous or arterial blood gases for your convenience. The diagnosis of DKA is confirmed by the presence of hyperglycemia, glucosuria, ketonuria, and metabolic acidosis. Other abnormalities can include hyponatremia, hypochloremia, hypokalemia, increased anion gap, and azotemia. Treatment of DKA patient can be tricky and time consuming. Correcting dehydration and electrolyte imbalances should be done first. A large bore peripheral intravenous catheter should be placed immediately. When the patient is stable a long indwelling jugular catheter, which fluids can be administered and blood samples can be drawn from should be considered. Hypoperfusion and dehydration should be replaced immediately with crystalloid fluid boluses and colloid fluids if needed. The typical shock dose of crystalloids is 60 ml/kg/hr in the feline patient. Remember that if your patient has concurrent heart disease, the rate of fluids may need to be tailored to fit that patient. Once hypovolemia is corrected, the fluid rate will need to be adjusted to correct the total fluid deficit.

Monitoring any patient that is receiving intravenous fluids is important. Any acute changes in body weight can be a sign of improper changes in water. Any patient that is losing weight while on fluid therapy is not receiving adequate amounts of fluid. Monitoring blood pressure, heart rate, respiratory rate is essential as well. Central venous pressure can be measured and used as a guide for fluid therapy replacement. Readings under 5 cmH2O are indicators of inadequate fluid replacement. Patients that still have
proper renal function, a dehydrated animal will have a urine specific gravity of >1.025. On the other hand fluid overload should be monitored as well. In patients that are experiencing fluid overload or over hydration you will see an increase of nasal discharge, chemosis, increased respiratory rate, pulmonary congestion, crackles, and eventually pulmonary edema will develop.

Knowing which type of fluid to pick off the shelf and why is important. Typically, 0.9% Sodium Chloride is the fluid of choice in the emergency phase of DKA. It is an isotonic fluid and has the highest concentration of sodium compared to other fluid types, which is important to correcting the sodium deficit. Lactated Ringer’s Solution should be avoided at this time due to the presence of lactate in the solution. The hepatic metabolic process to make bicarbonate from the lactate is the same process used to metabolize ketones, reducing the liver’s ability to correctly metabolize lactate. Poor perfusion will also aide in the retention of lactate because it is negatively charged and in the effort of the body trying to maintain electrical neutrality will dump even more sodium and potassium into the urine to be excreted.

Rehydration alone will improve hyperglycemia, acid/base status and electrolyte imbalances. Supplementation of electrolytes may need to be provided additionally due to the dilution effect of fluid administration. Tissue perfusion will be restored and proper urine production will be restored improving metabolic acidosis. Proper tissue perfusion will also help reduce the amount of lactate in the body helping to reduce the amount of sodium and potassium the body puts in the urine. All of this will help the body to restore normal amounts of electrolytes. Rehydration will also help reduce the concentration of ketones and glucose in the body because of the dilution effect. So as you can see the dilution effect of fluid therapy is important to remember in patients it helps reduce the high concentrations but can reduce them too much. Frequent chemistry panels should be ran on these patients, in the beginning it may be necessary to run chemistry panels every 4 to 6 hours and then as your patient becomes more stable, decreasing the frequency to every 12 hours and eventually to every 24 hours. In the author’s experience, a daily chemistry panel is performed until the electrolytes stay within normal ranges; the patient is considered euvoletic and eating on their own.

Regular insulin is suggested in the initial treatment of DKA and is continued until the patient is stable and ketosis has resolved. Therapy is adjusted to reach blood glucose of 250 – 300 mg/dl in approximately 24 hours. Insulin therapy should begin approximately 2 – 4 hours after fluid therapy. Fluid therapy alone will help decrease the concentration of glucose from dilution effect and urine production in the body. If the glucose is dropped too quickly it can result in cerebral edema and a coma. The maximum drop in blood glucose should not exceed 75 – 100 mg/dl/hr. There are several different recommendations to administration and dosage of regular insulin in the initial treatment of a DKA patient. The advantages of regular insulin are, it can be administered intravenously (IV), intramuscularly (IM), and subcutaneously (SQ), it has a rapid onset of action and a short duration of action.

Intramuscular and especially subcutaneous injection may not be absorbed properly if the patient is hypovolemic. Hourly IM injections of regular insulin can be done successfully if needed. The initial dose would be 0.2 – 0.25 U/kg with follow up doses of 0.1 – 0.2 U/kg hourly. The regular insulin is continued until the patient’s ketosis is resolved. When blood glucose drops <250 – 300 mg/dl the hourly dose is decreased by as much as 50%. At that point a 2.5 – 5% dextrose containing solution is started and if the blood glucose drops <100 mg/dl, insulin is temporarily discontinued until it rises above 150 - 200 mg/dl. If the blood glucose drops below 60 mg/dl a 1-2 ml/kg bolus of 25% dextrose should be administered and glucose measurement taken every 30 minutes to 1 hour until rises above 100 mg/dl. Blood glucose measurements are performed every 1 -2 hours until it is continuously in the 250 – 300 mg/dl range.

Regular insulin can be successfully administered IV as well. A popular treatment method is to add the dose of insulin into a 250 ml bag of 0.9 % NaCl to administer it to your patient. The dose of insulin to be added to the 250 ml bag of saline is 1.1 U/kg for the feline patient. This concentration will be started at a rate of 10 ml/hr and infused only with an infusion pump. 50 mls of the solution should be run through the line and discarded to allow insulin to properly bind to the plastic in the tubing. This will allow for immediate, proper dosing of insulin to the patient. Monitoring the patient is the same as with IM injections of regular insulin that was described above. Once the patient’s blood glucose is stable and they are eating, the insulin can be switched to intermediate acting insulin.

Hyperglycemic hyperosmolar syndrome (HHS)

This is an uncommon complication of diabetes in the cat. HHS is diagnosed when the feline patient has hyperglycemia, above 600 mg/dl, hyperosmolality, above 350 mOsm/kg, and dehydration without to ketosis. (5, 8) In HHS it is thought that hepatic glucagon resistance and small amounts of insulin prevents ketosis. It is not known specifically why some feline patients develop HHS instead of DKA as a complication of diabetes mellitus but most patients with HHA have a concurrent illness. Patients can show neurological clinical signs with this syndrome. Some patients are non-responsive to anticonvulsants and only respond to insulin therapy and rehydration in these situations. Neurological clinical signs are assumed to occur due to cerebral dehydration secondary to the hyperosmolality. HHS will occur more commonly, with concurrent disease such as, cardiac or renal failure, pancreatitis, sepsis, and/or steroid therapy.

Treatment of HHS and DKA are similar with the goals to correct dehydration restore electrolyte losses and provide adequate insulin to correct any metabolic defects. Correcting dehydration and hyperosmolality with 0.9% NaCl is necessary before starting
insulin therapy. If the blood glucose is decreased to rapidly this it will cause a decrease in extracellular fluid osmolality, which will cause cerebral edema. The prognosis of HHS is poor due to the high incidence of serious concurrent disease.

**Hypoglycemia**

Hypoglycemia can occur with an insulin overdose. This can be avoided by client education and making sure the client understands how to properly pull up and administer the correct insulin dose. Clinical signs usually include ataxia, weakness, behavior abnormalities, depression, shaking, seizures, coma, or death. When the clinical signs of hypoglycemia are first recognized owners can offer food or apply Karo® syrup to the mucous membranes of the patient until veterinary treatment can be performed. One the patient has arrived at the hospital, 50% dextrose can be administered per the request of your veterinarian. When an IV catheter is placed a dilution of 1:4 of 50% dextrose can be administered. This bolus can be repeated as necessary. A CRI of 2.5 % to 5% dextrose should be started to prevent recurrence and continued until the patient can eat.

References available upon request.
Feline Hyperthyroidism is a common disease seen in feline patients. Every technician should understand pathophysiology of the thyroid gland including how the gland affects the body in a normal and abnormal state. As well as diagnostic and treatment options which will help the technician become a better patient advocate and help educate clients on those options.

**Physiology of the thyroid gland**

The thyroid gland helps regulate many different parts of the body. It is one of many glands in the body that makes up the endocrine system. The thyroid gland is located below the larynx on each side of the trachea and is one of the largest endocrine glands in the body. Secretion of the hormones thyroxine (T4) and triiodothyronine (T3) is the primary function of the thyroid gland. These hormones control the rate of metabolism within the body. When the body secretes too much of the thyroid hormones it is termed “hyperthyroidism” and when the body does not secrete enough of the thyroid hormones it is termed “hypothyroidism”. When the thyroid gland needs to secrete more of the thyroid hormones, the anterior pituitary gland will release thyroid-releasing hormone (TSH). TSH will be secreted to the thyroid gland and in return the thyroid gland releases the hormones. Both T4 and T3 are as equally as important within the body even though T4 is secreted at a much higher rate than T3. T3 is four times more potent and but lasts a shorter amount of time than T4.

Iodine is needed to complete the formation of T4. Only a small amount is needed in the weekly diet. In humans, this was the reason why iodine was added to table salt. When iodides are ingested, they are secreted from the gastrointestinal tract into the blood and then thyroid gland will then transform it into an oxidized state and use them to complete the formation of T4. Once the hormone is released it binds with plasma proteins that are synthesized by the liver. The hormones are then introduced to the tissues of the body slowly. T4 is introduced every 6 days and T3 every day. Once they are introduced to tissue, they will bind again with intracellular proteins.

Calcitonin is the third hormone that is secreted from the thyroid gland. Calcitonin, Vitamin D and the parathyroid hormone (PTH) are all closely intertwined to help control the formation and regulation of calcium and phosphate metabolism as well as bone and teeth formation. Calcium specifically plays a role in this activity by decreasing plasma calcium concentrations and has opposing effects of PTH. (For the purposes of this lecture, specific effects of the PTH will not be discussed.) Increased calcium in the extracellular fluid is the primary stimulus for secretion of calcitonin. It only takes a 10% increase of calcium to cause secretion of calcitonin. The immediate effect of calcitonin is to change the amount of absorbed and deposited calcium, especially in the young animal. The long term effect of calcitonin is to decrease the amount of new osteoclasts being formed.

Functions of the thyroid gland include;

- Increasing metabolic rate in almost every tissue of the body
- Increases the amount and activity of the mitochondria that will cause an increase the rate of formation of adenosine triphosphate (ATP). So in return the body uses more energy
- Affects the Na-K-ATPase which will increase transportation of sodium and potassium ions through cell membranes of tissue in the body. This causes more energy to be used and will increase the core body temperature.
- Growth- promotes growth and development of the brain during fetal life and for the first few years after birth. Growth and maturation can be decreased in the event of not enough of the hormones and vice versa if there is too much of the hormones present.
- Carbohydrate metabolism- stimulation of most aspects of carbohydrate metabolism is effected by thyroid hormone secretion. Rapid glucose uptake of the cells, glycolysis, gluconeogenesis, rate of absorption of the gastrointestinal tract and insulin secretion are all affected by the rate of carbohydrate metabolism in which the thyroid hormones play a role in.
- Fat metabolism- they thyroid gland can alter almost every step of fat metabolism. The lipids will be mobilized rapidly from the fat tissue which will decrease the amount of fat stores in the body and this will affect the free fatty acid concentration in the plasma and cause oxidation of free fatty acids to increase with an increase in thyroid hormone secretion.
- Concentrations of cholesterol, phospholipids and triglycerides will be affected with increased amounts of thyroid hormones. And they will be increased with lesser amounts of thyroid hormones in the body.
- Increased blood flow and cardiac output- because the metabolic rate is increased in the body this will cause the oxygen consumption to increase as well. This will cause vasodilation causing an increase in blood flow peripherally.
Vasodilation will occur in the skin to aid in normalization of the increased body temperature. To compensate for the increased blood flow, the body will increase the cardiac output.

- Increased heart rate- The increased heart rate is not only due to the body trying to meet the needs of increased oxygen consumption and cooling the body. The rate at which the heart is beating is increased more than to be expected. It is believed that the increased secretion of thyroid hormone has a direct effect on the excitability of the heart.
- Increased heart strength- Enzymatic activity of the increased flow of thyroid hormone will increase the strength of the heart even if the hormone secretion is only slightly increased. With excessive amounts of hormone in the body the heart will become weak due to long term increased production of protein catabolism.
- Respiratory system- The respiratory system is affected due to the increase rate of oxygen consumption and formation of carbon dioxide. The rate and depth of respiration will be increased with hyperthyroidism.
- Gastrointestinal system- GI motility and rate of secretion of digestive enzymes will be increased to help aid the body in the increased metabolism.
- Central Nervous System- altered amounts of thyroid hormone will affect the patient’s ability to think. Hyperthyroidism will cause the patient to be nervous, fidgety or find it hard to sit still. Hypothyroidism will cause the patient to become dull or even lethargic.
- Muscles- a slight increase in hormone secretion the muscles of the body with react with increased reaction time. When the hormone secretion is excessive they will react slowly because the body is in a continuous state of protein catabolism. If decreased, the body will be sluggish to react.

Feline hyperthyroidism
Feline Hyperthyroidism is the most common endocrinopathy in feline patients over the age of 8 years old. It is a multi-systemic disease resulting in increased production and secretion of the thyroid hormone, T4 and T3 within the body. Typically lateral or bilateral small thyroid masses are palpable on physical examination. The mass causing the disease typically contains an adenoma or adenomatous hyperplasia cells. It is less common for the enlarged lobe to be caused by thyroid carcinoma. There is not a sex related predisposition to the disease. It has been reported that Siamese and Himalayans are at a decreased risk for development of Hyperthyroidism. And domestic long and short hair breed are most commonly affected.

Clinical Signs include the following;
- Weight loss
- Polyphagia
- Hyperactivity
- Increased vocalization
- Hair coat changes
- Polyuria
- Polydipsia
- Vomiting
- Diarrhea
- Behavior changes
- Tachycardia

Clinical signs can be variable and consist of the non-traditional findings listed above if the disease has progressed. The clinical signs of a progressed state could include anorexia, emaciation and severe dehydration.

Upon physical exam, it is important to do a thorough thyroid palpation. Not every patient with hyperthyroidism will have a palpable thyroid in the author’s experience. In the author’s opinion, a positive enlarged thyroid lobe and a matching history for hyperthyroidism is a positive start to diagnosis of the disease. Techniques for a proper thyroid palpation will be discussed during the lecture hour. Other physical exam findings can include; poor body condition, dull hair coat, dehydration, tachycardia and hyperactivity. In her experience, the author has noted that commonly the hyperthyroid cat will be aggressive, fidgety or vocalizing during examination.

Common abnormalities on a biochemistry panel include increased alanine aminotransferase, alkaline phosphatase, aspartate aminotransferase, BUN, creatinine and hyperphosphatemia. On urinalysis the urine specific gravity is typically greater than 1.035.

Diagnosis
Diagnosis of hyperthyroidism can be made on positive palpation of an enlarged node, matching history, clinical signs and documentation of an elevated total T4 on blood work. A total T4 should be run on serum and can be sent to ay commercial laboratory for evaluation. In cases that the Total T4 levels are not conclusive other testing should be pursued to determine a definitive diagnosis.
A T3 Suppression test can be performed if the serum total T4 is indecisive. This test evaluates the responsiveness of TSH that is secreted by the pituitary gland and if the synthetic T3 that was given suppresses its secretion. When the synthetic T3 is administered it should suppress pituitary TSH secretion. This would then cause a decrease in the serum T4 concentration in a normal cat. But if the patient has hyperthyroidism it will still have secretion of the thyroid hormone that has is not related to the pituitary gland. So the administration of synthetic T3 will have no effect on the hyperthyroid patient. To perform the test, collect a baseline serum total T4 and T3, the owner will administer 25 mg of sodium iothyronine three times a day for 2 days starting the next morning. The morning of day 3 the last dose of sodium iothyronine will be administered and a final serum T4 and T3 will be collected 2-4 hours after administration of the last dose.

Lastly a radioactive thyroid scan can be done to diagnose an enlarged thyroid lobe and the presence or absence of metastatic cancer. At the author’s facility, the scan is performed by injecting 2 millicuries (mCi) of technetium intravenously. After waiting 20 minutes for the technetium to take effect, ventral, left and right lateral images of the thyroid and thoracic regions are acquired. A radiologist will read the scans and determine if I131 is an appropriate treatment for the patient.

Treatment

Treatment options for feline hyperthyroidism include; drug therapy, thyroidectomy and radioactive iodine therapy. The mode of treatment will ultimately be determined by several different factors including; the age and health status of the patient, owner wishes, renal function, cardiac function, the presence or lack of hyperplasia, adenoma or carcinoma, the allowance of the patient to receive oral medications, the availability of I131 treatment and the availability of a surgeon to perform a thyroidectomy.

Initially the patient should be treated with antithyroid drugs to help control the side effects of excessive amounts of the hormone in the body. If surgery was chosen it will help reverse the effects on the body and make that patient a better anesthetic candidate. Oral therapy will also help reverse any cardiac or renal hyperthyroid induced derangements. Renal function abnormalities can be masked in the face of hyperthyroidism so when treated with antithyroid drugs any renal abnormalities will be uncovered and will help aid in final treatment options for the patient.

Antithyroid oral drugs include methimazole, propylthiouracil and carbimazole. Methimazole is the drug of choice for daily hyperthyroidism treatment in the feline patient. It can be given orally or placed topically on the pinna of the ear. A typical starting dose of methimazole is 1.25 to 2.5 mg/cat every 12 hours. Methimazole does not block the release of thyroid hormone it blocks the oxidation of iodine once the hormone is released. It typically takes 2 to 4 weeks before T4 concentrations normalize after beginning treatment. Side effects of methimazole include; neutropenia, thrombocytopenia scabbing lesions on the pinna of the ear, hepatotoxicity, anorexia, vomiting, lethargy, renal decompensation and rarely Myasthenia Gravis. Monitoring the CBC, biochemistry panel and serum T4 levels should be done at weeks 2, 4 and 6 initially. If owners choose to give the transdermal methimazole, it is important to educate them on proper administration and the importance of wearing gloves to not allow the medication to absorb into their skin and alter their thyroid levels.

Advantages of I-131 for the treatment of feline hyperthyroidism include the following: eliminates the difficulty of administering twice a day medication, eliminates the possibility of reactions to anti-thyroid drugs and eliminates the risk of anesthesia during the thyroidectomy. Disadvantages of I-131 treatment includes; the availability of I-131 is limited, it requires knowledge and safety precautions of the radiation therapy, the patient must be hospitalized for a specific period of time to allow the I-131 to be eliminated from the body (the typical hospitalization time is 7 to 10 days), the patient has to be isolated for that period of time without owner visitations, cost, and the patient may not respond properly to a single treatment.

Patient selection for I-131 treatment is very important; the patient must be able to be isolated and unmedicated during the entire duration of hospitalization. If the patient has concurrent medical diseases such as cardiovascular, renal, gastrointestinal, other endocrine or neurological diseases they may be excluded from this particular treatment plan. Pre-radioactive iodine treatment work up should include the following; CBC, biochemistry panel, urinalysis, serum T4, thoracic radiographs, echocardiogram and have been off of methimazole for 7 days. If all requirements are met at that time the patient can have I-131 treatment.

Safety precautions should be followed during hospitalization. They would include the patient being confined to an isolated area of the hospital particularly a nuclear medicine isolation ward, trained personnel should only touch the patient, this team of people should be properly trained on radiation safety and know the proper PPE. Long laboratory coats, disposable plastic gloves and dosimeter monitors are the proper PPE for radiation patients. Every day the radiation level should be monitored and recorded in the patient’s chart to ensure the level of radiation is decreasing. In the author’s facility, daily readings are performed by trained personnel until a measurement of 2.5 millirem per hour (mr/h) is obtained. Upon discharge owners should keep the cat strictly indoors, limit the amount of contact time with the cat and dispose of the cat waste properly. Children and pregnant women should not come into contact with the patient for two weeks after discharge. Typically I-131 will restore euthyroid in a single dose. The hormone concentrations are normal within two weeks of therapy and typically the patient starts to feel better within days after treatment. However, there are approximately 5% of cats who do not respond appropriately to a single dose and must have a second dose of I-131 to become euthyroid.
Patients that are good surgical candidates are considered a low anesthetic risk, the availability of I-131 is low and the availability of funding is low. Advantages of a thyroidectomy are it is 90% efficacious and/or curative. The disadvantages of a thyroidectomy include; high initial expense, the risk of hypoparathyroidism, it is nonreversible and the anesthetic risk of the patient. Post operatively patients should be monitored for 7 days for clinical signs of hypocalcemia. Other post-operative complications include; Horner’s syndrome, laryngeal paralysis, damage to the laryngeal nerve and permanent hypothyroidism.

Iodine restricted diets are available for hyperthyroid patients. If the patient is put on this diet, it must be fed this diet exclusively, physical exams and rechecking blood work must be done every 6 months for the rest of the patient’s life and they must be taken off antithyroid drugs over a course of 2 weeks while the patient is introduced to the iodine-restricted food. Only cats that are diagnosed with hyperthyroidism can eat the iodine restricted diet.

**Euthyroid sick syndrome**

Euthyroid Sick Syndrome is diagnosed in a patient with a nonthyroidal systemic illness with concurrent decreased serum thyroid level. Severe nonthyroidal illness will decrease serum thyroid levels to the low or undetectable range even in patients without concurrent hyperthyroidism. With concurrent systemic illness, patients with serum thyroid levels in the normal to high range should be suspected to have hyperthyroidism. A second serum thyroid level should be checked approximately 1-2 weeks later. If total T4 levels are still suspicious but inconclusive other diagnostic methods for diagnosis of Hyperthyroidism should be pursued.

References are available upon request.
Teams in the Trenches: What 12-Mile Mud Runs Teach Us About Working in a Veterinary Hospital
Kimberly Pope-Robinson, DVM, CCFP
1 Life Connected Consulting
San Clemente, CA

1. Working in a veterinary hospital has a list of struggles and often individuals make sacrifices on a number of levels to be present daily in that environment.
2. The team is what makes a hospital function and with that each individual brings their own unique strength to the overall goal of living the passion of helping pets and supporting the human animal bond.
3. Mud runs are mentally and physically difficult, requiring much of the same resources to complete as it takes to work as a team through the events of a day in a veterinary hospital.
4. Becoming aware of these similarities can help for each individual to recognize the needs of the individuals in making up the greater of the team.
5. Overview of the 10 points of what mud runs teach us about working in a vet hospital
6. One- Culture. We each openly accept ourselves to get “beat up” both physically and mentally for something that is bigger than ourselves.
7. Two- Each of us has a strength that is unique to us, rarely is one person “good” at everything.
8. Three- We readily assist others in need, as we recognize that we believe in the same vision.
9. Four- When an accomplishment is made from a commitment to significant training and focus, it feels great!
10. Five- Recognizing and learning to accept our personal limitations and how these do not define our dedication to our passion, is critical.
11. Six- Sometimes you are the motivator, other times you are the one in need of motivation.
12. Seven- We are willing to make sacrifices of ourselves in support of others on the team.
13. Eight- Sometimes we split up during the process to keep the event of the day moving, but in the end we all come together to celebrate the end result.
14. Nine- Spectators readily make sacrifices to support the spectators as they recognize the accomplishment the participants are working towards.
15. Ten- Although we all have the same goal in mind, it can come from different motivations.
16. People dedicated to mud runs are just as intense and focused as individuals that help to save pets lives every day. For we are all in the trenches, yet never alone!
17. 4 key points; (1) We control our response (2) We create our environment (3) We embrace our emotions (4) We find self forgiveness.
Allergies Explained:
How to Help Your Clients Understand Why Their Pet is Itchy and How We Can Help
Anthea Schick, DVM, DACVD
Dermatology for Animals
Tempe, AZ

Why do animals itch? I like to explain to my owners that there are three main reasons that their pet might be itchy: parasite allergy, food allergy and environmental allergy.

Parasite allergy
Fleas cause itching and hair loss due to the irritation caused by their bites, secondary bacterial infections, or to a flea bite allergy (FAD or flea allergy dermatitis). Symptoms are often worse in warm weather when fleas are most numerous. Animals will often itch and lose hair on their back near their tail. Cats may also develop small crusts on their skin that are similar in appearance to tiny millet seeds (miliary dermatitis). In flea-allergic animals, even one fleabite can cause a reaction.

Mite hypersensitivity can also cause itching. There are two common types of mange (microscopic skin mites), demodex and scabies/sarcoptes. Another, less common mite that can cause itchy skin disease is called Cheyletiella. Scabies infection is extremely itchy and contagious to other dogs, and may cause hair loss and a crust to form on their ears and elbows. Scabies mites can be very difficult to find, and often we will trial-treat for scabies based on the dog’s symptoms and appearance, even if we cannot find mites on the skin scrapes. With scabies, all dogs in the household must be treated at the same time, even if they are not showing signs yet, because some dogs can carry the mites and have no symptoms. Dogs with scabies may also have secondary bacterial or yeast skin infections, which contribute to the itch. Cheyletiella mites cause itchy skin and dry scaling on the back, and can infect dogs, cats, people, and rabbits. They can also be difficult to find on skin scrapings, and so trial treatment for Cheyletiella is warranted if symptoms are consistent with infection. Treatment options are the same as for scabies mites, and all animals in the household have to be treated at the same time, or they will pass the mites back and forth.

Allergies
Environmental allergies or canine atopic dermatitis (CAD) is one of the most common diagnoses in general veterinary practice, is a progressive condition that decreases the quality of life in 10% of companion dogs worldwide. CAD's most common sign is pruritus, most often affecting the ears, face, ventral neck, distal limbs, and ventrum, as well as the perianal and perivulvar regions. Secondary bacterial and yeast infections are also common. CAD may begin seasonally and progress to nonseasonal pruritus. CAD is thought to be a polygenetic disorder involving immune dysregulation and epidermal barrier dysfunction. The atopic immune response causes increased production of allergen-specific IgE, while barrier dysfunction facilitates transepidermal allergen and microbe penetration. CAD is diagnosed via exclusion of other causes of pruritus; it cannot be accurately diagnosed by allergy testing alone. Once CAD has been diagnosed (by ruling out parasite and food allergy), skin allergy testing can be helpful. Skin allergy testing tests the actual organ that is involved in the allergy (the skin), so is more accurate than blood testing, and is typically performed by veterinary dermatologists. Once the allergy test results are known, allergic pets can receive allergy shots (just like people!) to desensitize them to the pollen. Although not a cure or a quick-fix, allergy hyposensitization injections help 70-75% of allergic pets to decrease symptoms and need for other medications, and address the cause of the allergies, not just the symptoms.

Food allergy (cutaneous adverse food reaction)
Animals with food allergy can show very similar signs as pets with environmental allergies, but the itching is not seasonal and animals can develop a food allergy at any time in their life—even if they have been eating the same food all along. Cats may develop crusty dermatitis or hair loss similar to pollen allergy. Besides itchiness, food-allergic dogs may also have ear or skin infections. The symptoms of food allergy usually do not improve much with anti-itch medications, and the diagnosis and treatment is to feed the pet a hypoallergenic diet using a protein source that they have never been exposed to before. Switching to another commercial diet usually does not help, because most of these diets have similar ingredients. A better alternative is a hypoallergenic diet with single unique protein ingredients such as fish, rabbit, duck, or venison, with a single carbohydrate such as potato or rice, and no other treats, table scraps, rawhides, milkbones, chewable supplements or other foods for at least 6-8 weeks. Some food allergic dogs require home cooked hypoallergenic diets. Blood or skin testing or hair and saliva for food allergy is unfortunately not accurate in dogs and cats because of the high number of false positive and false negatives. If present, secondary bacterial or yeast infections also need to be treated. If the itchy symptoms have resolved in 6-8 weeks, new food allergens can be added one at a time every 2-3 weeks (ie. beef, chicken, lamb, wheat, corn, egg, milk etc.) to determine what the pet is allergic to and what other foods they may tolerate.
How to communicate with your clients about allergies—which require LONG TERM treatment

For the initial allergy work up, we need to explain to the client the long and often frustrating process of first trying to exclude any other causes of the pet’s signs (such as food allergy or parasites). This may include treatment trials of the pet and its environment for parasites, various diagnostic steps, and a hypoallergenic food trial.

Once CAD has been diagnosed, we need to make sure the client has appropriate and realistic expectations. The first and most important point to make with your client is that atopy is managed, and NOT CURED. Your main goal is to keep the pet as comfortable as possible during flare-ups.

Treatment of atopic pets includes developing an individual treatment plan. Clients need to be taught to recognize signs of flare-ups early so that they can be managed before becoming too frequent or intense or before their pet develops a secondary infection.

Since treatment of allergies usually involves multiple modalities (oral medications, shots, and topicals), which can get expensive. There needs to be a conversation with the client about what is affordable for them and what they are physically able to do. For example, a senior may not be able to bathe their 150 pound German shepherd every other day.

Communicating with clients who have allergic pets is often like being a cheer-leader. Trial and error therapy is tedious, can be expensive, and is often frustrating. There will often be failures or break-through flare ups with atopic patients. Clients need to understand that this will happen and that they can come to you when they experience problems or frustrations. Sometimes, all they really want is an ear and some reassurance. By facing up to this reality, you will better help your client through difficult times and better ensure they will continue trying to manage their pet’s atopy.

Longer appointment times might be necessary to teach owners about managing their allergic pets and reviewing often complicated treatment plans. Extra time with clients is the single most important diagnostic advantage veterinary dermatologists have over primary-care doctors, whose new clients often say that they wished their veterinarian had spent more time explaining what was wrong with their pet, so use this advantage. It is important for the veterinarian to explain the need for longer appointment times for several reasons: clients know well in advance that they should allow extra time in their schedules; reception team members know to reserve the extra time, so the appointment schedule is not affected; and appointments can be avoided during the practice’s busy times, such as the middle of Saturday mornings, which makes the visit less stressful for everyone.
Bullying has become more prevalent in today’s society, not only among school children, but within the workplace as well. Younger generations have been moving away from face-to-face communication with the overuse of social media and texting, so their ability to have direct communication tends to be less constructive. When faced with these situations, it may be harder to know how to communicate that this is unacceptable and stand up for themselves.

The repercussions can range from a lowered morale to intense emotional damage to the victim. Victims of bullying often experience feelings of depression and anxiety, and feel isolated. Sleep and eating patterns can be disrupted, and psychosomatic symptoms such as headaches, abdominal pain, and chest pain can arise. These individuals begin to dread coming into work, which results in increased absenteeism and decreased productivity. The employee is made to feel that nothing they bring to the table will be appreciated or recognized, so they stop trying. Highly talented, hardworking employees can appear to become complacent in the face of bullying behavior.

In more and more states, it is also becoming illegal to behave this way in the workplace. Currently, in 30 states, the Healthy Workplace Bill has been introduced. This bill defines what is considered an abusive work environment, and gives employers a more direct reason to terminate offenders. It also allows employees a path for legal compensation for health harming harshness at work. This bill holds the employer accountable for setting up internal correction and prevention procedures, and seeks to recover lost wages and benefits for the employee. Beyond protecting your employees because it’s the right thing to do, with this bill, it’s important to protect your practice by ensuring proper preventative procedures are put into place. This starts with an anti-bullying policy, which communicates to all employees that bullying behavior will not be tolerated. It is clearly defined and examples are provided. There is a focus on the effects felt by the disrespected individual, not on the intent of the offender. Regardless of intentions, perceived incivility will not be tolerated and the offender will undergo a corrective process.

In the workplace, individuals should be assessing their mental health and wellbeing on a regular basis, and be able to label bullying behavior when it’s occurring. When faced with bully-like behavior, individuals are often constantly criticized. It doesn’t matter what the employee does to try to improve or fix their work, instead of coaching or positive reinforcement into better performance, bullies will hold the individual to a higher, unfair standard and continue to criticize. While it sounds completely unprofessional to even fathom, another tell-tale sign of workplace bullying is using raised voices or even outright yelling to express concerns. Often times this is done while insulting or humiliating the individual in front of their coworkers.

Combating bully-like behavior starts with identifying it and eliminating any self-blame. Utilize mental health professionals for additional healing support if needed. Finding emotional stability is very important prior to making a decision as to how you will combat the problem at work. While in most circumstances speaking directly to the individual is recommended, with bullies this tends to be less effective. Research your company’s employee handbook for any internal policies on harassment, respect or specific bullying behavior. Speak to your manager and/or the owner of the practice regarding the issue, backing up your case with specific examples, and include reference to any relevant company policies. Quality employers will not stand for this behavior and will squash the problem quickly. However, if you find that your employer does not take action and/or the retaliation from the bully is ignored, it may be time to plan an escape route into new employment.

It’s important to keep an eye on your own mental wellbeing in any job, especially if you feel you are the target of bullying behavior. The stress can have negative effects not only on your mental health, but physical as well. Encourage your employer to create an anti-bullying policy before it personally affects you. Having new hires review and understand that this behavior is unacceptable will help build positive morale and eliminate bullying behavior before it starts. Not only will the employees be happier and more productive, the employer will not waste excessive time and financial assets in dealing with high turnover and corrective processes.
Employees under high stress equal lower productivity and increased turnover. Our field naturally produces high levels of stress at work, between sick patients, client demands, and coping with euthanasia. The expectation of our team members and doctors is to be able to go into a room to discuss end of life decisions, then be able to hop into a new puppy room with a smiling face an hour later. It can be emotionally draining and it wears on everyone. It’s important to be proactive as an employer and ensure team members are taking care of themselves. When a supportive culture is created, you will keep valued employees for much longer, and deal with much less absenteeism. You may also find that employees become devoted ambassadors of your company and brand in the community.

Prioritize health and wellbeing of the employees by implementing regular programs that promote a healthy lifestyle. Offer twice monthly activities for the employees to join in on, such as yoga or kickboxing. Not only will it enrich their health lives, it will also serve as a team building experience. Other concepts include wellness challenges such as trying to drink a certain amount of water each day, or consuming a certain serving amount of veggies and fruits. Wellness challenges are also often centered around performing a certain amount of exercise on a regular basis. The employer can establish a point system for increments of healthy choices, with a small prize at the end. Other programs include offering full health assessments, along with coaching and support to create individualized plans for your employees. Nutrition and exercise wellness lectures can be offered as part of your team meetings, or on a semi-annual basis.

Managers also should take some responsibility for their employees’ wellbeing and stress levels. Regularly check in with staff one on one to see how their job is going, and where there could be improvement not only in performance, but their level of contentment as well. There may be times that certain assigned tasks do not lend themselves to an individual’s work style or strengths in the workplace, so reassignment to an alternative task may make them feel more fulfilled and less overwhelmed. When employees see that their fulfillment is a priority of the organization, they are more likely to produce their highest quality work. While not always possible to immediately fulfill, the opportunity for growth should be available to employees so that they can envision a plan for their future and feel more invested in their position.

Fulfillment in an employee’s position at work is also correlated to the extent of staff development offered by the organization. Offer regular training and educational opportunities, whether it be clinical CE, or leadership CE for those interested in pursuing management opportunities one day (even if it is unlikely there will be an opening with your company specifically- if you invest in them, they will be more invested in you in the present). Outside of company created CE, it’s advisable to allow for a CE budget for all staff- assistants, client service representatives, and technicians. Not only does this help them grow and better themselves in their positions, it empowers them and makes them feel valued by their employer.

Ensure that the new employees you are trying to win over and engage are not left in the dark. For new hires, be sure to include phase training programs, and regular training check-ins that include seeking their feedback on how to improve the process. Offering cross training opportunities will not only help employees better understand all sides of the company’s operations, but will also help match up their personal strengths and goals with their job duties.

Those in leadership roles should set a positive example for the staff. When their supervisors value a positive work/life balance, and don’t stay late every night, and choose not to order fast food for lunch and skip out on exercising, the staff is more likely to prioritize their health as well. Creating a culture that checks in on each other and genuinely cares, on a personal level, how their coworkers are functioning will promote regular self-care in the workplace.
Most staff members are terrified of the concept of direct communication. There are so many “what if”s that take over an individual’s psyche. What if the person on the receiving end gets angry? What if they retaliate against you? Or even what if it ends up being a wasted discussion? While these are all possibilities, and potential challenges, the benefits far outweigh the pitfalls.

Team members who are willing to tackle direct conversations are more likely to see desired outcomes come to fruition. It is much easier to communicate a concern, and back it up with examples, when it’s coming directly from the person who was effected by the behavior. While a discussion led by a manager on a team member’s behalf may stall with excuses and retorts, this is less likely to occur when the team member approaches their coworker on their own. An employee will not push back and argue whether or not something actually occurred with the individual who was also present during the incident.

Moreover, even if the information received is hard to hear, employees feel respected when their coworkers come to them directly to solve problems versus going above their head to management. The team member feels that their coworkers are giving them a chance to fix the problem on their own before “getting in trouble” with the higher-ups. The individual leading the conversation also gains respect within their workplace as someone who is clear about where they stand in situations.

On the opposite end, the repercussions of triangulated conversations are dangerous to the success of a business. Often times, the message is distorted or misinterpreted by the third party relaying the information.

Implementation of a direct communicative culture starts with management and owners. New hires should be encouraged to speak directly to their coworkers (and management as well) when they have ideas or concerns. Managers must achieve a balance of ensuring the staff knows when they are stuck in a communication barrier with an employee, they can come to you for assistance, but the first step is never to immediately go above their coworker’s head.

When team members need help further resolving the problems they have between each other, management should help facilitate direct face-to-face communication with a supervisory employee as the moderator. Ensure both team members are equally heard and no sides are taken by managerial staff.

A culture of direct communication can also be achieved in part by conducting regular surveys on various issues that arise within the workplace. Whether it be opinions on protocols, training methods, or assessing morale, employees should feel comfortable voicing their opinion, and have a platform to do it. Equally important, the data obtained from employee surveys should be transparently utilized by the organization and not just collected, then ignored.

Management should also promote an open door policy with team members so that they are encouraged to directly communicate, even when they have a concern with management. There should never be fear of retribution, or losing their job, if a team member brings a concern, or even complaint, to management. Some methods to avoid team members shying away from these conversations include offering regular 360 reviews that allow staff to evaluate and comment on management’s performance in an anonymous manner. Having more than one “boss”, but rather a few individuals on the management team also allows employees to feel that there isn’t just one person in control of all elevated decisions. Therefore, if an employee has a concern with one of the supervisors, they understand it’s less likely if they bring up these issues that they will be treated differently or even terminated.

Unfortunately, in many veterinary practices, since the teams tend to be small, gossip and pent up hostility tend to take the place of honest conversations. When team members observe management or owners exhibiting this behavior, it makes it all the more difficult to turn a new page and switch to direct communication. Often in these environments, the “problem employee” is ganged up on, gossiped about, and alienated until they decide to move on. Not only does this approach create toxicity and lower productivity in the workplace, but it also does not even give the employee a chance to improve. We must remember that we go through a time-consuming, expensive process to select, hire and train new staff, so we should protect that investment. Not only is it the right thing to do by the employee, but also the smarter business move. If we can have a few more conversations and a little more coaching to get the employee back on track, it will increase morale for staff to see the investing nature of their employer, as well as save on rehiring processes.