The term "acute abdomen" is used to describe the clinical syndrome characterized by the sudden onset of severe abdominal pain and associated signs, which include: shock, abdominal pain, vomiting, diarrhea, and anorexia.

Immediate evaluation, diagnosis, and therapy is essential to reduce morbidity and mortality in these potentially life threatening situations. While these cases can be incredibly rewarding, they can also be frustrating for even the most experienced practitioner.

Immediate patient evaluation
The ultimate goal in diagnosis and therapy of the acute abdomen patient is to determine if the diagnosis lends itself to medical therapy or if surgical intervention is warranted. On presentation, a triage history should be taken while a rapid triage examination / evaluation is occurring. Major body systems for the triage examination include the respiratory system (e.g., airway, breathing), cardiovascular system (e.g., circulation), and neurologic system (e.g., dysfunction). Failure to recognize an abnormality in any of these systems can result in immediate life threatening deterioration of the patient.

Important triage information includes:
- Signalment
  - Age: younger patients may have a different differential list (e.g., trauma, poisoning) in comparison with older patients (e.g., neoplasia, metabolic disease)
  - Sex: Intact dogs (e.g., pyometra, prostatic abscess) may have a differential list different to that of spayed or neutered patients.
  - Breed: Breed variations may help guide the examination and diagnostics (Standard poodle and GDV or Hypoadrenocorticism vs. Dachshund and IVDD)
- Presenting complaint
- Time of onset
- Progression since initial onset

With this information, I try to place them into one of three categories:
- Nonsurgical (medical)
- Emergent
- Critical.

Nonsurgical patients
Examples of nonsurgical (medical) patients include those diagnosed with gastritis, gastroenteritis, and acute pancreatitis. These patients are commonly treated with intravenous fluid therapy, gastroprotectants, and analgesia.

Emergent patients
Examples of emergent patients include those diagnosed with a cardiovascullarly stable hemoabdomen, uroabdomen with the placement of a temporary urinary or dialysis catheter, and intestinal obstruction. These patients are surgical candidates, but tolerate medical therapy for stabilization prior to anesthesia and surgery.

Critical patients
Critical patients, as compared to the previous two groups, require immediate emergency abdominal surgery. Examples of critical patients include gastric dilation and volvulus, mesenteric torsion, septic peritonitis, and uncontrolled hemorrhage. While they may require some degree of medical therapy prior to anesthesia and surgical evaluation, rapid assessment and treatment is needed and delay in surgical correction will increase morbidity and mortality.

Once the initial assessment is completed and life-threatening abnormalities addressed, a more thorough history can be obtained and a more thorough physical examination can be performed.

With gastrointestinal distress and acute abdominal pain this includes:
- Medication history (both prescription and over the counter)
- Access to foreign material inside or outside
  - Garbage
  - Toys (children’s toys or dog toys)
  - Abnormal / new food
  - Trauma
  - Recent abdominal surgery

If vomiting is part of the history, this should be differentiated from regurgitation, coughing or retching. If diarrhea is part of the history, this should be characterized based on color, frequency, consistency, and supplemented with a rectal examination.
Following the triage examination, a thorough physical examination should be performed:

- **EENT (eyes, ears, nose, throat)**
  - Ulceration
  - Halitosis
  - String foreign material under the tongue
  - Dental disease
  - Ocular disease
  - Otic disease

- **CV (cardiovascular)**
  - Heart rate / rhythm
  - Pulse quality
  - RESP (respiratory):
    - Respiratory rate / effort
    - Bronchovesicular sounds

- **ABDOMEN:**
  - Organomegaly
  - Ascites
  - Gastric dilatation-volvulus
  - Neoplasia
  - Focal source of abdominal pain

- **MUSCULOSKELETAL / NEUROLOGICAL**
  - Joint swelling
  - Muscle atrophy
  - Fractures
  - Referred spinal pain.

- **UROGENITAL**
  - Pregnancy
  - Mammary masses
  - Vaginal discharge / preputial discharge

- **INTEGUMENT**
  - Ecchymoses / bruising
  - Hemorrhage in the area of the umbilicus (may indicate hemoabdomen – “Cullen Sign”).

- **LYMPH NODES**
  - Lymph node enlargement or discomfort

- **RECTAL**
  - Melena
  - Hematochezia
  - Sublumbar lymphadenopathy
  - Prostate pain or enlargement

The seasoned clinician has mastered the art of multi-tasking, and while taking the history and performing the examination, is often barking out orders to the support staff! To assess the patient, you may ask for:

- Placement of peripheral IV catheter(s)
- Intermittent or continuous ECG for cardiac monitoring
- Pulse oximetry
- Blood pressure
- PCV/TP/BG/Lactate/Electrolytes/Azo

Based on your examination and initial diagnostic results, primary treatments may include:

- Intravenous Fluid therapy to correct hypovolemia and improve perfusion.
  - Administration of balanced isotonic crystalloids (10-30ml/kg) in incremental boluses
  - Administration of synthetic colloids (hydroxyethyl starch 3-5 ml/kg) in incremental boluses

- Supplemental oxygen - if there is labored breathing or abnormal perfusion

- Analgesic therapy
  - Opioid therapy is most commonly used (SEE OPIOD CHART).
Nonsteroidal anti-inflammatory medication should be used with caution until the underlying cause has been established. Hypoperfusion, gastrointestinal compromise, and the potential need for anesthesia and surgery limit the usefulness of NSAIDS in this situation.

Secondary survey
Secondary survey and additional diagnostics may include:

- Complete blood count
  - Evaluation of white blood cell count, red blood cell count, and platelet count
- Serum biochemical profile
  - Evaluation of important organ values, blood glucose, and electrolytes
- CPL, Amylase, Lipase
  - Initial assessment if there is a concern for pancreatitis
- Coagulation profile
  - PT, PTT, Platelet count
- Urinalysis and urine sediment
  - Evaluation of the urine specific gravity, presence of bacteria, or other abnormalities.
- Fecal examination (fecal float, fecal cytology)
- Diagnostic imaging
  - Radiography
    - Presence of foreign material or an intestinal pattern consistent with obstruction
      - Small intestinal plication
      - Small intestinal dilation
    - Distention of bowel up to 1.6 times the height of the body of L5 is reported to be normal in dogs
    - Gastric Dilatation and volvulus
    - Peritonitis
      - Loss of abdominal detail due to increased peritoneal fluid
  - Abdominal ultrasound
    - Peritoneal effusion
    - Specific organ enlargement
    - Pyometra
    - Urinary tract obstruction
    - Gastrointestinal obstruction
    - Pancreatitis
- Abdominocentesis
  - Ultrasound guided or 4-quadrant technique
- DPL
  - When there is a small volume effusion or ultrasound is not available, a diagnostic peritoneal lavage (DPL) may be necessary to collect samples for evaluation.
- Cytology of the effusion
  - Presence of degenerate neutrophils
  - Intracellular bacteria
  - Food material
  - Measurement of lactate and glucose (compared to plasma in evaluation of sepsis)
  - Measurement of creatinine and potassium (compared to plasma in evaluation of urinary tract rupture)
  - Measurement of bilirubin (compared to plasma in evaluation of biliary tract rupture)

Indications for emergency surgical intervention include

1. Inability to medically stabilize intrabdominal hemorrhage
2. Free gas on abdominal radiographs (provided they were taken prior to abdominocentesis and there is no recent abdominal surgery)
3. Gastric dilatation-volvulus
4. Cytological evidence of intracellular bacteria or plant/food material in the abdominal fluid
5. Elevated creatinine and potassium levels compared to peripheral serum levels
6. Elevated bilirubin levels higher compared to peripheral serum levels
7. Complete bowel obstruction
8. Penetrating abdominal injury
9. Splenic torsion
10. Mesenteric volvulus

**How soon should surgery be performed once the diagnosis is made?**

This depends on two factors:
1. How stable is the patient?
2. What is and the underlying diagnosis.

Most patients presenting with an acute abdomen will require some degree of stabilization prior to anesthesia and surgery. Hypovolemia and acid-base or electrolyte abnormalities are common findings in patients that present with acute abdominal pain and gastrointestinal distress. These should be addressed prior to induction of anesthesia. Clinical judgment is needed to determine the appropriate balance between stabilization before surgery and the time that passes before the patient is placed under anesthesia to surgically correct the problem.

**Along with fluid therapy, electrolyte correction, and potential surgical correction, other therapies consider include: Antibiotics**

Translocation of gram positive and gram-negative aerobes and anaerobes may occur following a period of poor perfusion and alteration to the integrity of the gastrointestinal tract. **Common antibiotic broad spectrum combinations the author uses includes**

**a)** Cefazolin 20-30 mg/kg IV q8-12h or Ampicillin 22 mg/kg IV q8hr
   Metronidazole 10 mg/kg IV q12 hr
   Baytril 10-15 mg/kg IV q24h

**b)** Ampicillin/sulbactam 20-30 mg/kg IV q8h
   Baytril 10-15 mg/kg IV q24h
   Metronidazole 10 mg/kg IV q12 hr

**c)** Clindamycin 8-11 mg/kg IV q8-12h
   Cefotaxime 30-50 mg/kg IV q6h

**For persistent gastrointestinal upset, gastroprotectants are commonly used (SEE CHART)**

Ultimately, the prognosis for patients with acute abdomen will depend on the underlying disease process. Many diseases are treatable with fluid resuscitation, pain control, and exploratory laparotomy. Rapid evaluation and treatment of life threatening complications such as hypovolemic shock will decrease morbidity and give the astute clinician time to obtain the diagnosis to best assist the pet parents in helping their pet.

**Gastrointestinal protectants and associated medications**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Drug Class</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine</td>
<td>α2 and D2 Antagonist</td>
<td>0.1–0.5 mg/kg</td>
<td>Q8</td>
<td>SQ, IM, Rectal Suppository</td>
</tr>
<tr>
<td>Dolasetron</td>
<td>5-HT3 Antagonist</td>
<td>0.5–1.0 mg/kg</td>
<td>Q12-24h</td>
<td>IV, SQ</td>
</tr>
<tr>
<td>Maropitant</td>
<td>NK-1 Antagonists</td>
<td>1 mg/kg</td>
<td>Q24</td>
<td>SQ</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 mg/kg</td>
<td>Q24</td>
<td>PO</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>D2 Antagonist</td>
<td>0.2–1 mg/kg</td>
<td>Q6</td>
<td>PO, SQ, IM</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1–2 mg/kg/day</td>
<td>CRI</td>
<td>IV CRI</td>
</tr>
<tr>
<td>Ondansetron</td>
<td>5-HT3 Antagonist</td>
<td>0.1–0.3 mg/kg</td>
<td>Q6-24h</td>
<td>PO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>IV</td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>α2 and D2 Antagonist</td>
<td>0.1–0.5 mg/kg</td>
<td>Q8</td>
<td>SQ, IM, Rectal Suppository</td>
</tr>
</tbody>
</table>

**Opioid drug chart**

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buprenorphine</td>
<td>0.01-0.02 mg/kg</td>
<td>Q4-6h</td>
<td>SQ, IM, IV</td>
</tr>
<tr>
<td></td>
<td>Dose Description</td>
<td>Route</td>
<td>Frequency</td>
</tr>
<tr>
<td>------------------------</td>
<td>--------------------------------------------------------</td>
<td>-------</td>
<td>-----------</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.05–0.2 mg/kg</td>
<td></td>
<td>Q4-6h</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>25 mcg patch &lt; 10 kg</td>
<td></td>
<td>Q3–4 days</td>
</tr>
<tr>
<td></td>
<td>50 mcg patch 10–20 kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>75 mcg patch 20–30 kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>100 mcg patch &gt;30 kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fentanyl</td>
<td>3–10 mcg/kg/hr</td>
<td></td>
<td>IV CRI</td>
</tr>
<tr>
<td></td>
<td>(2–5 mcg/kg initial IV bolus)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>0.5–2.0 mg/kg</td>
<td></td>
<td>Q4-6h</td>
</tr>
<tr>
<td>Methadone</td>
<td>0.1–0.4 mg/kg SC, IM, IV</td>
<td></td>
<td>Q6h</td>
</tr>
</tbody>
</table>

Table 1. Differential diagnosis list for acute abdominal pain.

- **GI tract lesions**
  - Gastric or intestinal ulceration
  - Gastric or intestinal perforation
  - Gastric or intestinal tumors
  - Gastric or intestinal obstruction
  - Pyloric outflow obstruction
  - Gastric dilatation-volvulus
  - Intestinal torsion
  - Intussusception
  - Mesenteric thrombosis, torsion

- **Lymph node neoplasia or infection**

- **Pancreatic disease**
  - Pancreatitis
  - Pancreatic abscess
  - Infarction of the pancreas

- **Reproductive tract disease**
  - Pyometra
  - Uterine torsion
  - Uterine rupture
  - Testicular torsion
  - Testicular abscess

- **Reticuloendothelial system**
  - Splenic disease
    - Tumor
    - Torsion
    - Fracture
    - Thrombosis
  - Liver disease
    - Abscess / infection
    - Tumor
    - Torsion
    - Biliary obstruction
    - Biliary rupture

- **Urinary tract**
  - Renal disease
    - Calculi
    - Infarct
    - Pyelonephritis
    - Avulsion
  - Urethral / Ureteral Disease
• Obstruction
• Rupture
• Passage of calculi
  o Urinary bladder disease
    • Obstruction
    • Cystitis
    • Rupture
  o Prostatic disease
    • Prostatitis
    • Prostatic neoplasia
• Peritoneal cavity disease
  o Hemoperitoneum
  o Pneumoperitoneum
  o Bile peritonitis
  o Septic peritonitis
• External gastrointestinal / abdominal structures
  o Myositis
  o Steatitis
  o Fasciitis
  o Intervertebral disc disease
  o Herniation (umbilical, inguinal, abdominal wall)

FAST Scan – Introduction
FAST is an abbreviation for a procedure known as Focused Assessment with Sonography for Trauma. This is a tool, a diagnostic, that was initially used in human medicine to identify trauma patients with abdominal hemorrhage. The FAST has become a common diagnostic tool in the emergency room to scan for free fluid within any body cavity (abdomen, retroperitoneal, pleural, pericardial space).

In the past few years, the abdominal FAST protocol has been validated in dogs, reporting a good sensitivity and specificity to detect peritoneal effusion.

FAST Scan - Indications
The A-FAST scan is a non-invasive, rapid screening tool for abdominal effusion, indicated in patients presenting with trauma, acute abdomen, and other signs of abdominal disease or discomfort.

FAST Scan – Benefits
• As compared to radiographs, ultrasonography is more sensitive for detecting effusion.
• FAST scans are non invasive, safe, rapid, and do not require the use of radiation to the patient or veterinary personnel.
• A FAST scan can be performed during further patient assessment, catheter placement, and treatment/stabilization.

FAST scan - Technique
• The patient can be placed in lateral recumbency (right lateral preferred), although if the patient is in distress, they may remain standing or sit in sternal recumbency to facilitate the procedure.
• The abdomen is evaluated at four distinct sites
  o Just caudal to the xiphoid process (diaphragmatic-hepatic view)
  o The midline over the urinary bladder (cystocolic view)
  o Over the left and right flank regions (spleenorenal and hepatorenal views)
  o Each site is scanned in two planes at a 90-degree angle to each other.
• Serial exams using the FAST protocol may be needed, especially if hypovolemic or in shock to diagnose the presence of effusion
• Serial exams may also be used to monitor the amount of fluid present (i.e. is there ongoing accumulation of fluid)

While a FAST scan is not a complete, thorough ultrasound and it does not replace the utility of diagnostic radiographs, it is an important diagnostic tool for the small animal practioner and one that the clinician should be comfortable performing.

References
Patients with coagulopathies may present with a variety of clinical signs based on not only the underlying disease process, but also the type of coagulopathy. Hemostasis is commonly divided into primary hemostasis and secondary hemostasis. Primary hemostasis refers to the formation of the platelet plug whereas secondary hemostasis refers to formation of the stable fibrin clot. The third component of hemostasis is fibrinolysis, commonly referring to breakdown of the clot.

**Primary hemostasis**

Primary hemostasis is the initial formation of the platelet plug, involving platelets, the vascular endothelial cells, von Willebrand's factor, red blood cells, and white blood cells.

In health, the intact vascular endothelium inhibits platelet adhesion. When the endothelial wall is damaged, neurogenic mediators are produced, promoting platelet aggregation, platelet adhesion, and vasoconstriction. The binding of platelets at the site of injury is mediated by Von Willebrand's Factor (vWF). This aggregation of platelets forms the "platelet plug" which is responsible for initial cessation of bleeding.

**Useful diagnostic tests to detect primary hemostatic disorders**

1. Platelet estimate (blood smear) and count (automated or manual):
   a. 1 platelet per oil immersion 100x = 12,000-15,000 platelets
   b. If the platelets are clumped, an accurate count cannot be obtained.
   c. The presence of large platelets oftentimes indicates a regenerative process.

2. Platelet machine count (CBC machine or hemocytometer)
   a. Many machines can accurately count dog platelets
   b. Cat platelets are smaller and commonly clump, and therefore it is more difficult for a machine to produce an accurate count

3. Buccal mucosal bleeding time (BMBT)
   a. Performed in patients with a platelet count greater than 50,000-100,000/µl
   b. Thrombocytopenia can cause a prolonged bleeding time.
   c. If prolonged with a normal platelet count then the concern is a platelet dysfunction or a thrombocytopathia.

4. Bone marrow evaluation
   a. Can assess megakaryocyte numbers.
   b. Normal patients should have 3-5 megakaryocytes per HPF

5. Von Willebrand factor assay
   a. Measures and reports the amount of functional von Willebrand factor.

**Secondary hemostasis**

Secondary hemostasis results in the formation of fibrin through a series of enzymatic reactions involving coagulation factors, cofactors, calcium, and phospholipid membranes.

The coagulation cascade is traditionally viewed as the intrinsic, extrinsic, and common pathways, however more recent evaluation of the coagulation process appears less like a cascade and more likely a combination of functions and interdependencies of these pathways.

**Useful diagnostic test to detect secondary hemostatic disorders**

1. Intrinsic and Common Pathways
   a. Activated Clotting Time (ACT)
   b. Activated Partial Thromboplastin Time (aPTT)

2. Extrinsic and Common Pathways
   a. Prothrombin Time (PT)
   b. Protein Induced by Vitamin K Antagonism or Absence (PIVKA)

**Disorders of primary hemostasis**

Disorders of primary hemostasis include thrombocytopenia (decreased platelet count) and thrombocytopathia (decreased platelet function).

Clinical signs of a primary hemostatic disorder typically result in surface bleeding, notably petechia, ecchymoses and mucosal surface bleeding (e.g. gingival bleeding, gastrointestinal surfaces, and urogenital surfaces).
Thrombocytopenia
Thrombocytopenia is the most common primary hemostatic disorder, resulting from platelet destruction, decreased production, consumption or sequestration. Patients with thrombocytopenia (<50,000 μl) are at risk for spontaneous hemorrhage and must be treated cautiously.

The most common cause for thrombocytopenia in clinical practice is platelet destruction, specifically immune-mediated thrombocytopenia (ITP). ITP may be a primary, idiopathic, immune mediated disorder or as a secondary disease process. Secondary ITP may result from drug therapy, infectious diseases, neoplastic causes, or inflammatory conditions. Examples of infectious diseases include tick borne disease (such as *Ehrlichia*). Neoplasias associated with thrombocytopenia include splenic and liver neoplasia, specifically lymphoma.

Initial diagnostics to document thrombocytopenia include blood smear evaluation and complete blood count (CBC) evaluation. Additional diagnostics performed in an attempt to differentiate primary vs. secondary thrombocytopenia include infectious disease serology, imaging (thoracic and abdominal radiographs as well as abdominal ultrasound) and bone marrow aspirate cytology. Although not a diagnosis which can be made with testing, other causes for ITP exist including drug reaction and reaction to vaccination. While any drug may cause a reaction, medications associated with ITP include sulfa antibiotics, beta-lactam antibiotics and chlorambucil. Ultimately, idiopathic, primary ITP is a diagnosis of exclusion.

Treatment of immune mediated thrombocytopenia will vary based on the underlying cause. Tick borne infections are commonly treated with doxycycline. If common causes are ruled out and primary (idiopathic) ITP is suspected, immunosuppressive therapy is instituted, notably prednisone, azathioprine, and/or cyclosporine. The goal of these medications is to suppress the immune system and stop immune destruction of the platelets.

For severe thrombocytopenia with the risk of hemorrhage, vincristine can also be considered. While typically used as a chemotherapeutic agent, vincristine causes early release of platelets from the bone marrow. With that said, there is debate on the functionality of these platelets once released into circulation.

Thrombocytopathy
Thrombocytopathy, a platelet dysfunction, is an uncommon clinical problem. Patients that have a thrombocytopathia will often have a normal platelet numbers, but have clinical signs of illness (i.e. surface bleeding) similar to patients with a true thrombocytopenia.

Although uncommon, thrombocytopathia may be congenital, related to medications, or acquired subsequent to other diseases. Drug induced thrombocytopathia may result from medications such as aspirin, NSAIDS, or colloidal fluid therapy (i.e. Dextran) as synthetic colloids such as dextran are thought to adhere to the surface of the platelet resulting in decreased platelet aggregation.

The most common inherited platelet function disorder is von Willebrand's disease. With this disease, there is decreased platelet binding resulting in hemorrhage commonly seen following trauma, venipuncture, and/or surgery. In severe deficiencies, spontaneous hemorrhage may also be seen.

There are three types of VonWillebrand's Disease
1. Type one can be seen in any dog, but the most common breed is the Doberman pinscher. These patients have a low amount of vWF but the protein itself has a normal structure.
2. The second type is most commonly seen in German short-haired and wired hair pointers. This type of vWF disease has both a low amount of vWF and an abnormal protein structure.
3. Type three is the most concerning form of the disease, reported in Scottish Terriers, Chesapeake Bay Retrievers and Shetland Sheep dogs. In this type, the amount of circulating vWF is very low or completely absent resulting in the most significant risk of hemorrhage.

Other hereditary thrombocytopathic disorders are rare, but have been reported in the otter hound, basset hound, and both domestic and Persian cats (Chediak-higashi).

Disorders of secondary hemostasis
Disorders of secondary hemostasis include abnormalities within the coagulation cascade. As compared to abnormalities of primary hemostasis that results in surface bleeding, patients with disorders of secondary hemostasis present with signs consistent with cavity bleeding, including the abdominal cavity, pleural space, subcutaneous or intramuscular hematomas, and mucocutaneous bleeding.

The most common tests to evaluate disorders of secondary hemostasis include prothrombin time (PT) and activated partial thromboplastin time (PTT) as these tests are not affected by platelet number or function. The PT evaluates the common (factors II, V, and X) and extrinsic (VII) cascade while the PTT evaluates the common and intrinsic (VIII, IX, XI, and XII) pathways.

Acquired disorders of secondary hemostasis are more common than congenital causes in clinical practice. The most common cause for acquired secondary hemostatic effects is toxin ingestion, notably anticoagulant rodenticide ingestion. This toxicity results in a vitamin K dependent coagulation factor deficiency. Treatment of anticoagulant rodenticide ingestion includes decontamination if recent (emesis and activated charcoal) and potentially vitamin K1 supplementation. If ingestion was not recent and the patient is
clinical for anemia from hemorrhage, therapy including fresh whole blood transfusion or component therapy (plasma products and packed red blood cells) is warranted.

Congenital deficiencies of secondary hemostasis are less common, including the hemophilia disease states. Hemophilia A is associated with congenital deficiency of factor VIII. Hemophilia B is associated with congenital deficiency of factor IX. Depending on the degree of factor deficiency, the PTT may be prolonged, while the PT remains normal. With that said, there are times when standard clotting testing is normal despite suspicion for a specific factor abnormality. In these cases, testing exists evaluating specific coagulation factor concentrations.

Conclusion
The approach to the bleeding patient is directed at providing hemostasis, providing appropriate therapy, and performing the necessary diagnostic tests to determine the underlying cause.

In the emergent setting, bedside testing is the most useful for rapid patient assessment and direction of treatment, notably the platelet count, PCV, TP, BMBT, PT, and PTT.

Although there is some overlap in clinical signs, disorders of primary hemostasis are more likely to present with signs consistent with surface bleeding whereas disorders of secondary hemostasis are more likely to present with intracavitary bleeding.

References
Veterinarians are faced with clinical challenges every day with the goal of solving diagnostic dilemmas, reducing morbidity and mortality, and ultimately restoring patient health. One of the most challenging issues we face is determining the best sedation and/or anesthesia protocol for the sick, small animal patient. The objective of this lecture is to provide a clinical tool for understanding common sedation and/or anesthesia options for veterinary patients.

Regardless of the presenting complaint, an important concept to remember when approaching any emergency patient is a rapid primary survey, keeping in mind the ABCDs of evaluation and resuscitation. Briefly, "A" refers to Airway or Arterial Bleeding. "B", Breathing is equally important assessing the character of the patient's respirations. “C” refers to Circulation and the overall perfusion status of the patient. Finally, “D” refers to Disability notably the patient's mental status.

**What can we control?**
The importance of oxygenation and perfusion cannot be over emphasized. Supplemental oxygen either on presentation or pre-oxygenation prior to anesthesia are important concepts to remember in the sick, small animal patient.

**Oxygen supplementation techniques**

<table>
<thead>
<tr>
<th>Supplementation technique</th>
<th>Required flow rate</th>
<th>Maximum inspired oxygen concentration achieved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flow-by</td>
<td>3-15 l/min</td>
<td>40%</td>
</tr>
<tr>
<td>Oxygen cage</td>
<td>15 l/min</td>
<td>45-60%</td>
</tr>
<tr>
<td>Oxygen hood (unsealed bag)</td>
<td>5-15 l/min</td>
<td>85-95%</td>
</tr>
<tr>
<td>Oxygen collar</td>
<td>1 l/10 kg bodyweight/min</td>
<td>&lt;80%</td>
</tr>
<tr>
<td>Nasal cannula</td>
<td>50-100 ml/kg/min</td>
<td>40%</td>
</tr>
<tr>
<td>Nasal catheters</td>
<td>50-100 ml/kg/min</td>
<td>40-50%</td>
</tr>
<tr>
<td>Nasopharyngeal catheter</td>
<td>50-100 ml/kg/min</td>
<td>60-70%</td>
</tr>
<tr>
<td>Nasotracheal catheter</td>
<td>25-50 ml/kg/min</td>
<td>80-90%</td>
</tr>
</tbody>
</table>

Aside from oxygenation, perfusion is an essential part of health to assess and address. Perfusion is defined as the flow of blood through arteries and capillaries delivering nutrients and oxygen to cells (hence the importance of oxygen supplementation as listed above) and removing cellular waste products.

**Aside from oxygen, what else can we control?**

**Blood products**

Oxygen delivery to the issues is more than just administration of oxygen. Oxygen is carried in the blood in two forms: (1) dissolved in plasma and RBC water (about 2% of the total) and (2) reversibly bound to hemoglobin (about 98% of the total). It is therefore easy to see how oxygen molecules need a carrier to transport to the vital organs, hemoglobin. Patients that are anemic (PCV <20%) may require supplementation of red blood cells to improve their oxygen carrying capacity prior to sedation or anesthesia. This can be achieved with red blood cell products such as packed red blood cells or hemoglobin based oxygen carrying solutions (i.e. Oxyglobin®).

**Volume**

Aside from oxygen and a carrier molecule (hemoglobin within red blood cells), hypovolemic patients require volume replacement to improve perfusion and therefore oxygenation. Volume replacement is commonly achieved with crystalloid and/or colloid solutions.
Once the patient is deemed to be stable for sedation / analgesia / anesthesia, the clinician must critically evaluate which medication or medications would be most suitable.

**Anesthesia / analgesia drug review**

**Alpha-2 agonists (medetomidine, dexmedetomidine, xylazine)**

Alpha-2 agonist dexametomidine (Dexdomitor®) is a very specific drug affecting the alpha-2 receptor. More specifically, alpha-2 agonists work in the CNS via pre-synaptic receptors to decrease norepinephrine release, resulting in enhanced parasympathetic tone. Following administration, sedation lasts approximately 2 to 4 hours with analgesia lasting for a shorter period of time. Dexametomidine is reversible with atipamezole (Antisedan®).

Side effects of alpha-2 agonists include stimulation of peripheral alpha-1 and alpha-2 receptors in the vasculature causing peripheral vasoconstriction (increased systemic vascular resistance). Clinicians commonly note hypertension with a reflex bradycardia, often with heart rates of 50 beats per minute or less! Additional clinical findings include an appearance of pale mucous membranes and peripheral vasoconstriction with cold extremities.

The combination of the dissociative tiletamine and benzodiazepine, zolazeplan (Telazol®), is also commonly in small animal medicine, notably as a feline premedication. Telazol® provides mild analgesia and should not be used alone for procedures in which moderate to severe pain is expected, including castration, ovariohysterectomy, and dental extraction.

Xylazine, another alpha-2 agonist is less potent as compared to dexmedetomidine but induces a longer duration of hypertension through vasoconstriction. Xylazine is reported to induce a bi-phasic blood pressure with initial hypertension followed by prolonged hypotension. Anticholinergic agents such as atropine or glycopyrrolate are often used in combination with xylazine. Conversely, the use of anticholinergic agents with dexametomidine is discouraged due to the risk of hypertension and arrhythmias. The sedation and analgesia induced by xylazine can be reversed with yohimbine.

**Benzodiazepines (diazepam, midazolam)**

The benzodiazepines, diazepam (Valium®) and midazolam (Versed®), are tranquilizers, specifically enhancing the activity of the central nervous system inhibitory neurotransmitter, gamma-aminobutyric acid, as well as, combining with benzodiazepine receptors in the central nervous system. These medications induce mild sedation, muscle-relaxation, and acts as an anticonvulsant.

Importantly, the benzodiazepine class of drugs does not have analgesic activity. They are reversible with flumazenil (Romazicon®). Diazepam is supplied in a propylene glycol base, not a water based preparation, and therefore it is recommended to administer this intravenously as uptake from IM or SQ injection may be slow, unpredictable, and painful. Moreover, IV administration of propylene glycol based solutions have the risk of hemolysis, thrombophlebitis and cardiotoxicity. Conversely, midazolam is water-soluble and can be administered IV, SQ or IM with predictable uptake.

**Dissociatives (ketamine)**

Ketamine, a NMDA Receptor Agonist, provides both analgesic and sedative effects and cause dose-dependent depression of the central nervous system. Although the patient is dissociated from the environment, pharyngeal, laryngeal, corneal, and pedal reflexes persist and the eyes remain open. Tiletamine, which is chemically similar to ketamine, is more potent and has a longer duration of effect than ketamine.

These dissociative medications have minimal cardiovascular or respiratory depression. Ketamine should be used with caution in patients with cardiac disease such as hypertrophic cardiomyopathy, ischemic heart disease and renal insufficiency as it increases sympathetic tone and thus can increase blood pressure, heart rate and cardiac output. Ketamine also increases intra-cranial and intraocular pressure so should be used with caution with head trauma or seizure history.

**Etomidate**

Etomidate is a non-barbiturate anesthetic. Unlike other medications used for sedation or anesthesia, it does not affect cardiovascular function, notably having no effect on blood pressure, heart rate, or cardiac output. Concerns with this medication include its high osmolality (>4000 mOsm) which has the potential for hemolysis. It also interferes with cortisol production following induction.

**Opioids (hydromorphone, methadone, morphine, oxymorphone, buprenorphine, butorphanol)**

Opioids are considered to have three notable receptors, but clinically the mu and the kappa receptors are the ones most often considered when planning for sedation and analgesia.

Opioids commonly used in practice include hydromorphone, methadone, oxymorphone, morphine, buprenorphine, and butorphanol. Hydromorphone, methadone, oxymorphone and morphine are µ receptor agonists and are good choices for patients expected to experience moderate-to-severe pain. These opioids provide excellent analgesia as well as good sedative properties.

Common clinical side effects include hypersalivation, vomiting, nausea, and panting. Morphine is also known to cause histamine release following IV administration.

Butorphanol is a not a pure agonist, rather considered an µ agonist/ K antagonist, meaning that it will reverse some µ opioid effects. These provide less potent analgesia as compared to the primary µ agonists and should be used only for mild pain or short-term pain.
Buprenorphine is considered a partial µ agonist with four-to-six-hour duration of effect. Clinically, the author uses this more in cats than dogs.

**Phenothiazines (acepromazine)**

Acepromazine is the most common drug used in the class of drugs known as the phenothiazines. Acepromazine provides sedation via anti-dopaminergic (D2) effects and suppression of the sympathetic nervous system. It causes an alpha-adrenergic blockade which results in vasodilation and often hypotension. It has a relatively long duration of action, considered to be 6-12 hours and is not recommended for patients with liver disease as decreased hepatic metabolism may result in a prolonged recovery. Acepromazine does not result in analgesia and therefore should not be used as a pain medication. It should also be avoided in patients with hypotension, hypovolemia, shock, significant heart disease, or coagulopathy/platelet disease.

While previously it was believed that acepromazine may result in seizures in dogs with a history of seizures, a recent retrospective study has shown that acepromazine does not cause seizures in dogs with a history of seizures of various origins.

**Propofol**

Propofol is a non-barbiturate anesthetic and a popular medication in veterinarian medicine. Propofol undergoes hepatic metabolism as well as extra-hepatic metabolism. This drug has significant cardiovascular effects, decreasing cardiac output and causing vasodilation without a reflex tachycardia. Propofol should be used with caution in animals with hypotension, hypovolemia or cardiovascular dysfunction.

**Alfaxalone**

Alfaxalone is another drug that is becoming more popular in veterinary medicine and reported to have less cardiopulmonary depression than other intravenous induction agents such as thiopental or propofol. Alfaxalone, a progesterone analogue, is a neurosteroid which interacts with the gamma aminobutyric acid (GABA)_A receptor and produces anesthesia and muscle relaxation.

### Opioid drug potency chart

<table>
<thead>
<tr>
<th>Drug</th>
<th>Other names</th>
<th>Potency*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>Generic</td>
<td>1</td>
</tr>
<tr>
<td>Codeine</td>
<td>Generic</td>
<td>1/10</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>Vicodin, generic with acetaminophen</td>
<td>6x</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>Percocet, OxyContin</td>
<td>3–6x</td>
</tr>
<tr>
<td>Oxymorphone</td>
<td>Numorphan</td>
<td>10x</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>Dilaudid, generic</td>
<td>8x</td>
</tr>
<tr>
<td>Meperidine</td>
<td>Demerol</td>
<td>1/6</td>
</tr>
<tr>
<td>Propoxyphene</td>
<td>Darvon</td>
<td>1/3–1/6</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>Buprenex</td>
<td>25x</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Sublimaze</td>
<td>100x</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>Torbugesic, Stadol</td>
<td>5x</td>
</tr>
</tbody>
</table>

### Common drug doses

<table>
<thead>
<tr>
<th></th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acepromazine mg/kg</td>
<td>0.01–0.02 S/C 0.005–0.01 IV</td>
<td>Acep is not an effective sedative</td>
</tr>
<tr>
<td>Alfaxalone mg/kg</td>
<td>Premedicated: 2mg/kg IV Not premedicated 3mg/kg IV</td>
<td>Premedicated: 2mg/kg IV Not premedicated 3mg/kg IV</td>
</tr>
<tr>
<td>Buprenorphine mg/kg</td>
<td>0.01–0.02 mg/kg SQ, IM, IV</td>
<td>0.01–0.02 mg/kg SQ, IM, IV</td>
</tr>
<tr>
<td>Butorphanol mg/kg</td>
<td>0.2–0.2mg/kg SQ, IM, IV</td>
<td>0.2–0.4mg/kg SQ, IM, IV</td>
</tr>
<tr>
<td>Drug</td>
<td>Dosage</td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>-------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Dexmedetomidine micrograms/m²</td>
<td>375 IV; 500 IM micrograms/m²; 40 micrograms/kg IM micrograms/m²</td>
<td></td>
</tr>
<tr>
<td>Etomidate mg/kg</td>
<td>1–2 mg/kg; 1–2 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Fentanyl µg/kg</td>
<td>CRI: 0.1–0.7 µg/kg/min; CRI: 0.1–0.7 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Hydromorphone mg/kg</td>
<td>0.05–0.2mg/kg SQ, IM, IV; 0.05–0.2mg/kg SQ, IM, IV</td>
<td></td>
</tr>
<tr>
<td>Methadone mg/kg</td>
<td>0.1–1.0 mg/kg SQ, IM, IV; 0.05–0.5 mg/kg SQ, IM, IV</td>
<td></td>
</tr>
<tr>
<td>Midazolam mg/kg</td>
<td>0.1–0.5 SQ, IM, IV; 0.1–0.5 S/C, IV</td>
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<tr>
<td>Morphine mg/kg</td>
<td>0.1–1.0 mg/kg SQ, IM, IV; 0.1–1.0 mg/kg SQ, IM, IV</td>
<td></td>
</tr>
<tr>
<td>Propofol mg/kg</td>
<td>1-6mg/kg IV; 1-6mg/kg IV</td>
<td></td>
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</table>

**References**


Feline hepatic lipidosis is a potentially fatal intrahepatic cholestatic process that develops in cats in association with prolonged anorexia and catabolism. It is the most common form of liver disease in cats in North America\(^7\), although seen worldwide. Most affected cats are middle-aged adults (median age 7 years), domestic shorthaired cats, and obese or overweight\(^1, 3\). There is no gender or breed bias. The period of anorexia documented prior to evaluation may be as short as 2–7 days\(^3, 4\).

Feline Hepatic Lipidosis can occur as either a primary idiopathic disease syndrome or secondary to another disease process, such as pancreatitis, small intestinal diseases, renal disease, and neoplasia.

Although prolonged anorexia and decreased nutritional intake is the primary concern, decreased cellular nutrition can also lead to the development of hepatic lipidosis. Processes such as uncontrolled diabetes can lead to decreased cellular nutrition where intake is adequate, or even increased, followed by fat deposition in the liver.

Pathogenesis
The pathogenesis of hepatic lipidosis is likely multifactorial and many theories have been discussed.

One such theory is that there is a defect in hepatic lipid mobilization, decreased ability for hepatic fat oxidation, and decreased lipoprotein removal from the liver. Evidence for this theory includes ultrastructural changes of the liver, notably decreased number hepatic peroxisomes, altered mitochondria and altered endoplasmic reticulum.

Cats are also predisposed to accumulating triglycerides in their hepatocytes. With prolonged anorexia and decreased cellular nutrition there is hepatocellular fatty vacuolation despite an increased rate of Very-low-density lipoprotein (VLDL) secretion. In a normal feline liver, fat comprises < 5% of the total organ weight. In contrast, the liver of a cat with hepatic lipidosis may triple in weight due to lipid accumulation. This is more pronounced in overweight cats as prolonged anorexia results in a release of fatty acids from their abundant peripheral adipose stores, overwhelming the liver's ability to use or transport the excess fatty acids and lipid.

When fatty acids are released from the peripheral stores, there are several pathways they can follow. They may undergo beta-oxidation, be used for triglyceride synthesis, be converted to phospholipids, be used in the formation of cholesterol esters, or be packaged with apoproteins for dispersal as lipoproteins.

The most important pathway for triglyceride distribution is the formation of Very-low-density lipoprotein (VLDL). In order for this to take place, there must be an intact lipid transport system, adequate combination with apoproteins, formation of a secretory particle, and transportation out of the hepatocyte and into the perisinusoidal space. If any of these pathways are disrupted, this will result in abnormal fat mobilization.

Presenting complaint
Most patients presenting with feline hepatic lipidosis are bright and alert. The most common presenting complaints from the owners during questioning include inappetence, weight loss, vomiting, diarrhea and lethargy\(^3, 4\).

Less commonly they present with more serious illness as a result of hepatic encephalopathy or weakness as a result of hypokalemia.

Clinical signs- examination
Common physical examination findings include dehydration, icterus, an unkempt appearance, a pendulous abdomen with cranial organomegaly (hepatomegaly), and weight loss seen as dorsal muscle wasting.

Bloodwork
Complete blood count
Complete blood count (CBC) findings often include a nonregenerative anemia and a stress leukogram. The nonregenerative anemia may result from chronic disease, phlebotomy, or hypophosphatemia. Stress leukograms have a characteristic white blood cell count differential including a mature neutrophilia, lymphopenia, and eosinopenia. A monocytosis is variable in cats. Neutrophilia is due to decreased adherence to the vascular endothelium, which prolongs circulating time and increased bone marrow release of neutrophils. Lymphopenia is due to redistribution or lysis of lymphocytes. Poikilocytosis is common and may reflect altered red blood cell membrane lipids or oxidative stress to the red blood cells affecting cell membrane stability and flexibility. Heinz bodies are also common, and may reflect oxidation as a result of medications, inflammation, or the underlying disease\(^5\).
Serum biochemistry

Serum biochemical changes primarily reflect cholestasis. Cholestasis, is a term used to describe any condition in which there is impaired flow of bile from the bile duct preventing bile from entering into the intestines. Cholestasis may result from a variety of diseases related to the gallbladder, liver, and pancreas.

Most cats have a markedly increased Alkaline phosphatase (ALP/ALKP) as well as an increased serum bilirubin concentration. Transaminases such as Alanine transaminase (ALT) may be slightly elevated, but it would be uncommon for a cat to present with primary hepatic lipidosis and have a markedly elevated Alanine transaminase and only a mild elevation in Alkaline phosphatase. Gamma-glutamyl transferase (GGT) is often within the normal range in patients with hepatic lipidosis. This is in contrast to other diseases where the gamma-glutamyl transferase (GGT) elevations are normally in parallel Alkaline phosphatase (ALP). An elevated gamma-glutamyl transferase (GGT) in a cat with hepatic lipidosis would increase the suspicion of a secondary process, such as pancreatitis, cholangitis, an extrahepatic bile duct obstruction, or neoplasia of the pancreas, liver, or biliary tree.

Other biochemical abnormalities include a low Blood Urea Nitrogen (BUN) and low Albumin. The BUN is often low due to an abnormal urea cycle (also known as the ornithine cycle, this is a cycle occurring in many animals that produces urea ((NH2)2CO) from ammonia (NH3) and takes place primarily in the liver and to a lesser extent in the kidney). The albumin is low as a result of decreased synthesis and loss.

Hypoglycemia is uncommon, as more than 70% of the functional liver mass must be lost before hypoglycemia ensues. In contrast, hyperglycemia is present in about 50% of cases due to either a stress hyperglycemia or the underlying disease process, a primary example being diabetes mellitus.

Electrolyte panel

Important electrolytes to assess include potassium, phosphorus and magnesium. Prolonged anorexia results in total body depletion and untreated and persistent hypokalemia, hypophosphatemia, and less commonly hypomagnesemia increase the risk of morbidity and mortality. Signs of hypokalemia and hypophosphatemia include pallor due to red blood cell hemolysis, weakness, vomiting, and vetroflexion of the head and neck.

Coagulation profile

Evaluation of Prothrombin time (PT) and Partial Thromboplastin Time (PTT) is an essential part of the diagnostic evaluation in feline hepatic lipidosis. In less than 7 days cats can become vitamin K deficient and over 50% of cats with hepatic lipidosis have coagulation test abnormalities. Coagulation profile abnormalities are not uncommon as the liver plays a primary role in clotting factor synthesis, notably the Vitamin K dependent clotting factors II, VII, IX and X, Protein C and Protein S10. The importance of checking clotting factors and treating coagulopathies cannot be over emphasized in patients that may require the placement of large bore feeding tubes, liver aspirates or biopsies8, or jugular venipuncture.

Blood gas evaluation

Common venous blood gas abnormalities include a metabolic acidosis, consistent with elevated ketones and lactate. The lactate elevation is suspected as a result of impaired hepatic lactate metabolism, dehydration, hypovolemia, and poor perfusion. The elevated ketone level suspected as a result of poor cellular nutrition and accumulation of plasma ketones.

Urinalysis

Lipid accumulation may be present in the urine sample from renal tubule lipid vacuolation. Bilirubin pigmenturia and bile crystalluria may also be seen. Due to prolonged anorexia and fluid loss as a result of vomiting and diarrhea, dehydration is supported by an increased urine specific gravity.

Abdominal ultrasound

Following a thorough physical examination and biochemical analysis (complete blood count, serum biochemistry, urinalysis, and coagulation panel), further diagnostics are often considered in an attempt to rule out a primarily disease which resulted in the initial anorexia and subsequent development of secondary hepatic lipidosis.

An abdominal ultrasound allows a non-invasive evaluation of the abdominal organs, notably the liver, pancreas, stomach, small intestine, large intestine, spleen, and kidneys.

In health, the liver is isoechoic to the falciform fat and to the cortex of the right kidney, and hypoechoic to the spleen. With hepatic lipidosis, the liver is characteristically large (hepatomegaly) with diffuse hyperechoic parenchyma, hyperechoic to the falciform fat and renal cortex, and isoechoic to the spleen6.

Additional concerning findings on ultrasound include pancreatitis, triaditis, biliary disease, and inflammatory bowel disease. Triaditis is a term referring to inflammatory diseases involving three specific organs, namely the liver, pancreas and small intestine.

Combined with the history, examination findings, bloodwork results, and ultrasound findings, liver aspirates are often adequate for a presumptive diagnosis of hepatic lipidosis. The expected cytological finding is hepatocellular lipid vacuolation. Aspirates not only support the diagnosis with the presence of lipid, but also rule out other primary liver diseases that may appear similar ultrasonographically, (hyperechoic hepatomegaly) including hepatic lymphoma and hepatitis.
A true tissue biopsy (ultrasound guided, surgical, or laparoscopic) has an increased risk of complications, notably hemorrhage, and may be academic if all other information points towards a diagnosis of hepatic lipidosis. On gross evaluation (surgical or laparoscopic), the liver is tan-yellow in color, friable, and biopsy specimens float in formalin. Histopathology reveals marked hepatocellular vacuolation. If true tissue biopsies are needed, it is imperative to stabilize the patient, including hydration, electrolyte abnormalities, coagulation abnormalities, and the overall cardiovascular status.

**Therapy**

Successful recovery of cats with hepatic lipidosis requires nutritional support, correction of fluid loss, correction of electrolyte abnormalities, and appropriate detection and treatment of an underlying disease process (if present).

**Enteral feeding**

The cornerstone of therapy in reversing hepatic lipidosis is appropriate nutritional support. For this reason, enteral feeding is initiated as soon as possible in the treatment process. Food requirements are calculated based on energy typically referred to as the resting energy requirement (RER). Common formulas used to calculate the RER for a feline patient:

1. \[ \text{RER} = 70 \times \text{(current bodyweight in kilograms)}^{0.75} \] (for > 5 kg)
2. \[ \text{RER} = 30 \times \text{BWkg} + 70 \] (for < 5 kg)
3. \[ \text{RER} = \text{60kcal} \times \text{BWkg} \]

Once enteral feeding is initiated, the complete daily caloric intake (100% RER) is not offered on day one. Once the RER is calculated, a fraction (25-33%) is divided over the first 24 hours. If this is tolerated, the caloric intake is increased on day two (50-67%). Finally, on day three and thereafter, the caloric intake is increased to 100% RER. Over a 24 hour period, the feeding schedule will be adjusted based on the individual patient’s characteristics, often dividing the total caloric intake into 4-6 feedings rather than large infrequent boluses. Smaller volumes are preferred as prolonged anorexia in patients with hepatic lipidosis may reduce the gastric volume to as little as 10% of the original gastric volume.

For example: a cat that has a 5 kg ideal body weight:

- Day 1 RER = 300kcal * 25% = 75kcal total, or approximately 19kcal every 6 hours.
- Day 2 RER = 300kcal * 50% = 150kcal total, or approximately 38kcal every 6 hours.
- Day 1 RER = 300kcal * 100% = 300kcal total, or approximately 75kcal every 6 hours.

The importance of nutritional support cannot be overemphasized. Continued lack of nutrition will lead to further lipolysis and storage of lipid within the hepatocytes.

By the time these patients are presented to the hospital and diagnosed with hepatic lipidosis, they have often been anorectic for at least 3-5 days. As a result, force-feeding is not considered an effective or well-tolerated form of enteral nutrition. It is not only difficult to ensure adequate caloric intake, but continued nausea and systemic illness may develop into a food aversion. Appetite stimulants (i.e. Mirtazapine, Cyproheptadine) are also clinically ineffective and not recommended.

For this reason, adequate nutritional support often involves the use of a large bore feeding tube, nasoesophageal (NE) tube, esophageal tube (E-Tube) or Gastrostomy tube (G-Tube).

Esophagostomy tubes (E-tubes) are the most common feeding tube used in feline hepatic lipidosis. Placement requires a stable patient including coagulation factors, correction of electrolyte abnormalities, the cardiovascular system, and the ability to tolerate a short general anesthesia. As compared to a NE-feeding tube, an E-Tube allows the clinician to start a more suitable diet, and the E-Tube has fewer complications than G-tubes.

However, when the patient is not stable enough for the placement of an E-tube, initial feeding via a NE-tube is an accepted alternative. A NE-tube is inexpensive and does not require anesthesia in most cases.

Following the placement of any feeding tube (NE tube, E-Tube, or G-Tube) a radiograph is recommended to confirm placement. An E-collars are also recommended to avoid accidental trauma to the tube or premature removal of the tube.

Securing the E-Tube is imperative. While traditionally, gauze and Vet Wrap™ has been used, the Kitty Kollar (http://www.kittykollar.com) has been used by the author with success. This is a washable, fabric collar designed to wear in conjunction with an esophageal feeding tube. The collar replaces the gauze and bandaging normally used to hold the tube in place, keeping it more sanitary, more stable and comfortable, and more protected against scratching and damage.

When using an E-Tube in practice, while there are several diets to consider (see chart below), the author commonly uses Hill’s A/D. Undiluted Hill’s A/D contains 1.2 Kcal per ml. If the contents of 1 can are diluted with 50ml of water, the mixture will contain 1.0 KCal per ml and is a better consistency for placement through the E-tube with less risk of clogging of the tube.

Fluid and electrolyte therapy is essential for rehydration, maintenance, as well as correction of electrolyte abnormalities primarily resulting from a lack of nutritional intake. A balanced electrolyte solution is recommended. Due to decreased hepatic lactate metabolism, hyperlactatemia may already be present. For this reason, some clinicians avoid lactate-containing solutions such as...
Lactated Ringer's solution (LRS). With that said, more often we are concerned about improving overall hydration and intravascular volume replacement, and the use of one specific crystalloid as compared to another is more academic than clinical. Dextrose supplementation is also avoided unless hypoglycemia is documented, as many patients are already showing some degree of hyperglycemia as a result of glucose intolerance.

Electrolyte monitoring is also valuable, notably to assess and correct hypokalemia and hypophosphatemia. If supplementing potassium yet the patient is not responding, refractory hypokalemia can be seen with concurrent hypomagnesemia. Refractory hypokalemia is a negative prognostic indicator, thus hypokalemia must be addressed aggressively. Magnesium is found in enteral diets and once enteral feeding resumes this often corrects. When critical and not yet on an enteral diet, intravenous CRI supplementation is needed.

Although total body electrolyte depletion is expected, initial evaluation may show normal or low-normal electrolyte values. Prolonged anorexia and electrolyte depletion initially results in shifting of electrolytes out of the cells into the periphery. This is why the values, although expected to be low, can be normal or low-normal. Once enteral or parenteral nutritional therapy is started, a refeeding phenomenon is seen within 12-24 hours of therapy. Refeeding causes a shift in the body from a catabolic state to an anabolic state. Administration of enteral (or parenteral) nutrition stimulates the release of insulin, resulting in a dramatic shift of electrolytes from the extracellular space to the intracellular space, primarily phosphorus, potassium and to a lesser degree magnesium. Thus, the phosphorus (and other electrolytes) that were shifted extracellularly, are pushed back into the cell, resulting in dramatic and often sudden decreases in serum electrolytes.

Repeated blood sampling and electrolyte monitoring will depend on the patient’s clinical signs and disease severity; electrolytes are often checked every 6-12 hours initially, then 12-24 hours for continued monitoring.

While the focus thus far has been on stabilization, diagnosis, and nutritional support, many of these patients present with anorexia, nausea, and gastrointestinal signs such as vomiting and diarrhea. In order for enteral feeding to be effective, nausea and vomiting must be addressed. The vomiting is addressed in several ways; pharmacologic therapy, reducing meal volume with an increasing meal frequency, and treatment of any existing underlying illness. Although enteral feeding via an E-Tube is typically performed every 4-6 hours, patients that cannot handle these volumes, trickle feeding is an alternative feeding method. Trickle feeding is performed with slow, constant feeding over a longer period of time, often for convenience employing a syringe pump or fluid pump to deliver a constant infusion of enteral nutrition through the attached feeding tube.

Vitamin and anti-oxidant therapy should be considered as well. Cobalamin (Vitamin B12) deficiency is common in cats with intestinal or pancreatic disease. Thiamine (vitamin B1) deficiency is also common and can result in weakness, lethargy, ventroflexion, and poor pupillary light responses, vestibular signs10.

Cats with hepatic lipidosis also are suspected to have a vitamin K deficiency. Vitamin K treatment is imperative when a coagulopathy is diagnosed prior to insertion of feeding tubes, jugular venipuncture, or hepatic aspiration / biopsy. Vitamin K therapy is also empirically used in patients when a large bore feeding tube placement is scheduled.

Supplementation with L-carnitine has demonstrated improved fat metabolism and clinical survival. L-carnitine transports long chain fatty acids across the mitochondrial membrane for Beta oxidation, and is an essential cofactor for fatty acid oxidation. S-adenosyl-L-methionine (SAMe), an essential methyl donor and important for glutathione (GSH) synthesis may also aid in hepatic recovery.

**Prognosis**
The presence of a concurrent medical condition and the ability to treat this condition with directly affect the outcome. Overall, greater than 80% of patients can have a full recovery. With treatment, serum bilirubin concentration should decrease by 50% in approximately 7-10 days. The liver values may remain elevated at that time, but improve slowly with therapy. Feeding via the E-Tube may be needed for 3-6 weeks, and should be reduced and stopped only when there is consistent documentation of adequate oral caloric intake.

**Summary**
For successful treatment of feline hepatic lipidosis, client education and active owner involvement is essential. Treatment may require weeks to months of assisted feedings, electrolyte support, and treatment of concurrent medical conditions. A recovery rate greater than 80% is reported if the primary disease can be identified and treated.

**Caloric densities, for feeding volume calculations.**

<table>
<thead>
<tr>
<th>Formula</th>
<th>Calorie Density</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hill’s A/D&lt;sup&gt;TM&lt;/sup&gt;</td>
<td>1.2 kcal/ml</td>
</tr>
<tr>
<td>Rebound&lt;sup&gt;TM&lt;/sup&gt;</td>
<td>1 kcal/ml</td>
</tr>
<tr>
<td>Clinicare&lt;sup&gt;TM&lt;/sup&gt;</td>
<td>1 kcal/ml</td>
</tr>
<tr>
<td>Royal Canin/MediCal Recovery&lt;sup&gt;TM&lt;/sup&gt;</td>
<td>1.23 kcal/ml</td>
</tr>
<tr>
<td>Eukanuba Maximum Calorie&lt;sup&gt;TM&lt;/sup&gt;</td>
<td>2.1 kcal/ml</td>
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</table>
### Drugs used for vomiting

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorpromazine</td>
<td>0.5mg/kg</td>
<td>TID</td>
<td>IM</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Prochlorpromazine</td>
<td>0.1mg/kg</td>
<td>QID</td>
<td>IM</td>
<td>Vomiting</td>
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<tr>
<td>Metoclopramide</td>
<td>1-2mg/kg</td>
<td>Over 24 hours</td>
<td>IV CRI</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Ondasetron</td>
<td>0.1-0.2mg/kg</td>
<td>BID-QID</td>
<td>IV / PO</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Dolasetron</td>
<td>0.6mg/kg</td>
<td>SID-BID</td>
<td>PO, SC, IV</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Maropitant</td>
<td>1mg/kg</td>
<td>SID &lt; 5 days</td>
<td>SQ / PO</td>
<td>Vomiting</td>
</tr>
</tbody>
</table>

### Drugs used for appetite stimulation

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mirtazepine</td>
<td>1.875-3.75mg</td>
<td>q72h</td>
<td>PO</td>
<td>Appetite stimulant</td>
</tr>
<tr>
<td>Cyproheptadine</td>
<td>2 mg</td>
<td>BID-TID</td>
<td>PO</td>
<td>Appetite stimulant</td>
</tr>
</tbody>
</table>

### Drugs used for hepatic support

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin K1</td>
<td>0.5-1.5mg/kg</td>
<td>SID-BID</td>
<td>SC/PO</td>
<td>Coagulopathy</td>
</tr>
<tr>
<td>L-Carnitine</td>
<td>250-500mg</td>
<td>SID</td>
<td>PO/Tube</td>
<td>Fat metabolism</td>
</tr>
<tr>
<td>Taurine</td>
<td>250-500mg</td>
<td>SID</td>
<td>PO/Tube</td>
<td>Lipidosis</td>
</tr>
<tr>
<td>Ursodeoxycholic acid</td>
<td>15mg/kg</td>
<td>SID-BID</td>
<td>PO/Tube</td>
<td>Cholestasis</td>
</tr>
<tr>
<td>S-adenosyl- L-methionine</td>
<td>20–40 mg/kg</td>
<td>SID</td>
<td>PO/Tube</td>
<td>Glutathione donor</td>
</tr>
<tr>
<td>Milk thistle (silymarin)</td>
<td>5-15mg/kg</td>
<td>SID</td>
<td>PO/Tube</td>
<td>Hepatoprotective antioxidant</td>
</tr>
<tr>
<td>Lactulose</td>
<td>0.25-2ml/kg</td>
<td>BID-QID</td>
<td>PO/Tube</td>
<td>Hepatic Encephalopathy</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>7.5mg/kg</td>
<td>BID</td>
<td>PO/Tube/IV</td>
<td>Hepatic Encephalopathy</td>
</tr>
<tr>
<td>Cobalamin</td>
<td>1 to 2 mL Vitamin B complex in 1 L of fluids</td>
<td>IV CRI in crystalloid fluids</td>
<td>IV</td>
<td>Cobalamin deficiency, pancreatic or GI disease</td>
</tr>
<tr>
<td>Thiamine</td>
<td>50 to 100mg</td>
<td>Total dose per day</td>
<td>PO</td>
<td>Low thiamine levels</td>
</tr>
</tbody>
</table>

### Drugs used for electrolyte support

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td><strong>Potassium Replacement Chart</strong></td>
<td></td>
<td>IV CRI</td>
<td>Hypokalemia</td>
</tr>
<tr>
<td>K+</td>
<td>Add to 500mls</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.1-3.5</td>
<td>14mEq</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.6-3.0</td>
<td>20mEq</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.1-2.5</td>
<td>28mEq</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.6-2.0</td>
<td>40mEq</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>For critical hypokalemia, you can infuse KCL at higher than recommended doses (e.g. KMax = 0.5 mEq/kg/h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnesium</td>
<td>0.75-1mEq/kg/day</td>
<td>24 hour CRI</td>
<td>IV Cri</td>
<td>Hypomagnesemia</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>0.01–0.03 mmol/kg/hr</td>
<td>IV CRI</td>
<td>IV CRI</td>
<td>Hypophosphatemia</td>
</tr>
</tbody>
</table>
References
Practical Fluid Therapy: It’s More Than Just Water and Salt
Garret Pachtinger, DVM, DACVECC
Veterinary Specialty and Emergency Center
Levittown, PA

Fluid therapy is one of the most commonly used therapies for the small animal practitioner. Despite a large amount of research the general consensus is that there is not one fluid type that is better than another for resuscitation. This is often why there is debate as to what fluids a practice should purchase to have on the shelf. Moreover, the type of fluid desired may vary based on the underlying disease process.

The reason that fluid therapy is so important in medicine is that living organisms are comprised predominantly of...fluid! Total body water content is approximately 60% of body weight in a non-obese, adult dog or cat. Total body water is further distributed between two major compartments: the intracellular (ICF) and extracellular (ECF) fluid.

**Total body water (TBW) fluid compartments**

<table>
<thead>
<tr>
<th>ECF (33% TBW)</th>
<th>Intracellular Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICF (66% TBW)</td>
<td></td>
</tr>
</tbody>
</table>

The ICF compartment is the larger of the two compartments and comprises 66% of the total body water and 40% of body weight. It is separated from the ECF compartment by a cell membrane that is permeable to water but impermeable to most solutes. The ECF comprises the remaining 33% of the TBW and 20% of body weight. The ECF is subdivided into the plasma (25% of ECF) and interstitial (75% of ECF) fluid compartments.

The need for fluid therapy is often divided into 2 main categories:
1. Restoring the patient’s intravascular volume (hypovolemia)
2. Replacement of extravascular fluid (dehydration)

There are 4 types of hypoperfusion commonly recognized in veterinary practice:
1. Hypovolemia (i.e., loss of intravascular volume)
2. Maldistributive / Septic (i.e., loss of vascular tone, fluid shifting, third spacing)
3. Cardiogenic (i.e., myocardial dysfunction leading to lack of cardiac output and perfusion)
4. Obstructive (i.e., decreased venous return to the right side of the heart as a result of obstruction, e.g., due to gastric dilatation and volvulus or pericardial effusion)

It is important to distinguish which type of hypoperfusion is present as their initial treatment as well as long term therapy will differ based on the underlying disease process. As compared to cardiogenic causes, when clinical signs of hypovolemia are present (pale mucous membranes, prolonged capillary refill time, dull mentation, poor pulse quality, cold extremities, and tachycardia or bradycardia in cats) intravascular fluids must be replaced for emergency resuscitation. The estimated shock volumes of fluids are 90 ml/kg in dogs, and 60ml/kg for cats. The author initially replaces 1/4 to 1/3 of the calculated volume as rapidly as possible, the reassess perfusion parameters, notably heart rate, mucous membrane color, CRT, pulse quality, blood pressure, and eventually urine output. The reason the volumes calculated seem high is that approximately 75% of the crystalloid fluid administered will redistribute out of the intravascular space within 30-60 minutes of administration.

The administration of synthetic colloids is another option considered in hypovolemic patients, notably if there is a concern for hypoproteinemia (TP < 4.5) or in combination with crystalloid therapy. Common colloid bolus doses are 10–20 ml/kg in dogs and 5–10 ml/kg in cats followed by rapid and frequent reassessment. Synthetic colloids such as Hetastarch and Vetstarch cause expansion of the intravascular volume by pulling fluid from the interstitial and intracellular spaces into the intravascular compartment and keeping the fluid within the intravascular space longer due to the colloidal properties.

Besides isotonic crystalloids and synthetic colloids, another alternative fluid therapy is hypertonic crystalloids, specifically hypertonic saline. Hypertonic saline is considered for rapid expansion of the intravascular compartment and used in patients that have a normal hydration status. Hypertonic saline is contraindicated for a patient that is dehydrated or hyponatremic. Hypertonic saline has a potent effect, drawing fluids from other compartments into the intravascular space due to its potent osmotic forces. The typical dose...
recommended for rapid resuscitation is 4-7 ml/kg of 7.5% HS over 20 minutes. Additionally, hypertonic saline is theorized to have other beneficial properties including improved myocardial contractility, activation of a neurogenic reflex leading to peripheral vasodilation, improving microcirculatory flow by preventing capillary collapse, a reduction of endothelium cell swelling and alterations in function of polymorphonuclear cells (PMN) and endothelial cells. Complications include bradycardia, bronchoconstriction, sodium fluctuations, fluid overload and pulmonary edema, phlebitis and ventricular arrhythmias.

To prolong the effect of fluid resuscitation, the author also considers the combined use of a hypertonic saline/synthetic colloid. To achieve this fluid mixture, 1:2.5 ratio of 23.4% hypertonic saline (sodium chloride) and hetastarch or Vetstarch are used. To easily make this solution, 17ml of 23.4% hypertonic saline and 43ml of the colloid are mixed in a 60ml syringe. 3-5ml are then used as a bolus in the canine patient and 2-3ml are used as a bolus in the feline patient, followed by re-assessment.

Once immediate life-threatening fluid deficits are replaced, the focus then shifts to the patient’s dehydration level, maintenance level, and provisions for suspected ongoing losses.

The following chart is commonly used to assess patient dehydration characteristics:

<table>
<thead>
<tr>
<th>Percent dehydration</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;5</td>
<td>No detectable abnormalities</td>
</tr>
<tr>
<td>5-8</td>
<td>Decreased skin turgor, dry mucous membranes</td>
</tr>
<tr>
<td>8-10</td>
<td>Decreased skin turgor, dry mucous membranes, eyes may be sunken in orbits, slight prolongation of CRT</td>
</tr>
<tr>
<td>10-12</td>
<td>Severe skin tenting, prolonged CRT, dry mucous membranes, eyes sunken in orbits, possibly signs of shock</td>
</tr>
<tr>
<td>&gt;12</td>
<td>All of the above plus signs of shock, often life threatening</td>
</tr>
</tbody>
</table>

Measurement of dehydration is subjective and is not expected to be detected clinically below 5%.

For patients with evidence of chronic dehydration on examination but stable cardiovascular parameters (i.e. no evidence of hypovolemia), fluid deficits are corrected over a 6-24 hour period.

Following treatment of hypovolemia, the following formulas are used to create a fluid therapy plan:

1. Dehydration fluid replacement = Body weight (kg) x %dehydration x 1000
2. Maintenance daily requirements = Body weight (kg) x 2–4 ml/kg/h.
3. On-going losses = 3-4 ml/kg/vomit or diarrhea

**Complications of fluid therapy**

While fluid therapy is often considered a benign treatment, it is not without risk. Complications to consider based on the individual patient characteristics include:

- **Pulmonary edema**
  - Volume overload
  - Increased vascular permeability

- **Rapid sodium shifts**
  - Neurologic signs
  - Obtundation
  - Cerebral edema
  - Seizures

- **Phlebitis**
  - Use of hyperosmotic agents

**Conclusions**

Intravenous fluid therapy can be performed rapidly and can be life saving for the emergency patient. A thorough history, physical examination, and preliminary diagnostics can be used to help differentiate disease processes which may be worsened by fluid therapy (i.e. cardiogenic shock), as well as help the clinician choose the best fluid type to improve the clinical condition.
TABLE: Colloids and their chemical properties.

<table>
<thead>
<tr>
<th>Colloid</th>
<th>Mean MW (KDa)</th>
<th>Molar substitution</th>
<th>COP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5% Human albumin</td>
<td>69</td>
<td>N/A</td>
<td>23.2± 0.1</td>
</tr>
<tr>
<td>25% Human albumin</td>
<td>69</td>
<td>N/A</td>
<td>&gt; 200</td>
</tr>
<tr>
<td>Canine fresh frozen plasma</td>
<td>69</td>
<td>N/A</td>
<td>17.1± 0.6</td>
</tr>
<tr>
<td>6% Hetastarch in 0.9% NaCl</td>
<td>600</td>
<td>0.7</td>
<td>32.7± 0.2</td>
</tr>
<tr>
<td>6% Hetastarch in balance electrolyte solution--Hextend™</td>
<td>670</td>
<td>0.75</td>
<td>37.9± 0.1</td>
</tr>
<tr>
<td>6% Voluven™</td>
<td>130</td>
<td>0.4</td>
<td>37.1± 0.8</td>
</tr>
<tr>
<td>6% Vetstarch™</td>
<td>130</td>
<td>0.4</td>
<td>40*</td>
</tr>
</tbody>
</table>

In vitro

TABLE: Common crystalloids and their chemical properties.

<table>
<thead>
<tr>
<th>Solution</th>
<th>LRS</th>
<th>Plasmalyte A; Norm R</th>
<th>0.9% NaCl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na</td>
<td>130</td>
<td>140</td>
<td>154</td>
</tr>
<tr>
<td>K</td>
<td>4</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>Ca</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mg</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>CI</td>
<td>109</td>
<td>98</td>
<td>154</td>
</tr>
<tr>
<td>Gluconate</td>
<td>0</td>
<td>23</td>
<td>0</td>
</tr>
<tr>
<td>Lactate</td>
<td>28</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Acetate</td>
<td>0</td>
<td>27</td>
<td>0</td>
</tr>
<tr>
<td>Osmolarity</td>
<td>270</td>
<td>294</td>
<td>310</td>
</tr>
</tbody>
</table>

References

Sweet! Emergency Management of DKA
Garret Pachtinger, DVM, DACVECC
Veterinary Specialty and Emergency Center
Levittown, PA

Pathophysiology
Diabetes mellitus (DM) is a common endocrine disease in dogs and cats characterized by an absolute or relative deficiency of insulin. The classic signs of DM are polyuria, polydypsia, polyphagia, and weight loss. Ultimately, hyperglycemia results from a combination of factors including decreased insulin production, insulin resistance, lack of glucose transport, and decreased availability of glucose by cells for energy.

Aside from glucose transport, insulin also has other important roles in the body including inhibition of lipolysis. Absence of insulin results in increased activity of the hormone sensitive lipase system resulting in increased free fatty acids (FFAs) in circulation as they are released from adipocytes. FFAs are taken up by the liver where they are primarily made into triglycerides, metabolized via the tricarboxylic (TCA) cycle to CO2 and water, or formed into the ketone bodies acetoacetate, β-hydroxybutyrate, and acetone.

An uncomplicated diabetic patient produces mostly triglycerides, with a small portion being shifted to ketone production. The question then remains, what causes the transformation of a stable diabetic patient to a diabetic ketoacidotic patient? Development of diabetic ketoacidosis (DKA) requires more than just an increased FFA production. Along with an increased FFA production, there are increased concentrations of circulating levels of diabetogenic hormones such as glucagon, epinephrine, cortisol, and growth hormone. These are increased as a result of additional stressors or illnesses. Although not always identified, these stressors or illnesses include any inflammatory, infectious, or even neoplastic process.

Epinephrine and glucagon inhibit insulin-mediated glucose uptake in muscle and stimulate hepatic glycogenolysis and gluconeogenesis which results in persistent hyperglycemia. Cortisol and growth hormone inhibit insulin activity and potentiate the effects of glucagon and epinephrine on hepatic glycogenolysis and gluconeogenesis. Additionally, epinephrine, glucagon and growth hormone stimulate lipolysis which increases the amount of circulating FFAs available for ketone formation. Persistent hyperglycemia, increases in ketone formation combined with academia results in diabetic ketoacidosis. Along with glucosuria (from marked hyperglycemia), ketoacids exacerbate the osmotic diuresis and combined with the associated illness often seen with DKA patients (vomiting, diarrhea, decreased intake) contribute to development of severe dehydration.

Historical findings
Most dogs and cats with DM present with a history of polyuria, polydypsia, polyphagia, and weight loss. The polyuria and polydipsia results from hyperglycemia that exceeds the renal threshold (average ~ 200 mg/dl in the dog, 250 mg/dl in the cat) leading to glucosuria and an osmotic diuresis, medullary washout, and electrolyte loss. A patient that presents with DKA not only often has a chronic history similar to DM, but they often have an acute history of lethargy, mental depression, anorexia, vomiting, diarrhea, weakness, and other signs consistent with concurrent disease (i.e. abdominal pain with pancreatitis or urinary tract signs with a pyelonephritis.)

Physical examination findings
Persistent hyperglycemia and ketonemia will result in clinical signs including lethargy, dehydration, hypovolemia, muscle wasting, vomiting, diarrhea, and an acetone odor on the breath. On physical examination, hepatomegaly is often manifested as cranial organomegaly. Other abnormalities include diabetic cataracts (dogs) and signs consistent with a peripheral neuropathy. Icterus can develop as a result of the complicating factors of hemolysis, hepatic lipidosis or acute pancreatitis.

Epidemiology
DKA is commonly associated with a new diagnosis of DM. The signalment for dogs and cats with DKA is similar to that for other presentations of DM. In one study 40% of newly diagnosed diabetic cats had evidence of ketoacidosis. In another study, 15% of newly diagnosed diabetic dogs were also diagnosed with diabetic ketoacidosis. Breed characteristics may also play a role in development of DKA. Certain breeds, notably the Keeshond, Miniature Schnauzer, Poodle, and Dachshund seem overrepresented. DKA patients are more likely to be obese and either a female dog or male cats.

Common concurrent disorders in dogs with diabetes mellitus include urinary tract infections, hyperadrenocorticism, acute pancreatitis, neoplasia, and hypothyroidism. In cats, concurrent diseases common in DKA include pancreatitis, hepatic lipidosis, cholangiohepatitis, chronic renal failure, infection, and neoplasia.
Clinical pathology
The diagnosis of DKA is made in the presence of hyperglycemia, glucosuria, acidemia, and ketonuria or ketonemia. Blood pH can be determined with a blood gas machine in patients with suspected DKA. Point-of-care analyzers have made this readily accessible (e.g., i-STAT®).

Additional clinical pathology findings include an elevated anion gap and hyperosmolality.

An elevated anion gap indicates an accumulation of unmeasured anions in the form of ketones. The anion gap can be calculated by the equation:

\[
\text{Anion gap} = [\text{sodium (mEq/L)} + \text{potassium (mEq/L)}] - [\text{chloride (mEq/L)} + \text{bicarbonate (mEq/L)}].
\]

Normal is approximately 17–24 mEq/L.

Hyperosmolality is also common in the DKA patient, estimated by the equation:

\[
\text{Osmolality} = 2[\text{sodium (mEq/L)} + \text{potassium (mEq/L)}] + \text{BUN (mg/dl)/2.8} + \text{glucose (mg/dl)/18}.
\]

Normal osmolality is approximately 290–310 mOsm/kg.

Additional diagnostics performed in DKA patients to help formulate the most appropriate treatment plan include:

- Complete blood count
- Chemistry profile
- Serum electrolyte profile
- Urinalysis
- Urine Culture and Sensitivity
- Thoracic radiographs
- Abdominal radiographs
- Abdominal ultrasound
- Coagulation profile

These diagnostics are used to rule out complicating and causative diseases including pancreatitis, renal disease, neoplasia, pulmonary parenchymal disease and other associated illnesses that transition a stable DM patient to an unstable DKA patient.

Clinicopathologic findings
DKA patients have a relative or absolute deficiency of insulin and excessive hepatic production of glucose resulting in hyperglycemia. As blood glucose concentration increases, renal threshold is exceeded (average ~ 200 mg/dl in the dog, 250 mg/dl in the cat) and glucosuria ensues.

On a complete blood count (CBC), approximately 50% of dogs with DKA will have a non-regenerative anemia. Causes for the anemia may include hypophosphatemia, anemia of chronic disease, GI blood loss, hemolysis, or neoplasia. Other CBC findings include a leukocytosis with left shift with infectious or inflammatory processes.

Electrolyte abnormalities are common, notably abnormalities of potassium, phosphorus, and magnesium. DKA patients often have a total body depletion of potassium as a result of decreased intake (anorexia) and increased losses through the gastrointestinal tract (vomiting and diarrhea), and osmotic diuresis. Although a total body potassium depletion is present, initial bloodwork often shows a normal or low-normal potassium level due to shifting of potassium. To maintain electroneutrality to balance the concurrent acidosis, potassium is shifted from the intracellular space to the extracellular space and hydrogen ions are shifted from the extracellular space into the intracellular space, giving a false sense of a normal potassium level although a total body depletion exists.

With treatment, notably fluid and insulin therapy, potassium and glucose are shifted back intracellularly resulting in hypokalemia. The hypokalemia, which can be significant, can result in muscle weakness, cervical ventroflexion, cardiac arrhythmias, and respiratory muscle failure.

Similarly, hypophosphatemia develops when phosphate is shifted from the intracellular space to the extracellular space. Like potassium, fluid and insulin therapy results in electrolyte shifting, and phosphorous is shifted back intracellularly in exchange for hydrogen ions to maintain electroneutrality. Hypophosphatemia can result in weakness, hemolysis, arrhythmias, myocardial depression, and even seizures.

Magnesium is also an important electrolyte to monitor. Hypomagnesemia results from electrolyte shifting seen with decreased intake, acid-base changes and an osmotic diuresis resulting in refractory hypokalemia despite supplementation, weakness, and arrhythmias.

Serum chemistry abnormalities found in DKA patients are often related to co morbidities. One example is increased liver enzymes seen with inflammatory conditions such as pancreatitis, hepatic lipidosis (cats), cholangiohepatitis, or bile duct obstruction. Azotemia is commonly found and may be due to pre-renal hypovolemia or underlying primary renal disease.

Urinalysis abnormalities include glucosuria, ketonuria, and decreased urine specific gravity as a result of the osmotic diuresis and medullary washout. The urine should also be evaluated for the presence of an inflammatory sediment and a sample should be submitted for bacterial culture regardless as an osmotic diuresis may result in an artificially dilute urine specific gravity. Although
ketonuria is expected, this may not be initially seen on the urine strip because the nitroprusside reagent in the urine dipstick reacts with acetoacetate and not with beta-hydroxybutyrate, which is the primary ketone body in DKA.

**Diagnostic imaging**
Radiography is valuable in the diagnostic evaluation of the DKA patient. Thoracic radiographs are used to assess pulmonary parenchymal disease or cardiac disease including pulmonary pneumonia, neoplasia, cardiomegaly, and / or congestive heart failure.

Abdominal radiographs and/or ultrasound can be used to identify abdominal disease associated with DKA including pancreatitis, pyelonephritis, hepatitis, intestinal diseases, and/or neoplasia.

**Treatment**
Treatment of the DKA patient is multifactorial, with the combined therapy of:

1. Fluid therapy
2. Insulin therapy
3. Correction of electrolyte imbalances
4. Nutrition

When designing a hospitalization and treatment plan it is important to be proactive. DKA patients often spend several days in the hospital with the need for frequent reassessment of blood glucose and electrolytes. For this reason, the author recommends placing a sampling catheter or central venous catheter on admission. These catheters will allow repeated, painless, venous sampling, central venous pressure monitoring (CVP) in those patients with a possible fluid intolerance (i.e. underlying heart disease ), the use of multiple fluid types which may be incompatible when administered through one catheter, and parenteral nutrition if enteral feeding is contraindicated such as with protracted vomiting or prolonged anorexia.

**Fluid therapy**
Fluid therapy should be started immediately. Most DKA patients are markedly dehydrated due to the osmotic diuresis (caused by the hyperglycemia, ketonemia, and medullary washout) and concurrent fluid loss from illness (vomiting, diarrhea, decreased intake). Fluid therapy is generally instituted for several hours (4-6) before starting insulin therapy. Rehydration alone will aid in decreasing the blood glucose concentration by dilution and increased glomerular filtration through the kidneys. Fluid therapy should be calculated based on an estimate of dehydration, presence of ongoing losses and maintenance requirements. Fluid therapy with a replacement crystalloid such as Normosol-R®, Plasmalyte 148®, 0.9% NaCl, or lactated Ringers is appropriate in most cases. Although potassium is often normal on bloodwork, due to a total body depletion (see electrolyte section above), potassium should be added to these replacement solutions.

<table>
<thead>
<tr>
<th>Table</th>
<th>Formulas that relate to fluid balance and response to fluid therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily maintenance volume</td>
<td>Volume ml = (30 x kgBW) + 70</td>
</tr>
</tbody>
</table>
| Resuscitation (shock) volume | Crystalloid (dog) = 80–90 ml/kgBW delivered as 30 ml/kg boluses  
Crystallloid (cat) = 50–60 ml/kgBW delivered as 20 ml/kg boluses  
Colloid (dog) = 10–20 ml/kgBW delivered as 5 ml/kg boluses  
Colloid (cat) = 5–10 ml/kgBW delivered as 2.5 ml/kg boluses  
7.5% saline (dog) = 10 ml/kgBW bolus, single dose (slowly)  
7.5% saline (cat) = 5 ml/kgBW bolus, single dose (slowly) |
| Replacement (dehydration) vol | Volume ml = % dehydration (estimate) x Body weight (kg) |

While an isotonic crystalloid fluid is appropriate in most cases, the fluid choice should be isotonic to the patient’s sodium level to prevent rapid sodium shifts. Regardless of the fluid type chosen, the fluid rate is not a set-it and forget-it treatment. Constant re-assessment is needed to ensure the patient is not at risk for fluid overload or continued dehydration with ongoing losses. Re-assessment patient parameters include serial physical examinations, PCV/TS, body weight, urine output and other losses such as continued vomiting, diarrhea, or excessive panting. It is important to remember that hyponatremia may appear severe in cases of severe hyperglycemia, but this is often an artifact. Pseudohyponatremia may be seen as a result of marked hyperglycemia resulting in water retained within the vascular space, diluting the plasma sodium. The corrected sodium can be calculated by adding the measured sodium with 1.6 (glucose mg/dl–100)/100. The pseudohyponatremia corrects once normoglycemia is established.
Insulin therapy

Insulin therapy is essential to provide glucose to the starving cells, decrease lipolysis, reverse ketosis and correct the acidemia. While nobody will argue the importance of insulin in treatment of the DKA patient, insulin is not the most important (or even preferred treatment) on presentation. It is typically recommended that insulin therapy be delayed for at least 4-6 hours while fluids are started. Delayed insulin therapy is recommended to prevent rapid glucose and electrolyte shifts without adequate fluid replacement as well as prevent a rapid decrease in blood glucose and shift in osmolality causing CNS fluid shifts.

When insulin therapy is started, regular, short acting insulin (Humulin R®) therapy is recommended. Regular insulin is administered either intravenously (IV) as a CRI or intramuscular (IM). Subcutaneous insulin therapy is not recommended as dehydration may delay absorption from the subcutaneous space.

When administered as an intravenous CRI, it is recommended to have at least two catheters. As discussed above, a sampling catheter or central venous catheter is preferred to aid in frequent venous sampling. The CRI solution is formulated using 2.2 U/kg of regular insulin for dogs or 1.1 U/kg of regular insulin for cats diluted in 250 ml of saline. Approximately 50 ml of the combined solution is allowed to run through the fluid line and discarded as insulin binds to the plastic tubing. The rate of CRI insulin administration is based on a CRI chart (example below) and adjusted based on serial blood glucose readings, often every 2-4 hours.

<table>
<thead>
<tr>
<th>Blood Glucose (mg/dl)</th>
<th>Insulin CRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 350</td>
<td>10ml/hr</td>
</tr>
<tr>
<td>250-349</td>
<td>7ml/hr</td>
</tr>
<tr>
<td>150-249</td>
<td>5ml/hr + 2.5% dextrose</td>
</tr>
<tr>
<td>100-149</td>
<td>3ml/hr + 5% dextrose</td>
</tr>
<tr>
<td>&lt;10</td>
<td>0ml/hr</td>
</tr>
</tbody>
</table>

If the CRI protocol is not used, an alternative is an intramuscular regular insulin protocol. The intramuscular protocol is less labor intensive and often considered in patients that are more stable and less dehydrated. A common starting dose is 0.25 U/kg of regular insulin administered every 4 hours. The author in practice uses the chart below. Similar to the CRI protocol, the insulin dose is adjusted based on serial blood glucose monitoring.

<table>
<thead>
<tr>
<th>Blood Glucose</th>
<th>Insulin Dose</th>
<th>IV Fluids – Dextrose</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;300</td>
<td>0.4u/kg</td>
<td>No dextrose</td>
</tr>
<tr>
<td>200-299</td>
<td>0.3u/kg</td>
<td>+ 2.5% dextrose</td>
</tr>
<tr>
<td>100-199</td>
<td>0.1u/kg</td>
<td>+ 5% dextrose</td>
</tr>
<tr>
<td>&lt;100</td>
<td>None</td>
<td>+ 5% dextrose</td>
</tr>
</tbody>
</table>

Once the blood glucose is controlled, ketosis is resolved, and clinical signs improve, notably vomiting, dehydration, and anorexia, subcutaneous insulin can be started. Common insulin types including Glargine and NPH, q12 hours. Following discharge, a blood glucose curve is recommended 7-10 days to ensure appropriate insulin therapy.

Electrolyte supplementation

Electrolyte monitoring should be performed once to twice daily, depending on the severity of the electrolyte abnormalities. The main electrolytes to monitor are potassium, phosphorus, and magnesium. Although pseudohyponatremia is often found on presentation, careful sodium monitoring can help the clinician assess the fluid therapy plan.

Hypokalemia is primarily due to anorexia (lack of intake), correction of the metabolic acidosis with fluid therapy, and osmotic diuresis. Clinical signs of hypokalemia include muscle weakness, cervical ventroflexion, cardiac arrhythmias, or respiratory muscle failure. In addition to fluid therapy, insulin therapy further worsens hypokalemia as it drives potassium intracellularly.

<table>
<thead>
<tr>
<th>Serum potassium concentration (mEq/L)</th>
<th>Potassium added to fluids (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5–5</td>
<td>20</td>
</tr>
<tr>
<td>3.0–3.4</td>
<td>30</td>
</tr>
</tbody>
</table>
Hypophosphatemia occurs due to acidosis, insulin therapy (which like potassium, drives it intracellularly), and urinary losses due to osmotic diuresis. If hypophosphatemia is present, phosphorous should be supplemented (0.01-0.12 mmol/kg/hr CRI) as potassium phosphate. Phosphorous levels below 3.5 mmol/L may cause illness weakness, lethargy, and ataxia. More severe signs of illness including seizures and hemolytic anemia may be seen with phosphorous levels below < 1.5 mmol/L.

Hypomagnesemia, like potassium and phosphorus, is seen with anorexia, decreased intake, gastrointestinal loss, and osmotic diuresis. Hypomagnesemia can result in arrhythmias, weakness, hypotension, and exacerbate other electrolyte abnormalities. If hypokalemia persists despite adequate potassium supplementation, it is important to check the magnesium level. Hypomagnesemia is treated with magnesium sulfate supplementation at 0.75 mEq/kg/day with appropriate electrolyte monitoring once to twice daily.

Treatment for metabolic acidosis
In most cases, specific therapy for metabolic acidosis is not required. Metabolic acidosis is often present from a combination of ketone formation, lactic acid from decreased perfusion, and uremic acids. Fluid therapy restores perfusion and insulin therapy decreases formation of ketones, thus often resolving the metabolic acidosis. When the metabolic acidosis is severe and persists despite appropriate therapy (pH < 7, bicarbonate < 8-11 mEq/L, and BE < -15 mm Hg) treatment with bicarbonate can be considered. The amount of bicarbonate to administer is calculated with the following question:

Base deficit X body weight (kg) X 0.3 (ECF volume).

One quarter to one third of this calculated deficit is administered as a CRI diluted in IV fluids over 4-6 hours. Following this treatment, bloodwork is rechecked to assess response to therapy.

Nutrition
While patients with DM are historically polyphagic, patients with DKA are often anorexic with complicating clinical signs including vomiting and diarrhea. Ultimately, we must have improved nutrition to reverse and resolve the state of DKA. At the most basic level, the transition from short acting regular insulin to the insulin required for successful discharge from the hospital (i.e. NPH, glargine, etc.) requires that the patient is eating well. This is often improved with resolution of the underlying cause (i.e. pancreatitis, enteritis, pyelonephritis, etc). Although enteral nutrition is ideal, depending on the severity of DKA and concurrent disease, further enteral and even parenteral nutritional support may be needed. Nasoesophageal feeding tubes can be placed with local anesthetic in critical patients. If ongoing nutritional support is needed an esophagostomy tube can be placed. When prolonged anorexia is suspected and enteral nutrition is not possible, placement of a central venous catheter and the use of TPN is recommended.

Patient monitoring and supportive care
Treatment and monitoring of the DKA patient depends on the severity of the clinical condition, concurrent underlying diseases, and response to therapy. Frequent reassessment of vital signs (temperature, heart rate, pulse quality, respiratory rate, respiratory effort, body weight) is imperative. Electrolytes, notably potassium, phosphorus, and magnesium should be monitored at least once to twice daily. Venous blood gas analysis and urine or serum ketones should also be checked once to twice daily to assess response to therapy.

Conclusion
Successful treatment of the DKA patient requires a multipronged approach addressing fluid therapy, insulin therapy, electrolyte imbalances, and nutrition. Fortunately, approximately 70% of patients treated for DKA survive to discharge from the hospital. Owner education is important in both short and long term treatment plans. The owner should be educated on not only the average hospitalization time (6 days) but the long term commitment if insulin administration and the commitment to long-term veterinary care.

References
Abdominocentesis
Abdominocentesis is a minimally invasive, inexpensive, diagnostic and potentially therapeutic procedure for patients with ascites. Evaluation of the fluid aids in diagnosis and helps guide treatment. Abdominal effusion is classified as a transudate, modified transudate, or exudate based on the cellularity and protein content of the fluid. Transudates (protein concentration < 25 g/l, nucleated cell count < 1000/μl (1 x 10⁹/μl)), are commonly due to causes including hypoalbuminemia and early congestive heart failure. Modified transudates (protein concentration < 35 g/l, cell count < 5000/μl (5 x 10⁹/μl)) result from increased hydrostatic pressure (right-sided congestive heart failure, left-sided congestive heart failure in cats), decreased oncotic pressure (hypoalbuminaemia) or lymphatic obstruction (neoplasia). Exudates (protein concentration > 30–35 g/l, cell count > 5000/μl (5 x 10⁹/μl), are found with causes including sepsis, feline infectious peritonitis (FIP), neoplasia, lung-lobe torsion, and pancreatitis. Along with cellularity and protein content, biochemical evaluation of the fluid for creatinine, potassium, bilirubin, lactate and glucose can aid in the diagnosis of various conditions, including uroabdomen, bile peritonitis, and septic peritonitis.

The equipment needed to perform an abdominocentesis includes clippers, antimicrobial scrub, 70% ethyl alcohol, sterile gloves, 18 – 22 gauge, 1 - 1 ½ inch needles, extension tubing, 20 – 60 ml syringes (depending on size of the patient), and sterile EDTA and red top tubes for sample collection.

To perform an abdominocentesis, the patient is placed in left lateral (to allow the spleen to fall away from midline) or sternal recumbency. Using the prepared abdominocentesis site, the needle is inserted through skin and abdominal musculature into the abdominal cavity. This can be performed with or without ultrasound guidance. If ultrasound is not available, a four-quadrant technique can be used. This procedure is accomplished by preparing 4 aseptic sites, cranial and left, cranial and right, caudal and left, and caudal and right in respect to the position of the umbilicus.

Endotracheal and transtracheal washes
Procedures including endotracheal, transtracheal, or bronchoalveolar lavage are indicated in the diagnostic evaluation of lower airway disease. The sample obtained by the procedure can be used for cytological and microbiological evaluation (bacterial, fungal, protozoal, parasitic) and non-infectious disease such as allergic airway disease, inflammatory airway disease, and neoplasia.

Equipment needed for the endotracheal wash includes general anesthesia, sterile endotracheal tube, large bore suction catheter or Salem-samp suction catheter, sterile saline, 2-3 sterile syringes, mucus-specimen trap, oxygen tubing, suction, and sterile gloves.

Equipment needed for the transtracheal wash includes sedation and/or local analgesia with 2% lidocaine, clippers, scrub, 18 gauge sampling catheter, sterile saline, 2-3 sterile 10 cc syringes, and sterile gloves.

Approximate injection volumes of sterile saline include:
- Cat: 2-3 ml per attempt, start with lowest amount, up to 5 ml
- Small Dog: 2-4 ml per attempt, up to 5-20 ml based on size of dog
- Large Dog: 3-5 ml per attempt, up to 20-50 ml based on size of dog

To perform either an endotracheal wash or transtracheal wash, the clinician prepares the equipment prior to the procedure. This ensures that before sedation or anesthesia the clinician is able to perform the procedure quickly and efficiently to reduce patient morbidity. For example, prior to the endotracheal wash procedure, the sterile syringes are pre-loaded with sterile 0.9% NaCl, the oxygen tubing is connected to the suction device, and the mucus specimen trap and suction catheter are connected. Once the procedure set-up is complete and the veterinary team is ready, the assistant intubates the patient with a sterile endotracheal tube. Prior to contaminating the endotracheal tube by connecting the tube to the anesthesia machine, the endotracheal wash procedure is performed. The procedure itself is performed by inserting the catheter down the endotracheal tube until it cannot pass any further. The preloaded saline syringes are used to flush the saline down the tube. Once the saline is inserted, the assistant gently coupages the chest while the veterinarian is applying suction to the catheter. The procedure continues until an adequate sample is obtained provided the patient is not decompensating. Immediately after obtaining a sufficient sample the patient is connected to the anesthesia machine to provide 100% oxygen. The sample obtained is then submitted for cytology and aerobic culture, +/-mycoplasma and fungal.

To perform a transtracheal wash, the ventral neck is clipped and scrubbed, notably between two rings of cartilage 3-4 rings below the larynx. Along with manual restraint, chemical restraint can reduce stress and anxiety during the procedure. A local block combined with an opioid or benzodiazepine is considered for mild sedation. When inserting the sampling catheter, the bevel of the needle should be faced downward. The needle is advanced through the skin on the midline of the neck through two cartilage rings, perpendicular to the trachea into the tracheal lumen. As you enter the trachea, you will feel a pop. Once seated within the tracheal...
lumen, the needle is advanced 2-3 mm further to ensure appropriate positioning. The sampling catheter is advanced through the needle completely into the tracheal lumen. Once the catheter is completely advanced, the needle is pulled back until it is no longer in the trachea. Once it is completely exteriorized, the needle guard is attached to reduce the risk of tracheal laceration. The next step is to inject sterile saline, cuppage the patient, and aspirate with the attached syringe to obtain your diagnostic sample. The procedure is repeated often 1-3 times to achieve an adequate sample. The sample obtained is then submitted for cytology and aerobic culture, +/- mycoplasma and fungal.

Thoracocentesis

Thoracocentesis is a common emergency procedure to remove fluid or air from the thoracic cavity. Patients that present in respiratory distress should be evaluated for their breathing pattern. Clinical signs may include a short and shallow restrictive breathing pattern, paradoxical breathing pattern, increased respiratory rate, orthopnea, and an abdominal component to respiration. Thoracic auscultation may warrant thoracocentesis includes decreased or dull lung sounds ventrally (pleural effusion) or dorsally (pneumothorax). If the patient presents in respiratory distress with a short and shallow, restrictive breathing pattern, dull and muffled lung and heart sounds, and suspicion of pleural space disease, a thoracocentesis should be considered.

The equipment needed to perform a thoracocentesis includes clippers, antimicrobial scrub, 70% ethyl alcohol, sterile gloves, 18 – 22 gauge, 1 - 1 ½ inch needles, extension tubing, 20 – 60 ml syringes (depending on size of the patient), and sterile EDTA and red top tubes for sample collection.

To perform a thoracocentesis, the patient should be restrained in sternal recumbency. The procedure will vary slightly depending on the cause for pleural space disease. If air is present, a pneumothorax, the dorsal 1/3 of the chest will be prepared. If fluid is suspected, the ventral 1/3 of the chest will be prepared. The appropriate area of the chest wall is prepared by making a large (approximately 10 cm x 10 cm) window, clipped and aseptically scrubbed. Unless directed by ultrasound guidance to a more specific area, blind thoracocentesis is performed between rib spaces 7-11. The needle should be inserted in the intercostal space cranial to the rib, avoiding the blood supply and nerves found caudal to the rib.

Thoracostomy tube placement

A thoracostomy tube is most commonly considered on the emergency basis when ongoing accumulation of air or fluid requires frequent re-aspiration.

For large bore thoracostomy tube placement, the equipment required includes: clippers, antimicrobial scrub, 70% ethyl alcohol, 2% lidocaine, 3 ml syringe, 22 gauge needle, sterile surgical pack, sterile drapes/towels, trocar-type chest tube (Argyle), 2-0 nylon suture, bandage material, sterile gloves, 3-way stopcock, Christmas tree adapter, wire, wire cutters, and antimicrobial ointment.

To place a large bore thoracostomy tube, the patient is placed in lateral recumbency under general anesthesia. The entire lateral thorax is clipped, aseptically prepared, and draped to deliver a sterile field.

For local analgesia, 2% lidocaine is used to infiltrate the dermis and intercostal muscle at the intercostal space where you will be entering the chest, often the 8th-10th intercostal space. Following lidocaine infiltration, a small incision is made through the skin over the 10th intercostal space in the dorsal third of the chest. Through this incision, the chest tube is inserted into the subcutaneous space. Using a curved tip Carmalt forcep or Kelly hemostats, a tunnel is made through the subcutaneous space to the level of the 8th intercostal space. Using the instrument, force is placed on the tips to bluntly enter the pleural space. Once the tip of the instrument enters the pleural space, it is not removed, rather used to guide the chest tube into the pleural space. The trocar of the chest tube is removed once the tube is guided into the thoracic cavity. The chest tube is clamped prior to the complete removal of the trocar to prevent air entering the thoracic cavity. Adapters are then attached to the chest tube and secured to the chest tube with a suture or wire. The tube is secured with a purse-string suture and Chinese finger trap suture. The procedure is completed with the use of antibiotic ointment at the skin incision site, a non-adherent pad covering the incision and ultimately a gentle chest wrap for compression and securing of the tube to the patient.

While large bore chest tubes can be considered, the author has transitioned almost completely to the use of a smaller bore chest tube, specifically the Mila International® chest tube device, 14g x 20cm fenestrated chest tube catheter. This catheter can be placed easily without the use of general anesthesia via the modified seldinger technique. With the combination of an introducer/catheter, guide wire, catheter, and securing instrumentation, this chest tube has been used successfully for a variety of conditions including pneumothorax, chylothorax, pyothorax, and hemothorax.

Pericardiocentesis

Pericardiocentesis is a life saving procedure to remove effusion from the pericardial space. Pericardial effusion is abnormal fluid in the pericardial space resulting in inadequate cardiac filling, decreased cardiac output, and right heart tamponade.

Equipment needed for pericardiocentesis include clippers, antimicrobial scrub, 70% ethyl alcohol, sterile drapes, sterile gloves, electrocardiogram (ECG), ultrasound (if available), large intravenous catheter or pericardiocentesis catheter, extension set, three-way
stopcock, syringe, sampling tubes (red top and EDTA) and 2% lidocaine (both for local analgesia and preparedness if ventricular tachycardia develops.)

To perform a pericardiocentesis, the patient is placed in sternal recumbency or lateral recumbency. Anesthesia is not necessary although sedation with an opioid/diazepam combination can be helpful for mild chemical restraint. A local block with 2% lidocaine can be used to reduce discomfort as well. Cardiovascularly compromising medications such as propofol, acepromazine, and inhalant anesthesia should be avoided. Unless ultrasound guidance dictates a more appropriate location, the patient is prepared by clipping and scrubbing between the 4th and 6th intercostal space. While there is controversy as to the best side to use, the author prefers to enter the right side of the thorax. Similar to the thoracocentesis discussed above, the needle should enter cranial to the rib as the intercostal vessels and nerve runs caudal to the rib.

At the preference of the clinician, to prevent drag of the catheter through the skin a small skin stab incision can be made with a No. 11 scalpel blade. Also at the preference of the clinician, side holes can be placed in the distal portion of the pericardiocentesis catheter. If side holes are made, avoid a hole greater than 40% of the circumference of the catheter and holes directly opposite each other on the catheter, both which increase the risk of catheter weakness.

With appropriate patient monitoring including ECG, the catheter is inserted through the skin and into the pleural space. Once within the pleural space, the catheter is advanced slowly (1-2mm at a time) towards the heart while continuously monitoring the patient for discomfort and the ECG for arrhythmias. As the catheter is advanced, the clinician is watching carefully for fluid accumulation into the hub of the catheter. Typical fluid from the pericardial space will range from red to a port wine color. Once the fluid is seen within the hub of the catheter, the catheter is advanced another 1-2mm to make certain it is best seated within the pericardial space. The stylet is then removed and the catheter is connected to the extension tubing along with a three-way stopcock. Using a 10-20ml syringe, the fluid is aspirated. A sample of the aspirated fluid is to placed into a red top tube and a lavender top tube for further analysis. Specifically, the red top tube is monitored for clotting. A clot within the red top tube is a concern for trauma to the heart via the catheter and the catheter should be removed from the pericardial space. The amount of fluid obtained will vary but may be as much as 1/2 to 1 liter in a large breed dog. As they are often tachycardic on presentation, the clinician should notice a fairly dramatic decrease in heart rate within a few minutes of successful pericardiocentesis.

Central venous catheter placement

A central venous catheter is a catheter where the tip of the catheter sits in the thoracic part of the cranial or caudal vena cava and commonly placed in dogs and cats via the external jugular vein. A peripherally inserted central line (PICC) is also available, placed via the medial (cat) or lateral (dog) saphenous vein. Advantages of a central venous catheter include serial blood collection, hypertonic fluid administration (fluid osmolality > 600 mOsm/L), administration of total parenteral nutrition, and measurement of central venous pressure. Potential risks of central venous catheter placement include hemorrhage, thrombus formation, emboli, and infection.

Equipment needed to place a central venous catheter include clippers, antimicrobial scrub, 70% ethyl alcohol, sterile gloves, bandage material, antimicrobial ointment, 14, 16, or 18 gauge Venocath catheter, 3 ml syringe(s) with heparinized saline to use as flush, suture, and gauze 4 x 4s, and the central venous catheter kit.

A central venous catheter is most often placed via the Seldinger, or "over-the-wire" technique. Multi-lumen systems are frequently used to allow for infusion of multiple fluids, medications, CVP measurement, and parenteral nutrition. Surgivet, Abbott, and Arrow make over-the-wire catheter kits which have components that include the introduction catheter, vascular dilator, wire, wire introducer, and central catheter.

The central venous catheter is placed with the patient in lateral recumbency with the assistance of chemical restraint. Similar to other critically ill patients, this can often be easily accomplished with the use of a local block combined with an opioid or benzodiazepine. The lateral cervical area is clipped and aseptically prepared from the ventral ramus of the mandible caudally to the thoracic inlet and dorsally and ventrally to the respective midlines. Sterile drapes are then placed over the aseptically prepared area. The assistant extends the head and neck with the front legs pulled caudally. If available, a second assistant or the clinician occludes the jugular vein for visualization. Once the site is prepped, the provided 18 - 20 gauge over-the-needle catheter is inserted into the jugular vein. Once seated within the jugular vein, the stylet is removed. While monitoring the ECG for arrhythmias, the provided guide wire is inserted through the catheter into the jugular vein. Never let go of the wire. Repeat it with me, never let go of the wire. Once a majority of the wire is inserted via the catheter into the jugular vein, the over-the-needle catheter is removed, leaving the wire seated within the jugular vein exiting through the skin. The vascular dilator is fed over the wire into the vessel using a twisting motion, creating a larger hole in the vessel to prepare for placement of the multi-lumen catheter. Once the vascular dilator is bluntly used to create the larger diameter hole in the jugular vein, it is removed, again leaving the wire within the jugular vein, exiting through the skin. The large hold created is more likely to bleed and sterile gauze can be used to apply gentle pressure to the site. Once the vascular dilator is removed, the large multi-lumen catheter is fed over the wire into the jugular vein. Again, never lose the wire – keep this in your hand at all times. Once the multi-lumen catheter is fed into the jugular vein, the wire often has to be fed backwards.
through the most distal port of the catheter before the catheter can be completely seated within the jugular vein. The catheter is then secured with suture and wrapped with a gentle bandage.

**Intraosseous catheter placement**

Intraosseous catheters are considered when intravenous access is difficult or impossible due to hypovolemia, hypotension, or (small) patient size. Intraosseous catheters can be used for crystalloids, colloids, blood products, and medications. Placement of an intraosseous catheter is simple in pediatrics and slightly more complicated in larger and older patients.

The equipment needed for placement of an intraosseous catheter include clippers, antimicrobial scrub, 16 - 18 gauge bone marrow needle (or spinal needle, or 16 - 20 gauge needle), 2% lidocaine, heparinized saline flush, antimicrobial ointment, T-set connector, white tape, and nylon suture.

While there are several possible locations for IO catheter placement, the author prefers placement in the femur. The greater trochanter and the trochanteric fossa are palpated with the leg held in adduction to avoid the sciatic nerve. The desired needle is inserted through the skin to the level of the trochanteric fossa. The needle should be placed parallel to the length of the femur. The needle is rotated in a back and forth in a twisting motion, applying constant pressure to drive the needle into the cortex of the bone. Once the needle is seated within the cortex of the femur, movement of the leg should move the needle in the appropriate direction. A second test for appropriate placement is to flush the needle with sterile heparinized saline. If there is resistance, it may be necessary to rotate the needle 90–180 degrees to make certain the bevel of the needle is not lodged against the wall of the cortex. If the flush results in a swelling along the shaft of the femur, the catheter has penetrated the femoral cortex and should be replaced. Following successful placement, the needle is secured with suture and bandaged.

Potential complications of intraosseous catheter placement include osteomyelitis, bone trauma, and leakage of injected material into subcutaneous tissues.

**Nasal and nasopharyngeal oxygen catheter placement**

Placement of a nasal oxygen catheter is a quick and easy way to provide supplemental oxygen to the hypoxic patient. Nasal oxygen catheters are easy to maintain and often well tolerated.

The equipment required for nasal oxygen catheter placement includes a red rubber catheter (or similar tubing), 3-0 nylon suture, 2% lidocaine, sterile lubricant, 1 ml syringe case, flexible extension tubing, oxygen source, bubbler for humidification, and an Elizabethan collar.

In preparation for placement, the catheter is measured from the end of the nostril to the medial canthus of the eye. The tube that is then at the level of the tip of the nose is marked with a permanent marker to indicate how far the catheter is advanced during placement. For nasopharyngeal oxygen catheter placement, the tip of the tube is measured from the ramus of the mandible to the tip of the nose. Once measured, 0.5 - 1 ml of dilute 2% lidocaine can be instilled in the patient's nostril. The tip of the tube is lubricated with sterile lubricant and directed ventrally and medially, advanced to the level of the tube marked. Once the tube is in place, it is secured with suture (or staples). Oxygen flow rates of 50 - 100 ml/kg/minute are usually well tolerated making sure to humidify the oxygen source.

**Temporary tracheostomy tube placement**

A temporary tracheostomy tube is considered for severe upper airway obstruction, upper airway trauma, laryngeal or pharyngeal collapse, or when long-term positive pressure ventilation is planned.

Equipment required for tracheostomy tube placement includes: sterile surgical pack, sterile towels/drapes, small gelpi retractors, nylon suture, Shiley tracheostomy tubes, umbilical tape, hydrogen peroxide, sterile bowls, sterile pipe cleaners, sterile bottle brush, and sterile long cotton swabs.

To place a tracheostomy tube, the patient is placed under general anesthesia. The patient is placed in dorsal recumbency to expose the ventral neck. The ventral neck is clipped from the ramus of the mandible caudally to the thoracic inlet and laterally extending greater than 50% of the diameter of the neck. The ventral neck is aseptically clipped, scrubbed, then draped. The larynx is palpated and a skin incision is made on ventral midline, caudally for several centimeters. The subcutaneous tissues are dissected and sternohyoideus muscles are visualized. These layers are bluntly dissected using curved hemostats and Metzenbaum scissors. Gelpi retractors are used retract the skin and underlying tissues for adequate tracheal visualization. Once the trachea is visualized, a horizontal incision between tracheal rings is made with a Number 11 scalpel, between the 4th and 5th or 5th and 6th tracheal rings. The horizontal incision should not extend more than 50% of the circumference of the trachea. A stay suture should be placed around the tracheal ring at the cranial and caudal edges of the incision to allow retraction of the incision for placement (and re-placement) of the tracheostomy tube. The tracheostomy tube can be secured with umbilical tape and a light wrap. While opinions may differ, the author does not recommend suturing the tracheostomy tube directly to the neck. The tracheal ring stay sutures are left in place until the tracheostomy tube is no longer required.
References


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Williams J, Leveille R, Myer CW. Imaging modalities used to confirm diaphragmatic hernia in small animals. Comp Cont Ed Pract Vet 1998;20:1199-1209.


While it is challenging to review all of emergency medicine in a 1 hour lecture, important pearls are easy to discuss and review! This lecture will focus on clinical tips, common treatments, and practical information to help you fine tune your clinical knowledge in different treatment areas.

**Fluids, fluids, and more fluids. Is it important to have 8 different fluid types on the shelf?**
Does it really make a difference if you stock Norm-R, Plasmalyte, LRS, or 0.9% saline? There is a common misconception that one fluid is ALWAYS the best fluid choice. Ultimately, the answer to most situations that require fluid therapy is **perfusion**! Most isotonic crystalloids are safe in replacing what is missing, whether you are treating a patient with dehydration, shock, or both! As a result, it is generally safe to pick a fluid that is isotonic to your patient. This has been shown in numerous studies, notably in patients with feline urethral obstruction.

**Understanding and using the SHOCK dose of fluids!**
The “shock dose” of fluids is calculated from the estimated blood volume of the patient. This is classically considered to be 90 ml/kg in dogs and 60 ml/kg in cats. In most cases, when a fluid bolus is needed, the entire shock dose of fluids is not administered; rather smaller aliquots are used followed by patient re-assessment. For example, rather than give a dog 90ml/kg of an isotonic crystalloid in shock, typically 1/3 or ¼, or approximately 20-30ml/kg is administered. Re-assessment will look for improvements in patient characteristics and vital signs including heart rate, blood pressure, mentation, capillary refill time, and pulse quality.

**Sepsis is sepsis. And Cats are small dogs. Truth? Or far from it?**
I always say that cats are illiterate. They never read the book. They have to be different and for this reason, cats with sepsis are often quite different than dogs with sepsis. In dogs, we often see the classic hyperdynamic state of shock with brick red, injected mucous membranes, tachycardia, and bounding pulses. Cats on the other hand are more commonly found with sepsis to have signs including lethargy, pale mucous membranes, tachypnea, bradycardia, weak pulses, and hypothermia. As I was taught in school…septic cats are **cold, flat, and bradycardiac**! A stressed, sick cat in the hospital that is bradycardic should be a major concern and prompt immediate attention!

**Steroids. steroids. and…more steroids?**
Steroids and SHOCK are two terms that almost never should be used in the same sentence. While you can open older formularies and see traditional “shock doses” (e.g., dexamethasone sodium phosphate - DexSP 4–6 mg/kg), these are no longer accepted by most emergency and critical care specialists.

We have moved away from giving steroids in many situations including most cases of shock and trauma due to the potential adverse effects including gastric ulceration hyperglycemia, and delayed wound healing. Moreover, new doses of steroids have been evaluated and used. We no longer give 4-6mg/kg of DexSP! 
- **Antinflammatory doses of DexSP** - 0.1 mg/kg
- **Immunosuppressive doses of DexSP** 0.2-0.25 mg/kg IV q 12 to 24 hours.

It is also important to remember that DexSP is approximately 8 to 15 times stronger than prednisone! Would you give 40 mg/kg of prednisone to a patient that was hit by a car?

Specifically related to head trauma, steroids should be avoided! There are no experimental or clinical studies demonstrating a clear benefit of steroids in head trauma. Not only has there been no benefit shown, but the “CRASH” (Corticosteroid Randomisation After Significant Head injury) study demonstrated that overall mortality was statistically higher in patients who were treated with steroids! Aside from the concerns of gastric ulceration hyperglycemia, and delayed wound healing studies have shown that human patients with head trauma and hyperglycemia have a poorer return to cognitive function than do euglycemic patients. Elevated glucose concentrations provide a substrate for anaerobic metabolism and glycolysis in the brain, worsening brain perfusion via the accumulation of the by-product, lactic acid.

**Should ventricular premature complexes (VPCs) should always be treated?**
Anti-arrhythmic drug therapy is not benign, and like many medications you must evaluate the risk:benefit ratio before administration. Moreover, antiarrhythmics can also be arrhythmogenic in origin. In general, there are several principles to consider before using lidocaine or procainamide for VPCs.
a. Poor perfusion: cold peripheral limbs, a prolonged capillary refill time (CRT) > 2.5 seconds
b. Sustained tachycardiac > 170 (dogs) or >240 (cats).
c. The electrocardiogram (ECG) shows changes of malignancy such as “R on T” phenomenon
d. Other causes for the arrhythmia (systemic / metabolic / pain) have been ruled out / treated.

DOSES
- Lidocaine [Dogs: 1-4 mg/kg, IV, slow, followed by a constant rate infusion (CRI) 30-80 mcg/kg/min]:
- Procainamide (Dog: 6-8 mg/kg IV slow over 5 minutes, followed by a CRI 25-40 mcg/kg/min): If lidocaine appears to be ineffective, can consider procainamide.

Are you relying too heavily on technology, not using simple tests?

PCV/TP
Similar to the Ford TV commercial, **AND** is better. Who wants a PCV or TP? Having a PCV without the TP is like having to choose a Bed or Breakfast rather than a Bed and Breakfast. Evaluation of the PCV and TP together can help diagnose or fine tune your differential diagnoses.

You now are asking…”why are you talking about TP (total protein) and not TS (total solids)?” Modern refractometers measure total protein (TP) by the refraction produced by the total dissolved solids in plasma and have been calibrated to subtract 2.0 g/dL of the expected non-protein solids in plasma. These non-protein solids include urea, triglycerides, cholesterol, and glucose. So, TS and TP are different and should not be used interchangeably.

Here is a chart with a few examples of how PCV and TP together can help direct your diagnosis and treatment plan:

<table>
<thead>
<tr>
<th>Packed Cell Volume and Total Solids</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PCV/TP</strong></td>
</tr>
<tr>
<td><strong>PCV/ Normal TP</strong></td>
</tr>
<tr>
<td><strong>PCV/ TP</strong></td>
</tr>
<tr>
<td>Hemoconcentration</td>
</tr>
<tr>
<td>- Hemolytic anemia</td>
</tr>
<tr>
<td>- Anemia of chronic disease</td>
</tr>
<tr>
<td>- Pure red blood cell aplasia</td>
</tr>
<tr>
<td>- Blood loss</td>
</tr>
<tr>
<td>- GI</td>
</tr>
<tr>
<td>- Body cavity (abdominal, thoracic, etc)</td>
</tr>
<tr>
<td>- Protein Losing Enteropathy (PLE)</td>
</tr>
<tr>
<td>- Protein losing nephropathy (PLN)</td>
</tr>
<tr>
<td>- Acute blood loss</td>
</tr>
<tr>
<td>- Liver disease / failure</td>
</tr>
</tbody>
</table>
| Before breaking the hematocrit tube for evaluation on the refractometer, make sure to use all of the tools available to you! Evaluation of the color of the serum in the hematocrit tube can also help. A yellow discoloration of the serum within the hematocrit tube can indicate icterus or hemolysis. If the patient has a low packed cell volume in the hematocrit tube with yellow serum, for example your 5 year old Cocker Spaniel, immune mediated hemolytic anemia would be a concern. If the patient is not anemic, but is a 5 year old obese domestic shorthair cat that has been anorexic for 5 days, hepatic lipidosis would be a concern. Other abnormalities when evaluating the hematocrit tube include a buffy coat for a gross assessment of a white blood cell count elevation and lipemia which can be seen in sick patients with severe hypothyroidism or pancreatitis.

**Blood glucose concentration**
Prompt recognition and treatment of both hyperglycemia and hypoglycemia is essential to reduce morbidity and mortality in our patients. There are many cage-side portable blood glucose meters and point-of-care analyzers that provide reliable real time assessment for our patients. Patients that present hypoglycemic may have clinical signs including weakness, lethargy, tremors or seizures. Patients that present hyperglycemic have less reliable clinical signs, often suffering from the underlying cause for the hyperglycemia itself. Hyperglycemia may be found and historical information (polyuria, polydypsia, polyphagia, and weight loss) may help confirm a diagnosis of diabetes mellitus. On the other hand, hyperglycemia found in specific diseases such as head trauma and critical illness has been more recently found to be a negative prognostic indicator with a worse morbidity and mortality. In both human and veterinary medicine, studies have been performed ultimately concluding that tight glycemic control in critical illness reduces morbidity and mortality and prolonged hyperglycemia should be addressed in these patients. Thus, blood glucose monitoring and management in emergent patients can be quite helpful.

<table>
<thead>
<tr>
<th>Blood glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoglycemia</td>
</tr>
<tr>
<td>- Insulin overdose</td>
</tr>
<tr>
<td>- Hepatic failure</td>
</tr>
<tr>
<td>- Sepsis</td>
</tr>
<tr>
<td>- Insulinoma</td>
</tr>
<tr>
<td>- Paraneoplastic</td>
</tr>
<tr>
<td>- Pediatric hypoglycemia</td>
</tr>
<tr>
<td>- Portosystemic shunt</td>
</tr>
<tr>
<td>- Adrenal insufficiency</td>
</tr>
<tr>
<td>- Toxicity (xylitol)</td>
</tr>
</tbody>
</table>

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AZO / BUN

Reagent test strips (Azo stick®) are used to estimate BUN and provide a semi-quantitative estimation of the blood urea nitrogen concentration. Test strip ranges include 5-15 mg/dl, 15-26 mg/dl, 30-40 mg/dl, and 50-80 mg/dl. The normal Azo is considered less than 26mg/dl. Along with PCV/TP, history, and physical examination, the presence of azotemia will need further assessment to determine if the cause is pre-renal, renal, or post-renal in nature. Provided the patient does not suffer from a urinary obstruction, indicating a post renal cause for azotemia, ideally, a urine specific gravity should be obtained prior to starting IV fluids to help differentiate renal from pre-renal azotemia. Other causes for AZO/BUN elevation include gastrointestinal hemorrhage and following a high protein meal.

Ancillary testing

Depending on your background, the MDB (minimum database) can quickly turned into the EDB (extended database). I know what you are saying…let’s not get crazy! But it really is not much more work. An extended database is a minimum database plus additional testing. The minimum database discussed includes a packed cell volume (PCV), total protein (TP), blood glucose, and dipstick BUN/Azo. Additional testing that converts the MDB to the EDB includes a blood smear, venous blood gas, lactate and electrolytes including sodium, potassium, chloride, and ionized calcium. These additional tests in the EDB help provide a more rounded metabolic assessment of the patient and can better assist the clinician rapidly determine the underlying cause.

Temperature

When examining a patient, the core body temperature (often rectal) should be a standard part of the baseline examination and vital signs. An elevated temperature is more commonly seen as compared to a low body temperature, although the clinician must differentiate between exogenous and endogenous sources regardless of the change in body temperature. Whereas hyperthermia results often from an exogenous heat source, a true fever is caused by an endogenous heat source. Fever, while not a specific disease itself is a common clinical manifestation of disease. Common inciting causes include infection, inflammation, neoplasia, and drug administration. Usually, the underlying cause for fever is easily explained. Unfortunately, there are also cases where the underlying cause can not be easily found, thus a “Fever of Unknown Origin” (FUO). In human medicine, FUO was originally defined as an illness of more than 3 weeks’ duration with a temperature higher than 101°F (38.4°C) on several occasions, after 1 week of hospitalization and evaluation. While there is not a general consensus for a definition in our veterinary patients, FUO is considered when an obvious cause for the fever has not been found after thorough examination and routine diagnostic tests.

BODY WEIGHT

In a land of calculations, equations, and complexity, fortunately, we can make body weight evaluation quite simple! 1L = 1KG. If a patient loses 1 liter of fluid as a result of hydration, they will lose 1KG of body weight. Moreover, because we can calculate dehydration (kg weight × % dehydration), we can have a very easy way to estimate appropriate weight loss of weight gain in our clinical patients. This simple, cost neutral evaluation is important for all hospitalized patients, especially if they are receiving fluid therapy.

For example, if a 30 kg dog is 10% dehydrated:

Dehydration: 30 kg X 0.1 (percent dehydration) X 1000 mls = 3000 ml = 3 L

After performing this simple calculation, this 30kg dog has a 3L deficit of fluids. Not only do you know what you need to replace, but you also know that once the patient has received their fluid therapy, they should weigh 33 kg after complete hydration.

References

Trauma resulting in wounds or injury are common emergencies in small animal patients. Traumatic injuries may occur secondary to motor vehicle accidents, falls from heights, gunshot wounds and animal-animal interactions such as dog or cat fights. Some traumatic injuries are minor, such as a single bite wound, and are easily addressed and fixed. However, trauma can be extremely serious affecting multiple organ systems either directly or indirectly. Therefore, a thorough and systematic approach to the emergency evaluation and treatment of a traumatically injured animal is essential.

Early restoration of tissue perfusion and maintenance of tissue oxygenation is key to improving patient survival from trauma. Oxygen content of the blood and delivery of oxygen to cells in the body is critical for sustaining life. Under normal conditions, neural, humoral, local myogenic and metabolic autoregulatory mechanisms combine to continuously adjust vascular resistance and tissue blood flow to meet oxygen requirements in proportion to tissue needs. In trauma, an accumulating tissue oxygen debt either due to decreased tissue perfusion and/or hypoxemia, can trigger processes that lead to a systemic inflammatory response (SIRS) and multiple organ dysfunction syndrome (MODS). Therefore, the initial approach and fluid resuscitation of all traumatically injured patients should be aimed at improving and restoring tissue perfusion and oxygenation.

**General approach to the traumatically injury patient**

Oxygen delivery is dependent on cardiac output (CO), hemoglobin saturation with oxygen, and total hemoglobin concentration:

Therefore, an initial approach to the patient with traumatic injury should be focused on ensuring adequate saturation of hemoglobin with oxygen, adequate hemoglobin concentration, and improving cardiac output by restoring the intravascular volume.

**Ensuring adequate oxygen saturation**

Insuring adequate oxygen saturation of hemoglobin requires an assessment of the patient’s respiratory system and immediate correction of abnormalities that may contribute to hypoxemia. Is the patient breathing? If not, the patient should be immediately intubated and positive pressure ventilation should be instituted. In all other situations, oxygen supplementation, either by flow-by or mask, should be immediately administered until an adequate assessment of the patient’s oxygenation status can be established either through arterial blood gas analysis (ideally) or through pulse oximetry. The physical examination should include assessment of mucous membrane color, respiratory rate and effort, heart rate and thoracic auscultation. Changes in these physical examination variables may alert the emergency clinician to the presence of hypoxemia.

Pleural space disease, such as pneumothorax or hemothorax, is common following traumatic injuries. If pleural space disease is suspected based on thoracic auscultation, immediate needle thoracocentesis should be considered prior to radiography if the patient is severely compromised. Alternatively, thoracic ultrasound (T FAST) can be used to confirm the presence of fluid or air prior to performing thoracocentesis. An indwelling chest tube may need to be placed but generally only if the patient is requiring repeated thoracocentesis and the volume of fluid or air is not substantially decreasing. A tension pneumothorax is life threatening as the accumulation of air within the thoracic cavity affects venous return and ultimately cardiac output. Patients with tension pneumothorax may have a barrel-chested appearance and are likely to be in severe respiratory distress. In cases where cardiopulmonary arrest is imminent, a small thoracotomy may be performed to release the air.

Other common thoracic injuries contributing to hypoxemia in the traumatically injury patient include penetrating wounds, pulmonary contusions, flail chest and diaphragmatic hernia. Penetrating wounds resulting in pneumothorax should be covered (sealed with KY jelly and covered with a bandage). The presence of pulmonary contusions should be suspected in patients that have crackles auscultated. Supplemental oxygen therapy and limited volume fluid resuscitation should be considered in these patients (see below). In patients with flail chest, oxygen supplementation and pain management are essential. The flail portion of the chest should be placed in the down position when the patient is in lateral recumbency to minimize movement of the flail section which can exacerbate pain and contribute to further intra-thoracic injury. Future surgical management will be necessary once the patient is stabilized and other injuries are assessed. Traumatic diaphragmatic hernia may lead to severe compromise of the respiratory system depending on other concurrent thoracic injuries and the volume of organs within the thoracic cavity. Emergency surgery to reduce the herniated organs and repair the diaphragmatic defect is indicated once the cardiovascular system has been stabilized.

**Optimizing cardiac output**

The optimization of cardiac output is essential to the maintenance of adequate oxygen delivery. The most common cause of decreased cardiac output in traumatically injury patients is hypovolemia secondary to hemorrhage. Loss of 10-12 ml/kg of blood (<15% of blood volume) results in only mild changes in tissue perfusion parameters (such as tachycardia) and blood pressure may be normal or
even elevated. However, as increasing amounts of blood are lost (>15% of blood volume), clinical signs of shock become more apparent.

Initial assessment of cardiac output and circulating blood volume includes evaluation of the animal’s mucous membrane color, capillary refill time, heart rate and pulse quality. Pale or grey mucous membranes, prolonged capillary refill time, elevations in heart rate (heart rate > 120 bpm) and weak peripheral pulses indicate inadequate perfusion. Absent pedal pulses is very specific for diagnosing hypotension (the pedal pulses are lost when the blood pressure is less than 55 mmHg in dogs and less than 70 mmHg in cats). The measurement of arterial blood pressure may provide a more objective measurement of tissue perfusion. This can be performed either directly through the placement of an arterial catheter or indirectly by Doppler or oscillometric techniques. A measurement of blood pressure should always be part of the initial survey of a trauma patient. It is important to recognize that it is possible for the blood pressure to be normal or high in animals with early compensated shock and these patients are at risk of further blood loss and decompensation. Recently, the utility of shock index (SI) which is the ratio of heart rate to systolic blood pressure has been evaluated in dogs and an SI > 1.0 was found to be both sensitive and specific for diagnosing blood loss. Finally, an ECG should be performed during the initial survey to assess heart rate and evaluate for the presence of cardiac arrhythmias.

A large bore, short, intravenous catheter should be placed immediately for intravenous fluid and drug administration. This catheter is typically placed in the cephalic vein due to its ease of accessibility, however, other sites could be used such as the saphenous or jugular veins (caution: the jugular veins should be avoided in patients with suspected traumatic brain injury). If intravenous access cannot be obtained, a venous cut down can be done to facilitate catheter placement or an intraosseous catheter can be used. At the time of catheter placement, blood should be drawn for packed cell volume (PCV), total protein (TP), venous blood gas, and electrolytes analysis (when available). Hemorrhage should be suspected in a patient with a low PCV and low TP but also may be occurring in a patient with a normal PCV with concurrent low total solids as a result of splenic contraction (dogs only). In these cases, a low total solids (<6 g/dL) may be more specific than the actual PCV for active or previous hemorrhage. A blood lactate concentration is especially useful in assessment of tissue perfusion. Blood lactate concentration will increase in states of decreased oxygen delivery when cellular metabolism switches from aerobic to anaerobic metabolism. Increased lactate concentration greater than 2.5 mmol/L (or if unavailable, an increased base deficit and metabolic acidosis or decreased CO2) may provide additional confirmation of decreased tissue perfusion.

An additional part of the initial assessment of the traumatized animal should include an AFAST (abdominal focused assessment with sonography for trauma) examination to assess for abdominal blood loss which may be contributing to inadequate tissue perfusion. The use of the FAST technique performed by veterinary emergency clinicians in dogs following motor vehicle accident was first described by Boysen et al. This ultrasound examination consists of 2 ultrasonographic views (transverse and longitudinal) at 4 sites with the dog ideally placed in left lateral recumbency: caudal to the xiphoid process, cranial to the pelvis, right flank, and left flank caudal to the ribs at the most gravity dependent location of the abdomen with the goal of detecting free abdominal fluid. Using this technique, clinicians are able to easily and accurately identify free abdominal fluid. The gravity dependent view tends to be the most common location where fluid is identified followed by the view of the xiphoid region. It is this author’s opinion, that the use of AFAST as part of the initial patient assessment, in addition to physical examination findings and blood testing results, may help guide fluid therapy (see below) and direct future additional diagnostic testing such as abdominocentesis. Serial AFAST exams should be done during and following cardiovascular stabilization to assess for worsening effusion or in cases in which the AFAST was initially negative but the patient was cardiovascularly unstable.

A complete and thorough physical examination may not be able to be performed initially but once patient stabilization has occurred a secondary assessment, with particular attention to the neurologic system, renal system, and musculoskeletal system, should always be performed.

Fluid therapy

Based on our understanding of the pathophysiology of trauma, the goal of fluid therapy should be to restore adequate tissue perfusion and oxygenation. The institution of rapid fluid therapy is essential especially in patients suffering from severe hemorrhage where >30% of the blood volume is lost (grades III-IV hemorrhage).

Generally, a balanced isotonic crystalloid solution is chosen for initial fluid resuscitation of patients with inadequate tissue perfusion. Crystalloids are readily available and inexpensive compared to other fluid therapy options (such as whole blood). An initial fluid therapy plan consists of administering aliquots of a shock bolus (dog: 90 ml/kg/hr; cat: 40-60 ml/kg/hr) of crystalloids usually 20-40 ml/kg. As a general rule, crystalloids should be administered in a 3:1 ratio compared to the volume of blood lost due to rapid redistribution to the interstitial and intracellular spaces. An initial positive response to fluid therapy followed by recurrence of signs suggestive of decreased tissue perfusion may occur secondary to this redistribution of fluids or through ongoing hemorrhage. As fluids are administered, the patient should be continuously monitored for improvements in mucous membrane color, capillary refill time, heart rate and pulse quality until ends points of fluid resuscitation are achieved (ie. Normalization of tissue perfusion parameters).
The administration of colloids is another fluid therapy option that may be considered in traumatically injured patients. This would be the resuscitative fluid of choice in patients who are already hypoproteinemiac (TP <4.5) or in combination with crystalloid therapy. Colloids are generally administered in aliquots of 10-20 ml/kg in dogs and 5-10 ml/kg in cats. This lower volume of colloids will cause expansion of the intravascular volume by pulling fluid from the interstitial and intracellular spaces into the intravascular compartment. In addition, colloids have a longer volume expanding effect as compared with crystalloids due to its maintenance within the intravascular space for a longer period of time. Colloids may carry some risk to the patient when used. A recently published retrospective veterinary study suggests that similar to people, the use of colloids (in this study 10% hetastarch) in dogs is associated with an increased risk of acute kidney injury and death.

Hypertonic saline (7.5%) can also be considered as part of the initial fluid resuscitation of the traumatically injured patient. On a per volume basis, hypertonic saline is able to provide a more rapid intravascular volume expansion as compared to crystalloids. Similar to colloids, hypertonic saline due to its high osmolality is able to draw fluid from the interstitial and intracellular spaces into the intravascular space. However, this effect is short-lived as fluid will redistribute back to the interstitial space. Combining hypertonic saline with a colloid will prolong its volume expanding effect. This can be done by combining one part of 23.4% concentrated sodium chloride solution (American Reagent Laboratories, INC, Shirley, NY) to two parts hydroxyethyl starch. Hypertonic saline is typically administered only ONCE at a dose of 3-5 ml/kg. However, repeated doses can be given as long as hypernatremia is not occurring. Contraindications to the use of hypertonic saline include changes in sodium concentration (especially hyponatremia where a rapid elevation in sodium could occur) and dehydration. In cases of moderate to severe hemorrhage, either crystalloids or colloids must also be given along with hypertonic saline for adequate intravascular volume expansion.

**Optimizing hemoglobin concentration**

Hemoglobin concentration is the final important determinant in ensuring adequate tissue oxygenation. Decreased hemoglobin concentration will severely limit the oxygen carrying capacity of the blood. In anemia, the extraction ratio of oxygen increases as a compensatory response. However, eventually the tissue utilization of oxygen will outpace the oxygen delivery leading to tissue hypoxia. Therefore, initial evaluation of the PCV in trauma patients should be performed to get an initial estimate of hemoglobin concentration (roughly PCV divided by 3). As stated above, the initial PCV may not reflect significant blood loss due to splenic contraction and total solids (<6 g/dL) may more accurately reflect blood loss. Therefore, if hemorrhage is suspected, the PCV should be monitored serially for changes (during and following fluid resuscitation depending on the animal’s response to therapy). There has been no specific PCV that has been identified as the “transfusion trigger” and the optimal hemoglobin concentration in animals is unknown. Therefore, clinical parameters such as tachycardia, tachypnea, change in pulse quality, depressed mentation and the presence of cardiac arrhythmias should be evaluated to help guide in the decision to give blood products. Also, the rate at which the PCV has changed should also guide transfusion therapy (i.e acute blood loss may need to be treated at a higher PCV than animals with chronic blood loss that have time to adapt). In animals in which the PCV is changing rapidly, a blood transfusion should be given before the hemoglobin concentration becomes life threatening. Ideally, packed red blood cells and fresh frozen plasma would be administered in a 1:1 ratio.

**Conclusion**

A large proportion of traumatically injured dogs survive, with a reported survival rate of 88% in the most recent large veterinary retrospective study. In this study of dogs with blunt trauma, the prevalence of polytrauma was large with 72.3% of the cases affected with the chest being the most common site traumatized. This highlights the importance of assessing the small animal patient for traumatic injuries to multiple organ systems. A systematic approach to the trauma patient which focuses on ensuring adequate tissue oxygenation, optimizing cardiac output and hemoglobin concentration in the initial management will hopefully prevent the progression to multiple organ failure and continue to improved outcome in small animal trauma patients.

**References**

Traumatic Brain Injury
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Traumatic brain injury (TBI) is a common reason for small animal patients to be presented on an emergency basis. Head injuries from blunt trauma, such as motor vehicle accidents or animal-animal interactions, can result in intracranial hypertension which may lead to temporary or permanent alterations in neurologic function. Mortality of animals with TBI ranges from 18-24% in the veterinary literature. The emergency approach to and treatment of the patient with suspected TBI involves performing a meticulous physical examination followed by a combination of extra-cranial and intracranial stabilization therapies.

Pathophysiology

Injuries to the brain resulting from head trauma are typically separated into two categories: primary injury and secondary injury. Primary injury occurs as a result of the traumatic event. Concussion, contusion (at the site of impact or in the opposite hemisphere), lacerations and hematomas are examples primary brain injury. Hematomas may occur within the brain parenchyma itself or in the subarachnoid, subdural and epidural spaces. Secondary brain injury is triggered by a series of biochemical events that ultimately trigger neuronal cell death. Factors that contribute to secondary brain injury include: excitotoxicity, ischemia, production of reactive oxygen species and nitric oxide, and cerebral lactic acidosis. Immediately after brain injury there is release of excitatory neurotransmitters that cause influx of sodium and calcium into neurons resulting in depolarization and additional release of excitatory neurotransmitters. This influx of calcium leading to neuronal depolarization overwhelms cellular mechanisms for removal and ultimately results in neuronal cell death. In addition, depletion of neuronal ATP secondary to an increase in metabolic activity results in cell death. Systemic factors, such as hypotension, hyperglycemia, hypoglycemia, hypercapnia, hyperthermia, and acid-base disturbances can also contribute to the generation and perpetuation of secondary injury. Both primary and secondary brain injury, as well as any concurrent injuries resulting in hypotension or hypoxemia, can ultimately worsen cerebral injury and lead to permanent neurologic damage.

Cerebral perfusion pressure (CPP) is the force driving blood into the calvarium providing the brain with oxygen and nutrients. CPP is defined as the difference between mean arterial pressure (MAP) and intracranial pressure (ICP). Cerebral blood flow (CBF) is a function of CPP and cerebrovascular resistance. In the “healthy” brain, CBF is maintained over a wide range of MAP (50 to 150 mmHg) via autoregulatory mechanisms. Above and below this range, CBF is directly proportional to systemic blood pressure. In the “traumatized brain”, autoregulatory mechanisms are lost either focally or globally within the brain and CBF can be entirely dependent MAP. Therefore, even small decreases in MAP (hypotension) can lead to further ischemic injury in the traumatized brain.

Intracranial pressure (ICP) is the pressure exerted between the skull and the intracranial components: blood, brain parenchyma, and cerebrospinal fluid. In increase in volume of any one of these components can lead to elevations in ICP. Normal ICP in dogs and cats is 5 to 10 mm Hg. Elevations in ICP (or intracranial hypertension) can limit CBF resulting in ischemic and/or hypoxic injury to the brain further perpetuating secondary brain injury. Severe elevations in ICP can result in brain stem compression, depressed brain function, brain herniation and death. Initially, increases in ICP may trigger the Cushing’s reflex (AKA: the central nervous system ischemic response) which is a characteristic rise in MAP and reflex decrease in heart rate. If seen in a patient with TBI, this is a sign of a potentially life threatening increase in ICP and should be treated immediately.

As little can be done about the damage that occurred to the brain as a result of the primary injury, most treatments regimens for patients with TBI focus on limiting the progression of neurologic damage that can occur as a result of secondary injury. The treatment protocols are generally aimed at maximizing MAP to maintain CBF (extracranial stabilization) and minimize elevations in ICP (intracranial stabilization).

Initial patient evaluation

As trauma to other organ systems resulting in systemic disturbances are common in patients presenting with TBI, it is important on initial patient evaluation to assess for abnormalities of the cardiovascular and respiratory systems as well as the neurologic system. Ongoing hypotension and hypoxemia resulting from trauma to these systems can ultimately worsen patient outcome in TBI.

Assessment of the cardiovascular system should include evaluation of the mucous membrane color, capillary refill time, heart rate and rhythm, and pulse quality. Hypovolemia may be present in a patient with pale mucous membranes, prolonged capillary refill time, tachycardia and weak pulses. Objective assessment of the cardiovascular system should include an ECG and blood pressure measurement. Arrhythmias seen on an ECG may be present as a result of trauma to the abdomen, chest, or secondary to brain stem compression. An elevation in blood lactate (>2.5) concentration can provide additional evidence of abnormal tissue perfusion. Evaluation of the ECG in combination with blood pressure can alert to clinician to the presence of intracranial hypertension if a Cushing’s reflex is seen (bradycardia with hypertension).
Assessment of the respiratory system should include evaluation of the respiratory rate and effort, mucous membrane color and thoracic auscultation. Objective evaluation of the respiratory system can include pulse oximetry and arterial blood gas analysis if possible. The goal in the patient with suspected TBI is to ensure adequate oxygenation and ventilation as both hypoxemia and hypercarbia should be avoided.

Neurologic assessment should include an evaluation of the patient’s level of consciousness, posture, and pupil size and response to light (brain stem reflexes). Level of consciousness can be described ranging from normal, to depressed/obtunded, stuporous and comatose. Mentation needs to be evaluated in light of the patient’s hemodynamic status, as shock can reduce the patient’s level of consciousness and pupillary light responses. Pupils that are mid-range or that are bilaterally mydriatic and do not respond to light generally indicate severe brain injury and carry a guarded to grave outcome. Anisocoria is a common finding in head trauma patients and when still responsive generally indicates less severe injury and a good outcome. Finally, assessment of motor activity and posture should be performed. Ataxia, hemiparesis or tetraparesis may result from lesions affecting the cerebral cortex, brain stem or spinal cord. The presence of cranial nerve abnormalities may help to differentiate intracranial lesions from lesions in the spinal cord. Decerebrate rigidity, which is defined as extension of all four limbs and opisthotonus, is often associated with brain stem compression. Decerebellate rigidity, which is defined as extension of the front limbs with hind limb flexion indicates a cerebellar lesion and typically these patients have a normal level of consciousness.

**Treatment**

Treatment of the patient with traumatic brain injury should be focused on stabilization of abnormalities of the cardiovascular and respiratory systems (extracranial stabilization) in addition to intracranial stabilization. Following initial patient evaluation, an intravenous catheter should be placed and blood samples drawn for packed cell volume, total protein, venous blood gas, lactate concentration, and complete blood count and chemistry screen. The placement of an intravenous catheter will allow for administration of intravenous fluids and drug therapy.

**Extracranial stabilization**

During the initial patient evaluation, supplemental oxygen should be administered until a normal oxygenation and ventilation status of the patient is assured. Hypoxemia can result from concurrent thoracic injury such as pneumothorax or pulmonary contusions. Oxygen can be administered by a variety of routes including flow-by, masks and oxygen cages. Intranasal and intra-tracheal routes should be avoided as this can lead to coughing and sneezing that can transiently raise ICP. An oxygen saturation of hemoglobin of >95% (corresponds to a PaO2 of 90mmHg) should be the minimal goal in a patient with TBI either with or without oxygen supplementation. Hypoventilation should also be avoided as elevations in arterial CO2 can lead to cerebral vasodilation that will increase cerebral blood volume that can contribute to intracranial hypertension. Monitoring of PaCO2 will help to prevent episodes of hypoventilation and it should be kept within the normal range (not to exceed 40 mmHg).

If hypovolemia is suspected based on the initial assessment of the patient’s cardiovascular system (i.e physical exam findings suggestive of shock, elevations in blood lactate concentration, systolic blood pressure < 90 mmHg), volume resuscitation should be initiated. Considerable controversy exists regarding the best fluid therapy choice in patients with traumatic brain injury. Isotonic crystalloids, hypertonic crystalloids (7.5% NaCl) artificial colloids or blood products are appropriate and acceptable options with the goal of rapid volume expansion to a MAP of 80 to 100 mmHg. When isotonic crystalloids are chosen, aliquots of a shock doses (shock dose is 80-90 ml/kg in the dog, 45-60 ml/kg in the cat) should be administered rapidly. Artificial colloids are given intravenously in aliquots of 10-20 ml/kg in the dog and 5-10 ml/kg in the cats. Hypertonic saline (7.5%) has many properties that potentially make it a superior resuscitation fluid in patients with concurrent hypovolemia and elevations in ICP. Due to its high osmolality, when given intravenously it is able to promote fluid movement from the interstitial and intracellular spaces to cause volume expansion. Because sodium does not freely cross the blood brain barrier, hypertonic saline may also pull fluid from the brain interstitium concurrently reducing ICP. The recommended dose of 7.5% sodium chloride is 3-5 ml/kg administered over 10-15 minutes. This is typically only given once but could be given again as long the blood sodium concentration has not increased over 160-165 mEq/L. After initial fluid resuscitation, ongoing fluid therapy should be given to address dehydration, ongoing losses and maintenance fluid requirements.

**Intracranial stabilization**

Intracranial stabilization should only be pursued once extracranial stabilization has occurred. Goals of intracranial stabilization should include reduction of cerebral edema, decreasing cerebral blood volume, minimizing elevation in cerebral metabolic rate and eliminating space-occupying masses.

**Therapies to reduce cerebral edema**

Reduction of cerebral edema is typically achieved through the administration of hyperosmotic agents, such as mannitol and hypertonic saline. 25% Mannitol has been shown to decrease ICP, increase CPP and CBF, and have a beneficial effect on neurologic outcome in people with head injury and is still considered the gold standard therapy for treatment of intracranial hypertension. In addition, mannitol possesses free radical scavenging properties that can help with conditions of ischemia and reperfusion, a component of secondary injury. The recommended dose for mannitol is 0.5-1.5 g/kg as a bolus over 10-20 minutes. The initial benefit of mannitol is
thought to occur secondary to plasma expansion leading to reduced blood viscosity and increased cerebral blood flow and oxygen delivery. Within 15-30 minutes of administration, the osmotic effects of this drug will promote water movement from the intracellular and interstitial spaces of the brain into the vasculature, induces an osmotic diuresis, and reducing cerebral edema. The effect of mannitol can last from 1.4 to 6 hours. This drug can be given as repeated boluses if indicated, however, a constant rate infusion should be avoided.

Hypertonic saline is a second osmotic agent that can be administered either in addition to mannitol or in the place of mannitol to reduce cerebral edema. As stated previously, hypertonic saline may have additional benefits (i.e. its intravascular volume expanding effects, immunomodulatory effects, etc) when compared with mannitol. Hypertonic saline (7.5%) is administered as a bolus of 3-5 ml/kg once over 10-15 minutes. There is accumulating evidence in the human medical literature that hypertonic saline decreases ICP faster, to a greater degree and for a longer duration than mannitol.

Other medications which have been used to reduce cerebral edema include corticosteroids and furosemide. In the past, corticosteroids have been routinely used in veterinary medicine for its anti-inflammatory effects and anti-oxidative effects. However, a large clinical trial in human patients with head trauma (CRASH II) has since shown that corticosteroid treatment was associated with a worse outcome. There is no evidence in veterinary medicine at this time of the beneficial effects of corticosteroids and they have a potential to cause harm. Currently at this author’s institution, corticosteroids are not given in patients with TBI.

Furosemide has also been used in patients with head trauma either alone or in combination with mannitol. The goal of the administration of this medication is to decrease the initial increase in intravascular volume and increase in hydrostatic pressure associated with the administration of mannitol. However administration of furosemide can potentially contribute to intravascular volume depletion and therefore reduction in MAP and CPP and is not currently recommended.

Decreasing cerebral blood volume
Techniques which have been advocated to help decrease cerebral blood volume and hence ICP include elevation of the head and prevention of hypoventilation. Patients with traumatic brain injury should have the head elevated by 15-30 degrees. This will help to encourage venous drainage which will have the effect of decreasing cerebral blood volume and ICP. This is typically performed by placing the animal on a slant board and preventing the patient from sliding down. Occlusion of the jugular veins via kinking of the neck should be avoided. As stated previously, monitoring of the respiratory status of the patient and avoidance of hypoventilation is extremely important in patients with TBI. Elevations in CO₂ can lead to cerebral vasodilation and increased in cerebral blood volume. In cases of acute increases in ICP, short term hyperventilation (arterial CO₂ 25-35 mmHg) may be used to decrease cerebral blood volume and ICP.

Minimizing cerebral metabolic rate
Minimizing cerebral metabolic rate is an important component to the management of patients with TBI as it reduces oxygen and nutrient requirements of the brain and may help to minimize secondary injury. Seizures should be tightly controlled with anticonvulsants and about 10% of dogs with TBI will develop seizures. Hyperthermia, which may be caused by direct damage to the thermoregulatory center or from excessive vocalization and paddling, should be avoided. Generally, analgesics to minimize excitement and paddling are recommended. In people, currently therapeutic hypothermia is being used to minimize cerebral metabolic rate in TBI. Current evidence in people suggests that hypothermia does not appear to affect mortality but it is associated with a 46% increased chance of a good neurologic outcome with better outcomes seen with >48 hours of cooling.

Prognosis
Prognosis can be varied in small animal patients with traumatic brain injury. Initial neurologic status as well as response to stabilization therapy may help to predict outcome. The Small Animal Coma Scale or modified Glasgow coma scale score (MGCS) was developed to assess the impact of brain injury using an assessment of motor activity, level of consciousness, and brain stem reflexes. This scale has been evaluated retrospectively in dogs and scores correlate with 48 hour outcome. In the most recent study of TBI in dogs, a MGCS of <11 was both specific and sensitive for predicting outcome. Therefore, both an MGCS as well as serial Coma Scale scoring may help to provide prognostic information to assist owners of animals with TBI.

References
Urethral Obstruction: Emergency Treatment and Stabilization
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Urethral obstruction (UO) is a life threatening complication of feline lower urinary tract disease in male cats. This condition is a common reason for cats to present on an emergency basis and accounts for over 9% of the feline emergency cases seen in the ER at the University of Pennsylvania. Cats may be hemodynamically stable if the obstruction has only been present for a short time or they may be having life threatening cardiac arrhythmias from elevations in blood potassium. Therefore, prompt recognition and relief of the urethral obstruction is essential as cats can die from hyperkalemia and metabolic acidosis if urine flow is not re-established within 24-48 hours. With appropriate and timely treatment, the short term prognosis for cats with urethral obstruction is excellent, however, recurrence of urethral obstruction may occur and ranges in the veterinary literature from 14% to 30%.

Pathophysiology of urethral obstruction
Male cats are much more likely to develop urethral obstruction due to their long and narrow urethra and small urethral opening compared to the female cat. The obstruction of the urethra can occur either secondary to a physical obstruction with plugs or calculi (less commonly strictures or neoplasia) and/or secondary to a mechanical obstruction where urethral spasm and edema are thought to play a role. Urethral plugs are considered to be aggregates of proteins, crystals, white blood cells and red blood cells surrounded by amorphous material. Recent studies have shown that urethral obstruction is associated with either urethral plugs (18%) or idiopathic causes (53%) in the majority of cases whereas uroliths account for 29% of cases. Idiopathic cystitis (IC) is thought to be the underlying condition leading to the formation of urethral plugs and/or urethral spasm and inflammation resulting in obstruction. The pathogenesis of IC is complex and incompletely understood. However, multiple abnormalities of the bladder, central nervous system and hypothalamic-pituitary-adrenal axis are thought to contribute to the clinical manifestations of IC.

When complete obstruction of the urethra occurs, this leads to increased pressure within the urethra and the bladder. This increase pressure is ultimately translated to the ureters and kidneys resulting in decreased glomerular filtration rate (GFR) which results in reduction of urine production and excretion of potassium (which is dependent on urine flow and excretion in the distal tubules) and acids (primarily phosphates and sulfates). With continued obstruction, progressive decreases in renal blood flow occur mediated primarily by thromboxane and angiotensin II. This can result in renal ischemia and ultimately nephron loss.

The increase in blood potassium and accumulation of blood urea nitrogen (BUN), creatinine, phosphorus, and hydrogen ion contribute to the clinical signs associated with UO. Severe hyperkalemia results in cardiac arrhythmias and is the most life threatening aspect of this condition. When the potassium concentration rises, the body reacts by moving potassium intracellularly through the effects of insulin, epinephrine, and increased potassium concentration (diffusion gradient) in the extracellular fluid. Increases in the intracellular concentration of potassium causes the resting cell membrane potential in excitable tissues to become less negative (greater than the normal resting potential of -70 mV). The increased resting membrane potential inactivates the fast sodium channels making cell membrane depolarization more difficult. This results in bradycardia. In addition, the Na/K ATPase pumps which maintain the normal gradient of sodium (higher outside the cell) and potassium (higher inside the cell) are inactivated. When the cells of the cardiac atria are affected, they become unable to control automaticity of the heart and arrhythmias result. If the serum potassium level gets high enough, electrical activity in the heart ceases altogether resulting in asystole. The cardiac arrhythmias that may be seen include (in order of increasing potassium concentration): increase in T wave amplitude, decrease in R wave amplitude, prolongation of QRS and P-R intervals, S-T segment depression, decrease in P wave amplitude and duration, prolongation of the Q-T interval, atrial standstill and sinoventricular rhythm. CAUTION - clinical cases of urethral obstruction don’t always follow this pattern as other metabolic abnormalities (i.e. hypocalcemia, hypomagnesemia) exist that can affect the cardiac conduction. In other words, the serum potassium concentration does not always correspond to the cardiac abnormalities seen in actual clinical cases of urethral obstruction.

Stabilization of the “sick” blocked cat
The diagnosis of urethral obstruction is relatively straight forward and based on the physical exam finding of a large firm bladder that is unable to be expressed with gentle palpation. After bladder palpation, a complete physical exam should be performed. In cats with urethral obstruction, the heart rate should be carefully evaluated as bradycardia (a heart rate < 140 bpm) can alert you to the presence of hyperkalemia. In addition, hypothermia may be present in sick cats with urethral obstruction. The combination of bradycardia (HR <140) and hypothermia (T<96.6 °F) has been found to be 98% predictive of serum potassium level greater than 8 mEq/L in cats with urethral obstruction. Finally, the presence or absence of a murmur is important to identify in cats with urethral obstruction as sedation and/or anesthesia will be needed to relieve the urethral obstruction and this may determine the drugs you choose to use or avoid. Complete abdominal palpation should be repeated in cats with urethral obstruction after the cats is stabilized and the
obstruction has been relieved. After confirmation of urethral obstruction, an intravenous catheter should be immediately placed and blood work obtained. A minimum database (PCV/TS/GLU/AZO) should be drawn at catheter placement in addition to a venous blood gas to evaluate blood electrolytes, especially potassium, and acid/base status. It is not uncommon for sick blocked cats to have a severe metabolic acidosis, hyperkalemia, and low ionized calcium (likely secondary to hyperphosphatemia).

Once an IV catheter has been placed, an ECG should be performed, especially if hyperkalemia is present, to evaluate for cardiac arrhythmias. If cardiac arrhythmias are seen, treatment of the hyperkalemia should be initiated. Ultimately the establishment of urine flow will definitively treat the hyperkalemia. However, urethral catheterize SHOULD NOT take place until the heart rate and rhythm are stable. This process generally takes no more than a few minutes and up to approximately 30 minutes. Finally, in cats with a alterations in tissue perfusion parameters (pale mucous membranes, hypothermia, bradycardia, poor peripheral pulses), a systolic blood pressure measurement should be performed to evaluate for hypotension.

Options for treatment of hyperkalemia

1. Intravenous fluid therapy with isotonic crystalloids: intravenous fluids will dilute the potassium lowering its serum concentration. Any isotonic crystalloid is appropriate (even crystalloids containing small amounts of potassium such as Normosol R or Plasmalyte). There is some evidence to suggest that resolution of a concurrent metabolic acidosis may occur faster in cats given Normosol-R or LRS compared with 0.9% Saline. However, no difference was found in outcome or resolution of hyperkalemia. Therefore, it appears that as long as adequate volume is administered, the type of fluid does not matter. A reasonable initial fluid rate of 4-6 ml/kg/hr is appropriate in most cats but lower rates should be considered in animals with underlying heart disease or that are not severely ill (ie. 2 ml/kg/hr). In severely compromised, hypotensive patients, it may be necessary to administer a fraction of a shock bolus (10-20 ml/kg over 20-30 minutes).

2. IV 10% Calcium Gluconate: This medication works by increasing the cell’s threshold membrane potential re-establishing the normal difference between resting membrane potential and threshold potential. This will allow the cardiac cells to depolarize. Dose: 3 ml/cat (1-1.5 ml/kg) given over about 3-5 minutes while evaluating the ECG. This dose may be repeated if needed. The effects of this drug are immediately apparent on the ECG and once the cardiac arrhythmias are resolved, no more drug should be given.

3. IV Dextrose OR IV Regular Insulin + Dextrose: When insulin binds to its receptors on cells this stimulates the Na+/K+ ATPase causing intracellular movement of potassium. Dextrose is given to prevent hypoglycemia. When dextrose is given alone, this will stimulate endogenous insulin production from the pancreatic beta cells. Dose: 1 unit of regular insulin/cat, 0.5 g/kg of 50% Dextrose diluted 1:3. It takes about 20-30 minutes to see an effect from this medication and the insulin will be active for approximately 1 hour. The cat should be supplemented with 2.5% dextrose in its intravenous fluid bag for several hours to prevent hypoglycemia.

4. Terbutaline: Beta agonist. This will stimulate the movement of potassium intracellularly through the action of the Na/K ATPase pump. Dose: 0.01 mg/kg IV. In people, this has been found to lower serum potassium by approximately 1 mEq/L.

5. Intravenous Sodium Bicarbonate (rarely if ever given): Administration of this medication will increase the pH in the extracellular space. This stimulates the exchange of intracellular H+ ions for extracellular potassium pushing potassium into the cell. Dose: 1 mEq/Kg once or 0.3(BWkg) – BE (usually 1/3 – ½ of this dose will be given). It will take about 20-30 minutes to see an effect of this medication.

Urethral catheterization

Once the cat’s cardiovascular system has been stabilized, passage of a urethral catheter to relieve the obstruction is essential to re-establish normal urine flow and allow for resolution of hyperkalemia and azotemia. Cats are typically heavily sedated or placed under general anesthesia to maximize urethral relaxation (decreasing risk of trauma to the urethral wall) and eliminate pain associated with passage of the urethral catheter. Many different sedation protocols exist and no one protocol has been evaluated critically. We currently use a combination of methadone (0.4 mg/kg IV) and valium (0.2-0.4 mg/kg IV). At our institution, we will also perform a coccygeal epidural using a combination of bupivicaine (0.22 mg/kg) and preservative free morphine (0.1 mg/kg) to allow for maximal urethral relaxation further minimizing trauma to the urethra and allowing for longer term analgesia. This combination is then diluted with sterile saline to reach a dose of 0.2 ml/kg which allows for cranial extension of the block to L1-L2. Performing the epidural is straight forward and requires supplies that are already available in your hospital: 1 inch 24 gauge needle, 3 cc syringe and sterile gloves. A 2 inch X 2 inch area over the sacrum sacrum is clipped and aseptically prepared. The injection is made between the coccygeal vertebrae or the C1-C2 epidural space. It can take approximately 5-10 minutes for the epidural to take effect. Success of the epidural can be assessed by checking a perineal response and tail tone. In a double-blind placebo controlled study, we found that cats that received a morphine/bupivicaine combination coccygeal epidural had a significantly longer duration of analgesia and there were some cats that never required systemic opioids during hospitalization. When administering a morphine epidural however, it is important that the urinary catheter be left in place for 24 hours as urinary retention has been reported to occur.

Following sedation (+/- epidural) an open-ended tomcat catheter or MILA catheter with stylet can be used to relieve the obstruction. Sterile procedure (sterile gloves, sterile preparation of the perineum, draping the perineum) should be used to prevent introduction of bacteria into the urinary system. Admission urinary tract infection is uncommon in male cats with urethral obstruction and reports in the literature range from 0% to 21%. However, the incidence of catheter associated urinary tract infection ranges from
13% to 31% and therefore, it is important to monitor for evidence of developing infection during catheterization through daily urine gram stained sediment examinations.

Some institutions will perform a decompressive cystocentesis prior to urethral catheterization. The proposed benefits of a decompressive cystocentesis include pain relief, decreasing renal back pressures and improving bladder wall perfusion. In addition, decreasing intraluminal pressures prior to urinary catheterization may facilitate retropulsion of urethral plugs or uroliths and may ease urinary catheterization. A recent retrospective study evaluating outcome in 47 males cats with UO managed with DC and urinary catheterization, found that the procedure appeared safe with a 91% survival to hospital discharge rate, which is similar to other studies reporting outcome in UO in which DC was not performed. In this same study, 19 cats (56%) that had abdominal radiography had a focal loss of peritoneal detail consistent with effusion. Similarly, in a second prospective study evaluating DC without urinary catheterization, 53% (8/15) had evidence of caudal abdominal effusion following the initial decompression. However, no cats developed a clinically significant uroperitoneum. None of the above mentioned studies, however had a control group to allow for critical evaluation of their findings nor were these studies designed to evaluate for the proposed benefits of DC.

Generally, the cat should be hospitalized until the azotemia has resolved and the urine is clear with no obvious debris in the urine line. The ideal duration of catheterization is not known and varies based degree of urinary tract inflammation.

Additional considerations
Cats with prolonged obstruction (signified by azotemia causing a metabolic acidosis: pH<7.35) are at risk for post-obstructive diuresis resulting in massive urine production. In a retrospective study of 32 cats with urethral obstruction, the incidence of post obstructive diuresis, defined as a urine output of >2 ml/kg/hr, was 46% in the first 6 hours of hospitalization. This post-obstructive diuresis is thought to occur secondary to impaired tubular reabsorption of glomerular filtration as GFR and renal blood flow are still reduced in the period immediately following resolution of the obstruction. Impaired tubular reabsorption may result from urea osmotic diuresis, expansion of the extracellular fluid volume, altered intrarenal physical factors secondary to elevated intrarenal pressure, tubule insensitivity to ADH, and/or alterations in other natriuretic factors that have been ill-defined. Regardless of the underlying cause, the resulting massive urine production can result in severe dehydration and hypovolemia if IV therapy is not adjusted accordingly. It is not uncommon in the critically ill blocked cat for the urine production to be as much as 100 ml/hr! Therefore, frequent monitoring of urine output (every 2 hours ideally) and adjustments to the intravenous fluid rates should be made if a post-obstructive diuresis is occurring. The intravenous fluid rate should at least match urine output (depending on the hydration status of the patient) until the azotemia and hyperkalemia have resolved and the cat is feeling better. The post-obstructive diuresis typically is short term and will resolve within 24-48 hours. At some point, the IV fluids may start to drive urine production due to medullary washout and an increased GFR. Therefore, attempts should be made to decrease the rate of administration of the IV fluids and monitor to see if the urine production drops concurrently. As long as urine production decreases with the drop in IV fluid administration rate, the IV fluid rate may continued to be decreased and eventually discontinued. If urine production remains inappropriately high despite the decrease in IV fluid rate, the IV fluids should be increased back to previous rates and attempts at decreasing IV fluid administration can be tried again in a few hours.

Alpha antagonists, such as Prazosin (0.25-0.5 mg/cat PO q 12 hours x 7-10 days) or phenoxybenzamine (2.5-7.5 mg/cat P) q 12-24 hours x 7-10 days are routinely administered to cats with urethral obstruction in the hopes that it will prevent urethral spasm and re-obstruction by causing smooth muscle relaxation. Clinically, cats with urethral spasm (aka urethral hyper-reactivity) may strain to urinate and only produce small amounts of urine after the urethral catheter is pulled. Typically, urine can be expressed from these cats with bladder palpated, however, there is a large concern for re-obstruction as these cats do not completely empty their bladder. At this time, there is very limited evidence to support the use of treatment to prevent recurrent urethral obstruction and the appropriate duration of therapy is not currently known. A possible side effect of prazosin is low blood pressure (can cause vascular smooth muscle dilation as well) seen following the first dose administered and diarrhea.

Selected references
Respiratory distress is a commonly encountered, and truly life-threatening, emergency presentation. Successful management of the emergent respiratory patient is contingent upon rapid assessment and stabilization, and action taken during the first minutes to hours often has a major impact on patient outcome. While diagnostic imaging is undoubtedly a crucial part of the workup, patients at presentation may be too unstable to safely achieve imaging and clinicians may be called upon to institute empiric therapy based primarily on history, physical exam and limited diagnostics. This lecture will cover the initial evaluation and stabilization of the emergent respiratory patient, with a particular emphasis on clues from the physical exam that may help localize the cause of respiratory distress.

Establishing an airway
The first priority in the dyspneic patient is ensuring a patent airway. Signs of an obstructed airway can include stertorous or stridorous breathing or increased respiratory effort with minimal air movement heard when auscultating over the trachea. If an airway obstruction is present efforts should be made to either remove or bypass the obstruction. Clinicians should be prepared to anesthetize and intubate patients if necessary to provide a patent airway. Supplies to have on hand for difficult intubations include a variety of endotracheal tube sizes, stylets for small endotracheal tubes, a laryngoscope with both small and large blades, and instruments for suctioning the oropharynx. Emergent tracheostomy is indicated when orotracheal intubation is not feasible, although in the author’s experience this is a rare occurrence.

Physical exam and stabilization
Respiratory distress may be a manifestation of an abnormality in any one or a combination of the following areas: the upper airway, the lower airway, the pulmonary parenchyma, the pleural space, or the chest wall/diaphragm. Classically patients in respiratory distress will present with overt tachypnea or dyspnea; with severe distress they may adopt an orthopneic posture with elbows abducted and head and neck extended in an effort to minimize resistance to airflow into the lungs. Cats may present with less overt signs of distress than dogs. It should be noted that cyanosis is a specific but insensitive marker of hypoxia as it requires an arterial partial pressure of oxygen less than 50mmHg and greater than 5g/dL of deoxygenated hemoglobin. Thus, cyanotic mucous membranes always indicate severe hypoxemia but the absence of cyanosis cannot rule out severe respiratory compromise.

Careful attention to the physical exam may help to better localize the cause of distress (Table 1). It should be noted, however, that patients in respiratory distress can be quite fragile and may decompensate quickly particularly if stressed. Efforts should be made to minimize stress where possible. In some cases this may limit the initial physical exam to a brief cardiopulmonary auscultation and assessment of the cardiovascular status (mucous membrane color, capillary refill time, heart rate and rhythm, presence of a murmur, pulse palpation). A full physical exam is performed once the patient is more stable.

Upper airway disease
Patients with disease of the upper airway frequently have audible upper airway noise which is occasionally loud enough to make accurate auscultation of the heart and lungs challenging. Stertor is generally associated with obstruction in the nasopharynx while stridor more commonly points toward laryngeal obstruction. A harsh, honking cough is frequently noted with tracheal collapse. Evaluation of the respiratory pattern in patients with upper airway obstruction often reveals a long, shallow breathing pattern. A long, shallow inspiratory phase generally suggests obstruction of the extrathoracic airway while intrathoracic airway obstruction more commonly results in expiratory difficulty. In animals with severe respiratory distress these patterns may be difficult to distinguish and in some dogs with both extrathoracic and intrathoracic tracheal collapse both of these patterns may be present. Patients with upper airway obstruction often present in a very agitated state, may be quite dyspneic and very frequently are hyperthermic.

Common differentials for upper airway disease include brachycephalic obstructive airway syndrome, laryngeal paralysis collapse, tracheal collapse, nasopharyngeal collapse, nasopharyngeal polyp, laryngeal or nasopharyngeal edema, foreign body obstruction, or obstruction by a benign or malignant mass. Laryngeal paralysis is the most common cause of upper airway obstruction in large dogs while collapsing trachea more commonly occurs in small breed dogs. If a foreign body obstruction is suspected, an abdominal thrust maneuver may be employed in an attempt to clear the airway.

Patients with a dynamic airway obstruction, for example, dogs with laryngeal paralysis, tracheal collapse or brachycephalic obstructive airway syndrome, may benefit from sedation with one or a combination of butorphenol (0.1-0.3 mg/kg IM) and acepromazine (0.01-0.02 mg/kg IM). This helps to relieve the anxiety associated with increased work of breathing that often exacerbates their degree of distress. A low dose of a corticosteroid (dexamethasone sodium phosphate, 0.1 mg/kg IM) may be provided to reduce laryngeal edema or tracheal inflammation. Following sedation, placing these patients in a cool and quiet environment may provide relief while allowing the airway to be evaluated and interventions to be considered. Patients with severe upper airway obstruction or those in extremis may require more aggressive intervention, including emergent tracheostomy, intubation, or intubation with a large-bore endotracheal tube. In these cases, endotracheal intubation with a large-bore endotracheal tube may facilitate ventilation and oxygenation while minimizing resistance to airflow. In cases of severe laryngeal obstruction, emergent tracheostomy may be necessary to establish a patent airway.

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environment with oxygen supplementation may help alleviate their dyspnea. If available, placing the patient into an oxygen cage may be considered as this will allow for delivery of supplemental oxygen and frequently the white noise effect inside the cage may help to calm some stressed patients. Active cooling should be employed for patients with a rectal temperature greater than 103 degrees Fahrenheit.

Intubation is required for patients with dynamic upper airway obstruction that do not respond to sedation. Laryngeal function can be assessed at the time of intubation although this may be difficult to interpret in the setting of moderate or severe laryngeal edema – this may necessitate performing a laryngeal exam in a more controlled setting once the inflammation has resolved. Although definitive therapy for dynamic upper airway obstruction frequently requires a surgical or interventional approach, many patients may respond to medical management with a combination of weight control, cough suppression and sedation as needed.

Lower airway disease
Lower airway disease is a common cause for respiratory distress in cats. In fact, lower airway disease/asthma ranks among the top causes for feline respiratory distress along with congestive heart failure and pleural effusions. Typically cats will have a history of cough although owners may frequently not recognize this as coughing and rather suspect that the cat is attempting to vomit a hairball.

On presentation cats may present in variable degrees of respiratory distress, ranging from moderate tachypnea to orthopnea with open-mouth breathing. Cats are notorious for masking signs of respiratory compromise; often by the time distress is recognized by owners their disease is quite severe. The breathing pattern in these patients most commonly involves expiratory dyspnea and pulmonary auscultation may reveal wheezes or occasionally harsh crackles.

When an asthmatic crisis is suspected, oxygen is provided and a trial dose of a glucocorticoid (dexamethasone sodium phosphate, 0.15-0.2 mg/kg IM) and rapidly acting bronchodilator may be administered. At our facility, terbutaline, a selective B2 agonist (0.01 mg/kg, IM or SQ), is the preferred bronchodilator. Albuterol, an alternative B2 agonist, can be administered via inhaler; however, these cats are frequently quite stressed and may not tolerate the use of inhaled medication. Typically we expect to see some improvement within 30 minutes of administration. B2 agonists may cause transient tachycardia so these drugs must be administered with caution in cats with myocardial disease.

Pulmonary parenchymal disease
Pulmonary parenchymal disease can lead to a variable degree of dyspnea dependent on disease severity and distribution. Typically dyspnea is more inspiratory in nature and patients will often develop a restrictive breathing pattern due to decreased pulmonary compliance, leading to short, shallow but fast respirations. Auscultation often will reveal either increased bronchovesicular sounds or inspiratory crackles which may be focal or diffuse in distribution; however, breath sounds may be diminished when auscultating over a severely consolidated lung lobe. Dogs with pulmonary parenchymal disease will commonly cough but this is less frequently seen in cats.

Common causes of pulmonary parenchymal disease include pneumonia, left-sided congestive heart failure, neurogenic pulmonary edema, pulmonary contusions or hemorrhage, pulmonary thromboembolism, pulmonary fibrosis, acute respiratory distress syndrome, and neoplasia. Chest radiographs are typically required for diagnosis but frequently history and clinical signs may point to one differential over others. For example, dogs with pneumonia may be febrile or have mucopurulent nasal discharge. Evidence of trauma or petechiae/ecchymoses may indicate pulmonary contusion or pulmonary hemorrhage, respectively. The presence of a heart murmur coupled with increased bronchovesicular sounds or inspiratory crackles, arrhythmia or pulse deficits might suggest congestive heart failure as the cause of respiratory distress and a trial dose of furosemide (2-4 mg/kg IM for dogs, 1-2 mg/kg IM in cats) could be considered. For patients with congestive heart failure we generally expect to see some (although at times very minimal) improvement within 20-30 minutes of furosemide administration.

Pleural space disease
Patients with pleural space disease are unable to fully expand their lungs and, as a result, frequently develop a restrictive breathing pattern. A paradoxic or inverse respiratory pattern, that is, inward movement of the thoracic wall and outward movement of the abdomen during inspiration, may be seen with severe cases of pleural space disease. This respiratory pattern may also be seen with flail chest and severe upper airway obstruction. Auscultation commonly reveals diminished or muffled lung sounds. Pneumothorax typically results in diminished lung sounds dorsally while pleural effusion and diaphragmatic hernia more often cause diminished ventral lung sounds. Tension pneumothorax is rapidly life threatening; these patients are generally markedly dyspneic, may develop cardiovascular collapse as a result of inhibition to venous return, and their chest may have a ‘barrel shaped’ appearance. Patients with acute hemothorax may manifest signs of hypovolemic shock prior to the development of respiratory compromise.

Common causes of pleural space disease include pneumothorax, hemothorax, chylothorax, right-sided heart failure, pyothorax, neoplastic effusions, and diaphragmatic hernia.

If pleural effusion or pneumothorax are suspected, thoracocentesis should be performed as this procedure can be both diagnostic and therapeutic. Thoracocentesis does carry some minimal risk of hemorrhage and iatrogenic pneumothorax but in the vast majority of
cases the benefit to performing thoracocentesis far outweighs these risks. Thoracostomy tubes are placed in patients that have persistent pneumothorax despite thoracocentesis and may be considered for medically managed pyothorax.

**Chest wall and diaphragm disease**

Poor chest excursions may be seen with diseases that affect the neuromuscular control of the chest wall. Patients with a flail chest will have a free-floating segment of the rib cage that moves paradoxically inward on inspiration and outward on expiration. When an open pneumothorax occurs clinicians will typically note either a palpable defect in the chest wall or the presence of a sucking chest wound. Care should be taken in the exploration of traumatic chest wounds as occasionally manipulation of tissue can lead to an open pneumothorax.

Common causes of chest wall and diaphragm disease include central respiratory depression (for example, due to sedation or intracranial disease), cervical myelopathy, lower motor neuron disorders (for example, myasthenia gravis, botulism, polyradiculoneuritis, and tick paralysis), and severe hypokalemia leading to profound respiratory muscle weakness. An arterial or venous blood gas should be performed in these patients to evaluate for hypercapnea. Severe hypercapnea (PaCO₂ > 60mmHg) may warrant intubation and positive pressure ventilation. Open chest wounds should be occluded with Vaseline impregnated gauze and thoracocentesis is performed to address the pneumothorax. For larger chest wounds a thoracostomy tube is inserted through the wound and the wound is subsequently occluded with Vaseline impregnated gauze. Ultimately these wounds should be explored and closed surgically once the patient is stable. Patients with flail chest often respond well to analgesia and may not require surgical stabilization. Placing them in lateral recumbency with the flail segment down may help alleviate discomfort.

**The “look alikes”**

Many non-respiratory diseases may present with signs suggestive of respiratory compromise. Some examples include pain, anxiety, hyperthermia, severe hypovolemia and metabolic acidosis. Pulse oximetry or arterial blood gas analysis may be useful to rule out hypoxemia in these patients. However, when in doubt, oxygen therapy should be provided until a definitive diagnosis is reached.

**Table 1: Localizing respiratory distress**

<table>
<thead>
<tr>
<th>Localization</th>
<th>Physical Exam Findings</th>
<th>Common Differentials</th>
<th>Initial Stabilization</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper airway</td>
<td>• Stertor • Stridor • Long shallow respiratory pattern • Lack of air movement with complete obstruction</td>
<td>• Brachycephalic obstructive airway syndrome • Laryngeal paralysis • Tracheal collapse • Laryngeal edema • Benign or neoplastic mass • Foreign body</td>
<td>• Oxygen • +/- Sedation • +/- Intubation</td>
</tr>
<tr>
<td>Lower airway</td>
<td>• Expiratory dyspnea • +/- Crackles or wheezes • +/- History of cough (cats)</td>
<td>• Chronic bronchitis • Asthma</td>
<td>• Oxygen • +/- Steroid • +/- Bronchodilator</td>
</tr>
<tr>
<td>Pulmonary parenchyma</td>
<td>• Inspiratory dyspnea • +/- Increased bronchovesicular sounds or crackles • +/- Murmur, arrhythmia • Restrictive or paradoxic respiratory pattern</td>
<td>• Congestive heart failure • Pneumonia • Neurogenic pulmonary edema • ARDS • Pulmonary hemorrhage • Pulmonary thromboembolism • Pulmonary fibrosis • Neoplasia</td>
<td>• Oxygen • +/- Furosemide if congestive heart failure suspected</td>
</tr>
<tr>
<td>Pleural space</td>
<td>• Muffled breath sounds • Restrictive or paradoxic respiratory pattern</td>
<td>• Pneumothorax • Right-sided heart failure • Chylothorax • Pyothorax • Hemothorax • Neoplasia • Diaphragmatic hernia</td>
<td>• Oxygen • Thoracocentesis • +/- Chest tube</td>
</tr>
<tr>
<td>Chest wall and diaphragm</td>
<td>• Poor chest wall excursions • Paradoxic chest wall movement with flail chest • Palpable chest wall defect or sucking wound</td>
<td>• Flail chest • Open pneumothorax • Central respiratory depression • Cervical myelopathy • Lower motor neuron disease • Severe hypokalemia</td>
<td>• Oxygen • Arterial or venous blood gas to assess ventilation • +/- Intubation, positive pressure ventilation • Cover open wounds, thoracocentesis</td>
</tr>
</tbody>
</table>
Summary
In summary, respiratory distress may present a challenge as patients are often too unstable to allow for a comprehensive diagnostic workup. However, close attention to the physical exam and history often will narrow the differential list and allow for the initiation of empiric stabilizing therapy. Once the patient is stable, a definitive diagnosis and specific therapy can be pursued.

References Available by request.
Pets are often indiscriminate eaters and accidental (or, infrequently, malicious) poisonings are a relatively common occurrence. In many cases no specific antidote exists and treatment of intoxicated patients is largely supportive and symptomatic. However, some general principles apply to most toxicities, namely efforts aimed at decontamination and hastened toxicant elimination. This lecture will review the general approach to the poisoned patient with a focus on patient assessment as well as strategies for decontamination and promoting toxicant elimination. We will also discuss management of a few select toxicities.

**Patient assessment**

At presentation, all patients should be assessed for stability with particular focus on the respiratory, cardiovascular, neurologic and urinary systems. Signs of respiratory compromise can include tachypnea, dyspnea, an orthopneic posture (extended head and neck, abducted elbows), audible upper airway noises, and, in severe cases, cyanosis. Patients with cardiovascular instability may have hyperemic, pale or white mucous membranes, tachycardia or severe bradycardia, dysrhythmia, or poor or variable pulse quality. Neurologic instability may manifest as altered mentation, loss of consciousness, ataxia, altered or absent cranial nerve reflexes, or postural or gait abnormalities. Assessment of the urinary system at initial presentation is primarily directed toward determining whether a bladder obstruction is present. If the primary patient survey reveals compromise in any of these four major body systems, the patient is stabilized as necessary.

**Decontamination and elimination**

Decontamination, when possible, is performed to inhibit or minimize further absorption of the toxicant. For ocular exposures, eyes should be flushed for 20 minutes with either physiologic saline or tepid tap water. Ideally owners would perform this at home prior to transport to the veterinarian. Placement of an Elizabethan collar after flushing the eyes will help to prevent trauma to the cornea from the patient pawing at or rubbing the eye. Dermal decontamination can be accomplished by gently bathing the skin with tepid water and liquid dish soap. Gloves should be worn while bathing the patient to prevent skin exposure to the toxic agent and the patient’s temperature should be closely monitored as hypothermia may develop during the bathing process. Cats suffering from pyrethrin toxicosis may have moderate or severe muscle tremors and can benefit from a dose of methocarbamol (50 mg/kg IM) just prior to bathing.

Oral decontamination via emesis is generally indicated when ingestion of the toxicant has occurred within the prior 1-2 hours. However, there are several important contraindications to emesis induction that clinicians should be familiar with. Emesis is not appropriate in symptomatic patients, particularly those manifesting neurologic signs like seizures, sedation or obtundation as the risk of aspiration of gastric contents following emesis is quite high. Emesis is also contraindicated following ingestion of caustic substances which may further injure the esophagus during vomiting and following ingestion of hydrocarbon liquids as these may easily be aspirated into the airway. Patient anatomy and comorbid disease processes should also be taken into consideration when determining whether emesis is appropriate. Emesis is generally not recommended for patients already predisposed to aspiration pneumonia, for example, patients with brachycephalic obstructive airway syndrome, upper airway or laryngeal dysfunction, neuromuscular disease or megaesophagus.

When appropriate, emesis may be induced in dogs by oral administration of 3% hydrogen peroxide (1-5 mL/kg, maximum dose 50 mL) or by administration of apomorphine (0.03 mg/kg IV or via tablet placed in the subconjunctival sac).

Hydrogen peroxide causes emesis via direct gastric irritation. Adding it to a small amount of food may improve palatability; otherwise, if the patient is unwilling to drink the prescribed amount it can be administered by syringe with care not to induce aspiration. If vomiting does not occur within 10-15 minutes a second dose can be administered. If vomiting does not occur after the second dose, further administration of hydrogen peroxide is unlikely to be of benefit.

Apomorphine acts centrally at the level of the chemoreceptor trigger zone (CRTZ) via stimulation of dopamine receptors. It is frequently rapidly effective and dogs will generally vomit within several minutes. Some dogs may become sedate following administration of apomorphine – this can be reversed by administration of naloxone (0.02 mg/kg IM or IV) however naloxone will not affect the emetic action of apomorphine. A recent retrospective study compared apomorphine to 3% hydrogen peroxide and found that both were quite effective at inducing emesis in dogs, although anecdotal experience suggests that apomorphine more reliably will induce emesis.

Compared to dogs, emesis occurs less reliably in cats. Apomorphine is not thought to be an effective emetic in cats and hydrogen peroxide administration has anecdotally been associated with the development of severe hemorrhagic gastroenteritis. The mainstay emetic of choice in cats has historically been xylazine (0.44 mg/kg IM), an alpha-2 agonist, although a recent retrospective study conducted at our facility found that dexmedetomidine is an effective alternative to xylazine and may be a superior agent for inducing...
emesis. Further research is needed to identify the most effective route and dose for dexmedetomidine but we typically administer 7 mcg/kg IM. When emesis is successfully induced with xylazine or dexmedetomidine it typically occurs within 15 minutes. Both of these drugs may cause sedation and peripheral vasoconstriction which can lead to hypertension and a reflex bradycardia. Yohimbine (0.1 mg/kg IM or IV) is used to reverse xylazine and atipamezole (50 mcg/kg, or a volume equal on a per mL basis to amount of dexmedetomidine administered, IM or IV) is used to reverse dexmedetomidine. A reversal should be given following emesis or within 15 minutes if emesis is not successfully induced.

Syrup of ipecac, table salt and liquid dish soap are no longer recommended as emetic agents as safer and more effective emetics are readily available.

Gastric lavage can be considered when emesis is not successful or when it is contraindicated. Gastric lavage should not be performed following ingestion of caustic agents or hydrocarbons. It is best performed with the patient under general anesthesia and intubated so that the airway is protected. An orogastric tube is measured from the mouth to just beyond the last rib, lubricated and gently advanced into the stomach to the previously measured length. Placing a roll of white tape in the patient’s mouth will help facilitate keeping it open during the procedure; the orogastric tube can be fed through the hole in the roll. Warm water (60 mL/kg) is slowly infused into the stomach whilst palpatating to monitor for gastric overdistention. Fluid is then recovered by gravity and this cycle is repeated several times until the retrieved fluid is relatively clear. Complications of gastric lavage can include aspiration pneumonia and injury to the mouth, esophagus or stomach during placement of the tube.

In some cases, endoscopy or surgery are required for decontamination. For example, ingestion of corrosives, transdermal patches, or metals like zinc which are not able to be removed via emesis (or in the case of corrosives, if emesis is contraindicated). Gorilla glue is an adhesive which rapidly expands within the stomach and often forms a cast of the inside of the stomach obstructing gastric outflow. When this occurs gastrotomy is typically required for removal.

Activated charcoal is an adsorbent used to diminish systemic absorption of certain ingested toxicants. Many preparations of activated charcoal contain sorbitol, a cathartic, which is used to hasten gastrointestinal transit time. The dose of activated charcoal is 1-2 g/kg PO; for certain toxicants which undergo enterohepatic recirculation multiple dosages given q6-8 hours may be warranted. However, when administering multiple dosages of activated charcoal it is recommended to only administer a cathartic on the first dose due to the potential for hypernatremia due to hypotonic fluid loss in the gastrointestinal tract. Activated charcoal without a cathartic is used for the second and subsequent dosages. Many dogs will eat activated charcoal if it is mixed into canned food (and some indiscriminant eaters will eat it by itself!); for other patients it can be administered carefully by syringe. For patients undergoing gastric lavage, activated charcoal can be administered via the orogastric tube prior to tube removal. Activated charcoal administration is contraindicated for patients that will undergo gastrointestinal surgery or with gastrointestinal perforation due to the risk of leakage; other contraindications include hypernatremia, ileus, and in symptomatic patients or those with comorbidities that increase the risk for aspiration pneumonia. It does not adsorb reliably to alcohols, xylitol or heavy metals and administration is not recommended for these toxicants.

Fluid therapy is commonly employed for forced diuresis, particularly for toxicants that undergo renal elimination. Generally speaking, an IV fluid rate of 3-5 mL/kg using an isotonic crystalloid is recommended for healthy patients; however, caution must be employed and fluids used judiciously in patients with preexisting cardiovascular or pulmonary disease to avoid volume overload.

Extracorporeal therapies like intermittent hemodialysis (IHD), continuous renal replacement therapy (CRRT), or charcoal hemoperfusion (CH) may be considered in certain toxicities (Table 1). A comprehensive discussion on the use of extracorporeal therapies is beyond the scope of this lecture but given the increasing availability of these modalities clinicians should be aware of their potential utility.

**Intravenous lipid emulsion**

Intravenous lipid emulsion (ILE) has been used in the treatment of many lipophilic drug toxicoses. ILE was first explored in experimental rodent models of bupivacaine-induced cardiac arrest which demonstrated a potential benefit in the treatment of local anesthetic toxicosis. Since then, use of ILE has been reported for other toxicities involving lipophilic toxicants (Table 2). The lipophilicity of a particular drug can be to some degree estimated by its log $P$ value ($P$ represents the partition coefficient of the substance); the higher the log $P$ value, the more lipophilic the substance. Although the exact mechanism of action for ILE is uncertain, sequestration of toxicants within the intravascular space (the “lipid sink”) or improved myocardial performance are thought to play a role.

There are a variety of commercial lipid emulsions available for purchase. To decrease the risk of microbial contamination, ILE should be administered through a new, dedicated IV catheter ideally placed aseptically. Most references in the veterinary literature suggest to administer a 20% ILE initially as a bolus (1.5 mL/kg) followed by a constant rate infusion of 0.25 mL/kg/min for 30-60 minutes. Additional boluses of 1.5 mL/kg may be administered q4-6 hours for the first 24 hours, as needed, provided the patient’s serum has not become significantly lipemic.
Complications of ILE are thought to be uncommon but may include microbial contamination leading to thrombophlebitis, fever, hypersensitivity reaction to the emulsion, hyperlipidemia, hypertriglyceridemia and pancreatitis. ILE should be used cautiously in patients that are septic or suffering from acute respiratory distress syndrome as administration has been associated with increased venous admixture and decreased PaO2 in critically ill people.

Table 1 – Select toxicants for which extracorporeal therapy might be considered

<table>
<thead>
<tr>
<th>Toxicant</th>
<th>Acetaminophen</th>
<th>Acetylsalicylic acid</th>
<th>Aminoglycosides</th>
<th>Amphetamines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atenolol</td>
<td>Azathioprine</td>
<td>Barbiturates</td>
<td>Chloramphenicol</td>
<td></td>
</tr>
<tr>
<td>Cyclophosphamide</td>
<td>Digoxin</td>
<td>Diltiazim</td>
<td>Ethylene Glycol</td>
<td></td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>MAO Inhibitors</td>
<td>Penicillins</td>
<td>Tricyclics</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 – Select toxicants for which intravenous lipid emulsion might be considered

<table>
<thead>
<tr>
<th>Toxicant</th>
<th>Baclofen</th>
<th>Bupivacaine</th>
<th>Carprofen</th>
<th>Clomipramine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyclosporin</td>
<td>Diazepam</td>
<td>Carprofen</td>
<td>Digoxin</td>
<td>Diltiazim</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>Ivermectin</td>
<td>Lidocaine</td>
<td>Moxidectin</td>
<td></td>
</tr>
<tr>
<td>Naproxen</td>
<td>Permethrin</td>
<td>Trazodone</td>
<td>Vinblastine</td>
<td></td>
</tr>
</tbody>
</table>

References Available by request.
Acute kidney injury (AKI), previously referred to as acute renal failure, refers to an abrupt and potentially sustained decline in glomerular filtration; consequently, many patients with AKI may develop profound disturbances in fluid balance, acid-base and electrolyte derangements, and other clinical signs of illness secondary to accumulation of uremic toxins. The term AKI is preferred over acute renal failure as it suggests a potentially reversible etiology with hope for renal recovery in some cases. The clinical course of AKI can be quite dynamic and management frequently involves a combination of therapies aimed at addressing the pathophysiologic derangements that occur as a result of altered renal function as well as removal or reversal of the inciting cause, if possible. This lecture will cover the approach to the AKI patient, with a focus on the diagnostic evaluation and management of these complex cases.

Pathophysiology
AKI is classically thought to represent a progression through four stages: initiation, extension, maintenance and recovery. The initiation phase involves the initial renal insult; frequently at this stage there is no overt manifestation of renal dysfunction. A combination of inflammation, ischemia and hypoxia set the stage for ongoing renal injury during the extension phase. Renal cell death occurs either via apoptosis and necrosis, the latter exacerbating local inflammation. At this point there may or may not be clinical or laboratory evidence of renal dysfunction. Patients will frequently develop clinical manifestations of AKI during the maintenance phase. Azotemia is common and urine output is highly variable, ranging from anuria to oliguria (urine output <0.5 mL/kg/hr) to polyuria in some cases. The maintenance phase may last from days to weeks. The recovery phase follows and may persist for many weeks or longer. During this time, renal tubular cells regenerate and azotemia may improve. Significant polyuria is common during this period, developing as a result of impaired tubular function and osmotic diuresis. Should they survive to this point, patients may return to a non-azotemic state although some studies would suggest that approximately half of survivors go on to develop chronic kidney disease.

The etiology of AKI can be classified as pre-renal, intrinsic renal and post-renal. Pre-renal AKI develops as a result of renal ischemia. Common causes of intrinsic renal AKI include infection (Leptospirosis, FIP, pyelonephritis), toxins, metabolic disturbances (hypercalcemia) and neoplasia. Intrinsic renal AKI may also be a component of multiple organ dysfunction syndrome (MODS) as a result of SIRS or sepsis. A list of common nephrotoxins is found in Table 1; however, it should be noted that many compounds (including several therapeutic drugs) may have nephrotoxic potential. Post-renal AKI may occur as a result urethral obstruction, ureteral obstruction or uroperitoneum. Ureteral obstruction by ureterolith is an increasingly recognized cause of post-renal AKI in cats; invariably these ureteroliths are calcium oxylate in composition. Frequently on palpation these cats will have one large, often painful kidney and one small, irregular kidney. We suspect in many of these cases that the smaller kidney had a ureteral obstruction at some point in the past and the loss of renal function in that kidney was compensated for by a functional, contralateral kidney. Subsequently, obstruction of the functional kidney leads to hydronephrosis and azotemia develops.

AKI staging systems have been developed and validated for use in human medicine, with stage determined by one or a combination of decreased glomerular filtration rate, increased serum creatinine, or decreased urine output. A modification of one of these staging schemes, the veterinary acute kidney injury (VAKI) scheme (Table 2), was evaluated retrospectively in a recently published study. In that study, dogs that developed AKI in hospital had a significantly increased mortality rate compared to those that did not develop AKI; this held true even for dogs with very minor increases in serum creatinine, many of which had a peak serum creatinine within the reference range.

Diagnostic evaluation
The diagnostic evaluation for a patient with AKI is directed primarily toward identifying any reversible cause in addition to evaluating for metabolic derangements or other pathophysiologic sequella of renal dysfunction. Minimally, a biochemistry panel, complete blood count and urinalysis with culture is suggested. Urine sediment evaluation is an easy in-house test that should be considered. Calcium oxylate monohydrate crystals may be seen with ethylene glycol toxicity and the presence of neutrophils with intracellular bacteria may suggest bacterial pyelonephritis. Casts are commonly seen and typically indicate renal tubular cell death. Other first line tests to consider include a venous blood gas to look for acid-base derangements (ethylene glycol toxicity commonly causes an elevated anion gap metabolic acidosis), infectious disease testing (particularly in dogs living in Leptospirosis endemic areas), and a non-invasive blood pressure as hypertension is a potential complication of AKI. In-house ethylene glycol test kits are available but false negatives may occur with prolonged time following ingestion and false positives may occur following administration of propylene glycol (which is found in some activated charcoal preparations and in injectable diazepam). Some formulations of ethylene glycol contain a fluorescent dye that will glow under a black light - this may be seen on the patient’s muzzle or fur or in their urine. Abdominal
radiographs may reveal unilateral or bilateral renomegaly or the presence of radiopaque ureteral stones. Abdominal ultrasound provides a more thorough evaluation of renal size and architecture; additionally, some ureteral stones may be too small to visualize on radiographs but are more readily apparent with ultrasound.

**AKI management**

When a reversible cause for AKI is identified, treatment is aimed primarily at removing that cause while concurrently providing supportive care to minimize or reverse the metabolic complications of AKI. When pyelonephritis is suspected, broad-spectrum antibiotics are initiated pending the results of a urine culture. Penicillins or doxycycline are indicated if Leptospirosis is a possibility. Urethral or ureteral obstructions are either removed or bypassed to re-establish normal urine flow. Ureteral stenting or placement of a subcutaneous ureteral bypass system via interventional radiography may be warranted in some cases of ureteral obstruction. Uroperitoneum may occasionally be medically managed with an indwelling urinary catheter in the case of a small bladder or proximal urethral defect; however, many cases of uroperitoneum require a diagnostic evaluation to identify the source of urine leak and eventual surgery to correct it. In many cases, despite a diagnostic workup, an inciting cause for AKI is not identified. General thoughts on AKI management are found below:

**Fluid therapy**

Intravenous fluid therapy is a cornerstone of treatment for AKI, but it is imperative that the fluid prescription is tailored to the needs of the individual patient and frequently re-assessed throughout their clinical course. Many patients will present dehydrated due to one or a combination of inappetance, vomiting, diarrhea or polyuria; for these patients, the deficit is typically corrected over 12-48 hours. Care should be taken to avoid over-hydration as this can lead to renal edema, potentially exacerbating AKI, as well as pulmonary edema or pleural effusion which might lead to respiratory compromise. Common early signs of fluid overload include tachypnea, serous nasal discharge, a soft cough, or a newly auscultated gallop heart rhythm (particularly in cats). Monitoring body weight serially and at least twice daily is suggested; a body weight gain of more than 5-8% may be a sign of impending fluid overload. As AKI patients may easily progress to oliguric or anuric states, careful attention to their fluid balance is essential.

**Urine output**

Urine output in the AKI patient can be quite variable and range from anuria to extreme polyuria (this is most common in the recovery phase or following removal or bypass of an obstructive process). Consequently, urine output is a parameter to monitor carefully. This is a cinch if a urinary catheter is in place but can be challenging otherwise. For smaller patients, lining their cage with and then weighing incontinence pads can be helpful; every gram of weight gained from urine corresponds to one mL of urine output. Monitoring the size of the urinary bladder with serial ultrasound may also be helpful in determining if urine production is occurring. Urine output may be minimal at initial presentation particularly if the patient is dehydrated; however, urine production is expected within one-two hours of initiating IV fluid therapy and no urine output within 6-8 hours should raise the alarm for oliguria or anuria.

**Managing oliguria**

When oliguria occurs in a well-hydrated patient, the IV fluid rate should be decreased to match the urine output in order to prevent fluid overload. Euhydrated, anuric patients should have their IV fluids either discontinued entirely if they are drinking or run at a rate to replace insensible losses only (22 mL/kg/day total) if they are not drinking. Diuretic therapy can be considered in an attempt to convert an anuric or oliguric state to a polyuric state. Although studies in humans with AKI have failed to find a survival benefit when oliguria is converted to polyuria, polyuric patients are generally much easier to manage as we can continue to give them IV fluid therapy, medications by injection, etc. Furosemide (0.5-2 mg/kg, IV) is a commonly employed loop diuretic that may also reduce renal tubular cell energy expenditure via blockade of the Na/K/2Cl co-transporter in the ascending Loop of Henle. If an IV bolus of furosemide reestablishes urine output, a constant rate infusion (0.1-1.0 mg/kg/hr) could be considered. Mannitol is an osmotic diuretic that may also improve renal blood flow and scavenge free oxygen radicals, potentially attenuating the local renal inflammation associated with AKI. Mannitol is a hyperosmolar agent which is not metabolized in the intravascular space and is eliminated entirely by glomerular filtration. Therefore, administration in an anuric patient may precipitate intravascular fluid overload. The bolus dose of mannitol is 0.25-0.5 g/kg IV; with a good response, a constant rate infusion (60-120 mg/kg/hr) can be instituted. It should be noted that in a dehydrated patient, oliguria is potentially physiologic and diuretic therapy is contraindicated as it could precipitate a pre-renal azotemia.

**Acid-base and electrolyte derangements**

Patients with AKI commonly may develop a metabolic acidosis, most often due to retention of uremic acids or poor perfusion with subsequent lactic acidosis. A high anion gap metabolic acidosis is also commonly seen in cases of ethylene glycol toxicity. Severe metabolic acidosis can lead to cardiovascular compromise as evidenced by cardiac arrhythmias, poor cardiac contractility and loss of vasomotor tone resulting in hypotension. Deteriorating mental status is another potential complication of severe metabolic acidosis.
Patients with a venous pH < 7.1-7.15 and clinical signs that may be attributed to metabolic acidosis may benefit from administration of sodium bicarbonate. The dose of sodium bicarbonate can be calculated from the following formula: 0.3 x (BW kg) x (24-measured bicarbonate) = mEq bicarbonate deficit. Typically ¼ to 1/3 of this deficit is administered over several hours and the acid-base parameters are then rechecked. Potential complications of sodium bicarbonate therapy include hypernatremia, hypokalemia, hypocalcemia, paradoxical cerebral acidosis, and iatrogenic metabolic alkalosis if too high of a dose is used. Sodium bicarbonate is very hyperosmolar and needs to be diluted prior to administration in a peripheral vessel; consequently the volume may exceed that which the patient may tolerate particularly if oliguric or anuric. Because bicarbonate is converted to carbon dioxide, it is contraindicated in a patient that is hypoventilating.

Hyperkalemia is typically the most life-threatening electrolyte derangement that may occur in the AKI patient. Elevated serum potassium can cause moderate to severe bradycardia and potentially cardiac arrest by increasing the cardiomyocyte resting potential. Hyperkalemic patients with compatible ECG changes (loss of P waves, wide QRS, tall tented T waves, bradycardia) should be administered calcium gluconate (100 mg/kg IV slow while monitoring the ECG) which stabilizes the cardiomyocyte membrane by raising the threshold potential. Regular insulin (0.1 u/kg IV) and dextrose (0.5 g/kg IV, then 2.5% concentration added to IV fluids) may then be given to promote cellular uptake of potassium. Terbutaline (0.01 mg/kg IV) is another option for treating hyperkalemia; this medication works by stimulating the cell membrane Na/K ATPase to drive potassium into cells. Terbutaline may cause tachyarrhythmias and should be used cautiously in patients with cardiac disease. Refractory hyperkalemia is an indication for renal replacement therapy when this option is available.

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Tetany due to ionized hypocalcemia is uncommon; when noted, calcium gluconate (100 mg/kg IV slow) is the treatment of choice. Severe hypercalcemia is addressed via a combination of saline diuresis, furosemide, and potentially corticosteroids, calcitonin or bisphosphonates.

Uremic complications and ancillary treatments
Patients with AKI are typically quite nauseated and generally benefit from administration of centrally acting anti-emetics. Dental rinses can be considered when uremic oral ulceration occurs. At our facility we will commonly treat these with a small volume of lidocaine jelly, carafate suspension and aluminum hydroxide gel mixed in equal parts. This “magic mouthwash” seems anecdotally to help with oral pain. Patients may also experience moderate to severe renal pain and systemic analgesia is commonly required. Antihypertensive therapy is indicated if moderate or severe hypertension develops. Assisted nutrition may be required if patients are persistently anorexic. When tolerated, enteral nutrition is preferred and enteral feedings may be facilitated through placement of a temporary feeding tube. Parenteral nutrition is indicated when enteral nutrition is not feasible.

When to consider renal replacement therapy
Access to dialysis has increased in recent years and this treatment modality is now available at many academic and private veterinary specialty hospitals. While a comprehensive discussion of dialysis for AKI is outside the scope of this lecture, common indications include anuria, severe and refractory hyperkalemia, fluid overload, severe azotemia not improving with conventional therapy, and in the treatment of certain nephrotoxicities (ethylene glycol, for example).

Summary
AKI is a complex disease process that may result in a host of systemic and metabolic complications. Successful management is contingent upon finding and reversing, when possible, any precipitating cause whilst supporting the patient and providing symptomatic therapy for any pathophysiological derangements they may develop as a consequence of declining renal function. Renal replacement therapy may be indicated in a subset of patients not responding to conventional medical treatment and in the treatment of certain nephrotoxicities.

Table 1 – Common nephrotoxins

<table>
<thead>
<tr>
<th>Aminoglycosides</th>
<th>Amphotericin B</th>
<th>Azathioprine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbapenems</td>
<td>Cisplatin/carboplatin</td>
<td>Diuretics</td>
</tr>
<tr>
<td>Doxorubicin</td>
<td>Envenomation</td>
<td>Ethylene glycol</td>
</tr>
<tr>
<td>Grapes/raisins (dogs)</td>
<td>Heavy metals</td>
<td>Intravenous contrast agents</td>
</tr>
<tr>
<td>Lilies (cats)</td>
<td>Melamine</td>
<td>NSAIDs</td>
</tr>
</tbody>
</table>

Table 2 – Veterinary acute kidney injury (VAKI) staging system

<table>
<thead>
<tr>
<th>VAKI Stage</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 0</td>
<td>Creatinine increase &lt;150% from baseline</td>
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<tr>
<td>Stage 1</td>
<td>Creatinine increase of 150-199% from baseline or Creatinine increase of 0.3 mg/dL from baseline</td>
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<td>Stage 2</td>
<td>Creatinine increase of 200-299% from baseline</td>
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<td>Stage 3</td>
<td>Creatinine increase &gt;300% from baseline or Creatinine &gt; 4.0 mg/dL</td>
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**References** Available by request
Emergency medicine is not for the faint of heart! What emergency veterinarians thrive on, the unknown unstable patient arriving without notice and the need to make rapid therapeutic decisions- can instill panic in others. However, whether or not you work in an emergency practice, as veterinarians we will all be faced with patients that are presented on an emergency basis or previously stable patients that deteriorate rapidly and require emergent diagnostics and interventions. These situations will interrupt a planned day and can cause significant stress. In this session, we hope to give you some “tools of the trade” that are useful to have in your arsenal for common emergencies and hopefully will help alleviate some of the stress associated with emergency medicine.

**Triage evaluation, oxygen therapy, vascular access and AFAST & TFAST**

- **Tips:** Assume the patient is dying and attempt to prove otherwise
- **Tips:** Have the pet owner sign an emergency stabilization form
- **Tips:** Use that 2 drops of blood in the catheter stylet
- **Tips:** Update the pet owner during and following stabilization

Compared with other veterinary specialties, emergency veterinarians consider all of their patients to be “dying until proven otherwise”. Therefore, for animals that are presenting as emergencies or in patients with acute deterioration, the focus will be on assessing the cardiovascular and respiratory systems first, followed by the neurologic and urinary systems. This is termed the primary survey and generally should take less than 2 minutes and can be done either by a well trained veterinary technician at patient presentation to the hospital or by a doctor. Any patient with evidence of respiratory difficulty or cardiovascular instability should be moved immediately to the treatment area for stabilization. In cases in which the patient is thought to require emergent intervention, we will have the owner sign an “emergency stabilization form” which authorizes us to initiate treatment and diagnostics for their pet and outlines the cost of care. The procedures that are covered by the stabilization charge include: the cost of the emergency exam, IV catheter, minimum database and venous blood gas analysis, intravenous fluid therapy, pulse oximetry, blood pressure measurement, abdominal and thoracic FAST exams, and analgesia. This approach allows stabilizing treatments to be initiated without removing the veterinarian from the treatment area or getting into a long conversation with the pet owner before you have information. The triage area of the treatment room should be equipped with an oxygen supply, supplies for IV catheter placement, intravenous fluids, continuous electrocardiography, blood pressure measuring equipment, and a crash cart (or crash kit). The crash cart/kit should be supplied with items needed for cardiopulmonary resuscitation.

Signs of respiratory compromise include cyanosis, stridor/stertor and increased respiratory rate and effort and open mouth breathing in a cat. These patients should have oxygen administered until the underlying cause of the respiratory distress is addressed. Signs of abnormal tissue perfusion include depressed mentation, pale, injected or white mucous membranes, prolonged capillary refill time (>2 sec), tachycardia or severe bradycardia, weak or absent peripheral pulses. Absent pedal (or metatarsal) pulses is specific for diagnosing hypotension - cats with absent pedal pulses likely have a SBP < 70 mmHg and dogs with absent pedal pulses will likely have a SBP <55 mmHg. These guidelines assumes that the patient does not have an aortic thromboembolism which should be suspected in a patient with acute paresis or paralysis with concurrent absent pedal pulses. It is important to note that not all patients will have all abnormal tissue perfusion parameters and there is no one single indicator that can confirm “shock”. Ideally, any patient with changes in these parameters would have a blood pressure measurement performed. It is important to note that in patients with early compensated shock, there may only be mild changes in these parameters and the blood pressure may be normal or even elevated. These patients are at risk for developing decompensated shock if left untreated. In these cases, calculating a shock index, which is the heart rate divided by the blood pressure, may be useful. To date, the use of shock index has only been investigated in dogs and its utility in cats is unknown. A shock index >1.0 in a dogs with a clinical history that suggests shock may be present, should increase your suspicion of shock and prompt additional diagnostic evaluation and/or treatment in that patient.

Any patient with signs of cardiovascular or respiratory compromise, should have an intravenous catheter placed to allow for drug and intravenous fluid administration. At the time of catheter placement, blood should be drawn for a minimum database, blood lactate measurement, venous blood gas analysis, and a blood smear. A blood smear can be useful for evaluation of platelets (thrombocytopenia?), red blood cells (evidence of regeneration? spherocytes? heinz bodies?) white blood cells (leukopenia? bands?) In addition, blood can be drawn and held for a complete blood count and chemistry screen to be run later. If blood cannot be obtained from the catheter – don’t let the blood in the stylet go to waste! You may have enough to fill one hematocrit tube which allows
measurement of PCV and TP. The serum that is placed on the refractometer for TP measurement can then be used for a blood glucose measurement (results of a recent veterinary study suggest that serum blood glucose using a handheld glucometer may be more accurate than a whole blood measurement so go for it!). Remember a normal PCV does not rule out hemorrhage especially in patients with physical exam findings consistent with abnormal tissue perfusion (pale mucous membranes, tachycardia, and weak pulses). In acute hemorrhage, the PCV may remain normal due to splenic contraction and the TP may drop first.

Patients with respiratory distress and/or cardiovascular compromise or acute abdominal pain or distension should have an abdominal FAST (FAST = focused assessment sonography for trauma, triage and tracking) and/or thoracic FAST examination performed as part of the initial assessment. This will allow evaluation for free abdominal, pleural and pericardial fluid and it takes only a few minutes. If fluid is identified this should be sampled by performing an abdominocentesis or thoracocentesis. During the thoracic FAST scan, global cardiac contractility can also be assessed and evaluation of left atrial size can be useful especially if congestive heart failure is suspected. The early identification of fluid will expedite the diagnosis of the underlying cause of the animal’s condition and will allow for additional stabilization measures to be preformed.

One final note about the triage/stabilization period: It is important that the pet owner receives updates during the stabilization process as this can be a period of high anxiety for the owner. This can be done by the veterinarian technician responsible for performing the triage. A simple statement to the owner regarding what is currently being done to help their pet, i.e “the doctor evaluated your pet and we gave him/her pain medications and fluids” will go a long way toward easing the pet owners fears while they are waiting.

Emergency drug doses you should know (and a little bit of the why)

1. Epinephrine: This drug is the first line therapy for animals with cardiopulmonary arrest. Currently the recommendation is to administer low dose epinephrine (0.01mg/kg IV). High dose epinephrine (0.1 mg/kg IV) can be given to animals with prolonged CPR (>10 minutes). Most hospitals stock epinephrine at a concentration of 1 mg/kg and therefore a quick rule of thumb is to administer 0.05 mls IV to a cat (5 kg), 0.1-0.2 mls to a medium sized dog (30-50 lbs) and 0.3 mls to a large breed dog (i.e labrador/golden retrievers sized dogs)

2. Atropine: This drug is used to treat a severe bradycardia (not caused by hyperkalemia) or in cardiopulmonary arrest. The dose is 0.04 mg/kg IV. Atropine generally comes in a concentration of 0.54 mg/ml and therefore some general guidelines are to administer 0.5 mls to a cat, 1 ml to a medium sized dog, and 3 mls to a large breed dog.

3. Dextrose: As stated above, a blood glucose level should be checked on any sick patient presenting on an emergency basis. Hypoglycemia can be seen in sick pediatric patients, sepsis, liver failure, neoplasia, Addison’s disease, insulin overdose or insulinoma and in some toxicities such as xylitol. Clinical signs of hypoglycemia include lethargy and weakness, altered mentation, seizures and coma. If hypoglycemia is confirmed or suspected, 0.25 to 0.5 G/kg of 50% dextrose diluted 1:3 with sterile saline or other crystalloid should be administered IV quickly. There is very little risk to treating hypoglycemia if you cannot confirm it with a blood glucose measurement. A quick rule of thumb is to administer 0.5 G/kg dextrose is to draw up 1 ml of 50% dextrose per kilogram of body weight. For example, a cat weighing 5 kg would be administered 5 mls of 50% dextrose diluted. A blood glucose should be checked to confirm that the hypoglycemia has been corrected and the patient should be placed on a continuous infusion of 2.5%-5% dextrose to prevent recurrence of hypoglycemia.

4. Lasix (Furosemide): This drug is used most commonly to treat congestive heart failure in the dog and cat. It is reasonable to administer an intramuscular dose of lasix to a dog (2-4 mg/kg) or cat (1-2 mg/kg) in respiratory distress that has a heart murmur and crackles auscultated especially when thoracic imaging is unable to be obtained or there will be a delay in obtaining imaging. The patient can then be placed in oxygen and the patient’s response to treatment can be assessed. Generally, this drug should induce a diuresis in 30-45 minutes after administration.

5. Midazolam: This drug is useful for treating seizures emergently. Midazolam tends to be dosed slightly lower than valium but most of the time these drugs can be used interchangeably especially when valium should never be given IM. The dose of midazolam is: 0.25-0.5 mg/kg or 0.25-0.5 mls for a cat, ~1 ml for a medium sized dog (15kg), ~3 mls for a large dog (30 kg). It is our practice to administer midazolam IM in a patient that is actively seizing and does not currently have intravenous access. This drug works rapidly to control the seizure activity and once the seizures are controlled it is much easier to obtain vascular access. Its anticonvulsant effect lasts for about 1 hour. All seizing patients should have blood electrolytes checked (Na and Ca), in addition to a blood glucose measurement and this blood can be drawn at the time of intravenous catheter placement.

6. Lidocaine: This drug is used to treat ventricular tachycardia. Treatment of ventricular tachycardia is indicated when there is sustained rate of 180 bpm or greater and/or there is evidence of abnormal perfusion. The dose of this drug is 2 mg/kg IV.
7. **Calcium Gluconate (10%)**: This drug is useful to treat stabilize a critically blocked cat that is having severe cardiac arrhythmias from hyperkalemia. This drug has membrane stabilizing effects by increasing that threshold potential which antagonizes the effect of hyperkalemia. The dose is to administer 1-3 mls/cat of 10% calcium gluconate IV (or 1-1.5 mls/kg) over 3-5 minutes while monitoring the ECG for improvement in the cardiac arrhythmias. This drug works rapidly but its effects will dissipate within about 20-30 minutes.

A crash course in fluid therapy

Fluid therapy is a cornerstone of treatment for many ill patients and one of the most prescribed therapies in the emergency setting. The following are some general guidelines and a few tricks of the trade for developing a fluid therapy plan.

- The first consideration is whether your patient is in shock with evidence of poor tissue perfusion. Signs of hypoperfusion can include pale mucous membranes, a prolonged CRT, tachycardia, decreased rectal temperature, cool extremities, dull mentation and poor or absent peripheral pulses. Cats in shock frequently are bradycardic rather than tachycardic although this is not seen in every case. When signs of poor tissue perfusion are present, shock fluid therapy is indicated.

  - For **dogs**, the shock volume is 90 mL/kg of an isotonic crystalloid, given in aliquots of approximately ¼ to 1/3 of the total volume to effect. A quick way to estimate the volume of a shock bolus for a dog is to **take the weight in pounds and add a zero**. For example, a 40 lb dog would receive 400 mL. This is roughly equivalent to a 22 mL/kg dose (about a ¼ shock volume).

  - For **cats**, the shock volume is 45-60 mL/kg of an isotonic crystalloid, given in aliquots of approximately ¼ to 1/3 of the total volume to effect. A quick way to estimate the volume of a shock bolus in a cat is to **take the weight in kilograms and add a zero**. A 5kg cat would thus receive 50 mL. This is roughly equivalent to ¼ of the shock volume.

  - For patients that are hypoproteinemic, a synthetic colloid like hydroxyethylstarch could be considered. The shock bolus dose of hydroxyethylstarch is 5 mL/kg in dogs (up to total volume of 20 mL/kg) and 3 mL/kg in cats (up to total volume of 15 mL/kg).

  - For rapid volume expansion, hypertonic saline can be considered. We will typically dilute the 23.4% hypertonic saline into hetastarch (1 part hypertonic saline to 2 parts hetastarch) and administer 3-5 mL/kg of this mixture **total** as a shock bolus. Multiple doses of hypertonic saline can lead to hypernatremia so we generally only provide this as a bolus one time.

- For patients with normal perfusion, the fluid therapy plan is calculated by adding the maintenance requirement, deficit and ongoing losses.

  - The maintenance rate can be estimated at 1-2 mL/kg/hr or 45-60 mL/kg/day; however, these formulae may overestimate the requirement for very large patients and underestimate the rate for very small patients. A more accurate formula for these patients is (BW in Kg)0.75 x 70 = mL of fluid per day. **Example**: The maintenance volume for a 50 kg dog would be (50)0.75 x 70 = 1316 mL/day = 55 mL/hour. If you don’t have a calculator that can raise the BW to the 0.75 power, you can cube the body weight and then hit the square root function twice.

  - Deficits are calculated based on assessment of dehydration. Estimated dehydration is quite subjective but as a general rule of thumb mild dehydration is assumed to be 5%, moderate dehydration 8%, and severe dehydration 10%. The deficit is calculated by multiplying the BW in kg by the deficit. **Example**: For a 50 kg dog that is 8% dehydrated, the deficit would be (50 kg x 0.08) = 4 Liters. This is replaced over 12-48 hours.

  - Ongoing losses can be challenging to anticipate! Measuring urine output is a cinch if a catheter is in place, but frequently this is not the case and we have to give our best guess as to the volume of urine, vomit, diarrhea, or other losses the patient is experiencing. One tip is to line the patient’s cage with and then weigh the wee pads – every gram they gain from urine, diarrhea, etc, corresponds to one mL of fluid lost by the patient. Another way to estimate the volume of a loss is to compare it to the amount that a standard Dixie cup holds, which is approximately 100 mL. When in doubt about the loss estimate, frequent reassessments and adjustments to the plan are indicated.

- For obese patients, use the **ideal body weight** for your fluid therapy prescriptions. This is particularly important in sick obese cats to avoid fluid overload.

- Neonates have a much higher maintenance fluid requirement than adults. A general rule of thumb is to estimate their maintenance rate at 4-6 mL/kg/hr. Lactated Ringer’s solution is the preferred crystalloid for neonates as the lactate can

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serve as a metabolic energy source when they are hypoglycemic. Intraosseous catheters are a great option for fluid therapy in these patients.

- Although there are differences in the composition of the various isotonic crystalloids, in most instances any isotonic crystalloid is acceptable. Situations where a specific crystalloid might be preferred include include hyper- or hyponatremia (generally should select a fluid that has a sodium concentration close to that of the patient), liver disease (avoid lactate containing fluids as the liver may not be able to effectively convert it to bicarbonate), and hypochloremic metabolic alkalosis (0.9% saline is preferred due to the high chloride content).
- Don’t forget the PO route! For patients that aren’t vomiting but are unwilling or unable to drink enough to maintain their hydration, or for those that are dehydrated but intolerant of IV fluid therapy (for example, a patient with cardiac disease) placing a nasoesophageal or nasogastric tube allows for the provision of enteral water.

- To quickly estimate the volume of a blood transfusion, a good rule of thumb is 2 mL/kg of whole blood (or 1-1.5 mL/kg of packed red blood cells) is expected to increase the PCV by 1%.
- To calculate a constant rate infusion for a drug dosed in mcg/kg/minute:
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  \text{mg of drug to add to fluid} = \left( \text{dose in mcg/kg/min} \right) \times (\text{BW kg}) \times (\text{volume of fluid to add drug to in mL}) \]
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  \text{mg of drug to add to fluid} = \left( \text{dose in mcg/kg/min} \times \text{BW kg} \times \text{volume of fluid to add drug to in mL} \right) \times \frac{1}{16.67} \]
- To calculate a constant rate infusion for a drug dosed in mg/kg/hour:
  \[
  \text{mg of drug to add to fluid} = \left( \text{dose in mg/kg/hour} \right) \times (\text{BW kg}) \times (\text{volume of fluid to add drug to in mL}) \]
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**Working up abdominal effusions**

Abdominal effusions are a common finding in ill pets brought to the emergency service. Patients with a large volume of effusion may have a pendulous abdomen with a ballotable fluid wave; however, small volumes of effusion may not be readily apparent on physical exam and are instead found during the workup typically via diagnostic imaging. Decreased organ serosal detail on abdominal radiographs may suggest the presence of free abdominal fluid but this finding can also be noted in very young patients or those that are cachectic and lacking intraabdominal fat. Radiographs may also suggest the etiology of effusion; for example, if a splenic mass is seen, the effusion may be hemorrhage, and if a pneumoperitoneum is observed, the effusion is likely septic.

Ultrasound, when available, is generally quite helpful in confirming the presence of an effusion and potentially in recovering a sample. Abdominal FAST (AFAST) is a quick and relatively simple technique used commonly in the emergency setting to rapidly identify patients with free abdominal fluid. AFAST is indicated in any patient presenting with signs of shock, abdominal pain, abdominal distention, or fever of unknown origin.

When free abdominal fluid is detected, obtaining a sample is a crucial part of the diagnostic process. Abdominocentesis may be accomplished using ultrasound guidance although a blind technique is often successful when a large volume of effusion is present. To perform a blind abdominocentesis, the patient is placed in lateral recumbency and the area cranial and caudal to the umbilicus is clipped and aseptically prepped. A 22 gauge needle attached to a 3 or 6 mL syringe is inserted into the abdomen at an angle perpendicular to the skin just caudal to the umbilicus and just off midline on the dependent side. A four quadrant abdominocentesis can be attempted if single abdominocentesis is unsuccessful at recovering fluid. This procedure involves performing abdominocentesis in four locations; at a distance of 2 centimeters cranial to and caudal to the umbilicus, abdominocentesis is performed 2 cm lateral to midline on the right and left side. It should be noted that severely dehydrated or hypovolemic patients with a small volume of abdominal effusion may develop a larger volume of effusion once fluid resuscitated which may facilitate retrieval of a diagnostic fluid sample.

A variety of diagnostic tests may be employed in the workup of an abdominal effusion. When a hemorrhagic effusion is obtained, a sample should be placed into an empty red top tube and observed for clotting and the PCV of the effusion is measured and compared to the peripheral PCV. A non-clotting hemorrhagic effusion with a PCV approaching or even exceeding the patient’s peripheral PCV is diagnostic for hemoperitoneum. Hemoperitoneum may be the result of trauma; in the absence of trauma, spontaneous hemoperitoneum most commonly results from either a bleeding mass or systemic coagulopathy. Cytologic evaluation of hemorrhagic effusions typically has very low diagnostic yield.

Non-hemorrhagic effusions are classified on the basis of cellularity and protein concentration. Pure transudates are typically transparent, have a low protein concentration and are relatively acellular, most commonly resulting from hypoalbuminemia. Modified transudates are often transparent but may have a red tinge, have a protein concentration between 2.5 and 5 g/dL and a cell count between 300-5500 cells/uL. Common causes include right-sided congestive heart failure, heartworm infection, liver disease and neoplasia. Exudates are characterized by a high protein and cell count and are often cloudy in appearance – these effusions are often associated with either a neoplastic process or septic or nonseptic peritonitis. Microscopic evaluation is warranted and the presence of
neutrophils with intracellular bacteria is diagnostic for septic peritonitis. When septic peritonitis is suspected, glucose and lactate in the abdominal fluid can be measured and compared to that in peripheral blood. Abdominal fluid glucose that is 20 mg/dL less than peripheral blood glucose, and abdominal fluid lactate that is 2 mmol/L greater than peripheral blood lactate, is suggestive of septic peritonitis.

Creatinine, potassium and bilirubin may also be measured in abdominal fluid and compared to values from a peripheral blood sample. A fluid to blood creatinine ratio of >2:1 or fluid to blood potassium ratio of >1.4-2:1 are consistent with uroperitoneum. Similarly, a fluid to blood bilirubin ratio of >2:1 is suggestive of bile peritonitis.

Updates on CPCR
In 2012, the RECOVER working group published the first set of evidence-based guidelines for veterinary CPCR. These clinical guidelines were published in the Journal of Veterinary Emergency and Critical Care and are freely available online. Some highlights from the guidelines are listed below:

1. Chest compressions should be initiated as soon as possible and are best performed with the patient in lateral recumbency with its back to the rescuer. Barrel chested dogs (bulldogs, for example) are the exception – these patients should be placed in dorsal recumbency with compressions performed over the sternum. Compressions are performed over the heart in small dogs and cats, as well as in larger, keel-chested dogs; otherwise, compressions are performed over the widest part of the chest. Compress down 1/3 the width of the chest and make sure to allow for full elastic recoil. Aim for a rate of 100-120 uninterrupted compressions per minute (think Staying Alive!) and rotate in a new compressor every 2 minutes.

2. Practice intubation in lateral recumbency so that an airway can be secured whilst compressions are occurring. Administer one breath every 6 seconds and try to avoid hyperventilation as this may negatively affect venous return to the heart. Ambubags are quite helpful – they are portable, will interface with oxygen tubing and have a built-in pop off valve. If using an anesthesia machine to provide breaths, aim for a tidal volume of approximately 10 mL/kg and make sure to keep the pressure below 20 mmHg to avoid pulmonary injury.

3. Epinephrine and atropine may be administered every 3-5 minutes at the dose noted earlier in this document. Vasopressin may be used as an alternative to epinephrine at a dose of 0.8 u/kg given every 3-5 minutes. Theoretically this medication may be more effective than epinephrine in an acidemic environment however large studies have failed to find evidence for superiority of vasopressin. Ideally these medications are administered IV followed by a large volume of fluid flush to push them centrally; however, if an IV access is not available all may be diluted in saline and administered intratracheally via a red rubber catheter placed down the endotracheal tube between breaths. Optimal dosing for intratracheal medication is unknown but it is common to either double the intravenous dose or, in the case of epinephrine, use the high dose (0.1 mg/kg).

4. If an ECG is attached, the best time to look at it is during the brief period when compressors are being rotated. If ventricular fibrillation is occurring, defibrillation is recommended at an initial dose of 2-4 J/kg (biphasic defibrillator). Safety is paramount! Make sure no one is touching the table or the patient before administering a shock. Following a shock, immediately restart compressions and re-evaluate the rhythm at the end of a 2 minute compression cycle. If ventricular fibrillation is still occurring, increase the defibrillation dose by 50%. If no defibrillator is available, a precordial thump over the heart can be attempted.

5. End tidal CO2 (ETCO2) is a great monitoring tool as it provides a real-time assessment of the adequacy of the resuscitation effort. Return of spontaneous circulation may be more likely to occur when ETCO2 is greater than 15-20 mmHg.

6. During CPCR consider checking blood electrolytes and glucose to look for reversible causes of cardiopulmonary arrest like hypoglycemia or hyperkalemia.

7. If return of spontaneous circulation occurs, efforts are aimed at hemodynamic stabilization. Fluids, vasopressors or positive inotropes may be required to maintain normotension. One tip is to add 1 mL of epinephrine (1 mg/mL concentration) to 100 mL of 0.9% NaCl and administer via slow drip to effect to improve blood pressure and cardiac output. Supplemental oxygen is provided to maintain normoxia. Blood gas evaluation is helpful to assess ventilation; patients may require positive pressure ventilation to prevent hyper- or hypocapnea. Avoid aggressive rewarming in hypothermic patients and consider a dose of mannitol (0.5 g/kg IV) to help resolve cerebral edema. If mannitol is administered, monitor for hypotension that may result from its diuretic effect.