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 dexmedetomidine (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. Dexmedetomidine 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, zolazepam (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 dexmedetomidine 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, butorphanol, and buprenorphine. Hydromorphone, methadone, oxymorphone and morphine are mu 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 mu agonist/ K antagonist, meaning that it will reverse some mu opioid effects. These provide less potent analgesia as compared to the primary mu agonists and should be used only for mild pain or short-term pain.

Buprenorphine is considered a partial mu 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) 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>Dilauid, 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>Drug</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>Acp is not an effective sedative</td>
</tr>
<tr>
<td>Alfaxaline 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>0.2–0.4mg/kg SQ, IM, IV</td>
<td>0.2–0.4mg/kg SQ, IM, IV</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>375 IV; 500 IM micrograms/m²</td>
<td>40 micrograms/kg IM micrograms/m²</td>
</tr>
<tr>
<td>Etomidate mg/kg</td>
<td>1–2 mg/kg</td>
<td>1–2 mg/kg</td>
</tr>
<tr>
<td>Drug</td>
<td>µg/kg</td>
<td>CRI: 0.1–0.7 µg/kg/min</td>
</tr>
<tr>
<td>-------------------</td>
<td>-------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Fentanyl</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>0.05–0.2</td>
<td>SQ, IM, IV</td>
</tr>
<tr>
<td>Methadone</td>
<td>0.1–1.0</td>
<td>SQ, IM, IV</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.1–0.5</td>
<td>SQ, IM, IV</td>
</tr>
<tr>
<td>Morphine</td>
<td>0.1–1.0</td>
<td>SQ, IM, IV</td>
</tr>
<tr>
<td>Propofol</td>
<td>1–6</td>
<td>IV</td>
</tr>
</tbody>
</table>

**References**


All Bleeding Stops Eventually: Common Coagulopathies
Garret Pachtinger, DVM, DACVECC
Veterinary Specialty and Emergency Center
Levittown, PA

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 chloramphenicol. 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.

Thrombocytopenia
Thrombocytopenia, a platelet dysfunction, is an uncommon clinical problem. Patients that have a thrombocytopenia 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, thrombocytopenia may be congenital, related to medications, or acquired subsequent to other diseases. Drug induced thrombocytopenia 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 Von Willebrand’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 thrombocytopenic disorders are rare, but have been reported in the otter hound, bassett 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 K 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**

Cardiovascular Emergencies in the ER/ICU
Garret Pachtinger, DVM, DACVECC
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Levittown, PA

Cardiac diseases commonly seen in the small animal emergency room include congestive heart failure (mitral or tricuspid regurgitation, hypertrophic cardiomyopathy, dilated cardiomyopathy), myocardial failure (dilated cardiomyopathy, end-stage heart disease), pericardial effusion, arrhythmias, and aortic thromboembolism in cats secondary to HCM.

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.

Emergency therapy
Emergency management of the patient presenting with respiratory distress includes systemic oxygen delivery and minimizing patient stress. While flow-by and oxygen mask oxygen delivery will allow concurrent patient assessment, there are times when other methods of oxygen delivery are needed.

Oxygen supplementation techniques

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<td>Nasotracheal catheter</td>
<td>25-50 ml/kg/min</td>
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Once initial patient assessment is made, a more thorough physical examination is essential in the diagnosis and management of emergency cardiac patient.

Congestive heart failure
Patients with congestive heart failure often present in respiratory distress. Common examination findings include an increased respiratory rate and effort. If pulmonary edema is present, auscultation commonly is reported to have pulmonary crackles. More common in feline patients, dull lung sounds may be present ventrally with pleural effusion. Ascites may also be present with right sided heart failure as a result of tricuspid regurgitation, DCM, or heartworm disease. Other common physical examination findings include auscultation of a heart murmur, hypothermia in cats, pale mucous membranes, and other signs of respiratory distress (i.e. extension of the head/neck abduction of the elbows, and reluctance to lay down). Although uncommon in dogs, absent femoral pulses, cold rear extremities, and hind limb paresis are seen with aortic thromboembolism (ATE), seen most commonly as a consequence of hypertrophic cardiomyopathy (HCM) in cats.

Along with the history and physical examination, diagnostics to consider include blood pressure, pulse oximetry, thoracic radiographs, or thoracic ultrasound. Before performing diagnostics, it is important to make sure the patient is stable and can tolerate the diagnostics without a risk of decompensation.

Thoracic radiographs are often considered the mainstay diagnostics in evaluating the heart and lungs. In fulminant congestive heart failure, radiographs commonly show congestion/distension of the pulmonary vessels and interstitial to alveolar pulmonary infiltrates.
In dogs, the pulmonary interstitial to alveolar disease is often seen in the perihilar area while cats may have a more generalized pulmonary patent of edema. Specific cardiac disease may also become more apparent with the use of radiographs, notably a large, globoid heart with dilated cardiomyopathy (DCM) or pericardial effusion. When performing thoracic radiographs, at least two views should always be taken with many cardiologists preferring a lateral view and dorsoventral (DV) view.

While thoracic radiographs often confirm the diagnosis of CHF, thoracic ultrasound is an upcoming diagnostic in the ER. Along with the TFAST and AFAST, a new term, “Vet Blue” has recently been discussed. Using these ultrasound techniques, lung pathology is assessed based on the distinction between wet (ultrasound lung rockets (ULRs) vs. dry lung (A-lines with a glide sign). The goal of using this technique is to provide rapid, point-of-care global evaluation of the emergent patient with minimal restraint and risk of decompensation. For the TFAST (Vet Blue) procedure, the patient is placed in either right lateral recumbency and/or sternal recumbency. Dorsal recumbency is not recommended as it has not been validated for VetBlue and it also may increase patient stress. The Vet Blue "L"ung Scan (VBLS, and "blue" for cyanosis, "L" for the scan pattern) is a rapid respiratory evaluation primarily based on the concept of wet vs. dry lung. In human patients lung ultrasound has been shown to be superior to chest auscultation for the detection pulmonary pathology. The goal of the VBLS scan is to identify pulmonary patterns that improve the speed of diagnosis for the respiratory distressed patient preempting the stress of thoracic radiography.

The initial treatment of congestive heart failure will vary slightly depending on the specific patient as well as specific diagnosis but involves oxygen, furosemide (1-4 mg/kg IV as often as every 1-2 hours initially for fulminant edema), and monitoring including blood pressure, pulse oximetry, hydration status, electrolyte status, and renal status. In severe case, sodium nitroprusside may be considered. Sodium nitroprusside is a balanced vasodilator effective in reducing pulmonary edema by increasing venous capacitance and reducing ventricular afterload. The dose is 0.5-10 μg/kg/min IV as a CRI. The author starts at a dose of 1-2 μg/kg/min and increases based on the response to therapy by 1 μg/kg every 20-30 minutes until there is an improvement in respiratory rate, effort and thoracic auscultation. When using sodium nitroprusside, blood pressure must be monitored as it may cause moderate to severe hypotension.

In cases of low output failure (weak pulses, pale membranes, slow CRT, weakness, hypothermia, azotemia), dobutamine is a synthetic beta-adrenergic agonist is considered. This is commonly used in patients with DCM. Dobutamine has a dose range of 2-20 mcg/kg/minute. At lower doses, dobutamine improves cardiac contractility with minimal effects on chronotropy or heart rate. At higher doses, however, dobutamine can be pro-arrhythmogenic. Pimobendan (0.25 mg/kg PO BID) has been used with success in dogs with CHF secondary to DCM and mitral valve insufficiency. Pimobendan is a phosphodiesterase-III inhibitor that sensitizes the myocardium to calcium, and improves inotropic activity in addition to causing arteriolar and venous dilation. In addition to its use as a long-term inodilator in the treatment of dogs with CHF, Pimobendan is also recommended for use in emergency therapy of CHF, as it can have an onset of effects within one hour.

Cardiac tamponade
Cardiac tamponade results from the pressure of pericardial effusion on the heart leading to decreased filling, decreased cardiac output, and ultimately left and right heart failure. The degree of pressure exerted by the pericardial effusion depends on several factors. These include the volume of pericardial effusion, the rate of pericardial fluid accumulation, and the distensibility of the fibrous pericardium. In the author’s opinion, there are two common presentations of pericardial effusion. Patients presenting with acute cardiac tamponade often have a small volume of pericardial effusion (50–100 ml) which causes marked intrapericardial pressure and cardiac tamponade. However, we do also see patients with a more chronic, slower accumulation where there is increased compliance, allowing the pericardial sac to accommodate a significantly larger amount of fluid before intrapericardial pressure increases enough to result in cardiac tamponade.

Clinical signs of patients suffering from pericardial effusion may include tachycardia, tachypnea, poor or absent femoral pulses, pulsus paradoxus, jugular venous distension, dull heart sounds, exercise intolerance, weakness, and syncope. If more chronic in nature, patients may display signs of right-sided congestive heart failure including hepatomegaly, ascites, and jugular venous distension.

Aside from the traditional diagnostics listed above, echocardiography is recommended for the diagnosis of pericardial effusion. Pericardial effusion is diagnosed by the presence of hypoechoic fluid between the epicardium and the pericardium.

Diagnostic and therapeutic pericardiocentesis is indicated in patients with pericardial effusion.

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 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.

**Life-threatening arrhythmias**

The most common arrhythmia the small animal veterinarian will see is a tachyarrhythmia. These are also considered to be the most concerning as tachyarrhythmia’s require increased oxygen consumption and lead to reduced diastolic filling and coronary artery perfusion. Underlying causes of tachyarrhythmia’s include shock, anemia, hypoxia, hyperthyroidism, infection, inflammation, and pain. Supraventricular tachycardias should improve with treatment and resolution of the underlying cause (i.e. fluid therapy for hypovolemic shock or oxygen therapy for hypoxemia). If the heart rate does not decrease with appropriate therapy, a vagal maneuver can be attempted by applying pressure to the eyes or carotid sinus pressure. If there is no improvement despite appropriate therapy and despite a vagal maneuver, drug therapy is considered, notably digoxin. Other antiarrhythmics which may be effective include propranolol (20-60 mcg/kg IV slowly over 5-10 min.) or verapamil (.05 mg/kg IV q 10-30 min, up to 3 times). Both of these are negative inotropes and should be used with caution if there is concurrent evidence of congestive heart failure. Intravenous diltiazem (0.25 mg/kg administered slowly over 3 minutes) can be used instead of verapamil to control supraventricular tachycardias.

Ventricular tachycardia is another common arrhythmia seen, associated with primary cardiac disease or secondary to systemic disease. The arrhythmia is treated pharmacologically if signs of hemodynamic instability are present, notably with EKG findings including tachycardia (>160bpm), multiform QRS configurations, R on T phenomenon, and or hypotension. Lidocaine is the drug of choice for ventricular arrhythmias, dosed initially with a bolus of 2-4mg/kg IV given slowly to effect while monitoring the electrocardiogram. This bolus is followed by a CRI (25-80 μg/kg/min). Refractory ventricular arrhythmias can be treated with procainamide (2-15 mg/kg IV over 20-30 minutes).

Bradyarrhythmias are not as commonly seen in clinical practice, although bradycardia as a result of hyperkalemia seen with (feline) urethral obstruction is often seen. Aside from hyperkalemia as a result of urethral obstruction in male cats, other common causes include hypoadrenocorticism and renal failure. Treatment will depend on the underlying cause, but for hyperkalemia may include fluid therapy, Calcium gluconate (0.2-0.5 ml/kg IV), regular insulin (0.25 U/kg IV), dextrose (0.5g/kg), or sodium bicarbonate (1-2 mEq/kg IV slowly).

**Summary**

Patients presenting with evidence of emergent cardiac disease should be triaged quickly and treated immediately to reduce morbidity and mortality. Oxygen is a mainstay therapy for cardiac patients and should be administered on presentation and during the initial assessment phase. Prognosis will vary on the underlying cause of disease although patients may live for several years with careful monitoring.
### Common medications, dosages and indications

<table>
<thead>
<tr>
<th>Key Drug</th>
<th>Drug Class</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>Diuretic</td>
<td>2-4 mg/kg</td>
<td>Every 2-4 hours as needed, then every 8 hours</td>
<td>IV best in patients with CHF; IM Topical</td>
<td>Pulmonary edema due to CHF</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Vasodilator</td>
<td>1/8” strip on ear pinnae</td>
<td>Every 6 hours</td>
<td>Topical</td>
<td>Vasodilation to decrease afterload on the heart</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>β-1 agonist</td>
<td>2-10 μg/kg/min</td>
<td>CRI</td>
<td>IV</td>
<td>Positive inotrope to increase cardiac output in patients with primary myocardial failure</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Class I antiarrhythmic</td>
<td>40–80 mcg/kg/min</td>
<td>CRI</td>
<td>IV</td>
<td>Ventricular tachycardia</td>
</tr>
<tr>
<td>Procaainamide</td>
<td>Class I antiarrhythmic</td>
<td>25–40 mcg/kg/min</td>
<td>CRI</td>
<td>IV</td>
<td>Ventricular tachycardia</td>
</tr>
<tr>
<td>Diltiazem</td>
<td>Calcium channel blocker</td>
<td>0.25 mg/kg slow bolus</td>
<td>Q 20 min</td>
<td>IV</td>
<td>SVT +/- atrial fibrillation</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Beta-blocker</td>
<td>0.01 mg/kg slow bolus</td>
<td>Q 5 min</td>
<td>IV</td>
<td>SVT</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>Nitrate</td>
<td>2 to 10 mcg/kg/min</td>
<td>CRI</td>
<td>IV</td>
<td>Refractory CHF</td>
</tr>
</tbody>
</table>

**References**

Abdominocectesis

Abdominocectesis 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/l (1 x 10⁹/l)), are commonly due to causes including hypoalbuminemia and early congestive heart failure. Modified transudates (protein concentration < 35 g/l, cell count < 5000/l (5 x 10⁹/l)) 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/l (5 x 10⁹/l)), are found with causes including sepsis, feline infectious peritonitis (FIP), neoplasia, lung-lode 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 abdominocectesis 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 abdominocectesis, the patient is placed in left lateral (to allow the spleen to fall away from midline) or sternal recumbency. Using the prepared abdominocectesis 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-sump 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 cuppiges 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 benzodiazepaine 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 that 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 lifesaving 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 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 benzodiazepaine. The lateral cervical area is clipped and aseptically prepared from the ventral ramus of the mandible caudally to the thoracic inlet and dorsally 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.

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In health, our small animal veterinary patients have a very small amount of fluid within body cavities. We cannot see this radiographically, and ultrasound alone may not detect this fluid in health. The main goal of this fluid is to lubricate the surfaces of the organs and body walls like motor oil for your car engine. This allows the organs to glide over each other without friction, avoiding inflammation. That is in health. In states of disease we see effusion develop which needs to be identified and characterized for both diagnosis and targeted treatment.

Effusions are generally characterized into one of 3 categories:
- Transudate
- Modified Transudate
- Exudate

**Transudates**
Transudates are often found as a result of either increased hydrostatic pressure or decreased colloid osmotic pressure. Either of these situations will alter fluid balance resulting in effusion. By definition, transudate fluids are often low protein (< 2.5 g/dL) and low or absent in regards to cellularity (nucleated cell count < 2500/µl). Common causes of transudative effusions include congestive heart failure, liver failure, nephrotic syndrome, and in some cases of neoplasia.

**Modified transudates**
Modified transudates are identified when there is a mild increase in both total protein (typically 3.0-5.0 g/dl) and nucleated cell count (>2500/µl and less than 5000/µl). Modified transudates are commonly seen when a transudate has been chronic, resulting in an inflammatory reaction. As compared to the often clear gross appearance of a transudate, a modified transudate may appear cloudy in appearance. Common causes of modified transudates include increased hydrostatic pressure, right-sided congestive heart failure, left-sided congestive heart failure in cats, decreased colloid osmotic pressure (i.e. Hypoalbuminemia), lymphatic obstruction, and neoplasia.

**Exudates**
Exudates are identified when there is an abnormally high total protein and nucleated cell count. Total proteins range between 3.0 and 7.0 g/dl and total cell counts are typically greater than 5000/µl. Common causes of exudates include inflammation, hemorrhage, chylous, and neoplasia.

<table>
<thead>
<tr>
<th>Effusion type</th>
<th>Gross appearance</th>
<th>Total protein (g/dl)</th>
<th>Total nucleated cell count</th>
<th>Cell population</th>
<th>Other important findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transudate</td>
<td>Clear to colorless</td>
<td>&lt; 2.5</td>
<td>&lt; 2,500 cells/µl</td>
<td>Mononuclear</td>
<td>Reactive mesothelial cells</td>
</tr>
<tr>
<td>Modified transudate</td>
<td>Clear to cloudy, yellow to red</td>
<td>&gt; 2.5</td>
<td>&lt; 5,000 cells/µl</td>
<td>Mononuclear</td>
<td>Reactive mesothelial cells</td>
</tr>
<tr>
<td>Exudate</td>
<td>Hazy to opaque</td>
<td>&gt; 2.5</td>
<td>&gt; 5,000 cells/µl</td>
<td>Neutrophils, neoplastic, other</td>
<td>+/- other inflammatory cells</td>
</tr>
</tbody>
</table>

While classifying the effusion based on cell count and protein level may seem tedious, it can be quite helpful in developing a differential list and therefore a more specific diagnostic and treatment plan.

**Fluid collection and further evaluation**
When abnormal fluid (effusion) is suspected, either a blind centesis procedure can be performed (without ultrasound) or an ultrasound guided centesis (thoracocentesis or abdominocentesis) can performed. Focused assessment using sonography for triage, tracking, and trauma (FAST®) is a newly describe technique for rapid ultrasound technique to identify free fluid.

Once the fluid sample is collected, it is recommended to prepare the sample for either in-house evaluation of submission to the diagnostic laboratory for further evaluation.

If the sample is being submitted to an outside diagnostic laboratory, fluid should be placed in an EDTA tube and red top tube. The EDTA collected sample can be submitted for RBC count, nucleated cell counts, cytology, or other potential tests including flow cytometry and PCR testing. A red top tube can be saved for other diagnostics (i.e. aerobic and anaerobic bacterial culture,
mycoplasma, and fungal cultures, total protein, albumin, bilirubin, creatinine, potassium, triglyceride, glucose, lactate, and lipase). The author also prefers to use some of the fluid to prepare a slide for evaluation as delay in fluid sample submission may lead to artifactual changes in cell morphology when in the sample tubes.

While certain disease conditions may permit delay in diagnostic results, there are conditions which should be evaluated on a more emergent, in-house basis.

**Septic effusion**

Septic effusion, notably a septic abdominal effusion, is typically considered a surgical emergency and therefore delay in sample submission and evaluation may increase both morbidity and mortality. Septic abdominal effusions can be seen as a result of a ruptured gastrointestinal tract due to causes including trauma and neoplasia. When evaluating a septic effusion, in-house cytology will often demonstrate a markedly suppurative effusion with abundant neutrophils as well as bacterial, typically intracellular bacteria. While the author uses cytology as the gold standard in-house test for immediate patient assessment, supportive diagnostic testing may include comparison of abdominal fluid and peripheral blood lactate and glucose concentrations. When using lactate as a clinical tool, a septic patient will have an abdominal fluid lactate 2 times the level of the peripheral blood lactate on a paired sample. When comparing the patient’s glucose levels, a septic patient will have an abdominal fluid glucose concentration 20mg/dL lower than the paired peripheral glucose sample. Due to the disparity which may be seen in certain conditions such as severe suppurative disease processes, cytology remains the most reliable test.

**Uroabdomen**

The most useful evaluation of effusion for identification of a uroabdomen is creatinine, combined with potassium. Blood urea nitrogen is not as helpful when evaluating for the presence of a uroabdomen. A patient with a uroabdomen will have a creatinine level in the abdominal effusion that is two or more times greater than serum creatinine. Potassium concentrations consistent with a uroabdomen are greater than 1.4:1 (canine) and 1.9:1 (feline).

**Bile peritonitis**

Patients with a bile peritonitis will often have a green-tinged fluid present, although this may be masked by hemorrhage or other processes based on the underlying disease process. Bilirubin crystals may be seen in the fluid as well. A patient with a bile peritonitis will have a bilirubin level in the abdominal effusion that is two or more times greater than serum bilirubin, although in the authors experience the effusion bilirubin is often significantly higher than in the serum.

**Hemorrhagic effusions**

Hemorrhagic effusions, seen with causes such as neoplasia, coagulopathy, and trauma, commonly have a PCV > 10%. Following aspiration of hemorrhage effusion, an attempt should be made to place the effusion in a red top tube. The fluid obtained should NOT clot. Clotting fluid would be concerning for the inadvertent aspiration of an organ such as the spleen or liver when performing an abdominocentesis.

**References**

Hypoadrenocorticism is an endocrine disease process that results from a deficiency of both glucocorticoid and mineralocorticoid secretion from the adrenal gland. A majority of our clinical hypoadrenocorticism patients suffer from primary hypoadrenocorticism, also known as Addison’s disease. The main cause of hypoadrenocorticism is theorized to be an autoimmune destruction of the adrenal cortex. At least 90-95% of adrenocortical tissue must be destroyed before clinical signs develop. Typically, the destruction involves all three zones of the adrenal cortex and results in both glucocorticoid (cortisol) and mineralcorticoid (aldosterone) deficiency.

**Pathophysiology of hypoadrenocorticism**

The adrenal gland is comprised of an outer cortex and the inner medulla, with the outer cortex subdivided into three layers. The outer layer (zona glomerulosa) is involved with synthesis and secretion of the mineralocorticoid hormone, aldosterone. The middle layer (zona fasciculata) synthesizes glucocorticoids, and the inner layer (zona reticularis) produces adrenal sex steroids. The adrenal medulla, which is not affected in hypoadrenocorticism, secretes catecholamines such as epinephrine and norepinephrine.

Hypoadrenocorticism results from atrophy or destruction of the adrenal cortex and may be classified as either primary or secondary. **Primary hypoadrenocorticism** results from bilateral destruction of the adrenal cortices presumed in most cases to result from immune-mediated destruction of the adrenal gland. Less common causes of primary hypoadrenocorticism include trauma (e.g., surgical versus other), infections (e.g., fungal or bacterial), neoplasia, or following medical therapy (e.g., mitotane, trilostane, ketoconazole, megestrol acetate, etc.). **Secondary hypoadrenocorticism** results from lack of adrenal gland stimulation due to hypothalamic-pituitary adrenal axis dysfunction, which most commonly results from inflammation, tumors, or trauma. Exogenous steroid administration may also suppress ACTH release, resulting in adrenal atrophy.

**Signalment**

With hypoadrenocorticism, certain breeds of dogs are over-represented, including Standard Poodles, Great Danes, Rottweilers, West Highland White terriers, Wheaten terriers, Leonbergers, Portuguese Water Dogs, Labrador Retrievers, Bearded Collies, Old English Sheepdogs, and Standard Schnauzers. Hypoadrenocorticism is also seen more in young to middle aged female dogs.

**Clinical signs**

Common clinical signs include lethargy, inappetence, vomiting, diarrhea, bradycardia, hypotension, weight loss, and rarely, death. Cortisol is required in almost all tissues of the body and its deficiency is associated with stress intolerance, weakness, gastrointestinal signs, and hypotension.

**Clinicopathologic findings**

Clinicopathologic findings seen with hypoadrenocorticism include the failure to mount a stress leukogram (resulting in eosinophilia, lymphocytosis, and normal overall white blood cell and neutrophil count) and electrolyte abnormalities secondary to direct aldosterone effects (e.g., hyperkalemia, hyponatremia, hypochloremia, metabolic acidosis). Other common laboratory abnormalities include azotemia, isosthenuria (from osmotic diuresis secondary to sodium losses), hypoglycemia (due to impaired gluconeogenesis), hypercalcemia (due to altered renal excretion, reduced gastrointestinal absorption, and decreased resorption of calcium from bone), hypoalbuminemia, and hypocholesterolemia.

**Endocrine testing**

The ACTH stimulation test is commonly used to confirm the presence of hypoadrenocorticism. Typically, a cortisol level is drawn followed by intravenous ACTH (tetracosactrin) administration. This is followed by a paired cortisol sample following ACTH administration.

**Treatment**

Treatment for the critically ill hypoadrenocorticism patient should include symptomatic supportive care, aggressive fluid therapy, correction of electrolyte abnormalities and hypoglycemia, anti-arrhythmic therapy (if needed), steroid administration and mineralocorticoid supplementation, if needed.

Aggressive intravenous fluid therapy using an isotonic crystalloid should be used in the acute crisis. While some prefer the use of 0.9% NaCl, the author understands that not all practices will have an array of isotonic crystalloid options. Regardless of which crystalloid is chosen, it is important to monitor the sodium concentration and ensure it does not increase by more than 10–15 mmol/l in the first 24 hours. Dextrose may be required if the patient is hypoglycemic.
- Glucocorticoid therapy should be used early in the treatment of the acute crisis. Glucocorticoid options in the acute crisis include:
  - Hydrocortisone sodium succinate: 10 mg/kg IV repeated every 3–6 hours or as a constant rate infusion of 0.5 mg/kg/hour
  - Prednisolone sodium succinate: 5 mg/kg IV repeated every 3–6 hours
  - Dexamethasone sodium phosphate: 0.1–0.2 mg/kg IV given once

The author prefers to perform the ACTH stimulation test prior to the use of glucocorticoid therapy. If the patient health status does not permit waiting, dexamethasone sodium phosphate should be used as the other preparations cross-react with cortisol in the assay.

**Chronic primary hypoadrenocorticism (maintenance therapy)**

Once the patient is more stable, and the ACTH stimulation test confirms the diagnosis, the author will institute mineralocorticoid therapy. Options include fludrocortisone acetate (e.g., Florinef) and DOCP. Fludrocortisone acetate is an oral synthetic adrenocortical steroid with mineralocorticoid effects. An initial dose of 15 mcg/kg/day of fludrocortisone is given and serum electrolytes measured after 5 to 7 days. The dose rate should then be adjusted until the sodium and potassium levels are within the normal range. The daily maintenance dose required is usually between 15 to 30 mcg/kg/day. Alternatively, desoxycorticosterone pivalate (DOCP) is a slowly absorbed mineralocorticoid administered subcutaneously or intramuscularly at 21–28 day intervals. The dose for DOCP is 2.2 mg/kg per treatment. Mineralocorticoid dose should be adjusted to maintain normal sodium and potassium.

**In summary**

Clinicians should be able to rapidly recognize the “great pretender” based on history, signalment, clinical signs, and classic clinicopathologic testing. Rapid and appropriate diagnostic workup should be performed (e.g., baseline cortisol, ACTH cortisol evaluation) to rule out other “lookalike” diseases such as metabolic disorders (e.g., renal disease, pancreatitis), toxicosis (e.g., grapes, etc.), infectious disease (e.g., Leptospira, urinary tract infection, pyelonephritis), etc.

Without treatment, hypoadrenocorticism can be life threatening due to dehydration, hypovolemia, severe electrolyte derangements, and ongoing fluid losses. In order to ensure the best outcome, the rapid identification and recognition of the hypoadrenocorticism state should be made. Appropriate use of steroids needs to be weighed as not to impair diagnostic testing for baseline cortisol levels or for future ACTH stimulation tests.

While long-term management may be cumulatively expensive (e.g., prednisone, periodic electrolyte monitoring, and mineralocorticoid supplementation), the prognosis for hypoadrenocorticism is good to excellent with medical management.

**References**


Urethral obstruction is a common, yet challenging case for veterinarians and a potentially life-threatening manifestation of feline lower urinary tract disease.

History and clinical signs
Cats with lower-urinary-tract disease have common historical findings which may include stranguria, pollakiuria, licking of the genital area, hematuria, passage of only small amounts of urine, vocalizing (when urinating or posturing), inappropriate urination, as well as anorexia, lethargy, and vomiting. It is important that the clinician recognize that owner perception of posturing may be difficult as many owners misinterpret posturing for straining to defecate or constipation.

Most cats with lower-urinary-tract signs are between 2 and 6 years of age. True feline urethral obstruction occurs primarily in male cats and are commonly young to middle aged. Cats presenting to the veterinarian for the first time with lower-urinary-tract signs that are older than 8 years of age should be evaluated for the presence of urinary-tract infection, bladder/urethral stones and neoplasia, not just assumed to have idiopathic sterile cystitis.

Examination
On any feline patient, especially male, where there is a concern for the signs noted above, the bladder should be palpated immediately. A small, soft bladder is difficult to palpate in an otherwise healthy cat makes urethral obstruction unlikely. While the size of the bladder may vary, even potentially being small, cats with urethral obstruction typically have a firm and painful bladder, regardless of the size when obstructed. The clinician can also evaluate the tip of the penis if concerned about the patient. In cases of feline urethral obstruction, the penis is typically red or purple in color and a urethral plug or grit may be seen at the urethral opening.

Following palpation of the bladder, a complete physical exam should be performed. In cats with urethral obstruction, the heart rate and rhythm as well as evaluation for the presence of a heart murmur should be evaluated. Bradycardia (a heart rate < 140 bpm) should alert the clinician to the likely presence of hyperkalemia. A heart murmur or arrhythmia may also change your sedation protocol as well as long term treatment plan including fluid therapy. Using other simple tools, hypothermia is common in sick patients and the combination of bradycardia 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.

Stabilization of the blocked cat
Following history, examination, and diagnosis of urethral obstruction (based on clinical signs and physical examination findings), a stabilization protocol should be immediately performed.

A peripheral intravenous catheter is immediately placed, blood work performed, and fluid therapy started. The bloodwork, at minimum, should include an extended database (PCV/TP/GLU/AZO/Electrolytes +/- Blood gas). Common findings of the affected blocked cat may include metabolic acidosis, hyperkalemia, and azotemia.

Another diagnostic to consider in the emergency setting is the use of an electrocardiogram. Cardiac arrhythmias and bradycardia should be swiftly documented and treatment started if present.

Treatment of hyperkalemia
The author typically uses the therapies below in this order:

1) Intravenous fluid therapy
   a. Isotonic crystalloids are used to improve perfusion, hydration, and dilute the potassium. Even isotonic crystalloids containing small amounts of potassium (e.g. Normosol R or Plasmalyte) are acceptable as the small amount of potassium is unlikely to cause a worsening of the hyperkalemia. Moreover, the fluid therapy will improve perfusion and hydration, with the goal of resolving the described metabolic acidosis.
   b. Calcium gluconate is a potentially life saving therapy for hyperkalemic patients. Calcium gluconate increases the cell's threshold membrane potential reestablishing the normal difference between resting membrane potential and threshold potential. This will allow the cardiac cells to depolarize.
      i. Dose: 3 ml/cat administered intravenously over 3–5 minutes while evaluating the ECG.
   c. Dextrose and regular insulin are also considered with the goal of stimulating the Na+/K+ ATPase causing intracellular movement of potassium. Dextrose alone is not used, rather in combination with the insulin to prevent hypoglycemia.
      i. Dose: 1 unit of regular insulin/cat intravenously immediately followed by 0.5 g/kg of 50% dextrose diluted 1:4.
      ii. The patient will require supplementation of 2.5-5% dextrose in fluids for several hours to prevent hypoglycemia.
d. Sodium bicarbonate: The author rarely uses this therapy as the acidosis in question is commonly resolve with fluid therapy alone. Intravenous sodium bicarbonate will increase the pH in the extracellular space, stimulating the exchange of intracellular H+ ions for extracellular potassium pushing potassium into the cell.
  1. Dose: 1 mEq/kg once or 0.3(BWkg) – BE (usually 1/3–1/2 of this dose will be given).

Urethral catheterization

Once the patient has been stabilized, the next step is relief of the obstruction with placement of a urinary catheter. This will reestablish normal urine flow and subsequent resolution of hyperkalemia and azotemia.

The author typically uses a combination of sedatives including butorphanol, a benzodiazepine (i.e. midazolam or diazepam), ketamine, and/or propofol to provide heavy sedation. In rare cases, the author will use general anesthesia. The goal of heavy sedation and/or anesthesia is to allow for maximal urethral relaxation and decreased pain associated with placement of the urethral catheter.

Example protocol in a stable patient without cardiac disease

- Butorphanol 0.4 mg/kg IV
- Diazepam 0.3mg/kg IV
- Ketamine 5 mg/kg IV

It is important to recognize this is one protocol, and the individual patient will need careful assessment to determine if this combination and/or dosing is appropriate for that patient.

Along with sedatives and anesthesia, the use of a coccygeal epidural has been recently described to reduce urethral spasm and improve comfort during and following the placement of the urinary catheter.

Once sedated or placed under anesthesia, an open-ended polypropylene (3.5Fr) tomcat catheter is first used to relieve the obstruction. Sterile procedure is essential to reduce the risk of iatrogenic infection including sterile gloves, sterile preparation of the perineum, and draping the perineum. Syringes of sterile flush (e.g. 0.9% NaCl) are used to flush the tomcat catheter and retropulse the obstruction.

If a plug or debris is seen around the perineum or the tip of the penis, careful digital manipulation and rolling the penis between the fingers can assist in breakup of the plug and catheter placement.

Once the obstruction is flushed and the tomcat catheter is seated in the urethra, the bladder is emptied and flushed. As the tomcat catheter is rigid, this is not used as an indwelling catheter. This should be removed and replaced with a softer, indwelling urinary catheter such as a red rubber catheter. This red rubber, indwelling catheter is sutured in place and connected to a closed collection system.

Diagnostic evaluation

Following catheter placement and further patient stabilization, additional diagnostics to consider include complete bloodwork (CBC and biochemistry profile), urinalysis, and abdominal radiographs. Additional diagnostics such as urine culture and abdominal ultrasound can be considered based on the initial diagnostics results.

Hospitalization and follow-up

Most of our feline urethral obstruction cases remain in hospital for approximately 36–48 hours. Criteria for removal of the urinary catheter include:

- The urine has an improved color and character (i.e. not bloody, gritty, or cloudy)
- Normal fluid balance (i.e. not excessive urinary outputs)
- Correction of electrolyte imbalances
- Correction of azotemia

Thorough discussion should be performed with the owner regarding any underlying causes found and notably monitoring in the future due to the concern for re-obstruction. The most common time for re-obstruction is within the first few days following hospital discharge.

The author typically discharges the patient with buprenorphine and either prazosin or phenoxybenzamine along with counsel for diet change if indicated.

References


Hyperthermia is an increase in core body temperature that occurs when heat production exceeds heat loss, further classified into pyrogenic or nonpyrogenic. Pyrogenic hyperthermia, more commonly known as fever, is a result of an increased hypothalamic set point in the body, usually secondary to an infectious, inflammatory, or neoplastic cause. Heat stroke is the most serious form of nonpyrogenic hyperthermia, where there is a noninfectious or noninflammatory reason for an increased core body temperature. Heat stroke is clinically recognized by a core temperature > 41°C (> 105.8°F). In this lecture, we will focus on nonpyrogenic causes of hyperthermia, notably heat stroke. In these cases, if the markedly elevated temperature is not rapidly addressed, extreme core body temperatures may result in a systemic inflammatory state and ultimately multiple organ dysfunction.

History
Patients that present for significant hyperthermia, specifically heatstroke, classically have a history documenting increased heat production, decreased heat dissipation, or a combination of the two syndromes.

Risk factors for developing hyperthermia include high ambient temperature, high ambient humidity, upper airway obstruction, laryngeal paralysis, brachiocephalic airway syndrome, collapsing trachea, and obesity. Notably in warmer temperatures and high humidity situations, lack of shade and a cool-down period after exercise can also lead to exertional hyperthermia.

Physical examination
Patients with significant hyperthermia and heat stroke may have red/brick-red mucous membranes as they often present in distributive shock with peripheral vasodilation. Frequently, they are tachycardic, tachypneic, and agitated. Their body temperature often significantly exceeds 103.5°F, commonly 105°F to 107°F.

When presented with a heat stroke patient, the clinician should triage and assess the patient similar to any emergency patient, focusing on the ABCDs of illness. Particular attention should be placed on the cardiovascular, respiratory, and central nervous systems. Patients with heat stroke and significant hyperthermia may present in shock and require immediate therapy.

Emergency treatment
Emergency treatment for heat-induced illness is used to reverse the effects of shock, restore perfusion, and correct the markedly elevated temperature. Stertorous or stridous breathing, commonly seen secondary to processes such as laryngeal paralysis, edema, or tracheal collapse may also require rapid and immediate treatment.

Initial stabilization of the patient may begin prior to arrival at the hospital. If the owner recognizes the signs, it is often advisable to have the owner begin cooling at home. Cooling may begin by wetting down the dog with room temperature water followed by placement of a fan on the patient.

Once in the hospital, this can be continued along with other therapies including cool intravenous fluids to help decrease body temperature.

Cooling measures should be halted once the temperature reaches 103°F. The reason is that rebound hypothermia is more difficult to reverse.

Restoring perfusion
On presentation, an intravenous catheter should be placed. Ideally, during catheter placement, blood work is drawn for baseline values (hematologic, biochemical, and coagulation analysis).

In many cases, prerenal and renal azotemia is present, with elevated BUN and creatinine concentration secondary to renal tubular necrosis. Elevations in hepatocellular enzyme function secondary to hepatocellular thermal injury are documented with elevated ALT, AST, and total bilirubin. Elevations in creatine kinase (CK) and AST are commonly seen secondary to rhabdomyolysis. Packed cell volume (PCV) and total protein (TP) are commonly increased secondary to hypovolemia and dehydration. Thrombocytopenia, prolonged PT and APTT, and elevated FDPs may be seen if DIC is present.

Fluid therapy is often initiated with a balanced, isotonic electrolyte solution. If the patient is in shock, a shock bolus (20-40 ml/kg) is administered intravenously followed by patient reassessment. If cardiovascular parameters have normalized, a more maintenance fluid rate is considered. If the cardiovascular parameters remain abnormal, additional fluid bolus therapy may be warranted. Alternatively, synthetic colloids (5-10 ml/kg) can be administered to effect.

Following fluid resuscitation, maintenance intravenous fluids should be administered at rates which take into account ongoing losses, notably from the gastrointestinal and urinary tracts.

Respiratory system
During initial assessment, flow-by or mask oxygen therapy should be administered until a more complete evaluation of the respiratory system can be performed. Stridorous or stertorous breathing can be seen with conditions such as laryngeal paralysis, tracheal collapse, or airway obstruction, often underlying causes for reduced heat dissipation and hyperthermia. Objective measurements of oxygenation such as pulse oximetry and/or arterial blood gas analysis can be considered and performed to further assess the respiratory system.

**Central nervous system**

Abnormalities of the central nervous system (CNS) may be from a variety of causes including hypovolemia and poor perfusion, hypoglycemia, or cerebral edema. A blood glucose should be immediately checked on presentation if CNS signs are present. If hypoglycemia is present, 0.25-0.5g/kg of a 50% dextrose solution should be administered intravenously as a bolus. Following the bolus administration, dextrose (2.5-5%) should be added to the maintenance intravenous fluids. If mentation is still altered once tissue perfusion has been restored, consistent with cerebral edema, intravenous mannitol (0.5-1g/kg over 20 minutes) or 7.2% hypertonic saline (3-5 ml/kg) can be administered.

**Renal system**

Azotemia (elevations in BUN and creatinine) is common in heat stroke patients, resulting from several processes including decreased renal perfusion (pre-renal azotemia), gastrointestinal tract hemorrhage, or renal failure (renal azotemia). Prior to aggressive fluid evaluation, a urine sample should be obtained to evaluate for the presence of casts, myoglobinuria (muscle injury), infection, and urine specific gravity. A cystocentesis should only be performed in these cases if the coagulation system has been documented to be normal as thrombocytopenia and abnormalities in the PT/PTT are common. Once adequate tissue perfusion is documented, urine output should be monitored closely. If urine production does not seem adequate regarding output estimates, a urinary catheter should be placed to document specific urine output and assess for the presence of oliguric and anuric renal failure. Urine output should be at least 1-2 ml/kg/hour and certainly higher if aggressive intravenous fluid therapy is administered.

**Gastrointestinal system**

Patients presenting with heat stroke and severe heat related illness often have gastrointestinal signs such as vomiting and/or diarrhea, potentially hemorrhagic, notably in dogs as their shock organ is commonly considered the gastrointestinal system. Direct thermal damage as well as poor perfusion with shock contributes to the potentially severe gastrointestinal illness. Vomiting may be treated with anti-emetics such as ondansetron 0.2mg/kg IV, dolasetron 0.5mg/kg IV (5-HT3 receptor antagonists), and Cerenia (1mg/kg SQ q24h). Metoclopramide can also be administered as a constant rate infusion (1-2 mg/kg/24 hours IV) in fluids especially in patients with regurgitation and suspect ileus.

Hemorrhagic gastroenteritis is commonly seen with the concern for gastric ulceration as a result of decreased viscera perfusion. In these cases, treatment also consists of H2 blockers and/or proton pump inhibitors (Famotidine 1mg/kg IV q12-24h, esomeprazole 0.5-1mg/kg IV) and Sucralfate.

**Antibiotic therapy**

Intestinal mucosal permeability may be compromised with the ultimate concern for bacterial translocation and sepsis. Broad spectrum intravenous antibiotic therapy is generally considered as a result, examples being unasyn (25-30mg/kg IV q8 hours) with metronidazole (10mg/kg IV q12 hours) or ampicillin (22mg/kg IV q8 hours), Baytril (10mg/kg IV q24 hours), and metronidazole (10mg/mg IV q12 hours).

**Coagulation system**

Disseminated intravascular coagulation (DIC) is a common sequelae of heat stroke. Physical examination findings of DIC may include petechiae, ecchymoses, hematuria, and/or hematochezia. Laboratory evaluation of the coagulation system includes prothrombin time (PT), partial thromboplastin time (PTT), platelet count, fibrin split products (FSP) and D-dimer concentrations. If coagulation system abnormalities are present, it is important to avoid procedures with a risk of hemorrhage such as jugular vein phlebotomy, cystocentesis, or other invasive procedures such as organ aspirates. If hemorrhage is present as a result of coagulopathy, fresh whole blood or fresh frozen plasma (10-20 ml/kg IV) is considered.

**Hepatic system**

Alterations in hepatocellular enzyme function secondary to hepatocellular thermal injury is commonly seen. On biochemical analysis, ALT, AST, and total bilirubin are commonly elevated. Elevations in creatine kinase (CK) and AST may also be seen secondary to rhabdomyolysis.
Prognosis

Treatment of heat stroke patients can be both challenging and expensive. Disseminated intravascular coagulation, hypoglycemia at presentation, presence of nucleated red blood cells, and acute renal failure are noted risk factors for death. Other risk factors for death include delayed admission to the hospital (>90 minutes) increased serum creatinine at 24 hours, increased bilirubin, decreased albumin, and ventricular arrhythmias.

For this reason, rapid patient assessment, treatment, and aggressive supportive care are vital in the treatment of patients with heat stroke.

Drugs used for vomiting

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Range</th>
<th>Frequency</th>
<th>Route</th>
<th>Indications</th>
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</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 h</td>
<td>IV CRI</td>
<td>Vomiting</td>
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<tr>
<td>Ondasetron</td>
<td>0.1-0.2mg/kg</td>
<td>BID-QID</td>
<td>IV / PO</td>
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<tr>
<td>Dolasetron</td>
<td>0.6mg/kg</td>
<td>SID-BID</td>
<td>PO, SC, IV</td>
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<tr>
<td>Maropitant</td>
<td>1mg/kg</td>
<td>SID &lt; 5 d</td>
<td>SQ / PO</td>
<td>Vomiting</td>
</tr>
</tbody>
</table>

References

Lyme Disease: Treating Acute and Chronic Manifestations
Garret Pachtinger, DVM, DACVECC
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Levittown, PA

Lyme disease is caused by transmission of *Borrelia* spp. spirochetes from infected *Ixodes* spp. ticks. As the infected tick acquires a blood meal, the spirochetes travel to the salivary glands of the tick and are injected into the mammalian host. Once there, they colonize adjacent tissues and establish a persistent infection.

While similar in disease name, disease manifestation is quite different in humans as compared to dogs. While the spirochetes can persist in both hosts despite treatment with antibiotics, the clinical disease course is dissimilar in humans and dogs.

### Clinical signs

In humans, *B. burgdorferi* infection causes flu-like illness characterized by the classic erythema migrans rash. Other disease manifestations include arthritis, cardiac, or neurological abnormalities. In contrast, acute signs of illness are rarely seen in our canine patients. More common in dogs include polyarthritis, fever, anorexia, and lethargy. While less common, we can also see renal disease, cardiac disease, or neurologic disease.

Moreover, the signalment may also play a role in the degree of illness seen in our canine patients. For example, Labrador Retrievers, Golden Retrievers, and Shetland Sheepdogs appear susceptible to nephropathy from colonization of the kidneys.

As compared to the acute signs of illness in humans, the infrequent nature of the acute disease in our canine patients makes Lyme diagnosis challenging. In dogs, clinical signs are observed in approximately 10% of infected cases. Moreover, the 10% of patients with signs of illness show these signs two to five months after the infection. Even more challenging, the signs of illness may be vague including, lymphadenopathy, lethargy, and fever. This makes a specific diagnosis challenging as the signs of illness are seen months after exposure/infection with vague signs not pathognomonic to any specific disease.

### Diagnosis

There is not one individual test that will specifically determine, objectively, that a patient’s clinical signs are a result of the *Borrelia* infection.

From a clinical perspective, the author uses several criteria to increase the suspicion of disease:

1. Evidence of exposure
2. Presence or history of an engorged *Ixodes* tick
3. Patient lives or visited an endemic area
   a. Positive test result.
   b. Clinical signs consistent with infection
   c. Ruling out other disease
4. Expected response to therapy

With that in mind, what diagnostics exist to help with this diagnosis:

**ELISA based kit**

For diagnosis of Lyme Borrelia (*Borrelia burgdorferi*), the commercially available ELISA based kit manufactured by IDEXX Corp is a common first line diagnostic. This test is also useful for the detection of antibodies against *Anaplasma* spp (A phagocytophilum and A.platys) as well as *Ehrlichia* spp (E. canis, E. ewingii) and cross-reactive other *Ehrlichia* spp and heartworm. The test is intended to be used as a screening test, not a diagnostic test. This test detects a subgroup of antibodies against the outer surface protein VlsE using the C6 peptide. A positive antibody response occurs 3–5 weeks after infection. Unfortunately, this test does not show a truly active infection, rather just exposure. Test results will remain positive for weeks, months, or years. Antibodies remain in circulating in the body long after an infection is cleared with or without antibiotics.

**C6 antibody testing**

Once the ELISA based kit results in a positive test, many consider one of the available tests for canine antibodies against the Lyme C6 peptide. This peptide is expressed only during infection. As a result, these tests are used to distinguish previous exposure from active infection. There still may be quite a bit to learn about the quantitative C6 titer in all cases of Lyme infection. What has been shown is that the quantitative C6 titer decreases with therapy and it does correlate well with circulating anti-Lyme immune complexes. For this reason, it remains a reasonable clinical tool in the diagnosis and treatment plan recommendation for those patients that are Lyme positive.
Other testing includes the Cornell "MultiPlex" for Lyme. This is a fluorescent bead-based multiplex assay that quantifies antibodies to the outer surface proteins (OspA, OspC, and OspF) B. burgdorferi in canine serum. This test helps distinguish antibodies from vaccination vs. natural exposure. The Antech AccuPlex4® and ABAXIS VetScan® also detect antibody to Lyme.

**Lyme prevention**

Knowing that once ticks are attached to the host, transmission takes at least 48 hours from the beginning of the blood meal, daily tick removal following tick exposure is essential. Owners should be educated on tick evaluation and tick removal for safe and effective tick disposal and prevention of Lyme transmission. Tick control should also be recommended, even in low-incidence areas.

**Lyme treatment - Acute**

This is a very common question, or set of questions. Do you treat all Lyme positive dogs? Lyme positive dogs with symptoms only? Lyme positive dogs of a certain signalment regardless?

My treatment thoughts are as follows:

- I treat Lyme-positive dogs with clinical signs.
- I do not (commonly) treat Lyme-positive ELISA dogs that are asymptomatic
  - For these patients, I would consider a C6 Peptide test, and potentially treat with a high titer value.

If a patient warrants therapy, what do I treat them with?

- Amoxicillin and doxycycline are both reasonable options.
- I choose doxycycline based on my experience - 10mg/kg PO SID (or 5mg/kg PO BID) for 4 to 6 weeks. Doxycycline will also likely be better at treating co-infections.

**Lyme treatment – Chronic**

For chronic Lyme disease, it is recommended to monitor for proteinuria or microalbuminuria every 3-6 months. If there is persistent proteinuria despite appropriate antibiotic therapy (discussed above), a renal biopsy is recommended to determine if an immune-mediated glomerulonephritis exists. Further therapy for this condition would include a low-protein diet, angiotensin-converting enzyme inhibitor (ACEi) therapy, and an additional course of antibiotic therapy. The use of immunosuppressive therapy is also considered for patients with immune-mediated glomerulonephritis.

Unfortunately, the current prognosis for a patient with Lyme nephritis is guarded to poor with treatment aimed at improving quality of life, azotemia, proteinuria, and hypertension. The treatment plan focuses on judicious fluid therapy (due to hypoalbuminemia and the concern for third spacing), blood pressure monitoring with the use of anti-hypertensives (i.e. amlodipine), ACEI therapy (e.g. enalapril or benazepril), and antibiotic therapy. Lyme nephritis patients may also become hypercoagulable secondary to antithrombin loss, with anticoagulant therapy also considered (e.g. aspirin, Plavix).

**References**


Hemoabdomen, or hemoperitoneum, refers to the accumulation of blood in the peritoneal cavity. In veterinary medicine, causes of hemoabdomen can be divided into two categories: traumatic and non-traumatic. Traumatic causes can involve either blunt trauma, such as being hit by a car or falling from a height, or penetrating trauma, and can lead to hemorrhage from organs (spleen, liver, kidneys) or blood vessels. Non-traumatic causes, or spontaneous hemorrhage, are most commonly due to neoplasia or coagulopathy, but can also occur secondary to vascular avulsion from conditions such as gastric dilatation-volvulus or splenic or liver torsion. In both traumatic and non-traumatic cases of hemoabdomen, patients often present with signs of hypovolemic shock, and rapid triage and resuscitation are critical to patient survival.

As with any emergent patient, an initial assessment should include the ABCs (airway, breathing, circulation) with special attention to the cardiovascular system. Mucous membrane color, capillary refill time (CRT) and pulse rate and quality should be assessed. Characteristic signs of a hemoabdomen will include pale mucous membranes, prolonged CRT, tachycardia with poor pulse quality, and often cardiac arrhythmias characterized by ventricular premature contractions (VPCs) or ventricular arrhythmias. Depending on the volume of effusion, an abdominal fluid wave may be palpable. Bruising around the umbilicus, called Cullen’s sign, is suggestive of hemorrhage into the peritoneal or retroperitoneal space.

AFAST and abdominocentesis

During the triage exam, in conjunction with initial stabilization, AFAST (abdominal focused assessment with sonography for trauma/triage) and abdominocentesis can be performed. AFAST involves the use of ultrasound to briefly and rapidly assess a patient for peritoneal and retroperitoneal effusion, and should include visualization of the abdomen in four quadrants: cranially between the diaphragm and liver lobes, laterally in the right and left retroperitoneal spaces, and caudally near the urinary bladder. The most ventral portion of the patient, depending on their position, should also be assessed. If fluid can be visualized or is suspected based on physical exam findings, abdominocentesis should be performed to confirm a diagnosis of hemoabdomen. Abdominocentesis may be performed using a single paracentesis or four-quadrant approach. It is best performed with the patient in left lateral recumbency to avoid puncture of the spleen. The abdomen should be clipped and cleaned using aseptic technique along the ventral midline, centered at the umbilicus. For diagnostic abdominocentesis, a closed-needle technique using a 20- or 22-gauge needle attached to a 3-cc syringe may be used. The needle should be inserted just caudal to the umbilicus, preventing further movement of the needle tip to avoid laceration of internal structures. The syringe is gently aspirated and a sample obtained. If no sample is obtained, a four-quadrant tap (at regions cranial, caudal, ventral and dorsal to the umbilicus) may be performed. Diagnostic peritoneal lavage is another alternative. After aspirating fluid from the abdominal cavity, it should be observed carefully. If the fluid appears to be hemorrhagic, observe it carefully for clotting – fluid from the abdominal cavity should not clot, whereas blood aspirated from the spleen, liver or any vessel will clot readily.

Baseline diagnostics and initial resuscitation/Hypotensive resuscitation

While the above diagnostics are being performed, an intravenous catheter should be placed (ideally in a cephalic or jugular vein) and flow-by oxygen provided. A minimum database (PCV, TS, BG) and extended database (venous blood gas, lactate, creatinine), if possible, should be obtained at the time of catheter placement. PCV and TS should always be interpreted together, and can allow for assessment of volume status as well as differentiation of causes of anemia if present. In cases of hemorrhage, both the PCV and TS should be low, since both red cells and proteins are being lost. However, in cases of acute hemorrhage, especially in dogs, it is common to see a low TS in combination with a normal or even slightly increased PCV. This is due to acute splenic contraction and release of red cells into the circulation when hemorrhage occurs. Other common findings may include hyperglycemia (secondary to trauma or stress), hyperlactatemia and metabolic acidosis secondary to poor tissue perfusion. Mild to moderate azotemia, often prerenal in origin, may also be present. Further diagnostics should include a coagulation profile (PT/aPTT), blood type, complete blood count, and serum chemistries. Prolongation of clotting times, specifically the PT, may be suggestive of anticoagulant rodenticide toxicity in patients with a possible exposure history.

Since patients with hemoabdomen often have signs of hypovolemic shock, volume resuscitation should be provided immediately. Isotonic crystalloids remain the cornerstone of treatment. A shock dose of isotonic crystalloid solution is approximately one blood volume (i.e., 90 ml/kg in the dog and 50 ml/kg in the cat). However, it is important to remember that this volume should be administered in increments – generally 1/4 to 1/3 of a shock dose at a time, with frequent reassessment of vital parameters. In dogs, the author generally gives 20-30 ml/kg of isotonic crystalloids as a fluid bolus; in a cat, usually 10-20 ml/kg. In patients that are bleeding it may be advantageous to perform “hypotensive resuscitation” (to a MAP of approximately 60 mm Hg) until the hemorrhage is controlled since aggressive fluid therapy in this setting can worsen bleeding and outcome. To achieve this and avoid “popping the clot” that may be forming on a bleeding vessel or organ, smaller boluses or boluses over a slightly longer period of time may be...
advantageous. However, it is important to remember that this is a short-term solution to stabilize the patient, and should not be continued long term.

**Blood transfusion and damage control resuscitation**

In the hemoabdomen patient, there are several potential indications for blood component therapy. In the bleeding patient that cannot be stabilized with fluid therapy alone, packed red blood cell transfusion or whole blood transfusion should be considered in the case of severe anemia (generally, a PCV < 25% with evidence of patient instability – tachycardia, poor pulses, hypotension, pale mucous membranes, prolonged CRT, weakness). In human medicine, the standard treatment approach to hemorrhagic shock and those patients requiring massive transfusion historically included administration of liberal amounts of crystalloids and pRBCs followed by the administration of plasma and platelets based on coagulation testing. Damage control resuscitation is now recommended, the purpose of which is to try to stop the “vicious bloody cycle” in which the triad of hypothermia, acidosis, and coagulopathy leads to continued hemorrhage. Damage control resuscitation includes hypotensive resuscitation, decreased use of crystalloids, administration of fresh frozen plasma, platelets and packed red blood cells (FFP:pRBC:pRBC) in a 1:1:1 ratio, use of other hemostatic agents as needed, heat support, and early surgical intervention. In veterinary medicine, no studies currently exist examining blood product component therapy and transfusion ratios. However, based on the human evidence, it may be advisable to consider plasma and platelet transfusions early on in patients requiring massive transfusion, or administering whole blood when available. The recommended dose for FFP is 10-20 ml/kg and the dose for pRBC is 15 ml/kg to raise the PCV by 10%. If whole blood is available, the recommended starting dose is 20-30 ml/kg. In cases where blood products are not available or not financially feasible, autotransfusion can be life-saving and can be considered in select cases of hemoperitoneum.

If fibrinogen is able to be measured and is low, then cryoprecipitate can be given. Tranexamic acid or aminocaproic acid can also be considered. If possible, synthetic colloids should be avoided given their reduction in FVIII and vWF activity.

**Management of arrhythmias**

Tachyarrhythmias may be encountered in the patient with hemoabdomen secondary to increased sympathetic tone, shock, myocardial ischemia, or secondary to underlying abdominal pathology. An electrocardiogram should be obtained in any patient presenting with hemoabdomen and can be useful in monitoring the patient during resuscitation in addition to recognizing arrhythmias and the need for potential therapy. Sinus tachycardia is expected in the patient with hemoabdomen and should respond to fluid resuscitation and analgesic therapy if indicated. Ventricular arrhythmias are also common, and ventricular complexes are recognized by a wide and bizarre QRS morphology. Accelerated idioventricular rhythms are recognized as a regular, monomorphic ventricular rhythm that is very similar in rate to the underlying sinus rhythm, and fusion complexes may be recognized during transitions between the ventricular and sinus rhythm. In general, this rhythm does not produce significant hemodynamic abnormalities and does not require therapy. In many cases, such as isolated ventricular premature complexes, these rhythm disturbances also do not require treatment. However, treatment is recommended if there are frequent or multifocal ventricular premature complexes, if the coupling interval is rapid and creates an R-on-T morphology, or if there is sustained ventricular tachycardia (rates greater than 180 bpm) with clinical or hemodynamic sequelae (weakness, hypotension, pale mucous membranes, poor pulse quality). The first line of treatment for ventricular tachycardia in dogs and cats is generally lidocaine, although cats are more likely to develop adverse side effects (GI signs, neurologic signs including seizures) so their doses should be lower (dogs, 2 mg/kg IV bolus up to 8 mg/kg and followed by 25 to 80 mcg/kg/min CRI; cats, 0.25 to 0.5 mg/kg IV bolus followed by 10 to 20 mcg/kg/min CRI). If the patient fails to respond to lidocaine, other anti-arrhythmics to be considered include procainamide, esmolol or amiodarone.

**Prognosis**

For patients with both traumatic and non-traumatic hemoabdomen, prognosis will depend on the extent of injury and need for surgery. In all of these patients, rapid recognition and appropriate resuscitation and treatment are key to ensuring a good outcome.

References available upon request from the author.
Head Trauma without the Headaches
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Causes of traumatic brain injury (TBI) in veterinary patients most commonly include motor vehicle accidents and animal interactions (bite wounds), with less common causes including things like blunt trauma, falls from heights, gunshot wounds and malicious human activity. Having a basic understanding of the pathophysiology of TBI and remembering to focus on both intracranial and extracranial injuries can help to ensure positive outcomes in these often severely affected patients.

When thinking about TBI, it is helpful to remember that acute head injuries can be separated into two categories of injury: the primary injury, which occurs immediately at the time of the trauma and cannot be prevented, and secondary injury, which occurs in the hours to days following the primary injury and should be addressed medically. Primary brain injuries include concussion, contusion, and laceration. Concussion is the least severe type of primary brain injury and is characterized by a brief loss of consciousness. Contusion, which is more severe, involves brain parenchymal hemorrhage and edema and usually causes unconsciousness for more than several minutes. Contusions can be characterized as “coup” lesions (directly under the site of impact), “contrecoup” lesions (directly opposite the site of impact), or both, due to movement of the brain within the skull during the trauma. The most severe primary brain injury is laceration, which involves disruption of the brain parenchyma and can be characterized by axial or extraxial hematomas. Secondary injuries include excitotoxicity, ischemia, inflammation, ATP depletion, production of reactive oxygen species, accumulation of intracellular sodium and calcium, nitric oxide accumulation, and cerebral lactic acidosis; these injuries all eventually cause neuronal cell death. Secondary injuries can also be exacerbated by systemic abnormalities that occur commonly with head trauma including hypovolemia and hypotension, systemic inflammation, acid-base and electrolyte disturbances, hypoxemia, hypop- and hypercapnia, hypo- and hyperglycemia, and hyperthermia.

Head trauma involves trauma to an enclosed space, the skull, and thus can be thought of in relation to the Monroe-Kellie doctrine, which states:

\[ V_{\text{intracranial}} = V_{\text{brain}} + V_{\text{CSF}} + V_{\text{blood}} + V_{\text{mass lesion}} \]

Where \( V = \) volume. Primary or secondary injury that increases any of these volumes (for example, hemorrhage within the skull) will lead to an increase in intracranial pressure (ICP), which can be life-threatening. If we also remember that cerebral perfusion pressure (CPP) is the difference between mean arterial pressure (MAP) and ICP (CPP = MAP – ICP), we can see how an increase in ICP will compromise perfusion to the brain. While the normal brain is able to maintain a constant cerebral blood flow over a wide range of systemic blood pressures, anywhere from MAP 50-150 mmHg, using autoregulatory mechanisms, these mechanisms are impaired with trauma and even small decreases in systemic MAP will make the brain sensitive to further injury. The Cushing’s reflex, also known as the CNS ischemic response, is characterized by an increase in MAP and decrease in HR due to increased ICP and the body’s attempt to maintain CPP. This response is an indication of potentially life-threatening intracranial hypertension and should be addressed immediately.

Initial evaluation of the patient with TBI should include assessment of both intracranial and extracranial abnormalities. As with any trauma patient, the “ABCs” (airway, breathing, circulation) should be immediately assessed, and life-saving measures (provision of oxygen, intubation, placement of IV catheter, fluid support, CPCR) provided if indicated. Extracranial life-threatening injuries such as compromised oxygenation or ventilation, hypovolemia, penetrating injuries, airway obstruction and hemorrhage should be identified. After any life-threatening injuries have been assessed and dealt with, intracranial evaluation should take place. An initial brief neurologic exam should be performed and should include assessment of level of consciousness (recalling that hypovolemia and shock can impair mentation as well), pupil size and response to light, and posture/locomotion.

Following placement of an IV catheter, initial diagnostics and monitoring should be performed to assess patient stability. When drawing blood or placing catheters in patients with TBI, the jugular veins should be avoided as occlusion can decrease venous outflow from the brain and increase ICP. A minimum database (PCV, TS, BG) and venous blood gas are recommended at baseline to assess for hemorrhage as well as acid-base, perfusion and ventilation status. Ideally, comprehensive blood work including electrolytes, renal values, hepatic parameters and lactate should also be measured. Monitoring should include assessment of tissue perfusion (mucous membranes, capillary refill time, pulse quality and heart rate), oxygenation (pulse oximetry or arterial blood gas analysis) and blood pressure. In patients with TBI, blood pressure should be maintained at or above a MAP of 80 mmHg or Doppler > 100 mmHg; evidence of hypertension with tachycardia should prompt treatment of pain or anxiety, while hypertension with bradycardia should prompt treatment to lower ICP. For hypovolemic patients with normal electrolyte status, normal (0.9%) saline should be administered, since it has the smallest amount of free water of the isotonic fluids (154 mEq/L) and as such, is least likely to worsen cerebral edema. Alternatively, hypertonic saline can be considered, as discussed below.

Radiographs of the skull are rarely useful in cases of head trauma as they are insensitive and difficult to interpret. In patients that require imaging, CT is preferred due to its superior ability over MRI to assess bone and areas of edema or acute hemorrhage.
Advanced imaging should be considered in patients with severe abnormalities on presentation, failure to improve over the course of treatment or worsening of signs during treatment, or lateralizing signs.

Two major treatments for intracranial hypertension exist – mannitol and hypertonic saline. Mannitol is an osmotic diuretic that has two main effects: (1) within minutes, it reduces blood viscosity through plasma expansion, improving cerebral blood flow and oxygen delivery to the brain (rheologic effects), and (2) reducing brain water content after 15-30 minutes through a delayed osmotic effect that establishes a gradient between plasma and cells and pulls water out of the brain parenchyma and into the vasculature; this effect lasts anywhere from 1-6 hours. Mannitol has been shown to have a beneficial effect on neurologic outcomes in human patients with head injury, and decreases ICP, and increases CPP and cerebral blood flow. It may also have some free radical scavenging properties.

Because mannitol is a diuretic, patients who are hypovolemic should be volume resuscitated prior to administration, and urine output should be carefully monitored. Monitoring of serum osmolality is also recommended in patients receiving repeated boluses, since osmolality > 320 mOsm/L has been associated with acute renal failure in human patients; for this reason and because it could leak into the brain parenchyma and worsen edema, use of mannitol as a CRI in patients with head trauma is not recommended. In veterinary patients, mannitol is usually administered at 0.5-1.5 g/kg over 15-20 minutes through a fluid line filter since the medication can crystallize. Treatment should be followed by crystalloid fluids to maintain intravascular volume.

In patient who require intravascular volume resuscitation in addition to treatment of intracranial hypertension, hypertonic saline (HTS) is a great choice. In addition to the rheologic and osmotic effects of mannitol, HTS also improves hemodynamics, has immunomodulatory effects and can help with vasoregulatory function. HTS is generally supplied as a 23.4% solution, and must be diluted to 7-7.5% before administration. The author prefers to dilute HTS in a 60-ml syringe (17 ml of HTS and 43 ml of isotonic crystalloid) and then administer 3-5 ml/kg of the solution over 10 minutes. Repeated dosing can cause hypernatremia, so electrolytes should be monitored in patients requiring multiple doses.

Two medications that are NOT indicated in the treatment of head trauma are corticosteroids and the diuretic medication furosemide. While corticosteroids have potent anti-inflammatory effects and have been commonly used in patients with head trauma in the past, recent human studies have demonstrated worse outcomes in patients with head trauma treated with steroids. Furosemide, which has been used historically in patients with head trauma to increase brain water loss, should be avoided because of its potential to cause volume depletion, hypotension and impair cerebral perfusion.

Other potential therapies that may be useful in patients with head trauma include the use of a slant board to decrease ICP, maintenance of normocapnia, and prevention of seizures. Elevating the head and neck on a slant board (instead of pillows or blankets which can kink the neck and occlude the jugular veins) at 15-30° will help to increase venous drainage and decrease ICP without impairing CPP. Maintaining normocapnia (arterial carbon dioxide of 35-40 mmHg) is also recommended, since hypercapnia (hypoventilation) can cause cerebral vasodilation and hypocapnia (hyperventilation) can cause cerebral vasoconstriction, worsening ICP and decreasing cerebral blood flow, respectively. In severely affected patients, mechanical ventilation may be necessary to maintain normocapnia. If patients develop seizures, aggressive treatment is recommended, and a recent study has demonstrated that post-TBI seizures are more common in people and veterinary patients after head trauma. While prophylactic anticonvulsants have not been shown to prevent delayed seizures, treatment should be instituted in any patient showing signs of seizure activity.

Monitoring of patients over the course of their treatment for TBI can be challenging, and the modified Glasgow Coma Scale Score can help. Through assessment of motor activity, level of consciousness and brainstem reflexes, scores can be assigned to patients at regular intervals and offer a more objective means of monitoring progress, especially when different technicians or clinicians are involved. Retrospectively, this scale has been shown to correlate with short-term outcome in dogs with head trauma. Poor prognostic indicators include deterioration in level of consciousness, pupillary dilation and loss of pupillary light responses. Hyperglycemia has also been associated with more severe head injuries in veterinary patients. Because prognosis is difficult to predict after TBI, and severely affected patients can respond dramatically to treatment, reassessment of the patient after initial stabilization is always recommended. It is the author’s experience that young animals especially can make dramatic and rapid recoveries, although owners should be aware that long term neurologic deficits can exist.

References available upon request.
Medical Management of Thoracic Trauma
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Thoracic trauma is frequently caused by blunt or penetrating trauma and is common in veterinary medicine. Rapid and appropriate treatment is key to ensure a positive outcome. Patients with thoracic trauma frequently have more than one thoracic injury, making a thorough physical exam vital in directing treatment. Traumatic thoracic injuries may include pulmonary contusions, pneumothorax, rib fractures and flail chest, hemothorax, diaphragmatic hernia, penetrating chest wounds, gunshot wounds, high-rise syndrome, tracheobronchial injury and myocardial contusions. As with any trauma patient, an initial assessment should include the ABCs (airway, breathing, circulation) with special attention to the respiratory system. Airway patency, function of the chest wall and pleural space, and assessment of the pulmonary parenchyma should all be incorporated into the triage exam. It is also important to remember that the severity of injuries may progress over time. In addition, the use of TFAST (thoracic focused assessment with sonography for trauma) can be helpful in detecting pneumothorax and other injuries such as pleural and pericardial effusion. Other minimally invasive baseline diagnostics such as a minimum data base, blood pressure, pulse oximetry and EKG should be considered at the time of triage.

Pulmonary contusions
Pulmonary contusions are common with thoracic trauma and are the result of blunt trauma to the alveolar capillary membranes, resulting in pulmonary interstitial and alveolar hemorrhage and edema. Contusions are the most common type of injury following blunt thoracic trauma, affecting 58% of dogs in one study. They can range from mild to severe, and start immediately following impact but can worsen in the 24-48 hours following injury, highlighting the importance of continued monitoring. Clinical signs may include tachypnea, dyspnea, cyanosis, hemoptysis and harsh lung sounds or crackles; arterial blood gas analysis or pulse oximetry may reveal hypoxemia. Diagnosis is based on thoracic radiographs or computed tomography when the patient is stable. Classic radiographic signs of pulmonary contusions include areas of patchy or diffuse interstitial or alveolar infiltrates. It is important to remember that radiographic changes may lag behind clinical signs and thus underestimate the severity or extent of lesions. In addition to primary lung dysfunction from hemorrhage and edema, lung function may also be worsened by bronchoconstriction, increased mucus production, and alveolar collapse from decreased surfactant production. Disruption of the alveolar capillary membrane increases pulmonary vascular permeability, and leads to fluid movement into the interstitium and alveoli. Any increase in pulmonary capillary hydrostatic pressure can lead to increased fluid in the interstitium and alveoli, prompting concerns about fluid therapy and possible worsening of respiratory signs. While no particular type of fluid (isotonic crystalloids versus hypertonic saline versus synthetic colloids) has been shown to have an obvious benefit, using smaller volumes of fluid and administering fluids at a slower rate while monitoring respiratory signs is recommended. Fluid resuscitation should be adequate to support optimal cardiac output and perfusion with normalization of hypotensive end-points. Therapy for pulmonary contusions includes supportive care (oxygen therapy with mechanical ventilation if needed, analgesics, fluids). The incidence of pneumonia associated with pulmonary contusions is low, thus antibiotics are not indicated unless other injuries prompt their administration.

Pneumothorax
Pneumothorax is another common complication of blunt thoracic trauma, and occurs when air leaks from the pulmonary parenchyma or airways into the pleural space, resulting in lung lobe collapse. In one study of high-rise falls in cats, 60% had pneumothorax, highlighting the importance of recognition of this condition with any type of trauma. Classic clinical exam findings suggestive of a pneumothorax include tachypnea, short shallow breathing, and muffled breath sounds dorsally or diffusely. Radiographic signs include elevation of the cardiac silhouette off of the sternum, collapse and retraction of the lung lobes, and absence of lung markings in the periphery. In a patient with respiratory compromise and suspicion for pleural space disease, thoracocentesis should be performed to achieve stabilization prior to further diagnostics, including radiographs. The procedure is simple, fast, diagnostic and therapeutic. Prior to thoracocentesis, oxygen supplementation should be provided. The author prefers to place an intravenous catheter if possible should sedation be required or in case of complications during the procedure. Supplies for intubation should also be available. To perform thoracocentesis, a needle, butterfly catheter or peripheral catheter can be used to access the pleural space. Other necessary supplies include clippers, scrub, sterile gloves, an extension set, a three-way stopcock and a collection syringe (10 to 60 mL depending on patient size). The patient should be positioned in sternal recumbency or the most comfortable position to minimize stress. Blind thoracocentesis for air can be performed at the 7th to 9th intercostal spaces in the dorsal part of the thorax. The needle, attached to a closed system, should be inserted cranial to the rib to avoid the nerves and vessels that run caudally, and perpendicular to the chest wall. The needle is then advanced slowly through the skin and into the intrathoracic space while aspirating gently; air should be aspirated until negative pressure is obtained. If negative pressure cannot be obtained, the system connections should be checked for leaks; if no leaks are present, a tension pneumothorax should be suspected and placement of a chest tube is indicated.

Management of a patient with pneumothorax includes continued and careful monitoring and may require repeat thoracocentesis, tube thoracostomy, continuous evacuation, or, rarely, thoracotomy. Generally, surgical placement of a chest tube should be considered
when negative pressure cannot be obtained during thoracocentesis or when multiple taps are performed in a short period of time ("three strikes" rule). Two types of indwelling thoracostomy tubes can be placed: larger tubes with a sharp stylet or trochar to allow surgical placement, or smaller long flexible catheters that can be placed using the Seldinger technique under light sedation (Guidewire Inserted Chest Tube, MILA International, KY). Surgical placement of a standard chest tube should be performed under anesthesia with the patient intubated. Necessary materials include clippers, surgical scrub, a sterile blade, small surgical pack, suture, a 12-24 French trocar thoracostomy tube, catheter adapter, 3-way stopcock, injection caps and a continuous drainage device if indicated.

Following surgical preparation of the 7th to 11th intercostal space, an assistant pulls the skin cranially about two intercostal spaces, and a skin incision is made over the 8th to 10th intercostal space. Hemostats are used to bluntly dissect into the pleural space, and then opened to allow the tube and trocar to fit through the opening. The tube is directed cranially and ventrally and the stylet is removed as the tube is clamped. Releasing the skin allows a subcutaneous tunnel to form around the tube, reducing air leakage. The tube is then connected to a closed system and sutured into place using a purse-string suture around the entry site and a finger trap pattern to secure the tube. Alternatively, the Seldinger technique can be used to place a smaller, long, fenestrated catheter under light sedation and/or local block. Similar surgical preparation is performed following patient sedation, and a local block with 2% lidocaine can be used in the dermis and intercostal muscle. The provided short catheter is used to enter the thoracic cavity between the 7th to 8th intercostal space, and the stylet is then removed to allow for feeding of the wire. The short catheter is then removed, dilation performed, and the long fenestrated catheter placed over the wire. Following removal of the wire, the catheter can be secured with suture and bandaging.

In both cases, placement should be checked with radiographs, and 24-hour monitoring is essential.

**Rib fractures**

Rib fractures may occur secondary to any type of thoracic trauma and are the most common type of thoracic injury in human patients. They rarely occur in isolation, and hence should prompt one to look for other thoracic injuries such as pulmonary contusions or pleural space disease. Rib fractures are of clinical importance because they can cause hypoxemia due to lung injury as well as hypovolemia due to pain. A flail chest involves fractures of two or more adjacent rib segments, both dorsally and ventrally, leading to thoracic instability and paradoxical chest wall movement. On inspiration, negative intrapleural pressure expands the lungs and causes the flail segment to collapse inward; on expiration, the intrapleural pressure becomes less negative, the lungs deflate and the flail segment moves outward. Rib fractures should be confirmed via radiographs, and techniques such as inverting or rotating digital films can be helpful in diagnosing fractures. Other injuries should also be identified. Emergency treatment of rib fractures involves supportive care: the fractured side, especially in the case of a flail segment, should be placed down to minimize painful outward movement. Supplemental oxygen should be provided as needed. Analgesia, including systemic medications and local blocks, should be rapidly employed. For many rib fractures, surgical stabilization is not necessary unless penetrating wounds are present or the flail segment is large.

**Hemothorax**

Hemorrhage into the pleural space, or hemothorax, can result from injury to the lung parenchyma, chest wall and associated vessels, or the great vessels within the thoracic cavity. Since the pleural space can accommodate large volumes of blood without causing outward signs of respiratory compromise, patients with hemothorax often present with evidence of hypovolemic shock without an obvious source of blood loss. Clues may include tachypnea with shallow chest excursions and dull breath sounds ventrally. A diagnosis of hemothorax can be achieved via thoracocentesis, yielding not clotting blood; radiographs may not be necessary and are often risky prior to stabilization. Treatment involves fluid therapy for hypovolemic shock; rarely, autotransfusion or exploratory thoracotomy may be indicated.

**Diaphragmatic hernia**

Diaphragmatic hernias can occur with trauma when a sudden increase in abdominal pressure forces the diaphragm forward, resulting in rupture and displacement of abdominal organs into the thoracic cavity. Concurrent injuries are common and are most often caudal to the thorax, including fractures, hip luxations, and damage to the liver and urinary bladder. Organs in the cranial abdominal cavity (liver, small intestine, stomach, spleen) are most likely to herniate and compress the pulmonary parenchyma, resulting in decreased lung volume and signs of respiratory distress. Hypoxemia, dyspnea, tachypnea, dull ventral heart sounds (possibly with borborygmia heard within the thoracic cavity), and shock are all possible sequelae. Thoracic radiographs will often reveal loss of the normal diaphragmatic outline, air-filled intestines or stomach within the thoracic cavity, and displacement of the heart, lungs and/or trachea by other soft tissues or effusion. Thoracic ultrasound and positive contrast gastrography may also be helpful. While surgical repair is necessary to repair the rupture, patients may have signs of shock on presentation and should be stabilized first with fluid therapy, oxygen support and analgesics. Surgery should be pursued as an emergency if gastrointestinal contents are within the thoracic cavity or respiratory stabilization cannot be achieved.

**Penetrating thoracic trauma**

Penetrating thoracic trauma secondary to bite wounds, impalement/stabbing injuries or projectile injuries can result in severe respiratory distress secondary to the location of the injury, other underlying injuries, and severe pain. While surgical exploration and
repair will eventually be required, initial treatment and stabilization should include supplemental oxygen if necessary, early antibiotic therapy and pain management. Sterile lubricating ointment and bandage material should be placed on the wound to prevent further contamination until exploration can be performed. Gunshot wounds are the most common type of projectile injury in veterinary patients and can result in extensive trauma to tissues in the direct path of the bullet as well as adjacent structures due to laceration and crushing of tissues. Damage to large vessels may result in a hemothorax, whereas penetration of the lung may result in a pneumothorax. Although surgical exploration of the thorax may not be necessary, it is indicated in patients with continued hemorrhage or air leakage.

**Other thoracic injuries**

High-rise syndrome involves injuries that result from a fall from a height. In cats, thoracic injuries are most common, including pneumothorax and pulmonary contusions. Pancreatic rupture and pancreatitis has also been reported in a small number of cats with high-rise syndrome. Dogs may also suffer thoracic trauma but are also commonly affected with extremity and spinal cord injuries. Treatment should be directed at the underlying injuries as discussed previously.

Tracheobronchial injury is uncommon with thoracic trauma but can be lethal. Tracheal rupture occurs due to stretching and tearing of the trachea, increased intrabronchial pressure, and shearing forces on the trachea during deceleration. Patients may have varying signs of respiratory distress or may even be asymptomatic initially; coughing and subcutaneous emphysema can also be seen. Thoracic radiographs may reveal presence of a pneumomediastinum, pneumothorax, or tracheal discontinuity. If clinical signs are mild, cage rest may be appropriate to allow the trachea to heal; in more affected cases, surgical correction may be necessary.

Myocardial contusions are caused by deceleration force on the chest wall, causing both direct compression of the myocardium and shearing stress secondary to increased intrathoracic pressure. Contusions may result in arrhythmias, which can be delayed following trauma (12-48 hours). Patients may have evidence of hemodynamic compromise, and an EKG should be performed if arrhythmias are suspected. Treatment consists of therapy as indicated for shock, pain management, and anti-arrhythmics is necessary.

Thoracic injuries are common in veterinary patients, and the presence of a single thoracic injury should prompt one to look for comorbidities. Key concepts in management of thoracic trauma include treatment of shock, the use of thoracocentesis as a diagnostic and therapeutic tool, and the importance of pain management in treatment. In severe cases, mechanical ventilation may be necessary.

**References**


Acute renal failure (ARF) has been a recognized challenge to human and veterinary clinicians since the 1950s. Despite medical advances, the mortality rate is still 50-60% in human patients, and presumably higher in veterinary patients, although large studies are lacking. There are more than 35 definitions of ARF in the human literature, which makes it challenging to identify and classify patients with ARF. Subsequently, the term “acute kidney injury” (AKI) has been created to encompass the dynamic process of kidney injury that often leads to a loss of function of the organ. Acute kidney injury is defined as damage to nephron units that may or may not lead to renal failure. A staging system for veterinary AKI (VAKI) was recently examined which classifies the severity of injury based on the increase in serum creatinine from baseline value into four categories. The study demonstrated that the greater the increase in creatinine during hospitalization (and the higher the VAKI stage), the worse the prognosis.

There are several known predisposing factors for the development of acute kidney injury: preexisting renal disease, dehydration, concurrent cardiovascular or hepatic disease, sepsis, and the use of diuretic or drugs with potential nephrotoxicity. Clinical recognition of high-risk patients is the first step in prevention. A majority of preventable cases of acute kidney injury are caused by prolonged hypotension secondary to anesthesia, hemorrhage, or severe inflammatory disease (i.e. septic or non-septic systemic inflammatory response syndrome or SIRS). Acute kidney injury in the critically ill patient may be functional (prerenal, hemodynamic instability), intrinsic renal (vasogenic, renal vasculopathy, tubular epithelial damage, glomerulopathy, interstitial pathology, hepatorenal, infectious, toxic), or postrenal (ureteric or obstruction).

Acute kidney injury is characterized by a sudden onset (days) of illness and may include anorexia, lethargy, depression, vomiting, diarrhea, and/or halitosis, muscle tremors, ataxia, seizures, known toxin exposure, medical or surgical diseases, recent trauma, and observed oliguria or polyuria. A thorough history from the owner is paramount, including any possible toxin exposure or administration of nephrotoxic drugs. Physical examination findings might include hypothermia, dehydration, lingual or oral ulcerations, necrosis of tongue margins, scleral injection, tachypnea, and bradycardia. The kidneys may be large, swollen, and painful upon abdominal palpation. The urinary bladder may be distended in animals with post-renal azotemia. Animals who have been receiving fluids prior to presentation may have peripheral or pulmonary edema, chemosis, and increased or decreased breath sounds on auscultation of the chest. Patients with acute kidney injury and no concomitant preexisting medical conditions may have normal body condition and a healthy haircoat and skin. Hypertension may be present (+/-retinal detachment or intraocular hemorrhage) and blood pressure should be monitored routinely. If hyperkalemia is present or an abnormal cardiac rhythm is ausculted, electrocardiographic evaluation should be pursued.

Clinical laboratory data from the animal with acute kidney injury often reveals a normal hematocrit (initially), elevated blood urea nitrogen (BUN) and creatinine, hyperphosphatemia, hypo- or hypercalcemia, hyperkalemia, metabolic acidosis, and isothenuria +/-casts, pyuria, bacteriuria, or other urine sediment abnormalities. A urine culture should be performed in all animals with acute kidney injury since bacterial infection is one of the more easily treatable etiologies. Many animals with acute kidney injury develop multiorgan abnormalities during hospitalization, such as gastrointestinal, hematologic, cardiovascular, pulmonary and neurologic derangements. Additional diagnostic modalities may include abdominal radiographs (with special attention to kidney size or the presence of uroliths), contrast studies (i.e. nephropelvogram, retrograde cystourethrogram), abdominal ultrasound to evaluate the renal architecture, and specific infectious disease testing (leptospirosis, rickettsial disease). Renal biopsy may be indicated in specific cases, especially those that will receive advanced treatment such as hemodialysis or renal transplant surgery. Renal biopsies may be especially useful in determining prognosis and appropriate treatment plans in some animals, as well as monitoring progress over time. Potential complications do exist, but are rare with appropriate precautions and expertise.

Management of the patient with acute kidney injury is fourfold: 1) reverse underlying cause or causes and ongoing risk factors (drugs, hemodynamic instability, concurrent diseases), 2) correct uremic intoxications and fluid, electrolyte, and acid-base imbalances, 3) establish adequate urine production, and 4) provide nutritional support until renal function is returned. Failure to initiate appropriate management can prolong and worsen renal damage, close finite windows of reversibility, and jeopardize a successful outcome. Animals with acute kidney injury are commonly dehydrated due to anorexia, vomiting, and diarrhea. An estimate of the fluid deficits should be made based on body weight, skin turgor, mucous membrane moistness, hematocrit, total solids, capillary refill time, arterial blood pressure and heart rate. Volume deficits (in liters) are calculated by multiplying the volume deficit by the body weight in kilograms, and should be restored within 4 to 6 hours using an isotonic replacement fluid. Blood products should be used as clinically indicated. Maintenance fluid requirements, including replacement of ongoing losses (vomitus, fecal, urinary), must also be administered.

Fluid balance should be closely monitored using physical examination, body weight, urine output, and indices of hydration. CVP monitoring may be helpful in guiding fluid therapy (N = 0-5 cm H₂O), although more recent evidence suggests that compounding
factors may make these values difficult to interpret. It is important that oliguric or anuric animals not become volume overloaded as this can be life threatening, causing pulmonary/organ edema and failure.

Severe metabolic acidosis and life-threatening hyperkalemia are frequently encountered complications in patients with acute kidney injury, although variable primary or mixed acid-base and electrolyte disturbances may develop. These abnormalities are dependent on the underlying disease process and extent of vomiting, diarrhea, and respiratory components. Treatment regimens should be based on assessment of serum bicarbonate, blood gases, serum potassium, and the electrocardiogram. Severe metabolic acidosis is (bicarbonate < 10 mEq/L and pH < 7.0) despite fluids therapy may require treatment with sodium bicarbonate. Sodium bicarbonate replacement (in mEq) is calculated by multiplying body weight (kg) x 0.3 x bicarbonate deficit. One half of the calculated replacement can be administered over 20-30 minutes, and the remainder provided over 4-6 hours. Sodium bicarbonate should be re-evaluated following replacement. It is important to remember that administration of sodium bicarbonate is not without risks; it can promote metabolic alkalosis, sodium excess, and volume overload in oliguric or anuric animals, potentiate a decrease in ionized calcium (and potassium) concentration, and induce paradoxical cerebral acidosis and cerebral edema in animals that are not adequately ventilating. Severe hyperkalemia may lead to life threatening cardiac disturbances and require immediate treatment. Potential treatment options include fluid diuresis (in mild cases), dextrose with or without insulin, sodium bicarbonate, and calcium gluconate. These treatments provide only transient protection from life threatening arrhythmias until renal potassium excretion is restored or hemodialysis is performed.

Failure to induce adequate diuresis with repletion of fluid deficits indicates severe renal parenchymal injury. Numerous therapeutic agents have been advocated to reduce the severity of renal injury or induce urine formation in oliguric or anuric animals. Although many of these therapeutic agents demonstrate beneficial effects in experimental forms of ischemic or toxic acute kidney injury, their efficacy in the clinical setting remains largely unknown. Conversion of oliguric or anuric acute kidney injury to a nonoliguric state greatly facilitates management of fluid, electrolyte, and acid-base imbalances; however urine output has no correlation with survival or prognosis. Drugs commonly used to promote urine output include mannitol, furosemide, and diltiazem. The use of dopamine remains controversial and is no longer routinely used to promote diuresis. Hypertonic mannitol (10-25%) is given to fluid replete animals as an initial slow bolus of 0.1 – 0.25 gm/kg intravenously (IV) over 20-30 minutes to promote diuresis. Mannitol acts to increase renal blood flow and tubular fluid flow, ameliorate epithelial swelling and intratubular obstruction, and scavenge free radicals. If significant diuresis is accomplished within 30 minutes, mannitol can then be started as a constant rate infusion (CRI) at 60-120 mg/kg/hour IV or as intermittent repeated boluses every 4-6 hours (as above). Mannitol is contraindicated in patients who are hypervolemic or have evidence of cardiac failure or pulmonary edema. Fluid balances must be carefully monitored to prevent dehydration and further renal compromise. Furosemide is a potent natriuretic agent with mild renal vasodilating effects. It is given as a bolus initially (0.1 – 0.5 mg/kg) IV in fluid replete patients. If adequate diuresis is not evident within 30 minutes, re-administration of the initial or higher dose is indicated. If diuresis ensues, the dose can be repeated every 6-8 hours or a CRI initiated (0.1 – 1 mg/kg/hour) IV. As with previous diuretics, fluid balances must be carefully monitored to prevent dehydration and further renal compromise. Calcium channel blocker drugs can cause renal afferent arteriolar dilation, inhibition of mesangial cell contractility, renin release, redistribution of RBF to juxtamedullary nephrons, natriuresis, and cytoprotection. Although prospective small animal studies are limited, the use of diltiazem in dogs with leptospirosis was recently reported in a retrospective study to improve renal recovery and have minimal side effects at low doses (0.1 – 0.5 mg/kg IV slowly, followed by 1 – 5 μg/kg/min CRI). Fenoldopam is a selective dopamine-1 receptor agonist that may prove useful in the management of acute kidney injury in small animals, but further research is necessary. It does not stimulate dopamine-2 receptors or alpha/beta receptors and may therefore avoid the potential deleterious effects associated with the use of dopamine. Preliminary research in experimental dogs and cats appears promising.

If a diuretic response cannot be established or maintained within 4-6 hours following restoration of fluid balance, dialysis should be considered. Peritoneal dialysis, intermittent hemodialysis (IHD), or continuous renal replacement therapy (CRRT) is required when conservative therapy medical management fails to increase urine production or the clinical consequences of azotemia, hyperkalemia, acid-base disturbances, and fluid overload cannot be immediately controlled. Although peritoneal dialysis is possible to perform in general practice, it is rarely life-saving in severely affected animals, but may stabilize them temporarily. As a more successful alternative, IHD and CRRT are offered at several critical care referral centers throughout the United States. IHD and CRRT serve to stabilize the complications resulting from acute kidney injury until the renal injury is repaired and excretory function regained.

Recovery from acute kidney injury may follow a prolonged clinical course during which afflicted animals are hypercatabolic and suffer from a variety of chemical and acid-base disturbances. Although precise nutritional requirements for small animals with acute kidney injury are unknown, a high-energy, moderate protein, potassium and phosphate deficient diet is a logical choice. In animals that are reluctant to eat appropriate amounts of food, enteral feeding with a nasoenteral, esophageal, or gastrostomy tube is indicated. If vomiting and nausea preclude enteral feeding, total or partial parenteral nutrition may be administered. Phosphate binders such as aluminum hydroxide or aluminum carbonate (30-90 mg/kg/day divided with feedings) should be used in conjunction with dietary phosphorus restriction in hyperphosphatemic animals.

In contrast to chronic renal insufficiency, animals with acute kidney injury have the potential for complete or partial recovery. The prognosis for recovery is good in animals with pre- and postrenal causes of azotemia if the underlying cause is identified and
Mild nonoliguric kidney injury carries a guarded prognosis, but complete or partial recovery of renal function may be expected over 3-6 weeks. Oliguric kidney injury carries a guarded to poor prognosis, but a sudden onset of diuresis and gradual return of renal function over 4-12 weeks is possible. Anuric kidney injury or conditions associated with multiple organ failure carry a grave prognosis for recovery without hemodialysis or CRRT for extended periods. Renal transplantation is available for cats and dogs that meet preoperative criteria at the University of Pennsylvania and additional select referral centers in the United States.

References available from the author.
In general, human and veterinary patients are administered fresh frozen plasma (FFP) transfusions for two main reasons: prophylactically, to prevent clinical bleeding, and therapeutically, to stop active bleeding. In human medicine, clinical use of FFP continues to grow, but many patients who receive plasma, while coagulopathic, do not have evidence of clinical bleeding. These patients are also of interest in veterinary medicine, since many canine and feline patient who receive FFP do not have evidence of bleeding.

**Fresh frozen plasma, models of coagulation and coagulation testing**

Fresh frozen plasma (FFP) is plasma that has been separated from whole blood and frozen within eight hours. It contains coagulation factors, anticoagulation factors such as antithrombin, fibrinogen, albumin, and alpha-macroglobulins. It can be stored for up to one year when frozen at -20 to -30 degrees Celsius. Frozen plasma (FP) is less commonly used in veterinary medicine; it is plasma that is separated from whole blood but not frozen within eight hours, or that has been frozen quickly but stored between one and four years. FP contains the stable coagulation factors II, VII, IX and X as well as albumin.

There are two models of coagulation: the classic model and the cell-based model. The classic model of coagulation is based on the coagulation cascade, which involves a sequence of steps in which enzymes cleave proenzymes to generate the next enzyme in the cascade. Most steps require calcium and occur on phospholipid membrane surfaces. The process is divided into two pathways: the extrinsic pathway, which is localized outside of the blood and involves tissue factor (TF) and factor VIIa; and the intrinsic pathway, which is localized within the blood and initiated through contact activation of factor XII on negatively charged surfaces. The intrinsic and extrinsic pathways both lead to the common pathway, in which activation of factor X leads to activation of prothrombin to thrombin, and then fibrinogen to fibrin, resulting in a cross-linked fibrin clot. The cascade model is helpful because it allows for a more useful interpretation of commonly used laboratory tests. The prothrombin time (PT) test looks for abnormalities in the extrinsic and common pathways, and the activated partial thromboplastin time (aPTT) test looks for deficiencies in the intrinsic or common pathways. The usefulness of the coagulation cascade is limited by its inability to explain how coagulation works in the body during interactions with the vascular wall or cell surfaces. The newer model of coagulation is the cell-based model, which allows for a more integrated understanding of the mechanisms of coagulation in the dynamic vascular system. It suggests that coagulation occurs in distinct overlapping phases and requires two main cell types: the tissue factor (TF)-bearing cell and the platelet. The three main phases of coagulation in the cell-based model are initiation, amplification and propagation. In the first phase, initiation, injury occurs and blood is exposed to a tissue factor-bearing cell, causing factor VII to become activated and eventually resulting in production of a small amount of factor IXa and thrombin that diffuse from the surface of the TF-bearing cell to the platelet. During amplification, the small amount of thrombin from the initiation phase activates platelets, releasing von Willebrand factor and leading to the generation of activated forms of factors V, VIII and XI. During the final phase, propagation, enzymes activated during earlier phases assemble on the procoagulant surface of the activated platelet to form intrinsic tenase, which leads to factor Xa generation on the platelet surface, and a burst of thrombin generation directly on the platelet which results in fibrin formation.

The main tests of coagulation used for detecting hypocoagulable states include the prothrombin time (PT) and activated partial thromboplastin time (aPTT). Both of these tests were developed to investigate coagulation factor deficiencies in human patients with a history of bleeding, and provide an end-assessment of thrombin generation by fibrin formation. Numerous studies in human medicine have demonstrated that they are poorly predictive of bleeding complications in patients.

**Evidence in veterinary medicine to support FFP use**

In veterinary medicine, the evidence to support or refute FFP use is lacking. In one study examining the indications for FFP use in dogs over a 3-month period in a large urban teaching hospital, the authors found that 42/74 dogs received plasma for provision of coagulation factors, but only 22/42 of those patients had evidence of clinical bleeding. Other indications for FFP transfusion included albumin support (45/74 dogs), provision of immunoglobulins (12/74 dogs) and provision of alpha-macroglobulins (10/74 dogs). A more recent retrospective study characterizing the use of plasma at another veterinary teaching hospital found that FFP was used primarily for coagulopathies rather than hypoalbuminemia or pancreatitis, but there was no association between volume of FFP given and patient outcome, although the presence or absence of hemorrhage was not investigated. While hypoalbuminemia remains an often-cited reason for administration of FFP, this study found no difference in serum albumin levels pre- and post-transfusion (median FFP dose was 15-18 ml/kg). Given that FFP contains a relatively small amount of albumin and the volumes required to cause a significant change is large, this result is not surprising. One additional study investigating the effectiveness of FFP for pancreatitis (to replenish antiproteases) revealed a higher mortality rate in dogs that received FFP, although illness severity scoring was not
performed, and these patients may have been more critically ill. The preliminary results of a study examining the coagulation response in dogs with and without SIRS found that both groups of ill dogs had evidence of hypocoagulability, leading them to conclude that severe coagulopathies may be present in critically ill dogs without concurrent SIRS.

While guidelines for the use of FFP in human patients suggest that it should be reserved for patients with active bleeding or prior to surgery in coagulopathic patients, no such guidelines exist for veterinary use. The controversy regarding the effectiveness of FFP in non-bleeding critically ill patients is ongoing, and further discussion in both the human and veterinary medical communities is needed. Given the risks associated with transfusion, the labor required to collect blood products, and the associated costs of administration, evidence-based veterinary guidelines are essential. The risks and potential benefits of FFP transfusion should be carefully considered in each and every patient before administration.

Stabilization and ER Management of Blocked Cats
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Feline urethral obstruction (UO) is commonly encountered in veterinary medicine and can be life-threatening without rapid treatment. While the prognosis for UO is generally quite good, mortality can vary, ranging from 5.8-26% in several studies (including cases that died or were euthanized) and recurrence is also common, affecting about 1/3 of patients. As with any patient presenting for emergency evaluation, a primary survey should be conducted initially to ensure that the patient is stable. Major body systems should be quickly assessed, including airway and breathing, circulation (heart rate, mucous membrane color, capillary refill time, pulse quality), auscultation (cardiac and pulmonary), level of consciousness and urogenital. In cases of acute uremia, hyperkalemia may need to be immediately treated. Since most of us are comfortable treating the “routine” blocked cat, this lecture will focus briefly on initial stabilization, and then on some more novel treatments and potential complications of urethral obstruction in cats.

Caudal epidural
Prior to the unblocking process, a caudal epidural is a quick, simple procedure that can be performed using preservative-free lidocaine, bupivacaine, and/or morphine. Injection into the caudal epidural space provides anesthesia to the urethra and penis without affecting motor function to the hindlimbs, and can facilitate passage of the urinary catheter. A recent abstract presented at IVECCS revealed that caudal epidurals appear to be safe, might decrease the amount of propofol needed during the unblocking process, and can provide nice adjunctive analgesia while patients are unblocked and in the hospital. Usually the author uses lidocaine for this procedure (0.1-0.2 ml/kg of 2% preservative-free lidocaine), but it can be combined with bupivacaine or morphine to prolong the effects. Once the patient is sedated, position the cat in ventral recumbency and clip and prep the sacrococcygeal region. To find the injection site, palpate the space between the sacrum and the first coccygeal vertebrae, which is where the tail moves at the base of the body. Draping can be performed to facilitate aseptic technique, and sterile gloves must be worn. Insert a 25-ga, 1-inch needle into the sacrococcygeal space at a 30-45° (a “pop” may be felt when penetrating the ligamentum flavum) and attach a syringe, applying gentle negative pressure. If no blood or CSF is aspirated, slowly infuse the lidocaine. Success of the procedure can be verified by pinching the tail base; there should be no response. Routine unblocking can then be performed. This technique is very helpful in patients with significant urethral spasm or in those who have issues maintaining urinary catheters in the hospital due to discomfort or agitation.

Using general anesthesia
While many emergency doctors learned to unblock cats under heavy sedation with careful monitoring, general anesthesia can be helpful for those tricky unblockings. In addition to providing more smooth muscle relaxation, it also provides more time to work out the kinks in the unblocking process.

The value of a radiograph
A lateral abdominal radiograph should always be performed following the unblocking procedure if finances will allow. Not only will it help to evaluate the urinary system for radiopaque stones, which affect around 20-29% of cats, but it can help avoid complications of catheter placement. The catheter should be visualized in the urinary bladder and adjusted if necessary.

Urine analysis and culture
Obtaining a urine sample at the time of unblocking can provide valuable information about the patient’s underlying disease process, including the presence of cystic calculi, crystalluria or infection. Knowing this information from the beginning can help to provide owners with recommendations for further treatment and therapy. At the end of hospitalization, when the urinary catheter is removed, it can also be helpful to obtain a urine culture. While we try to be as aseptic as possible during the unblocking process, infection can be introduced, and positive culture results occurred in 13-33% of cats in two recent studies. Culture samples can be obtained by either taking a sample through the catheter right before removal, or performing a cystocentesis; culturing the tip of the catheter is not recommended due to risk of contamination during catheter removal. While sending all blocked cats home on antibiotics is not recommended since the chances of a UTI are low, culture can help provide appropriate antimicrobial therapy to those patients who do need it.

Anemia and urinary bladder hemorrhage
While uncommon, a small percentage of cats with urethral obstruction can develop urinary bladder hemorrhage. With this condition, severe anemia requiring blood transfusion may result, and some cats can reobstruct due to a blood clot in the bladder, developing recurrent signs secondary to hyperkalemia and worsening azotemia, with a urinary catheter in place. A history of previous urethral obstruction and longer duration of clinical signs may be important risk factors for severe anemia. Additionally, compared to “routine”
UO cats, cats with suspected urinary bladder hemorrhage appeared to be more severely affected as evidenced by lower blood pressure, more severe metabolic acidosis, higher BUN and creatinine, and worse outcome in one retrospective study.\textsuperscript{6}

**Post-obstructive diuresis**

Post-obstructive diuresis, or a urine production > 2 ml/kg/hr, occurs in a fair number (46\% of cats in one study) of blocked cats within six hours of unblocking.\textsuperscript{3} While the exact causes of post-obstructive diuresis are unknown, it is thought to be due to a number of factors including accumulation of osmotically active particles in the blood, medullary washout and tubular dysfunction. It can lead to massive urine output and hypovolemia if not addressed. While fluid therapy in cats can be challenging, and high fluid rates cause concern for fluid overload, it is incredibly important to meet these cats’ fluid requirements. Matching the fluid rate to the urine output (plus a small amount if the patient is still azotemic) is recommended initially, with slow weaning as their azotemia resolves. Monitoring is crucial here: frequent assessments of urine output, PCV/TS, and body weight are essential.

**References**


Hypoglycemia
In healthy patients, euglycemia occurs through a balance of glucose storage, release from storage, and production; hypoglycemia occurs when this balance becomes disrupted. In patients with diabetes mellitus, the most common causes of hypoglycemia are exogenous insulin overdose, either accidentally or because the patient is ill and anorexic or hyporenal. In cats, especially, hypoglycemia can also occur secondary to insulin dosing during spontaneous remission of diabetes mellitus. Veterinary patients at higher risk of hypoglycemia include obese patients and cats receiving more than 6 units of insulin per dose.

While hypoglycemia is technically defined as a blood glucose (BG) concentration ≤ 60 mg/dl, clinical signs often do not develop until BG are less than 50 mg/dl. To confirm hypoglycemia, a patient should have three things: clinical signs of hypoglycemia, a BG ≤ 60 mg/dl, and resolution of signs with treatment. In most emergency rooms, glucometers are used to determine blood glucose levels. These small handheld tools are convenient, inexpensive, and can be used with serum, plasma or whole blood. Recent research has shown that measurement of whole blood glucose using a glucometer generally results in values that are roughly 30 mg/dl lower than serum glucose values in both dogs and cats. Since handheld glucometers may be inaccurate, results should be confirmed via other methods. Sample collection can also affect BG readings – if serum is not separated from red blood cells within 30 minutes of collection, falsely low BG values may result due to consumption of glucose by red blood cells.

Clinical signs of hypoglycemia are often related to neuroglycopenia, or hypoglycemia of the central nervous system. Because glucose is the brain’s obligate energy source, a continuous stream of glucose is needed to maintain brain function, and lack of glucose results in neurologic signs. Common clinical signs include mental dullness, abnormal mentation, weakness or recumbency and seizures. Permanent brain damage can occur if neuroglycopenia is prolonged. Other signs in veterinary patients include vocalizing, shaking or trembling, anorexia, vomiting, panting, urination and diarrhea. In severe cases, bradycardia or cardiovascular collapse are possible.

Treatment of hypoglycemia in the diabetic patient should include three steps: (1) discontinue insulin therapy, (2) supplement dextrose intravenously as needed, and (3) feed the patient as soon as possible. Initially, because patients are often severely affected, mentally apathetic and unable to eat, dextrose should be supplement intravenously. A bolus dose of 0.5-1.0 mL/kg of 50% dextrose should be diluted 1:2 to 1:4 and given slowly IV over 3-5 minutes. Most patients will respond favorably to this treatment within minutes. If the patient is then alert and able to eat, nutrition should be provided in small, frequent meals to maintain euglycemia. If not, dextrose can be supplemented intravenously at a continuous rate infusion (CRI) of 2.5-5% by adding 50% dextrose to an isotonic fluid. BGs should be checked frequently and supplementation adjusted as needed; if greater than 5% dextrose is required as a CRI, it should be administered through a central line to prevent phlebitis.

The duration of hypoglycemia following insulin overdose varies, and may not be dependent on the amount and type of insulin given, especially if the patient is systemically ill and has other factors contributing to hypoglycemia. Once the patient is improving, BG is stable on supplementation, and the patient is eating, the dextrose CRI can be slowly weaned with careful monitoring of BG levels. In some patients, insulin therapy will need to be reinstituted fairly quickly, while other patients may take days before they require insulin again. Careful monitoring should be provided to verify the need for continued insulin treatment. When restarting therapy, the dose should be reduced by 25-50%, and follow up with a glucose curve should be performed in 5-10 days.

Diabetic ketoacidosis (DKA)
DKA is a serious complication of diabetes mellitus that often requires emergency and critical care. DKA occurs in diabetic patients – often in new diabetics – when glucose cannot be transported into cells, and ketones are synthesized from fatty acids as an alternate source of energy. Ketones accumulate in the body, causing life-threatening electrolyte abnormalities and acidosis. There are three major ketones produced in the body from acetyl-CoA: acetone, acetoacetate, and β-hydroxybutyrate. In diabetics that develop DKA, low levels of endogenous insulin, as well as increased glucagon, catecholamines and cortisol, contribute to production of ketones and secondary ketotic acidosis. Because concurrent illness is common, acidosis may also be worsened by systemic illness and vomiting, dehydration and poor renal perfusion.

Dogs and cats that develop DKA are generally older (median age in dogs 8 years, mean age in cats 9 years) and of variable breeds or sex. Concurrent diseases are common and affect about 90% of cats with DKA and 70% of dogs. Common concurrent diseases include pancreatitis, urinary tract infections, hyperadrenocorticism in dogs, hepatic lipidosis in cats, chronic renal failure and neoplasia.

Clinical signs of DKA are often secondary to the underlying concurrent problem, or may be reflective of unmanaged diabetes mellitus. Polyuria, polydipsia, vomiting, anorexia or decreased appetite, weight loss and lethargy are common in both dogs and cats. A
diagnosis of DKA is characterized by hyperglycemia, acidemia (pH < 7.3) and ketonemia or ketonuria. Electrolyte abnormalities are also common. Patients are often critically ill on presentation, and fluid therapy as well as electrolyte supplementation are paramount to stabilization. Some of the more commonly encountered electrolyte abnormalities include hypokalemia (even in patients that initially appear hyperkalemic) due to total body depletion of potassium, which may worsen with insulin therapy as potassium is shifted into cells, hypophosphatemia, hypomagnesemia, hypocloremia and hypocalcemia. Hyponatremia is also commonly noted, due to hyperglycemia and movement of water into the extracellular fluid compartment; this is called pseudohyponatremia.

Initially, fluid therapy is critical to patient stabilization. Those with evidence of hypovolemia should receive fluid boluses as needed to achieve stabilization, and while controversy exists about the “best” type of fluid to use, any balanced isotonic crystalloid is acceptable. While some newer evidence suggests that early insulin therapy may be beneficial in speeding resolution of ketonemia in some patients, the author still prefers to begin fluid therapy and rehydration over 4-6 hours before beginning insulin therapy. Fluids alone will often significantly decrease the blood glucose concentration, and may also affect electrolyte concentrations. Patient sodium should be monitored frequently with fluid therapy, as it should increase as the hyperglycemia resolves.

Along with initial stabilization with fluids, electrolyte abnormalities should be addressed. Hypokalemia should be treated with an intravenous CRI, not exceeding 0.5 mEq/kg/hr. Hypophosphatemia often also needs to be addressed, and it should be noted that phosphorus supplementation is provided as potassium phosphate (4.4 mEq/ml of potassium and 3 mM/ml of phosphate), so potassium supplementation may need to be adjusted; the recommended rate is 0.03-0.12 mEq/kg/hr. Magnesium may also need to be supplemented as a CRI of magnesium sulfate (0.5-1 mEq/kg/day).

Once the patient has been stabilized and initial rehydration has begun, insulin therapy can be instituted with regular (Humulin R, Novolin R) insulin. Insulin can be given as an IV CRI, or as intermittent IM injections, and BGs can be checked using a glucometer or a continuous intrastitial glucose monitoring system. If IM insulin is being administered, doses are generally 0.2 U/kg regular insulin once, and then 0.1 U/kg one hour later. BGs should be monitored hourly, and insulin dosing adjusted based on readings (usually 0.05 U/kg if the BG drops by more than 75 mg/dl/hr., 0.1 U/kg if it drops by 50-75 mg/dl/hr, or 0.2 U/kg if it drops by less than 50 mg/dl/hr. A general table for insulin administration CRI is provided below; this table can be adjusted based on the patient’s blood glucose changes (Table 1). Patients can be transitioned to long-acting insulin once they are eating and ketonemia/ketonuria has resolved.

While acidosis usually resolves with fluid therapy and insulin, severe metabolic acidosis (bicarbonate < 10 mEq/L and pH < 7.0) despite fluid therapy may require treatment with sodium bicarbonate. Sodium bicarbonate replacement (in mEq) is calculated by multiplying body weight (kg) x 0.3 x bicarbonate deficit. One-third to one half of the calculated replacement can be administered over 20-30 minutes, and the remainder provided over 4-6 hours. Serum bicarbonate should be re-evaluated following replacement. It is important to remember that administration of sodium bicarbonate is not without risks; it can promote metabolic alkalosis, sodium excess, and volume overload in oliguric or anuric animals, potentiate a decrease in ionized calcium (and potassium) concentration, and induce paradoxical cerebral acidosis and cerebral edema in animals that are not adequately ventilating.

Because concurrent diseases are so common in patients with DKA, identification of any comorbidities is important and will affect prognosis. While prognosis is generally good (70% of dogs and cats survive to hospital discharge), hospitalization can be prolonged and may involve intensive care.

### TABLE 1. Administration of IV Insulin to Patients with DKA²

<table>
<thead>
<tr>
<th>BG concentration (mg/dl)</th>
<th>Fluid composition</th>
<th>Rate of administration (ml/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;250</td>
<td>0.9% NaCl</td>
<td>10</td>
</tr>
<tr>
<td>200-250</td>
<td>0.9% NaCl + 2.5% dextrose</td>
<td>7</td>
</tr>
<tr>
<td>150-200</td>
<td>0.9% NaCl + 2.5% dextrose</td>
<td>5</td>
</tr>
<tr>
<td>100-150</td>
<td>0.9% NaCl + 5% dextrose</td>
<td>5</td>
</tr>
<tr>
<td>&lt;100</td>
<td>0.9% NaCl + 5% dextrose</td>
<td>Stop insulin</td>
</tr>
</tbody>
</table>

*2.2 U/kg regular insulin added to 250 ml of 0.9% NaCl solution. The administration set must be flushed with 50 ml of the mixture before administering to the patient.

**Hyperglycemic hyperosmolar syndrome (HHS)**

The least common form of diabetic crisis, HHS, is characterized by severe hyperglycemia (> 600 mg/dl), minimal or no ketones, and hyperosmolality (> 350 mOsm/kg). While HHS occurs in a similar fashion to DKA, the key difference is that ketosis is prevented by glucose resistance and small amounts of insulin. Severe hyperglycemia results, causing further osmotic diuresis, dehydration and hyperosmolality. Due to hyperosmolality, neurologic signs may be noted in addition to general signs of illness in patients with HHS and may include circling, mentation changes, seizures, cranial nerve abnormalities, weakness or ataxia.

With HHS, glucose levels can become extremely elevated (1000-1600 mg/dl), and will often be unreadable on glucometers. Actual glucose levels should be confirmed using serum chemistry to allow for monitoring over the course of treatment. Osmolality can be estimated based on the following formula:

\[
\text{Serum osm (calc) = 2(Na+) + (BUN/2.8) + (Glucose/18)},
\]

where BUN and glucose are measured in mg/dl and Na is measured in mEq/L. Normal serum osmolality is around 300 mOsm/kg. Similar to patients with DKA, electrolyte abnormalities are also common.
In patients with HHS and frequent monitoring is essential. Because of the severity of hyperglycemia, sodium concentrations should be monitored frequently and pseudohyponatremia is likely. The corrected serum sodium concentration can be calculated as:

\[
Na_{(corr)} = Na\ (measured) + 1.6([\text{measured glucose} - \text{normal glucose}]/100)
\]

Where sodium is measured in mEq/L and glucose is measured in mg/dl. As hyperglycemia resolves, sodium levels should also return to normal.

While HHS patients, similar to DKA patients, often require fluid resuscitation and careful monitoring of electrolytes with supplementation as needed, insulin therapy is often not as important. Initial treatment should be focused on fluid resuscitation with careful monitoring of patient sodium, with care taken not to drop sodium concentration too rapidly, which can exacerbate neurologic signs. In general, sodium should be decreased at a rate no greater than 1 mEq/L/hr. Normal saline (0.9%) is generally the initial fluid of choice due to its sodium concentration. As with DKA patients, fluid therapy alone will begin to resolve the patient’s hyperglycemia. Following fluid resuscitation and treatment of dehydration, insulin therapy can be instituted, similar to in patients with DKA. Usually, a similar protocol with lower insulin levels (50% that of the DKA protocols) are indicated to prevent the blood glucose from dropping too quickly (goal 50-75 mg/dl/hr). Concurrent diseases can also contribute to HHS and should be investigated over the course of treatment. Survival for HHS patients is significantly lower than for patients with DKA, due to both severity of disease and the presence of comorbidities.

References


Intravenous fluid therapy is vital for the management of shock, interstitial dehydration, and daily maintenance fluid needs in critically ill. This lecture will focus primarily on the distribution of total body water, patient assessment, and the delivery of intravenous fluids to resuscitate critically ill dogs and cats that are hemodynamically unstable. Because critically ill animals often have fluid and electrolyte balance derangements, overall recovery often depends on recognition and appropriate treatment of these disorders, in addition to diagnosing and treating the primary disease process. There are three main clinical indications for fluid therapy: resuscitation, rehydration, and to meet maintenance needs. In determining the reason(s) that a patient may require fluids, certain questions should be considered: What fluid space is deficient? If resuscitation is required, what type of shock is present? And, are there any contraindications to giving fluids?

As we know, living organisms are predominantly composed of water. Total body water content is approximately 60% of body weight in a non-obese adult dog or cat. Total body water is distributed between two main compartments: intracellular fluid (ICF) and extracellular fluid (ECF). Each compartment consists of solutes, primarily electrolytes, dissolved in water. The ICF compartment is the larger of the two and comprises 66% of the total body water (40% of body weight). It is separated from the ECF compartment by the cell membrane, which is very permeable to water but impermeable to most solutes. The ECF comprises the remaining 33% of the total body water and 20% of body weight. The ECF is subdivided into the plasma (25% of ECF) and interstitial (75% of ECF) fluid compartments. The interstitial fluid bathes all cells and includes lymph.

Water moves freely within most compartments in the body. Small particles such as electrolytes move freely between the intravascular and interstitial compartment but cannot enter or leave the cellular compartment without a transport system. Larger molecules (>20,000 Daltons) do not easily cross the vascular endothelial membrane and may attract small, charged particles, thus creating the colloid osmotic pressure (COP). There are three main natural colloid particles: albumin, globulins, and fibrinogen. An increase in the pressure of fluid within a compartment that pushes against a membrane is known as hydrostatic pressure. In health, fluid distribution within the ECF is determined by the balance between forces that favor reabsorption of fluid into the vascular compartment (increased COP or decreased hydrostatic pressure) and those that favor filtration out of the vascular space (decreased COP or increased hydrostatic pressure). Changes in the osmolarity between any of the fluid compartments within the body will cause free water movement across the respective membrane.

Before choosing a fluid for resuscitation, considering the type of shock is important. By definition, shock involves impaired tissue perfusion due to inadequate delivery or utilization of oxygen. In all cases, a major goal of resuscitation is to improve oxygen delivery to or utilization by the tissues. While it is clear that cardiogenic shock should be identified early as fluids are often contraindicated, differentiating hypovolemic and distributive shock is also very important. In hypovolemic shock, an absolute or relative reduction in blood volume exists, whereas in distributive shock, inappropriate vasodilation leads to poor perfusion despite an often normal blood volume. In hypovolemic shock, isotonic crystalloids, hypertonic crystalloids, synthetic colloids and blood products can all be considered. In distributive shock, arguments may exist for specific types of fluid or for conservative fluid resuscitation and early pressor therapy. Isotonic crystalloids are electrolyte containing fluids that are similar in composition to the ECF and have a similar osmolality to plasma. They are commonly used for resuscitation, and cause extracellular expansion, but redistribute quickly to the interstitial space. While isotonic crystalloids are often well-tolerated for resuscitation in hypovolemic patients, they can cause damage when they redistribute to the interstitial space. In critically ill patients (including those with distributive shock and a low COP, increased capillary permeability, etc.), increases in interstitial fluid can cause tissue edema, including cerebral and pulmonary edema.

Hypertonic crystalloids (most often 7.2-7.5% hypertonic saline) have an osmolality approximately eight times that of plasma (2400 mOsm/L) and can be used for rapid intravascular volume expansion – up to 3.5 times greater than the volume infused. By creating an osmotic gradient from the intracellular to extracellular space, hypertonic saline decreases intracellular volume and increases vascular volume. As a crystalloid, the effects are short lived, but rapid volume expansion can be helpful in cases of hypovolemic shock, especially for small volume resuscitation or in large patients. Hypertonic saline is commonly used for resuscitation of the head trauma patient or in those with intracranial hypertension. It has also been shown to decrease endothelial swelling, improve cardiac contractility, and improve cardiac output and tissue perfusion by decreasing afterload and increasing preload. Immunosuppressant effects (inhibition of neutrophil respiratory burst activity and cytokine effects) have also been reported. Since hypertonic saline is usually provided as a 23.4% solution, it must be diluted before use. This can be done by mixing it with an isotonic crystalloid in a 1:2 ratio (hypertonic saline: crystalloid). Typical doses are 3-5 mL/kg over 5-10 minutes. Too rapid administration (> 1 mL/kg/min) can result in hypotension and bradycardia. Synthetic colloids are isotonic crystalloids to which large molecules have been added. Administration results in increases in COP and a pull of fluid into the vascular space from the interstitial space. Colloids also increase the intravascular volume greater than the volume infused (about 1.4-1.5 times). In addition to volume expansion, synthetic colloids remain in the intravascular space longer than crystalloids and provide a more sustained effect. The synthetic colloids most commonly
used in the United States include VetStarch, a 6% tetrastarch veterinary product, and 6% hetastarch. Colloids can adversely affect coagulation via dilutional effects and interference with platelet function, von Willebrand’s factor and factor VIII, with prolongation of bleeding times seen at doses higher than 20 ml/kg/day. In human patients, concern regarding the occurrence of acute kidney injury has led to the removal of synthetic colloids from the market in some countries. In veterinary medicine, recent studies have shown mixed results correlating the use of synthetic colloids to acute kidney injury. Based on these concerns and a number of large human trials that have shown no clear benefit to using synthetic colloids, the author no longer uses these products.

Regardless of the type of fluid chosen for resuscitation, two main points should be remembered. Many years of research and clinical studies in human patients and animal models (and some veterinary patients) have failed to find an optimal fluid type overall. Thus, it is of utmost importance to choose a fluid that one is comfortable using and base resuscitation needs and success on monitoring the end points of resuscitation. A combination of variables including physical exam parameters, lactate, base deficit, arterial blood pressure, cardiac output, mixed venous oxygen saturation and central venous pressure can be helpful in guiding therapy. In addition, fluid therapy is not benign and potential complications include volume overload (organ edema and cavitary effusion), electrolyte changes and dilutional effects on hematocrit, albumin and coagulation factors. In human studies, more recent evidence has highlighted the importance of conservative fluid therapy, especially in patients with septic shock.

References available upon request.