Emergency Medicine and Critical Care

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CIRCULATORY SHOCK AND FLUID THERAPY – PART I HYPOVOLEMIC
Tim B. Hackett DVM MS DACVECC

The Emergency Phase

When a patient is has clinical signs of shock, attempts to restore circulating volume starts with crystalloid fluids. Circulatory shock can be due to inadequate volume, poor cardiac function, maldistribution of blood flow or a combination. The initial “shock volume” of fluids is often given and serves to answer the question: “Is this patient volume responsive?” Aggressive fluid loading has the potential to cause harm. One of the worst things we can do is cause pulmonary edema when pushing fluids too quickly. It is essential for all members of the team to be cognizant of the possibility and aware of signs of early fluid overload.

The “shock” volume of fluid necessary to reverse the signs of shock is different for every patient. Rather than giving a full blood volume each hour of crystalloid solutions it is safer to carefully titrate fluids while observing the patient for evidence of resolution and fluid overload. Rather than giving textbook shock volumes (In the dog this has been 90 ml/Kg/hr and for the cat about 44 ml/Kg/hr) we recommend giving 25% of this volume to find out if the patient is volume responsive. Once we see clinical resolution of shock (normal heart rate, improved pulse quality, normal capillary refill) we can move to the second phase of fluid resuscitation (dehydration phase).

One must be cautious about overhydration and hemodilution. Overhydration during the emergency phase is most likely to occur when large volumes are administered to animals with pulmonary contusions, preexisting pulmonary edema, aspiration pneumonitis, hypoproteinemia, brain injuries, and congestive heart failure.

Hypovolemic Shock

The problem in hypovolemic shock is an inadequate circulating volume. This can be from sudden massive blood loss as in surgery or trauma or fluid loss from vomiting, diarrhea or renal disease. Because cardiac output relies on stroke volume and heart rate, the patient with inadequate volume will be tachycardic to compensate. Neurohormonal pathways detecting a drop in blood pressure will lead to increased vascular tone in an attempt to shunt circulation from the periphery to vital tissue beds. This results in cool extremities, tachycardia, prolonged capillary refill, oliguria and weakness.

Treatment should be directed at the primary source of fluid loss while correcting the fluid deficit. Crystalloid fluids can be used initially to restore circulating volume. Crystalloids will improve cardiac output and should not be withheld for fear of diluting the red blood cell mass. Oxygen delivery is a function not only of oxygen content but of cardiac output as well. With a treatment goal of improving oxygen delivery to the tissues we can increase cardiac output by increasing stroke volume (fluids). Oxygen content can be increased by increasing the hemoglobin concentration (Red cell transfusion) and increasing oxygen saturation (Oxygen supplementation).

Volumes of fluid for resuscitation should be tailored to the individual patient. An initial goal with crystalloid fluids is to give a blood volume (40 ml/lb) in an hour. This is often more than enough fluid and in extremely debilitated patients may lead to fluid overload (pulmonary and cerebral edema). It may be more practical to titrate this dose in 1/4 increments. (makes the math easier too!). Give 10 ml/lb of crystalloids rapidly and reassess the patient’s clinical signs. Are the pulses stronger? Slower? Is the patient more alert? If not and we determine the shock state still exists give another 10 ml/lb.
Following the second dose of fluids the packed cell volume and total solids should be compared to prefluid values. If a patient receiving large quantities of crystalloids becomes anemic or hypoproteinemic, the fluid should be switched to an appropriate colloid such as whole blood, packed red blood cells, plasma or a synthetic product like hetastarch or dextrans. If the total solids have dropped to less than 50% of pretreatment a colloid should be considered for further resuscitation. If the PCV has dropped precipitously, whole blood and a search for the source of blood loss is indicated. Often, in the case of traumatic hemorrhage, correction of blood loss and pressure can open torn vessels leading to more hemorrhage. Therefore close attention is important. Once the shock is controlled, fluid deficits can be replaced along with maintenance volumes and ongoing losses over the course of one to two days.

**Crystalloid Fluids for Resuscitation**

Crystalloid fluids are mixtures of sodium chloride and other physiologically active solutes. They are generally isotonic with plasma and have sodium as their major osmotically active particle. The distribution of sodium determines the distribution of infused crystalloid fluids. Sodium is the major solute in the extracellular space and 75% of the extracellular space is extravascular. Therefore, infused sodium will reside primarily outside the vascular compartment.

**Colloids**

Fluid solutions containing large molecules help to pull water into the vascular space. VetStarch and similar fluids are used when hypoproteinemic patients continue to need fluids. While every patient is different I usually consider adding a colloid when the total protein (or total solids read by refractometer) fall below 4.5 g/dl. Other colloids include plasma, whole blood and packed red blood cells, and albumin transfusions.

**Hypertonic Saline**

The use of concentrated crystalloid solutions is appealing because of the reduced volumes of fluid required. This decreases the risks of pulmonary edema and the need for specialized equipment for delivery of very large volumes of fluids. Combining hypertonic saline with something like 6% dextran-70, Hetastarch or VetStarch will prolong the response. Hypertonic saline was popular for rapid volume replacement with 4 ml/kg quickly giving the volume expanding effects of a 90 ml/kg isotonic crystalloid. More recently hypertonic saline has been used to improve microvascular blood flow. It is dehydrating and can shrink swollen endothelial cells improving blood flow at the tissue level. This effect is seen at a lower dose and it is currently used at a dose of 1-2 ml/kg for such conditions as head trauma and organ dysfunction.

**The Replacement Phase**

The volume of fluid administered during the dehydration phase is based on an assessment of fluid needs for (1) returning the patient's status to normal (deficit volume), (2) replacing normal ongoing losses (maintenance volume), and (3) replacing continuing abnormal losses (continuing losses volume). Maintenance volumes are normal ongoing losses. Ongoing losses are divided into sensible and insensible losses. Sensible losses can be measured and are water losses in the urine and feces. Insensible losses are normal but are not easily quantitated. These water losses occur during panting or sweating. One-third of the maintenance volume is made up of the insensible volumes and two-thirds, sensible volumes. Traditionally, maintenance volumes have been estimated at about 66 ml/kg/day, or 30 ml/lb/day.
The Maintenance Phase

The last phase of fluid therapy is the maintenance phase. At this point the patient has received enough fluid to compensate for shock (if necessary) and has had a partial replacement of any deficit volume. Chronologically, this phase begins no sooner than 24 hours after fluids were begun. Objective signals that the patient is ready to be placed in the maintenance phase are an absence of clinical signs of shock or dehydration, and the body weight will have increased by at least the percentage of dehydration already corrected. During the maintenance phase, you will be providing both maintenance volumes and continuing losses volumes.
CIRCULATORY SHOCK AND FLUID THERAPY – PART II CARDIOGENIC AND THE COMPLICATED DISORDERS OF OXYGEN DISTRIBUTION
Timothy Hackett DVM MS Dipl. ACVECC

Introduction
Circulatory shock is divided into 3 major classifications; hypovolemic shock, cardiogenic shock or pump failure, and distributive shock. Though the mechanisms for each are distinctly different, each results in reduced oxygen delivery \( (\text{DO}_2) \) to tissues through low blood flow or uneven distribution of flow. In actual practice, each primary event can lead to a cascade of complex physiologic problems, neurohormonal compensations and cascades that activate various biochemical mediators and inflammatory responses integral to the shock syndromes. A single patient may have several pathologic processes simultaneously resulting in reduced perfusion of tissues. We have already discussed hypovolemic shock in a previous lecture.

Cardiogenic Shock
Cardiogenic shock occurs when the pumping function of the heart is severely impaired leading to circulatory failure. As with hypovolemic shock, the patient will be tachycardic, weak, oliguric, have cool extremities and weak pulses. The patient with cardiac failure may also have evidence of cardiac disease with a murmur, ascites, jugular venous distention, pulmonary edema or cardiac arrhythmias. The primary defect in oxygen delivery is a reduced cardiac output.

\[ \text{Cardiac Output (CO)} = \text{Heart rate} \times \text{Stroke volume} \]

Stroke Volume is determined by preload, afterload and contractility

Within limits, cardiac output increases as heart rate increases. Very high heart rates actually decrease cardiac output by impairing cardiac filling and subsequently stroke volume. Excessively fast heart rates may be the result of cardiac arrhythmias or physiologic responses to low volume. Specific antiarrhythmic therapy and correction of underlying causes of tachycardia should be used to normalize heart rate. Clinically significant bradyarrhythmias are less common but include sick sinus syndrome and third degree atrioventricular block. It is uncommon for these slow heart rates to require emergency treatment. Often these patients have compensated with increased stroke volume and can be referred for pacemaker treatment.

Stoke volume is dependent upon three determinants of cardiac function: Preload, afterload and contractility. With congestive heart failure, the pump is failing due to decreased contractility. The body attempts to compensate by increasing pre-load (sodium and fluid retention). Normally, the heart is able to pump all fluid presented to it through the Frank-Starling mechanism (increase stretch leading to increased contractility) so that by increasing pre-load, the heart will increase stroke volume. With failure however, the excess fluid cannot be moved and accumulates downstream of the failing ventricle. This results in pulmonary edema in the case of left-ventricular failure and ascites, pleural effusion and hepatic congestion in the case of right-ventricular failure.

Stoke volume (and cardiac output) can be maximized by recognizing and treating the primary defect. In the case of congestive failure, pre-load can be optimized by monitoring central venous pressure, administering diuretics like furosemide and venodilators such as nitroglycerine. With obstructive failure as is seen with pericardial effusion, removal of even a small amount of pericardial fluid will relieve the pressure on the right ventricle and allow more normal filling. Cardiac output can also be enhanced by decreasing afterload with calcium channel blockers or ACE inhibitors. These are especially useful in treating failure due to mitral insufficiency where contractility may be normal to increased but the cardiac output is going backwards into the left
atrium instead of to systemic circulation. In documented myocardial failure, contractility can be enhanced with positive inotropic drugs such as digoxin or dobutamine.

**Distributive Shock**

Distributive shock is probably the most challenging of the shock syndromes and one of the most difficult to reverse. The defect with distributive shock is an abnormal or systemic vasomotor response leading to peripheral vasodilation and a maldistribution of blood flow. There may also be increased vascular permeability. Both of which result in decreased perfusion of vital tissues. There can be components of the other forms of shock. Fluid loss into body cavities and interstitial spaces results in a relative hypovolemia. The release of inflammatory mediators as in septic shock can depress the myocardium resulting in a cardiogenic component. Therapy must be directed at the underlying systemic defect. In the case of sepsis, drainage and control of the infected focus. Because systemic inflammation resulting from sepsis and other inflammatory disease can affect oxygen delivery in so many different places, serial monitoring of many variables becomes necessary to treat the variety of problems an individual may face. The following table lists many of these important variables and optimal values for each. Interventions are also listed:

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<th>Physiologic Variable</th>
<th>Optimal Values</th>
<th>Intervention</th>
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<tr>
<td>Systemic arterial pressure</td>
<td>&gt;90 mmHg</td>
<td>Fluids, inotropes</td>
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<tr>
<td>Central venous pressure</td>
<td>&lt;3 cmH20 (5-10 cm H2O if loading)</td>
<td>Diuretics, venodilators</td>
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<tr>
<td>Urine output</td>
<td>&gt;1 ml/lb/hr</td>
<td>Diuretics, dopamine</td>
</tr>
<tr>
<td>Blood Glucose</td>
<td>&gt;3.9 mmol/L</td>
<td>Nutrition, dextrose</td>
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<tr>
<td>Packed Cell Volume</td>
<td>25-35%</td>
<td>Transfusion</td>
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<tr>
<td>Total Serum Solids</td>
<td>&gt;35 g/L, &lt; 50 g/L</td>
<td>Plasma, colloids</td>
</tr>
<tr>
<td>Albumin</td>
<td>&gt;10 g/L</td>
<td>Plasma</td>
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<tr>
<td>Arterial blood gasses</td>
<td>PaO2 &gt; 70 mmHg</td>
<td>Supplemental oxygen</td>
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<td></td>
<td>PaCO2 &lt; 35 mmHg</td>
<td>Ventilatory support</td>
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<tr>
<td></td>
<td>HCO3 &gt; 14, &lt; 24</td>
<td>Fluids, perfusion</td>
</tr>
<tr>
<td></td>
<td>pH &gt; 7.3, &lt; 7.5</td>
<td>Fluids, perfusion</td>
</tr>
<tr>
<td>Heart rate</td>
<td>&gt;70, &lt;150 BPM</td>
<td>Fluids, analgesics, anti-arrhythmics</td>
</tr>
<tr>
<td>Activated clotting time</td>
<td>90-120 seconds</td>
<td>Blood, plasma, heparin.</td>
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Circulatory Shock Treatment Summary

1) Primary goal: Improve oxygen delivery (DO$_2$)

\[
DO_2 \text{ (ml/min)} - \text{oxygen delivery} = \text{CaO}_2 \text{ (oxygen content) } \times \text{CO \ (cardiac output)}
\]

Improve oxygen content:
- Increase oxygen saturation (SaO$_2$)
  - Nasal oxygen, E-Collar oxygen tent, oxygen cage
- Increase hemoglobin concentration
  - Whole blood transfusion
  - Packed red blood cell transfusion
  - Oxyglobin infusion

Improve Cardiac Output
- Optimize heart rate (monitor pulse rate, quality, electrocardiogram)
  - Arrhythmias
    - Sinus tachycardia
      - Normalize fluids, control pain and anxiety
    - Supraventricular arrhythmias
      - Slow heart rate, fluids, beta blockers, digoxin
    - Ventricular tachyarrhythmias
      - Lidocaine
      - Procainamide
- Optimize stroke volume
  - Optimize preload (monitor central venous pressure)
    - Balance fluids, diuretics and venodilators
  - Optimize afterload (monitor toe web temperature, blood pressure)
    - ACE inhibitors, calcium channel blockers
  - Optimize contractility (echocardiogram, blood pressure)
    - Positive inotropes (pimobendan, dobutamine, dopamine)

2) Treat primary problem
- Sepsis/trauma
  - Debridement/drainage
  - Antibiotics
  - Antiinflammatory drugs
- Neoplasia
  - Surgery
  - Chemotherapy

3) Monitor for new problems and multiple organ failure
- DIC, renal or hepatic failure or pulmonary failure.
Introduction

Cardiac arrhythmias are a common finding in critically ill and emergency patients. If you’re not seeing arrhythmias in your critical patients it’s because they aren’t being monitored. Electrocardiograms should be available for your patients under anesthesia and during the post-operative period. Mounting these machines to the wall in surgery only deprives your critical hospitalized patients of a useful monitoring tool. The purpose of this brief talk is to cover the recognition and treatment (if any) for the most common arrhythmias we see in the emergency department and in our post-operative patients.

As with any monitor, ECG’s are only helpful if they provide meaningful information that may change a patient’s treatment plan. It is important to note that most rhythm disturbances are a sign that the heart and its conduction system are unhappy. Many times there are correctable reasons for this and specific antiarrhythmic drugs may not be indicated. Other times the rhythm is noted that is not affecting the patient clinically. Clinicians should always look at the patient to help guide the need for more specific therapy.

Too Slow? - Bradyarrhythmias

Less common than rapid heart rates, there are times when the heart is beating slowly. Unless a patient with a slow heart rate is clinical (syncope, exercise intolerance) many of the arrhythmias require no specific treatment. As with people, healthy athletic animals may have surprisingly slow normal heart rates. It is not uncommon to see a large breed dog resting quietly and pain free with a heart rate less than 60 beats per minute. These animals usually respond to stimuli with a jump in rate and require no treatment. Dogs (unlike humans) often have a sinus arrhythmia when resting. This is a regularly irregular sinus rhythm that changes rate in sync with respiration. Sinus bradycardia is often seen at rest or in conjunction with ocular, cervical, or gastrointestinal problems (common denominator is high vagal tone). If the patient responds to stimuli and is not having unexplained syncopal spells or a decrease in normal activity the slow heart rate requires no therapy. If high vagal tone is causing clinical problems, a vagolytic like atropine or glycopyrolate should be used (while treating the underlying problem).

There are 4 different types of atrioventricular (AV) block that can lead to slower heart rates. 1\textsuperscript{st} degree AV block is seen as a prolonged PR interval, is a common finding in dogs and usually requires no treatment. If a patient is receiving digoxin, this finding may indicate overdosage and blood levels should be checked. 2\textsuperscript{nd} degree AV block comes in 2 forms a Mobitz type I with a steadily widening PR interval until a p-wave occurs without a QRS complex and the more common Mobitz type II, a uniform PR interval with an occasional dropped beat (p-wave without the QRS complex). While responsive to atropine or glycopyrolate, 2\textsuperscript{nd} degree AV block is a very common finding in young and healthy dogs and usually requires no treatment. The final (and most serious) heart block is the 3\textsuperscript{rd} degree AV block. Also called complete heart block there is no signal making it through the AV node. The patient usually has a regular and normal rate of p-waves but the ventricular rate (and corresponding pulse rate) is much slower. Because no signal passes through the AV node, atropine or glycopyrolate are unlikely to increase the ventricular escape rate or pulse rate. This can be a big surprise when performing an ECG on a patient with bradycardia. It should be noted that most cases of 3\textsuperscript{rd} degree AV block do not require emergency therapy. Many times these animals have compensated to the slow rhythm with increased stroke volume. The definitive treatment is a cardiac pacemaker. If the patient is clinical (syncope, collapse, weakness) the ventricular escape rate can be increased with a \(\beta\)-agonist such as isoproterenol.
Atrial standstill most commonly occurs in patients with hyperkalemia. Blocked tomcats and dogs with hypoadrenocorticism (Addison’s disease) are the most common reasons for seeing bradycardia without p-waves. Treatment is directed at protecting the heart from the effects of the high potassium (with calcium gluconate) and correcting the high circulating potassium with IV bicarbonate, dextrose, or a combination of regular insulin and dextrose.

Too Fast? - Tachyarrhythmias

Since cardiac output (and with it oxygen delivery) increases with an increasing heart rate, tachycardia is a common finding in stressed and shocky patients. However, there is a limit to how high a heart rate can go and still be helpful. As heart rates climb, the time for ventricular filling shortens. Eventually, there isn’t enough time for the ventricle to fill and stroke volume suffers. This point varies with patient but is probably somewhere above 150-200 beats per minute for most dogs and cats.

As with slow arrhythmias, underlying cause and clinical impact on the patient should be primary considerations before reaching for specific Antiarrhythmics.

Supraventricular tachycardias (SVT) or narrow-complex tachycardias include sinus tachycardia, atrial fibrillation, and re-entrant or junctional tachycardia. Sinus tachycardia is the natural response to stress and shock. Patient’s volume status, pain, and anxiety should be closely evaluated. Most case of sinus tachycardia respond to titrated intravenous fluids (the type determined by the patients packed cell volume/ total protein, and electrolytes), appropriate control of pain and anxiety. Atrial fibrillation is the irregularly irregular rhythm seen most often with cases of cardiomyopathy. This has a very characteristic sound of Early cases of atrial fibrillation can be electrically converted to the more functional sinus rhythm. This requires specialized defibrillation equipment and general anesthesia. Most case of atrial fibrillation are treated by slowing the rate with negative chronotropic drugs like digoxin, calcium channel blockers, or β-blockers. Diseased heart muscle can provide an alternate electrical pathway resulting in a paroxysmal Supraventricular tachycardia. These fast rhythms are not uncommon and can result in very fast, regular, narrow-complex rhythms that can start and stop suddenly.
They can result in syncope and usually require treatment to alter (slow) conduction. As for atrial fibrillation a negative chronotrope is chosen based on the patients underlying cardiac disease.

**Ventricular ectopy and ventricular tachycardia** can often be diagnosed without an electrocardiogram. One should get in the habit of palpating pulses while listening to the heart. Early heart sounds without a corresponding pulse are typical of the dropped beats of premature ventricular contractions. A run of ventricular beats and fast than normal heart rates is termed ventricular tachycardia. Because the beats are initiated in the ventricular muscle and not within the conduction system the complexes are much wider than normal. The one look-alike rhythm that should be considered when contemplating treatment is the **bundle-branch block**. With right and left bundle branch blocks there is a p-wave for every QRS complex. The rhythm is Supraventricular although the wide complexes (caused by the delay in the conduction pathway) make it look ventricular.

The ventricular arrhythmia is likely a sign that the heart muscle isn’t happy. Much the way azotemia indicates renal malaise or a high bilirubin suggest hepatic disease, ventricular arrhythmias indicate cardiac dissatisfaction. Treatment of ventricular tachycardia is directed at several fronts. Specific antiarrhythmics and all possible underlying causes. In cases of clinically significant v-tach (weak pulses, evidence of poor perfusion, R on T pacing) a ventricular antiarrhythmic such as lidocaine or procainamide are usually started. Note: Lidocaine is initially dosed as a bolus, but then followed up by continuous infusion. Patients with v-tach and ventricular ectopic activity should also be evaluated for ineffective oxygen delivery (shock! Check volume status, and oxygenation). Because hypokalemia and hypomagnesemia are associated with ventricular arrhythmias, these electrolytes should be evaluated and supplemented as necessary. Stress and anxiety can increase circulating catecholamines. Since catecholamines lower the cardiac threshold for ventricular arrhythmias the careful use of appropriate analgesic and sedative drugs can go a long way toward resolving the arrhythmias.
Cardiac Arrest

During cardiopulmonary arrest (CPA) it will be important to quickly obtain an electrocardiogram after beginning basic life support (chest compressions and rescue breathing). Advanced life support involves the use of electrical defibrillation and cardioactive drugs the order of which will depend on the cardiac rhythm present. Without an ECG, specific therapy cannot be initiated.

The three most common arrhythmias seen during cardiac arrest are **ventricular asystole**, **ventricular fibrillation**, and **electrical-mechanical dissociation** (also called **pulseless electrical activity**... EMD or PEA for short). For all three, good basic life support in the form of cardiopulmonary resuscitation is necessary to restore oxygen delivery to the myocardium. Ventricular asystole should be treated with atropine followed by epinephrine. Ventricular fibrillation is common in people and dogs, but normally cat’s hearts are not large enough to support the chaotic activity. If you see ventricular fibrillation in cats think about underlying cardiomyopathy. The treatment is rapid electrical defibrillation. Underlying problems such as hypomagnesemia may complicate this rhythm making defibrillation more difficult. EMD should be managed with good basic life support, atropine, and epinephrine. Consider naloxone if patients have recently received narcotic analgesics.

**Intervals and baselines**

As an emergency clinician I don’t have the patience to measure intervals. I primarily use the electrocardiogram to monitor heart rate and watch for clinically important arrhythmias. However there are a couple of intervals that I do find useful in the emergency room. One is the **prolonged QT interval**. This is seen with hypothermia and hypocalcemia. Clinically I’ve found this useful in the triage of ethylene glycol intoxication with its profound hypocalcemia. Seeing
an abnormally long QT interval gets me looking at body temperature, and if normal blood calcium.

I think it’s also good to look at the ST segment when managing the shock patient. ST segment depression (the segment below baseline) is a common finding in human myocardial infarction (MI or heart attack) patients. It is a sign of myocardial ischemia and a powerful indication that the heart isn’t getting enough oxygen. Treatment should be geared toward improved oxygen delivery (fluids, blood transfusion, oxygen or ventilator therapy).
VASCULAR ACCESS TECHNIQUES
Tim B. Hackett DVM MS DACVECC

Introduction
Intravenous catheterization is one of the most important skills to master in veterinary emergency and critical care. Veterinarians and veterinary technicians that can reliably gain vascular access in the sickest patients are extremely valuable members of the patient care team. Often, the patients most in need of vascular access are the hardest to catheterize. In these situations, intraosseous catheterization is an option for quick access to the vascular compartment. The landmarks are easy to identify regardless of blood pressure or degree of hydration. The purpose of this seminar is to provide a review of available catheters, indications for their use and the techniques and tricks to placing each.

Catheterization Basics
Intravenous catheters are invasive devices and their use must be managed with potential complications in mind. These foreign materials provide direct access to the bloodstream for fluids, intravenous medications and repeated blood sampling. Direct access can also allow infectious agents a means of bypassing defenses to colonize the host. These “foreign” materials can also cause a variety of inflammatory complications from mild vasculitis to serious thrombosis and vascular occlusion.

A surgical preparation of the skin over the catheter site, and sterile handling of catheter and connection tubing will minimize infectious complications. Hair should be clipped within 2 to 4 cm of the site. The area should then be cleaned and disinfected with surgical scrub for 3 minutes. While it is not necessary to wear sterile gloves when placing intravenous catheters, the person handling the catheter should have clean hands. We recommend wearing disposable vinyl or latex gloves when scrubbing the skin, then drying or changing the gloves when handling catheters and tubing.

After the catheter is in place, the site should be covered with an antibiotic ointment and clean dressing. The catheter needs to be held securely, however think about how you will remove or change connections when you are applying the bandage. Incorporating “T-port” connectors in the final bandage will take the strain of movement off the catheter. These connections are also easily replaced without having to replace the entire dressing.

Catheters should be checked every 24 to 48 hours. The bandage should be removed, the catheter site examined and the vessel palpated. The catheter should be removed and a new one placed in another site if there is any evidence of inflammation or thrombosis.

Catheter selection
Modern intravenous catheters are made of a combination of material. These materials are designed to minimize inflammation of the vessel wall (phlebitis) and prevent blood clot formation (thrombosis). Some newer catheters even have special coatings to prevent bacterial colonization and infection. Plastic tubing that is not specifically designed for intravascular use should be avoided as these complications would be hard to explain given the economy, variety and availability of intravascular products.

Peripheral Catheters
Short, polyvinyl chloride catheters are designed for use in small, peripheral arteries and veins. These catheters typically vary in size from 18 to 22 gauge and are 1-2 inches in length. The easiest to place, the catheters should be used for short-term procedures, in patients requiring several “lines” at one time and in those where central catheters may be contraindicated (see
Central catheters). The main advantage to peripheral catheters is the ease of placement. Peripheral vessels are easy to visualize, prep and catheterize. Peripheral vessels are easier to bandage and a better choices when it is desirable to stay away from the head as with anesthetized or seizing patients. The biggest disadvantage to peripheral catheters is the changes in fluid flow with position of the limb. Tightly flexed limbs can occlude venous drainage and impede the continuous flow of intravenous fluid. Although easy to place, these catheters may be relied up too much in the critical care setting. When vascular access is going to be needed for several days, a larger, central catheter should be considered.

An alternative to a large jugular catheter is the use of a long, teflon catheter in the lateral saphenous vein in dogs and the medial saphenous vein in cats. These catheters provide many of the advantages of a central catheter for blood sampling, and uninterrupted flow. They also may be easier to place in patients in which you want to avoid the head or neck. Rear leg catheters are easily soiled and should be avoided in patients with urinary incontinence or diarrhea.

**Central intravenous catheterization**

Central venous catheterization is essential in many critically ill patients. A large-bore, teflon, jugular venous catheter can be maintained for days. Incorporated into a bandage around the animal’s neck, these catheters tend to stay dryer, cleaner and can remain in longer than peripheral catheters. Central venous catheters are useful in obtaining a central venous pressure (CVP) which provides important information about fluid loading and the hearts ability to pump the fluid presented to it. Large central catheters can also be useful for repeated blood sampling. By drawing 3 cc of blood back into a syringe with 0.5 cc-heparinized saline, any volume of blood can then be sampled. The heparinized blood can then be returned to the patient and the line flushed. This “Three-syringe” technique will minimize patient discomfort and iatrogenic blood loss. The most common central venous catheters are through-the-needle catheters. The largest of these is 16 gauge. Larger central catheters include triple lumen catheters and Swan-Ganz catheters with multiple injection ports and balloon tips to allow placement in the pulmonary artery. These larger catheters are inserted into the jugular vein over guide wires threaded through a 14-18-gauge needle. Once in place these advanced catheters facilitate monitoring of such variables as mixed venous oxygen content, central venous pressure, right atrial, right ventricular and pulmonary artery pressure as well as cardiac output and pulmonary capillary wedge pressure.

Central venous catheters should be avoided in patients with bleeding disorders, seizures or hypercoagulable states (autoimmune hemolytic anemia, hyperadrenocorticism, DIC, protein losing nephropathy).

**Intraosseous catheterizations**

Intraosseous catheters are for short-term use. They are an excellent means of delivering fluids, blood products and intravenous medications. Unlike other intravascular catheters, this procedure is easily mastered using fresh cadavers. The downside is that there are afferent pain fibers in the periosteum and endosteum making these catheters less comfortable than their intravenous counterparts. It is also difficult to impossible to sample blood from these catheters. These two issues make the placement of an intravenous catheter desirable in patients requiring ongoing intravenous therapies. The good news is that the improved vascular volume and blood pressure provides by intraosseous resuscitation make intravenous catheterization easier.

Intraosseous catheters are invasive devices and their use must be managed with potential complications in mind. These foreign materials provide direct access to the central bone cavity bloodstream for fluids, intravenous medications. Direct access can also allow infectious agents a means of bypassing defenses to colonize the host. These catheters in the proximal central spaces
of long bones can dislodge bone and fat into the bloodstream. This is especially true if fluids are delivered through these catheters at high pressure.

A surgical preparation of the skin over the catheter site, and sterile handling of catheter and connection tubing will minimize infectious complications. Hair should be clipped within 2 to 4 cm of the site. The area should then be cleaned and disinfected with surgical scrub. While it is not necessary to wear sterile gloves when placing intraosseous catheters, the person handling the catheter should have clean hands.

The locations used for intraosseous fluids are difficult to bandage. Since these catheters are for short-term use, we often just attach a “t-port” to the catheter and suture the tubing to the skin. This takes tension off the catheter to prevent prematurely pulling the catheter when the patient moves. The catheter needs to be held securely, however think about how you will remove or change connections when you are suturing tubing.

**Intraosseous choices**

**Hypodermic needles** (20-22 gauge) can be used in the smallest patients. In a bird, small mammal, puppy or kitten the needle can be worked through the soft, thin cortex. The disadvantage to this inexpensive choice is that bone material may block the lumen.

To avoid the potential for obstruction, 18-20 gauge disposable **spinal needles** are an excellent choice. With an internal stylet, these needles can enter the bone without plugging. These are stocked in our emergency room for this purpose. The needles can be inserted in all the above cases, but because they are fairly weak, not designed to cut through bone, they are not practical choices for dogs and cats with mature, hard bone and thick cortices.

If a bone marrow biopsy needle or intraosseous system is not available, a **Steinman pin** can be used to create a hole in the bone that can provide catheter access. Pin size should be checked with the outer diameter of the over-the-needle IV catheter to be used. With close attention to landmarks placement through the pre-drilled hole is easier than it sounds.

The best way to get through the cortex of mature bone is with a styleted **bone biopsy needle**. The classic choice is a metal Rosenthal needle. We tend to use 18 or 20 gauge sizes as intraosseous catheters. These needles are robust enough to cut through bone. They are reusable options built to sample bone marrow. They can be sharpened, sterilized and reused. A number of purpose made intraosseous catheters have taken this design and incorporated larger handles to make handling and insertion easier.

**Vidacare’s EZ-1O, Pyng’s First Responder, and WaisMed's BIG (Bone Injection Gun)** are elegant mechanical systems that have been developed and have quickly become commonplace with human first responders and trauma centers. Though more expensive than bone marrow needles, these purpose made devices can provide vascular access faster than any catheter system. The EZ-IO is a purpose made electric drill and low profile needles. This system is easily used in veterinary patients. The First Responder is a hand-powered system specifically designed to access the manubrium in people. The Bone Injection Gun is a spring-loaded system that has been used at other access points.

**Intraosseous access points**

The intraosseous catheter can be placed in medullary space of the femur (dogs and cats), the humerus (dogs and cats), the tibia (ferrets, pocket pets) or the ulna (birds). Birds use the spaces within the humerus and femur as part of the respiratory system so these bones must not be used for fluid replacement.
With advances in intraosseous access technology it has become the method of choice for vascular access in hard-to-catheterize patients. Many human first responders are using the intraosseous route for their first line vascular access in adult and pediatric critical illness. Veterinary patients are even smaller and more difficult to catheterize making intraosseous access a necessary skill for the emergency/critical care team.
ANESTHESIA IN THE CRITICAL PATIENT
Tim Hackett, DVM MS, Dip. ACVECC

Veterinarians needing to induce anesthesia in critical patients must recognize the serious risks they are taking with their patients. Since most anesthetic agents have some negative effects, choices need to be made to be sure that the anesthetic event doesn’t lead to more serious problems. Many times it is necessary to secure an airway or perform extensive surgical procedures in order to stabilize emergent patients. With careful monitoring, support of ventilation and blood pressure, and use of agents with cardiopulmonary side effects these patients can be induced safely.

The anesthetic protocol should be modified to the needs of the patient. Determine anesthetic considerations prior to anesthetizing the patient. For example, cardiovascular compromise, head trauma, renal failure, or blood loss could affect the outcome. By watching for expected complications and reacting quickly the veterinarian and nursing staff may save the life of the patient. The ideal anesthetic agent would be a rapid acting one, easily reversed, provide analgesia, and have minimal cardiovascular or respiratory depression. There should also be no residual effects or toxic side effects. Unfortunately, there is no one agent that meets all these criteria.

Premedication
Anesthetic premedication with sedative/analgesic drugs provides stress free induction, and lower dosages of induction agents. There is not a perfect single anesthetic drug. Instead, we strive to use smaller amounts of multiple drugs. This helps avoid the negative effect of any single drug while achieving our goals of sedation, analgesia, amnesia, and anesthesia.

Alpha-agonists produce sedation, and muscle relaxation. Opioids produce analgesia and euphoria. In combination (neuroleptanalgesia) alpha-agonists tranquilizers and opioids produce profound calming, analgesia, and a light plane of anesthesia. Premedication sedatives and analgesics are commonly combined with an anticholinergic (atropine, glycopyrrolate, scopolamine) to limit airway secretions and prevent bradycardia. In the emergency setting, anticholinergic drugs may not be necessary. If the patient is tachycardic, it may be more wise to have the dose of anticholinergics standing by while carefully monitoring heart rate.

The clinician should also consider the plan for post-operative analgesia when formulating a plan for premedication. Narcotic agonists should be used if narcotics will be used for post-operative pain control. Agonist-antagonist drugs may make postoperative narcotics less effective.

Phenothiazines
Acepromazine (and promazine), have a calming effect by depressing the reticular activating system of the CNS, suppressing the sympathetic nervous system. Their important effect is calming stressed patients and reducing anxiety. It is important to note that they have little to no analgesia when used alone. Side effects are noteworthy and include alpha-adrenergic receptor mediated hypotension, a lowering of the seizure threshold, bradycardia, and reduced respiratory rate. These effects can be minimized by using low doses (generally we do not exceed 2 mg/dog) designed to alleviate anxiety without undesirable cardiovascular and pulmonary side effects.

Benzodiazepines
Diazepam, midazolam, and zolazepam are considered minor tranquilizers. They are centrally acting muscle relaxants exerting their effects by enhancing the activity of CNS inhibitory neurotransmitters, depressing the limbic system. They cause minimal CNS depression
and are useful for their anticonvulsant effect. Their effects can be antagonized by flumazenil. At preanesthetic doses, there have minimal cardiopulmonary effects. Bradycardia and mild hypotension may be seen with rapid intravenous administration.

**Alpha-2 Agonists**

Xylazine, detomidine, medetomidine, and romifidine produce CNS depression by stimulating presynaptic alpha2-adrenoceptors. By decreasing norepinephrine release throughout the body, they result in CNS depression and a pronounced, phenothiazine-like sleep state. These drugs have more pronounced cardiopulmonary side effects including bradycardia, conduction block, increased cardiac sensitivity to catecholamine-induced arrhythmias, reduced cardiac output, increased peripheral vasoconstriction, and respiratory depression.

**Narcotic Agonists**

Opioids (Morphine, oxymorphone, fentanyl) act by reversibly binding with of receptors in the brain to produce analgesia, sedation, euphoria, dysphoria, and even excitement. The different formulations have different potencies and different effects at individual receptors. These agents have some profound cardiopulmonary effects with are completely reversible with the narcotic antagonist naloxone. These effects include bradycardia, respiratory depression, salivation, nausea, and gastrointestinal hypermotility.

**Partial Narcotic Agonists and Agonist-Antagonists**

Partial narcotic agonists (buprenorphine) and agonists-antagonists (butorphanol) compete for narcotic binding sites but exert less analgesia and CNS depression. They can be useful to partially antagonize cardiopulmonary side effects of pure narcotic agonists without completely reversing analgesia.

**Combination premedication.**

Neuroleptanalgesia is a CNS depression (sedation) and analgesia produced by using sedative/tranquilizers with more potent analgesics. By providing pre-induction sedation, the dose of induction drugs and maintenance anesthesia can be minimized. Combinations include the use of acepromazine with morphine, fentanyl, meperidine, oxymorphone or butorphanol. Diazepam can also be used with opioid agonists for similar effects.

**Induction**

Most anesthetic agents depress cardiopulmonary function and cause dose dependent hypotension. It is imperative to use these agents with extreme caution in patients with cardiac, respiratory or other major organ system dysfunction. The ideal agent would be fast acting, short acting, reversible and having minimal effects on tissue oxygen delivery. Unfortunately this ideal agent does not exist. Instead we have several agents with some of these properties. The following section will review the most common agents, side effects and dosing schedules used to prevent serious complications.

**Diprivan (propofol)**

Propofol is rapid acting, ultra-short, non-barbiturate induction agent. It produces profound sedation-hypnosis similar to the barbiturates by interacting with CNS GABA receptors with minimal analgesia. When given by rapid bolus infusion, it can cause profound hypotension and apnea (like fast-acting barbiturates). We have found that by giving propofol by a slow bolus with intravenous fluids, not only can we avoid hypotension and apnea, but also we achieve an induction plane of anesthesia with less drug. Propofol is rapidly cleared from the body by hepatic
and extrahepatic metabolism (much faster than barbiturates), and is non cumulative. Due to the rapid onset of action, unique metabolism, and its titratability, propofol is an invaluable agent in the critically ill patient.

**Ultra-short acting barbiturates**

Barbiturates like Pentobarbital sodium (Nembutal) and Thiopental sodium (Pentothal) have been in use for many years. They provide a quick induction and were favored for many years. The induction dose can be minimized with the use of neuroleptanalgesia. The disadvantages were related to a longer duration of action and prolonged recovery times. Additionally they cause a dose-dependent hypotension, ventricular arrhythmias and often resulted in apnea upon induction. These drugs are metabolized by the liver and may persist in obese patient because of their high lipid solubility.

**Ketamine**

Ketamine, a dissociative anesthetic, causes profound analgesia, and amnesia. Muscle tone is maintained and often increases. The muscle tone can be controlled with the simultaneous use of sedative/tranquilizers. Ketamine is often used with benzodiazepines. Telozol uses a dissociative (tiletamine) combined with a tranquilizer (zolazepam) in a 1:1 combination used in aggressive animals (IM administration). Cardiopulmonary side effects include increased heart rate and blood pressure. Effects on cardiac contractility can be profound and may cause acute heart failure and pulmonary edema. Cleared by the liver and kidney, ketamine should be avoided in patients with hepatic or renal disease.

**Etomidate**

A rapid acting non-barbiturate intravenous anesthetic, etomidate produces anesthesia without significant analgesia by interacting with CNS GABA receptors, and depressing the reticular activating system within the brainstem. Cardiopulmonary effects include transient apnea, and mild reductions in cardiac contractility.

**Neuromuscular blocking agents**

Having neither anesthetic nor analgesic properties, neuromuscular agents have a place in emergency control of the critically ill. Patients may be so fragile that control of airway may be needed while the patient is too hypotensive or hypoxemic to consider an anesthetic agent. Neuromuscular blocking drugs interfere with neuromuscular transmission providing skeletal muscle relaxation. With neuromuscular blockade, breathing ceases, so controlled ventilation must be provided. Succinyl choline or atracurium may provide the clinician a few minutes of patient cooperation to intubate, ventilate, oxygenate and start fluid therapy. It must be remembered however that NEURMUSCULAR BLOCKING DRUGS DO NOT PROVIDE ANESTHESIA OR ANALGESIA. Patients controlled with neuromuscular blocking agents can be completely aware and painful and it is vital to provide sedative and analgesic drugs as soon as the patient’s condition will allow.

**Monitoring**

**Body temperature** can drop quickly, especially if the abdomen or thorax are open. Large lavage volumes of saline and small patient size can also predispose anesthetized animals to hypothermia. Active warming is important, as is the use of re-breathing systems, warmed IV fluids, and warmed saline to flush abdomen.

**Heart Rate and Rhythm** can be monitored by the use of continuous Electrocardiogram or with an esophageal stethoscope. Placement of a Doppler flow-detector for
blood pressure will also allow the veterinarian to hear heart rate and rhythm as the blood flows past the crystal.

**Blood Pressure** can be measured directly or indirectly. Assessment of pulse quality can be made by periodic palpation of the dorsal pedal or femoral pulse.

**Oxygenation/Ventilation** should be assessed. This is done easily with pulse oximetry and end-tidal carbon dioxide (ET-CO2) monitors. These monitors are excellent for continuous assessment. The ideal standard for oxygenation and ventilation is arterial blood gas measurements. These should be performed periodically to verify the readings of oxygen saturation and ET-CO2.

**Renal function** and adequacy of fluid therapy is easily assessed by monitoring urine production. Anesthetized and critically ill patients receiving intravenous fluids should produce at least 1-2 ml/kg/hour. An indwelling urinary catheter will help manage bladder size while providing an objective monitoring tool to assess renal function. The staff should also pay attention to respiratory tract fluid losses and surgical blood loss (1 ml of blood weighs 1.3 grams).

**The Underlying diseases** should receive continuous monitoring while under anesthesia. Special consideration should be given to any underlying renal disease, liver disease, coagulopathy, endocrinopathies, heart disease, and respiratory problems.
<table>
<thead>
<tr>
<th>Drugs</th>
<th>Dosage</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Anticholinergics</td>
<td></td>
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<tr>
<td>Atropine</td>
<td>0.04 mg/kg IV, SQ</td>
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<tr>
<td>Glycopyrrolate (dogs)</td>
<td>11 mcg/kg IV, IM,</td>
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<td>Tranquilizers</td>
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<tr>
<td>Acepromazine</td>
<td>0.05-0.25 mg/kg IV, SQ</td>
<td>Acepromazine dose should not exceed 3 mg dogs) and</td>
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<tr>
<td>Diazepam</td>
<td>0.75-1 mg/kg IV, IM</td>
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<tr>
<td>Midazolam</td>
<td>0.1-0.5 mg/kg IV, IM, SQ</td>
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<tr>
<td>Narcotic agonists</td>
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<tr>
<td>Morphine</td>
<td>0.2-2 mg/kg IM, SQ</td>
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<tr>
<td>Fentanyl</td>
<td>2-4 mcg/kg IV, SQ</td>
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<tr>
<td>Oxymorphone</td>
<td>0.1 to 0.2 mg/kg IV, IM, SQ</td>
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<tr>
<td>Cat doses of narcotics are</td>
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<tr>
<td>usually half listed dog doses</td>
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<td>If dysphoria and excitement, reduce dose by half. Or switch narcotic.</td>
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<td>Agonist-Antagonists</td>
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<tr>
<td>Butorphanol</td>
<td>0.1 to 0.5 mg/kg IM, SQ</td>
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<td>Partial Agonists</td>
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<tr>
<td>Buprenorphine</td>
<td>0.007-.02 mg/kg IM, SQ</td>
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<tr>
<td>Narcotic Antagonists</td>
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<tr>
<td>Naloxone</td>
<td>15 mcg/kg IV, IM, SQ</td>
<td></td>
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<tr>
<td>Cat dose is 1/2 dog dose</td>
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<tr>
<td>Less analgesia than pure agonists</td>
<td></td>
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<tr>
<td>Less analgesia than pure agonists</td>
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<tr>
<td>Antidote for narcotic overdose or to reverse cardiopulmonary effects of narcotics</td>
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<tr>
<td>Barbiturate Anesthetics</td>
<td></td>
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<tr>
<td>Thiopental sodium</td>
<td>18 mg/kg IV to effect</td>
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<tr>
<td>Propofol</td>
<td>6 mg/kg IV to effect</td>
<td></td>
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<tr>
<td>Etomidate</td>
<td>1 mg/kg IV</td>
<td></td>
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<tr>
<td>Neuromuscular blockade</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>0.2 to 1 mg/kg</td>
<td>Succinylcholine is faster acting with more side effects (bradycardia, pyrexia)</td>
</tr>
<tr>
<td>Atracurium</td>
<td>0.1 to 0.4 mg/kg IV</td>
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SMALL ANIMAL TRAUMA  
Tim B. Hackett DVM MS DACVECC

Trauma is a common small animal emergency. Panicking owners may go to the first veterinary hospital they find. With a basic understanding of the systemic complications of trauma, and rationale treatment, patients that can be saved can be managed in most veterinary hospitals.

Each trauma patient should be evaluated in an orderly and systematic manner. Injuries that interfere with vital physiological functions should receive the highest priority. These are injuries that involve the respiratory system, cardiovascular system, or neurological system. Serious injuries that are not immediately life threatening include: fractures, luxations, and intra-abdominal injuries (ruptured spleen, liver or damage to the urological system). Minor injuries may merely require observation, monitoring, and serial evaluations to assure they do not slip to a more serious status.

**Initial Assessment**

The purpose for the initial assessment of the trauma patient is to identify life-threatening physiological injuries. Whenever a problem is identified immediate therapy is begun. This "primary survey" follows the ABC (and D’s) of triage and resuscitation:

- **Airway**--Is the patient having difficulty breathing? Are there mandibular injuries that are interfering with the airway? Has a penetrating wound disrupted the larynx or trachea? Obstruction of the upper airway typically results in a slow, deep (obstructive) breathing pattern.

- **Breathing**--Is the patient dyspnéic? What is the color of the mucous membranes? Is there evidence of thoracic penetration or is there a flail chest? Pulmonary contusions, pneumothorax, diaphragmatic hernia, and broken ribs can all result in a rapid, shallow (restrictive) breathing pattern.

- **Circulation**--Is there evidence of hemorrhage? Is the hemorrhage arterial or venous? How large is the swelling associated with the extremity fracture? Are the mucous membranes pale and tacky? Are the femoral pulses weak and rapid? Are the extremities cold? Is the abdomen distended?

- **Disability**--Is there evidence of neurological injury? What is the posture of the animal? Is the animal bright, alert and responsive? Does the animal respond to painful stimuli? Are the pupils dilated, constricted, of equal size, and responsive to light? Is there an extremity fracture that might threaten a peripheral nerve?

Management of the life-threatening problems identified during the primary survey is continued as shock therapy begins. The secondary survey identifies all other problems related to the trauma. The entire animal's body is examined again from head to toe. Necessary diagnostic samples are collected and submitted. Only when the patient is stable are indicated radiographs taken. In-depth management of the patient's less life-threatening injuries is undertaken in the definitive care phase. The fractures are stabilized, and careful inspection for "hidden" injuries is begun.

After arterial hemorrhage, respiratory function represents the highest priority in trauma. These injuries require immediate recognition and treatment. As aggressive intravenous fluid therapy can make some of these injuries worse, it is important to assume some degree of thoracic injury in all trauma patients. In one study, thoracic injuries were present in 57.7% of the dogs presented for treatment of orthopedic injuries. Pulmonary contusions, pneumothorax, and fractured ribs were most commonly observed.
Pulmonary Contusions

Lung contusion is the most common acute pulmonary complication of blunt chest trauma. Such a contusion may occur under the site of a flail chest or independent of obvious external injury. A large bruise in a very bad place, the contused alveoli fill with blood, and fluid resulting in atelectasis. Hypoxemia will result from pulmonary shunt as blood flows through these non-ventilated portions of lung. With time, pulmonary contusions appear radiographically as a diffuse alveolar pattern. The location varies with the injury. It is important to note that contusions may not be evident on radiographs for several hours after the injury.

Complicating trauma management is the evidence that these syndromes of respiratory insufficiency may be iatrogenic. The use of large volumes of rapidly administered crystalloid solutions can exacerbate the hypoxemia associated with the contusions. Maintaining plasma colloid oncotic pressure with the use of plasma or other colloid solutions may lessen the occurrence of respiratory insufficiency by preventing water loss into the injured lung. We use conservative fluid replacement in trauma patients with pulmonary contusions. Using a combination of crystalloid fluids (22-44 ml/kg, 1/4 to 1/2 of a typical shock volume) and colloid solutions (plasma, whole blood, Oxyglobin®, or hetastarch) we strive to maintain a minimally acceptable blood pressure (mean pressure of 60 mmHg) while avoiding iatrogenic pulmonary fluid overload. Patients with severe contusions may present with or develop hemoptysis. Blood from the mouth, agitation and respiratory distress are all indications that pulmonary parenchymal hemorrhage is ongoing and aggressive treatment is necessary. These patients are quickly restrained (fast acting anesthetic or a paralytic) and intubated. Ventilating with 100% oxygen and 5-10 cm H₂O positive end-expiratory ventilation will help keep remaining alveoli open, and open atelectic lung units. Patient tidal volume should be monitored closely as positive pressure ventilation and damaged lungs can lead to a tension pneumothorax.

Pneumothorax

Simple pneumothorax occurs when gas accumulates in the pleural space but pleural pressure does not significantly exceed atmospheric pressure. Gas can enter the space either from outside the chest wall, as occurs with bite wounds, sharp objects, or weapons, or via the lung through a tear in the lung parenchyma. Small amounts of gas cause pleural pressure to increase slightly, but it remains sub atmospheric during inspiration because it is in equilibrium with the negative alveolar pressure. Although pleural and alveolar pressures become positive during forced expiration, slight separation of the pleural spaces does not compromise ventilation. If the pneumothorax is small and the pleural leak seals itself, the gas will be absorbed as a result of partial pressure differences between gas in the pleural space and in the blood. Tension pneumothorax is characterized by a progressive increase in pleural pressure sufficient to impair circulation. This occurs as gas enters the pleural space and remains there during expiration because tissue or fluid occludes the pulmonary parenchyma. While tension pneumothorax can occur during spontaneous negative pressure inspiration, it is more likely with intubated patients receiving positive pressure ventilation. The accumulating gas not only collapses the lungs but also interferes with venous return to the right atrium. Thoracocentesis is preferred in the initial evaluation of thoracic injury. With a 20-gauge needle attached to an intravenous extension set, 3-way stopcock, and 60 ml syringe, one will aspirate air, fluid, or both. It is advisable to aspirate from both right and left sides of the thorax.

Fractured Ribs

Rib fractures are painful and limit diaphragmatic and chest wall motion. Failure to adequately expand the lungs results in atelectasis of the underlying lung and hypoxemia. Flail chest
occurs when three or more ribs, or the junction of ribs and the sternum, are each fractured at two points. This results in paradoxical inward movement of the flail segment during inspiration when the rest of the thoracic cage expands. Because the hypoxemia associated with flail chest results from atelectasis due to pain and contusions of the lung underlying the flail segment, therapy is aimed at relieving pain through analgesics and local blocks, supplemental oxygen, and supportive measures while the contused lung heals.

**Cardiovascular system trauma**

It is important to assess not only the vascular system and blood volume but also the heart. Contusions to the heart occur with blunt chest trauma in dogs and may cause cardiac dysrhythmias. In many cases, the dysrhythmias may be delayed as the stress and pain can lead to an "overdrive suppression" of ectopic foci. As the pain and shock resolve, sinus tachycardia subsides, and the ectopic foci discharge at a rate greater than the sino-atrial node leading to dysrhythmias. Therapy is generally directed at the treatment of the cause of the dysrhythmia. Treatment of shock, hypoxemia, electrolyte imbalances, pain, and anxiety may be all that is necessary.

**Intracranial Injuries**

Normal pupillary function implies that the midbrain and third cranial nerve are intact. Midbrain damage can produce midposition and unreactive pupils. Dilated unreactive pupils that develop from miotic pupils imply brain stem lesions and a grave prognosis. Decerebrate rigidity is characterized by quadrilateral rigidity and opisthotonos. Treatment of the brain trauma patient is supportive. In order to preserve brain function, and prevent ongoing neuronal damage, patients are given supplemental oxygen, maintenance intravenous fluids to optimize perfusion and oxygen delivery. Many of our head trauma patients receive hypertonic (7%) saline at 1-2 ml/kg. Hypertonic saline can decreased endothelial cell swelling and improve microvascular blood flow. It is used more often than mannitol (0.5-1 gm/kg IV) that is used with caution. Theoretically, mannitol could exacerbate bleeding in patients with intracranial hemorrhage, and should not be used if there is evidence of focal disease. Anisocoria, and strabismus are suggestive of focal bleeding. Mannitol is reserved for comatose patients with bilaterally symmetrical pupils or patients with deteriorating neurologic signs. Corticosteroids may actually increase cytologic damage and are no longer part of our treatment protocol.

**Spinal Cord Injury.**

In assessing a patient with spinal cord injuries, one should look at the motor, sensory, and autonomic responses associated with the various levels of the cord. Generally, lesions of the cervical spinal cord produce tetraplegia as their principal symptom. When the lesion is above the C5 cord segment, hyperreflexia is exhibited. As the cord segments of the brachial plexus becomes involved, lower motor neuron lesions are present. It is important to assess for superficial and deep pain sensation in the forelimbs. Additionally, the cervical cord injury patient is prone to apnea and must be closely monitored.

Lesions of the T2 - L3 cord segments will produce the Shiff-Scherington motor response with forelimb extensor rigidity and flaccid paralysis of the rear limbs. For prognosis, the ability of the patient to perceive superficial and/or deep pain is important. The inability to perceive pain is associated with a poor prognosis and the need for aggressive diagnostics and therapy. Therapy in spinal cord trauma is directed to the cause. The key to success is a correct diagnosis and the presence of pain perception.
Less life-threatening emergencies

Fractures and luxations of the bony pelvis and extremities are not considered life-threatening emergencies. Of greater importance is the damage to associated neural, vascular, and soft tissues surrounding these bony injuries. In fractures of long bones, blood loss may exceed 25% of the total blood volume. This blood is often not obviously lost but rather sequestered about a fracture site.

Fractures are classified as either open or closed. Most closed fractures pose no immediate threat to life and definitive repair is generally delayed at least 24 hours. Application of a splint will relieve pain, lessen additional swelling of the limb, and prevent a closed fracture from becoming an open fracture. The principle of immobilization of the joint above and the joint below the fracture decreases the usefulness of splints in animals. In splinting, the toes should remain partially exposed for assessment of color, pain, swelling, discharge, odor, and temperature. Open fractures require a thorough cleansing and covering of the wound along with appropriate antimicrobial therapy. Contaminated wounds should be cultured with antimicrobial sensitivity performed. The last step in the emergency management of the open fracture is the application of sterile gauze and immobilization if possible.

Bite wounds, gunshot wounds, and wounds with massive contusions should not be closed. Early closure of these wounds generally results in disruption and prolonged convalescence. Definitive fracture treatment is undertaken after the animal is stabilized.

Abdominal Trauma

Abdominal injuries are occult. Injuries caused by blunt trauma include lacerations of the liver and/or spleen, urological trauma, infarcted bowel, or reproductive organ damage during pregnancy. Penetrating injuries from gunshot, impalement injuries, and bite wounds are more obvious. The wounding potential of missiles is related both to velocity and mass of the bullet. High velocity missiles produce cavitation within the abdomen that is sufficiently energetic to disrupt hollow organs, break bones and spread contamination. Physical examination findings and diagnostic studies are required in deciding which abdomen should be surgically explored following penetrating injury. This decision is generally based upon signs of peritoneal penetration, unexplained shock, ileus, organ evisceration, free gas on radiographic examination or evidence of bacteria or plant debris following abdominocentesis or peritoneal lavage. Blunt abdominal trauma cases are challenging diagnostic problems because the clinical manifestations may be delayed for hours or days. Abdominal tenderness is an important clinical signs of peritoneal irritation by blood or intestinal contents.

A four quadrant abdominocentesis is our preferred means for confirming blunt abdominal injury. From the fluid obtained, a packed cell volume, total solids, cytology, bilirubin, and creatinine are submitted. If the packed cell volume of centesis fluid exceeds the peripheral packed cell volume, very likely there is either a splenic, hepatic or renal parenchymal laceration. In the dog or cat our approach is to treat these patients as conservatively as possible. With an abdominal pressure bandage and individualized fluids therapy, it is unusual to require surgery for a splenic or hepatic laceration. Caution should be employed in applying an excessively tight bandage when thoracic injuries are also present.

With biliary injury, the clinical signs of icterus are often delayed 4 to 6 weeks. If the abdominal fluid bilirubin is approximately 30 times greater than peripheral bilirubin, then surgical exploratory will be required to close the lacerated organ and lavage the abdomen. This surgery is not considered an emergency procedure. With urological injury, the packed cell volume of the abdominal fluid will be lower than the peripheral packed cell volume due to hemodilution with urine. The diagnosis is confirmed by comparison of abdominal fluid urine nitrogen or creatinine to peripheral blood values collected at the time of the abdominocentesis.
Emergency management of intraperitoneal rupture of the bladder, urethra, and/or ureters involves drainage of the abdominal fluid via an indwelling catheter until the patient is sufficiently stable to undergo anesthesia and surgical repair. Prior to surgery, contrast studies of the kidneys, ureter, and bladder should be performed to assess the severity of injury using an excretory urogram. If there is evidence of lower urinary tract injury, positive contrast urethrography and cystography may be necessary.

Should plant debris or significant numbers of mixed bacteria be found with centesis of the abdominal fluid, a ruptured viscus is likely and exploratory surgery is indicated. Use of peritoneal lavage for diagnosis of abdominal injury should be considered if abdominocentesis is negative. If no blood, bile, urine or intestinal fluid can be aspirated, the abdominal fluid is irrigated with 10 to 20 ml/kg of warmed crystalloid fluid.

Hypothermia, Acidosis and Coagulopathy

The relationship of hypothermia to the development of coagulopathy is seen both in vitro and in the clinical patient. Hypothermia impairs platelet aggregation and decreases function of coagulation factors in pre-resuscitation (undiluted) blood. Clinically, human patients with a temperature lower than 34°C had elevated PT and PTT. Studies documenting this effect showed a linear relationship between the elevation in the coagulation profile times and the drop in the patient’s core temperature. Acidosis, which occurs in the setting of trauma as a result of bleeding and hypotension, also contributes to the failure to clot. Experimentally, animals with a pH less than 7.20 have impaired hemostasis. It has been shown that the presence of acidosis is one of the strongest risk factors for the development of life-threatening hemorrhage in patients receiving massive transfusions. Even therapeutic options, such as factor VIIa, may be less effective in a low pH environment.

Correction of Coagulopathy

Coagulopathy and microvascular bleeding continue to be major contributors to early inhospital death after an injury and new treatment approaches are needed to reduce mortality rates. Recent studies of early coagulopathy in trauma provide new reasons for the ongoing bleeding. This knowledge considers post trauma coagulopathy as a primary, rather than secondary, event after an injury. Earlier and more aggressive correction of hypoperfusion along with coagulation factor replacement should lessen hemorrhage-related mortality.

Will survival improve if coagulation parameters are corrected in veterinary patients? This question still remains to be answered. As discussed before many of our clinical practices used to treat shock in trauma are thought to worsen coagulation parameters. Systematic reviews of the human literature have documented very few reports of changes to standard coagulation profiles following fluid resuscitation. The few reports that have provided evidence of prolongation of clotting tests with larger and earlier fluid usage (including those that showed improved hemostasis with recombinant factor VIIa treatment) failed to show that these changes affected mortality. Early reviews evaluating TEG and transfusion practice in trauma were not able to provide any specific information about how clotting tests should best be used.

It is important to be able to predict or diagnose coagulopathy so we can identify patients at greater risk of major bleeding in whom intensive transfusion management may improve outcome. Since management of coagulopathy in veterinary medicine is almost entirely directed at augmenting clotting factors with blood component therapy it may be important to consider the thrombomodulin–protein C pathway. If the anticoagulation is from activation of the thrombomodulin–protein C pathway, adding factors to enhance thrombin activation in the presence of hypoperfusion may activate anticoagulant and fibrinolytic pathways. However, if protein C is exhausted, clot formation in underperfused vascular beds may result in
microvascular thrombosis, and subsequent organ dysfunction. Therefore, management of acute traumatic coagulopathy should focus on limiting the degree and duration of shock and tissue hypoperfusion to avoid the drop in protein C. With a clearer understanding of the thrombomodulin-protein C pathway and the concerns with microvascular bleeding and thrombosis, TEG may prove to be a useful clinical tool to identify and manage the hypoocoagulability and hyperfibrinolysis related to the activation and depletion of Protein C.

**Tranexamic acid and CRASH-2**

Tranexamic acid is a synthetic derivative of lysine. It is used to reduce bleeding in human elective surgery patients. It is an antifibrinolytic that competitively inhibits the activation of plasminogen to plasmin, preventing the degradation of fibrin, the major framework of blood clots. It is similar to but more potent than ε-aminocaproic acid.

The CRASH-2 trial was undertaken in 274 hospitals in 40 countries. 20,211 adult trauma patients with, or at risk of, significant bleeding were randomly assigned within 8 h of injury to either tranexamic acid or placebo. It was hypothesized that the inhibition of fibrinolysis would lead to improved hemostasis in trauma patients. An alternative hypothesis is that tranexamic acid might act by reducing the pro-inflammatory effects of plasmin, rather than by improving hemostasis.

The results showed that administration of tranexamic acid to adult trauma patients with, or at risk of, significant hemorrhage, within 8 h of injury, significantly reduced all-cause mortality with no apparent increase in pathologic thrombosis. With the publication of this trial, tranexamic acid has been incorporated into human trauma treatment protocols worldwide. Studies in veterinary patients are ongoing and hopefully we will have information about efficacy and dosing. In the meantime we are using Aminocaproic acid:

<table>
<thead>
<tr>
<th>Hemostasis, transfusion, and the ideal red cell to plasma ratio</th>
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<tbody>
<tr>
<td>Hemorrhage control has to be an early goal when resuscitating bleeding patients. In addition to the CRASH-2 results, multiple human studies evaluating the efficacy of recombinant factor VIIa found an average reduction in the number of packed red blood cell units needed for resuscitation. Transfusion of RBCs to patients that are exsanguinating offers life-saving potential. Recent reviews have focused on the PRBC:FFP ratio. Several studies looking at this ratio have reported the lower the PRBC to FFP ratios did have lower mortality. That is there was lower mortality with higher levels of FFP and platelets (lower PRBC:FFP ratios). Still, with large numbers of observational studies in this area, an optimal consistent FFP/RBC ratio has not been reported.</td>
</tr>
</tbody>
</table>

![Aminocaproic acid](https://example.com/aminocaproic-acid)
PRACTICAL TRANSFUSION MEDICINE
Tim Hackett, DVM MS, Dip. ACVECC

Introduction

Transfusion of blood to the anemic dog or cat can make a tremendous difference carrying oxygen to tissues to support vital functions. Transfusions can be safely done in any practice provided potential donors are appropriately screened and the clinician practices care in the collection, processing, storage, and administration of the blood.

Canine Transfusion Medicine

1. Type potential canine donor dogs with card test as DEA 1.1(+) or 1.1(-).
2. Consider only using DEA 1.1(-) donors OR use DEA 1.1(+) donors for DEA 1.1(+) recipients and DEA 1.1(-) donors for DEA 1.1(-) or 1.1(+) recipients (our approach at CSU).
3. Infectious disease screening. in the Colorado, we screen for Brucella spp. disease, Ehrlichia canis, Babesia canis, and Heartworm disease.
4. Potential donors should be less than 8 years of age, have a normal CBC, Serum Biochemical Profile, and Urinalysis, no previous medical problems, a body weight greater than 40 pounds.
5. There should be no concurrent medications, an appropriate vaccination history, and have a personality that will allow for sustained restraint for blood collection.
6. All screening with the exception of blood type should be repeated on a yearly basis.

Feline Transfusion Medicine

1. Type potential feline donors as Type A or B. Try to identify a donor of each type to be available for donation.
2. Only consider using Type A blood for Type A cats and only consider using Type B blood for Type B cats.
3. Infectious disease screening at CSU looks for FeLV, FIV, Bartonella spp. and Hemobartonella felis (now Mycoplasma sp.).
4. Good flea control measures should be in place and ideally feline donors should not be allowed outdoors or exposed to cats that are.
5. Donor cats should weigh greater than 10 lbs, have normal CBC, Serum Biochemical Profile, and Urinalysis, no previous medical problems.
6. There should be no current medications, a good vaccination record, no previous transfusions, and a personality conducive to phlebotomy.

Xenotransfusion of canine blood into cats

A recent report in the Journal of Feline Medicine and Surgery (2012 Vol. 15(2) 62-67) has brought the subject of using dog blood in cats needing a transfusion back to the table. Finding five studies from the 1960s where over 50 cats received canine blood. Multiple transfusions within 5 days were tolerated. The authors conclude this is a viable alternative when the appropriate feline blood product is not available. The cats should never receive another dog unit after 5-6 days because they will develop antibodies and will likely have a transfusion reaction.
**Collection of Blood for Transfusion**

The area over the jugular vein should be clipped and a surgical preparation performed. For dogs, we routinely use a commercially available **closed** collection system containing CPDA-1 anticoagulant. Collection in cats is performed with the same system the only difference is that most of the anticoagulant is drained from the bag leaving only enough in the line to anticoagulate the smaller (55-60 ml. Unit). Risks of bacterial contamination using an open system are significantly greater than those with a closed system so blood collected in an open manner should be used immediately.

400-450l of whole blood may be collected from dogs and 40-50ml of whole blood may be collected from cats. Smaller cats and dogs may show signs of hypovolemia after their first donation. Donors should be observed closely and if necessary rapid intravascular volume expansion with IV fluids may be necessary. If this happens, donation protocol may need to be adjusted. Whole blood and packed red blood cells collected into CPDA-1 from a closed collection system can be stored in a temperature controlled refrigerator at 4C for up to 35 days.

**Blood Product Administration**

Blood and blood products should be delivered via a commercially available 170µm blood filters. An in-line filter is best used in cats and small dogs receiving less than 60ml of blood or blood products. Filtration of the blood will reduce the risk of blood clots and other particulate matter that could embolize in the recipient. We begin non-emergency transfusions of whole blood or packed red blood cells slowly (1-3ml over 5min in cats and .25ml/Kg over the first 30min in dogs) while monitoring for signs of transfusion reaction. If no reaction is noted, the transfusion may be delivered over 1-4 hours while intermittently evaluating for any evidence of a transfusion reaction. Recipients with underlying heart disease or other conditions that may result in clinical volume overload should have the rate reduced and closely monitored. We try to avoid hanging blood products at room temperature longer than 4 hours due to the risk of bacterial proliferation in the product.

**Transfusion Reactions**

There are four types of transfusion reactions:

1. Acute immunologic
2. Acute non-immunologic
3. Delayed immunologic
4. Delayed non-immunologic

An acute immunologic transfusion reaction may occur when antibodies to donor RBCs are present in the recipient plasma. An example would be the administration of Type A blood to a type B cat. The donated cells can be rapidly destroyed in 1-2 hours and could be fatal. A similar situation could occur in a DEA 1.1 negative dog transfused multiple times with DEA 1.1 (+) blood. Other less severe signs of acute immunologic transfusion reactions include fever and urticaria. Acute immunologic transfusion reactions can be avoided in most cases by the administration of type specific blood. When a patient has been transfused repeatedly, a major crossmatch is indicated even when using type-specific blood.

Acute non-immunologic transfusion reactions include air embolism, blood clot embolism, hypocalcemia because of citrate (anticoagulant used in blood collection) toxicity, and bacterial contamination of the product.

Delayed immunologic transfusion reactions result from development of antibody that could shorten transfused RBC lifespan. They could result from the transfusion of blood contaminated
with organisms like FeLV or FIV. Delayed non-immunologic reactions can be prevented through appropriate donor screening.

We recommend monitoring for acute reactions by collecting baseline temperature, pulse, and respirations. These should be reassessed every 5 min for 15-30 min as the transfusion is started. If no reaction is noted, the patient should be reassessed every 15 min. The patient should also be assessed for vomiting during this time and if urine is voided or collected, it should be evaluated for the presence of hemolytic pigment. If any evidence of a hemolytic transfusion reaction is noted, the transfusion should be stopped immediately and the recipient supported aggressively. If a febrile, non-hemolytic reaction is noted without any evidence of cardiovascular or respiratory compromise, the rate of the transfusion can be slowed. If the fever persists or worsens, the transfusion should be discontinued. Urticaria can be managed with short acting steroids and antihistamines if necessary and safe for the recipient.

**Crossmatching**

Blood crossmatching tests look at the true compatibility or incompatibility between donor and recipient. The crossmatch tests check for the presence or absence of naturally occurring and induced alloantibodies in serum (or plasma). The major crossmatch tests for alloantibodies in the recipient's plasma against donor cells, whereas the minor crossmatch test looks for alloantibodies in the donor's plasma against the recipient's red blood cells. A major crossmatch incompatibility is of greatest importance because it predicts that the transfused donor cells will be attacked by the patient's plasma, thereby causing a potentially life-threatening acute hemolytic transfusion reaction. A minor crossmatch incompatibility should not occur if donors have not been previously transfused and is of lesser concern because donor's plasma volume is small, particularly in packed red cell products, and will be markedly diluted in the patient.

The initial blood crossmatch between two dogs that have never before received a transfusion should be compatible, because dogs do not have naturally occurring alloantibodies. Therefore, we usually omit the crossmatch before the first transfusion in clinical practice. Because the crossmatch does not determine the blood type of the patient and donor, a compatible crossmatch does not prevent sensitization of the patient against donor cells within 1 to 2 weeks. Previously transfused dogs should be crossmatched, even when receiving blood from the same donor. The time span between the initial transfusion and incompatibility reactions may be as short as 4 days and lasts for many years (i.e., years after the last transfusion alloantibodies may be present). Obviously, a blood donor should never have received a blood transfusion.
# BLOOD CROSS-MATCHING

<table>
<thead>
<tr>
<th>Canine</th>
<th>Feline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Add one drop of Donor RBCs to 3 mL saline.</td>
<td>For each potential donor, prepare three microscope slides.</td>
</tr>
<tr>
<td>Repeat with a second tube.</td>
<td></td>
</tr>
<tr>
<td>Add one drop of Recipient RBCs to 3 mL saline.</td>
<td>Label slides: Major cross-match; Minor cross-match; Recipient control.</td>
</tr>
<tr>
<td>Repeat with a second tube.</td>
<td></td>
</tr>
<tr>
<td>Spin each tube for 15 secs at 3500 rpm. Pour off saline; add fresh</td>
<td>Place two drops of Recipient Plasma + one drop of Donor RBCs on Major cross-match slide;</td>
</tr>
<tr>
<td>saline as before; spin down; decant saline, add fresh saline and repeat</td>
<td>two drops of Donor Plasma + one drop of Recipient RBCs on Minor cross-match slide; and two</td>
</tr>
<tr>
<td>until each RBC tube is washed three times. Pour off saline, leaving</td>
<td>drops of Recipient Plasma + one drop of Recipient RBCs on control slide.</td>
</tr>
<tr>
<td>cells in tubes.</td>
<td></td>
</tr>
<tr>
<td>Add two drops of Recipient Plasma to washed Donor RBCs = Major cross-</td>
<td>Mix each slide gently back and forth with rocking motion and examine for several mins,</td>
</tr>
<tr>
<td>match. Add two drops of Donor Plasma to washed Recipient RBCs = Minor</td>
<td>looking for hemagglutination. This should be obvious to the naked eye, and the recipient</td>
</tr>
<tr>
<td>cross-match. Add two drops of Recipient Plasma to washed Recipient</td>
<td>control slide should be negative.</td>
</tr>
<tr>
<td>RBCs = Recipient control. Add two drops of Donor Plasma to washed</td>
<td></td>
</tr>
<tr>
<td>Donor RBCs = Donor control.</td>
<td></td>
</tr>
<tr>
<td>Mix, place drop of each tube on separate microscope slides to check</td>
<td>Repeat procedure for other potential donors.</td>
</tr>
<tr>
<td>for agglutination or hemolysis. Both controls should be negative.</td>
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</tbody>
</table>
Introduction

Respiratory distress in dogs is not only frightening for owners. Veterinarians faced with having to make quick decisions fear making the wrong choice or not making the right choice fast enough. There is a fairly short list of differential diagnoses for causes of dyspnea in dogs. Some of these causes can look very similar but treatments may vary widely. Some treatments for one condition may not help (or even harm) another cause of dyspnea. With an understanding of the common causes, how they look alike, and how they can be differentiated, the medical team can quickly rank possible causes and treat accordingly.

Initial observation and diagnostics

Try to determine the nature of the problem first with observation. A rapid shallow respiratory pattern suggests restrictive disease while a slow deep inspiratory pattern is seen with airway obstruction. With the restrictive pattern, auscultation can help differentiate pleural space disease (pneumothorax, hydrothorax) from parenchymal diseases (pneumonia, pulmonary edema). Signalment and history can help determine a cause of upper airway obstruction (playing with small toys, brachycephalic airway diseases, laryngeal paralysis). Once the patient has been sedated and calmed, treated for shock and hyperthermia, definitive diagnostics can be performed. Some of the important emergency diagnostics will be reviewed.

Imaging

Animals presenting with upper and lower respiratory signs should have a thoracic radiograph. Bronchial patterns develop as the peribronchiolar tissues become inflamed and the airways thicken. Interstitial patterns develop with thickening of the fibrous structures of the lung. Alveolar patterns characterized by “Air bronchograms” are caused by fluid accumulation in the alveoli. Thoracic and cervical radiographs can be used to diagnose collapsing trachea, tracheal or laryngeal foreign bodies, and tracheal or laryngeal masses. Taking inspiratory and expiratory views of the trachea or through the use of fluoroscopy it is possible to assess dynamic changes in airway diameter.

Thoracocentesis

When pleural fluid or air accumulation is suspected, a thoracocentesis can treat the impaired tidal volume while making the diagnosis. The character of the fluid or presence of air will be valuable in understanding the cause of the problem. Thoracocentesis should be approached as a quick “yes-no” question. With one person manipulating the needle and another holding the syringe and tubing. The syringe is aspirated as soon as the needle is through the chest wall and the team quickly determines if air or fluid is causing the problems. If no air or fluid is easily withdrawn, the needle is withdrawn to avoid iatrogenic lung injury. Ultrasound is a useful modality to determine the presence and location of fluid if available.

Airway cytology

Transthreeal and transoral airway wash are useful techniques for the diagnosis of diseases of the respiratory system and easily performed in most dogs in about 15 minutes. It should be performed following assessment of the thoracic radiographs and is indicated for all coughing dogs and cats with interstitial, bronchial, or alveolar lung patterns that are not suspected to be due to cardiogenic disease or coagulopathy. The goal of the wash is to collect
fluids from the trachea, bronchi and lower airways for cytology, culture, and antibiotic susceptibility. Animals rarely develop subcutaneous emphysema and pneumomediastinum following airway wash and so should be observed in the hospital for several hours following completion of the procedure.

**Laryngeal paralysis** can be either congenital or acquired and is a common cause of emergency visits in large breed dogs. The paralysis may be either unilateral or bilateral. Acquired laryngeal paralysis is more common with many proposed etiologies. The recurrent laryngeal nerve innervates the arytenoid processes of the larynx. One of the longest nerves in the body, it is susceptible to a variety of degenerative processes. Damage to the nerve anywhere along its course by trauma, surgery, neoplasia, polyneuropathy or even hypothyroidism can lead to a loss of innervation of the intrinsic laryngeal muscles. Animals with laryngeal paralysis will present with varying degrees of exercise intolerance, stridor, voice change, inspiratory effort, cyanosis and hyperthermia. They will have a pronounced inspiratory stridor with a loud, deep (obstructive) breathing pattern. Often an obvious inspiratory wheeze will be heard loudest over the larynx. Diagnosis is by direct examination under a light plane of anesthesia. A low dose of propofol is administered to allow the mouth to be held open while visualizing the glottis. If the animal becomes apneic with the sedative a 1 mg dose of doxapram HCL IV can initiate a large breath. Normally the arytenoid cartilage abducts on inspiration. With laryngeal paralysis the arytenoids may actually be drawn together during inspiration causing inflammation and edema. Laryngeal paralysis may be unilateral or bilateral. Increased airway pressures can lead to everted laryngeal sacules further compromising the laryngeal lumen.

Because panting is such an important method of controlling body temperature, subclinical laryngeal paralysis may only become evident on hot days or following strenuous exercise. The body temperature can quickly climb to dangerous levels, necessitating treatment for heat stroke. Dyspnea from upper airway obstruction can cause the animals to become anxious and more dyspneic. A vicious cycle begins as the more distressed they become, the harder they try to breathe. Handling these animals can be difficult and often the best treatment is sedation. Acepromazine is a predictable sedative. A dose of 0.02 – 0.04 mg/kg not to exceed 0.25 mg will break the cycle of distress. Acepromazine should be given cautiously in dehydrated or shock patients as it may cause a drop in blood pressure. Patients with prolonged hyperthermia should be hospitalized and observed for complications. The kidneys, GI tract, liver and nervous tissue can all be damaged by excessive heat. Disseminated intravascular coagulation is another common complication. Once the patient is stable and signs of heat stroke have resolved definitive treatment for laryngeal paralysis can proceed.

**Pulmonary Edema.** Non-cardiogenic pulmonary edema occurs occasionally in dogs and cats secondary to electric cord bites, sepsis, following near drowning or choking, snake bites, uremia, smoke inhalation, upper airway obstruction, and the adult respiratory distress syndrome (ARDS). Dogs that chew on electric cords often present with acute onset of dyspnea and oral burns, which may or may not be associated with dysphagia or ptyalism. The syndrome occurs most commonly in the young. Pulmonary edema develops rapidly, generally within hours. Common physical examination abnormalities include oral burns, dyspnea, and pulmonary crackles. Thoracic radiographs show mixed interstitial and alveolar patterns that are most prominent in the dorsal portions of the caudal lung lobes. The pathogenesis of edema is thought to be increased pulmonary capillary hydrostatic pressure and increased alveolar-capillary permeability. Increased pulmonary capillary hydrostatic pressure is likely due to a centrally mediated burst of sympathetic activity, which causes constriction of resistance and capacitance vessels leading to a shift of blood from the splanchnic viscera into the circulation. This ultimately results in overcirculation of the pulmonary vasculature. Increased peripheral vascular
resistance increases pulmonary capillary hydrostatic pressure and pulmonary venous pressures increase as the left ventricle pumps against increased outflow resistance. Treatment includes administration of low dose narcotics, diuretics, and oxygen (mask, nasal insulation or oxygen cage). Morphine and other pure narcotic agonists at lower doses can have a good clinical effect. At low doses it sedates dyspneic animals while drawing excess fluid from the lungs via splanchnic vasodilatation.

The clinical signs and physical examination abnormalities associated with near drowning, smoke inhalation, and snakebite are similar to those with electric cord bites with the exception of oral burns. Historical findings confirm near drowning and smoke inhalation. Puncture wounds and a swollen face or extremities may be found on animals with snakebite. Administration of bronchodilators may also aid in the treatment of some cases. Smoke inhalation causes dyspnea by inducing carbon monoxide poisoning and damage to respiratory tissues by heat and noxious gasses. Laryngeal spasm, loss of ciliary function, decreased surfactant activity, bronchospasm, increased alveolar-capillary permeability, impaired phagocytosis, and sloughing of airway mucosa frequently occur. Bronchial patterns occur first with interstitial and alveolar edema developing later if edema develops. Treatment is similar to electric cord bite and near drowning.

Pulmonary edema occasionally develops secondary to upper airway obstruction in dogs. Laryngeal and pharyngeal diseases are most common. Inspiratory and expiratory stridor, dyspnea, crackles, and cyanosis are common physical examination abnormalities. Mixed interstitial and alveolar lung infiltrates are detected in the perihilar and dorsocaudal lung fields. Treatment can include administration of oxygen, diuretics and glucocorticoids, as well as tracheostomy if needed. Edema is primarily related to decreased intrathoracic pressure resulting in decreased interstitial hydrostatic pressure and hypoxia resulting in increased alveolar capillary permeability.

**Pneumonia.** Bacterial pneumonia in dogs is rarely a primary disease. Occasionally, *Bordetella bronchiseptica* or *Mycoplasma* spp. can induce pneumonia due to their adverse affects on mucociliary function. Most cases of bacterial bronchopneumonia are secondary to immunosuppressive diseases or previous inflammatory insults including viral infection, aspiration, and irritant inhalation. Owners should be carefully questioned concerning potential exposure to other animals and clinical signs associated with immunosuppressive diseases or aspiration.

Most animals with bacterial pneumonia will be clinically ill. Common complaints include depression, anorexia, dyspnea, productive, moist cough with a terminal retch, and exercise intolerance. Some animals with pneumonia will present only with cough. Physical examination findings commonly include fever, crackles and wheezes, and muffled lung sounds in cases with consolidated or abscessed lung lobes. Many dogs will have increased tracheal sounds, a tracheal cough, and pharyngeal inflammation due to transport of inflammatory cells up the mucociliary apparatus to the mouth. Thoracic radiographs usually reveal a mixed alveolar, bronchial, and interstitial pattern. Aspiration pneumonia generally has radiographic lesions that are most pronounced in the right middle lung lobe. Animals with opacity of the right middle lung lobe should be evaluated for esophageal and gastrointestinal disease or respiratory stridor. Esophageal diseases leading to regurgitation and aspiration may be evident on evaluation of thoracic radiographs. Laryngeal paralysis, which is characterized by inspiratory stridor, can predispose dogs to aspiration.

One of the most important treatments of bacterial pneumonia is hydration. The mucociliary apparatus function best in a well-hydrated animal and is essential for the clearance of infection. Affected animals should receive parenteral fluid therapy until able to maintain hydration orally. Airway hydration can be accentuated by nebulization or by placing the animal
in a closed bathroom while running hot water through the shower. Common bacterial isolates include *Bordetella bronchiseptica*, *Pasteurella multocida*, *Klebsiella* spp., *Streptococcus* spp., and *Escherichia coli*.

**Canine bronchitis** causes a cough occurring on most days usually in the absence of other active disease. With long standing inflammation histologic changes include fibrosis, epithelial hyperplasia, glandular hypertrophy, and inflammatory infiltrates. Canine chronic bronchitis is likely a consequence of a chronic inflammatory process initiated by infection, allergy, or inhaled irritants or toxins. Uncontrolled, inflammation leads to mucosal damage. Excessive mucus secretion, and airway obstruction impair normal clearance mechanisms. Tracheobronchial weakness can further contribute to the ongoing cycle of cough and inflammation. The inflamed airways are also prone to dynamic collapse. These patients typically present with an expiratory wheeze and increased expiratory effort. They can be distinguished from the cardiac patient with pulmonary edema who will have primarily an inspiratory dyspnea with pulmonary crackles. Treatment is aimed at reversing inflammation while opening the airways with a bronchodilator.

**Pleural Space Disease.** Diseases of the pleural space cause a decreased tidal volume and a restrictive breathing pattern. Characterized by the rapid, shallow respirations and dull lung sounds, fluid and air in the pleural space can be diagnosed and treated by rapid thoracocentesis following the procedure above. Once removed, fluid can be examined to determine the likely cause. A negative thoracocentesis may be due to fibrous adhesions and small pockets of fluid. Diaphragmatic hernias may also cause significant pleural restriction but yield a negative tap. These patients should be given supplemental oxygen while ultrasound is performed or radiographs taken. Large volumes of air or fluid, continuous production of air, or suppurative inflammation are indications for tube thoracostomy. Pneumothorax and some inflammatory conditions may require continuous suction. Disposable suction devices are available to hook onto surgical suction units. These “3-bottle” devices allow easy regulation of suction (20 cm H<sub>2</sub>O desirable), a water trap to prevent air from being drawn into the chest should suction become interrupted, and a collection chamber to quantitate fluid production.
FELINE RESPIRATORY EMERGENCIES
Tim B. Hackett DVM MS DACVECC

Introduction
Respiratory distress in cats is particularly difficult for veterinarians trying to make quick decisions afraid of making the wrong choice or not making the right choice fast enough. There is a short list of differential diagnoses for causes of dyspnea in cats. Dyspneic cats either have heart failure (with pleural effusion and/or pulmonary edema), bronchitis (most commonly asthma), or a pleural space disease. Some of these causes can look very similar but treatments may be different. Treatments for one condition may not help (or even harm) another cause of dyspnea. With an understanding of the common causes, how they look alike, and how they can be differentiated, the medical team can quickly rank possible causes and treat accordingly. Handling these cats can be difficult and often the best treatment is sedation. In cats, I prefer butorphanol to calm and sedate the dyspneic cat. A dose of 0.1-0.2 mg/kg IM or IV will break the cycle of distress.

Observation and diagnostics
Inspiratory dyspnea in cats is usually seen with pulmonary edema and pleural space disease. As it requires more effort to breath in, these cats adopt a rapid shallow (restrictive) breathing pattern to minimize the work overcoming the increased elastic forces. Expiratory dyspnea in the cat is due to collapse and narrowing of the small airways within the chest. This is the hallmark of bronchitis. The causes of bronchitis may vary and require further diagnostics to arrive at optimal care. However the emergency management of feline bronchitis is the same regardless of etiology.

Thoracocentesis
When pleural fluid or air accumulation is suspected, a thoracocentesis can treat the impaired tidal volume while making the diagnosis. The character of the fluid or presence of air will be valuable in understanding the cause of the problem. Thoracocentesis should be approached as a quick “yes-no” question. With one person manipulating the needle and another holding the syringe and tubing. The syringe is aspirated as soon as the needle is through the chest wall and the team quickly determines if air or fluid is causing the problems. If no air or fluid is easily withdrawn, the needle is withdrawn to avoid iatrogenic lung injury. Ultrasound is a useful modality to determine the presence and location of fluid if available.

Imaging
Cats presenting with upper and lower respiratory signs should have a thoracic radiograph. Bronchial patterns develop as the peribronchiolar tissues become inflamed. Interstitial patterns develop with thickening of the fibrous structures of the lung. Alveolar patterns characterized by “Air bronchograms” are caused by fluid accumulation in the alveoli. Thoracic and cervical radiographs can be used to diagnose collapsing trachea, tracheal or laryngeal foreign bodies, and tracheal or laryngeal masses. Taking inspiratory and expiratory views of the trachea or through the use of fluoroscopy one can assess airway dynamics.

Transoral tracheal wash
Transoral tracheal wash (transtracheal wash, TTW) is one of the most useful techniques for the diagnosis of diseases of the respiratory system. The TTW can be easily performed in most cats in about 15 minutes. The TTW should be performed following assessment of the thoracic
radiographs and is indicated for all coughing cats with interstitial, bronchial, or alveolar lung patterns that are not suspected to be due to cardiogenic disease or coagulopathy. The goal of the TTW is to collect fluids from the trachea, bronchi and lower airways for cytology, culture, and antibiotic susceptibility.

**Pulmonary Edema.**
Because the lungs are considered the “shock organ” in cats, any hypotensive event can result in alveolar flooding and edema. Thoracic radiographs show alveolar or a mixed interstitial/alveolar pattern that can be diffuse throughout the chest cavity, perihilar (in the case of left-sided cardiac disease) or most prominent in the dorsal portions of the caudal lung lobes (with non-cardiogenic causes). The pathogenesis of edema is increased pulmonary capillary hydrostatic pressure, increased alveolar-capillary permeability or both. Increased pulmonary capillary hydrostatic pressure is likely due to congestive heart failure or an acute burst of sympathetic activity that shifts blood from the splanchnic viscera into the circulation. Either cause results in over circulation of the pulmonary vasculature and leakage of fluid into the airways. Treatment includes supplemental oxygen, administration of diuretics, morphine, or positive end expiratory pressure ventilation.

**Feline Bronchial disease.**
There is no clear terminology for the bronchial obstructive diseases in the cat. Bronchitis is inflammation of the airways. Asthma generally implies a reversible bronchoconstriction related to hypertrophy of smooth muscle in airways, hypertrophy of mucous glands, and infiltrates of eosinophils. Asthma in cats is primarily due to Type I hypersensitivity reactions; the etiology is generally undetermined. Cats with bronchitis not due to asthma generally have infiltrates of neutrophils or macrophages as well as hypertrophy of mucous glands, hyperplasia of goblet cells, excessive mucous, and ultimately fibrosis secondary to chronic inflammation. Etiologies include bacterial infection, mycoplasmosis, viral infection and parasitic infections.

Cats with bronchitis can be of any age; chronic bronchitis usually develops in middle-aged to older cats. There is no obvious breed or gender predilection. Primary presenting complaints include cough, dyspnea, and wheezing. Some cats will have a terminal retch following cough. Physical examination abnormalities include cough, dyspnea, and crackles, and wheezes in the pulmonary tissues. Increased bronchovesicular sounds may be the only abnormality noted on auscultation. If dyspnea occurs, it commonly has a pronounced expiratory component. Open mouth breathing or panting commonly occurs during periods of stress.

CBC is generally normal with the exception of eosinophilia in some cats with asthma. Thoracic radiographs reveal primarily a bronchial pattern. Over inflation and air trapping is seen in some dyspneic cats with chronic disease. Air bronchograms are commonly seen in some dyspneic cats with bronchitis due to bacterial infection. Cytology of transtracheal wash samples reveals increased mucus with variable numbers of eosinophils, neutrophils, and macrophages. Bacteria may or may not be visualized. Aerobic and *Mycoplasma* culture as well.

**Pleural Space Disease.**
Diseases of the pleural space cause a decreased tidal volume and a restrictive breathing pattern. Unlike dogs, cats can have pleural effusion from heart disease. Other common causes include chylothorax, pyothorax, and neoplastic effusions. Characterized by the rapid, shallow respirations and dull lung sounds, fluid and air in the pleural space can be diagnosed and treated by rapid thoracocentesis. Once removed, fluid can be examined to determine the likely cause. A negative thoracocentesis may be due to fibrous adhesions and small pockets of fluid. Diaphragmatic hernias may also cause significant pleural restriction but yield a negative tap.
These patients should be given supplemental oxygen while ultrasound is performed or radiographs taken.

Large volumes of air or fluid, continuous production of air, or suppurative inflammation are indications for tube thoracostomy. Pneumothorax and some inflammatory conditions may require continuous suction. Disposable suction devices are available to hook onto surgical suction units. These “3-bottle” devices allow easy regulation of suction (20 cm H₂O desirable), a water trap to prevent air from being drawn into the chest should suction become interrupted, and a collection chamber to quantify fluid production.

<table>
<thead>
<tr>
<th>Type of Effusion</th>
<th>Protein (g/dl)</th>
<th>Cell count (/ul)</th>
<th>Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Modified Transudate</td>
<td>&lt;2.5</td>
<td>&lt;500-1000</td>
<td>Right Heart failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pericardial disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hypoalbuminemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Neoplasia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Diaphragmatic hernia</td>
</tr>
<tr>
<td>Exudate</td>
<td>&gt;3.0</td>
<td>&gt;5000</td>
<td>Feline infectious peritonitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Neoplasia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Diaphragmatic hernia</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Lung lobe torsion</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pyothorax</td>
</tr>
<tr>
<td>Chylous</td>
<td>&gt;2.5</td>
<td>&gt;500</td>
<td>Idiopathic</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cardiomyopathy</td>
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<tr>
<td></td>
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<td></td>
<td>Heartworm disease</td>
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</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Tuberculosis</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>&gt;3.0</td>
<td>&gt;1000</td>
<td>Trauma</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Coagulopathy</td>
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<td></td>
<td>Neoplasia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lung lobe torsion</td>
</tr>
</tbody>
</table>

Table 1. Common causes of pleural effusion in small animals. Effusions are classified based on protein content and cell count.
Conclusions

- An induction chamber or oxygen cage for cats can be valuable to give the patient added oxygen while the clinician can observe the patient in an attempt to localize the problem.
- Inspiratory dyspnea in cats is usually either pulmonary edema or pleural effusion while expiratory dyspnea is seen with cases of bronchitis including feline asthma.
- Cats are uniquely susceptible to pulmonary edema and iatrogenic fluid overload.
- While the emergency treatment of the various causes of feline bronchitis usually involves a fast acting steroid and bronchodilator, a fecal exam, blood work and airway cytology are necessary to identify the underlying cause.
- The use of long acting, respositol steroids in dyspneic cats may unmask cardiac disease leading to pulmonary edema. Their use in the chronic management of inflammatory disease should only be considered after a complete workup including echocardiography.
RECOGNIZING AND TREATING ENVENOMATIONS
Tim Hackett, DVM MS, Dip. ACVECC

Introduction
Veterinary patients can encounter a wide variety of potentially toxic creatures in and around the home and in their travels. “Bug bites” and snakebite are often included in differential diagnoses when animals present with vague signs such as unexplained swelling, or nausea. The purpose of this talk will be a review of common zootoxins seen in the United States, their diagnosis, over diagnosis, and treatment.

Insects
Bees, hornets and wasps (genus *Hymenoptera*) are found around the world and throughout North America. Dogs and cats are exposed when they disturb a nest, encounter a swarm or play with them when they find them around the house. *Hymenoptera* toxins are not as well characterized as snake venom, but are known to contain formic acid. Formic acid is painful when injected and is also found in the bite of stinging ants. Anaphylaxis and death can occur in animals exposed to massive envenomation (multiple stings) or in sensitized individuals. Since most of our patients are not able to communicate, diagnosis can be difficult. Signs are non-specific and include mild to severe pain and swelling around the sting. In some cases, urticaria and swelling can be seen all over the body. Bees may leave a venom sac with the stinger. Laboratory abnormalities are non-specific and not usually helpful. It is important to note that insect bites can cause echinocytosis of red blood cells. Echinocytes are very common with pit viper (Crotalid) envenomation, but not pathognomonic. Finding echinocytes on a blood smear does not rule out an insect bite. Treatment is purely supportive and includes treatment of anaphylaxis and circulatory shock, respiratory support, and analgesia. Specific antidotes are not available.

Fireflies (genus *Photinus*) are poisonous if ingested in large quantity. The toxin seems to be related to cardiotoxins found in *Bufo* toads. Veterinary reports of intoxication are limited to cases involving insectivorous pet lizards.

Spiders
Black widow spiders (*Latrodectus mactans* and *L. Hesperus*) are 1–2 centimeters in size, shiny and black and have a red hourglass mark on their abdomen. These spiders are found across the United States but more common in the East and Southwest. In Colorado, black widows may invade suburban lawns and gardens, woodpiles, basements and garages. They present a risk to pets and humans that accidentally encounter a nesting area. They have a neurotoxin (latrotoxin), which is believed to exert effects by binding to calcium channels. Calcium-channel binding increases the membrane's permeability to calcium and enhances depolarization resulting in a paralysis and destruction of peripheral nerves endings. Clinical signs include severe pain, which may be confused with signs of acute abdominal distress. There may be ascending motor paralysis, muscle spasms, muscle rigidity, and hypersalivation. Death may result from respiratory or cardiovascular failure. Laboratory evaluation is not helpful and lesions are not conclusive or distinctive. Treatment is both specific (antivenin which results in rapid relief) and supportive. Supportive care includes muscle relaxants, atropine to reduce salivation, and fluid therapy for circulatory shock.

Brown recluse spiders (*Loxosceles rectusa*) are 1–4 centimeters long, with a fiddle shaped mark on the dorsal aspect of the thorax. There are many species of *Loxosceles* found throughout the United States. They like dark places, as they are reclusive. The venom contains
digestive and hemotoxic proteins (hyaluronidase, proteases, and hemolysins). The result is damage to endothelial cell membranes, intravascular coagulation, micro-thrombi and tissue necrosis. The tissue necrosis can be dramatic leading to large open wounds and impressive scar formation. Experimentally, less than 5 µg of the venom can cause lesions. Interestingly the bite is often not noticed (painless?) and does not cause ulcerative necrosis in all cases. Therapy is supportive as there is no antidote available. If the patient is showing signs of hemolysis or hemoglobinuria, they should be started on intravenous fluids. Surgical debridement, excision of the affected area, analgesia, and antibiotics may be indicated. Prognosis is good to fair as the wound may take weeks to months to resolve.

**Pit Vipers**

Pit vipers occur throughout North America and in many parts of the world and include rattlesnakes (*Crotalus* spp.); copperheads, cottonmouths, and water moccasins (*Agkistrodon* spp.); and pygmy rattlesnakes and massasaugas (*Sistrurus* spp). Rattlesnakes comprise 20 species that vary in habitat, size, and most importantly, relative toxicity of venom. Because rattlesnakes usually warn before they strike, most bites can be avoided. Human snakebite victims are disproportionately represented by young males < 30 years old, and the bites are often associated with use of intoxicants and other illicit substances. Curious dogs could be considered the animal equivalent of a drunken teenager and usually get bitten on the face as they antagonize the innocent reptile.

The Western diamondback rattlesnake (*C. viridis*) is the most widely distributed rattlesnake in North America, with 9 recognized subspecies. Of all the subspecies, the prairie rattlesnake (*C. viridis viridis*) has the largest range, extending north to Alberta, Canada, and south to Coahuila province in Mexico. The prairie rattlesnake is the only wild venomous snake in northeastern Colorado.

Most Northern Colorado rattlesnake bites occurred between May and September. Swelling in the area of the bite is the primary physical abnormality. Snakebites generally occur on the face (81%); almost equal numbers of bites affected the forelimbs (12.4%) and the hind limbs (9.4%). Despite serious clinical signs at referral, most dogs referred to our hospital survive the bite with basic supportive care. The most common initial laboratory findings are echinocytosis (91%), thrombocytopenia (88%), and leukocytosis (53%). Echinocytes can be seen quickly, even before swelling of the affected area occurs. Small dogs are more severely affected (longer hospitalization) than large dogs. Antivenin administration for prairie rattlesnake bites did not effect the duration of hospitalization but was associated with higher platelet count sooner.

The lack of clinical information in makes it difficult to recommend ideal treatments for all rattlesnake envenomation in dogs. Data provided by a manufacturer of antivenom (Fort Dodge) indicated that 16 practicing veterinarians had positive results with antivenin in dogs with mild clinical signs at the time of treatment. With no mention of snake species involved, clinical problems, or other treatments, it is difficult to draw practical treatment recommendations from this information. There are many genera and species of snakes in North America, with a wide array of species-specific venom potency and clinical envenomation patterns; therefore, optimal treatments might differ. For example, the ideal treatment for a bite by Arizona’s deadly Mojave rattlesnake (*C. scutulatus*) or the Eastern diamondback rattlesnake (*C. adamanteus*) may need to be much more aggressive than for that of the southern copperhead (*A contortix contortix*), which is reported to have much less toxic venom. Conservative treatment (IV administration of fluids, use of analgesics, and local wound care) has been advocated for humans bitten by copperheads and other pit vipers with low-toxicity venom. Human snakebite victims report localized pain that is most severe after envenomation by the Eastern and Western diamondback, timber, pacific, and prairie rattlesnakes. For this reason some analgesic is indicated in all cases.
Many variables have been reported to affect the amount of venom injected at the time of the bite. Species differences have been discussed, but the age of the snake and time since the last meal are variables unknown to the clinician. Twenty to 25% of North American pit viper bites are “dry bites”, with no clinical evidence of envenomation. For these reasons canine snakebite victims must be evaluated and treated individually. Clinical findings should provide the clinician enough information to institute appropriate supportive care.

Elapidae

*Coral snakes* are the only member of the *Elapidae* family indigenous to the United States. They are found in the southeast coastal states and west to Texas. The non-toxic king snakes look almost identical to the toxic coral snake but has yellow rings separated by a black band: “Red and yellow kill a fellow/ Red and black, venom lack.” Their toxins include a cholinesterase, which has parasympathetic effects, and a neurotoxic polypeptide that induces paralysis. Signs of envenomation include weakness, disorientation, and paralysis that can progress to respiratory failure. Parasympathetic signs can be seen including salivation, vomiting, and defecation or diarrhea. There is minimal to absent local swelling at the bite site. Treatment is both supportive (fluids, oxygen supplementation, ventilatory support) and specific (elapid antivenin). Prognosis is good if early, aggressive therapy is instituted.

Beaded Lizards

The *Gila monster* (*Heloderma suspectum*) and *Mexican beaded lizard* (*H. horridum*) are both easily recognized and found in the desert Southwest. They chew their venom into the victim using small grooved teeth. Like most reptiles they are peaceful and easygoing but will bite if provoked. Like pit bull terriers, they may not release their victim without a fight. Toxic properties and symptoms are similar to crotalid venom. Bites are painful and swollen. Variable systemic signs similar to pit viper bites can also occur. Treatment is supportive (fluids, and analgesia). Meperidine should NOT be used for analgesia. Crotalid antivenom has been used, but efficacy is unknown. Prognosis depends on prompt intervention and supportive care.

Toxic Toads

The *marine toad* (*Bufo marinus*) is found mainly in Florida and Hawaii. Though probably less toxic than *B. marinus*, the *Colorado River toad* (*Bufo alvarius*), found in the desert Southwest, causes similar signs in dogs that ingest them. Big and slow moving toxic toads are easy prey for curious dogs and cats. The bufotoxins are cardioactive glycosides that have digitalis like action. Toad venom also contains epinephrine, cholesterol, ergosterol and serotonin. The toxins produced in the parotid glands of the toad are absorbed across the pet’s mucous membranes. Hypersalivation, nausea, and vomiting are seen almost immediately. Dogs may shake their head while others may act disoriented and blind. Seizures have also been reported. The cardioactive glycosides can cause hypertension, ventricular fibrillation, and death. Morbidity rates are high, while mortality is low. There is no specific antidote. The dog’s mouth should be flushed with water. Gastrointestinal decontamination with activated charcoal and an osmotic cathartic may hasten recovery. Cardiac arrhythmias and hypertension can be treated as indicated.
TREATMENT PRINCIPLES IN SMALL ANIMAL POISONING CASES
Tim Hackett, DVM MS, Dip. ACVECC

Introduction

Many compounds which when absorbed or ingested can cause harm to animals and people. Veterinarians are commonly faced with companion animals that have been exposed to these harmful compounds. It is the responsibility of the first clinician encountering these cases to prevent further exposure to the poison, enhance its elimination and provide supportive and antidotal care. We will introduce the general principles of triage and emergency care of poisoning cases. This will include available procedures to stop the exposure, prevent further absorption and hasten elimination of poisons from the patients body.

Initial Assessment

Antidotes are useless if vital organ function is lost. Regardless of the type of poison, an initial assessment must be made of cardiopulmonary and neurologic function and appropriate actions must be taken early to treat any problems identified. Using an “ABC” approach to any sick animal will keep the emergency team focused on life-threatening problems:

AIRWAY – Be sure the animal has a patent airway. A quick oral exam and finger sweep can remove any obstruction. Visually inspect the larynx for swelling or obstruction. If the animal is obtunded, protect the airway by intubation with a low-pressure cuffed endotracheal tube.

BREATHING – Is the animal breathing? Are the breaths of adequate rate and depth? Is oxygen getting from the lungs into the tissues? Observation, auscultation, and examination of the mucous membranes can answer these questions. If the mucous membranes are pale or cyanotic, supplemental oxygen should be provided immediately.

Some poisons can cause respiratory depression and significant hypoventilation. Antiemetics such as apomorphine and xylazine can also result in cardiopulmonary depression. Simply supplementing oxygen can correct the hypoxia associated with hypoventilation however significant hypercarbia (PaCO2 > 50 mmHg) and respiratory acidosis may persist. A ventilator may be required to support the patient until normal function returns. Arterial blood gas analysis is required to identify hypoxia, hypoventilation and acid base disturbances associated with altered respiratory function.

CIRCULATION – Check pulse quality, heart rate and rhythm. If pulses feel weak or irregular, and electrocardiogram should be evaluated. Patients in shock should have an intravenous catheter placed, blood samples drawn and fluids ready to administer. If the heart rate is rapid and the pulse quality weak, Shock volumes of crystalloid fluids should be given (90 ml/kg over an hour in a dog, 40 ml/kg in a cat). Patients should be continually reevaluated and therapy changed as needed. With aggressive fluid therapy we should expect the heart rate to slow, the pulses to become stronger, and organ perfusion to improve. Clinically, the patients color and capillary refill should improve and the animal should act more alert. Packed cell volume and total solids should be rechecked after half of the total shock volume has been given. If the packed cell volume has dropped below 25-30% whole blood, or packed red blood cells may be needed. If the total solids drop below 3.5 gm/dl or less than half of the starting value, consider a colloid such as plasma, hydroxyethyl starch or dextrans.

Neurologic Complications

Partial or generalized seizures can be seen with a variety of poisonings. Generalized (grand mal) seizures are the most serious form and can result in severe hyperthermia, hypoxia, metabolic acidosis, permanent neurologic injury and organ failure. It is important to distinguish true CNS mediated seizures from severe muscle tremors as treatments vary. Short acting fast
acting anticonvulsants (such as Diazepam) are used to control generalized seizures. Phenobarbital is used for longer control and as a maintenance anticonvulsant. For severe seizures needing further control, Propofol (a general anesthetic is titrated to effect). Patients with severe muscle tremors can be managed with muscle relaxants like methocarbamol or guaifenisin. Depressed consciousness, miotic pupils or coma may all indicate increased intracranial pressure. Mannitol is indicated in cases with suspected cerebral edema.

**Treatment goals**

Regardless of the poison, the goals include: 1) Prevent further exposure, 2) Decrease absorption, 3) Hasten elimination and 4) Provide supportive care, and when available, an antidote. Removing poisons or preventing their metabolism to more toxic compounds can prevent further exposure. Thorough bathing for topical poisons and gastrointestinal decontamination for ingested poisons decrease absorption. Ion trapping is another technique to prevent absorption by maintaining substances in an ionic form less likely to pass into systemic circulation. Elimination is hastened with forced intravenous fluid diuresis, the administration of cathartics to decrease intestinal transit and potentially through the use of either hemodialysis or peritoneal dialysis.

**Topical Poisons**

To prevent further exposure and decrease the absorption of topical poisons, the animal should be thoroughly bathed. Large volumes of warm water and a mild detergent should be used to completely remove toxic compounds from the skin and hair of the animal. Acids or bases should not be neutralized as the resulting chemical reaction could lead to local skin burns. Instead, dilution, the pollution solution, should be instituted with copious amounts of warm water. When bathing depressed systemically ill animals, pay close attention to maintaining body temperature and protecting the airway. Obtunded, recumbent patients can quickly become hypothermic.

If the poison is dry or powdered, it is better to brush or vacuum the animal. Wetting dry compounds can make it easier for them to cross the skin and enter systemic circulation. With dry poisons, care should be taken keeping the compound away from eyes and nose of the patient and the medical team.

**Ingested Poisons**

Gastrointestinal decontamination involves either emesis, gastric lavage or both followed by activated charcoal and a cathartic. Gastrointestinal decontamination should be considered with nearly any suspected intoxication. Transit of stomach contents usually takes about 2 hours. Gastrointestinal transit time can vary with the quantity and type of food present in the stomach. If a patient has recently ingested any toxin, forced emesis and gastric lavage are practical ways to remove the toxin, preventing further exposure and decreasing absorption.

**Emesis**

Emesis can remove 40-60% of the contents of the stomach. For animals presented soon after ingesting a toxin, emesis is more effective than gastric lavage in removing stomach contents. Emesis is also likely to be more effective with large stomach volumes, with larger food particles or thick mucus. Emesis is unlikely to be beneficial after 2-3 hours of ingesting a poison.

For vomiting to occur there must be enough material in the stomach to forcibly expel. If an animal has ingested a small amount of a toxic substance, feeding a low fat gruel may be beneficial.
Emesis should not be induced in depressed or weak patients; the risk of aspiration is too great to justify the maneuver. Patients that have ingested caustic compounds such as cleaning solutions, acids, or alkalis should not be forced to vomit. These compounds can burn the esophagus, leading to the formation of strictures. Caustic solutions should instead be diluted with water or milk and activated charcoal solutions. Petroleum products can be very viscous so that emesis and lavage can result in regurgitation, vomiting, and aspiration. Activated charcoal may be indicated in significant ingestion with kerosene and terpentine. In general petroleum compounds are poorly absorbed and pose little threat to life as long as they stay out of the lungs. Care should be directed at avoiding aspiration.

**Gastric Lavage**

As with emesis, gastric lavage is only going to be effective early in the management of intoxication. With stomach transit, the recovery rate drops as more time elapses. In one study, only 8% of barium sulfate was recovered 60 minutes of ingestion. Gastric lavage is performed using a large bore, fenestrated stomach tube and large volumes of tepid water. The patient should be sedated or anesthetized and the airway protected with a cuffed endotracheal tube. Water is instilled and removed gently until the returning water is clear and free of debris. Volumes of 5 to 10 ml/kg should be instilled at each exchange. Stomach contents should be saved in plastic and refrigerated until a decision is made regarding toxicological testing.

**Activated Charcoal**

Activated charcoal is an excellent absorbent for the great majority of toxic substances ingested by small animals. Destructive distillation and oxidization of the charcoal residue produce “Activated” charcoal with gas at high temperature and low pH. The final product has pores, which increases the binding surface area. The large pores on the activated charcoal stick to ingested material in a non-specific manner, which makes it an effective treatment for almost any intoxication. Keep in mind that these pores will fill with anything so activated charcoal products should not be mixed with food. Food will occupy binding sites on the activated charcoal decreasing its efficacy. Enterohepatic circulation occurs when toxins eliminated in the bile are reabsorbed by the small intestine. Repeating the activated charcoal can interrupt this cycling.

**Fluid Diuresis**

Crystalloid fluids given at a rate high enough to result in urine production of at least 2 mL/kg/hour will optimize glomerular filtration and the clearance of many poisons. These high fluid rates can lead to signs of fluid overload. Central venous pressure (CVP) offers an objective measure of the compliance of the right-sided circulation. Volume overload to the left side of the heart is harder to measure objectively. Serial thoracic auscultation for the presence of pulmonary edema should be performed frequently so that fluid rates can be adjusted and diuretics administered before an iatrogenic pulmonary edema becomes a problem.

**Miscellaneous treatment and supportive care**

Ion trapping takes advantage of the fact that the ionic form of weak acids and bases will not cross cellular membranes. Ammonium chloride, an acidifying compound, can trap weak bases such as strychnine in the urine. Alkalization with sodium bicarbonate may be useful in eliminating weak acids such as salicylates and ethylene glycol.

Dialysis. Small water-soluble drugs and poisons with low protein binding are ideally suited for removal by dialysis. Some of these compounds (e.g. ethylene glycol) are not readily bound by activated charcoal so that dialysis is an alternative worthy of serious consideration by the emergency clinician.
While hemodialysis is now being used successfully in veterinary medicine, the manpower, expertise and equipment required limit its use to specialized referral institutions. Peritoneal dialysis involves relatively simple equipment and although time consuming can be performed in most any practice.

**Supportive Care**

Many toxins result in respiratory and cardiovascular depression. Patients should be continually monitored for adequate oxygenation and ventilation. Arterial blood gas analysis, pulse oximetry and close attention to mucous membrane color should all be performed in recumbent patients.

A closed urine collection system should be employed to assess urine output, prevent soiling and prevent the reabsorption of toxins from the urine. Urine production less than 1 ml/kg/hour suggest either inadequate fluid therapy or early renal failure. Close attention to this objective value will alert the clinician to serious renal problems while they may still be corrected.

Changes in packed cell volume, and total serum solids may require blood transfusion or other colloid support. To maximize oxygen delivery, hemoglobin concentration, intravascular volume and cardiac output should be optimized. Fluids should be changed based on serum electrolytes, total solids, packed cell volume and hemoglobin concentration to optimize oxygen delivery to the tissues.

Once everything has been done to prevent absorption and hasten elimination of any poison, intensive monitoring and attentive nursing care will provide the patient the time needed to recover from the toxic insult.
Topical Poisonings

Topical insecticides either overdosed or used for species for which they were not intended are common presenting complaints to emergency practices. Organophosphate and pyrethrin containing insecticides are the most common. The organophosphates cause a variety of clinical signs including hypersalivation, lacrimation and bradycardia. The pyrethrins can also cause salivation and muscle twitching. Although there is an antidote available for organophosphate poisoning, treatment is mainly supportive. The patient should be thoroughly bathed, the airway should be kept clear and respiratory system assessed to determine the need for oxygen or ventilatory support. Tremors and seizures controlled with muscle relaxants and anticonvulsants. Body temperature should be monitored and addressed if either hypo or hyperthermic.

Ethylene Glycol

Ethylene glycol, an active ingredient in most antifreeze is also found in photographic chemicals. Because it can cause irreversible kidney damage if left untreated, astute recognition of the early signs of ethylene glycol poisoning is the only chance these animals have to survive. Ethylene glycol intoxications can present in several ways. Early symptoms are due to the ethylene glycol itself. An alcohol it causes neurologic symptoms like a wobbling ataxic, gait and drunken disposition. Animals in this early stage have a reasonable prognosis. As the ethylene glycol is metabolized by the liver to oxalic acids, calcium crystals deposit in the kidney’s leading to irreversible renal failure. These metabolites also cause life-threatening acidosis. Animals presented in the later stages demonstrate symptoms consistent with acute renal failure. Lethargy, vomiting, anuria and azotemia are all seen in the late stages. When ingestion was witnessed, aggressive gastrointestinal decontamination is indicated. Intravenous fluid and antidotal therapy with either 20% ethanol or 4-methylpyrazole should be started immediately.

Anticoagulant Rodenticides

Most rat and mouse bait products use warfarin-like compounds as their active ingredient. They kill by interrupting the normal coagulation pathway causing uncontrolled hemorrhage. Mice and rats will eat the bait and begin to bleed internally within several days. Dogs can also eat enough bait to bleed internally. However, it does take several days for the poison to act. If days have passed since ingestion, coagulation testing will reveal prolonged activated clotting time, prothrombin time (PT), and partial thromboplastin time (PTT). If an owner witnesses an ingestion and calls, you can reassure them that with appropriate care and antidotal therapy the animal will be just fine. Animals that ate the bait within 2-3 hours should have standard gastrointestinal decontamination performed. After assessing how much bait was ingested and how much was removed, the decision to give the antidote (Vitamin K1) can be made. Treatment should be continued for several weeks. Retesting coagulation function is mandatory after the vitamin K therapy is discontinued. A PT should be done 36-48 hours after the last vitamin K was given. Prothrombin time is the first coagulation test to be prolonged. Some of these compounds have extremely long half-lives and clinical relapse can occur if therapy is discontinued too soon. Animals presented days after ingestion may be bleeding from their nose and mouth, into their lungs, or the pleural space.

Bromethalin Rodenticides

In cases of acute ingestion of bromethalin, therapy should primarily be directed towards gastrointestinal decontamination with emesis induction and the administration of repeated doses of activated charcoal (3-5g/Kg orally QID x 24hrs) the first dose of which should be combined with a catharticb. There is some experimental evidence that the administration of Gingko biloba
(100mg/Kg) may attenuate some of the brain pathology seen in experimental animals (rats) administered a toxic dose of Bromethalin. Once clinical signs are present, seizure treatment and prophylaxis are recommended along with medications like mannitol +/- loop diuretics to attempt to combat cerebral edema. Corticosteroids have also been administered to dogs with cerebral edema. A poor prognosis is given for animals with severe clinical signs. Dogs with the syndrome of ascending paresis / paralysis should be treated supportively while awaiting recovery.

**Cholecalciferol Rodenticides**

In cases of acute ingestion of cholecalciferol, therapy should primarily be directed towards gastrointestinal decontamination with emesis induction and the administration of repeated doses of activated charcoal (3-5g/Kg orally QID x 24hrs) the first dose of which should be combined with a cathartic. If there was any possibility of prior ingestion, a serum biochemical profile should be performed to assess calcium, phosphorus, BUN, and creatinine concentrations. In cases with azotemia or hypercalcemia (total or ionized), saline diuresis and loop diuretic therapy should be instituted. Corticosteroids (prednisone) may decrease absorption of calcium from the gastrointestinal system, and resorption from the bone and kidney. Pamidronate disodium is a bisphosphonate compound that lowers serum calcium concentration very effectively through inhibition of osteoclastic bone resorption. A single dose of 1.3-2.0mg/Kg over two hours will decrease calcium concentration to normal within a maximum of 48 hours and will last for 5-7 days. Doses may need to be repeated as dictated by monitoring serum calcium concentration. Calcitonin (4-6IU/Kg every 2-3 hours) of salmon origin is also a treatment modality for lowering serum calcium concentration through increased renal excretion and inhibition of osteoclastic bone resorption. Use of pamidronate disodium has supplanted routine use of calcitonin due to its longer duration of action and because tolerance to the effects of pamidronate have not been routinely observed. Despite its initial expense, pamidronate may be the most cost-effective treatment because once it has normalized calcium concentration, the patient can often be weaned from other medications and supportive care once concurrent problems have resolved (azotemia).

**Ant and Roach Baits**

These products contain a variety of insecticides. However, the main ingredients in most of these products are peanut butter, lard, and jelly to attract the insects. Therefore, ingestion of these products is rarely a concern unless a very tiny animal such as a pocket pet is involved. The two exceptions are when the product contains chlorpyrifos or arsenic. Cats are exceptionally sensitive to chlorpyrifos, an organophosphate, and should be decontaminated via emesis and activated charcoal if ingestion occurs. A few products contain arsenic, which is a stomach irritant. Therefore, most animals will vomit spontaneously, decontaminate themselves, and not require further treatment. If the animal does not vomit, then emesis should be induced and the animal monitored for further problems. Activated charcoal does not bind well to arsenic and should not be given.

**Chocolate**

Chocolate contains two forms of methylxanthines, theobromine and caffeine, and their amounts vary with the type of chocolate. Unsweetened baking chocolate contains almost seven times more theobromine as milk chocolate, while white chocolate contains very little. The LD$_{50}$ of caffeine and theobromine in dogs and cats is approximately 100-200 mg/kg.

Methylxanthines cause CNS stimulation, tachycardia, and vasoconstriction. Signs seen with chocolate toxicosis include vomiting, diarrhea, hyperactivity, polyuria, polydipsia, lethargy, tachycardia, cardiac arrhythmias, seizures, and death.
Decontamination by emesis or gastric lavage is recommended. Repeated doses of activated charcoal and IV fluids can also increase elimination. Methylxanthines can be absorbed through the bladder wall; therefore, a urinary catheter can enhance elimination. Cardiac abnormalities are treated symptomatically. Seizure control may also be needed.

Cleaning Products
Most household cleaning products contain acidic or alkaline ingredients, which can cause caustic or corrosive lesions, respectively, in the GI tract. Fortunately, most exposures occur after the product has been diluted in a bucket of water or a toilet bowl and so only cause mild vomiting.

If the product is not dilute, it can cause severe burning of the mouth, esophagus, and stomach. Lesions from acids usually appear soon after exposure, while lesions from alkalis may not appear until 8-12 hours later. Do not induce vomiting because further damage will occur to the esophagus. Activated charcoal does not bind to these products and should not be used. The best course of action is to dilute with milk or water and maintain the animal on GI protectants for several days. If the animal becomes depressed and anorectic, evaluate it for esophageal and/or stomach ulcers.

Glues
Most glues do not cause problems when ingested by pets. Superglue can glue the lips or tongue together, but otherwise is digested without incidence. An exception is Gorilla Glue, which expands in the stomach and can require surgical removal.

Liquid Potpourri
Liquid potpourris may contain essential oils and cationic detergents. Essential oils can cause mucous membrane and gastrointestinal irritation. Severe clinical signs can be seen with potpourri products that contain cationic detergents. Dermal exposure to cationic detergents can result in erythema, edema, intense pain, and ulceration. Ingestion of cationic detergents can lead to tissue necrosis and inflammation of the mouth, esophagus, and stomach. Following dermal exposure, the animal should be bathed with a mild liquid hand-dish detergent or a noninsecticidal pet shampoo. For ocular exposure to liquid potpourri, the eyes should be flushed thoroughly with tepid tap water or physiological saline. Initial treatment of ingestion of cationic detergents includes oral dilution with milk or water. Supportive care including gastric protectants, analgesics, nutritional and hydration support may also be required.

Zinc
US pennies minted after 1982 are composed of copper plating around a zinc core. The total content of zinc is 97.6 to 99.7% and the weight of the penny is 2.5 grams. Therefore one penny would contain 2,440 mg of zinc and toxicosis has been reported as a result of ingestion of pennies. Subacute or chronic zinc toxicosis can affect the renal, hepatic, and the hematopoietic tissues. Zinc can cause hemolytic anemia, which could lead to hemoglobinemia and/or hemoglobinuria. Treatment of penny ingestion involves removing the pennies from the stomach, which often requires surgery. Supportive care includes blood transfusions, intravenous fluids, and gastrointestinal protectants as needed. Zinc can be chelated with calcium EDTA at 25 mg/kg SQ QID for 5 days.

Metaldehyde
Metaldehyde is one of the active ingredients in slug and snail baits (molluscicides). They come in a variety of forms and may be mixed with other toxins. Metaldehyde and its conversion
to acetaldehyde lead to a metabolic acidosis. The exact mechanism is unclear although reduced levels of gamma-aminobutyric acid (GABA) may play a role in subsequent muscle tremors. Signs associated with metaldehyde ingestion include anxiety, tachycardia, tachypnea, ataxia and severe muscle tremors. With increased muscular activity, there is an increase in body temperature giving this poisoning the nickname of “shake and bake” syndrome. Although any seizure activity can produce the same signs. Patients with muscle tremors from metaldehyde can be distinguished from those with seizures because they tend to remain aware of their surroundings while seizing animals generally seem to lose consciousness. For this reason typical anticonvulsants (diazepam and phenobarbital) are not as effective as a muscle relaxants (methocarbamol or guaifenesin). Treatment of metaldehyde poisoning involves gastric decontamination. If patients are obtunded or unable to swallow, further sedation, endotracheal intubation and gastric lavage should take place instead of forced emesis. Animals should remain overnight with close attention to respiratory function, body temperature and ongoing muscle tremors. Blood work and follow up is strongly recommended as other poisons may be present in these products causing damage to other systems including the liver.

**Non-Prescription Analgesics**

Over-the-counter pain relief products designed for people can easily overwhelm a small animals system. Nonsteroidal anti-inflammatory drugs (NSAIDS) decrease pain by preventing the production of prostaglandins. Unfortunately, several prostaglandins are required to maintain a normal gastric barrier and maintain perfusion of the kidney. For this reason, gastric ulceration and renal failure are two common complications of NSAID overdose. When an animal was seen ingesting large amounts of any NSAID, quick gastric decontamination is advised. Gastrointestinal protectants and intravenous fluids are also recommended. Patients may present with severe gastrointestinal ulceration and even perforation or in acute renal failure. Therapy is supportive and if necessary surgical. Patients should be monitored closely for excessive blood loss and signs of either renal failure or peritonitis. Client education and prevention is the most important aspect to these cases. People should only give medications to their pets AFTER consulting a veterinarian. Newer products like ALEVE® should never be given to dogs or cats. Cats should only be given anti-inflammatory medication under veterinary supervision as they have more problems metabolizing these compounds than dogs.

Acetaminophen is an analgesic but not an NSAID. Because cats lack glutathione and the enzyme glucuronyl transferase, acetaminophen is metabolized differently. Cats will accumulate toxic molecules that result in methemoglobinemia and cell death. The blood becomes dark and the cats become dyspneic and develop facial edema. Depression can give way to coma and death. Immediate gastrointestinal decontamination is needed if the ingestion was within 2-3 hours. Clinical signs develop rapidly and supportive care is started with N-acetylcysteine (Mucomyst). By providing glutathione precursors, the acetylcysteine allows for the formation of non-toxic metabolites. Vitamin C (ascorbic acid) can be used to convert methemoglobin to hemoglobin. In-hospital nursing care with frequent monitoring of vital signs and, if necessary, oxygen and fluid support, should continue until clinical signs of intoxication have resolved.
Hypophosphatemic Diabetic Ketoacidosis

For the small animal emergency practitioner, diabetic ketoacidosis (DKA) is one of the most common endocrine emergencies. DKA can often present either as an animal that is found to be insulin-dependent diabetic after presenting in a ketoacidotic crisis or as a known diabetic animal with poorly regulated disease that becomes ketoacidotic because of inadequate insulin therapy.

When the renal threshold of glucose is exceeded, the resulting glucosuria causes an osmotic diuresis and primary polyuria. With progression of the disease, even the secondary polydipsia is insufficient to maintain fluid balance and severe dehydration results. With insulin deficiency, the body is unable to use glucose. This relative lack of calories to insulin-dependent tissues stimulates the mobilization of fat. Increasing levels of glucagon activates hormone-sensitive lipase, which mediates the transport of long-chain free fatty acids (FFA’s). In the presence of excess glucagon, FFA’s are oxidized in the liver to produce the ketone bodies β-hydroxybutyrate, acetone, and acetoacetate. In the presence of insulin and glucose substrate ketone bodies are metabolized in peripheral tissues to form carbon dioxide and water, which together can form bicarbonate. In the absence of insulin, ketone production exceeds metabolism, the result is ketonuria and metabolic acidosis. Emergency treatment of DKA is aimed at correcting the severe dehydration, hyperkalemia and metabolic acidosis. A central intravenous catheter should be placed in anticipation of a large fluid requirement. The added advantage of a large bore (16-18 gauge) jugular catheter is the ease with which multiple blood samples can be collected over time. These patients should have blood glucose determinations no less than every 2 hours. Intravenous fluid resuscitation is initiated with 0.9% NaCl. Potassium will need to be supplemented but this should wait until the acidosis and perfusion problems are corrected. The hyperglycemia and ketoacidosis should be addressed with regular insulin. Regular insulin will result in the most rapid ketone metabolism and should be continued until the urine tests negative for ketones.

As the insulin and dextrose act to correct the hyperglycemia, the combination also drives potassium and phosphorous into the cells. The resulting drop in potassium and phosphorous should be addressed in the maintenance fluids as therapy continues. Hypokalemia can result in weakness while severe hypophosphatemia can result in hemolysis. At Colorado State University, we find that most DKA patients require phosphorous supplementation soon after initiation of insulin therapy. Phosphorous levels should be checked throughout the hospitalization and supplemented early and aggressively. Anemic patients or those with hemolytic serum are assumed to be hypophosphatemic. Phosphorous can be supplemented using potassium phosphates at a dose 0.03 to 0.12 mmol/kg/hour. Note the concentration of potassium contained in the potassium phosphates. In many instances no further potassium will need to be added to the fluids.

Atypical Hypoadrenocorticism

Adrenocortical insufficiency can cause profound shock and a history of vague, but severe, gastrointestinal problems. Typical or primary hypoadrenocorticism presents with both mineralocorticoid and glucocorticoid deficiency. Atypical or secondary hypoadrenocorticism involves only glucocorticoid deficiency. Glucocorticoid deficiency by itself may be more difficult to identify. Glucocorticoids are important hormones in the body’s battle with daily stress. Cortisol is an important counter regulatory hormone to insulin. Patients deficient in cortisol are often hypoglycemic. Cortisol is also important in cellular integrity especially within the gastrointestinal tract. Many of the non-specific gastrointestinal signs associated with
hypoadrenocorticism are related to the glucocorticoid deficiency. A normal blood count is another interesting finding in patients with a glucocorticoid deficiency. An affect of normal cortisol release during times of stress is a lymphopenia, eosinopenia and neutrophilia referred to as a stress-leukogram. Animals in a hypoadrenocortical crisis should have a profound lymphopenia and neutrophilia. A normal leukogram in this setting should alert the clinician to possible glucocorticoid deficiency and secondary (atypical) hypoadrenocorticism. Therapy for secondary hypoadrenocortical crisis consists of volume and glucocorticoid replacement and treatment of gastrointestinal hemorrhage. Emergency treatment should include aggressive intravenous fluid therapy with 0.9% NaCl, and administration of a rapid acting corticosteroid. The choice of steroid is important to prevent problems with definitive diagnosis. A Corticotropin (ACTH) stimulation test should be performed as part of the admission database. Since the test involves drawing blood samples for serum cortisol levels at 0 and 1 hour post aqueous corticotropin (0.5 U/kg intravenously), the steroid chosen for replacement should not cross react with the cortisol assay. Dexamethasone sodium phosphate (0.5 to 2.0 mg/kg) is the ideal choice. It is an inexpensive, fast acting steroid which, unlike prednisone sodium succinate, does not interfere with the cortisol assay.

Critical Illness-Related Corticosteroid Insufficiency

Organ dysfunction and organ failure can occur in any system within the body. After many years and many thousand patients treated with corticosteroids for shock and trauma, meta-analysis of the hundreds of scientific reports have generally failed to support the use of corticosteroids for many emergent and critical patients. It’s use as all but stopped for conditions like head trauma. While the data failed to show benefit for most septic and shock patients, there was usually a small cohort of patients that clearly showed benefit from steroid replacement. These patients were not overt Addisonians, but were not showing a normal cortisol release with the stress of their primary disease. The term Critical Illness-Related Corticosteroid Insufficiency or CERCI was coined to explain this phenomenon. It now seems rational to provide physiologic steroid replacement in critical patients not exhibiting the normal release of this very important stress hormone.

- CIRCI
- 0.25 - 1 mg/kg IV q 24 hours Prednisone
  - Daily dose can be divided BID
- 0.04 - 0.14 mg/kg IV q 24 hours DexSP
  - Daily dose can be divided BID
- Taper dose by 25% per day.
Electrolyte Disorders

Hypokalemia is a common finding in critically ill patients. Hypokalemia becomes clinically important when levels fall below 3.5 mEq/L. Hypokalemia can be caused by gastrointestinal or renal potassium loss or through translocation of extracellular potassium into the cells. Gastrointestinal losses commonly occur in patients with vomiting and diarrhea. Anorexia or potassium deficient diets are potential reasons for decreased intake. Renal potassium loss is associated with chronic renal failure, tubular acidosis, intravenous fluid diuresis, and post-obstruction diuresis. Rapid shifts between the intracellular and extracellular compartment can cause changes in circulating potassium. Potassium can be driven into the cells by insulin and glucose administration. Any cause of alkalosis including sodium bicarbonate administration can result in intracellular movement of potassium in exchange for hydrogen ions. Upper gastrointestinal obstruction can result in potassium loss through vomiting and an intracellular shift due from the hypochloremic metabolic alkalosis.

Symptoms of hypokalemia are non-specific and include weakness, lethargy, ileus, and anorexia. Muscle weakness can become so severe with extreme hypokalemia that respiratory paralysis can lead to death. Signs may be referable to the primary disease such as polyuria and polydipsia or vomiting and diarrhea. In cats fed potassium deficient diets, hypokalemia can result in a retroflexion of the neck as well as a stilted forelimb gait.

Potassium can be supplemented orally or added to intravenous fluids. Oral potassium supplementation, especially in cats, is desirable because it is safe, and avoids the dilutional effects of intravenous fluids and further loss of potassium from diuresis. High levels of potassium can be cardiotoxic therefore it is important not to exceed 0.5 mEq/kg/hr when administering potassium-containing fluids. When replacing estimated gastrointestinal losses or giving bolus shock volumes of fluids it is important not to use fluids with extra potassium. Normally, 14 to 20 mEq/L of maintenance fluid is sufficient to maintain normal potassium levels. Patients with pre-existing deficits may require much more. The amount of potassium to add to maintenance fluids should be based on measured serum potassium (Table 6).

Magnesium is essential in the normal sodium-potassium exchange. Hypomagnesemia, another common finding in the critically ill, can lead to refractory hypokalemia. If potassium fails to correct with aggressive replacement therapy, hypomagnesemia should be considered. Because serum magnesium represents less than 1% of all magnesium in the body, there is no easy way to document hypomagnesemia. Magnesium replacement (0.75 to 1 mEq/kg over 24 hours) may result in rapid resolution of the hypokalemia.
UROGENITAL EMERGENCIES
Tim Hackett, DVM MS, Dip. ACVECC

Feline Lower Urinary Tract Disease
Urethral obstruction in male cats is often caused by mucoproteinaceous plugs and uroliths. Magnesium ammonium phosphate (struvite) and calcium oxalate crystals are the more common types seen. With obstruction of the urethra, severe prerenal azotemia, dehydration, metabolic acidosis and hyperkalemia occur.

Immediate therapeutic goals are the relief of the obstruction, drainage of the bladder and reversal of the metabolic derangements. Intravenous fluid support and cardiac protection against, acidosis and hyperkalemia are immediate concerns in the compromised animal. In the obtunded animal, cystocentesis may be necessary to relieve the pressure on the bladder when attempts to pass a urinary catheter are taking too long and the patient is becoming more debilitated. Iatrogenic rupture is possible and these patients should be managed as for uroabdomen.

Life threatening hyperkalemia and acidosis can be managed with either Sodium Bicarbonate (1-2 mEq/kg IV) or Regular insulin (1U/kg) with 2 gm dextrose/U insulin IV. The fluid of choice initially is 0.9% NaCl. After resolution of the acidosis and with treatment of the hyperkalemia, potassium supplementation may be necessary. Serum potassium should be monitored closely and adjusted accordingly.

Indwelling catheterization is advisable in severely affected cats. Soft polyvinyl catheters or argyle feeding tubes are preferred to minimize urethral trauma. Urine should be quantitated in a sterile, closed collection system. The diuretic effect after relief of urinary obstruction can be dramatic. Retained urea, sodium, potassium, phosphate and hydrogen ions stimulate an increased excretion. Intrarenal tubular defects can also impair concentrating ability. Intravenous fluid replacement should be based on urine output plus insensible fluid losses (10ml/lb/day). Close attention to rehydration, body weight and urine output is mandatory to prevent severe dehydration.

Canine urethral obstruction
Obstructive diseases of the canine urinary tract occur with less frequency in the cat but can have the same metabolic complications. The animal may be presented for straining to urinate. If the obstruction remains untreated they can become azotemic, acidic and hyperkalemic. Emergency treatment is identical to the cat with catheterization, intravenous fluid therapy and restoration of normal acid base and electrolytes. Male dogs will frequently obstruct with uroliths at narrow parts of the urethra. One of the most common sites for the stones to lodge is the proximal os penis. Catheterization and retro propulsion can be used to dilate the urethra and allow stone passage out the penis or the stone can be worked back into the bladder for surgical removal. Plain and contrast radiography can help localize the lesion.

Female dogs rarely obstruct with uroliths. It is more common for a slow growing mass lesion in the bladder or urethra to obstruct the flow of urine. A history of chronic hematuria unresponsive to antibiotics is often seen in animals diagnosed with transitional cell carcinoma.

Acute Renal Failure
The most common causes of acute renal failure (ARF) in small animals are nephrotoxic and ischemic injury. Ethylene glycol is the most abundant, tasty nephrotoxin and is much more common than ischemic injury. Iatrogenic ARF can be due to nephrotoxic drugs (aminoglycosides, NSAIDS, amphotericin B, chemotherapeutic agents, radiographic contrast materials) or ischemic injury (hypovolemia, hypotension, vasodilatory therapy). By recognizing
patients at risk (preexisting renal disease or other systemic diseases), volume deficits, electrolyte disturbances and major medical problems can be stabilized before the use of anesthetics, contrast materials or other potentially nephrotoxic agents.

Acute renal failure is a sudden drop in GFR that leads to rapid development of azotemia and uremia. Anorexia, vomiting, diarrhea, dehydration and sudden changes in urine output are seen with ARF. The breath may have a uremic odor and ulcerative lesions on the lips, and tongue develop. Kidneys can swell within their capsule leading to diffuse abdominal pain.

Rapid onset azotemia, hyperphosphatemia, hyperkalemia and metabolic acidosis are seen in oliguric patients. Urine samples may reflect decrease concentrating ability, and the presence of urinary casts with acute renal tubular injury. Early recognition of oliguric ARF and prompt aggressive treatment are mandatory.

Treatment of volume deficits and any underlying disease should begin immediately. Urine output should be monitored closely either by palpation of the bladder, urine collection in a metabolic cage or a catheter and closed collection system. Oliguria (urine output < 1 ml/kg/hour) should be treated initially with crystalloid fluids. The fluid of choice is 0.9% NaCl. Potassium supplementation should be based on serum levels, as hyperkalemia is a common finding.

If following IV fluid diuresis, urine production is still low, pharmacological intervention is indicated to increase urine output. Diuretics and vasodilatory agents are used to enhance urine output. Furosemide, 10% dextrose and mannitol are diuretic agents employed. Low dose dopamine (2-5 µg/kg/min) may improve urine flow.

Furosemide is readily available and given as an intermittent IV bolus (2-3 mg/kg IV every 6-8 hours) it helps increase tubular and renal blood flow without significantly affecting GFR. Dopamine can enhance the effectiveness of furosemide so these two agents are often given concurrently.

Mannitol is an effective osmotic agent but should only be used in rehydrated, normovolemic patients. By creating volume expansion, mannitol increases tubular flow and urine production by improving GFR. Mannitol also has weak free radical scavenging activity and may help minimize swelling of injured renal cells.

<table>
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<tr>
<th>Drug</th>
<th>Action</th>
<th>Dosage</th>
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<tbody>
<tr>
<td>Furosemide</td>
<td>Loop Diuretic</td>
<td>2-3 mg/kg IV q 6-8 hr</td>
</tr>
<tr>
<td>Dopamine</td>
<td>(controversial) Improved renal blood flow</td>
<td>2-5 µg/kg/min CRI</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Osmotic diuretic</td>
<td>0.5-1.0 gm/kg IV</td>
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Table 1. Summary of drugs commonly used to initiate urine production.

When rehydration and pharmacological agents fail to restore urine flow, dialysis is the next step to restore fluid and electrolyte balance in patients with life-threatening fluid overload, hyperkalemia, or metabolic acidosis. Prognosis, underlying disease, animal temperament and financial commitment should all be considered before proceeding with dialysis. Dialysis is based on the interaction of plasma water with a solution across a semipermeable membrane, which allows movement of soluble substances from the plasma into the removable dialysate. Dialysate
solutions are buffered, hyperosmolar crystalloid fluids which pull urea, phosphate, potassium and water from the plasma. These solutions vary in concentration from 1.5% to 4.25% dextrose.

**Uroperitoneum**

Uroperitoneum can be diagnosed by comparing serum to fluid BUN or creatinine. This can be accomplished quickly using calorimetric reagent strips (Azostick) for BUN. If the nitrogen or creatinine level in the fluid is higher than that in the serum, the fluid contains urine and additional contrast studies are indicated to localize the source of the leakage. Uroperitoneum will require surgical intervention but is not a surgical emergency. Patients often have severe electrolyte abnormalities and are in shock. By providing abdominal drainage, dialysis and intravenous fluids, these patients will be hemodynamically stable and able to undergo extensive contrast studies of the bladder and urethra. If evaluation of the lower urinary tract does not reveal a source of leakage, an intravenous pyelogram can be performed on a well-hydrated, non-azotemic patient. Patients with pelvic trauma should have the entire urethra evaluated by contrast studies. Catheters may pass small tears, which will not show up, unless the catheter is withdrawn while additional contrast is injected. Avulsion of the bladder is also seen with trauma. Plain radiographs may show a moderately full bladder sometimes displaced cranial. The presence of a visible bladder on plain radiographs does not exclude bladder avulsion, rupture or leakage.

**Pyometra**

The accumulation of pus within the lumen of the uterus is one of the most life-threatening conditions of the female reproductive tract. The sequestration of massive amounts of neutrophils and inflammatory debris leads to many systemic effects including shock, septicemia toxemia, glomerulonephritis and peritonitis.

Common in middle-aged females, pyometra usually occurs within a month of estrus. Opportunistic vaginal bacteria (usually *Escherichia coli*) ascend into a uterus affected by endometrial hyperplasia. Under the influence of progesterone, local immunity is suppressed, there is decreased myometrial contractility and the cervix is closed. Estrogen will increase the severity by increasing the number of progesterone receptors in the endometrium. This especially evident in bitches given estradiol as an abortifacient.

Any intact female presenting lethargic, anorexic, with vague gastrointestinal signs, polyuria/polydipsia or weight loss should be suspected of having a pyometra. Diagnosis is made through a combination of recent estrus, an inflammatory leukogram (though they may be leukopenic with massive consumption) abdominal distention or caudal abdominal mass noted on radiographs and a septic suppurative vaginal discharge.

Prompt treatment of dehydration and septicemia with intravenous fluids and antibiotics will provide cardiovascular support for an ovariohysterectomy. Surgery should be performed within 12 hours, sooner if the uterus has ruptured. Open-cervix pyometra may be treated successfully with prostaglandin F2-alpha and long term antibiotics.

**Dystocia**

The average gestation period in the bitch is 64 days with a range of 58-72 days considered normal. If ovulation timing has been performed, whelping should occur 56 to 58 days from the first day of diestrus, or 64 to 66 days from the initial rise in serum progesterone and the leuteinizing hormone surge. The average gestation in the queen is 63 to 65 days. The narrow range in the queen results from induced ovulation.

Five to 6% of canine and feline pregnancies require intervention. Animals at higher risk include brachycephalic dogs and short-faced cats. Devon rex cats and Bulldogs are at much higher risk than other breeds. Uterine inertia, the failure to initiate and maintain sufficient uterine
contractions, is the most common cause of dystocia accounting for 2/3 of the dystocias in the bitch and queen. Fetal malpresentation is the most common fetal cause of dystocia. Other maternal causes of dystocia include mechanical obstruction (abnormal pelvic canal), and maternal anxiety. An oversized fetus, fetal monstrosity and fetal death are the other fetal causes of dystocia.

When an expected due date has come and passed, the dam should be evaluated. Even without evidence of maternal distress, early intervention will likely improve fetal survival. A complete physical exam is important to assess the health of the dam. Radiographs will confirm a term pregnancy and are best to assess fetal number, the pelvic canal and fetal presentation. Ultrasound is more sensitive in determining fetal viability.

When a client calls, concerned their pet is having a difficult labor; serious thought must be given before bringing the dam to the hospital. If the dam is in active labor, the stress of transport can lead to a maternal dystocia a fetus in the birth canal is put at risk. However, the sooner indicated medical and/or surgical interventions are performed the more likely a successful outcome. Some of the signs and indications for immediate veterinary care include:

- History of previous dystocia
- Signs of systemic illness in the bitch or queen
- Flank biting or severe abdominal discomfort
- No sign of labor 24 hours after the temperature drops below 100°F in the full-term bitch
- More than 24 hours after the temperature drops below 100°F in the full-term queen
- Hemorrhagic or foul smelling vaginal discharge
- Normal lochial (brown-green) vaginal discharge without a fetus
- A fetus or fetal membranes protruding from the vulva for more than 15 minutes
- More than 4 hours have passed after the onset of Stage II labor (rupture of chorioallantois and contractions)
- Strong, active, nonproductive contractions for more than 30 minutes
- More than 2 hours between fetuses or failure to deliver all fetuses within 12-24 hours (bitch) or 24-36 hours (queen)

When the term bitch or queen is presented to the hospital, every effort should be made to minimize maternal stress. If the bitch or queen is extremely agitated or nervous, low doses of acepromazine (0.1 to 0.25 mg/patient) will minimize anxiety without profound sedation. Indications for emergency cesarean section include:

- Pelvic obstruction
- Oversized fetus
- Fetal malpresentation or obstructions that cannot be manipulated
- Fetal death (ultrasound or Doppler stethoscope)

Attempts to at manual removal are limited to puppies and kittens protruding from the vaginal vault. Use of water-based sterile lubricant and gentle traction with fingers is the safest approach.

Once maternal and fetal obstructions have been ruled out with radiographs, uterine inertia is usually successfully managed with dextrose, calcium gluconate and if necessary, oxytocin. Oxytocin is given at a dose of 1-2 U/kg (maximal dose 20 U) IM in the bitch and 2-4 U IM in the queen. The dose can be repeated at 30 minute intervals. Alternatively 10 Units of oxytocin in a liter of 5% dextrose and water (D5W) will allow intravenous titration of the oxytocin. These
patients must be closely monitored and the drip rate slowed if signs of oxytocin overdose develop (titanic contractions). Oxytocin is given when the contractions are less frequent than expected. Fetal heart rate monitoring using a hang-held Doppler can aid in the decision to use oxytocin (fetal heart rates are normal) and signal the need for an emergency cesarean section (fetal heart rates begin to slow indicating fetal stress).

Calcium gluconate increases the strength of myometrial contractions while oxytocin increases the frequency of the contractions. Calcium gluconate 10% (2-10 ml IV for the bitch, and 1-2 ml IV for the queen) is given for ineffective, weak uterine contractions or after several unsuccessful doses of oxytocin. If the dam fails to produce a fetus with medical management, a cesarean section is indicated.
FREQUENTLY ASKED QUESTIONS SERIES

Center for Companion Animal Studies

What is the outpatient treatment protocol utilized for the treatment of parvoviral enteritis at Colorado State University?

Introduction
Funding to evaluate the study developing this outpatient treatment protocol was provided by Zoetis Animal Health.
This randomized clinical study will be presented as an oral abstract at the American College of Veterinary Internal Medicine Forum, Seattle, WA in June, 2013.
The treatment guidelines provided within this protocol are only to be used under the knowledge and supervision of a licensed veterinarian.
This protocol is not intended to be a substitute for the gold standard of care (hospitalization and administration of fluids/medications intravenously), but rather used as an alternative for clients that cannot afford the recommended treatment protocol.
In the previous study, the survival rates for the standard of care protocol and the outpatient protocol were 90% and 80%, respectively.
Standard of care treatment should be offered and refusal to follow that protocol documented in the medical record prior to offering this as an alternative.
The faculty associated with this outpatient protocol will not assume any responsibility for the outcome or complications associated with the use of this protocol.

Initial Stabilization
Upon presentation to the hospital, all dogs should have an IV catheter placed for intravascular volume resuscitation.
An initial electrolyte panel should be obtained to determine the presence or severity of hypokalemia or hypoglycemia.
Use the standardized chart (Table 1) to determine the intravascular volume loss to be replaced
- Isotonic crystalloid boluses should be delivered over 15-20 minutes, with subsequent reevaluation of cardiovascular parameters.
- Additional IV fluid resuscitation should be performed at the discretion of the veterinarian.
- Based on the electrolyte concentrations, 25% dextrose can be supplemented IV (1-2 ml/kg) based on the presence and degree of hypoglycemia.
After cardiovascular resuscitation and restoration of normoglycemia, the outpatient portion of the study is entered.

Basic outpatient protocol
Start subcutaneous crystalloid fluid therapy immediately after IV fluid resuscitation.
- Normosol-R (120 ml/kg/day) divided TID (40 ml/kg/dose)
- In addition, replace dehydration over 24 hours
Use the standardized chart (Table 2) for determination of hydration status.
Divide the amount of fluids needed to rehydrate the patient by 3, and add that amount onto the maintenance SQ fluid dose for the next 3 doses.
Do not add additives (such as dextrose or KCl) to the crystalloids.
Provide aggressive external warming to help promote absorption of the SQ fluids.
Monitor rectal temperature to maintain ≥ 99 °F.
If part or all of the previous dose of SQ fluids remains at the next treatment, only give partial dose of SQ fluids (subjectively determined) or withhold additional SQ fluids that treatment period.
Cefovin is administered once at 8 mg/kg SQ once while at hospital.
Maropitant is administered once at 1 mg/kg SC q24h for the duration of treatment period.
Syringe feed small amounts of Hill’s a/d q6h (1 ml/kg PO), as tolerated by patient.

Rescue protocols
Rescue analgesia
   o In dogs with visceral pain that is deemed “uncontrolled,” buprenorhine 0.02 mg/kg SQ should be administered as frequently as q6-8h.
   o In the previous study, about 20% of dogs required buprenorphine.
Rescue antiemetic
   o In dogs with nausea that is deemed “uncontrolled,” ondansetron 0.5 mg/kg SQ should be administered as frequently as q8h.
   o In the previous study, about 20% of dogs required ondansetron.

Electrolyte supplementation
Ideally, blood glucose and electrolytes should be checked once daily by the veterinarian.
Glucose supplementation should be provided for dogs that have a BG <80 mmol/L.
   o Dogs should be administered simple syrup (Karo) 1-5 ml buccally, every 2-6 hours.
   o In the previous study, about 75% of dogs required glucose supplementation.
Potassium supplementation should be provided to dogs that have a serum K+ < 3.4 mEq/L.
   o Dogs should be administered oral Tumil-K (0.5-1 tsp per 10 lbs, every 4-6 hours).
   o In the previous study, about 60% of dogs required potassium supplementation.
Glucose and/or potassium supplementation should be continued until the electrolyte abnormalities have resolved and the patient is eating enough on their own to maintain these values within the normal range.
In addition to having their electrolytes checked once daily, dogs should also have a cursory physical examination performed by the DVM once daily.

Failure of the Outpatient protocol
In dogs receiving the outpatient protocol, worsening clinical symptoms warrants that treatment will be switched to hospitalized treatment protocol (to allow for IV catheterization). Criteria for “worsening symptoms” may include the following:
   o Progressive dehydration, defined as loss of ≥ 10% of body weight from admission or ≥ 8% dehydration on two serial measurements, based on physical examination findings.
   o Hyperlactatemia, defined as ≥ 4 mmol/L.
   o Decline in mentation to stuporous/obtunded.
   o Fever, defined as ≥ 104°F.
   o Other subjective criteria that sway the attending clinician towards transition to the Inpatient protocol are the discretion of the attending veterinarian.
   o In the previous study, 5% of dogs on the outpatient protocol were transitioned to the inpatient protocol.
Table 1. **Determination of volume of crystalloids fluids required for IV fluid resuscitation and normalization of cardiovascular parameters.** If required, 6% Hetastarch (5-10 ml/kg) can also be provided as a bolus over 10-15 minutes. Additional isotonic crystalloid boluses can be administered as indicated by the clinical status and at the discretion of the overseeing veterinarian.

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<tr>
<th>Class</th>
<th>Intravascular volume loss to replace (BV = Blood Volume)</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>≤ 15% BV loss (15 mL/kg IV fluid bolus)</td>
<td>Mild ↑ HR</td>
</tr>
<tr>
<td>II</td>
<td>15-30% BV loss (25 mL/kg IV fluid bolus)</td>
<td>↑ HR, ↑ RR</td>
</tr>
<tr>
<td>III</td>
<td>30-40% BV loss (35 mL/kg IV fluid bolus)</td>
<td>↑ HR, ↑ RR, pale mucous membranes, ↑ CRT</td>
</tr>
<tr>
<td>IV</td>
<td>&gt; 40% BV loss (45 mL/kg IV fluid bolus)</td>
<td>↑ HR, ↑ RR, pale mucous membranes, ↑ CRT, cold extremities, mental dullness</td>
</tr>
</tbody>
</table>

Table 2. **Determination of dehydration to be replaced over the first 24 hours.**

Liters of crystalloid to be replaced are determined by multiplying % dehydration by body weight (in kg). As an example, a 12kg dog that is 5% dehydrated would need 0.6L (600 mL) replaced over the first 24 hours. This 600 ml would be divided into three doses (200 ml each), and added onto the maintenance SQ fluid dose to be administered (480 ml + 200 ml = 680 ml), for the next three doses.

<table>
<thead>
<tr>
<th>% Dehydration</th>
<th>Clinical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5</td>
<td>Not detectable</td>
</tr>
<tr>
<td>5-6</td>
<td>Subtle loss of skin elasticity</td>
</tr>
<tr>
<td>6-8</td>
<td>Delay in return of skin to normal position, dry mucous membranes, slight prolongation of CRT</td>
</tr>
<tr>
<td>8-10</td>
<td>Tented skin stands in place, very dry mucous membranes, definite prolongation in CRT</td>
</tr>
<tr>
<td>&gt; 10</td>
<td>All of the above, with definite signs of shock (tachycardia, hypotension, weak pulses)</td>
</tr>
</tbody>
</table>

This protocol is pending publication. The information provided within this document is simply guidelines under which a licensed veterinarian may construct their own outpatient treatment protocol, while taking into consideration the clinical indication and financial situation of a particular pet and owner.