Interventional Radiology (IR) is defined as the use of contemporary imaging and endoscopic modalities to gain access to various anatomic structures to deliver therapeutic agents. The advantages of minimally invasive techniques are well documented in the human medical field, and the benefits of decreased hospitalization and pain and faster recovery will likely be seen in veterinary medicine with improved proficiency in these skills and thorough research is conducted. Interventional radiology and endoscopy also provide alternative treatments to traditional management or even a last option for palliation and improved quality of life with some terminal disease states.

Tracheal collapse & tracheal masses
Medical management is the mainstay of management of tracheal collapse, and may include the use of any combination of cough suppression, anti-inflammatory medications, antibiotics, bronchodilators, and sedation. For dogs with progression of tracheal collapse, medical management may not be able to effectively control clinical signs of airway obstruction, resulting in life threatening respiratory distress.

For patients with intra-thoracic collapse, or patients with cervical collapse where there are increased risks associated with surgical prosthetic ring placement, intra-luminal tracheal stenting provides a rapid, non-invasive, permanent method to relieve tracheal collapse. Advantages of tracheal stenting include it being minimally invasive, surgical dissection is not required, anesthetic time is lessened, and it can treat disease of the intra-thoracic trachea.

The extent of tracheal collapse, including the presence of mainstem bronchi collapse is assessed with fluoroscopy and pre-operative tracheoscopy. At the time of the procedure, a thorough laryngeal examination and endotracheal wash are also performed, as tracheal infections are being more commonly recognized in dogs undergoing tracheal stenting. Stent size is selected under general anesthesia, and the stent is placed through the endotracheal tube using a bronchoscope adapter to permit continued delivery of oxygen during stent placement. Patients are generally monitored in the intensive care unit (ICU) for 24 hours after stent placement. Repeat thoracic radiographs prior to discharge are used to confirm stent positioning and evaluate for the presence of bronchopneumonia.

Lifelong continued medical management for cough control is essential for most tracheal stent patients to prevent complications associated with stents. Complications include stent migration, fracture, progressive collapse in the unstented portion of the trachea, and the development of intra-luminal inflammatory tissue cranial or caudal to the stent ends. Tracheal stents can also be used to increase tracheal lumen diameter in patients with strictures and neoplasia and have been used successfully in both cats and dogs.

Percutaneous antegrade urethral catherization
When retrograde urethral catheterization is not possible, such as in small female dogs, patients with obstructive neoplasia, or patients with urethral tears secondary to trauma, antegrade urethral catheterization performed with the assistance of fluoroscopy can prevent the need for cystostomy tubes or emergency surgery.

With the patient anesthetized or heavily sedated, cystocentesis is performed with an over-the-needle catheter after aseptic preparation of the caudolateral abdomen. Urine is removed for urinalysis and culture (if indicated), and an iodinated contrast agent is injected into the bladder under fluoroscopy to delineate the bladder and urethra. An angled hydrophilic guidewire is advanced through the catheter and into the bladder. Under fluoroscopic guidance, the wire is advanced into the trigone and out the urethra. A urinary catheter is passed retrograde over the wire, positioned appropriately within the bladder, and secured routinely.

Urethral stenting
Intra-luminal or extra-luminal urethral obstructions and compressions can quickly become life-threatening emergencies. For many patients, surgical resection of the cause of the obstruction, particularly in the case of neoplastic obstructions, is not a viable option. Permanent cystostomy tubes are associated with complications and can negatively affect an animal’s quality of life.

The most common indications for urethral stents include transitional cell carcinoma, prostatic carcinoma, urethral strictures, and urethral compression from malignant enlargement of other abdominal organs, such as lymph nodes.

Urethral stenting provides a permanent, non-invasive, comfortable option to relieve urethral obstructions in dogs, and more recently, cats. Stent size is based on contrast retrograde cystourethrogram generated measurements of normal and diseased urethral diameter and length. A repeat contrast cystourethrogram is performed after the stent is deployed to confirm patency of the urethra followed by abdominal radiographs to confirm stent positioning should future comparisons be needed.

The most significant complication associated with urethral stenting is incontinence; with approximately 25-30% of patients being affected by severe incontinence regardless of patient sex or length of urethra stented. Due to the risk of incontinence, the procedure is
Thrombocytopenia, and coagulopathies can develop. Physiologic derangements, particularly in electrolytes, can result from massive transfusion. There are substantial financial implications for clients of patients requiring large amounts of blood products, and the strain placed on a veterinary practice’s blood product supply can impact future patients in need.

**Urinary Obstructive Disease**

Ureteral stenting provides intra-luminal bypass of obstructions by inducing passive ureteral dilation and urine flow through the stent, though this effect is lost as the stent becomes filled with cellular debris over time. Most ureteral stents are made of multifenestrated polyurethane and have a double pig-tail construction such that a coil is in place in the renal pelvis and one is in place in the bladder to prevent migration. Ureteral stents can be placed cystoscopically, percutaneously via access into the renal pelvis, or surgically depending on patient size, sex, and the nature of the obstruction. Due to variations in patient size, ureteral stent sizes have been developed exclusively for veterinary patients.

Subcutaneous ureteral bypass (SUB) devices are an alternative technique for relief of ureteral obstructions. The device is placed via laparotomy; minimally invasive placement is not possible. A locking loop catheter is placed in renal pelvis via access from the caudal pole of the kidney and secured with a dacron cuff and cyanoacrylate glue. A second straight catheter is placed in the apex of the bladder and secured with sutures and cyanoacrylate glue. The catheters are connected to a port placed on the external body wall, which allows future percutaneous access for sample collection and device flushing.

Many azotemic patients experience significant post-obstructive diuresis upon relief of the obstruction and often require multiple days of intensive care and fluid management. Careful management of fluid balance and hydration is essential in these patients to prevent both fluid overload and intravascular volume depletion that can result when there are excessive urinary losses. Due to concerns about infections in patients with urinary implants, urinary catheters are avoided if at all possible in this patient population to avoid ascending infections which have the potential to create a biofilm on the ureteral stent or SUB device.

**Arterial and Venous Obstruction**

Arterial and venous obstructions from clots or invasion of perivascular neoplasms can have catastrophic effect on regional blood supply, such as in aortic thromboembolism, and venous and lymphatic drainage, as with invasive adrenal masses and cranial mediasinal neoplasia. Surgical excision is often not possible in these diseases given the critical nature of the patient and extent of local disease.

Interventional techniques for intravascular delivery of thrombolytic compounds to break down clots, such as tissue plasminogen activator (TPA) and mechanical thrombectomy have been performed in dogs and cats with aortic thromboembolism. Surgical cut-down to the carotid artery is performed, followed by placement of a vascular sheath, and wire and catheter advancement to the location of the clot under fluoroscopic guidance. TPA infusion cranial to and within the clot is performed or mechanical removal via rheolytic thrombectomy is performed to restore flow. Endovascular stenting can be used in combination with these techniques to maintain vessel lumen patency. With arterial obstructions, intensive monitoring for electrolyte imbalances that occur secondary to return of perfusion to a previously ischemic region is essential.

Endovascular stents for venous obstructions secondary to vascular sarcomas and adrenal gland tumors can be placed under fluoroscopic guidance to relieve peripheral edema and ascites, and improve venous return to the heart.

**Transarterial embolization for hemorrhage and neoplasia**

When traditional medical and surgical techniques fail to alleviate hemorrhage, life threatening hypovolemic shock, anemia, thrombocytopenia, and coagulopathies can develop. Physiologic derangements, particularly in electrolytes, can result from massive transfusion. There are substantial financial implications for clients of patients requiring large amounts of blood products, and the strain placed on a veterinary practice’s blood product supply can impact future patients in need.

Selective, transarterial catheterization can be performed via access through the femoral or carotid artery with the aid of fluoroscopic guidance. Hemostasis can be achieved through placement of thrombogenic coils, gelfoam, or polyvinyl alcohol particles. This technique has been used for intractable epistaxis, hemorrhage from gastric ulceration, and traumatic laceration of the genicular artery in a dog with a femoral condylar fracture.

In patients with non-resectable neoplasms, particularly hepatocellular carcinomas, intra-arterial delivery of chemotherapy with or without embolic polyvinyl alcohol particles is an emerging interventional option for tumor management. This technique has also been used for soft tissue sarcomas, tumors of the head and neck, and urinary tract neoplasias. Using arterial access (femoral or carotid), selective catheterization into the arterial blood supply of a given tumor is achieved with angiography and fluoroscopy. Depending on the nature of the mass, collateral blood supply, and other structures being supplied by the artery in question, chemotherapy with or
without embolic particles are delivered under fluoroscopic guidance. Often, a systemic dose of chemotherapy is given locally to increase drug levels within the tumor. Embolic particles are added to achieve stasis of blood flow to the tumor.

Experience with intra-arterial chemotherapy and chemoembolization is still in its infancy in veterinary medicine, though preliminary experiences are promising and clinical trials are being conducted at several institutions to determine safety and efficacy.

References
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Heat stroke is the most serious form of heat-induced illness, with the hallmark clinical sign being central nervous system dysfunction. Heat stroke results when the mechanisms for heat dissipation are overwhelmed by heat load. Cooling measures are the mainstay of emergency treatment, followed by intensive management of hypovolemia, bacterial translocation, coagulopathies, acute kidney injury, and nervous system dysfunction. Prolonged hospitalization, transfusion support, and intensive care are imperative for survival of these critically ill veterinary patients.

Pathophysiology

Heat stroke is defined as “hyperthermia associated with a systemic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates”. There are less severe versions of heat-induced illness, which are often under-recognized in veterinary patients, are heat cramp and heat exhaustion. Heat cramp occurs when sodium and chloride loss causes muscle spasms and tremors. Heat exhaustion is characterized by weakness, fatigue, vomiting, and diarrhea.

Cats rarely suffer from heat-induced illness, therefore the following discussion focuses mainly on dogs. Most dogs present when warm, humid weather begins and becomes less frequent later in the summer, which is likely due to fact that it takes 60 days for full acclimatization in animals. Exertion and exposure to warm, humid conditions does not automatically equate to heat induced illness. Heat induced illness results when the core body temperature increases the heat load in excess of the heat dissipating mechanisms.

The major heat loads for animals are:
- Warm, humid environment (non-exertional)
- Exercise (exertional)

The major mechanisms of heat dissipation are:
- Convection – heat transfer from the body to air as air is moved over the patient
- Conduction – occurs when heat is transferred from patient to a cooler surface via direct contact
- Radiation – body heat release into the environment
- Evaporation – conversion of water from its liquid form to a vapor
- As the ambient temperature increases above 89.6°F (32°C) evaporation becomes most important method for heat dissipation

Factors that diminish heat dissipation include:
- Confinement/poor ventilation
- Upper respiratory disease/anomalies – brachycephalic airway syndrome, laryngeal paralysis, collapsing trachea
- Obesity
- Thick haircoat
- Cardiac disease
- Lack of acclimatization

There are multiple protective mechanisms that are inherent to the physiology of buffering against an excessive heat load. In addition the mechanisms of heat dissipation listed above, other core thermoregulatory responses include tachycardia, vasodilation, and shunting of blood to the cutaneous circulation, all of which are designed to increase heat loss through the skin. In order to increase respiratory heat losses, minute ventilation (minute ventilation = respiratory rate x tidal volume) is increased. Heat shock proteins, which are intracellular proteins to protect and improve enzyme function during heat insults, and both pro- and anti-inflammatory cytokines are important to halt protein denaturation and damage.

Predisposing factors

While any animal can succumb to heat induced illness when their mechanisms of heat dissipation are exceeded by heat load, there are multiple predisposing factors that can make certain animals more susceptible to heat stroke. Since panting and evaporative heat loss is a major mechanism of thermoregulation in dogs, any airway disease that narrows the airway or impedes air-flow is a risk factor for heat injury. Dogs with brachycephalic airway syndrome, particularly laryngeal and palate disease that impedes ventilation, are at risk of heat stroke even in environmental conditions that would not be problematic for dogs with mesocephalic or dolicocephalic facial anatomy. Dogs with laryngeal paralysis, laryngeal collapse, and tracheal collapse are also susceptible to heat stroke from an inability to eliminate heat load due to ineffective panting from airway obstruction. Other disease processes that may impair heat dissipation include cardiovascular disease and neurologic or neuromuscular disease. Obesity and a thick haircoat can decrease radiant and convective heat loss, thereby increasing heat load.
Physical examination

- Temperature may be normal, increased or decreased depending on prior interventions and perfusion. When body temperature exceeds 105.8°F (41°C) permanent brain injury may result.
- On cardiovascular assessment, sinus tachycardia common and ventricular arrhythmias are also possible. The capillary refill time (CRT) may be fast from vasodilation. Peripheral pulse quality is often weak due to fluid losses (panting, vomiting, diarrhea) and vasodilation.
- On respiratory assessment, panting is common and upper airway noise may be heard from a distance or auscultated as referred upper airway noise on lung auscultation. Pulmonary parenchymal harshness and/or crackles may be heard if aspiration pneumonia or parenchymal hemorrhage secondary to coagulopathy are present.
- Mental depression is common, but mentation can vary from normal to comatose on neurologic assessment. Pupil size and PLR may vary and ataxia may also be present in ambulatory patients.
- Vomiting and diarrhea, with or without evidence of gastrointestinal hemorrhage (hematemesis, hematochezia, melena), are common findings on assessment of the gastrointestinal system.
- Urinary system findings can be variable since bladder size will depend on the patient’s last void and will not immediately reflect renal dysfunction. Urinary bleeding may be evident with coagulation derangements.
- Petechia and ecchymoses may be seen in the oral cavity, sclera, or anywhere on the skin secondary to thrombocytopenia or thrombocytopenia.

Common clinicopathologic findings

- Minimum database (MDB)
  - Packed cell volume (PCV) may be increased due to hemoconcentration, decreased from gastrointestinal blood loss, or may be normal due to a combination of these two effects.
  - Total solids (TS) may be increased due to hemoconcentration, due to protein loss through diarrhea, or may be normal due to a combination of these two effects.
  - Blood urea nitrogen (BUN) may be increased from pre-renal azotemia, renal azotemia, and/or gastrointestinal bleeding.
  - Blood glucose (BG) can be decreased from increased utilization during hyperthermia or as an early marker of sepsis.
- Electrolytes
  - Hypernatremia may be seen secondary to free water losses from excessive panting, urinary and/or gastrointestinal losses.
  - Hypokalemia may be seen secondary to gastrointestinal and renal losses.
  - Hypochloremia can be present due to losses from vomiting.
- Lactate may be elevated from decreased perfusion during hypovolemic shock.
- Serum chemistry
  - Increased BUN and creatinine may be present due to pre-renal or renal azotemia.
  - BUN may also be increased with gastrointestinal bleeding.
  - Alanine aminotransferase (ALT) and creatinine kinase (CK) can be increased secondary to muscle injury.
  - Bilirubin may be increased with hepatic dysfunction and associated with cholestasis of sepsis.
  - Cholesterol can be decreased from gastrointestinal blood loss and hepatic dysfunction.
- Complete blood count
  - Increased nucleated red blood cells (nRBC) are present early after heat stroke and are an indicator of bone marrow insult.
  - Thrombocytopenia can secondary to blood loss and/or consumptive processes.
- Urinalysis
  - Glucosuria may be suggestive of recent hyperglycemia and/or tubular injury.
  - Tubular casts on urine sediment can be indicative tubular injury.
- Coagulation screening
  - Prothrombin time (PT) and partial thromboplatin time (PTT) are increased secondary to coagulation factor activation and consumption.

Emergency management

Since several upper airway obstructive disease processes, such as brachycephale airway disease, laryngeal paralysis, and tracheal collapse can impair heat dissipation and lead to heat induced illness, it is important to ensure adequate oxygenation and ventilation.
Heat stroke can be difficult to treat and requires aggressive critical care and monitoring.
The outcome is often dependent on the dog’s prior health status and severity of heat insult. Factors associated with a more poor prognosis include presentation in a coma or progressive neurologic decline, hypothermia on presentation, persistent hypoglycemia, worsening azotemia in the face of fluid therapy or oliguria in the face of appropriate volume resuscitation, evidence of disseminated intravascular coagulation (DIC), refractory hypotension, increased total bilirubin, and ventricular arrhythmias. Hypoglycemia and PT prolongation at presentation were associated with death in another study. Other risk factors for death include creatinine > 1.5 mg/dL after 24 hours of hospitalization, delayed admission (>90 minutes), seizures and obesity. Number of nucleated red blood cells (nRBC) has also been shown to valuable at predicting death in heat stroke patients; >18 nRBC per 100 WBC at presentation had a sensitivity of 91% and specificity of 88% for predicting death in dogs. Mortality rates may be as high as 50% in dogs with heat stroke.

References
Veterinarians working in the ER and ICU are faced with treating hypotensive patients on a daily basis. Depending on the etiology of the hypotension, aggressive fluids resuscitation may not be an appropriate management strategy and the clinician must be familiar with vasopressor therapy options. Alternatively, vasopressors may be necessary in patients who have been maximally volume resuscitated and continue to be hypotensive and/or hypoperfused. Vasopressor agents’ pharmacology, indications, and complications will be discussed.

Blood pressure results from a tightly regulated balance of the neural inputs of the autonomic nervous system, cardiac function, peripheral vascular resistance (SVR) and endothelial signaling, and renal control of electrolytes and plasma volume. Sympathetic input, vasomotor tone, and intravascular volume, through their combined effects on heart rate, stroke volume (SV), and cardiac output (CO), together, determine blood pressure (BP) (BP = CO x SVR). Throughout the cardiac cycle, blood pressure varies resulting in systolic (SAP), diastolic (DAP) and mean arterial pressures (MAP), with the mean pressure being most closely related to diastole, as the majority of the cardiac cycle is in diastole. MAP can be calculated as MAP = ((SAP-DAP)/3) + DAP.

Canine normal blood pressure ranges from 131-150 mmHg (systolic) and 74-91 mmHg (diastolic) using direct, oscillometric, and Doppler technology. In cats, normal blood pressure ranges from 115-162 mmHg (systolic) and 74-91 mmHg (diastolic) using direct, oscillometric, and Doppler technology. In general, the average canine blood pressure is 133/75 mmHg (systolic/diastolic) and average feline blood pressure is 124/84 mmHg (Labato, 2009 and Labato, 2004). Hypotension is generally defined as MAP < 60 mmHg.

Methods commonly used in small animal clinical practice to measure blood pressure include both non-invasive and invasive technologies. For non-invasive blood pressure monitoring, oscillometric and Doppler ultrasonography are readily available. Doppler is particularly useful in small patients, cats, and those with cardiac arrhythmias. Oscillometric methods are convenient as they can be programmed to cycle at pre-determined intervals, such that repeated measurements can be obtained automatically. For both methods, cuff size selection in relation to limb diameter is essential for accurate results. Cuff diameter should be approximately 40% the limb circumference in dogs and 30% in cats. Cuffs that are too large will generate falsely low blood pressure results and falsely high results will be obtained from a cuff that is too small. In hypotensive patients, non-invasive methods have been shown to have the greatest variability as compared to direct measurements. Direct arterial blood pressure is considered the gold standard for blood pressure determination, and offers the additional benefits of continuous, real-time results that are accurate with arrhythmias and decreased perfusion. However, placement of an arterial catheter is technically challenging, especially in hypotensive patients and cats, uncomfortable for the patient during placement, and requires constant monitoring to ensure the catheter is not inadvertently removed. Care must also be taken to ensure that medications are not injected into the arterial catheter. In cats, necrosis of tissues distal to the arterial catheter is a concern and therefore arterial catheters should not be left in place in cats for more than 6-8 hours.

It is important to remember that hypotension is not a diagnosis or disease, but is instead a clinical manifestation of the underlying pathologic process(es). It is also important to remember that many patients with hypotension do not have a single cardiovascular dysfunction responsible for the decrease in blood pressure; their hypotension is often the result of a multitude of contributing factors. For example, a cat in septic shock is may be hypotensive from bradycardia, hypovolemia, and catecholamine deficiency or non-responsiveness resulting in vasodilatation. Clinical signs recognized in most hypotensive patients correlate with end organ perfusion, and include decreased responsiveness, tachycardia, weak or poor pulse quality, cool extremities, pale mucous membranes with prolonged capillary refill time (CRT), and decreased rectal temperature.

Since hypovolemic shock is the most common cause of hypotension in veterinary patients, intravenous fluid therapy to provide intravascular volume resuscitation is the mainstay of treatment for many patients with hypotension. Improvement of intravascular volume status will improve stroke volume, which will in turn improve cardiac output and ultimately blood pressure if hypovolemia is the sole or largest contributor to hypotension (CO = HR x SV and BP = CO x SVR). Catecholamine therapy is rarely needed for patients with inadequate volume resuscitation unless there are other factors impacting blood pressure, such as general anesthesia. For patients with cardiogenic shock due to forward (left sided) or backward (right sided) cardiac pump failure or severe arrhythmias, fluid therapy to correct hypotension may be ineffective or even contraindicated. For these patients, improvement in heart rate, cardiac contractility and/or systemic vascular resistance may be needed to normalize blood pressure. For patients with hypotension that is refractory to fluid therapy, or in those where further volume resuscitation cannot be tolerated (as with fluid overload or severe cardiac dysfunction creating risk for fluid overload), vasoactive catecholamines or vasopressin can improve vascular tone and blood pressure, but may not normalize perfusion due to shunting and excessive vasoconstriction in certain tissues.
Catecholamines

Endogenous catecholamines (epinephrine, norepinephrine, and dopamine) are synthesized from tyrosine, and depending on location, can function as hormones or neurotransmitters. Dopamine is a precursor to norepinephrine. They act through stimulation of $\alpha$- or $\beta$-receptors, and receptor type determines response initiated by the catecholamine. Individual catecholamines have variable effects on each receptor type, and depending on the outcome desired, different catecholamines may be preferable in certain situations.

**Dopamine:** Dopamine’s (Abbott Laboratories, Chicago, IL) target receptor and action is dose dependent. At low doses (1-4 mcg/kg/min), the drug predominantly affects D1 and D2 dopaminergic receptors, resulting in dilation of renal, cerebral, coronary, and mesenteric vascular beds. The efficacy of its use for preservation of renal blood flow and diuresis at this dose range in renal failure is debated. At doses of 5-10 mcg/kg/min, the $\beta$-receptor effects of this drug predominate, resulting in improved inotropy and chronotropy. There is also some $\alpha$-receptor stimulation within this dose range, though resultant vasoconstriction and increased systemic vascular resistance are less predictable. At higher doses of 10-20 mcg/kg/min the $\alpha$-receptor effects are most prominent, resulting in potent vasoconstriction, which may compromise perfusion to the GI, peripheral, and renal circulations. Dopamine has a rapid onset and offset of action, with a plasma half-life of approximately 2 minutes, which is why constant rate infusion (CRI) is necessary. Its metabolism is through monoamine oxidase (MAO) and catechol-O-methyltransferase (COMT) in the plasma, kidney, and liver. Side effects include nausea, vomiting, arrhythmias, hypotension, or hypertension. It can cause soft tissue injury and necrosis, so extreme care must be exercised to prevent extravasation. There are no strict guidelines for incremental dose increases or weaning of dopamine. In general, given the rapid onset of action, improvements in cardiac function or blood pressure should be seen quickly (within 5-10 minutes) at a given dose of dopamine. If insufficient improvement in cardiovascular parameters is seen, the dose is generally increased in increments of 10-25% until desired effect or maximum safe dose is reached, with the goal being to use to lowest effective dose. When cardiovascular parameters have stabilized, dopamine is generally weaned in 2-5 mg/kg/min increments every 30 minutes to hour as tolerated by the patient’s blood pressure. Most vasopressors are less effective over time due to down regulation of adrenergic receptors and higher doses and/or additional vasoactive catecholamines may be needed.

**Dobutamine:** Dobutamine (Lilly, Indianapolis, IL) is more effective at therapeutic doses as a $\beta_1$-receptor agonist than $\beta_2$-receptor or $\alpha_1$-receptor agonist. It does not significantly alter systemic vascular resistance and does not contribute to norepinephrine release. It produces marked increase in cardiac ionotropy, and is therefore useful in patients with inadequate cardiac contractility. However, the increased contractility likely also increases myocardial oxygen demand, which may be detrimental in some patients, especially hypoxicemic animals. Dobutamine has a half-life of approximately 2 minutes, is rapidly metabolized by the liver, and needs to be given as a CRI. Side effects secondary to dobutamine therapy include tachycardia, arrhythmias, and hypertension. In cats, seizures and tremors, particularly at higher doses, have been reported. For this reason, the dosing range for cats is 1-5 mcg/kg/min and the range for dogs is 2-20 mcg/kg/min. Due to the similar half-life and onset of action, the same considerations for dose escalation and weaning as used with dopamine can be used for dobutamine.

**Norepinephrine:** Norepinephrine (Teva, Irvine, CA) is primarily an $\alpha$-receptor agonist with its main effect being increased vasoconstriction. It is able to provide significant vasoconstriction without compromising renal and cardiac blood flow, which makes it particularly attractive as a vital organ perfusion sparing vasopressor. It produces little effect on cardiac contractility and heart rate. The recommended dose is 0.05-2 mcg/kg/min. The same considerations for dose increase and weaning as with dobutamine and dopamine are used.

**Epinephrine:** Epinephrine (Abbott Laboratories, Chicago, IL) has both $\alpha$- and $\beta$-receptor activity of similar magnitude at a given dose, and is most commonly used for cardiopulmonary resuscitation and anaphylaxis in veterinary medicine due to substantial side effects. Epinephrine causes increased cardiac ionotropy and chronotropy, as well as increased systemic vascular resistance. Other effects include bronchial smooth muscle relaxation and increased glycogenolysis. After IV bolus, it can cause significant increase in blood pressure through vasoconstriction, as well as increased oxygen consumption at the capillary level, which can be deleterious with vasoconstriction that effects perfusion. It is rapidly absorbed when given intravenously, however, it can also be given subcutaneously (SQ) or intramuscularly (IM). It is metabolized in the liver and other tissues through MOA and COMT. Side effects include agitation, anxiety, hypertension, arrhythmias, and tissue necrosis at the site of repeated IM or SQ injections. The dose is 0.005-1 mcg/kg/min when used as a vasopressor CRI.

**Phenylephrine:** Phenylephrine (Baxter, Deerfield, IL) is primarily an $\alpha$-receptor agonist with its main effect being powerful vasoconstriction which can result in reflex bradycardia. Vasoconstriction can be significant enough to decrease perfusion to the gastrointestinal, renal, and hepatic vasculature, however, coronary perfusion is often maintained or increased. It has a rapid onset of action, and is generally administered as a CRI even though its effects can persist for up to 20 minutes after injection. Side effects include agitation, bradycardia, arrhythmias, and skin necrosis in cases of vascular extravasation. The dose is 1-3 mcg/kg/min.

**Isoproterenol:** Isoproterenol (Abbott Laboratories, Chicago, IL) is reserved for cases of third degree AV block, as it causes increased conduction rate through the SA and AV nodes. It is a non-specific $\beta$-agonist that causes both improved contractility and heart rate through increased cAMP production. It has little, if any, $\alpha$-receptor effects. Side effects include bronchial relaxation, arrhythmias, tremors, anxiety, weakness, and vomiting. The dose is 0.04-0.08 mcg/kg/min
**Vasopressin**

Vasopressin, which is also known as anti-diuretic hormone (ADH), is a peptide synthesized in the pituitary that has a variety of systemic effects. With respect to vascular tone, vasopressin binds receptors on vascular smooth muscle, and depending on the dose, causes vasodilation (low doses) or vasoconstriction (high doses). Vasopressin stores can become depleted with prolonged shock or sepsis resulting in vasoplegia despite intravenous fluid and vasoactive catecholamine therapy. Vasopressin deficiency has been documented in people with refractory hypotension, and positive benefit has been shown with the addition of intravenous administration of vasopressin. The use of vasopressin has also been described in 5 dogs with dopamine-resistant hypotension secondary to vasodilatory shock and shown to improve blood pressure without causing arrhythmias. Clinical experience in cats is lacking. It is not bound to plasma proteins, and is rapidly degraded in the liver and kidneys. The dose used in dogs is extrapolated from human doses and is 0.5-2.0 mU/kg/min IV CRI. There is no established dose in cats, however, similar doses as used in dogs have been used in cats.

When hypotensive patients fail to respond to fluid therapy to improve perfusion and hemodynamic status, or in patients intolerant of or for whom intravenous fluid therapy is contraindicated, vasopressors are often required to improve blood pressure. It is important to know whether enhancing α- or β-receptor response is preferred for a given patient, depending on whether cardiac contractility or vascular tone needs improvement, as this will impact the decision about which pressor and dose. Vasopressor agents can be associated with serious side effects, and patients receiving these medications require intensive monitoring.

**References**

Tracheal collapse is a common, frustrating disease process in small breed dogs. Signalment, history, and physical examination findings can support a tentative diagnosis of tracheal collapse but localization of disease along the trachea and lower airways may require pointed client questions and diagnostic imaging. Once the extent and severity of disease is understood, medical management is attempted in many cases except those so severely affected that discharge without intervention is not possible. Tracheal stenting has become a popular, non-invasive method for treating tracheal collapse in dogs. Initial experience with tracheal stenting was met with significant complications, leading to it being branded a salvage procedure. Design enhancements, progress in sizing protocols, and improved patient selection criteria has increased the success of their use.

**History and physical exam**

In all dogs suspected to have tracheal collapse, specific questions that should be directed to the client to help better understand the localization of disease and severity include:

1. Duration and progression of clinical signs
2. Nature of cough (honking, dry, moist, soft, expiratory/pushing)
3. How frequent are events and how long do they last
4. Has there ever been respiratory difficulty or distress during or after an event
5. Does a coughing event stop activity, or is the dog able to continue activity despite coughing
6. What are the triggers for a coughing event
7. Does the dog snore and can it sleep through the night
8. Is there a seasonal component to the coughing events
9. What is the home environment like (is there smoking, air conditioning, air fresheners, etc.)

Visual assessment of patient breathing at rest and during a coughing event to evaluate for nature of the cough, prolongation of inspiratory or expiratory phase of respiration, increased respiratory effort, abdominal push on expiration, and herniation of the cranial lung lobes out of the thoracic inlet on expiration should be performed first. Auditory assessment from a distance should assess for nature of abnormal respiratory noises (honking, high pitched, wheezing moist, stertor or stridor). The larynx, trachea, and entire thorax should be carefully ausculted with simultaneous observation of respiratory phase for air movement, fluid sounds, and crackles. The presence of an inducible cough on tracheal palpation is not pathognomonic for tracheal collapse as aggressive palpation can induce patients with normal tracheas to cough and patients with both cervical and intrathoracic collapse may not cough on tracheal palpation.

In animals with tracheal collapse, their diseased airway is not able to withstand deformation due to dynamic airway pressure changes during respiration. In general, patients with cervical tracheal collapse have clinical signs upon inspiration whereas those with intrathoracic collapse are affected on exhalation. Patients with thoracic inlet collapse can have signs during both or either phase of respiration. Patients with mainstem bronchial collapse are affected mostly during expiration.

The remainder of a complete history and physical exam are essential for ensuring there is not another disease process contributing to the cough and to assess for other disease processes which may exacerbate tracheal collapse signs (obesity, endocrinopathies such as hyperadrenalcorticism, chronic lower airway disease, valvular heart disease, etc.)

The ultimate goal of a thorough history and physical examination is to determine the primary source of clinical signs, as many dogs will have a combination of nasopharyngeal, tracheal and mainstem bronchial collapse. Honking dogs with airway obstruction and respiratory compromise need to be differentiated from the chronic coughing patient with no respiratory or activity compromise whom are mainly affected by bronchial collapse as the treatment and intervention recommendations are different in these two groups of patients.

**Diagnostics**

Thoracic radiographs are an important first line diagnostic in patients with suspected tracheal collapse. They are essential to rule out concurrent bronchopneumonia, bronchiectasis, lower airway disease, cardiomegaly, and to assess pulmonary vasculature size. The presence of tracheal collapse on films does not determine severity, the absence of collapse on films does not preclude a diagnosis of tracheal collapse, and thoracic radiographs cannot determine the dynamic nature of the collapse. Paired inspiratory and expiratory thoracic radiographs improves their utility, but can still underestimate severity and extent of disease. Radiographs misdiagnosed the location of tracheal collapse in 44% of dogs and failed to diagnose tracheal collapse in 8% of dogs when compared to fluoroscopy. Thoracic radiographs may not give a complete assessment of mainstem bronchial disease and nasopharyngeal collapse.
Tracheal fluoroscopy is an extremely useful diagnostic for thoroughly assessing the dynamic nature nasopharyngeal, tracheal, and bronchial collapse. It allows for real time understanding of the extent of tracheal collapse during all phases of respiration and during coughing in awake patients. It is especially helpful for determining the presence and extent of mainstem bronchial collapse.

Computed tomography (CT) can be very helpful in dogs with tracheal disease, especially in cases where a tracheal mass is a differential for the cause of clinical signs and when there is concern for severe tracheal collapse leading to ventral tracheal cartilage inversion (tracheal malformation). Unfortunately, dynamic disease processes like nasopharyngeal, laryngeal, and tracheal collapse will not be seen on sedated or anesthetized CT examinations. Studies evaluating a clear plastic patient positioning device (MouseTrap™) that restricts movement have shown promising results for dynamic CT evaluation of upper airway obstruction secondary to laryngeal, tracheal and bronchial disease without the need for anesthesia or sedation. Recent studies evaluating the utility of CT for tracheal diameter measurements to determine tracheal stent sizing show promise for improved measurement accuracy when compared to lateral radiographic or fluoroscopic measurements alone.

Endotracheal washes for cytology and aerobic should be performed in all dogs undergoing intubation for tracheal collapse diagnostics or definitive intervention. Dogs with tracheal collapse likely have abnormal airway clearance mechanisms due to their tracheal disease, so it is possible to have positive airway cultures without radiographic evidence of pneumonia. This is especially important for dogs undergoing stenting since they are managed post-operatively with steroids and cough suppressants, which could exacerbate infection, especially in the presence of a permanent implant. Airway culture and sensitivity is also important in dogs who may have been treated with multiple antibiotics while being medically managed for their tracheal collapse to guide therapy.

Tracheoscopy is a very useful diagnostic in tracheal collapse patients, however, small patient size may mean that some patients must be extubated for tracheoscopy, which can make anesthesia more difficult and dangerous in patients with compromised airways. Tracheoscopy allows for direct visualization of the entire airway and for bronchoscopy if indicated. In patients with suspected or confirmed tracheal collapse where tracheoscopy is being used for staging of collapse and complete airway assessment, the clinician must be prepared for intervention (prolonged intubation, prosthesis ring placement, or endoluminal stenting) should the patient be unable to be extubated due to their disease and/or iatrogenic irritation induced from tracheoscopy. Therefore, in most cases of tracheal collapse, tracheoscopy is reserved for immediate airway assessment before intervention, and in the case of tracheal stenting, immediately post-stent placement to assess position and mucosal contact before recovering the patient from anesthesia.

Medical management
Medical therapy is a mainstay of tracheal collapse management and efforts should be made to attempt medical therapies before surgical or interventional options are pursued. Oftentimes, “breaking the cycle” of dyspnea, distress, and anxiety with sedation, oxygen, cough suppression, and possibly corticosteroids is adequate to control tracheal collapse symptoms enough to permit discharge with medical management institution or adjustment or referral for further surgical or interventional care. However there are cases where respiratory distress cannot be controlled or there is significant patient compromise and immediate relief of the airway obstruction is needed. Prior recommendations that medical management should be “exhausted” before intervening with extra-luminal rings or a stent are falling out of favor due to the negative impacts of long term steroids and improved outcomes with stents.

In the emergency setting when tracheal collapse patients are dyspnic, sedation, anxiolytics, and oxygen are essential. Butorphanol (0.1-0.4 mg/kg IV or IM q2-4 hr) and acepromazine (0.005-0.01 mg/kg IV, IM, or SQ q4-6 hr) are effective initial anxiolytic and sedative options. Acepromazine should only be used patients for whom cardiovascular compromise is not a concern. The use of an oxygen cage where the percentage of inspired oxygen can be adjusted as dictated by the patient’s comfort is also very helpful during emergency stabilization. Corticosteroids may also be considered if there is concern for airway edema and inflammation secondary to respiratory distress and increased work of breathing. Dexamethasone sodium phosphate (0.05-0.1 mg/kg IV q12-24hr) is typically used in these instances.

Hydrcodone and butorphanol are effective antitussives, though it is imperative to educate owners that they better at preventing coughing than stopping an episode, so they should be used regularly in the beginning phases of medical therapy and in some patients, lifelong. Dosing is flexible (0.25-0.5 mg/kg PO q6-12hr) and dependent on patient response and will likely need to be increased over time should tolerance develop. Hydrocodone is available in tablets and an elixir, with the elixir allowing for smaller dose increment changes and titration by the owners. If a prescription for hydrocodone is filled at a human pharmacy ensure that the medication is hydrocodone/homatropine, and not hydrocodone/acetaminophen, which is an additional formulation available for use in people.

The use of corticosteroids such as prednisone in the long-term management of tracheal collapse is controversial. In patients recovering from an episode of dyspnea, a tapering course of anti-inflammatory steroids may be necessary to control the airway inflammation that results from cycling of the collapsing trachea during coughing and edema from increased work of breathing. However, when cough is controlled, airway inflammation from tracheal collapse should be minimal and may not require steroids long term. In addition, long-term steroids contribute to muscle weakness, weight gain and predispose to the development of pulmonary thromboemboli; all of which may exacerbate respiratory signs associated with tracheal collapse. Inhaled steroids such as fluticasone
have been used with some success long-term in dogs with mainstem bronchial collapse when systemic cough control has failed to manage their clinical signs, though clinical studies and data are lacking.

The use of bronchodilators is also controversial in managing dogs with tracheal collapse. Bronchodilators are generally reserved for dogs with confirmed bronchial collapse or concurrent lower airway disease. Methylxanthine derivatives, such as theophylline (10-20 mg/kg PO q12) or aminophylline (5-10 mg/kg IM, IV, PO q8hr) may be preferable to B2 agonists such as terbutaline or albuterol due to risk of tachyarrhythmias with sympathomimetic agents. When oral theophylline is used, the extended release formulation is preferred. Theophylline and aminophylline have multiple drug interactions, so drug compatibility must be confirmed before initiating therapy. Inhaled bronchodilators have also been used with some success as a rescue drug during coughing or long term in dogs with mainstem bronchial collapse.

**Tracheal stenting**

Tracheal stenting can be performed in dogs with both intra-thoracic and extra-thoracic tracheal collapse as well as those with cervical collapse that are deemed poor surgical candidates for extra-luminal rings or for those whom clients are unwilling to accept the complications associated with tracheal ring placement. Since tracheal stenting can be performed quickly and non-invasively, it has been shown to be beneficial in tracheal collapse dogs that present in respiratory crisis that is nonresponsive to medical stabilization. Additionally, dogs who have required hospitalization for a respiratory crisis and whose clinical signs are consistent with airway obstruction can also be considered candidates for stenting after appropriate client counseling about the pros and cons of intervention.

Complications when balloon expandable and human biliary wall stents were used in research dogs and clinical tracheal collapse patients, such as foreshortening, migration, fracture, and excessive airway irritation, led to labeling tracheal stenting as a salvage procedure only, and hence the recommendation to exhaust medical management before considering stenting. Stents designed specifically for the canine trachea (http://infinitimedical.com) have gone through multiple design enhancements to improve their sizing and placement predictability and risk of fracture. Complications seen with tracheal stenting are believed to be greatly reduced when precise sizing is performed. Oversized stents that do not fully expand are at increased risk of fracture since stents are strongest when fully expanded. Alternatively, undersized stents are at risk of poor incorporation into the tracheal mucosa, leading to mucous accumulation, inflammation, and likely infection. Unequal tracheal mucosal contact, in conjunction with airway inflammation and infection, may also lead to granulation/inflammatory tissue formation but objective data correlating these circumstances is lacking.

Current sizing recommendations include positive pressure breath holds to 20 cmH2O to determine maximal tracheal diameter using digital radiography or fluoroscopy. Stent size is chosen by selecting a stent with a diameter that is 10-20% larger than the maximal tracheal diameter measurement obtained. In some cases, the tracheal diameter is not uniform along its length, with the most common scenario being a larger cervical and cranial thoracic inlet diameter compared to the inthrathoracic tracheal diameter. Stent choice in these cases mandates that either the cervical trachea is undersized to accommodate the intrathoracic trachea, or the thoracic trachea is oversized to accommodate the cervical trachea when using traditional tubular stents. Since accurate sizing is thought to be paramount in the success of tracheal stenting, a self-expanding, nitinol stent with a tapering diameter where the cervical tracheal diameter stent portion is larger than the intrathoracic portion was designed. Initial clinical case experience with the tapered tracheal stent (VetStent Duality, http://infinitimedical.com) has been very promising.

Since measurements are performed under general anesthesia at the time of stenting, multiple sizes of tracheal stents should be readily available so that the appropriate size can be placed without having to attempt to recover a compromised patient. Tracheal stents are placed through an endotracheal tube and when a bronchoscope adapter is used, the patient can continue to have oxygen insufflation during stent positioning and deployment.

Patients are often discharged the day following stent placement, unless there is concurrent pneumonia requiring prolonged hospital care. All tracheal stent patients have thoracic radiographs taken prior to discharge. If no pneumonia is present, the first recheck examination is generally one month after stent placement. If pneumonia is present prior to discharge, but patients are only mildly affected and able to be discharged for care at home, recheck radiographs are usually performed 7-10 days post-operatively to ensure appropriate resolution of pneumonia. Patients are discharged with antibiotics (10-15 mg/kg enrofloxacin PO q24) pending airway culture, a 2-3 weeks tapering course of steroids, and regular (q6-8 hour) cough suppression. A short, dry, self-limiting cough is to be expected for 4-6 weeks post-stent placement and is something about which clients should be educated pre-operatively.

Long-term routine thoracic radiographic monitoring is important to be able to detect migration, early fracture or the development of inflammatory tissue. For the first year post-operatively, radiographs are checked every 3-4 months. For every year after that, radiographs are taken every 6 months. If at any point post-operatively, there is a change in the patient’s cough or respiratory comfort, repeat radiographs are taken immediately. If radiographs and/or tracheal fluoroscopy do not reveal the explanation for changes in coughing and respiratory comfort, repeat endotracheal wash and tracheoscopy is indicated. Since tracheal stents are permanent implants, stent fracture causing clinical signs of airway obstruction and coughing can only be managed by placement of an additional stent within the original stent. Very limited experience with obstructive intra-luminal granulation tissue has also shown promising response to immunosuppressive steroid therapy, culture-guided antibiotic therapy, and in some cases, repeat stenting.


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Don’t see emergency cases every day? Have a dog presenting to you with pale mucous membranes, a weak pulse, a heart rate of 190 bpm, and you’re not sure what to do next? Have a dyspneic cat fish-mouth breathing in front of you? This article discusses how to avoid 10 common errors in emergency patients that will save your patient’s life, including when to tap that dyspneic cat’s chest, when to reach for that “FAST” ultrasound, or the best time to do chest radiographs. Having practiced in the trenches of a busy inner-city emergency room to the ivory tower of academia, I’ve seen these mistakes made, and I’ve made them myself. Here, some common mistakes to avoid in the emergency room.

Not doing chest radiographs
One of the most common mistakes in the emergency room is not performing chest radiographs (a “met check”) as part of routine geriatric diagnostics. Geriatric patients (defined as a dog > 6–7 years of age [size-dependent] or a cat > 12 years of age) with, for example, hepatosplenomegaly, icterus, hemoabdomen, immune-mediated disease, or fever of unknown origin should have chest radiographs done at the same time as abdominal radiographs. Typically, a three-view chest set is the method of choice; however, this may be difficult in emergency patients with dyspnea. That said, a right- and left-lateral chest radiograph is also an effective way to screen for metastasis. While a met check is often a “low-yield test” (i.e., the likelihood of identifying chest metastasis is relatively low), it is an important screening tool that can help veterinarians counsel pet owners on end-of-life decision-making and overall prognosis.

Using the shock dose of fluids
The “shock dose” of fluids is extrapolated from the blood volume (60–90 ml/kg for dogs; 60 ml/kg for cats). More recently, emergency critical care specialists have moved away from using the entire shock dose when trying to stabilize hypovolemic patients—smaller aliquots (e.g., one-quarter to one-third of a shock dose) of intravenous (IV) crystalloids are preferred. A patient rarely requires replacement of the whole blood volume with crystalloid fluids.

Using the wrong dose of steroids
Traditionally, “shock doses” of steroids have been listed in emergency books (e.g., dexamethasone sodium phosphate [DexSP] 4–6 mg/kg). However, criticalists have moved away from giving steroids with trauma because of potential deleterious effects (including gastric ulceration in a poorly perfused “shock gut” in the dog, exacerbation of hyperglycemia, and delayed wound healing). More recently, we have moved to different doses of DexSP. Antiinflammatory doses of DexSP are generally considered 0.1 mg/kg, whereas immunosuppressive doses are as low as 0.25 mg/kg IV q 12 to 24 hours. For that reason, the 4-6 mg/kg dose for shock is no longer indicated. Remember that DexSP is approximately 8 to 15 times stronger than prednisone, and one is unlikely to need 40 mg/kg of prednisone in trauma cases.

Clinical application
1. For cases warranting immunosuppression (e.g., immune-mediated hemolytic anemia), consider using lower doses of DexSP (0.25 mg/kg IV q 12–24 hours).
2. Avoid the “shock doses” of steroids—if you are giving more than a few milliliters, it’s probably too much.
3. Concurrent use of nonsteroidal antiinflammatory drugs and steroids should still be avoided to minimize GI effects.

Giving steroids to head trauma patients
Recently, the use of steroids in both human and veterinary head trauma has been widely debated. Although research in this topic is voluminous, there are no experimental or clinical studies demonstrating a clear benefit of steroids in head trauma. In one human study, a meta-analysis of randomized, controlled trials did not show a beneficial response from steroid therapy (1). Unfortunately, steroids have been associated with the following deleterious side effects: gastrointestinal (GI) bleeding, hyperglycemia, immunosuppression, delayed wound healing, and perpetuation of a catabolic state. Currently, the brain trauma foundation guidelines state that glucocorticoids are “not recommended for improving outcome or reducing intracranial pressure in head-injured patients” (2). The “CRASH” (Corticosteroid Randomisation After Significant Head injury) study demonstrated that overall mortality was statistically higher in patients who were treated with steroids (3). Recent studies have shown that human patients with head trauma and hyperglycemia have a poorer return to cognitive function than do euglycemic patients. Why is hyperglycemia dangerous in head trauma, or in any case of brain ischemia? Unfortunately, elevated glucose concentrations provide a substrate for anaerobic metabolism and glycolysis in the brain, worsening brain perfusion via the accumulation of the by-product, lactic acid. Hyperglycemia is also associated with proconvulsant effects, which are due to
increased neuronal excitability. In a veterinary study by Syring and coworkers, 52 dogs and 70 cats with head trauma were compared with 122 age- and species-matched control dogs and cats (4). Severity of head trauma was classified as mild, moderate, or severe, and blood glucose concentrations were recorded within 1 hour after admission (4). The study found that the blood glucose concentrations were significantly associated with severity of head trauma in dogs and cats and were significantly higher in dogs and cats with head trauma than in the control animals. However, blood glucose concentration was not associated with outcome, which is divergent from human studies. This veterinary study may also differ from human medicine in that overall cognitive function varies between humans and dogs/cats. These studies reiterate that iatrogenic hyperglycemia must be avoided in patients with head trauma or cerebral ischemia and that severe hyperglycemia in head trauma should potentially be treated with regular insulin therapy if warranted and persistent. When in doubt, withhold steroid therapy in head trauma patients to prevent hyperglycemia and other detrimental effects. Instead, osmotic agents such as mannitol have been found to be helpful in decreasing intracranial pressure (ICP).

If IV fluid resuscitation alone does not reduce glucose levels in hyperglycemic patients with head trauma, a low dose of regular insulin (0.2 U/kg, intramuscular) may be given every 3 to 4 hours for the first few hours to help lower blood glucose. Blood glucose levels should be monitored frequently to ensure improvement and to preclude hypoglycemia, which would further complicate neurologic monitoring.

Therapies other than steroids to consider in head trauma patients include:
- Aggressive fluid resuscitation to help normalize or maintain blood pressure and maximize perfusion
- Oxygen therapy
- 15- to 30-degree head elevation (to lower ICP)
- Minimal jugular restraint or pressure (to prevent increased ICP)
- Tight glycemic control

Not assessing your patient more frequently with simple tests

In veterinary medicine, the temperature, pulse rate, respiratory rate, and weight are typically evaluated during the initial presentation. These simple, inexpensive physical examination parameters are an important part of serial assessment and often provide clues on hydration status, disease process, and response to treatment.

Temperature

When examining a hyper- or hypothermic patient, differentiate between exogenous and endogenous sources. Hyperthermia is typically caused by an exogenous heat source (e.g., sun exposure, humidity, locked inside a car), upper airway obstruction resulting in lack of ability to thermoregulate. This is semantically different from fever, which is caused by an endogenous heat source (e.g., neoplasia, inflammatory cytokines). With hyperthermia, patients should be cooled by using cold water baths, cold IV fluids, fans, and relieving the upper airway obstruction. Patients should only be cooled to 103.5°F (39.7°C) to prevent severe rebound hypothermia. The use of “fever-breaking” medications (e.g., dipyrone) is not indicated, as resetting of the hypothalamus may have already occurred.

Patients with fever should not undergo cooling methods, as the fever is a physiologic response to an underlying pathology (e.g., viruses, bacteria). Three key differential diagnostics should be considered with fever:

1. Infection
2. Inflammation
3. Neoplasia

For hypothermia, it is important to determine whether it is due to an exogenous source (e.g., living in a cold environment with inadequate shelter, hair coat, or underlying hypothyroidism) or an endogenous one. Hypothermic patients should be warmed passively (e.g., blankets, concurrent warm IV fluids) and slowly. With hypothermic patients, it is important not to rapidly warm patients via surface warming alone (e.g., BAIR hugger), particularly if they are hypotensive, as rewarming can result in peripheral vasodilation. During states of poor perfusion or hypotension, patients should physiologically vasoconstrict peripheral blood flow to direct blood to more important organs—the heart and lungs. Rapid surface rewarming of hypothermic patients without adequate IV fluid replacement can result in inappropriate shunting of blood.

Weight

Weight is often underutilized as a means of assessing hydration. Because we can calculate dehydration (kg weight X % dehydration), we can also estimate appropriate weight gain, as 1 liter = 1 kg. Patients should be weighed daily while hospitalized, ideally on the same scale. This is important because it is an easy way to evaluate hydration and appropriate (or inappropriate) weight gain. For patients in which volume–fluid balance is tenuous (e.g., acute renal failure with anuria, congestive heart failure, volume overload), weight should be evaluated every 6 to 8 hours.

For example, if you determine that a 30 kg dog is 10% dehydrated, the amount of fluid required to hydrate him is:

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

In other words, a 30 kg dog needs 3L of IV crystalloids for rehydration alone and thus should weigh 33 kg after hydration (in 8–12 hours, depending on patient stability). If that same dog weighs 32 kg by the next day, he may still be inadequately hydrated. Likewise,
if the patient weighs 34.8 kg the next day, he may be overhydrated, volume overloaded, and retaining water inappropriately (e.g., acute renal failure).  

Pulse quality
Assessing pulse quality frequently is imperative in unstable, shocky emergency patients. Palpating the femoral pulse enables assessment of pulse quality, which is the difference between the systolic and diastolic pressures. Pulse palpation, quality, and duration are a gross estimate of blood pressure and, indirectly, stroke volume. In a normal healthy animal, the pulses should be strong and synchronous, with a palpable pulse for each heart beat (therefore, make sure that you are simultaneously ausculting your patient and palpating for femoral pulses). A palpable femoral pulse is consistent with systolic blood pressure of at least 60 mm Hg. Poor femoral pulses typically indicate profound hypotension and should be treated aggressively and appropriately. A palpable dorsal metatarsal pulse is consistent with a systolic blood pressure of at least 90 mm Hg, and can be used as a basic “poor man’s Dinamap,” particularly during volume resuscitation.

Patients with systolic blood pressure < 90 mm Hg should be treated with IV fluids (if hypovolemic) and vasopressors (once adequately volume resuscitated) if evidence of shock (e.g., hypovolemic, septic, hyperdynamic, hemorrhagic) is present (provided cardiovascular shock has been ruled out as a differential). Patients with systolic blood pressure > 180 mm Hg (normal, 120 mm Hg) should be treated with antihypertensives, such as hydralazine, nitroprusside, amlodipine or enalapril, to minimize secondary complications from hypertension, such as detached retinas, cardiovascular and renal effects, and ischemic events. Frequent monitoring of blood pressure is imperative to ensure adequate care.

Serial physical examination is imperative to adequately evaluate a patient’s hydration status—checking for return of skin turgor, appropriate weight gain, and moisture of mucous membranes. However, physical examination findings are subjective, and <5% dehydration is subjective and difficult to assess on physical examination. The concurrent use of evaluation of PCV/TS, blood glucose, blood urea nitrogen (BUN or AZO) weight, UOP, and urine specific gravity (USG), and thirst can be used in conjunction with physical examination findings to better assess hydration status.

Packed cell volume/total solids, blood glucose, and blood, urea, nitrogen (BUN/AZO) (“Big 4”)
Patients on IV fluids should have a minimum database (including PCV/TS and blood glucose) measured daily, along with basic electrolytes to make sure Na⁺ and K⁺ are normal. Because patients often experience hemoconcentration when they are dehydrated (e.g., PCV/TS 55%/7.8 g/dl [78 g/L]), the goal of fluid therapy is to ensure that these numbers improve with appropriate therapy (consistent with hemodilution). Ideally, the PCV/TS in a normal, systemically healthy patient on IV fluids at sea level should be 35%/5.0 g/dl (50 g/L). In fact, oxygen delivery is maximal at such a “hemodilute” PCV/TS, as there is less viscosity of red blood cells and “sludginess.” We can still evaluate the PCV/TS in abnormal, metabolically inappropriate patients. Classically, a 10% to 12% dehydrated, cachectic, geriatric cat with chronic renal failure may present to you with a PCV/TS of 28%/11 g/dl (110 g/L). Once that patient is adequately hydrated, the PCV/TS may decrease to 20%/7 g/dl (70 g/L), unmasking the anemia from lack of erythropoietin.

Urine specific gravity (USG)
USG can be evaluated in patients on IV fluids to help assess hydration status. Ideally, USG should be measured before fluid administration to allow for evaluation of renal function. Dehydrated patients with concentrated urine demonstrate adequate renal function (cat > 1.040, dog > 1.025)—in other words, the kidneys are working and trying to absorb as much water from the urine as possible. Once started on IV fluids, normal, systemically healthy patients should have isosthenuric urine. Patients on IV fluids for > 6 to 12 hours should have adequate dilution of USG, and the ultimate goal of fluid therapy and adequate hydration should be USG of 1.015 to 1.018 on IV fluids. Patients on IV fluids with USG > 1.020 are still likely dehydrated and should be treated more aggressively with IV fluids if other parameters of dehydration persist (e.g., hemoconcentration). Hydration can be determined by assessing the color, volume, and USG of urine. A patient that is still dehydrated while hospitalized on IV fluids may have decreased UOP and dark-yellow urine (provided, for example, that no pigmentation, myoglobinuria, or bilirubinuria are present). This is a result of antidiuretic hormone release and renin-angiotensin stimulation, resulting in maximum absorption of free water and sodium.

Urine output (UOP)
UOP should be monitored carefully, particularly in azotemic patients. Fluid therapy should be directed toward achieving normal UOP (i.e., 1–2 ml/kg/hour). Again, one can assess the hydration status of the patient by evaluating the volume and USG of urine. Excessive urination with dilute, clear urine may indicate copious or excessive IV fluid therapy, whereas hypersenturia may suggest ongoing dehydration, and aggressive fluid resuscitation may be further warranted. If UOP is decreased (particularly in azotemic patients), fluid therapy and vasopressor support (to increase renal blood flow) should be initiated to prevent anuria (< 0.5 ml/kg/hour) or oliguria (< 1 ml/kg/hour). If UOP is decreasing and renal function is normal (based on creatinine, BUN, and pre–fluid therapy USG), the patient should be reassessed for hydration status, and fluid therapy adjusted as indicated. Classically, a cat with urethral obstruction may have a profound postobstructive diuresis. A sudden decrease in UOP should elicit assessment for reobstruction. If no obstruction is found, USG should be remeasured. If hypersenturia is found (>1.025 on IV fluids), decreased UOP is likely due to continued dehydration from a postobstructive diuresis—the patient is attempting to absorb as much free water as possible from the kidneys, resulting in decreased UOP. In this example, the IV fluid rate should be increased.

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- Normal UOP: 1–2 ml/kg/hour
- Oliguria: 0.5–1 ml/kg/hour
- Anuria: < 0.5 ml/kg/hour

Note that underlying diseases such as postobstructive diuresis (posturethral obstruction); diabetes mellitus (with secondary osmotic diuresis due to glucosuria); hyperthyroidism (increased glomerular filtration rate due to increased metabolic rate); and chronic renal failure (inability to adequately concentrate and absorb water) may result in dramatic water losses through the kidneys, and these patients may need a higher rate of fluids to compensate for ongoing losses. Likewise, these disease processes prevent us from differentiating renal versus prerenal disease on the basis of USG alone, as these patients have isosthenuria due to metabolic disease. Regardless, appropriate fluid therapy and urine monitoring (e.g., “measuring ins and outs”) may be necessary, particularly in azotemic, oliguric renal failure.

**A water bowl**
Any hospitalized animal should always have access to fresh, clean water unless it is contraindicated due to vomiting, pancreatitis, fasting for anesthesia or sedation, or to maximize mannitol or furosemide effects (fasted for 20 minutes only). If a hospitalized patient on IV fluids continues to drink water in front of you, you should be concerned that the patient is still dehydrated. Due to the timidity of cats, they often will not drink water when stressed and hospitalized. If a dog or cat drinks in your presence, that patient is probably still dehydrated, and their thirst mechanism continues to be stimulated in an attempt to hydrate. Take that as a hint that your patient is trying to tell you to increase the fluid rate! Rare situations when hydration status cannot be based on the thirst mechanism include diabetes insipidus and psychogenic polydypsia.

**Not using enough SQ fluids**
We often use SQ fluids in veterinary outpatient medicine to help hydrate a patient. Because fluids are so slowly absorbed when given in this manner, SQ administration is not appropriate for hypovolemic or severely dehydrated patients. SQ fluids are ideally utilized for outpatient medicine (e.g., the vomiting patient that needs to be fasted overnight but still needs to maintain hydration). But just how much fluid can you give SQ? The calculation for how many ml/kg to give SQ is typically *maintenance fluids*. We do not adjust for dehydration or ongoing losses with SQ fluids.

**Example:**
- 5-kg, male castrated cat presents for 4 episodes of vomiting
  - Physical examination: no string on oral examination, nonpainful abdomen
  - Amount of SQ fluids to potentially give: 5 kg x 60 ml/kg/day = 300 ml SQ

- 40 kg, female spayed Labrador presents for 3 vomiting episodes in 12 hours after ingesting garbage
  - Physical examination: nonpainful abdomen; abdominal radiographs: no significant findings, no obstruction, but some fluid-filled loops of intestine
  - Amount to give: 40 kg x 50 ml/kg/day = 2000 ml SQ

Giving too small of an amount of SQ fluids often does not benefit the patient. Having owners give < 50 ml/adult cat for SQ fluids is often not aggressive enough (not worth the needle poke!). That said, if a patient has a heart murmur (particularly in cats), this maintenance amount should be reduced to prevent volume overload.

**Not doing enough fast (focused assessment with sonography for trauma) ultrasounds**
The focused assessment with sonography for trauma (FAST) ultrasound is a 2-minute procedure that detects the presence of fluid in the abdominal cavity to allow for rapid therapeutic intervention (e.g., fluid resuscitation, abdominocentesis, cytology, clinicopathologic testing) (6). This has also been modified for the pleural (T-FAST) and pericardial space. This rapid method of ultrasound is designed to be used by health care professionals with limited ultrasonographic training and is not designed for extensive examination of the abdomen. The added benefit of the FAST examination is the ability to detect very small amounts of fluid. Typically, 5 to 25 ml/kg of fluid needs to be present to be removed by blind abdominocentesis; > 10 to 20 ml/kg of fluid has to be present before it can be detected by fluid-wave assessment on physical examination; and approximately 8.8 ml/kg of fluid needs to be present before it can be detected radiographically. On the contrary, as little as 2 ml/kg of fluid can be detected on a FAST examination, allowing for rapid diagnosis and identification of underlying pathology.

The FAST examination typically involves assessment of 4 sites of the abdomen: caudal to the xiphoid, cranial to the bladder, and the right and left dependent flank (6). The presence of fluid at any of the sites is considered positive. Evaluation of the xiphoid region allows you to check for fluid between the liver and diaphragm and the liver lobes, as well as for pericardial or pleural effusion (6). Evaluation of the bladder view evaluates for fluid cranial to the bladder and for the presence of a bladder (6). The right dependent flank allows for fluid detection between the intestines and the body wall, whereas the left dependent flank view allows for identification of the spleen, abdominal effusion near the spleen and body wall, the kidney and spleen, and the liver and spleen (6).
Reluctance to penetrate body cavities
The use of abdominocentesis or thoracocentesis is a benign procedure that is both diagnostic and therapeutic. Referring a stressed, hypoxemic, frantic, dyspneic cat with 300 ml of pleural effusion for a 1-hour car ride to a specialist can easily result in the cat’s demise. Shaving and surgically preparing a wide area near the umbilicus (abdominocentesis) or thorax (thoracocentesis) should be done quickly but aseptically. For the thorax, thoracocentesis should be performed either dorsally (for air) or ventrally (for effusion) at the 7th to 9th intercostal space (ICS). Likewise, an imaginary line can be drawn from the end of the xiphoid to the lateral body wall, which is approximately the 8th ICS. This will allow for rapid identification of where to perform an emergency thoracocentesis. Pericardiocentesis should be performed on the right side at the region of the 3rd to 5th ICS at the point of the flexed elbow. Abominocentesis should be aseptically performed via a four-quadrant tap in the periumbilical region. The use of a 3-way stopcock, 20- to 60-ml syringe, extension tubing, and appropriately sized needles dependent on patient size and volume of effusate (usually 20–22 gauge for cats and 16–22 gauge for dogs) is indicated.

Conclusion
Veterinarians should avoid these key, common mistakes in emergency medicine. By avoiding these errors, the overall quality of care and survival of the emergency patient may improve. Simple, easy monitoring tools (e.g., Big 4, pulse quality, weight) can be used to more carefully monitor our critically ill patients in a cost-effective, simple, repeatable manner.

References

NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as Plumb’s Veterinary Drug Handbook.
Gastric dilatation-volvulus (GDV), often known as “bloat” by the layperson, is a life-threatening syndrome seen most commonly in large or giant breed dogs. Immediate recognition, aggressive volume resuscitation, medical stabilization, and surgical intervention are required for best outcome. Life-threatening complications can occur – even with treatment – and include cardiac arrhythmias, sepsis, disseminated intravascular coagulation (DIC), peritonitis, multi-organ dysfunction syndrome (MODS), and death.\(^1\)

**Signalment**

Certain breeds are over-represented or predisposed to GDV and include:\(^1\)
- Great Dane
- Standard poodle
- Gordon setter
- Weimaraner
- Saint Bernard
- Irish Setter
- German shepherds

**Risk factors**

The following risk factors for developing GDV have been previously reported:\(^1\)
- High thoracic depth-to-width ratio
- Older dogs
- Lean body
- Those with a first-degree relative having had GDV
- Fast or “greedy” eater
- Temperament (e.g., nervous, fearful or aggressive)
- Dietary factors
- Dry food
- Feeding of a single large meal
- Raised dog bowl
- Pyloric outflow mechanics
- Abnormal eructation
- Laxity or agenesis of perigastric ligaments
- Gastric foreign body

**Pathophysiology**

GDV can result in acute signs of hypovolemic shock and decreased oxygen delivery (DO\(_2\)) due to cardiovascular compromise. Severe gastric dilatation can cause compression of the intraabdominal veins [e.g., portal vein, splenic veins, caudal vena cava (CVC)], resulting in decreased CVC blood flow and secondary decreased venous return.\(^1\) Likewise, respiratory comprise can occur due to gastric distension and increased intraabdominal pressure; this results in diminished diaphragmatic excursion and decreased total thoracic volume.\(^1\)

**Clinical signs and physical examination findings**

Clinical signs of GDV include
- Non-productive retching
- Agitation
- Restlessness
- Hypersalivation
- Attempting to vomit
- Tachypnea
- Distended stomach
- Tachycardia
- Collapse
Common physical examination findings of GDV include:

- Sprung ribs
- Signs of early decompensated shock (e.g., prolonged CRT, pallor, tachycardia, weak pulses, depressed mentation)
- Tachypnea
- Pulse deficits
- Irregular cardiac arrhythmias
- Tympanic abdomen
- Splenomegaly
- Obtunded
- Comatose

**Diagnosis**

The diagnosis of GDV is typically based on a combination of history, clinical signs, physical examination findings, and a single right lateral abdominal radiograph. On abdominal radiographs, with GDV, the pylorus moves craniodorsal, and is separated by a soft tissue opacity from the gastric fundus (e.g., Popeye sign, double bubble, reverse C). If the radiograph is unclear, a dorsoventral (DV) or ventrodorsal (VD) radiograph may be performed; the pylorus will be found left of midline on the DV view (as compared to right of midline in a normal healthy dog). It is also important to take the time to perform three-view chest radiographs in any dog > 5-7 years of age to help rule out underlying metastasis or pathology. A recent study found that 14% of dogs had evidence of aspiration pneumonia on radiographs, reiterating the importance of chest radiographs in these pre-surgical candidates.

A minimum database (e.g., PCV/TS/BG/AZO and lactate) should ideally be performed on presentation (e.g., during IV catheter placement). Further clinicopathologic testing should include a CBC, biochemistry panel, venous blood gas, and possibly a coagulation panel (if DIC is suspected). Evidence of hemoconcentration and stress-related hyperglycemia (e.g., elevated PCV/TS/BG) are often seen, along with hyperlactatemia (due to hypoperfusion). Severe hyperlactatemia (e.g., initially reported at 6 mmol/L; now 7.4 mmol/L) has been associated with gastric necrosis and nonsurvival in dogs. More recently, Green et al found that initial plasma lactate > 6 mmol/L was not predictive of gastric wall necrosis or survival in dogs; rather, the trend of the lactate level to decrease by > 50% within 12 hours was a good indicator for survival. Further clinicopathologic testing may reveal a stress leukogram, mild thrombocytopenia (secondary to platelet consumption and blood loss), and coagulopathy. Mild elevation in hepatic transaminases (secondary to hepatocellular injury from hypoperfusion) and azotemia (secondary to pre-renal causes) may be seen.

**Treatment**

In the GDV patient, aggressive and prompt treatment should be initiated to ensure the best outcome. Treatment should be directed towards the following:

- Fluid resuscitation
- Gastric decompression
- Surgical intervention
- Treatment of life-threatening complications (e.g., cardiac arrhythmias)
- Analgesia
- Monitoring and supportive care

**Fluid resuscitation**

Depending on the stability of the GDV patient, immediate IV access should be established with two, large bore cephalic catheters. Fluid resuscitation with isotonic crystalloids should be initiated until clinical signs of shock resolve. Small crystalloid aliquots of the “shock dose” should be used (e.g., 20-30 ml/kg, IV) and repeated as necessary. Colloids (e.g., Hetastarch, VetStarch, 5-10 ml/kg, IV) can also be used if the patient fails to respond to multiple crystalloid aliquots. The patient should be stabilized prior to anesthesia and surgery. Following surgery, continued fluid therapy is warranted to maintain perfusion and colloid osmotic pressure (COP) until the patient is discharged.

**Gastric decompression**

Gastric decompression can occur concurrently while the patient is being fluid resuscitated. This author prefers trocharization as compared to orogastric intubation, as it is easy to perform, effective, has minimal side complications, and is less stressful to the patient. The clinician should locate the most tympanic region (estimating where the stomach is) and clip and prep the region. Aseptic technique should be used. A large gauge needle or catheter (e.g., 14 or 16 ga.) should be directed into this area to alleviate gas from the stomach; the sound of hissing gas indicates appropriate placement into the stomach. Rare complications can be seen secondary to trocharization including splenic laceration, gastric perforation, or septic peritonitis. Alternatively, once the patient has been...
appropriate stabilized, orogastric intubation can be performed to aid in gastric decompression. Goodrich et al evaluated dogs undergoing orogastric intubation versus trocharization in 116 dogs and found that orogastric tube placement was successful in 77% of dogs, with 38% of the dogs requiring sedation.\(^7\) In comparison, trocharization was successful in 86% of the cases, with no need for sedation.\(^7\) In the author’s opinion, all dogs should be sedated for orogastric intubation unless comatose or obtunded to prevent undue stress and anxiety to the patient.

**Surgical intervention**

Once the patient has been stabilized, immediate surgical intervention is aimed at decompressing and repositioning the stomach.\(^1\) A complete abdominal exploratory should be performed; viability of the stomach, spleen, and entire gastrointestinal tract (GIT) should be assessed. Lastly, a gastropexy should be performed to prevent future reoccurrence. The readers are referred to a surgical resource for additional information.

**Treatment of life-threatening complications (e.g., cardiac arrhythmias)**

Cardiac arrhythmias have been reported in GDV patients both pre- and post-operatively (11-38%, 50.6-77%, respectively).\(^8-10\) Ideally, continuous ECG monitoring should be performed. There have been conflicting reports of whether the presence of perioperative arrhythmias affects the outcome or prognosis.\(^1\) Regardless, the use of anti-arrhythmics (e.g., lidocaine, procainamide) should be used in the following situations:

- When perfusion parameters are affected by the arrhythmia (e.g., prolonged CRT, poor pulse quality, pulse deficits, hypotension),
- Heart rate > 180 bpm
- R-on-T phenomenon, sustained ventricular tachycardia, multiform ventricular premature contractions (VPC)
- Impaired cardiac output

**Monitoring and supportive care**

As the majority of GDV patients are critically ill, appropriate monitoring (e.g., continuous ECG, blood pressure monitoring, etc.) and supportive care is imperative. Therapy should be aimed towards improving perfusion and oxygen delivery (e.g., crystalloid and colloid therapy). PCV/TS, blood pressure, perfusion parameters, and level of pain should be carefully monitored. Often, the use of gastrointestinal support (e.g., anti-emetics, pro-kinetics, antacids) is warranted to prevent secondary complications (e.g., aspiration pneumonia, ileus, etc.).

**Analgesia**

Once the patient has been stabilized, analgesia can be considered. Ideally, this should be titrated to the lowest effective dose and should be reversible (e.g., opioids). The author prefers opioids for post-operative analgesia (e.g., buprenorphine, hydromorphone, fentanyl CRI, etc.). The use of NSAIDs is not generally recommended due to the severity of gastric injury and poor perfusion to the GIT during GDV syndrome.

**Prognosis**

Overall, the prognosis for GDV is fair to good with rapid recognition, volume resuscitation, gastric decompression and surgical correction. However, other parameters such as hypotension, presenting collapsed, combined splenectomy and partial gastrectomy, DIC, peritonitis, and sepsis have been associated with fatalities.

**Conclusion**

The clinician and veterinary team must be able to rapidly recognize and treat the GDV patient. With aggressive fluid resuscitation, gastric decompression, stabilization and supportive care, the patient will be a better anesthetic candidate for surgical correction and gastropexy.

**References**


NOTE: When in doubt, all drug dosages should be confirmed and cross-referenced with a reference guide such as *Plumb’s Veterinary Drug Handbook*. 

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Leptospirosis, a thin, motile spirochete with a hook-shaped end,¹ is a zoonotic disease that affects wildlife, companion animals, and livestock. There are over 200 serovars of Leptospira, with some being saprophytic while others are pathogenic.¹ Numerous reservoir hosts exist for Leptospira including raccoons, voles, skunks, dogs, pigs, cattle and rats, resulting in growing exposure to humans and to the environment.²

In dogs, leptospirosis is caused by Leptospira interrogans (including serovars icterohaemorrhagiae, canicola, pomona, bratislava, and possibly autumnalis) and Leptospira kirschneri (including serovar grippotyphosa). Prior to vaccines (developed in the 1960s), the most common serovars infecting dogs including L. icterohaemorrhagiae and L. canicola. More recently, different serovars seem to be more associated with canine leptospirosis, including L. grippotyphosa, pomona, bratislava, and possibly autumnalis. Currently, veterinary vaccines provide coverage against L. icterohaemorrhagiae, L. canicola, L. grippotyphosa and L. pomona. With canine leptospirosis, infection with certain serovars are thought to be associated with certain types and severities of clinical disease, although this is not definitive.³ L. pomona appears to result in more severe renal disease and worse outcome (50% as compared to 78-81%) as compared to other serogroups.³

Geographic distribution
The prevalence of leptospirosis is higher in warm, tropical locations with high rainfall.¹ The top geographical locations where humans are diagnosed with leptospirosis include the Caribbean, Latin America, India, Southeast Asia, Oceania, and Eastern Europe.¹ In North America, Hawaii is the state with the highest human cases.¹ In the United States, high antibody prevalence (>1,600) has been seen in dogs from the following regions: Hawaii, West coast states (e.g., northern California, Oregon, Washington), the upper Midwest (e.g., Minnesota, etc.), the Northeast, the mid-Atlantic coastal regions, and other regions (e.g., Texas, Colorado).¹

Risk factors
While canine leptospirosis used to be considered more prominent in large breed, male, working dogs that free roam in rural environments,⁴,⁵ more recent studies have found that urban areas have a growing prevalence,⁶ with smaller dogs < 15 pounds being one of the fastest growing populations of canine leptospirosis.⁷ Of increased concern are studies showing that >20% of dogs may be chronic healthy carriers (based on studies in Michigan).⁸ Additional risk factors for leptospirosis include exposure to slow-moving or stagnant water,¹ conditions where higher rainfall has occurred,¹ late autumn,⁹ exposure to urbanized wild animals,⁸ or rodent exposure. One hypothesis is that global warming has contributed to the growing prevalence of leptospirosis due to the creation of warmer, wetter (e.g., flooding) weather conditions. Likewise, urban sprawl – the invasion of humans into the environment of wildlife - has increased the prevalence of canine leptospirosis.

While rare, leptospirosis has been reported in cats.¹⁰ Serologic evidence of exposure has been confirmed in cats to L. canicola, L. grippotyphosa, and L. pomona.¹¹ Exposure is thought to occur due to rodent exposure,¹ and can result in clinical illness and histopathologic changes consistent with changes seen in dogs with leptospirosis.

Transmission
Pathogenic leptospires are shed from renal tubules of both domestic and wild animals,¹ and can remain viable in the soil and environment for weeks to months.¹¹ That said, leptospires are inactivated by UV radiation and freezing.¹ Infection can also occur through intact mucous membranes or abraded skin with direct or indirect exposure to urine.¹ Rarely, leptospirosis can be transmitted via bite wound, ingestions of infected tissue (e.g., eating raw meat), or by venereal or placental transfer.¹

Clinical signs
Canine leptospirosis classically presents with both acute kidney injury (AKI) and hepatic injury. Clinical signs include:

- Generalized malaise/listlessness
- Inappetence to anorexia
- Vomiting
- Halitosis (e.g., uremia)
- Hypersalivation
- Diarrhea
- Melena
- Icterus
- Febrile
- Dehydration
- PU/PD*
- Abdominal pain (e.g., secondary to AKI)
- Uveitis
- Conjunctivitis
- Oliguria/anuria
- Weight loss

*Note, the polyuria and polydipsia seen with canine leptospirosis may be seen irrespective if the patient is azotemic.\(^1\) This may be due to several causes: impaired renal concentrating ability secondary to a decreased glomerular filtration rate or decreased vasopressin responsiveness of the inner medullary collecting ducts (e.g., acquired nephrogenic diabetes insipidus).\(^1\)

Less common signs include hematuria, vasculitis (e.g., peripheral edema, pleural effusion, ascites, etc.), cardiac abnormalities, abortion (e.g., predominantly reported in cattle), and pulmonary signs.\(^1\) Pulmonary lesions may be secondary to leptospiral pulmonary hemorrhage syndrome (LPHS) or vasculitis.\(^1\) Clinical signs include tachypnea, dyspnea, acute respiratory distress syndrome (ARDS), and pulmonary hemorrhage. With leptospirosis, a secondary coagulopathy may also be seen due to hepatic failure (e.g., decreased production of activated Vitamin K factors II, VII, IX, and X), disseminated intravascular coagulation (DIC), or vascular damage (e.g., presumed to be secondary to the spirochetes). Clinical signs of coagulopathy include:

- Petechial hemorrhage
- Hemothysis
- Melena
- Epistaxis
- Hematochezia
- Hematemesis

**Diagnostic testing**

The diagnosis of leptospirosis is based on clinical suspicion, clinical signs, and clinicopathologic results consistent with leptospirosis. Clinicopathologic findings consistent with leptospirosis include the presence of: neutrophilia, a left shift, lymphopenia, a mild to moderate non-regenerative anemia, hemoconcentration (seen with dehydration), hemolysis (seen with cattle), thrombocytopenia (seen in up to 58% of dogs),\(^1\) azotemia (seen in > 80-90% of dogs),\(^1\) increased liver enzymes (including increases in ALT, AST, ALP, and total bilirubin; these changes are almost always seen with concurrent azotemia with leptospirosis),\(^1\) electrolyte abnormalities (e.g., hypokalemia, hyponatremia, hypochloridemia, hyperphosphatemia), and increased creatinine kinase.\(^7\) Additional findings consistent with leptospirosis include isosthenuria, bilirubinuria, hematuria, glucosuria, proteinuria, and evidence of coagulopathy (e.g., increased fibrinogen, FDP, FSPs). Prolonged PT or PTT may be seen in 6-50% of dogs with leptospirosis.\(^1\)

Other advanced diagnostics may include radiology (to look for evidence of pulmonary lesions secondary to leptospirosis, which may appear as a nodular interstitial or alveolar pattern) and abdominal ultrasound (to rule out other underlying disease processes such as neoplasia, etc.).\(^1\) Ultrasound findings may reveal non-descript findings including renomegaly, pyelectasia, perirenal fluid accumulation, a medullary band of increased echogenicity, increased cortical echogenicity, and rare other findings (e.g., splenomegaly, mild abdominal lymphadenopathy, etc.).\(^1\)

The most “definitive” diagnosis of leptospirosis is typically based on serology by the microscopic agglutination test (MAT), which tests for antibodies to leptospires. The MAT tests for the highest serum dilution causing agglutination of 50% of the leptospires. MAT testing typically includes *L. canicola*, *L. icterohaemorrhagiae*, *L. pomona*, *L. grippotyphosa*, *L. hardjo*, and *L. bratislava*. Unfortunately, there are several limitations of the MAT, including the hazardous need to maintain live cultures of pathogenic serovars, difficulty in standardizing the test, expense, cross-contamination of serovar cultures, and false negatives (e.g., due to acute disease) or false positives (due to previous vaccination, exposure, etc.).\(^1\) Another limitation of the MAT is that some cross-reactivity may occur between different serogroups. Keep in mind that one of the key limitations of MAT is that during the first week of acute disease, dogs may test negative. For this reason, convalescent titters are generally recommended 2-4 weeks later (at the same laboratory) to look for the presence of seroconversion. Typically, a 4-fold increase in titer is suggestive of infection; however, recent vaccination or antimicrobial therapy may affect the results (e.g., antimicrobial therapy may blunt the expected response). Interpreting MAT results must be done with care, as a result. Titters post-exposure can persist for at least one year, but are thought to declined by 4 months secondary to vaccination.\(^1\)

There are other diagnostic tests that can be used to screen for leptospirosis including dark field microscopy, silver staining of biopsy specimens (e.g., renal), immunohistochemistry, PCR, in situ hybridization, culture, and Idexx’s leptospirosis PCR & antibody ELISA in-clinic test. Note that each has their limitations. Dark field microscopy is technically difficult and has low specificity; this has fallen out of favor and is rarely used now. Silver staining of renal biopsy tissue can be performed, but lacks sensitivity and can result in false negatives. Fluorescent antibody testing and PCR can be performed on urine or tissue. Note that culture, PCR, and even
antibody ELISA tests can all be affected by recent microbial therapy. For this reason, pre-treatment blood work should always be utilized for submission in the patient suspected to have leptospirosis.

**Treatment**

Treatment of the leptospirosis patient is aimed towards fluid therapy, antibiotic therapy, gastrointestinal support, supportive care, and monitoring.

**Fluid therapy**

In the leptospirosis patient, aggressive intravenous (IV) fluid therapy is indicated as many patients are often massively polyuric, dehydrated, and azotemic. In general, a balanced, maintenance, isotonic crystalloid (e.g., LRS, Norm-R) can be used at 2.5-4.5X maintenance, and monitoring of ins and outs may be necessary to guide treatment (based on the severity of polyuria seen in patients with leptospirosis). The patient should be assessed carefully to ensure that volume overload does not occur, particularly in patients with cardiopulmonary disease. Fluid therapy should be continued until azotemia and clinical signs resolve (typically 2-4 days); IV fluids should then be slowly tapered to ensure that polyuria has resolved and the patient can maintain hydration.

**Goals of fluid therapy**

Serial physical examination is imperative to adequately evaluate a patient’s hydration status—checking for return of skin turgor, appropriate weight gain, and moisture of mucous membranes. However, physical examination findings are subjective, and <5% dehydration is subjective and difficult to assess on physical examination. The concurrent use of evaluation of PCV/TS, blood glucose, blood urea nitrogen (BUN or AZO), weight, urine output (UOP), urine specific gravity (USG), and thirst can be used in conjunction with physical examination findings to better assess hydration status.

**Packed cell volume/total solids, blood glucose, and blood, urea, nitrogen (BUN/AZO)**

Patients on IV fluids should have daily blood work (including PCV/TS, blood glucose, electrolytes, renal or biochemistry panel) assessed while hospitalized. Because patients often experience hemoconcentration when they are dehydrated (e.g., PCV/TS 55%/7.8 g/dL; 78 g/L), the goal of fluid therapy is to ensure that these numbers improve with appropriate therapy (consistent with hemodilution). Ideally, the PCV/TS in a normal, systemically healthy patient on IV fluids at sea level should be 35%/5.0 g/dL (50 g/L). In fact, oxygen delivery is maximal at such a “hemodilute” PCV/TS, as there is less viscosity of red blood cells and “sludginess.” Note that some patients with leptospirosis may have a mild to moderate non-regenerative anemia; the goal should still be to hemodilute the patient, and total protein/solids should be used as a more appropriate guide in this situation. We can still evaluate the PCV/TS in abnormal, metabolically inappropriate patients. Classically, a 10% to 12% dehydrated, cachectic, geriatric cat with chronic renal failure may present to you with a PCV/TS of 28%/11 g/dL (110 g/L). Once that patient is adequately hydrated, the PCV/TS may decrease to 20%/7 g/dL (70 g/L), unmasking the anemia from lack of erythropoietin.

**Urine specific gravity (USG)**

In normal healthy patients, USG can be evaluated in patients on IV fluids to help assess hydration status. Ideally, USG should be measured before fluid administration to allow for evaluation of renal function. Dehydrated patients with concentrated urine demonstrate adequate renal function (cat > 1.040, dog > 1.025) - in other words, the kidneys are working and trying to absorb as much water from the urine as possible. Once started on IV fluids, normal, systemically healthy patients should have isosthenuric urine. Patients on IV fluids for > 6 to 12 hours should have adequate dilution of USG, and the ultimate goal of fluid therapy and adequate hydration should be USG of 1.015 to 1.018 on IV fluids. Patients on IV fluids with USG > 1.020 are still likely dehydrated and should be treated more aggressively with IV fluids if other parameters of dehydration persist (e.g., hemoconcentration). Hydration can be determined by assessing the color, volume, and USG of urine. A patient that is still dehydrated while hospitalized on IV fluids may have decreased UOP and dark-yellow urine (provided, for example, that no pigmentation, myoglobinuria, or bilirubinuria are present). This is a result of antidiuretic hormone release and renin-angiotensin stimulation, resulting in maximum absorption of free water and sodium. Unfortunately, in the leptospirosis patient, PU/PD may occur due to acquired nephrogenic diabetes insipidus, so utilizing USG as a guideline for hydration status will be difficult.

**Urine output (UOP)**

UOP should be monitored carefully, particularly in azotemic patients with leptospirosis. Fluid therapy should be directed toward achieving a hydrated state and matching ins and outs, based on the patient’s UOP. Note that normal UOP is 1–2 ml/kg/hour, but many of these leptospirosis patients present with severe polyuria. Again, one can assess the hydration status of the patient by evaluating the volume and USG of urine. Excessive urination with dilute, clear urine may indicate copious or excessive IV fluid therapy, whereas hypersenturia may suggest ongoing dehydration, and aggressive fluid resuscitation may be further warranted. If UOP is decreased (particularly in azotemic patients), fluid therapy and vasopressor support (to increase renal blood flow) should be initiated to prevent anuria (< 0.5 ml/kg/hour) or oliguria (< 1 ml/kg/hour). If UOP is decreasing and renal function is normal (based on creatinine, BUN, and pre–fluid therapy USG), the patient should be reassessed for hydration status, and fluid therapy adjusted as indicated.

1. Normal UOP: 1–2 ml/kg/hour
2. Oliguria: 0.5–1 ml/kg/hour
3. Anuria: < 0.5 ml/kg/hour
Note that underlying diseases such as leptospirosis; postobstructive diuresis (posturethral obstruction); diabetes mellitus (with secondary osmotic diuresis due to glucosuria); diabetes insipidus; hyperthyroidism (increased glomerular filtration rate due to increased metabolic rate); and chronic renal failure (inability to adequately concentrate and absorb water) may result in dramatic water losses through the kidneys, and these patients may need a higher rate of fluids to compensate for ongoing losses. Likewise, these disease processes prevent us from differentiating renal versus prerenal disease on the basis of USG alone, as these patients have isosthenuria due to metabolic disease. Regardless, appropriate fluid therapy and urine monitoring (e.g., “measuring ins and outs”) may be necessary, particularly in azotemic, oliguric renal failure.

**Antibiotic therapy**

In the patient suspected of having leptospirosis, prompt, appropriate antibiotic therapy should be initiated (ideally after pre-treatment blood work has been submitted). Goals of antibiotic therapy is to eliminate leptospriemia and to eliminate leptospires from the renal tubular cells and renal carrier state). Appropriate antibiotics include penicillins (e.g., including ampicillin, amoxicillin, amoxicillin/clavulanic acid, penicillin, etc.) and doxycycline. In humans, the use of ceftriaxone and cefotaxime are also efficacious. The use of fluoroquinolones is controversial, as efficacy in a hamster model failed to clear leptospires from the kidneys and blood. Based on the ACVIM Consensus Statement, the antibiotic of choice is doxycycline (5 mg/kg PO or IV q. 12 hours for 2 weeks). Leptospires can shed in urine for months if appropriate antibiotic use is not implemented.

**Gastrointestinal support**

Azotemic patients should be treated with phosphate binders (e.g., aluminum hydroxide) if hyperphosphatemic, along with gastrointestinal protectants (e.g., omeprazole, pantoprazole, famotidine, sucralfate, etc.) for presumptive uremic gastritis. Anti-emetics (e.g., maropitant, ondansetron, dolasetron) should be implemented for patient comfort and to treat nausea.

- **Anti-emetics**
  - Maropitant: 1 mg/kg SQ q. 24 hours
  - Ondansetron: 0.1-0.2 mg/kg IV q. 8-12 hours
  - Dolasetron: 0.5-1 mg/kg SQ, IV q. 24 hours
  - Metoclopramide: 0.1-0.5 mg/kg SC, IV q. 8 hours or 1-2 mg/kg/day as CRI IV

- **Gastric pH altering medication**
  - **H2 blockers**
    - Famotidine: 0.5-1 mg/kg IV, SQ q. 12-24 (least p-450)
    - Ranitidine: 0.5-2 mg/kg, IV, PO, SQ q. 8-12 (moderate p-450)
    - Cimetidine: 5-10 mg/kg IV, PO, SQ q. 6-8 (most p-450)
  - **Proton-pump inhibitors**
    - Omeprazole: 0.5-1 mg/kg PO q. 24 hours
    - Pantoprazole: 1 mg/kg IV q. 24 hours
  - **Anti-ulcer:** Sucralfate 100-1 g PO q. 8 hours

**Zoonotic risks**

In animals developing acute leptospirosis, caution must be taken to prevent zoonotic spread. The use of appropriate hygiene (including protective eye ware, gowns, gloves, etc.) should be used when handling the patient and bodily fluids while hospitalized. Pet owners should also be cautioned about the zoonotic risk. A 10% bleach solution, iodine-based disinfectant, accelerated hydrogen peroxide, and quaternary ammonium solutions can all be used against leptospires. Likewise, other pets in the house should be assessed for clinical signs, and if healthy, vaccinated to mount an immune response.

**Prognosis**

The prognosis for leptospirosis is fair to good, provided aggressive treatment can be initiated. The survival is reported to be approximately 80% in dogs, both with dogs treated conservatively (e.g., IV fluids) and those treated more aggressively with hemodialysis. In those dogs developing pulmonary complications, the prognosis is poorer, with reported mortality rates (from Europe) of 36-42%. Pet owners should be cautioned about the risks for chronic renal insufficiency as a secondary consequence of chronic renal inflammation.

**Prevention**

As shedding of organisms can persist (e.g., leptospirosis) for weeks to months, prevention is imperative. Despite the good prognosis for leptospirosis, aggressive preventative care is warranted in dogs. This will help minimize zoonotic risk to pet owners and veterinary professionals; help minimize the chronic carrier state in dogs (which can result in further spread); prevent costly hospitalization; and minimize the risk of chronic injury (chronic renal failure). A leptospirosis prevention package should be initiated with the following:

- Environmental changes; This should be initiated to include rodent control; appropriate fencing; and landscaping changes to remove stagnant/standing water.
Annual vaccination: The decision to vaccinate should be based on an endemic area, exposure of the dog, and risk factors (e.g., access to streams/stagnant water or urbanized wildlife). Ideally, vaccination with a 4-way leptospirosis strain should be utilized. Vaccination is important to help prevent/aid in the prevention of shedding to reduce infection of other animals and possible human exposure.

References
2. Winzelberg SE. Leptospirosis treatment and prevention with data on an ongoing leptospirosis prevalence study at the Animal Medical Center in New York City. Atlantic Coast Veterinary Conference Proceedings, 2013.
Canine parvovirus (CPV) is a common pathogen affecting young dogs that are unvaccinated, under-vaccinated, or immunosuppressed. Without treatment, CPV can be life threatening due to severe fluid losses and electrolyte derangements secondary to anorexia, vomiting, and diarrhea. In order to ensure the best outcome, treatment should be aimed towards symptomatic supportive care, aggressive fluid therapy, anti-emetics, antibiotic therapy, and nutritional support. This lecture will review the etiology, clinical signs, treatment, overall prognosis and preventative measures for CPV.

Etiology
CPV was originally discovered in 1967 and resulted in mild diarrhea. Since then, the virus has evolved to CPV-2 in 1978, with additional evolution of subtypes CPV-2a, CPV-2b, and more recently, CPV-2c. CPV-2b is thought to be more pathogenic and has replaced CPV-2a as the cause of parvovirus throughout the United States.

Pathogenesis
CPV is a small, single-stranded, non-enveloped DNA virus that preferentially infects rapidly diving cells (e.g., bone marrow, gastrointestinal tract, myocardium, etc.). There is an increased prevalence during warm summer months (e.g., July through September). Spread occurs via ingestion of bodily fluids (e.g., vomitus, diarrhea, etc.) containing the virus. CPV replicates quickly and infects the intestinal crypt epithelium by day 4 of infection. Clinical signs are thought to appear within 4-10 days of exposure, while antibody development occurs approximately 5 days after exposure.

Risk factors/signalment
Parvovirus is often seen in more urban environments with affected pups coming from poor husbandry backgrounds. As a result, pet owners may also have financial limitations. Dogs affected typically are < 6 months of age, between 6 to 20 weeks of age. Typically, there is no gender predilection, although one study reported that in dogs > 6 months of age, intact, male dogs were overrepresented. Certain breeds are thought to be at increased risk:
- American Pit Bull terrier
- Rottweilers
- German Shepherd dogs
- Doberman pinschers

In studies, breed, age, gender, and body weight did not appear to correlate with outcome or duration of hospitalization in one study.

Clinical signs
Clinical signs seen with parvovirus include:
- Anorexia
- Lethargy/Listlessness
- Malaise
- Hypersalivation (e.g., secondary to nausea)
- Vomiting
- Abdominal pain
- Diarrhea
- Hematochezia

In mild cases, diarrhea may not be seen.

Physical examination findings
Classic physical examination findings for the parvovirus patient include:
- Dehydration (e.g., prolonged skin tenting, sunken eyes, etc.)
- Cachexia
- Hypothermia
- Fever
- Tachycardia

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• Tachypnea
• Pallor
• Prolonged capillary refill time (CRT)
• Hypersalivation
• Poor pulse quality
• Hypovolemic shock
• Fluid filled loops of intestine
• Malodorous diarrhea staining
• Dyspnea
• Death

Differential diagnoses
Other rule outs for patients exhibiting similar clinical signs include:
• Other viral (e.g., coronavirus) infections
• Other bacterial (e.g., E. coli) infections
• Parasitism
• Intestinal bacterial infection (e.g., Salmonella, Campylobacter)
• Intussusception
• Foreign body obstruction

Diagnostic testing
The use of a fecal antigen ELISA test is the most rapid, cost-effective way of diagnosing CPV for the practitioner. The fecal antigen ELISA is sensitive to detect both CPV-2b and CPV-2c.6 Other tests that can be considered include PCR, virus isolation, and hemagglutination inhibition, but these are less commonly performed. That said, in a dog that tests negative on an in-house fecal antigen ELISA test, a PCR (on feces) can be considered due to its high sensitive. A real-time PCR can improve the sensitivity and specificity, and allows for rapid detection of CPV-2.7

The diagnosis of CPV can be more challenging if these diagnostic tests are not readily available, as the decision to put an immunocomprised, young, immunologically naïve puppy into isolation poses large risk if the patient truly does not have CPV. Note that the modified live (ML) vaccine for CPV also replicates in the mucosal epithelium of the GIT; theoretically, the presence of low levels of antigen can be detected by various tests, resulting in a false-positive result.2 However, a recent study showed that various types of ML CPV-2 vaccines did not produce levels of antigen that were detectable on a SNAP ELISA parvovirus antigen test within 7 days of vaccine.2,8

Depending on the financial limitations of pet owners, the ideal “gold” standard for the parvovirus patient includes:

Gold or cadillac™ standard
• Parvoviral fecal antigen test
• Complete blood count + blood smear
• Biochemistry panel
• Venous blood gas (e.g., acid-base status, electrolytes)
• Fecal float/smear
• PCV/TS/BG/AZO
• Blood pressure
• PCR if negative fecal antigen test and still suspicious
• Abdominal radiographs
• Colloid oncotic pressure (COP)
• + abdominal ultrasound (if intussusception is suspected)

Silver or Honda™ standard
• Parvoviral fecal antigen test
• CBC with smear evaluation
• Biochemistry panel or venous blood gas
• Fecal float
• Blood pressure

Bronze or YUGO™ standard
• Parvoviral fecal antigen
• Blood smear + PCV/TS/BG/AZO
• Venous blood gas with electrolytes
Clinicopathologic findings
As parvovirus affects the pediatric patient, blood work changes associated with young patients are observed (e.g., isosthenuria, mild anemia, hypoproteinemia, elevated alkaline phosphatase, hyperphosphatemia, etc.). Additional clinicopathologic findings seen with parvovirus include:

- Lymphopenia
- Neutropenia
- Overall leukopenia
- Left shift
- Hypoglycemia
- Hemoconcentration (for a puppy)
- Hypoalbuminemia
- Hypoproteinemia
- Elevated liver enzymes
- Electrolyte abnormalities (e.g., hyponatremia, hypokalemia, hypochloremia)
- Mildly increased blood urea nitrogen
- Thrombocytopenia (due to consumption/blood loss into the GIT or DIC)
- Prolongation of PT/PTT
- Acid-base abnormalities (e.g., metabolic acidosis)
- Azotemia (secondary to multi-organ dysfunction)

Goals
Treatment of the canine parvovirus patient is aimed towards fluid therapy, antibiotic therapy, nutritional support, gastrointestinal support, supportive care, and monitoring. Specific goals of pediatric medicine include temperature control, fluid therapy, nutritional support (with the goal of weight gain), and control of infectious disease and parasites. In the more critically ill pediatric patient, goals should be focused on the following 4H’s: Hypovolemia/Hydration, Hypothermia, Hypoglycemia, and Hypoxemia.

Hypovolemia/Hydration
One of the most common causes of neonatal hypovolemic shock is dehydration, which can occur quickly in these small patients due to gastrointestinal losses or higher fluid requirements; therefore, aggressive fluid therapy is warranted because these small patients can deteriorate quickly. For neonates, maintenance fluid requirements are 120-180 ml/kg/day, while for pediatric patients, fluid requirements range from 60-100 ml/kg/day. In critically ill pediatric patients, fluid therapy for shock must initially be given by IV (or intraosseous) route. Intraperitoneal or SQ routes are not adequate due to slower absorption and, ideally, should not be used in the critically ill, dehydrated, or hypovolemic patient. In severely dehydrated or hypovolemic patients, initial shock doses of a balanced crystalloid such as 30-45 ml/kg should be used. Serial assessment should be done after the bolus to reassess response and to evaluate the need for further fluid resuscitation. Potassium and dextrose supplementation typically is required, and careful monitoring of blood glucose and electrolytes is warranted. Lastly, colloids can be used in pediatric patients; however, keep in mind that puppies have a lower colloid osmotic pressure (COP) than adult dogs. If necessary, a colloid (e.g., Hetastarch, 1 mL/kg/H; VetStarch, 2 mL/kg/H) can be used to keep colloid osmotic pressure above 15 mm Hg in non-azotemic patients.

Hypothermia
In pediatric patients, careful temperature regulation and awareness of normal homeostatic temperatures is imperative. Normal rectal temperature in the first week of life is 96° ± 1.5°F (35.6° ± 0.7°C), 98.6° - 100°F (37-38.2°C) in the second and third week of life, and by 7 weeks of age, reach normal adult levels. Hypothermia can lead to bradycardia and intestinal ileus.

Hypoglycemia
Young patients are prone to hypoglycemia, which can be aggravated by anorexia, vomiting, diarrhea, dehydration, and infection. Ideally, IV dextrose boluses should be used (0.5-1.0 g/kg or 0.5-1.5 ml/kg IV of 50% dextrose, diluted 1:2-1:3) preferentially over oral dextrose. Isotonic fluids supplemented with 2.5-5% dextrose as a CRI can also be used (i.e., not D5W); however, caution should be used to prevent over-supplementation as prolonged hyperglycemic can result in worsening of dehydration via osmotic diuresis (due to puppies having insulin insensitivity).

Hypoxemia
Young patients exhibiting clinical signs of hypoxemia (e.g., cyanosis, orthopnea, tachypnea, dyspnea, and abnormal auscultation) should be immediately treated with oxygen therapy. Because neonates and pediatric patients are “normally anemic,” it may be clinically more difficult to “see” cyanosis since detection of cyanosis is dependent on hemoglobin concentration. In dyspneic patients, initial first line therapy should include oxygen therapy via facemask, oxygen cage, incubator, or endotracheal tube. The FiO2 should not exceed 40-60% for more than a brief period due to the risk of oxygen toxicity.

Antibody therapy
In general, beta lactam antimicrobials are considered the safest choices in young, growing puppies. If possible, avoid chloramphenicol, aminoglycosides, tetracyclines, and drugs like clindamycin that undergo enterohepatic cycling. Metronidazole can be used, but dose interval should be prolonged. Finally, quinolones have been shown to result in cartilage lesions in puppies and should be used only with the benefit outweighs the risk and ideally avoided altogether in growing, large breed dogs. Commonly recommended dosages include:

- Amoxicillin: 6-20 mg/kg IV, PO q. 12
- Amoxicillin + clavulanic acid: 12.5-25 mg/kg PO q. 12
- Cephalexin/Cefazolin: 10-30 mg/kg IV q. 8-12
- Cefoxitin: 22 mg/kg IV q. 8
- Ampicillin: 22 mg/kg IV q. 8
- Ampicillin/Sulbactam: 22-30 mg/kg IV q. 8

**Gastrointestinal support**

Anti-emetics (e.g., maropitant, ondansetron, dolasetron) should be implemented for patient comfort and to treat nausea.

**Anti-emetics**

- Maropitant: 1 mg/kg SQ or IV q. 24 hours
- Ondansetron: 0.1-0.2 mg/kg IV q. 8-12 hours
- Dolasetron: 0.5-1 mg/kg SQ, IV q. 24 hours
- Metoclopramide: 0.1-0.5 mg/kg SC, IV q. 8 hours or 1-2 mg/kg/day as CRI IV

The use of gastric pH-altering medication is not necessarily warranted in the CPV patient; most are unlikely to have gastric ulcers. As these gastric pH-altering medications have no anti-emetic effect, the author believes these are not typically necessary. That said, therapeutic doses include:

**H2 blockers**

- Famotidine: 0.5-1 mg/kg IV, SQ q. 12-24 (least p-450)
- Ranitidine: 0.5-2 mg/kg, IV, PO, SQ q. 8-12 (moderate p-450)
- Cimetidine: 5-10 mg/kg IV, PO, SQ q. 6-8 (most p-450)

**Proton-pump inhibitors**

- Omeprazole: 0.5-1 mg/kg PO q. 24 hours
- Pantoprazole: 1 mg/kg IV q. 24 hours

**Miscellaneous therapies**

Fresh or fresh frozen plasma from recovered dogs has been suggested in the past to provide anti-parvoviral antibodies, but recent studies have not found a beneficial effect and have found that even recently recovered animals have minimal anti-CPV antibody concentrations.\(^1\)\(^,\)\(^13\) Moreover, such treatment may prime the dog for future transfusion reaction at a later point in its life. Equine endotoxin antiserum, recombinant human granulocyte-stimulating factor (rhG-CSF), or anti-virals (e.g., Tamiflu) have not been shown to be effective in improving survival or outcome.\(^14\)\(^,\)\(^16\) In small studies, the use of feline interferon has been weakly associated with improved survival; however, this is not readily available in veterinary hospitals.\(^17\)\(^,\)\(^18\)

**Prognosis**

The prognosis for canine parvovirus infection is fair to good with treatment, with recent reports of 80-90% survival with various modalities of treatment.\(^4\) Perhaps surprisingly, severity of neutropenia is not a negative prognostic factor, but severity of dehydration and lymphopenia may be.\(^19\) Recently, studies have compared standard in-hospital treatment versus a modified outpatient treatment (using volume resuscitation followed by subcutaneous fluid therapy and supportive care). Both protocols can be successful, with a survival only slightly lower in outpatients.\(^4\) A modified outpatient protocol may be a good alternative for less severely affected cases or those with financial limitations.

**Infectious diseases**

In animals developing acute parvovirus, caution must be taken to prevent further spread. The use of appropriate hygiene (including protective eye ware, gowns, gloves, etc.) should be used when handling the patient and bodily fluids while hospitalized. A 10% bleach solution, iodine-based disinfectant, accelerated hydrogen peroxide, or quaternary ammonium solution can all be used against parvovirus.\(^2\) Pet owners should be made aware to quarantine all affected pets for several weeks and to avoid dog parks, doggy daycares, training schools, city p

**Prevention**

While vaccination against parvovirus is highly effective, failure of passive transfer, early weaning, lack of vaccination, inappropriate client education (e.g., frequency of veterinary visits), or maternal antibody interference can result in disease. Parvovirus can be easily prevented by appropriate client education and vaccination. As DHPP is considered a core vaccine, puppies should be vaccinated
frequently while maternal antibodies are waning. In high-risk areas (e.g., shelters), a vaccine every 2 weeks is indicated until 16-22 weeks of age (depending on if the breed is at risk) to prevent outbreaks.

AZOSTICK = BUN, bpm = beats per minute, BPM = breaths per minute, BG = blood glucose, HR = heart rate, IO = intraosseous, IP = intraperitoneal, IV = intravenous, PCV = packed cell volume, SQ = subcutaneous, TS = total solids

Footnotes


References

Cardiogenic shock (CS) occurs when oxygen delivery to the tissues is inadequate as a result of cardiac dysfunction with adequate intravascular volume. While patients in cardiogenic shock are often in congestive heart failure this does not have to be the case. The decrease in cardiac output associated with CS can result from failure of the heart to eject blood during systole (forward failure) or from inadequate ventricular filling during diastole (backward failure).

Forward CS can occur due to true systolic failure (decreased inotropy) as seen with DCM in which cardiac is directly related to stroke volume, however forward CS may also occur when valvular integrity is suddenly lost as happens with rupture of one or more first order chordae tendinae. Cardiogenic shock uncommonly occurs due to abrupt onset bradycardia but can be seen with 3rd degree AV block or sinus arrest (sick sinus syndrome). Regardless of the underlying cause, forward failure and CS is characterized by an increased end diastolic volume. This form of CS is often exacerbated by pulmonary edema and accompanying hypoxemia.

Backward failure and CS results when a disease process prevents diastolic filling of either the left or the right ventricle. This form of CS can result from obstruction of the right ventricular outflow tract or severe tachycardia effectively shortening the time available for diastolic filling, but most commonly is the result of acute cardiac tamponade secondary to pericardial effusion.

Due to the mechanism of CS many of these patients have a chronic progressive heart disease with an acute exacerbation and onset of shock. Acute tamponade can occur in a patient that was apparently healthy with collapse, shock or even sudden death as the only clinical signs. Canine heartworm disease can lead to CS when a large worm burden (60-100 worms) matures rapidly resulting in caval syndrome.

Cardiogenic shock can occur in patients of any age although older middle aged to geriatric animals are likely at increased risk. Small breed dogs are more likely to have forward failure resulting from valvular disease while large or giant breed dogs (Boxers and Dobermans excepted) are more likely to develop backward failure due to pericardial disease. When CS develops in a young animal it is most often secondary to a severe congenital abnormality and is often a terminal event.

Common historical information provided by owners includes episodes often described as “seizures”. These episodes are in fact syncope and can be differentiated from true seizures with some detailed questioning. Syncope often occurs during periods of excitement or increased activity whereas seizures often occur when animals are quiet and resting. Syncope patients often have episodes lasting for a few seconds after which they return to normal within several minutes. Episodes of aborted syncope may also have occurred in which the patient looked like it was going to collapse but then was able to recover. Patients can urinate or defecate during syncope so these findings should not be used to differentiate between the two. All patients presenting in shock with a history of “seizures” should be carefully evaluated for causes of cardiogenic shock.

As a rule these patients have physical exam evidence of impaired perfusion including: pale or cyanotic mucous membranes, cool distal extremities and obtundation. These patients will also have at least one abnormality found during cardiac auscultation such as: bradycardia or tachycardia, abrupt onset or cessation of an arrhythmia, new or worsening murmur, absent heart sounds or pulmonary crackles.

Unfortunately a single diagnostic test does not exist to diagnose cardiogenic shock, rather results from several tests, physical examination and history will have to be reconciled. Often, patients with CS are not hemodynamically stable enough to withstand prolonged physical restraint so the evaluation may be conducted over an extended period of time. Proper prioritization of diagnostic tests can aid in reaching a diagnosis, for instance delaying blood sampling but performing a single lateral radiograph instead may be a better use of the patient’s minimal physiologic reserves. Brief thoracic ultrasound and lead II EKG can often be obtained with little to no physical restraint necessary. When performed a brief thoracic ultrasound should focus on identifying left atrial enlargement, tamponade and estimating systolic function. Formal echocardiography is often necessary to identify the specific cause of CS although clinical decisions can be made without it.

Treatment of CS differs fundamentally from all other forms of shock in which volume expansion and vasopressor therapy are the mainstays of treatment. In fact, volume expansion and vasopressor therapies are often contraindicated for CS patients and their use could lead to worsening of the patient’s condition up to and including death. Cardiogenic shock must therefore be managed primarily by improving cardiac performance first and altering vasomotor tone second. Oxygen should be administered at the highest FiO2 possible immediately upon recognition of a patient in shock to maximize arterial oxygen content as much as possible. Animals with confirmed or suspected pulmonary edema should be administered a loop diuretic (furosemide 2 mg/kg IV or IM) and the dose should be repeated every 30-60 minutes until the clinical signs improve. The use of mild sedatives such as butorphanol (0.2 mg/kg IV or IM) may facilitate placement of an intravenous catheter to optimize medication administration.
Positive inotropes should be administered to patients with known or highly suspected systolic dysfunction. Due to the critical state of these patients these medications are almost invariably administered as constant rate IV infusions. Dobutamine can be administered at a starting dose of 2.5 mcg/kg/min and can be titrated up every 5-10 minutes until clinical signs improve or a rate of 20 mcg/kg/min is reached. In some instances it is possible to use pimobendan to improve cardiac contractility. Unfortunately, in the U.S. pimobendan is only available as an oral medication making its use limited. An injectable form of pimobendan is available for use in Europe with a recommended starting dose of 0.15 mg/kg IV.

Vasoconstricting medications are almost never used to treat CS however, balanced vasodilators may be beneficial. The most effective medication available is nitroprusside which can be used at doses starting at 1 mcg/kg/min up to a maximum of 10 mcg/kg/min. Unfortunately, the cost of nitroprusside has recently made its use prohibitive. Injectable hydralazine may be used to reduce vascular tone with anecdotal doses reported from 0.05 to 0.2 mg/kg/hr. As with nitroprusside, hypotension may occur. Early signs that hypotension is developing include vomiting. When vasodilators are used the doses should be adjusted to target a systolic arterial blood pressure of 90 mmHg or a mean blood pressure of 70 mmHg. Sildenafil citrate may be useful for management of acute, severe pulmonary hypertension in dogs with a dose of 1-2 mg PO TID. Again this medication is generally available as an oral formulation making its use for CS patients limited. An injectable form of sildenafil is available and has been used anecdotally with success. When used a loading dose of 1 mg/kg is administered slowly IV followed by a CRI of 0.3 mg/kg/hr.

Cardiogenic shock secondary to tamponade is treated by pericardiocentesis. Ultrasound may be used to guide the catheter during this procedure or it can be done blindly following previously described landmarks and techniques. Removal of even small amounts of pericardial effusion can have a dramatic impact on cardiac performance since pressure increases exponentially in a sphere as the radius changes.

Arrhythmias can be managed in one of two major ways, conversion of the rhythm to normal sinus rhythm or control of the ventricular contraction rate. Anti-arrhythmic medications can be used in the setting of CS to achieve either of these two goals. Due to the urgency inherent in CS injectable medications are always preferred over oral formulations. Lidocaine should be considered the first line anti-arrhythmic of choice for treatment of ventricular tachycardia and should be administered as a 2 mg/kg slow IV bolus monitoring the EKG for conversion. If no response is seen a second dose can be administered. If conversion occurs but is transient then a CRI should be considered at rates of 50-100 mcg/kg/min. Occasionally patients with SVT present in CS in which case beta blockade would be recommended. Administration of beta blocking drugs should be withheld until all other causes of shock have been ruled out. Once again, injectable, ultra-short acting drugs are preferred. Esmolol can be administered as a bolus of 0.2 to 0.5 mg/kg followed by CRI of 50-200 mcg/kg/min. Diltiazem may also be used (5-20 mcg/kg/min) and may be more beneficial for managing SVT that is not sinus in nature. Some tachyarrhythmias are amenable to electrical cardioversion but this procedure should be performed only in a referral setting by someone experienced in electrical cardioversion as the risk for developing asystole is moderate.

Management of bradyarrhythmias resulting in CS is essentially limited to the use of temporary or permanent pacemaker implantation. Transthoracic or transesophageal pacemakers can be used with many currently available defibrillators using the pacing function. Temporary or permanent transvenous pacing requires a dedicated generator and fluoroscopy and is most likely to be found in a referral hospital setting.

The management of caval syndrome can be done in a private practice setting using a transvenous jugular approach. This technique has been well described in review articles and textbook chapters and provided the patient survives the perioperative period the prognosis appears to be good.

Unfortunately, published mortality rates for veterinary patients with CS are lacking but human literature suggests mortality rates of 50-80%. In most cases, CS develops secondary to severe underlying cardiac disease. For this reason the short and long-term prognosis for these patient should be considered guarded to poor. Dogs receiving pacemaker implantation for 3rd degree AV block or surgery for caval syndrome provide two notable exceptions.
In 2010 the American College of Veterinary Emergency and Critical Care undertook the task of developing cohesive, veterinary specific recommendations for the performance of cardiopulmonary resuscitation utilizing an evidence-based approach. The result of this effort was the publication of the Reassessment Campaign on Veterinary Resuscitation (RECOVER) in 2011, developed through the participation of over 100 veterinary specialists and are composed of 101 clinical recommendations spanning 6 categories.

The progression for severely ill to cardiopulmonary arrest (CPA) is complex. Although not all animals progressing to CPA follow the same clinical course some physical exam findings that may indicate impending CPA include decreasing level of consciousness, hypothermia, hypotension, bradycardia and changes in respiratory pattern. Although the underlying cause of CPA may not be immediately apparent to the treating clinician, the nature of the arrest can have a significant impact on the expected outcome. Although success rates for veterinary patients receiving CPR are low with published survival rates ranging from 3 to 27% for animals suffering in hospital arrest, neurological outcomes are generally good to excellent, making CPR a worthy endeavor. In order to maximize the likelihood of a good outcome every effort should be made to optimize the effectiveness of CPR.

The success or failure of CPR is often determined before the first chest compression or rescue breath is provided. Organized and pre-stocked crash carts should be located in the areas that CPR is most likely to be performed. In most veterinary practices one of these locations will be in the vicinity of the operating room or areas where anesthesia is most frequently performed. In addition to crash carts, an easily visible and legible poster or individually tailored form with pre-calculated doses of common CPR drugs should be available. The veterinary team should make efforts to develop leadership and communications skills for members of the staff to improve their effectiveness during CPR. Communication skills can be further honed, and performing a debriefing following each CPR event can reveal important learning points. During this debriefing the team should critically evaluate their performance and determine what aspects of the CPR went well and which areas need to be targeted to improve future performance. The adage “practice makes perfect” or at least “practice makes better” in the case of CPR holds true. Therefore, the use of high fidelity mannequins or veterinary CPR training dummies and regular training events is recommended as their use can improve effectiveness of CPR by developing psychomotor skills allowing the clinician to slow down the event and think in a more clear fashion.

Second in importance only to preparation is the provision of high quality basic life support that is the foundation of cardiopulmonary resuscitation. The rapid recognition and diagnosis of CPA is of utmost importance as success rates decline considerably the longer CPA goes untreated. Because performance of CPR in patients that are not in CPA rarely causes complications and almost never causes serious complications it is recommended that no more than 10 seconds be allowed for pulse or heartbeat detection in animals that are apneic and unconscious. If cardiopulmonary arrest cannot be ruled out during this 10-second assessment then chest compressions should be started immediately. While historical practice has emphasized establishing an airway as the first step in resuscitation, current recommendations are to begin chest compressions and attempt orotracheal intubation concurrently. The need to re-establish blood flow to the brain and heart, even if that blood is somewhat oxygen depleted, trumps the need to increase blood oxygen levels, at least initially. Chest compressions should be provided at a rate of 100 compressions per minute and should compress the thoracic diameter by 1/3 to 1/2 its diameter. Once an airway is established, rescue breaths should be provided at a rate of 10 breaths per minute. Higher respiratory rates do not improve blood oxygen levels but can have detrimental effects due to the increased intrathoracic pressure during positive pressure ventilation and impaired venous return. Once begun, every effort should be made to minimize interruptions to chest compressions. It is recommended that brief (5-10 second) interruptions happen at pre-planned intervals every 2 minutes to allow for ECG evaluation and compression provider rotation. Significant compression provider fatigue occurs quickly and leads to a significant degradation in the quality of chest compressions.

Advanced life support is comprised of any and all interventions beyond chest compressions and ventilation. The most frequently utilized advanced life support technique is provision of drugs intended to improve hemodynamics and ameliorate vagal tone. Epinephrine is arguably the most frequently administered medication during CPR. Its use is intended to cause peripheral vasoconstriction and centralization of the blood to allow better perfusion of the brain and heart. The currently recommended dose of epinephrine is 0.01 mg/kg IV every 3-5 minutes or every two CPR cycles. All anesthetic drugs should be reversed immediately upon recognition of CPA and correction of documented, severe acid-base or electrolyte disturbances should be considered. Defibrillation is only indicated in animals with a heart rhythm amenable to such treatment (i.e. ventricular fibrillation or pulseless ventricular tachycardia). When progression from a perfusing rhythm to ventricular fibrillation (VF) or pulseless ventricular tachycardia (PVT) is observed then immediate defibrillation should be performed. If the progression to VF or PVT is not observed then defibrillation should be delayed to allow for completion of one CPR cycle of two minutes. Once defibrillation has been performed, chest compressions should be resumed for a two-minute cycle before the ECG is evaluated for success. The application of open chest CPR
can improve outcomes but comes with difficulties inherent with thoracotomies. Attempts at open chest CPR should only be attempted if the means for managing a patient post-resuscitation are readily available.

In order to optimize CPR it is necessary that patients be monitored for signs of success or failure. All patients with suspected CPA should be instrumented with ECG. Analysis of ECG may help to rule out CPA or may identify rhythms that are amenable to a specific treatment (i.e. defibrillation). In the ideal setting all patients undergoing CPR will have end-tidal CO₂ monitoring performed. While useful for confirming correct placement of endotracheal tubes within the tracheal lumen in non-CPA animal, E₄CO₂ should not be used as the sole confirmation of endotracheal intubation in CPA animals. Since E₄CO₂ is linearly associated with cardiac output in CPA it can be a useful monitoring tool to gauge effectiveness of chest compressions and can be the earliest indicator of return of spontaneous circulation. When performing chest compressions and maintaining the minute ventilation at a constant level, E₄CO₂ levels can predict the likelihood of success with E₄CO₂ level of less than 15 mmHg and 20 mmHg suggesting worse prognosis in dogs and cats respectively. Once ROSC occurs E₄CO₂ will undergo a rapid and sustained rise as the heart more efficiently delivers CO₂ laden blood from the periphery to the pulmonary circulation. If ROSC is achieved then post-resuscitative monitoring should be tailored to the individual patient’s needs.

The care of an arrest patient does not end with ROSC; rather this is when the true care of the patient begins. In the immediate post-resuscitation phase every effort should be made to maintain arterial oxygen content within the normal range. No evidence exists that supra-physiologic oxygen levels are beneficial and theoretical detrimental effects exist. While post-arrest therapeutic hypothermia has become the standard of care in human medicine it is still beyond the capabilities of most veterinary facilities. However, if hypothermia occurs during the course of the arrest then rewarming efforts should not be vigorous and the patient should be allowed to return to normothermia at a gradual rate. Hyperthermia should be avoided if at all possible. There is no evidence in support of the routine use of corticosteroids, hypertonic fluids (mannitol or hypertonic saline), or prophylactic treatment with anti-seizure medications. If patients exhibit signs of intracranial hypertension then hypertonic saline or mannitol can be considered. Finally, referral to a comprehensive care facility with 24-hour capabilities should be considered for ongoing care of the post-arrest patient.
North America is home to 2 different families of venomous snakes, the elapidae (coral snakes) and the crotalinae (pit vipers). Of the two, the crotalinae are much more clinically significant due to their less reclusive behavior and wide range. In fact, pit-vipers can be found throughout North, Central and South America and include all rattlesnakes, the cottonmouth/water moccasin, and the copperhead. Pit-vipers are characterized by having moveable, hollow fangs through which venom is injected into the victim. Mature pit-vipers are capable of metering the dose of venom that they inject in order to preserve venom for the purpose of obtaining food. Juvenile snakes are incapable of dose adjustment and envenomate in an all-or-nothing fashion.

Historically the venom of the pit-vipers was considered to be vasculotoxic or necrogenic. While these terms are still useful, pit-viper venom is complex and varies with the species and local habitat. Some rattlesnakes for example the Mojave Green Rattlesnake, have venom that is highly neurotoxic with little tissue damage accompanying the bite. Ultimately, the purpose of snake venom is to immobilize the victim and begin the process of digestion. Typical pit-viper venom is composed of 90% water with the toxic components divided into both enzymes and peptides. The enzymatic factors are responsible for degrading connective tissue and allowing spread of the venom while peptides cause endothelial cell damage and ultimately circulatory collapse.

In addition to the massive soft tissue injury and necrosis that can accompany pit viper envenomation, life threatening hemostatic injury can occur. Typically, the coagulopathy resulting from pit viper envenomation is secondary to degradation of fibrin and fibrinogen directly by venom components. Through an unknown mechanism, platelet counts are often low following pit viper envenomation. These findings mimic those of disseminated intravascular coagulation (DIC) although importantly, fibrin cross-linking does not occur with snake envenomation and microthrombosis is not typically present. Neurotoxins associated with pit vipers result in non-depolarizing post-synaptic neuromuscular blockade and flaccid paralysis. Direct cardiovascular injury is possible and is manifested by ventricular arrhythmias and endothelial injury with circulatory collapse.

Establishing a diagnosis of pit viper envenomation can be challenging and is often heavily dependent on the history provided by the owner since clinical signs can be delayed by several hours. Physical examination may reveal one or two small puncture wounds or fang marks, most often on the face but occasionally on the feet or legs. Envenomating bites are often severely painful with progressive swelling advancing from the site of the bite. Some hematologic findings are present early in an envenomating bite and include echinocytosis and thrombocytopenia. Common chemistry changes that accompany envenomation include elevation of CPK, AST and ALT. The coagulation changes occur slightly later and are correlated with the severity of the envenomation. Expected findings would include decreased fibrinogen, prolongation of PT and aPTT and elevation of fibrin degradation products with normal to mildly elevated D-dimer.

Evaluating the severity of the bite can initially be difficult as 25 to 30% of pit viper bites are non-envenomating. Most initial defensive bites are dry with subsequent bites being highly envenomating. Agonal bites should always be considered highly envenomating since the entire volume of venom remaining in the venom glands will be injected. Several methods for describing severity have been described ranging from a numerical scale evaluating multiple systems to a simple minimal-moderate-severe scale. Regardless of the scoring mechanism used it is imperative to remember that signs may be delayed and may progress rapidly. Serial monitoring is recommended even for patients with no signs initially. Measuring circumference of an appendage or outlining the leading edge of swelling every few hours may be helpful to determine progression however, redistribution of edema to dependent locations occurs must be differentiated from progression of swelling. One distinguishing characteristic is the pain that accompanies progression of signs is usually absent when dependent redistribution is the occurring.

Owners may contact a veterinarian prior to presentation seeking first-aid advice. Currently the only accepted first aid measure to be taken is immobilization of the affected site if possible followed by rapid transportation to a veterinarian. The owner should be instructed not to attempt to lance the site or attempt to suck venom from a bite. Placement of a tourniquet as this could lead to severe tissue damage and large areas of necrosis as the normal dilution of spreading venom components is prevented. It is not unusual for a severely envenomated patient to present in hypovolemic shock. In this case judicious resuscitation with crystalloids should be performed. Maintaining clear endpoints for resuscitation such as normalization of heart rate, respiratory rate and mentation are important to prevent over-resuscitation and subsequent exacerbation of tissue edema. Utilization of goal directed endpoints such as lactate, base excess and central venous oxygen saturation may aid in determining when resuscitation is complete.

The dosing of antivenom is based on the amount of venom injected not on the size of the patient. Typically dogs receive between 1-5 vials of antivenom. Paradoxically, smaller dogs often require higher doses of antivenom than large dogs due to the smaller volume of distribution of the venom. Administration usually begins slowly to allow for monitoring of possible reactions but is ideally completed within 30-60 minutes. Treatment with antivenom is indicated if progression of venom effects is seen including progressive...
swelling and pain, coagulopathy or systemic effects such as hypotension or altered mental status. When administered antivenom binds to venom components and can reverse some of the clinical signs while preventing or slowing progression of signs. Ideally, antivenom should be used early following envenomation (i.e. within 4 hours) but may be beneficial up to 24 hours after a bite. Two different types of antivenom are currently available, a whole molecule antibody derived from immunized horses and a partial molecule antibody (Fab) derived from immunized horses or sheep. Both types of antivenom can lead to immune reactions including anaphylactoid dose related reactions and delayed type III hypersensitivity reactions.

Comprehensive management of envenomated patients includes effective analgesia. Liberal administration of opioids should be considered provided the patient’s neurological status does not preclude their use. Adjunctive analgesic medications to consider would include ketamine, lidocaine or dexmedetomidine. Limbs should be maintained in a functional position. Areas of necrosis should be cleaned and covered with a sterile bandage and managed as any other open wound would be. The prophylactic use of antibiotics in pit viper envenomation is controversial but both the recent human and veterinary literature suggests that prophylactic antibiotic use is not beneficial. Rather, antibiotics should be reserved for treatment of a known or suspected infection. Administration of steroids is not beneficial and should be avoided. Non-steroidal anti-inflammatory drugs are useful for treating associated inflammation but their use should be restricted to patients that are hemodynamically stable. NSAID medications should not be given to patients with impaired renal function or myoglobinurainbus. Since the coagulopathy associated with pit viper envenomation is due to the presence of circulating venom molecules, the mainstay of treatment is antivenom administration. Fresh frozen plasma is not expected to be beneficial so long as circulating venom is present and when used should be given after all intended antivenom doses.
Recognition of dystocia is based on an understanding of normal parturition. The average gestational length for the dog is 65 days (57-72) and for the cat is 66 days (56-70). Once the fetuses are full term parturition begins. Normal labor can be divided into 3 stages. Stage 1 includes restlessness, nesting and panting. No detectable uterine contractions occur during stage 1 labor which usually lasts for 6-12 hours but may last as long as 24 hours. Stage 2 begins with strong abdominal contractions and clear discharge and is complete when a fetus is expelled. The first fetus should be expected 2-4 hours after the onset of stage 2 labor with subsequent fetuses being delivered every 30 minutes to 4 hours. This stage of labor may last as long as 12-24 hours in dog. Cats have the unique ability to voluntarily interrupt stage 2 labor if they are feeling distessed and stage 2 may last for up to 48 hours. Stage 3 labor consists of placental expulsion which should occur 5-10 minutes after delivery of a fetus. Generally stage 2 and stage 3 labor alternate between fetuses but it is possible for 2 fetuses to be delivered consecutively followed by 2 placentae, one from each uterine horn.

Dystocia occurs when parturition does not proceed normally. This can be due to a problem with either the fetus or the dam. Fetal causes of dystocia include fetal monsters, true oversize, fetal pelvic mismatch, fetal malposition or malposture or fetal death. Maternal causes of dystocia are more common and include small pelvic diameter, abnormalities of the caudal reproductive tract, primary uterine inertia (failure of the uterus to begin contractions), secondary uterine inertia (uterine fatigue) or malnutrition. Secondary uterine inertia is the most common cause of dystocia and results from uterine fatigue usually in small breed dogs with fewer fetuses of larger size relative to the dam.

Frequently, inexperienced owners will call a veterinarian concerned that parturition is not occurring normally. Generally, owners should be advised to have a patient evaluated if one of the following occurs:

1. Prolonged gestation (due date reached with no signs of labor)
2. Stage 2 labor > 4 hours with no fetus
3. More than 2 hours between fetuses (cats may interrupt labor so this may not apply if they have been disturbed)
4. Green or brown vaginal discharge for 2-3 hours with no fetus delivered
5. Strong contractions > 30 minutes with no fetus delivered
6. Signs of systemic illness in the dam
7. Fetal membranes or part of fetus protruding from the vagina

When a patient presents for suspected dystocia a detailed history should be obtained including last known breeding date and expected due date if available. Following the history a thorough physical examination should be performed focusing on identifying any underlying systemic diseases of the dam that may be causing dystocia. Digital vaginal examination should be performed using sterile gloves and liberal lubrication. Fetal positioning, vaginal anatomy (vaginal hyperplasia, septum, neoplasia), cervical dilation, soft tissue pelvic anatomy (lymph nodes, urethra) and presence of meconium should be evaluated. Light feathering of the dorsal vaginal wall can be performed to attempt to elicit a contraction.

Diagnostic imaging is often useful to aid in determination of fetal age if the owner is unaware of the breeding or due dates. A fetus should be considered full term when the caudal vertebrae, fibula, calcaneus, bones of the feet are visible radiographically. Visible teeth are the final radiographic sign of a full term fetus and are present at approximately day 61 of gestation. Beyond estimating fetal age, radiography can be used to evaluate fetal viability. Signs of fetal death include gas present in the fetus or placenta, collapse of the spinal column, overlapping of skull bones or loss of fetal flexion. Orthogonal radiographs should be evaluated for potential causes of dystocia including bony pelvic anatomy and fetal positioning. Fetal viability may be more reliably evaluated with ultrasonography. Lack of a visible heartbeat in a full term fetus is an indication of fetal death. Other ultrasound findings consistent with fetal death include: decreased placental fluid volume, increased fetal echogenicity and increased gas in the fetal stomach. Once viability is determined each fetus should be evaluated for evidence of distress. Normal fetal heart rates are greater than 180 bpm while rates between 150-170 indicate moderate distress and less than 150 bpm indicate severe distress. If a low fetal heart rate is identified it should be monitored for 30-60 seconds as fetal heart rates will be transiently depressed during uterine contraction.

Medical management can be considered for dystocia if the fetuses are viable with no evidence of distress, the dam is in good health, the labor has not been protracted, the cervix is dilated and the fetal size is consistent with a possible vaginal delivery. If medical management is attempted the dam should be well hydrated with normal electrolytes. This may require administration of intravenous fluids. Oxytocin should be administered 1-3 IU SC or IM and the dam should be monitored for progression of labor. If a fetus is not delivered after 30-45 minutes calcium gluconate may be administered slowly IV (0.5-1.5 mL/kg) while monitoring EKG. If no further progress is made the oxytocin dose can be repeated or may be increased to 5-20 IU SC or IM. Oxytocin dosing may be
repeated up to 3 times. If this fails to result in delivery of a fetus the patient should proceed to surgery for caesarean section. Only 1/3 of dogs with dystocia will respond to administration of oxytocin alone.

Caesarean section is required in approximately 60-65% of canine dystocia cases and 70% of feline dystocia cases and 60% of caesarean sections are performed on an emergency basis. The most common reason for proceeding to C-section is identification of fetal distress. When a fetal heart rate less than 150 bpm is identified the patient should proceed to emergency surgery, if the fetal heart rate is between 150-170 bpm surgical treatment should be strongly recommended above attempted medical management. If there is a doubt about the necessity for surgery C-section is usually better for both the dam and the fetuses. Pre-operative considerations include the dam’s volume status, shock status and intended future breedings. Prior to induction the owner should be asked whether to prioritize the dam or the fetuses in the case of an emergency.

The patient should have the abdomen clipped prior to induction to minimize the time between induction and delivery. Blood pressure should be monitored if possible since placental blood flow is dependent on maternal blood pressure. Anesthetic drugs that are reversible and/or short acting are preferred with pure opioid agonists and propofol being most common. Inhalant gas should be minimized until after delivery since elimination of the gas is dependent on ventilation and neonates delivered by C-section are often apneic. Line block of the intended abdominal incision with local anesthetic may aid in reducing the amount of inhalant needed for the approach. Epidural anesthesia/analgesia can be considered and is generally safe for neonates and dam. Pure opioids are the preferred analgesic drugs due to their complete reversibility.

Two surgical techniques have been described. Traditional caesarean section in which a hysterotomy is performed and the fetuses are delivered one at a time will allow for placental blood flow to be maintained in the undelivered fetuses increasing the likelihood of survival. When hysterotomy is performed the expected neonatal survival rate exceeds 90%. Following delivery of all the fetuses, an ovariohysterectomy can be performed or the uterus can be closed if future breedings are intended. An alternate method of delivery is en-bloc ovariohysterectomy in which each ovarian pedicle and the uterine body is clamped and the uterus is removed en-bloc with the fetuses still inside. The uterus is then handed off to an assistant who delivers the fetuses and begins neonatal resuscitation. If this method is used every effort should be made to limit the amount of time from the placement of the first clamp to delivery of the last fetus to less than 60 seconds. Survival rates using the en-bloc technique are reported to be 75% in the dog and 42% in the cat. This technique should be used preferentially if uterine infection is suspected, the entire litter is dead or if the maternal condition necessitates surgical brevity. When using this technique, care should be taken to ensure that no part of the fetus is clamped in the uterine body. This can be achieved by manually milking the fetus proximally into the body of the uterus. If any part of the fetus has descended beyond the vulva this technique will likely not be possible.

Following delivery neonatal resuscitation begins. Ideally, each neonate will have a dedicated assistant responsible for its resuscitation. The umbilical cord should be clamped if this has not already been done and the face and mouth should be cleared of placental membranes. The neonates should be rubbed vigorously with a clean, dry towel focusing on the face, thorax, genitals and umbilicus. The mouth and nostrils can be cleared by using a bulb syringe. Neonates should not be swung in an attempt to clear airways as this could lead to significant injury. Neonates should have a respiratory rate between 10-18 bpm. Manual, mouth to snout ventilations can be used and supplemental oxygen should be provided. Aggressive needling of the acupuncture point GV26 (at the philtrum of the nose) with a 25-gauge needle can stimulate ventilation in an apneic neonate. As long as a heartbeat is detectable resuscitation should continue even in the presence of prolonged apnea. In neonates that are responding poorly to resuscitative efforts, reversal of anesthetic/analgesic drugs can be attempted. Naloxone can be administered at 0.1 mg/kg. Dosing can be intravenous, intramuscular, intraosseous or sublingual. Vascular access can be obtained at the jugular vein or the umbilical vein. The administration of doxapram is not recommended due to the risk of cerebral acidosis in an apneic patient. Once resuscitation is complete and the neonates are vocalizing and vigorous, they should be evaluated for the presence of cleft palate, atresia ani and umbilical hernia prior to being placed in a warm environment. The dam and surviving neonates should be discharged from the hospital when anesthetic recovery is complete and milk letdown has been confirmed and all neonates have been
Under normal conditions total body water is divided into the intracellular and extracellular fluid compartments with 66% of total body water being located within the intracellular space. The remaining 33% of total body water is distributed between the intravascular and extravascular spaces. Sodium is the most ubiquitous electrolyte in the extracellular compartment. It is responsible for maintaining plasma osmolarity between fluid compartments and regulating water movement between the intracellular and extracellular spaces. Serum sodium and subsequently serum osmolarity is closely regulated by the kidneys and brain under the influence of several hormones the most important of which is antidiuretic hormone (ADH). Changes in serum osmolarity by as little as 1-2 percent are detected at osmoreceptors located in the hypothalamus. Stimulation of these receptors results an increase in thirst and the release of ADH from the posterior pituitary gland. Once released, ADH causes aquaporin-2 channels into the collecting tubules of the kidney resulting in free water retention and restoration of plasma osmolarity. Serum sodium is therefore a better reflection of total body water content than total body sodium content.

When serum sodium concentrations increase serum osmolarity also increases. When this happens, water moves down its osmotic gradient out of the intracellular compartment resulting in cellular dehydration and thus cellular shrinkage. While most tissues are capable to tolerating some change in cellular volume the brain is not. As serum osmolarity increases brain volume decreases. This is compensated for within the first several hours by accumulation of electrolytes within the brain. The movement of electrolytes into the intracellular space is sufficient to partially restore brain volume. This process is completed over the next several days as osmotic particles called organic osmolytes are generated re-establishing the normal balance between intracellular and extracellular osmolarity.

Hypernatremia, defines as plasma or serum sodium concentration above the reference range (145-155 mEq/L), is a relatively uncommon finding in dogs and cats but when present has been associated with a significant increase in mortality. The development of hypernatremia may be divided into two major causes: excessive free water loss, absolute sodium gain. Of these, absolute sodium gain is by far the less common but can result from ingestion of high salt materials such as homemade playdough or seawater. Hypotonic fluid loss on the other hand is more common and can result from insufficient water intake, increased insensible losses, renal causes, gastrointestinal causes and cutaneous losses.

Whether or not clinical signs are associated with hypernatremia depends on the degree of hypernatremia present (> 180 mEq/L) and the rate at which it developed. Most patients with mild to moderate hypernatremia that occurs slowly do not have clinical signs associated with the electrolyte abnormality itself, rather signs will be referable to the underlying disease. In patients with severe acute hypernatremia it is possible to see obtundation, head pressing, seizures, coma or death.

Since the normal physiologic response to hypovolemia is sodium retention through the action of aldosterone, free water deficit should not be calculated until normovolemia has been established. When a patient is hypovolemic the initial replacement fluid of choice should have a sodium concentration as close as possible to that of the patient. In most cases 0.9% NaCl will be a suitable choice. Many patients will have correction of hypernatremia when the volume status has been returned to normal. If hypernatremia persists in a normovolemic patient then a free water deficit exists. Free water deficit is calculated using the following formula: Free water deficit (L) = 0.6 x body weight in kg x (measured Na+/ideal Na+ - 1). When hypernatremia develops acutely the correction can occur relatively rapidly with the free water deficit being replaced to achieve a rate of change of plasma sodium of 1 mEq/hr. When chronic hypernatremia is present it must be corrected more slowly due to the generation of organic osmolytes in the brain. The maximum rate of correction of chronic hypernatremia is therefore 0.5 mEq/hr.

Management of mild to moderate hypernatremia in a patient with an intact thirst mechanism may be achieved by administration of oral water. When the thirst mechanism is no longer intact enteral water may still be provided through placement of a feeding tube with the calculated free water deficit being replaced over the number of hours calculated using the correction limits above. If provision of enteral water is not an option due to decreased mentation or vomiting, or is not likely to be sufficient the free water deficit can be replaced using 5% dextrose in water. When this approach is taken the free water deficit is replaced as above with the volume being administered over the number of hours deemed to be safe. Regardless of the method used to correct free water deficit it must be remembered that this volume is an absolute deficit and should be added to the patient’s maintenance needs keeping in mind that ongoing losses and underlying medical conditions must be accounted for.

Once correction of hypernatremia has been started serum sodium should be monitored every 4 hours and the rate of correction adjusted to maintain an appropriate drop of sodium without exceeding the established maximum rate of correction (0.5 to 1 mEq/hr). It is typical for correction of severe hypernatremia to require 24 to 48 hours to correct. In addition to serum sodium concentration, the patient should be monitored for neurological signs associated with too rapid a correction including mentation changes (obtundation, stupor, coma), head pressing confusion or seizures. When hypernatremia is corrected too rapidly the intracellular osmolarity exceeds
blood osmolarity and water moves back into the cells down its osmotic gradient. This cellular swelling manifests as cerebral edema and increased intracranial pressure.

If a patient develops any signs associated with cerebral edema the serum sodium should be immediately measured to determine if the sodium concentration has gone down. If the serum sodium is lower, even if the rate of correction has not been excessive, cerebral edema should be considered. If cerebral edema is suspected treatment with 7.2% hypertonic saline at a dose of 3-5 mL/kg over 20 minutes should be started. If hypertonic saline is not available, mannitol can be considered at a dose of 0.5 to 1 g/kg IV over 20 minutes.
Under normal conditions total body water is divided into the intracellular and extracellular fluid compartments with 66% of total body water being located within the intracellular space. The remaining 33% of total body water is distributed between the intravascular and extravascular spaces. Sodium is the most ubiquitous electrolyte in the extracellular compartment. It is responsible for maintaining plasma osmolarity between fluid compartments and regulating water movement between the intracellular and extracellular spaces. Serum sodium and subsequently serum osmolarity is closely regulated by the kidneys and brain under the influence of several hormones the most important of which is antidiuretic hormone (ADH). Changes in serum osmolarity by as little as 1-2 percent are detected at osmoreceptors located in the hypothalamus. Stimulation of these receptors results an increase in thirst and the release of ADH from the posterior pituitary gland. Once released, ADH causes aquaporin-2 channels into the collecting tubules of the kidney resulting in free water retention and restoration of plasma osmolarity. Serum sodium is therefore a better reflection of total body water content than total body sodium content. Consequently hyponatremia reflects free water excess. Whether this free water excess represents absolute fluid excess depends on the underlying cause.

When intracellular osmolarity exceeds extracellular osmolarity water will move down its osmotic gradient and into the cell. This is most important in the cells of the CNS, which are highly sensitive to changes in volume. When cellular swelling occurs in the CNS intracranial pressure increases and if not addressed will lead to brain herniation and death. In acute hyponatremia, the cells of the CNS actively pump intracellular ions out of the cell in an effort to match the osmolarity of the plasma. This process occurs over several hours but is insufficient to compensate for large changes in serum sodium over long periods of time. When hyponatremia is chronic, cellular generation of organic osmoles is down regulated. This process takes several days but is much more effective as a long-term solution.

Hyponatremia is defined as blood sodium less than the reference range (145-155 mEq/L). Similar to hypernatremia, the degree of clinical signs associated with hyponatremia is dependent on both the magnitude of sodium change and the rate at which that change occurred. Mild to moderate hyponatremia (Na+ > 120 mEq/L) is unlikely to result in clinical signs. Severe or rapidly occurring hyponatremia on the other hand may cause CNS signs consistent with the development of cerebral edema including: mentation changes (obtundation, stupor, coma), head pressing and seizures. Unlike hypernatremia however, the pathogenesis of hyponatremia is more complex and affects the approach to treatment so a much greater degree.

The first step in managing a patient with hyponatremia is determining effective plasma osmolarity using the following formula: eOsm = 2(Na+) + (glucose/18). Unfortunately this formula fails to account for unmeasured osmoles such as ketone bodies, mannitol molecules or ethylene glycol molecules. Measuring blood osmolarity may identify an “osmole gap” when compared to the calculated value. Based on the calculation or measurement of osmolarity a patient can be classified as hyperosmolar, normosmolar or hypoosmolar.

Hyperosmolar hyponatremia results from increased blood osmolarity causing water to move from the intracellular to the extracellular space diluting blood sodium concentration. This type of hyponatremia is most common in patients with diabetes mellitus and in it’s worst form can manifest as hyperosmolar hyperglycemic syndrome. Normosmolar hyponatremia is often called pseudohyponatremia and is a laboratory artifact that can be seen when a patient’s plasma compartment contains a large fraction that is not water as occurs with hyperglobulinemia or hyperlipemia. Pseudohyponatremia only occurs when flame photometry is used to determine sodium concentration but does not occur when an ion-selective electrode is used so familiarity with the laboratory methodology employed is important.

Hypoosmolar hyponatremia is further categorized based on the patient’s volume status. Hypervolemic hypoosmolar hyponatremia results from a decrease in effective circulating volume and is seen in three clinical conditions: congestive heart failure, severe liver disease, nephrotic syndrome. In all of these conditions a decrease in effective circulating volume results in non-osmotic release of ADH in an attempt to restore volume at the cost of decreasing osmolarity. As a rule glomerular filtration rate must be decreased for hypervolemic hypoosmolar hyponatremia to develop. When GFR is maintained excess volume is eliminated through an increase in urine production.

Normovolemic hypoosmolar hyponatremia develops due to an absolute excess of free water when GFR is maintained. Conditions that can lead to development of normovolemic hypoosmolar hyponatremia include psychogenic polydipsia, syndrome of inappropriate ADH secretion, myxedema coma or administration of hypotonic fluids.

Hypovolemic hypoosmolar hyponatremia is the most common category of hyponatremia encountered in daily practice. This type of hyponatremia develops due to volume depletion from either renal or non-renal causes. When volume is lost from the vascular space through the gastrointestinal tract (vomiting or diarrhea) or through loss into a third space (pleural effusion or peritoneal effusion) the
body again attempts to preserve volume at the expense of tonicity through release of ADH and free water retention. Renal fluid and sodium loss occurs from administration of loop diuretics or mineralocorticoid deficiency (hypoadrenocorticism).

Treatment of hyponatremia is dependent on the classification of hyponatremia and the underlying disease process as well as the magnitude and duration of hyponatremia. Hyperosmolar hyponatremic patients are treated for their underlying disease. Once the source of the unmeasured osmoles is removed (i.e. glucose, mannitol) the patient’s plasma sodium will return to normal. Management of hyperosmolar hyponatremic patients is more similar to management of hyponatremia due to the presence of a hyperosmolar state. In this situation, inappropriately rapid correction of hyperosmolarity will lead to formation of cerebral edema and the neurological sequelae that accompany it. The most appropriate rate of correction for hyperosmolar hyponatremia would be to reduce the patient’s osmolarity by 0.5 to 1 mOsm/hr (the same rate as sodium correction in hypernatremia). Normosmolar or pseudohyponatremia dose not require treatment, as this type of hyponatremia is a laboratory artifact and should resolve when the underlying condition leading to the artifact is removed (non-lipemic sample) or the laboratory method is changed.

Correction of hypoosmolar hyponatremia can be more challenging. Due to the brain’s adaptive response to chronic or profound hyponatremia overzealous correction can lead to acute decrease in CNS cell volume. Myelin sheaths are particularly sensitive to acute changes in cell volume and demyelination can occur. Unfortunately, the neurologic signs that accompany this dehydration do not present immediately but rather, are delayed for several days. Common clinical signs secondary to overly rapid correction of hyponatremia include drowsiness, inability to stand, ataxia, extensor spasticity, coma and death. Although this condition is not uniformly fatal when it occurs the prognosis for return to normal is poor with most animals having residual neurologic deficits.

Hypervolemic hyponatremia is managed by restoration of effective circulating volume and GFR. For patients with congestive heart failure this will include a diuretic medication but must also necessarily include medications to improve cardiac output such as dobutamine or pimobendan. In the acute setting pimobendan can be administered at 0.25 to 0.5 mg/kg PO BID to improve cardiac output. The addition of afterload reducing drugs (hydralazine 0.5 to 2 mg/kg PO BID to effect) may also improve GFR. Vasopressin antagonists are available although their clinical use in veterinary medicine has been limited due to high cost and potential liver toxicity.

Normovolemic hyponatremia can be managed by careful water restriction to limit intake to a volume less than urine output. When restricting water intake it is important to monitor the patient for ability to concentrate urine. All medications that have potential antidiuretic effects should be discontinued.

Hypovolemic hyponatremic patients should be volume resuscitated with fluid containing sodium in concentrations as close to plasma sodium as possible. Once normovolemia (intravascular volume and interstitial volume) has been re-established the patient’s sodium should be rechecked. If hyponatremia is persistent then the addition of higher sodium containing fluids can be considered.

Regardless of the cause of hyponatremia, when correction is begun the patient should have serum sodium levels monitored every 4-6 hours targeting a return to normal of 0.5-1 mEq/hr. The late onset of clinical signs associated with correction of chronic or profound hyponatremia means that a more conservative correction rate is often preferred. Due to the delayed presentation of neurological signs owners should be instructed to monitor the patient carefully and to return if the patient seems to be declining in any way. The best way to manage central demyelination is to be aware of the possibility and to prevent it from occurring. Once demyelination occurs treatment becomes entirely supportive.
Trauma is a common cause of veterinary emergency room visits. Common causes of trauma include bite wounds and blunt injuries suffered from encounters with vehicles or falls from heights. Due to the high degree of variability possible in the injuries associated with different mechanisms of trauma a systematic approach at the time of presentation followed by a deliberate medical or surgical plan is essential. The initial evaluation of a trauma patient begins with a brief history in which the owner or agent conveys what transpired if known. This initial history should be kept brief and should focus on important facts about the traumatic event including mechanism of injury (fall, hit by car, bite wound), loss of consciousness during or after the event, time from event to presentation and voluntary ambulation post event.

Following this brief history an initial survey exam is performed focusing on identifying potentially life-threatening injuries. Evaluation of the airways is usually performed first. This is achieved by observation of the respiratory pattern paying special attention to the effort the patient is making and if an increase in effort is evident which phase of the respiratory cycle is affected. Stertor or stridor may be evident and if severe may necessitate immediate intubation to aid in stabilization. Mucous membranes should be evaluated next. The presence of cyanosis or pallor should raise concern for impaired oxygenating ability or shock. Capillary refill time may be prolonged or even absent if the patient is in the later stages of shock. Breath sounds should be carefully ausculted. Absence of breath sounds either unilaterally or bilaterally may indicate pleural space disease such as pneumothorax or hemothorax. Loss of breath sounds in conjunction with circulatory collapse and severe respiratory distress suggest tension pneumothorax. Crackles, indicating pulmonary contusion or hemorrhage may be detected.

If a life threatening condition is present steps should immediately be taken to correct or manage it. Severe stertor/stridor or respiratory arrest requires endotracheal intubation or emergency tracheostomy. Identification of tension pneumothorax is treated by rapid thoracocentesis. Circulatory collapse or cardiac arrest is treated with cardiopulmonary resuscitation. If an imminent threat to life is not identified the secondary survey is begun utilizing a systematic approach to ensure no occult injuries are missed.

The secondary survey typically begins with evaluation of the patient’s neurological status. The patient’s mentation and level of consciousness should be evaluated and the patient should be assessed for voluntary motor. Postural changes such as rigid extension of all four limbs (opisthotonus) or rigid extension of the forelimbs only (Schiff-Sherrington) are associated with severe forms of brain stem compression or cerebellar herniation. Animals may be obtunded to stuporous following trauma and this may be due to the presence of primary CNS injury or as a result of shock. Small patients that were lifted from the ground and shaken should be treated as if they have a spinal cord injury until proven otherwise, especially if they have not been observed to ambulate following the injury.

Evaluation of the thorax is performed next beginning with a visual inspection to identify any lacerations or puncture wounds that might penetrate the thoracic cavity. Repeated auscultation should be performed paying special attention to the quality of breath sounds in each hemithorax. Decreased breath sounds ventrally may indicate the presence of pleural effusion while decreased sounds dorsally is common with pneumothorax. Focused assessment of the thorax with ultrasound has been shown to be an easy and rapid method of evaluating both the pleural space and the lung parenchyma. Loss of a glide sign or identification of fluid indicate pneumothorax or pleural effusion respectively. So called “lung rockets” suggest the presence of primary parenchymal injury or contusion. This procedure may be performed in either lateral or sternal recumbency. The ultrasound probe is initially placed at the “chest tube site” (widest part of the chest) on the left and right to evaluate for pneumothorax or contusions. The probe is then relocated to the “pericardial site” directly over the heart on the left and right to evaluate for possible pericardial effusion.

Abdominal evaluation again begins with a visual inspection of any lacerations and puncture wounds to generally assess depth and possible penetration. Any puncture wounds over the abdomen, especially those resulting from animal bites have the potential to penetrate the abdomen even if the surface injury appears to be minor. Abdominal distension with the presence of a fluid wave may be present with traumatic hemoabdomen. Palpation of the urinary bladder should be attempted although the presence of a palpable bladder does not rule out the possibility of uroabdomen or uroretroperitoneum. Rectal exam should be performed to assess pelvic ring continuity and to assess anal tone. Abdominal focused assessment with sonography for trauma (AFAST) has been well reported and is widely accepted as a rapid and sensitive method for evaluating the abdomen for the presence of free fluid. This evaluation can be performed in lateral recumbency. The ultrasound probe is placed over one kidney and is fanned cranial to caudal, at which time the probe is turned 90 degrees and the fanning is repeated in a ventral to dorsal plane. The probe is then moved to the next site of evaluation and the process is repeated until all of the following sites have been evaluated: left kidney (spleno-renal), xyphoid (diaphragmatico-hepatic), right kidney (hepato-renal), urinary bladder (cysto-colic). The finding of free fluid is a positive result. If free fluid is identified then attempts should be made to collect a sample for evaluation either via ultrasound guidance of blind 4-quadrant tap. Fluid should be assessed for PCV/TS, creatinine and potassium to rule out hemoabdomen and uroabdomen respectively.
Orthopedic and cutaneous wounds are finally identified. Patients should be evaluated for puncture wounds, abrasions, lacerations or bruises. Any areas of possible cutaneous injury should be gently clipped to allow for complete evaluation. Long bones and joints are assessed for fractures or disarticulations.

Laboratory evaluation may include PCV/TS, lactate, BUN as separate tests or may be included as part of a venous blood gas analysis. PCV is often normal following trauma, even when hemorrhage has occurred, due to splenic contraction however TS will generally fall when significant hemorrhage has occurred and may be a better indicator of the degree of bleeding. Lactate is a byproduct of anaerobic metabolism and indicates that oxygen delivery to the tissues is inadequate to keep up with patient needs and is a sensitive indicator of shock. Routine radiography of the thorax following any blunt trauma should be considered to rule out occult pneumothorax or pulmonary contusion. Radiographs should be obtained of any identified long bone or joint abnormalities with the region of interest centered in the beam.

Treatment of trauma patients can be divided into primary and secondary phases. The primary phase or resuscitative phase of treatment focuses on restoration of systemic and local oxygen delivery to pre-injury levels. The second phase begins once normal oxygen delivery has been restored and lasts until the patient is discharged from the hospital. The focus of the second phase of management is maintenance of oxygen delivery and definitive treatment of wounds, fractures and pain.

The ultimate success or failure of managing the severely traumatized patient is often decided within the first hours of presentation during the initial evaluation and stabilization. Successful resuscitation begins with identification of shock. The main goal of resuscitation should be re-expansion of the effective circulating volume with re-establishment of systemic and local blood flow. Resuscitation should not be considered to be complete until certain endpoints have been reached. Traditional endpoints of resuscitation include normalization of heart rate, respiratory rate, pulse quality and blood pressure. Newer, goal directed endpoints have been useful in identifying occult shock and should be incorporated into resuscitation goals. These include normalization of blood lactate and base excess and central venous oxygen saturation.

Initial treatment of a patient in shock should be administration of supplemental oxygen with early volume expansion. Crystalloid only resuscitation has been the mainstay of treatment for shock for many decades. It has the advantage of being relatively cheap and is readily available. Typically a replacement crystalloid such as lactated Ringer’s solution or 0.9% NaCl is administered rapidly in aliquots of 20-25 mL/kg IV until endpoints are reached or the maximum dose of 90 mL/kg has been reached. While effective the duration of volume expansion associated with crystalloid only resuscitation is short (30 minutes); this can be extended by incorporating synthetic colloids into the resuscitation protocol. A useful technique is to alternate doses of crystalloids with doses of colloids in 5 mL/kg aliquots until endpoints are reached or maximum doses of 90 mL/kg crystalloid and 20 mL/kg colloid are reached. Low volume resuscitation has been utilized for several years and is effective at restoring oxygen delivery while limiting over-resuscitation and associated tissue edema that delays healing. This is accomplished by administering 4-6 mL/kg 7.2% NaCl with 10-20 mL/kg synthetic colloid followed by crystalloids as needed. This approach generally reduces the overall fluid needed to reach endpoints and can be used in any trauma patient that was not severely dehydrated at the time of the traumatic event but is particularly useful in animals with cavitary hemorrhage or brain injury.

Early analgesia should be considered an important part of the resuscitative phase of treatment. As soon as it is evident that death is not imminent analgesia should be administered. Since trauma patients are dynamic the best analgesic choice is a pure opioid agonist such as morphine or hydromorphone. These drugs are effective, have no ceiling effect and are fully reversible. Typical starting doses are 0.05-0.1 mg/kg hydromorphone or 0.2-0.3 mg/kg morphine IV, IM or SC.

The treatment of wounds during the resuscitation phase focuses on preventing further wound contamination and tissue injury. All visible wounds should be clipped and cleaned with an antiseptic solution and lavaged with saline or tap water. Following lavage the wounds should be gently probed for depth and extent and then covered with a sterile non-adherent dressing until stabilization has been achieved and definitive management is possible. Any confirmed or suspected fractures of the distal limbs should be immobilized by placement of a modified Robert-Jones bandage or splint ensuring that the joint above and below the fracture are included. Fractures of the proximal limbs are not stabilized with external coaptation due to the risk of creating a stress riser and causing injury to important nearby structures (arteries, veins, nerves).

The secondary phase of trauma management begins when resuscitation endpoints have been met and the patient is either admitted for further care or is moved to surgery for definitive management of wounds or fractures. Almost all patients that suffered trauma severe enough to require surgery or hospitalization will require some fluid therapy. Due to the large volumes of crystalloids that many of these patients receive during resuscitation care must be taken to adjust ongoing fluid plans to meet patient needs without exacerbating edema or causing fluid overload. To ensure that fluid administration is not exceeding patient needs, serial weights may be used with any weight gains likely reflecting retained fluid. Many severely traumatized patients are moderately to severely hypoproteinemic making interstitial edema more likely to occur. Administration of synthetic colloids can be considered to minimize fluid leakage from the intravascular space but significant controversy exists about their routine use.

As with the resuscitative phase, appropriate analgesia is an important part of the management of trauma during the secondary phase. Pain causes a neuroendocrine response that increases levels of catabolic hormones including cortisol while decreasing anabolic

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hormones such as insulin and impairing healing. Patients suffering trauma should be considered painful even if they are not demonstrating overt signs of pain. The best approach to pain management in trauma is multimodal therapy. Pure opioid agonists are the mainstay of treatment and can be safely used in even severely traumatized patients due to their cardiovascular sparing characteristics and reversibility. Respiratory depression is uncommon in veterinary patients when appropriate doses are used. Ketamine is dissociative agent NMDA antagonist that has some analgesic properties and modifies central sensitization that can lead to chronic pain syndromes or disproportionate pain responses. When used ketamine should be administered for at least 24 hours as a CRI at a rate of 5-15 mcg/kg/min in conjunction with an opioid. Alpha-2 agonist agents such as dexmedetomidine are also useful analgesic drugs at doses lower than those used for sedation. Dexmedetomidine is typically used as a CRI at a rate of 0.5 to 2 mcg/kg/min/hr.

When possible local or regional anesthetic techniques should be used including epidural administration, nerve block or diffusion catheter placement.

Non-steroidal anti-inflammatory drugs may be used in trauma patients once hemodynamic stability has been returned and abrupt changes are no longer anticipated. Typically NSAID administration is delayed until 24 hours after trauma for patients admitted to the hospital. The decision to use NSAIDS should be deliberate following careful consideration of the possible negative side effects. NSAIDS should not be used in any patient with evidence of hematuria or pigmentation. The routine use of corticosteroids should be avoided due to the potential side effects including immunosuppression and delayed wound healing.

Traumatized patients require adequate caloric intake to heal. Following admission to the hospital a nutritional plan should be formulated beginning with calculating the basal energy requirements of the patient [(30 x BW in kg) + 70]. If the patient is not eating voluntarily then a feeding tube may be required to meet nutritional needs. The goal should be to reach the full BER within 48 to 72 hours from admission.

The routine use of antibiotics is not necessary unless the patient presented with grossly contaminated wounds or has evidence of infection. When necessary, first generation cephalosporins are generally adequate for wounds not associated with animal bites. Patients that have been bitten by an animal should be treated with a potentiated penicillin or fluoroquinolone to cover common oral pathogens. Ideally antibiotic therapy would be guided by culture and sensitivity results. The risk of infection can be reduced through careful handling of all catheter sites and by keeping wounds and surgical incisions covered with a clean and dry bandage at all times. As always, the use of barrier protections (i.e. gloves) is recommended to prevent inadvertent colonization of an immunocompromized patient with potential pathogens such as methicillin resistant staphylococcus species.

Perhaps the most important aspect of the secondary management phase is monitoring the patient for evidence of hemodynamic compromise, organ failure or infection. Regular monitoring of temperature, respiratory rate and heart rate will identify patients potentially developing complications. Monitoring of blood pressure will be useful if the patient has had episodes of hypotension. Serum biochemistry analysis and complete blood count should be performed every 2-3 days as the patient’s condition dictates to identify possible organ failure early. Traumatic coagulopathy can occur in severely traumatized patients and any patient with unexplained bleeding or bruising should be evaluated with a platelet count and PT/aPTT.

High quality nursing care is imperative for the successful management of a severely traumatized patient. Every effort should be made to keep the patient clean and comfortable. Fecal and urine contamination should be cleaned as soon as they are identified. Patients should be kept on deep, soft bedding and should be repositioned every 4 hours if they are not ambulatory or moving on their own to prevent decubital ulcers from forming. All incisions and wounds should be evaluated at least once a day and soiled or wet bandages should be changed immediately upon recognition. Early mobilization of the patient will maintain joint health and aid in the management of ileus associated with recumbency and opioid administration. Passive-range-of-motion exercises are also useful to maintain range of motion and lymphatic flow.

Determination of prognosis can be difficult due to the confounding effect of euthanasia. Factors that have been associated with poorer outcomes include: head trauma, vertebral fractures, hemobdemen and the need for mechanical ventilation. The use of scoring systems may aid in guiding decision making but care should be taken to prevent using scores as a decision making tool for individual patients. The animal trauma triage (ATT) score assigns a score from 0-3 in six categories (perfusion, cardiac, respiratory, eye/muscle/integument, skeletal, neurological) with a maximum total score of 18. The risk of death has been shown to increase by 2.3 to 2.6 times for every one-point increase in ATT score. The modified Glasgow coma score (MGCS) can be used to serially monitor patients with head trauma and traumatic brain injury by assigning a score from 1-6 in three categories (motor activity, brain stem reflexes, level of consciousness) with a minimum score of 3 indicating the worst possible neurological performance. Total MGCS scores of less than 8 have been associated with a 50% mortality rate at 48 hours. In general the prognosis for animals suffering trauma is good with survival rates above 90%.

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Identifying patients that might be at risk for development of refeeding syndrome is the first step in the management of the condition. Certain risk factors have been identified in people and are also present in the veterinary reports. The classic risk factor for refeeding syndrome is chronic malnutrition in which total energy and nutrient quality are both deficient. An often-overlooked cause of chronic malnourishment is conditions that result in malabsorption of ingested nutrients. Dogs and cats with severe intestinal disease or pancreatic insufficiency may be unable to absorb adequate nutrients despite having a normal appetite and nutrient rich diet. Similarly, patients that have been completely anorexic (no caloric intake) for > 7-10 days have an increased risk of developing refeeding. The last significant risk factor for veterinary patients is obesity with rapid weight loss.

Under homeostatic conditions net nutrient balance is 0. Meaning that the sum of energy intake is equal to the sum of energy consumption by the body and energy excretion. In order for growth to occur a positive energy balance is needed. Similarly, for protein synthesis to occur, a positive nitrogen balance is necessary. When energy consumption exceeds that needed for maintenance of daily needs, excess energy is stored in the form of adipose tissue for use in times of decreased energy availability. This storage mechanism allows for an energy reserve to be established for use at a later date if needed. Excess nitrogen intake unfortunately, does not result in storage of nitrogen. Rather, excess nitrogen intake only results in an increase in nitrogen excretion. During times of starvation or inadequate nutrient intake it is possible for animals to be severely protein deficient but still have adequate caloric energy to meet RER in the form of stored adipose tissue. Therefore, animals that are consuming protein deficient diets may be at increased risk of developing refeeding syndrome despite having some enteral caloric intake in the form of carbohydrate or fat. When starvation conditions are present the energy needs of the vital organs are met in the following order: brain, kidneys, maintenance of blood sugar, physical activity and finally maintenance of body condition.

The physiology of starvation can be divided into an acute response (that occurring within the first 2 weeks of starvation) and a delayed response (that occurring greater than 10 days after the onset of starvation). During the initial phase of starvation or anorexia there is a voluntary reduction in physical work and an involuntary reduction in basal metabolic rate. The decreased metabolic rate is characterized by a decrease in protein turnover, and decreased levels of thyroid hormones and somatomedins. If these changes do not result in equilibrium of energy demand and energy intake then endogenous fuels (adipose tissue, muscle) are used to balance the equation. As mentioned above, triglycerides are the major form of fuel storage in mammals and the body’s fat supply is the major determinant of the length of survival under starvation conditions. Carbohydrates are not stored in any significant amount and are therefore of little value with liver and muscle glycogen stores accounting for less than one days worth of caloric needs. While body protein could account for up to 2 weeks of caloric requirements, its depletion would have profound adverse effects due to its role in essential non-fuel functions. The metabolic alteration that occurs under starvation conditions is mediated by changes in circulating hormone levels with decreases in insulin and triiodothyronine (t3) and increases in glucagon, growth hormone, catecholamines and plasma cortisol. The end result of these hormonal alterations is enhanced hepatic glycogenolysis, gluconeogenesis and skeletal muscle proteolysis thereby facilitating lipolysis. The brain is subsisting on glucose generated from protein catabolism and gluconeogenesis in the liver. During the delayed response to starvation there is a major shift from glucose to fat as the main energy source. Gluconeogenesis is reduced during this period and protein catabolism is minimized. Ketone bodies from hepatic oxidation of fatty acids are used by most of the tissues for energy. At this stage the brain is reliant on ketone bodies as an energy substrate. It is also during this delayed response to starvation that the kidneys begin to assume the role of an important glucose-producing organ.

Besides the hormonal and metabolic changes taking place, compositional changes are also occurring. Weight loss during starvation is greatest during the first few days due to a disproportionate loss of water. At the same time, body fat and lean body mass are reduced. Bone mass is preserved in nearly all cases unless malnutrition or starvation is prolonged. The reduction in total body water is greatest during the first 10-14 days. Within the first 48 hours of complete anorexia natriuresis occurs leading to significant extracellular and subsequently, intracellular fluid loss. Urinary sodium excretion decreases significantly after 7-14 days of complete anorexia. Natriuresis can be prevented by consumption of even very small amount of dietary carbohydrate. In addition to sodium loss, potassium, phosphorus and magnesium are lost due to catabolism of cell mass for use as an energy substrate. Although total body potassium, phosphorus and magnesium levels are depleted, serum levels are maintained.

The metabolic response to refeeding following starvation or lengthy malnourishment results from a major shift back to the use of carbohydrates as the primary energy substrate instead of fat. When patients are fed with carbohydrate and protein there is an immediate increase in circulating insulin levels. This insulin secretion inhibits glycogenolysis, gluconeogenesis and fatty acid mobilization by reducing glucagon levels. The same insulin secretion results in enhancement of cellular uptake of glucose, potassium, phosphorus and magnesium. Abrupt cessation of natriuresis occurs causing rapid expansion of the extracellular fluid compartment.
When the diet used for refeeding is composed of a high proportion of carbohydrate the cessation of natriuresis is abrupt and can lead to the development of peripheral edema and fluid overload. Refeeding with fat or protein alone will allow natriuresis to continue and may prevent fluid overload or edema formation from occurring.

In addition to the overall, global metabolic consequences of starvation and refeeding, there are effects on specific organ systems. During starvation the cardiovascular system is affected as cardiac muscle mass is reduced in parallel with loss of lean body mass. Cardiac function is impaired as catabolism of contractile sarcoplasmic proteins occurs and left ventricular mass is reduced. Cardiac dysfunction is characterized by a reduction in cardiac output (primarily through reduced stroke volume) and decreased ventricular compliance (reducing pre-load). Hypotension and bradycardia are the body’s attempt to reduce oxygen consumption and are not reflective of circulatory collapse. Upon refeeding there is a dramatic increase in heart rate, blood pressure, oxygen consumption, cardiac output and plasma volume. This rapid increase in metabolic demand may exceed supply and can lead to the development of congestive heart failure. Although the same metabolic and cardiovascular changes likely occur in veterinary patients there are no reports to date of congestive heart failure secondary to “naturally occurring” starvation and refeeding.

The effect of starvation on the gastrointestinal tract is early reduction in enterocyte formation and nutrient absorption. Decreased levels of brush border disaccharidases and pancreatic enzymes occur due to mucosal atrophy, loss of intraluminal stimulation and generalized protein depletion. Gut atrophy with decreased crypt cell proliferation and reduced villus height occurs as starvation progresses. Intestinal mass is reduced and thickening and coarsening of small intestinal mucosal folds occurs. Gastric acidity is reduced and both gastric and intestinal motility is decreased. Diarrhea occurs due to impaired absorptive ability, bacterial overgrowth, presence of unconjugated bile salts, hypoalbuminemia and gut edema. The development of diarrhea exacerbates electrolyte loss and leads to further whole body depletion of phosphorus, potassium and magnesium.

Arguably, the most important metabolic consequences associated with starvation and refeeding are the effects on total body phosphorus (during starvation) and serum phosphorus (upon refeeding). Phosphorus is present in both an inorganic and organic form within the body with organic phosphate being the most abundant intracellular anion. Organic phosphate is found within phospholipids, nucleic acids and many enzymes. It is important for maintenance of plasma membranes, the electron transport chain, ATP and 2,3-diphosphoglycerate. Inorganic phosphate is found in the extracellular fluid and is used as a substrate for many important functions including glycolysis, 2,3-DPG production and oxidative phosphorylation. As mentioned above, during starvation, total body phosphorus becomes depleted despite serum phosphorus usually being maintained within the normal range. Upon refeeding phosphorus moves from the extracellular fluid compartment to the intracellular fluid compartment to be used for the synthesis of phosphorylated compounds. When hypophosphatemia occurs secondary to refeeding it can be dramatic and may be seen within the first 24-72 hours although delayed hypophosphatemia can be seen up to 10 days following reintroduction of food. The consequences of hypophosphatemia can be severe and most of the clinical consequences associated with refeeding syndrome are attributed low serum phosphorus. Some of the consequences of hypophosphatemia include cardiac arrhythmias and contractile dysfunction, central nervous system dysfunction (including seizures), cellular hypoxia (due to 2,3-DPG depletion and shift of the oxyhemoglobin dissociate curve), leukocyte dysfunction, thrombocytopenia and impaired clot contraction; all of which are due to limitations in ATP and 2,3-DPG levels. In addition to its importance in cellular respiration, ATP is important for the maintenance of red blood cell (RBC) membrane integrity, cell shape and RBC deformability. ATP depletion can lead to decreased cell deformability and osmotic lysis due to impairment of RBC sodium-potassium pump function. The subsequent increased red blood cell fragility leads to perhaps the most devastating consequence of hypophosphatemia: intravascular hemolysis.

Alterations in serum potassium and magnesium levels also occur upon refeeding and can be severe in patients that have whole body depletion of potassium and magnesium. When serum insulin levels increase secondary to refeeding extracellular potassium is shifted to the intracellular compartment. Trans-cellular shifting of potassium with subsequent hypokalemia can lead to muscle weakness, ileus, cardiac arrhythmias, and rhabdomyolysis. Magnesium plays an important role in many body systems, acting as a cofactor involved in energy storage and utilization, protein synthesis, CNS function and neuromuscular function. Serum magnesium undergoes a similar trans-cellular shift when refeeding occurs and new tissue synthesis begins. The clinical signs of hypomagnesemia are similar to those seen with hypokalemia making it difficult to determine what is responsible for the clinical signs that are present. Although hypophosphatemia, hypokalemia and hypomagnesemia are each capable of individually causing the clinical signs associated with refeeding it is probable that the combination of electrolyte abnormalities is ultimately responsible. Thiamine deficiency may play a role in the development of clinical signs associated with refeeding syndrome although no definitive evidence exists. Thiamine is an important cofactor in carbohydrate metabolism and thiamine deficiency can lead to CNS dysfunction, muscular weakness and cardiomyopathy.

The prevention of refeeding syndrome begins with awareness and recognition of patients that are at risk. As previously mentioned risk factors for veterinary patients to develop refeeding syndrome include chronic undernourishment, obesity with rapid weight loss, prolonged fasting or complete anorexia of greater than 7-10 days duration. When a patient with one or more of these risk factors is identified and it is imperative to formulate a comprehensive nutritional plan in addition to a treatment plan for any underlying medical conditions. Patients should have complete blood work performed and any electrolyte abnormalities should be corrected prior to
initiation of nutritional support. Because of the risk for fluid overload, the administration of intravenous fluids should be judicious and should be geared at correcting dehydration and meeting maintenance needs.

The first step in formulating a nutritional plan is to determine the patient’s caloric needs by calculating the basal energy requirement (BER) using one of the following formulas: 1. Kcal/day = 30(BW in kg) + 70 or 2. Kcal/day = 70(BW in kg)\(^{0.75}\). Basal energy requirement is the amount of energy required to maintain the body’s minimum normal metabolic activity. When calculating the BER there is some controversy about whether the ideal body weight or actual body weight should be used. It is likely that either method is acceptable provided the patient is monitored for refeeding syndrome. The goal of the first week is to meet the calculated BER. Glucose should be provided at 150 to 200 g dextrose per day and lipid should account for 20-30% of non-protein calories. Protein should be provided at 1.2 to 1.5 grams/kg/day. There should be no attempt made to achieve weight gain during the first week of treatment and any weight gain that does occur should be considered to be due to fluid retention rather than addition of lean mass. There is little evidence that supplementing electrolytes prevents the development of refeeding syndrome and should not be used as a replacement for development of a comprehensive nutritional plan. The provision of electrolytes should be guided by measurement of serum levels. Although not documented, thiamine deficiency is a theoretical concern and supplementation with vitamin B complex can be considered. Due to the risk of fluid overload, measurement of serial body weights should be considered mandatory and consideration should be given to measurement of central venous pressure.

Once refeeding has begun electrolytes should be monitored at least once a day and should include measurement of serum phosphorus, potassium, sodium and ionized calcium and magnesium. After all, early recognition of refeeding syndrome is essential for successful treatment. Refeeding syndrome is most likely to occur within the first several days of refeeding although its onset can be delayed up to 10 days. If electrolyte changes consistent with refeeding syndrome develop then nutritional support should be stopped immediately and aggressive correction of electrolyte abnormalities should be pursued. Supplementation of potassium may occasionally exceed the recommended maximum of 0.5 mEq/Kg/hr. Phosphorus supplementation rates of 0.3 to 0.6 mM/kg/hr. can be used and magnesium supplementation can be attempted at 0.75 to 1 mEq/kg/day. If hypophosphatemia is not recognized and hemolysis develops the patient should receive blood products as needed to prevent signs associated with anemia and phosphorus supplementation should be begun. Following the first week of nutrition the patient’s caloric prescription should be increased by 10 to 15 percent. Upon correction of electrolyte abnormalities, nutritional support can be restarted but should be reduced by 20-30%. Following the initial refeeding period of 7-10 days, the nutritional prescription can be increased to create an anabolic state and allow for lean body mass repletion.

Perhaps the most difficult aspect of preventing and treating refeeding syndrome is convincing the owners/rescuers and hospital staff that a slow and methodical approach to refeeding is in the patient’s best interest. A frank discussion with staff members is recommended immediately upon recognition of an at risk patient to ensure that a thoughtful nutritional plan can be formulated. Although it is with the best intentions that rescuers and staff offer food to dogs and cats rescued from starving conditions, that approach can have dire consequences. Veterinarians who are in a position to evaluate patients rescued from hoarding conditions or those responding to natural disasters should brief support personnel prior to seizure of pets to ensure proper introduction of nutrition. Once hospitalized, the best way to prevent overzealous feeding is to delegate responsibility of feeding to one person within the hospital. The attending veterinarian should continue to update staff on the expectations and possible consequences of refeeding syndrome ensuring the staff that they have the patient’s best interest at heart to prevent staff members, family or rescuers from sneaking food to the animal. Ultimately, the best treatment for refeeding syndrome is prevention, and prevention begins with awareness.
The use of targeted endpoints to guide resuscitation from shock has been in routine use in human medicine for the last 10 years with most evidence directed at resuscitation from septic shock. The utility of endpoints lies in their ability to alert the clinician to the possibility of incomplete resuscitation or occult shock. By evaluating specific endpoints the treating veterinarian is able to determine if a different approach to shock resuscitation is necessary or if addition of vasoactive agents is indicated. The ease with which endpoints are reached may also provide the veterinarian with prognostic information.

The determination of when resuscitation from shock is complete has traditionally relied on normalization physical examination findings. Evaluation of the patient’s mentation has always been (consciously or unconsciously by the veterinarian) the most utilized traditional endpoint. The goal is to have a patient that is bright and alert and, assuming that the neurologic and skeletal systems allow, ambulating. Evaluation of mentation is cheap, easy and non-invasive but may be complicated by co-morbidities including traumatic brain injury or metabolic disease. In addition the brain may be affected much later in shock than other organ systems due to the body’s ability to maintain cerebral perfusion over a wide range of blood pressures. Serial monitoring of the heart rate with the goal of normalization for species, age and breed is also cheap, easy to perform and non-invasive. Unfortunately, the heart rate can be elevated due to physiologic responses not secondary to shock, such as pain and anxiety. When heart rate is evaluated it may provide a better indication of volume status than actual tissue oxygen delivery. Mucous membrane color is often evaluated in conjunction with heart rate and offers the same advantages with the added advantage of providing an insight as to the oxygen content of hemoglobin. The major drawbacks to evaluation of mucous membrane colors are its lack of sensitivity and its subjective nature.

Traditional resuscitation endpoints that are more specifically used to evaluate perfusion include pulse quality, capillary refill time, blood pressure and body temperature. Evaluation of pulse quality is cheap and non-invasive but requires a certain degree of skill and experience and is highly subjective. While useful for providing a crude evaluation of macrovascular function, it does not provide any information about microvascular function and may be altered by regional perfusion disturbances. Capillary refill time is the only perfusion parameter evaluated on the physical examination that attempts to evaluate microvascular function but is also crude and non-specific. Measurement of blood pressure can be done with the use of Doppler technology or oscillometric monitors. Targeting a systolic blood pressure of greater than 90 mmHg or mean arterial pressure of greater than 60 mmHg is still one of the mainstays of resuscitation. Blood pressure determination gives the clinician a good overall estimation of macrovascular performance but again, does not correlate with microvascular perfusion. Additionally, depending on the technology in use, obtaining an accurate blood pressure reading requires both skill and experience on the part of the operator. Finally, body temperature can be used to evaluate perfusion indirectly. While core body temperature will be affected in the later stages of shock when compensation is no longer complete, the gradient between core body temperature and peripheral limb temperature may be abnormal earlier. As with most of the traditional resuscitation endpoints, evaluation of body temperature or temperature gradients can be affected by co-morbidities or by pharmacologic interventions.

Given the subjective and non-specific nature of most of the traditional resuscitation endpoints, alternatives were sought that would provide more specific information regarding the delivery and use of oxygen on a global level. Venous oximetry (evaluation of venous hemoglobin saturation) provides information about tissue oxygen utilization. As tissue oxygen levels decrease a greater portion of oxygen is extracted from hemoglobin as it traverses a tissue bed. This holds true insofar as lung function is normal and arterial hemoglobin is fully saturated with oxygen upon leaving the lung. Targeting a central venous oxygen saturation of greater than 65% ensures that blood oxygen content is not the factor responsible for shock. Unfortunately, venous oximetry has drawbacks including the impact of co-morbidities, the requirement for central venous catheterization and the need for blood gas measuring equipment. Additionally, the value of venous oximetry is highly dependent on the location the sample was collected from. The further away from the hear the sample is collected from the more it reflects the specific tissue beds it drains, meaning in that blood collected from a cephalic vein only reflect oxygen consumption in the limb distal to the venipuncture site.

Central venous pressure measures the amount of hydrostatic pressure within the cranial vena cava and provides a direct assessment of vascular volume and an indirect assessment of cardiac pre-load. Measurement and normalization of central venous blood pressure allows for the exclusion of hypovolemia as the cause of shock. When CVP is utilized as a resuscitation endpoint the goal should be to achieve a CVP of 8-12 mmHg (11-16 cm H2O). Utilization of CVP as a resuscitation endpoint can help prevent morbidity associated with over-resuscitation. Although relatively easy to perform, measurement of CVP requires a water manometer or multiparameter monitor capable of measuring invasive pressures. A moderate to high skill level is needed to successfully and correctly place a central venous catheter in a patient in shock and is often a limiting factor in the use of CVP in veterinary medicine.
Assuming that no co-morbidities exist and that the patient was euhydrated at the onset of shock, urine output is a good resuscitation target. Normal urine production of greater than 1 mL/kg/hr indicates that renal perfusion is adequate to maintain normal GFR making it unlikely that a significant perfusion derangement exists. To be utilized however, the patient must have an indwelling catheter placed and a closed collection system attached. Although this is easy to achieve in most male dogs, it is much more difficult in female dogs and cats of both sexes making its use infrequent.

Finally, the use of biochemical markers of shock allows for the assessment of cellular respiration and utilization of oxygen. Base deficit and lactate have both been utilized as resuscitation endpoints in both human and veterinary medicine. No clear evidence exists that would justify the use of one over the other and in fact they may be most useful when evaluated in conjunction. Base deficit reflects the degree of intracellular acidosis and therefore the presence or absence of anaerobic metabolism. It is easy to obtain and is a good indicator of tissue hypoxia. Diseases affecting both the kidneys and acid-base status independent of cellular respiration can affect base deficit. Lactate is a byproduct of anaerobic metabolism and can be used to estimate the degree of anaerobic metabolism that is occurring. Similar to base deficit however, lactate can be elevated for reasons other than tissue hypoxemia (i.e. type B lactic acidosis). When biochemical monitoring is being used to guide resuscitation the goal should be to achieve a base deficit of no greater than 5 mmol/L and a lactate of less than 2.5 mmol/L.

Perhaps the most clinically useful approach to resuscitation incorporates both the traditional and goal directed endpoints. When abnormalities are present with the traditional endpoints it is reasonable to provide resuscitation until resolution of these abnormalities occurs. At that time the evaluation of goal directed endpoints could be performed to investigate the possibility of occult shock or ongoing oxygen debt. By having endpoints of resuscitation in mind prior to treatment of a patient in shock the clinician can better determine when resuscitation is complete or if more aggressive means must be implemented.