Feline Lower Urinary Tract Disease

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

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

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

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

Canine urethral obstruction

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

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

**Acute Renal Failure**

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

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

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

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

If following IV fluid diuresis, urine production is still low, pharmacological intervention is indicated to increase urine output. Diuretics and vasodilatory agents are used to enhance urine output. Furosemide, 10% dextrose and mannitol are diuretic agents employed. Low dose dopamine (2-5 μg/kg/min) may improve urine flow.
Furosemide is readily available and given as an intermittent IV bolus (2-3 mg/kg IV every 6-8 hours) it helps increase tubular and renal blood flow without significantly affecting GFR. Dopamine can enhance the effectiveness of furosemide so these two agents are often given concurrently.

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

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

**Uroperitoneum**

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

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

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

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

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

Dystocia

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

Five to 6% of canine and feline pregnancies require intervention. Animals at higher risk include brachycephalic dogs and short-faced cats. Devon rex cats and Bulldogs are at much higher risk than other breeds. Uterine inertia, the failure to initiate and maintain sufficient uterine contractions, is the most common cause of dystocia accounting for 2/3 of the dystocias in the bitch and queen. Fetal malpresentation is the most common fetal cause of dystocia. Other maternal causes of dystocia include mechanical obstruction (abnormal pelvic canal), and maternal anxiety. An oversized fetus, fetal monstrosity and fetal death are the other fetal causes of dystocia.
When an expected due date has come and passed, the dam should be evaluated. Even without evidence of maternal distress, early intervention will likely improve fetal survival. A complete physical exam is important to assess the health of the dam. Radiographs will confirm a term pregnancy and are best to assess fetal number, the pelvic canal and fetal presentation. Ultrasound is more sensitive in determining fetal viability.

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

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

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

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

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

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

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

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

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

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

**Atypical Hypoadrenocorticism**

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

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

Electrolyte Disorders

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

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

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

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

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

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

Initial Assessment

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

- **Airway**—Is the patient having difficulty breathing? Are there mandibular injuries that are interfering with the airway? Has a penetrating wound disrupted the larynx or trachea? Obstruction of the upper airway typically results in a slow, deep (obstructive) breathing pattern.
- **Breathing**—Is the patient dyspneic? What is the color of the mucous membranes? Is there evidence of thoracic penetration or is there a flail chest? Pulmonary contusions, pneumothorax, diaphragmatic hernia, and broken ribs can all result in a rapid, shallow (restrictive) breathing pattern.
- **Circulation**—Is there evidence of hemorrhage? Is the hemorrhage arterial or venous? How large is the swelling associated with the extremity fracture? Are the mucous membranes pale and tacky? Are the femoral pulses weak and rapid? Are the extremities cold? Is the abdomen distended?
- **Disability**—Is there evidence of neurological injury? What is the posture of the animal? Is the animal bright, alert and responsive? Does the animal respond to painful stimuli? Are the pupils dilated, constricted, of equal size, and responsive to light? Is there an extremity fracture that might threaten a peripheral nerve?
Management of the life-threatening problems identified during the primary survey is continued as shock therapy begins. The secondary survey identifies all other problems related to the trauma. The entire animal's body is examined again from head to toe. Necessary diagnostic samples are collected and submitted. Only when the patient is stable are indicated radiographs taken. In-depth management of the patient's less life-threatening injuries is undertaken in the definitive care phase. The fractures are stabilized, and careful inspection for "hidden" injuries is begun.

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

**Pulmonary Contusions**

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

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

**Pneumothorax**

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

**Fractured Ribs**

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

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

Intracranial Injuries

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

Spinal Cord Injury.

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

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

**Less life-threatening emergencies**

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

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

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

**Abdominal Trauma**

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

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

With biliary injury, the clinical signs of icterus are often delayed 4 to 6 weeks. If the abdominal fluid bilirubin is approximately 30 times greater than peripheral bilirubin, then surgical exploratory will be required to close the lacerated organ and lavage the abdomen. This surgery is not considered an emergency procedure. With urological injury, the packed cell volume of the abdominal fluid will be lower than the peripheral packed cell volume due to hemodilution with urine. The diagnosis is confirmed by comparison of abdominal fluid urine nitrogen or creatinine to peripheral blood values collected at the time of the abdominocentesis.

Emergency management of intraperitoneal rupture of the bladder, urethra, and/or ureters involves drainage of the abdominal fluid via an indwelling catheter until the patient is sufficiently stable to undergo anesthesia and surgical repair. Prior to surgery, contrast studies of the kidneys, ureter, and bladder should be performed to assess the severity of injury using an excretory urogram. If there is evidence of lower urinary tract injury, positive contrast urethrography and cystography may be necessary.

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

**Hypothermia, acidosis and coagulopathy**

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

**Correction of Coagulopathy**

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

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

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

**Tranexamic acid and CRASH-2**

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

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

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

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<th>ε-Aminocaproic acid</th>
<th>Colorado State University</th>
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<td><strong>Choose one of 3 Protocols:</strong></td>
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<td>• 50mg/kg diluted 1:10 in 0.9% saline</td>
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<td>• 15-40mg/kg IV bolus then 500-100mg PO q8hr</td>
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Hemostasis, transfusion, and the ideal red cell to plasma ratio

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

Antivenom is the definitive treatment for most envenomations. Most commonly used for snakebite, products are also available for some poisonous fish, insect, spider and scorpion envenomations. In the past decade, we have seen new antivenom products compete for use in the treatment of crotaline (viper) snake envenomation. They have addressed some of the availability issues and other concerns with the product we have been using for decades. In addition to antivenom, other treatments continue to come forward to address this common emergency. This lecture will focus on the antivenoms available for use against pit vipers in North America. We will discuss crotalidae polyvalent (ACP) equine origin whole IgG, Crofab® ovine origin crotalidae polyvalent immune F(ab')1, Antivipmyn® equine origin F(ab')2, Venomvet™ equine origin F(ab')2, and PoliVet-ICP® equine origin polyspecific antivenom. I will also discuss a newer, untested plasma product, Rattler Antivenin, Crotalidae, Polyvalent, Equine Origin.

Venom

Francisco Redi, Italian physician and naturalist, wrote in 1664 “the venom of the serpent consists of a yellow liquor contained in a bladder, at the bottom of its tooth, which liquor, upon its biting, by the pressure of the bladder, is forced through a tube within the tooth, into the wound, and thence ensue direful effects”. These conclusions were challenged by Moyse Charas, a French apothecary, who considered venom a harmless saliva and attributed the virulence to the enraged spirits of the viper. Charas tasted the venom, fed dogs and chickens venom soaked bread and wrote “no mischief at all followed it”. By the 1700’s venom was seen for what it was, a true toxin. The English physician Richard Mead observed that “venomous animals, when they bite or sting, inflict a wound and instill into it a drop of liquor which infects the fluids of the nerves, and by this means inflames the membranes.” In 1727 venom science came to America when Captain G. Hall published an observational report in the Philosophical Transactions of the Royal Society. He allowed a 4-foot-long rattlesnake from South Carolina to bite 3 dogs one day, two dogs, a cat, and a chicken four days later, and a bullfrog another chicken and a common black snake a week later before making the snake bite itself. Captain Hall observed many of the clinical differences from one bite to the next that we understand today.

Venom has a few important functions for the snake. It plays offense by aiding in the capture and digestion of large and agile prey. Large teeth and a painful bite can work to defend the snake against the birds, larger mammals and other snakes that might prey on them.
Antivenom

Toward the end of the nineteenth century, a student of Louis Pasteur named Albert Calmette saw the devastating effects of snake envenomation on the Indochina Peninsula near Saigon. A flood had forced monocled cobras (*Naja kaouthia*) into a village where he observed many cases of snakebite and four fatalities. Inspired by the work underway on vaccinations, he began work on antivenom. He eventually discovered the process of injecting horses with venom to produce antibodies, extracting the horse blood injecting it into the snake-bitten victim. The process has improved, but is much the same. The development of a crotaline antivenom contributed to the remarkable decrease in mortality rate from crotaline snakebite that has occurred over the past century. Mortality rates went from 5% to 25% in the 19th century to 0.1% to 1% for patients treated with antivenom in a health care facility.

In 1954, Wyeth Laboratories introduced Antivenin (Crotalidae) Polyvalent. This was a concentrated preparation of serum globulins from the blood of horses immunized with the venom of 5 new world crotalids. Ammonium sulfate precipitation was used to partially purify the serum. The resulting product contained 2 grams of equine protein, much of which was not immunoglobulin. This required administration of large volumes of antivenom to deliver sufficient immunoglobulin G (IgG) to neutralize venom. This exposure of horse proteins and whole, Fc portion containing, immunoglobulin, was likely responsible for delayed serum sickness and acute hypersensitivity reactions. Incidence of acute hypersensitivity reactions to the Antivenin (Crotalidae) Polyvalent (Wyeth) was reported to be 23% to 56%. This high complication rate led many clinicians to withhold this treatment when weighing risk versus perceived benefit. Human risk factors for serum sickness include those with a history of treatment with horse serum– derived antivenom, significant atopy or asthma, and those on beta blockers.

Modern antivenom contains immunoglobulins, collected and purified from sensitized horses and sheep. It is available as a whole IgG product, or as antibody fragments. Donor animals receive doses of selected venoms along with adjuvants to improve antibody response. Venoms are chosen to be representative as there may be significant variation in venoms even within a single species. Once desired antibody titers are reached, plasma is harvested, pooled and purified.

Fragmented antibody products by digesting the immunoglobulins with pepsin or papain. The globulins are then precipitated with ammonium sulfate or caprylic acid then put through an ultrafiltration process. Affinity purification results in a product that is 100% antivenom without extraneous proteins. The further purification significantly adds to the expense of the antivenom. The cost of these products is not only problematic in veterinary medicine, but indeed around the world. Snake antivenoms are the only specific treatment for poisonous snakebites and there is an urgent
need to ensure availability of safe, effective and affordable antivenoms, particularly to those in developing countries. The risk of snakebite envenoming is a public health hazard that many people in the rural tropics face on a daily basis. This is a common cause of occupational injury and disproportionately affects women and children in developing countries.

Figure 1: Whole IgG cleaved by pepsin to produce an F(ab’)2 antibody fragment. The cleaved FC fragments are removed in the purification process. This process is used in Antivipmyn® equine origin F(ab’)2 and Venomvet™ equine origin F(ab’)2.

Figure 2: Whole IgG cleaved by papain to produce two Fab antibody fragments. The cleaved FC fragment is removed in the purification process. This process is used in CroFab® - Crotalidae Polyvalent Immune Fab (Ovine).

Advantages of whole IgG antibodies are a longer elimination half-life, excellent movement through interstitial spaces, and cellular immune system clearing. Disadvantages are its small volume of distribution and longer half-life. By retaining the compliment fixing (Fc) portion of the antibody, it is also associated with more reactions. Fragmented antibody antivenins have a larger volume of distribution and deliver less protein per dose. Without the Fc portion and the rapid renal elimination
there is less risk of serum sickness. The shorter half-life becomes a disadvantage as signs of re-
venomation require continued dosing, even after discharge from the hospital.

**Cross Protection**

Snake venoms are a complex mix of compounds that can differ between species and even
within individuals of the same species. A major challenge to using antivenoms is how effective they
will be against the venom in the patient being treated. Ideally, an antivenom would be concentrated
IgG or active antibody fragments from animals sensitized with the specific specie that caused the
bite. Monovalent vaccines are available for some snakes, especially in the tropics and Australia, but
obviously require that the biting species is known. In North America, all the crotaline antivenom
products are polyvalent, meaning that they include antibodies harvested from animals sensitized to
multiple crotaline species. In Colorado, we deal exclusively with the Prairie rattlesnake (*Crotalus
viridis viridis*). Prairie rattlesnake venom is not included in the manufacture of most antivenom
products available but we have seen an acceptable clinical response when polyvalent products are
used. A new equine source frozen plasma antivenom product, Rattler Antivenin does include Prairie
rattlesnake venom. As an unpurified plasma product, it has yet to be tested alongside the other
products.

Arce et al. (2003) demonstrated that a Costa Rican polyvalent crotaline antivenom made
exclusively from South American snakes effectively neutralized various toxic activities of the venoms
of North American snakes including the Western Diamondback rattlesnake (*C. atrox*), the Eastern
Diamondback rattlesnake (*C. adamanteus*), The Prairie rattlesnake (*C. viridis viridis*), the Timber
rattlesnake (*C. horridus*), The Southern copperhead (*Agkistrodon contortrix*) and the Eastern
cottonmouth (*A. piscivorus*). It was effective in the neutralization of hemorrhagic and myotoxic
activities of all venoms studied but was found to be ineffective against the neurotoxins of the Mojave
rattlesnake (*C. scutulatus*). They concluded that a venom containing presynaptically-active
neurotoxic phospholipases A(2) related to "mojave toxin" needs to be introduced in the immunizing
mixture in order to increase the neutralizing scope of this product in North America. A challenge
accepted by the makers of the new version of Antivipmyn® equine origin F(ab')2.

**Crotalidae polyvalent (ACP) equine origin whole IgG - Boehringer-Ingelheim.**

Crotalidae polyvalent (ACP) was the stand alone antivenom used in veterinary medicine for
decades as the Fort Dodge product. It is the only veterinary licensed antivenom in the United States
and is now produced by Boehringer-Ingelheim. Horses are injected with the venom from two North
American snake species, the Western Diamondback rattlesnake (*C. atrox*) and the Eastern
Diamondback rattlesnake (*C. adamanteus*) in addition to venom from two South American snake
species, the Fer-de-lance (*Bothrops atrox*) and the South American rattlesnake (*C. durissus*). It is a
whole IgG antibody containing other, nonimmunoglobulin proteins including albumin. Only 20% of the IgG is active antivenom. The incidence of both acute reaction rates and delayed serum sickness are much higher with this antivenom than any of the others (approaching 50% in humans). Antivenom components are retained for extensive periods of time in the recipient. This is a lyophilized product that takes significant effort to reconstitute.

**CroFab® - Crotalidae Polyvalent Immune Fab (Ovine). BTG International Inc.**

Crotalidae Polyvalent Immune Fab (Ovine) was the first fragmented antibody antivenom commercially available in the US for people. It is made from pooled venom from Western (*C. atrox*) and Eastern Diamondback (*C. adamanteus*), and Mojave (*C. scutulatus*) rattlesnakes. It also contains venom from the Cottonmouth (*A. piscivorus*). CroFab® is the only polyvalent crotaline antivenom containing the venom of the Mojave rattlesnake (*C. scutulatus*) approved for use in the United States though the new version of Antivipmyn® equine origin F(ab')2 also contains Mojave antivenom, see below. Ovine donors are injected with the venom from only one snake species creating monovalent antibodies. These antibodies are then combined to create the final, polyvalent, product. The short half-life created some problems with recurrent signs of envenomation. Lavonas et al. (2004) reported CroFab® use with human patients bitten by copperhead snakes (*A. contortrix*) saw most local tissue effects of envenomation respond promptly though some treatment failures occurred. They noted recurrence of swelling and defibrination syndrome. Late coagulopathy, even after hospital discharge, could occur or recur after clearance of Fab antivenom. This delay sometimes lasted more than 2 weeks. Other reports of serious, even fatal, bleeding complications were associated with recurrence phenomena. Clinicians need to be prepared to follow-up with additional testing, interventions and re-hospitalization. CroFab® is a lyophilized product.

**Venomvet™ equine origin F(ab')2 - MT Venom, LLC, Argentina and Antivipmyn® equine origin F(ab')2 - Veteria Laboratories, Mexico**

Venomvet™ is an Argentinian product that is now licensed in the United States for use in dogs. Venomvet™ uses donor horses immunized with Central American rattlesnake (*C. durissus*) and the Fer-de-lance (*B. asper*) along with the Bushmaster viper (*Lachesis muta*). Cross protection for this product provides a positive clinical response for most North American crotaline bites with the possible exception of the more neurologic venoms found in the Mojave rattlesnake (*C. scutulatus*) and the Southern Pacific rattlesnake (*C. oreganus helleri*).

Antivipmyn® is an Equine F(ab')2 antivenom made in Mexico that has been used by veterinarians in the United States who have obtained import licenses. Antivipmyn® recently updated its formula and now includes antibodies against 10 North and 12 South American snakes. In addition to both diamondback rattlesnakes, *C. atrox* and *C. adamanteus*, horses are sensitized to four
Agkistrodon species, the Timber rattlesnake (C. horridus), and the North American snakes with a high concentration of neurologic acting venom, the Mojave rattlesnake (C. scutulatus) and the Southern Pacific rattlesnake (C. oreganus helleri).

Neither of these products are affinity purified so they are not as expensive as CroFab®. They do contain immunoglobulin fragments against antigens other than snake venom. Extraneous proteins are removed in the ultra-filtration and precipitation process reducing many unwanted side effects. While there is more foreign protein injected than CroFab®, there is less than ACP. F(ab')2 fragments clear the body faster than whole IgG but slower than CroFab®. This size may prove beneficial. Bush et al. (2015) hypothesized that since F(ab')2 immunoglobulin derivatives are larger and have longer plasma half-life than do Fab, F(ab')2 antivenom would be superior to Fab in the prevention of late coagulopathy following treatment of human patients with Crotalinae envenomation. They found that longer-half-life F(ab')2 antivenom, with or without maintenance dosing, reduced the risk of subacute coagulopathy and bleeding following treatment of envenomation. Antivipmyn® is lyophilized but mixes easier than ACP. Venomvet™ is a liquid.

PoliVet-ICP® equine origin polyspecific antivenom – Instituto Clodomiro Picado Costa Rica

Polyvet-ICP® is a whole IgG product made in Costa Rica. Like Antivipmyn®, no North American species are used to inoculate the donor horses. Snakes used are the Fer-de-lance (B. asper), the Neotropical rattlesnake (C. simus), and the Bushmaster (L. stenphrys). Precipitation and purification results in a IgG product without extraneous protein. Like ACP, only 20% of the IgG is antivenom. It is not lyophilized, making administration easier, but does require refrigeration. Studies suggest it may be effective against some North American pit vipers although, as with the two F(ab')2 products, its efficacy against neurotoxic crotaline snakes like the Mojave rattlesnake (C. scutulatus) is questionable.

Rattler Antivenin, Crotalidae, Polyvalent, Equine Origin – MG Biologics

Rattler Antivenin is an equine-derived plasma based Crotalidae polyvalent antivenin. The product is plasma from equine donors vaccinated against the Western Diamondback rattlesnake (C. atrox), the Eastern Diamondback rattlesnake (C. adamanteus), the Prairie rattlesnake (C. viridis viridis) and the Mojave rattlesnake (C. scutalatus). As plasma, it comes frozen so it must be handled like other frozen plasma products. As an equine plasma, it carries with it the potential for hypersensitivity reactions. The product’s package insert has the following: “In one in-house study of six healthy dogs, the most common adverse events reported were mild to moderate allergic skin reactions around the injection site, which occurred in all six patients. Additionally, two patients experienced an increased respiratory rate and increased heart rate, while one patient experienced increased respiratory rate and one other patient experienced a decrease in blood pressure.”
Canine parvovirus (CPV) is a serious and often fatal viral illness that most of us are all too familiar with. Canine parvovirus is still a common emergency continuing to fill isolation wards and critical care units with serious yet predictably complicated cases. There is a range of presentations from mild GI upset to shock or organ failure from massive dehydration and sepsis. CPV presents small animal practitioners, shelter veterinarians, and emergency/critical care clinicians with a steady supply of critically ill patients suffering a variety of problems related to gastrointestinal dysfunction, sepsis, and the systemic inflammatory response syndrome (SIRS). Untreated or undertreated, CPV infection can rapidly progress with a mortality rate that exceeds 90%. With hospitalization and aggressive supportive care the mortality rate can be reduced to between 0 and 30%. Survival in affected dogs can vary depending on place of treatment, with higher survival rates reported in tertiary care hospitals compared to private practices.

Epidemiology

Canine parvovirus disease emerged in 1978 as a severe gastroenteritis of dogs characterized by depression, anorexia, vomiting, hemorrhagic diarrhea and leukopenia. Canine parvovirus 2 (CPV-2) was identified later as the causative agent of the enteritis as well as of sporadic cases of myocarditis in neonatal puppies. Canine parvovirus 2 is believed to have emerged from feline panleukopenia virus or from a parvovirus of another wildlife species was the first variant of this virus associated with the development of hemorrhagic diarrhea. By 1985, CPV-2 was largely replaced by 2 more virulent strains of parvovirus, CPV-2a and CPV-2b. More than 80% of the isolated cases of CPV in the United States today are CPV-2b. Parvoviridae are small, nonenveloped, single-stranded DNA viruses that replicate in actively dividing cells. These viruses are ubiquitous and hardy, persisting for many months in the environment.

The earliest hematological abnormality seen in CPV patients is a lymphopenia associated with viral replication in lymphoid tissues 3 days post inoculation in experimental infections. Neutropenia is also seen in severely affected and appears to occur shortly after severe diarrhea begins. At least one study found significant inverse correlation between survival and white blood count, suggesting the need for aggressive therapy in cases with leukopenia. In experimental infection, profound neutropenia was seen in the most severely affected dogs after the appearance of clinical enteric disease (vomiting and diarrhea).

The parvovirus affects rapidly dividing cells, first replicating in the lymphoid tissues before moving to the plasma. Secondary replication occurs in the bone marrow, intestinal crypt epithelium and in neonates, the myocardium. Pathologists have described toxic and degenerative changes in the bone marrow of these dogs with depletion of the storage pool of mature neutrophils. These changes can be seen with other non-specific processes such as severe septicemia and endotoxemia. CPV induced neutropenia with depletion of the marrow storage pool may be the result of an accumulation and exudation of neutrophils at the intestinal mucosa. The damaged intestinal
mucosa can allow egress of mature neutrophils while giving gram-negative bacteria and endotoxins the means enter the general circulation. The resulting mortality and morbidity results from the viremia, gram-negative bacteremia and endotoxemia.

Whether the cause of the neutropenia in severely affected dogs is the direct destruction of myeloid elements or due to increased loss across damaged intestinal epithelium, neutropenia impacts the clinical course of the disease and may affect survival.

**Outpatient Management**

Mildly affected dogs are often treated as outpatients with subcutaneous crystalloid fluids and dietary restriction. Food can be withheld for 12–24 hours for rest and healing of the GI tract though it should be noted that enteral nutrition is very important in the restoration of the intestinal lining. Water should also be cautiously withheld when there is a history of vomiting provided the patient is stable enough to absorb subcutaneous fluids.

Clinically unstable patients that may be dehydrated, tachycardic, obtunded or showing other signs of circulatory shock should have an IV catheter placed for intravascular volume resuscitation. If financially possible, an initial electrolyte panel should be obtained to determine the degree of hypokalemia or hypoglycemia. Isotonic crystalloid fluid should be delivered over 15-20 minutes, with continuous assessment of heart rate, pulse quality and clinical response. Additional intravenous fluids may be required. Severe hypoglycemia should be treated with 25% dextrose at a dose of 1-2 ml/kg IV. External warming should be provided to help promote absorption of the SQ fluids and to maintain a rectal temperature above 99 °F.

After cardiovascular resuscitation and restoration of normal glucose levels and body temperature, patients may be discharged on a subcutaneous protocol. At Colorado State University, we will use crystalloid fluids (120 ml/kg/day) divided TID (40 ml/kg/dose) with extra fluids to correct dehydration over 24 hours. Divide the amount of fluids needed to rehydrate the patient by 3, and add that amount onto the maintenance SQ fluid dose for the next 3 doses. We will not add additives (such as dextrose or KCl) to the subcutaneous crystalloid fluids. If any of SQ fluids remains at the next treatment, we give partial dose of SQ fluids or withhold additional SQ fluids that treatment period. To cover for bacterial translocation and to prevent sepsis, Cefovicin is administered once at 8 mg/kg SQ while at hospital. For its visceral analgesic and antiemetic properties, Maropitant is administered at 1 mg/kg SC q24h for the duration of treatment period. Feeding should begin as soon as the patient is stable and nausea is controlled. Syringe feed small amounts of Hill’s a/d or similar diet at q6h (1 ml/kg PO), as tolerated by patient.

Motility modifying drugs should be used with caution. Anticholinergic antidiarrheal drugs may cause gastric atony and small intestinal ileus. This can increase the risk of bacterial overgrowth and intussusception. Synthetic opioids and narcotic analgesics can also inhibit the flow of intestinal contents that may also facilitate microbial proliferation and septic complications.

In dogs with uncontrolled visceral pain, buprenorphine is given at 0.02 mg/kg SQ as frequently as q6-8h. Preisner et al reported 20% of dogs on the above protocol required
buprenorphine. Dogs with uncontrolled nausea should receive ondansetron at 0.5 mg/kg SQ TID. In Preisner’s study, 20% of the dogs required ondansetron.

If it is possible, blood glucose and electrolytes should be rechecked. Oral glucose supplementation should be provided for hypoglycemia using 1-5 ml of corn syrup every 2-6 hours. Potassium supplementation should be provided to dogs that have a serum K+ < 3.4 mEq/L using oral Tumil-K at 1-2 tsp per 10 kg, every 4-6 hours. Oral sugar syrup was needed in 75% and oral potassium supplementation was needed in 60% of dogs in Preisner’s study.

Outpatient Survival.

Preiser et al. found 16/20 (80%) of dogs randomized to the outpatient protocol survived, compared to 18/20 (90%) of dogs managed as inpatients. Two of the outpatient protocol dogs died and one was euthanized before they could be transitioned to the in-patient protocol, a fourth was transitioned to inpatient treatment and survived. A prospective study utilizing a similar outpatient protocol at Penn found that approximately two thirds of the dogs survived. These survival numbers excluded dogs that were severely moribund or in severe shock at presentation. This survival data is still under review and is subject to refinement.

In-Hospital Care

Patients that fail outpatient management due to persistent or recurrent diarrhea and/or vomiting, and puppies that initially present with more severe clinical signs, including severe dehydration/hypoperfusion, fever, abdominal pain, and protracted diarrhea and/or vomiting should be hospitalized and managed aggressively. Outpatients with, worsening clinical symptoms should be evaluated and, if possible, hospitalized. Some of the criteria we used for hospitalizing these patients includes progressive dehydration, decline in mentation, and fever > 104°F. One dog out of the 20 in Preisner’s outpatient protocol was transitioned to the inpatient protocol and survived.

Antibiotics

Normal resident microbial flora of the intestinal tract includes anaerobic bacteria, which outnumber the aerobic gram-negative organisms. The normal anaerobic flora on the mucous layer adjacent to the epithelial cells can prevent the adherence of other potential pathogens. Antibiotic therapy should not simply target the anaerobes. Instead, the clinician should employ a balanced approach toward both gram-negative and anaerobic pathogens. The use of antibiotics is controversial with simple diarrhea. However, with severe hemorrhagic diarrhea, as seen with CPV, the clinician must assume the patient has a serious loss of intestinal mucosal barrier integrity and parenteral bactericidal antibiotic therapy is indicated. The goal of antibiotic therapy is aimed at eliminating enteric bacteria that have passed through the mucosa, entered the bloodstream, and occupy portal and pulmonary circulations. Animals with fecal cultures positive for bacterial pathogens may be treated according to the sensitivity pattern of the culture. Animals with positive blood cultures should have their antibiotic regimen refined based on the organisms identified.
Other Treatments

The high cost associated with treatment for CPV has led to investigation of alternative treatments and home remedies. Therapies have included use of hyperimmune plasma theorized to provide passive immunotherapy with immune CPV plasma obtained from dogs with high titers of anti-CPV antibodies has been proposed. Passive immunotherapy has shown efficacy in the treatment of several diseases including tetanus and Clostridium difficile infection. In the case of CPV enteritis, the rationale for use of CPV immune plasma is that the infused antibodies would neutralize free virus in plasma, slow viral spread, and reduce new infectious virions coming from infected cells. Immune plasma has reported previously to improve survival and reduce vomiting and diarrhea in dogs with experimentally induced CPV infection when administered immediately after viral inoculation. Macintire et al. (1999) reported that administration of concentrated lyophilized canine IgG significantly decreased hospitalization duration in dogs with CPV enteritis.

At Colorado State University, we had routinely administered a single, fixed dose of CPV hyperimmune plasma as adjunctive therapy for years before challenging anecdotal success with a controlled study. Bragg et al. (2012) published a prospective, randomized, double-blinded, placebo controlled clinical trial of CPV cases between March 2008 and September 2009 that showed no clinical differences in the patients receiving hyperimmune plasma. While we expected that anti-CPV antibodies might clear CPV rapidly from the bloodstream, we found that while viremia remained relatively constant in the placebo-treated group, it actually increased over the first 3 days in the CPV immune plasma-treated group. The magnitude of CPV viremia was not significantly different between the two treatment groups.

Other proposed therapies have included human recombinant granulocyte colony stimulating factor, equine LPS antitoxin, recombinant bacterial/permeability-increasing protein, interferon ω, and oseltamivir. However, all of these adjunctive treatments have failed to show a significant improvement in outcome. The only treatment that has been shown to date to improve treatment outcomes is early use of enteral nutrition, which has changed the paradigm of withholding food from these patients.
What is the outpatient treatment protocol utilized for the treatment of parvoviral enteritis at Colorado State University?

Introduction

Funding to evaluate the study developing this outpatient treatment protocol was provided by Zoetis Animal Health.

This randomized clinical study will be presented as an oral abstract at the American College of Veterinary Internal Medicine Forum, Seattle, WA in June, 2013.

The treatment guidelines provided within this protocol are only to be used under the knowledge and supervision of a licensed veterinarian.

This protocol is not intended to be a substitute for the gold standard of care (hospitalization and administration of fluids/medications intravenously), but rather used as an alternative for clients that cannot afford the recommended treatment protocol.

In the previous study, the survival rates for the standard of care protocol and the outpatient protocol were 90% and 80%, respectively.

Standard of care treatment should be offered and refusal to follow that protocol documented in the medical record prior to offering this as an alternative.

The faculty associated with this outpatient protocol will not assume any responsibility for the outcome or complications associated with the use of this protocol.

Initial Stabilization

Upon presentation to the hospital, all dogs should have an IV catheter placed for intravascular volume resuscitation.

An initial electrolyte panel should be obtained to determine the presence or severity of hypokalemia or hypoglycemia.

Use the standardized chart (Table 1) to determine the intravascular volume loss to be replaced

- Isotonic crystalloid boluses should be delivered over 15-20 minutes, with subsequent reevaluation of cardiovascular parameters.
- Additional IV fluid resuscitation should be performed at the discretion of the veterinarian.
- Based on the electrolyte concentrations, 25% dextrose can be supplemented IV (1-2 ml/kg) based on the presence and degree of hypoglycemia.

After cardiovascular resuscitation and restoration of normoglycemia, the outpatient portion of the study is entered.

Basic outpatient protocol

Start subcutaneous crystalloid fluid therapy immediately after IV fluid resuscitation.

- Normosol-R (120 ml/kg/day) divided TID (40 ml/kg/dose)
- In addition, replace dehydration over 24 hours

Use the standardized chart (Table 2) for determination of hydration status.
Divide the amount of fluids needed to rehydrate the patient by 3, and add that amount onto the maintenance SQ fluid dose for the next 3 doses.
Do not add additives (such as dextrose or KCl) to the crystalloids.
Provide aggressive external warming to help promote absorption of the SQ fluids.
Monitor rectal temperature to maintain ≥ 99 °F.
If part or all of the previous dose of SQ fluids remains at the next treatment, only give partial dose of SQ fluids (subjectively determined) or withhold additional SQ fluids that treatment period.
Cefovicin is administered once at 8 mg/kg SQ once while at hospital.
Maropitant is administered once at 1 mg/kg SC q24h for the duration of treatment period.
Syringe feed small amounts of Hill’s a/d q6h (1 ml/kg PO), as tolerated by patient.

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

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

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

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

Table 2. Determination of dehydration to be replaced over the first 24 hours. Liters of crystalloid to be replaced are determined by multiplying % dehydration by body weight (in kg). As an example, a 12kg dog that is 5% dehydrated would need 0.6L (600 mL) replaced over the first 24 hours. This 600 ml would be divided into three doses (200 ml each), and added onto the maintenance SQ fluid dose to be administered (480 ml + 200 ml = 680 ml), for the next three doses.

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

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

Tim B. Hackett DVM MS DACVECC
Colorado State University

Introduction

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

Initial Assessment

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

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

BREATHING – Is the animal breathing? Are the breaths of adequate rate and depth? Is oxygen getting from the lungs into the tissues? Observation, auscultation, and examination of the mucous membranes can answer these questions. If the mucous membranes are pale or cyanotic, supplemental oxygen should be provided immediately. Some poisons can cause respiratory depression and significant hypoventilation. Antiemetics such as apomorphine and xylazine can also result in cardiopulmonary depression. Simply supplementing oxygen can correct the hypoxia associated with hypoventilation however significant hypercarbia (PaCO2 > 50 mmHg) and respiratory acidosis may persist. A ventilator may be required to support the patient until normal function returns. Arterial blood gas analysis is required to identify hypoxia, hypoventilation and acid base disturbances associated with altered respiratory function.
CIRCULATION – Check pulse quality, heart rate and rhythm. If pulses feel weak or irregular, and electrocardiogram should be evaluated. Patients in shock should have an intravenous catheter placed, blood samples drawn and fluids ready to administer. If the heart rate is rapid and the pulse quality weak, Shock volumes of crystalloid fluids should be given (90 ml/kg over an hour in a dog, 40 ml/kg in a cat). Patients should be continually reevaluated and therapy changed as needed. With aggressive fluid therapy we should expect the heart rate to slow, the pulses to become stronger, and organ perfusion to improve. Clinically, the patients color and capillary refill should improve and the animal should act more alert. Packed cell volume and total solids should be rechecked after half of the total shock volume has been given. If the packed cell volume has dropped below 25-30% whole blood, or packed red blood cells may be needed. If the total solids drop below 3.5 gm/dl or less than half of the starting value, consider a colloid such as plasma, hydroxyethyl starch or dextrans.

Neurologic Complications

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

Treatment goals

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

Topical Poisons

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

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

Ingested Poisons

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

Emesis

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

For vomiting to occur there must be enough material in the stomach to forcibly expel. If an animal has ingested a small amount of a toxic substance, feeding a low fat gruel may be beneficial.

Emesis should not be induced in depressed or weak patients; the risk of aspiration is too great to justify the maneuver. Patients that have ingested caustic
compounds such as cleaning solutions, acids, or alkalis should not be forced to vomit. These compounds can burn the esophagus, leading to the formation of strictures. Caustic solutions should instead be diluted with water or milk and activated charcoal solutions. Petroleum products can be very viscous so that emesis and lavage can result in regurgitation, vomiting, and aspiration. Activated charcoal may be indicated in significant ingestion with kerosene and terpentine. In general petroleum compounds are poorly absorbed and pose little threat to life as long as they stay out of the lungs. Care should be directed at avoiding aspiration.

**Gastric Lavage**

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

**Activated Charcoal**

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

**Fluid Diuresis**

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

**Miscellaneous treatment and supportive care**

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

**Dialysis** is useful to remove small water-soluble drugs and poisons with low protein binding are ideally suited for removal by dialysis. Some of these compounds (e.g. ethylene glycol) are not readily bound by activated charcoal so that dialysis is an alternative worthy of serious consideration by the emergency clinician.

While hemodialysis is now being used successfully in veterinary medicine, the manpower, expertise and equipment required limit its use to specialized referral institutions. Peritoneal dialysis involves relatively simple equipment and although time consuming can be performed in most any practice. Newer continuous renal replacement therapy (CRRT) units can extract protein bound toxins through an albumin countercurrent mechanism.

**Supportive Care**

**Intravenous lipid (fat emulsion) IFE**, has shown promise in recent animal and clinical case studies as an effective antidote for treating cardiotoxicity from overdose of lipophilic drugs, such as local anesthetics and psychotropic agents. Several mechanisms have been proposed to explain why IFE might work as an antidote to an overdose of myocardium-poisoning drugs, including calcium channel blockers, beta-blockers, digoxin, local anesthetics, cyclic antidepressants, antipsychotics, atypical antidepressants, and mood stabilizers.

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

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

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

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

Canine Dyspnea

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

Initial observation and diagnostics

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

Imaging

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

Thoracocentesis

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

**Airway cytology**

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

**Laryngeal paralysis** can be either congenital or acquired and is a common cause of emergency visits in large breed dogs. The paralysis may be either unilateral or bilateral. Acquired laryngeal paralysis is more common with many proposed etiologies. The recurrent laryngeal nerve innervates the arytenoid processes of the larynx. One of the longest nerves in the body, it is susceptible to a variety of degenerative processes. Damage to the nerve anywhere along its course by trauma, surgery, neoplasia, polyneuropathy or even hypothyroidism can lead to a loss of innervation of the intrinsic laryngeal muscles. Animals with laryngeal paralysis will present with varying degrees of exercise intolerance, stridor, voice change, inspiratory effort, cyanosis and hyperthermia. They will have a pronounced inspiratory stridor with a loud, deep (obstructive) breathing pattern. Often an obvious inspiratory wheeze will be heard loudest over the larynx. Diagnosis is by direct examination under a light plane of anesthesia. A low dose (1-2 mg/kg) of propofol is administered to allow the mouth to be held open while visualizing the glottis. If the animal becomes apneic with the sedative a 1 mg dose of doxapram HCL IV can initiate a large breath. Normally the arytenoid cartilage abducts on inspiration. With laryngeal paralysis the arytenoids may actually be drawn together during inspiration causing inflammation and edema. Laryngeal paralysis may be unilateral or bilateral. Increased airway pressures can lead to everted laryngeal saccules further compromising the laryngeal lumen.
Because panting is such an important method of controlling body temperature, subclinical laryngeal paralysis may only become evident on hot days or following strenuous exercise. The body temperature can quickly climb to dangerous levels, necessitating treatment for heat stroke. Dyspnea from upper airway obstruction can cause the animals to become anxious and more dyspneic. A vicious cycle begins as the more distressed they become, the harder they try to breathe. Handling these animals can be difficult and often the best treatment is sedation. Acepromazine is a predictable sedative. A dose of 0.02 – 0.04 mg/kg not to exceed 0.25 mg will break the cycle of distress. Acepromazine should be given cautiously in dehydrated or shock patients as it may cause a drop in blood pressure. Patients with prolonged hyperthermia should be hospitalized and observed for complications. The kidneys, GI tract, liver and nervous tissue can all be damaged by excessive heat. Disseminated intravascular coagulation is another common complication. Once the patient is stable and signs of heat stroke have resolved definitive treatment for laryngeal paralysis can proceed.

**Pulmonary Edema.** Non-cardiogenic pulmonary edema occurs occasionally in dogs and cats secondary to electric cord bites, sepsis, following near drowning or choking, snake bites, uremia, smoke inhalation, upper airway obstruction, and the adult respiratory distress syndrome (ARDS). Dogs that chew on electric cords often present with acute onset of dyspnea and oral burns, which may or may not be associated with dysphagia or ptyalism. The syndrome occurs most commonly in the young. Pulmonary edema develops rapidly, generally within hours. Common physical examination abnormalities include oral burns, dyspnea, and pulmonary crackles. Thoracic radiographs show mixed interstitial and alveolar patterns that are most prominent in the dorsal portions of the caudal lung lobes. The pathogenesis of edema is thought to be increased pulmonary capillary hydrostatic pressure and increased alveolar-capillary permeability. Increased pulmonary capillary hydrostatic pressure is likely due to a centrally mediated burst of sympathetic activity, which causes constriction of resistance and capacitance vessels leading to a shift of blood from the splanchnic viscera into the circulation. This ultimately results in overcirculation of the pulmonary vasculature. Increased peripheral vascular resistance increases pulmonary capillary hydrostatic pressure and pulmonary venous pressures increase as the left ventricle pumps against increased outflow resistance. Treatment includes administration of low dose narcotics, diuretics, and oxygen (mask, nasal insulation or oxygen cage). Morphine and other pure narcotic agonists at lower doses can have a good clinical effect. At low doses it sedates dyspneic animals while drawing excess fluid from the lungs via splanchnic vasodilatation.
The clinical signs and physical examination abnormalities associated with near
drowning, smoke inhalation, and snakebite are similar to those with electric cord bites with the
exception of oral burns. Historical findings confirm near drowning and smoke inhalation.
Puncture wounds and a swollen face or extremities may be found on animals with snakebite.
Administration of bronchodilators may also aid in the treatment of some cases. Smoke
inhalation causes dyspnea by inducing carbon monoxide poisoning and damage to respiratory
tissues by heat and noxious gasses. Laryngeal spasm, loss of ciliary function, decreased
surfactant activity, bronchospasm, increased alveolar-capillary permeability, impaired
phagocytosis, and sloughing of airway mucosa frequently occur. Bronchial patterns occur first
with interstitial and alveolar edema developing later if edema develops. Treatment is similar to
electric cord bite and near drowning.

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

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

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

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

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

**Pleural Space Disease.** Diseases of the pleural space cause a decreased tidal volume and a restrictive breathing pattern. Characterized by the rapid, shallow respirations and dull lung sounds, fluid and air in the pleural space can be diagnosed and treated by rapid thoracocentesis following the procedure above. Once removed, fluid can be examined to determine the likely cause. A negative thoracocentesis may be due to fibrous adhesions and small pockets of fluid. Diaphragmatic hernias may also cause significant pleural restriction but yield a negative tap. These patients should be given supplemental oxygen while ultrasound is performed or radiographs taken. Large volumes of air or fluid, continuous production of air, or suppurative inflammation are indications for tube thoracostomy. Pneumothorax and some inflammatory conditions may require continuous suction. Disposable suction devices are available to hook
onto surgical suction units. These “3-bottle” devices allow easy regulation of suction (20 cm H₂O desirable), a water trap to prevent air from being drawn into the chest should suction become interrupted, and a collection chamber to quantitate fluid production.

FELINE DYSPNEA

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

Observation and diagnostics

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

Thoracocentesis

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

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

Transoral tracheal wash

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

Pulmonary Edema.

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

Feline Bronchial disease.

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

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

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

Pleural Space Disease.

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

Large volumes of air or fluid, continuous production of air, or suppurative inflammation are indications for tube thoracostomy. Pneumothorax and some inflammatory conditions may require continuous suction. Disposable suction devices are available to hook onto surgical suction units. These “3-bottle” devices allow easy regulation of suction (20 cm H2O desirable), a water trap to prevent air from being drawn into the chest should suction become interrupted, and a collection chamber to quantitate fluid production.
<table>
<thead>
<tr>
<th>Type of Effusion</th>
<th>Protein (g/dl)</th>
<th>Cell count (/ul)</th>
<th>Etiology</th>
</tr>
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</table>
| Modified Transudate | <2.5           | <500-1000        | Right Heart failure  
|                  |                |                  | Pericardial disease  
|                  |                |                  | Hypoalbuminemia  
|                  |                |                  | Neoplasia  
|                  |                |                  | Diaphragmatic hernia  |
| Exudate | >3.0         | >5000            | Feline infectious peritonitis  
|     |              |                  | Neoplasia  
|     |              |                  | Diaphragmatic hernia  
|     |              |                  | Lung lobe torsion  
|     |              |                  | Pyothorax  |
| Chylous | >2.5         | >500             | Idiopathic  
|      |              |                  | Cardiomyopathy  
|      |              |                  | Heartworm disease  
|      |              |                  | Neoplasia  
|      |              |                  | Lung lobe torsion  
|      |              |                  | Tuberculosis  |
| Hemorrhage | >3.0        | >1000            | Trauma  
|       |              |                  | Coagulopathy  
|       |              |                  | Neoplasia  
|       |              |                  | Lung lobe torsion  |

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