The emergency clinician is frequently called upon to treat burn wounds secondary to thermal, chemical, electrical, or radiation injury. Most burn wounds seen in veterinary medicine are relatively minor, possibly because animals with severe burns and smoke inhalation are less likely to be rescued from the scene of a house fire. However, life threatening burns and inhalation injury are being seen with increasing frequency and the emergency clinician should therefore be familiar with their pathophysiology and management.

**Classification of burns**

Burns are commonly classified according to the extent of body surface involved and the depth of injury to the skin. Extent of injury is initially estimated in human burn patients using “the rule of nines”. This rule divides the adult human body into areas corresponding to 9% of the total body surface area, or multiples of 9%. For example, each forelimb comprises approximately 9% of total body surface area; each hind limb, 18%; head and neck, 9%; chest and abdomen, 18%; back, 18%; and perineum, 1%. Body surface area percentages vary in children, and as such, the rule of nines is not typically used in children less than 10 years of age. Although the rule of nines has been cited in veterinary texts, it seems similarly unlikely that these percentages accurately describe the majority of veterinary patients. Other methods of estimating extent of injury include serial halving (Do burns cover more than half the patient’s surface area? If not, do burns cover ¼-½ the surface area? and so forth), or measuring the burn area in centimeters and using a chart to calculate meters\(^2\) from the patient’s body weight in kilograms.

Depth of injury may be described as first-, second-, or third-degree, or using the more recent terms, partial- and full-thickness. First-degree burns involve only the epidermis (like a sunburn), and are bright red, non-blistered, and painful. First-degree burns typically heal within 5 days without scarring, and are therefore not included in the calculation of extent of burn injury unless they exceed 25% of body surface area. Second-degree, or partial-thickness, burns involve all epidermal layers and extend to various depths within the dermis. Superficial partial-thickness burns involve the epidermis and less than ⅓ of the dermis, and are characterized by blisters, pain, blanching in response to pressure, and intact hairs. The surface may appear moist, red, or mottled. Injuries of this depth typically heal without serious scarring within 2-3 weeks. Deep partial-thickness burns involve destruction of the deep dermal layers and may appear dry, or blistered and moist. As skin thickness is not uniform, partial-thickness burns may interdigitate with full thickness burns, appearing mottled-red intermixed with whitish areas. Deep partial-thickness burns do not blanch, lose hair easily, and heal more slowly, producing scarring and loss of function. They may easily progress to full-thickness injuries as a result of edema, infection, thrombosis, or mechanical injury. Third-degree, or full-thickness, burns involve destruction of the entire dermis, usually extending into the subcutaneous tissues. They are dry, leathery, lack sensation, and appear white or charred. Healing of these injuries can occur only by contracture and epithelial migration from the periphery, or through excision and grafting. Burn injuries that extend into the muscle, fascia, or bone can be seen as well, and are termed fourth-degree burns. These appear similar to third-degree burns, but may result in severe systemic illness if unrecognized due to severe underlying tissue necrosis. Depth of injury can be difficult to assess initially, and usually requires repeated evaluation over the first 24 hours for accurate determination. Once this information is collected, burned patients may be divided into minor, moderate, or severe categories for the purposes of treatment planning.

**Pathophysiology of burn shock**

Following severe burns (>20% TBSA), a severe systemic inflammatory response may develop within minutes, leading to cardiovascular collapse and multiorgan system failure if not quickly addressed. These systemic manifestations are driven by loss of the protective skin barrier, as well as release of inflammatory mediators from within the damaged tissues. Release of prostaglandins, leukotrienes, and other vasoactive substances leads to a diffuse “capillary leak” syndrome, increasing in proportion to size of burn injury, delay in initiation of resuscitation, age of the patient, and the presence of inhalation injury. This increased vascular permeability results in marked declines in effective circulating volume as well of the development of edema in injured and non-injured tissues. Edema is further exacerbated by the development of hypalbuminemia, resulting from loss of albumin through “leaky” vessels, compounded by decreased hepatic albumin production in favor of acute phase protein synthesis. Extensive tissue edema leads to tissue hypoxia at the junction between burned and non-burned tissues (the “zone of ischemia”), and may have adverse effects on depth of burn injury. Thromboxane $A_2$ and $B_2$ prostaglandins, cytokines, and reactive oxygen species are produced at the burn site and are associated with local ischemia and further tissue damage. Cardiac output decreases within the first eight hours of burn injury secondary to hypovolemia and myocardial depression associated with release of inflammatory mediators. Arterial blood pressure may be misleading however, as burn patients may have normal or increased blood pressure despite significant hypovolemia due to vasoconstrictive substances released from the burn wound.
Following successful resuscitation, microvascular leak typically “seals” after 18-24 hours. Hypermetabolic response develops during this time with near doubling of cardiac output and resting energy expenditure. Increased gluconeogenesis, protein catabolism, insulin resistance, and weight loss may also be seen. These changes are believed to result from increased cortisol, glucagon, catecholamine, and cytokine release, GI mucosal barrier dysfunction, bacterial translocation, burn wound sepsis, and heat loss. The hypermetabolic response typically persists until all wounds are closed, and continues for some time afterwards.

Sepsis is one of the major causes of death among burn patients. In addition to wound infections, respiratory infections, and catheter-related infections, decreased gastrointestinal perfusion in the first 24 hours following burn injury leads to compromised integrity of the mucosal barrier and allows passage of bacteria and endotoxin. Peak endotoxin levels have been reported to develop as early as 12 hours post-burn, and may contribute to the development of multiorgan failure. It has been reported that patients with extensive burns also have altered humoral and cell mediated immunity attributed to increased levels of cortisol and inflammatory mediators such as TNF, IL-1, and IL-6. This immunosuppression may further contribute to the development of septic complications in these patients.

Inhalation injury contributes significantly to morbidity and mortality in the burned patient. Smoke inhalation triggers release of thromboxane, causing pulmonary vasoconstriction and pulmonary hypertension. Chemical and thermal injuries directly damage the respiratory epithelium, leading to sloughing of the tracheobronchial mucosa, impairment of the mucociliary escalator, and formation of cellular casts that may obstruct the airways and promote bacterial growth. Disruption of respiratory epithelium and vascular endothelium leads to exudation of proteinaceous fluid into the terminal airways and further contributes to respiratory compromise, impaired surfactant production, and bacterial proliferation. Acute lung injury or ARDS may also result indirectly from systemic inflammation related to the burn wound or from sepsis arising from various sources including the lungs, burn wounds, GI tract, or catheters.

**Prehospital treatment of the burned patient**

The first consideration in treatment of the burned patient is to stop the burning process. Flames should be extinguished and any collars or harnesses that may become constrictive should be removed. Because the skin is slow to cool, the burning process may continue for some time after the patient is removed from the heat source. For this reason burned areas should be cooled with running water for up to 10 minutes. Alternatively, cool wet towels can be placed over the burn areas. Ointments should not be applied at this time as these may hinder the subsequent assessment of extent of injury. Cold water or ice should also not be used as this can rapidly decrease the patient’s body temperature and may contribute to increased wound depth by inducing vasoconstriction. To avoid hypothermia during transport, the patient should be wrapped in several clean, dry sheets or blankets.

**Primary and secondary surveys**

A primary survey should be performed to determine the extent of injury and to institute treatment as needed. Ensuring a patent airway and supporting breathing should be the first priority, followed by shock resuscitation. 100% oxygen should be administered to any patient suspected to have smoke inhalation injury to hasten the elimination of carbon monoxide. Intubation or emergency tracheostomy may be required if airway edema is severe. In the event of orotracheal intubation, tubes should be carefully secured, as worsening edema may make re-intubation more difficult.

Vascular access may be difficult in hypovolemic, burned patients. Ideally, short peripheral catheters should be placed in non-burned areas, though burned areas may be used in the first 24 hours. If burned sites are used for catheterization, the catheters should be removed within 24-48 hours due to bacterial colonization of these areas. Intraosseous catheters are another good alternative for patients in whom vascular access is limited. Central lines may be required in patients with large burns, those needing parenteral nutrition, or those requiring central venous pressure monitoring, but their use should be avoided whenever possible due to the risks associated with hypercoagulability in burned patients.

Following initial stabilization, a secondary survey should be performed to identify concurrent injuries. Patients should be assessed for neurologic injuries secondary to trauma, hypoxemia, or carbon monoxide poisoning. The abdomen should be assessed for compartment syndrome, gastric distension, or other traumatic injuries. The airways and thorax should be carefully ausculted for stridor, crackles, or wheezes, and adequacy of ventilation should be assessed. The face, oral cavity, and pharynx should be examined for the presence of burns or particulate debris that may indicate inhalation injury. Baseline radiographs should be obtained to evaluate for changes related to smoke inhalation or traumatic injury. Chest radiographs may be normal initially, or bronchial markings may be present. The development of pulmonary infiltrates or lobar consolidation may suggest pneumonia. Arterial blood gas evaluation is useful for determination of parameters related to oxygenation and perfusion. However, because both partial pressure of oxygen (pO₂) and oxygen saturation can be misleading in the presence of carbon monoxide (pulse oximetry will misread carboxyhemoglobin as oxyhemoglobin), cooximetry should also be performed if available to determine carboxyhemoglobin levels. Baseline complete blood count, serum biochemistry panel, and urinalysis should be obtained upon admission. The presence of myoglobinuria may indicate a need for higher fluid rates to avoid renal tubular damage. Coagulation testing should be performed, as burned patients may suffer from
hyper- or hypocoagulable states. Blood typing may be indicated if surgery is anticipated for large burns, as these procedures frequently result in significant blood loss. The eyes should be evaluated for the presence of conjunctivitis, particulate material, or corneal ulceration. Corneal ulcers are common secondary to thermal injury or abrasion by particulate material, so fluorescein staining should always be performed. A topical anesthetic such as proparacaine may be used to facilitate examination behind the third eyelids for foreign material, and the eyes should be copiously flushed with sterile saline. Corneal ulcers may be treated with triple antibiotic ophthalmic ointment and atropine ophthalmic drops.

**Fluid therapy**
The goal of fluid therapy in the burn patient is to restore and maintain perfusion to the tissues while keeping edema fluid to a minimum. The greatest amount of fluid loss in burn patients occurs during the first 24 hours as a result of increased microvascular permeability. Fluids given during this time rapidly leave the vasculature, with colloids having no benefit over crystalloids due to the leakiness of the endothelium. Crystalloids, such as lactated Ringer’s solution, are therefore usually the fluids of choice for the first 24 hours. Hypertonic saline, used in some human institutions to decrease crystalloid requirements, is also of questionable benefit and has been associated with adverse outcomes in burn patients. Fluid requirements can be estimated based on percentage of body surface area burned using the Parkland formula. LRS is given at 4 ml/kg x % TBSA, with one half of the calculated volume given within the first eight hours, and the second half given over the next 16 hours. The starting point is the time of injury, not the time of hospital admission. Urine output should reach 0.5-1 ml/kg/hr within the first three hours. If it falls below 0.5 ml/kg/hr, more fluid is needed.

Lasix should not be used to increase urine output, as this will further deplete effective circulating volume as well as invalidate the use of urine output as an indicator of shock resuscitation. If total resuscitation needs are estimated to exceed 6 ml/kg/% TBSA, central venous pressure (CVP) measurement should be performed to assess intravascular volume. If blood volume is assessed as adequate, dopamine (5-15 ug/kg/min) or dobutamine (3-10 ug/kg/min) may be added to maintain cardiac output and arterial blood pressure.

Many resuscitation formulas recommend adding colloids at 0.5 ml/kg/day x %TBSA after 24 hours, as colloids are more likely to be retained within the vasculature at that time. (Note: some formulas advocate colloid supplementation as early as 8 hours post-burn). Hetastarch, fresh frozen plasma, or albumin may be used, though it is interesting to note that albumin supplementation in burn patients has not been associated with decreased mortality nor mobilization of tissue edema within the first week. Crystalloids are continued only at doses needed to maintain urine output, approximately 1.5 ml/kg/day x %TBSA.

It is important to emphasize that these fluid formulas should be used only as guidelines, and should be frequently reevaluated and adjusted based on physiologic parameters. Additionally, because these formulas have been derived from experiences with human patients and experimental models in animals, they should be applied cautiously in clinical veterinary patients, and dose reduction may be appropriate in cats.

**Wound care**
Patients with small burns rarely develop overwhelming wound sepsis, and medical management for several days usually allows better determination of wound depth and extent. Wounds should be gently clipped of hair and then rinsed or soaked in dilute povidone-iodine solution. Animals with thick coats may hide more extensive wounds than initially suspected, so liberal clipping should be performed in these cases. After the wounds are cleaned, topical agents may be applied to decrease pain, prevent desiccation, and delay bacterial growth. Silver sulfadiazine is used most commonly as it has broad antibacterial activity, is soothing, and has no systemic effects. Eschar penetration is poor however. In contrast, mafenide acetate has excellent eschar penetration and similarly broad antibacterial effects, but can be painful when applied. Topical agents can be applied directly to wounds with a clean tongue depressor, or the burn can be covered with impregnated dressings. Gloves should be worn at all times during wound care to avoid spread of resistant organisms.

The choice of dressing is a much-debated topic. Of critical importance is the maintenance of a moist environment to promote rapid wound healing. This may be accomplished through the use of semi-occlusive dressings, or with various types of hydrogel shown to speed healing and to decrease scarring of partial thickness wounds. Wounds with heavy exudation may be managed with dry, absorbent bandaging material applied in layers. Following application of silver sulfadiazine, a non-adherent and porous inner layer is applied, allowing passage of fluid and exudates. Absorbent padding or gauze should then be applied, followed by an elastic outer layer. Bandages should be loose enough to avoid putting additional pressure on the wounds.

Patients with more extensive burns generally do better if full thickness wounds are excised within the first week, starting 24-48 hours following burn injury. Early wound excision has been shown to circumvent the development of wound sepsis and SIRS, attenuate the hypermetabolic response, and reduce morbidity and mortality, length of hospital stay, and pain in patients with large burn wounds. Burns >20% total body surface area may require staged procedures, and burns > 50% TBSA make closure with autograft impossible. Once autograft closure is no longer feasible, temporary closure may be performed using cadaver allografts, porcine xenograft, or synthetic skin substitute, though these procedures are not routinely performed in veterinary medicine. Research is currently underway to evaluate the use of synthetic membranes such as Integra (Integra Life Sciences, Plainboro, NJ) that mimic
vapor transmission characteristics of normal skin and allow fibrovascular ingrowth from the host, ultimately undergoing biodegradation.7

Prophylactic antibiotic usage is controversial as penetration of the eschar is unlikely and the potential for development of antibiotic resistance exists.8 As such, antibiotic therapy is generally reserved only for documented infections and should be based upon culture and sensitivity of full thickness eschar biopsies. Excision of eschar has been associated with bacteremia however, so intraoperative antibiotic administration has been recommended.

**Inhalation injury**

Management of smoke inhalation is typically supportive. The head should be elevated and excessive fluid therapy avoided to minimize development of edema. However, it should be noted that patients with inhalation injury typically have higher fluid requirements than those with burn injury alone due to increased severity of systemic inflammatory response. Bronchospasm may be treated with systemic β agonists such as terbutaline, or inhaled albuterol administered via spacer (Aerokat, Trudell Medical, London, Ontario). Prophylactic antibiotics have not been shown to reduce morbidity or mortality associated with smoke inhalation, and may contribute to resistant infections. Antibiotics should therefore be reserved for documented infections, and should be based on tracheal wash culture and sensitivity when possible.

Supplemental oxygen should be provided as needed, based on blood gas analysis. Carbon monoxide poisoning, if present, may be treated with hyperbaric oxygen therapy, but in most cases administration of 100% oxygen for 6 hours9 constitutes appropriate therapy without the increased risks and cost involved in transporting a critically ill patient to a facility with a hyperbaric oxygen chamber. Administration of 100% oxygen has been shown to shorten the half life of carboxyhemoglobin from several hours to approximately 74 minutes (range 26 to 148 minutes).10

If ventilation is required, lung protective strategies should be use to minimize ventilator induced lung injury. Peak airway pressures greater than 40 cm H2O and FiO2 greater than 0.60 should be avoided, using PEEP, faster rate, and permissive hypercapnea to maintain an oxygen saturation greater than 90% with a PCO2 less than 65 mmHg. Strict attention should be given to suctioning of airways, and asepsis should be maintained to minimize the likelihood of nosocomial infection.

**Nutritional support and the hypermetabolic response**

Nutritional support is an important component of burn care, and should ideally be provided as soon after resuscitation as possible. Enteral nutrition using a nasogastric or esophagostomy tube is ideal, as this is believed to decrease gut atrophy, possibly decreasing bacterial translocation and subsequent sepsis. Resting energy requirements may be calculated using the formula [RER= Weight (kg) x 30 + 70]. Although the use of an illness energy requirement calculation (IER) has largely fallen by the wayside in veterinary medicine, multiplying resting energy requirements by an IER of 1.3-1.7 may be appropriate in the burned patient to compensate for the anticipated hypermetabolic response. The use of such formulas has been shown to correlate poorly with actual energy requirements in both human and veterinary patients however, and as such, indirect calorimetry would be a more accurate method of determining resting energy requirements if available. Critically ill patients or those with very large burns may not tolerate their full nutritional requirements because of ileus or vomiting, and these patients may benefit from the supplementation of parenteral nutrition through a designated central line.

**Pain management**

Pain can be reduced initially using cool compresses and soothing ointments such as silver sulfadiazine. Once burn shock has been adequately controlled, narcotics may be administered. Pure agonists such as fentanyl (CRI: 3-5 ug/kg/hr), hydromorphone (CRI: 0.025 mg/kg/hr), or morphine (0.5-1 mg/kg SQ q4h) are recommended for patients with moderate to severe pain. Ketamine can be useful for the relief of somatic pain, and may be used in conjunction with narcotics at a constant rate infusion of 0.15-0.6 mg/kg/hr. Lidocaine may provide adjunctive analgesia in addition to free radical scavenging properties, and may also be added at a rate of 1.5-3 mg/kg/hr. If using constant rate infusions, a loading dose equal to the hourly rate should initially be administered.

**References**


Reproductive problems often arise after normal business hours, so it is not uncommon for them to fall into the domain of the emergency veterinarian. As most owners lack medical knowledge, they frequently look to the veterinarian to answer questions and to identify potential problems. The emergency clinician must therefore be familiar with normal reproductive behavior in addition to the common emergencies that may arise. With this goal, we will review the events surrounding normal parturition as well as the common complications that may develop during this period.

Normal reproductive physiology
Normal gestation length in the dog may range from 57-72 days from the time of first breeding, with an average length of 65 days.1,2 Because cats are induced ovulators, there is generally less variability in gestation length, which ranges from 63-65 days. Ovulation may not take place after the first breeding however, so in the event of multiple breedings, uncertainties with regards to gestation length may still be present in the cat. As the whelping date approaches, a number of clues may point toward impending parturition. Mammary development, vulvar enlargement, mucous vaginal discharge, and relaxation of the pelvic ligaments are early signs of approaching parturition. Onset of lactation may be noted in primiparous bitches within 24 hours of parturition, but in multiparous bitches may occur several days before parturition. A sudden drop in body temperature (≤2°C) is generally noted within 24 hours of parturition in dogs and cats as a result of decreases in progesterone levels, but this finding is not always reliable. In one recent study, nadir temperature occurred >48 hours before parturition in 24% of dogs, and an appreciable drop in temperature (>1°F) was not seen in 35% of dogs.4

Normal parturition proceeds in three stages. The first stage is characterized by subclinical uterine contractions and progressive dilation of the cervix. During this stage, which typically lasts for 6-12 hours, bitches may show signs of restlessness, apprehension, panting, nesting behaviors, hiding, and anorexia. Queens may be tachypneic, restless, and vocal, or may lay in their nesting boxes, purring. Active expulsion of the fetuses occurs during the second stage of labor. The first fetus is usually delivered within 1 hour of onset of stage 2 labor in cats, and within 4 hours in dogs, with subsequent deliveries every 15 minutes to 3 hours.5,6 Active straining generally results in expulsion of a fetus within 15 minutes. The entire process generally occurs over 2-12 hours, but may take as long as 24 hours with large litter sizes. The third stage of labor results in expulsion of the placenta. One placenta should be identified for each fetus delivered. Placentas are usually still attached to the fetus by the umbilical cord and emerge with the fetus, but may emerge within 15 minutes to several hours if they become detached. Lochia, a greenish vaginal discharge, indicates placental separation and may be seen during all stages of labor. Following parturition, the discharge gradually becomes red-brown, decreasing in volume over 4-6 weeks as uterine involution takes place.

Dystocia
Historical and physical exam findings that should prompt a clinician to suspect dystocia are as follows:1

- A definite cause is apparent (ie. fetus lodged in birth canal, pelvic fractures)
- Gestation is prolonged (>70 days) with no evidence of labor
- Temperature has dropped to <100°F and returned to normal with no evidence of labor within 24 hours
- Lochia is noted and 2 hours have elapsed without expulsion of a fetus
- Strong and persistent contractions fail to result in the delivery of a puppy within 30 minutes
- Weak and infrequent contractions fail to produce a fetus within 4 hours.
- More than 4 hours have elapsed since the birth of a puppy with no evidence of ongoing labor
- Signs of systemic illness or severe pain are present

Dystocia may result from either maternal or fetal factors that prevent delivery from taking place. Uterine inertia is the most common maternal cause of dystocia,3-9 seen when the myometrium produces only weak and infrequent contractions that fail to expel a normal fetus through a normal birth canal. Primary uterine inertia is considered complete when gestation that has exceeded its expected length with no evidence of progression into active labor. Primary uterine inertia is termed partial if the bitch initiates parturition and expels one or more healthy fetuses, but then subsequently fails to deliver the remaining fetuses as a result of myometrial fatigue. Uterine inertia may also be considered secondary if myometrial failure results from prolonged attempts to expel an obstructed fetus, and persists following relief of obstruction. Morphologic causes of dystocia are those in which an anatomic abnormality of the bitch or queen results in obstruction of the birth canal (eg. small birth canal, pelvic fractures)

Fetal factors that may result in dystocia include malpresentations, oversize, fetal malformations, and fetal death. Some of the commonly described malpresentations include transverse presentation, lateral or ventral flexion of the neck, anterior presentation with flexion of one or both forelimbs, posterior presentation with retention of both hindlimbs, and simultaneous presentation of two fetuses.
It should be noted that posterior presentations are considered to be a normal variation in dogs and cats, occurring in approximately 40% of deliveries. Fetal oversize is another potential cause of dystocia, most commonly seen with single pup pregnancies. Fetal death is an infrequent cause of dystocia, increasing the likelihood of malpresentation because of failure to rotate and extend the head and legs, which commonly occurs immediately prior to parturition. Fetal malformations are another potential cause of dystocia, with anasarca (generalized subcutaneous edema), hydrocephalus, cerebral and cerebrospinal hernias, abdominal hernias, duplications, and rib cage malformations among the more commonly noted.

**Diagnosis of dystocia**

Workup of a patient that is presented for dystocia begins with a complete history and physical exam, including digital vaginal exam. If a fetus is lodged within the birth canal, digital manipulation should be attempted. The fetus may be grasped around the head and neck, around the pelvis, or around the proximal portions of the hind limbs, depending on fetal presentation. Excessive traction should never be applied to a single extremity because of the ease with which these may be avulsed. With the dam restrained in a standing position, traction is applied in a posterior-ventral direction. The fetus may be gently rocked back and forth, and twisted diagonally to free shoulders and hips “locked” in the pelvic canal. If flexion of head or extremities is preventing delivery, a finger may be used to extend them. One cannot overemphasize the importance of using copious amounts of sterile lubricant during obstetrical maneuvers, applied digitally or infused around the fetus using a red rubber catheter.

Radiographs should be obtained in any animal experiencing dystocia. Radiographs are accurate for assessing the number, size, location, and position of fetuses, as well as maternal pelvic morphology and general status of the abdomen. Fetal viability is more difficult to assess from radiographs, unless evidence of fetal decomposition is present. Signs of decomposition include intrafetal or intraterine gas patterns, awkward fetal postures, collapse of the spinal column due to loss of muscular support, and overlapping of the bones of the skull. Ultrasound may be a more useful tool for assessment of fetal viability, fetal malformations, and fetal distress. Normal fetal heart rates have been reported at 180-245 beats per minute in dogs and up to approximately 265 bpm in cats. Deceleration of fetal heart rates to less than 180 beats per minute and the presence of fetal bowel movements on ultrasound have been shown to correlate with severe fetal distress, and may indicate a need for rapid intervention.

Medical management should be considered if there is no evidence of obstruction, and fetal and pelvic size appear normal. Oxytocin is a peptide hormone that increases the frequency and strength of uterine contractions by promoting influx of calcium into myometrial cells. Oxytocin also promotes post partum uterine involution, aids in control of uterine hemorrhage, and assists in expulsion of retained placentas. The dose for oxytocin has traditionally been reported at 5-20 units IM in the dog and 2-4 units IM in the cat. However, with an increase in the use of uterine contraction monitoring (Whelpwise, Veterinary Perinatal Specialties Inc, Wheat Ridge, CO) in veterinary patients, there is a growing body of evidence to suggest that traditional doses may be too high, potentially causing uterine tetany, ineffective contractions, and decreased fetal blood flow. Recent data suggests that doses of 0.5-2 units are effective in increasing the frequency and quality of contraction. The oxytocin dose may be repeated in 30 minutes if expulsion of a fetus has not resulted. If labor proceeds and a fetus is delivered, oxytocin may be repeated every 30 minutes as needed to assist in expulsion of the remaining fetuses.

Calcium gluconate may be considered if weak, infrequent contractions are noted or when labwork reveals hypocalcemia. Retrospective studies have indicated that many patients who fail to respond to oxytocin alone may respond to a combination of calcium and oxytocin. The dose for calcium gluconate (10% solution) as a uterotonic agent is 11 mg/kg diluted in saline and given subcutaneously, or added to IV fluids and given slowly while monitoring an ECG for arrhythmias. If hypocalcemia is documented, a dose of 50-150 mg/kg intravenously should be used. Subcutaneous administration has been reported to result in irritation and potential granuloma formation, though this is an infrequent complication. Dextrose infusion should also be initiated if hypoglycemia is evident on labwork.

Surgical management should be considered for the following conditions:

- Complete primary uterine inertia
- Partial primary uterine inertia or secondary uterine inertia where large numbers of fetuses remain and response to drugs is unsatisfactory,
- Fetal oversize
- Gross abnormalities of maternal pelvis (fractures, masses)
- Fetal malformations
- Malpresentation that is not amenable to manipulation
- Past history of dystocia or c-section
- Fetal putrefaction
- Maternal evidence of systemic illness
- Suspicion of uterine torsion, rupture, prolapse, or herniation
- Evidence of fetal distress with poor response to medical intervention
An anesthetic protocol for caesarian section should be selected with the goal of maximizing survival of neonates and dam. Attempts should be made to minimize exposure of the fetus to anesthetics by keeping the time from induction to delivery as short as possible. Ideally, the dam should be clipped and prepped prior to induction, equipment should be out, and the surgeon should be scrubbed and ready. Induction agents should be given to effect. Regional techniques such as line blocks and epidurals may help to minimize the need for other drugs. A line block can be performed using 2 mg/kg lidocaine infused along the ventral midline. Alternately, epidural lidocaine may be administered in dogs at a dose of 2-3 mg/kg, not to exceed a total volume of 6 ml. Propofol (4-6 mg/kg IV) or mask inductions are most commonly used for caesarian section at this time, and have been associated with reduced neonatal mortality in dogs. Anesthetic agents that have been associated with increased neonatal mortality include thiopental, ketamine, xylazine, medetomidine, and methoxyflurane.\textsuperscript{13-15}

**Neonatal resuscitation**

A warm (90°F) incubator, hemostats, suture material, suction bulb syringes, emergency drugs, and an adequate supply of soft dry towels should be prepared beforehand. As each neonate is handed off, the umbilical cord should be clamped and ligated 1-2 cm from the umbilicus. Fetal fluids and amnion should be removed by rubbing briskly with a soft, clean towel. The oral cavity and nares may be suctioned with a bulb syringe. The old practice of “swinging” puppies to clear their airways is best avoided because of the potential for cerebral hemorrhage due to concussive injury. If vigorous rubbing is not successful at stimulating respiration, positive pressure ventilation may be initiated with a snug fitting mask, keeping the neonates head and neck extended to ensure adequate inflation of the lungs. Alternately, intubation may be accomplished using a catheter or small, uncuffed endotracheal tube. Because isoflurane is minimally metabolized, ventilation is the primary route of elimination. Thus, its depressant effects can not be reversed until the neonate breathes. Cardiac massage may be instituted if a heart beat is not detected once warming and ventilation measures have been instituted. Epinephrine (0.1 mg/kg) may be given intratracheally, intrasosseously, or intravenously if cardiac massage is unsuccessful. Naloxone (0.1 mg/kg) should be considered if the dam received opioid analgesics as part of the anesthetic regimen. Although doxapram (dopram) is routinely administered in many practices as a respiratory stimulant, it is not used for this purpose in the resuscitation of human neonates and there is no evidence to support its use in veterinary patients.

The prognosis for medical management of dystocia is guarded, with success rates of 20-40% in the veterinary literature.\textsuperscript{3,7-9} Additionally, stillbirth rates have been shown to rise when dystocia is allowed to continue for greater than 4.5-6 hours from the time of onset of second stage labor in the dog.\textsuperscript{3,7} For these reasons, the decision to proceed to caesarian section should not be delayed if response to medical management is poor or unlikely to result in successful delivery. In recent studies, neonatal survival rates following surgical treatment of dystocia have been reported at 92% at birth, with 80% still alive at 7 days post c-section.\textsuperscript{13,14}

**Periparturient emergencies**

**Mastitis**

Mastitis is a postpartum complication seen in both dogs and cats that results from bacterial infection of the mammary glands. Bacteria most commonly enter through the nipple as a result of nursing, trauma, or poor hygiene, but may also be spread hematogenously. In mild cases, discomfort, swelling, and inflammation may be seen, while in severe cases, signs of systemic illness such as fever, anorexia, and lethargy frequently develop. Dogs often refuse to allow their young to nurse and may be reluctant to lie down. Severe mastitis often progresses to abscession and necrosis.

Diagnosis of mastitis is generally based on history and clinical signs (fever and swollen, painful glands in the postpartum animal), but baseline CBC and chemistry as well as milk cytology and culture are useful for assessing severity of illness and appropriateness of antibiotic selection. Milk expressed from the gland may be purulent and cytology typically shows large numbers of white blood cells and intracellular bacteria. The most common bacteria isolated on culture include \textit{E. coli}, \textit{Staphylococci}, and \textit{Streptococci}.

Treatment is initiated immediately with broad spectrum antibiotics. Amoxicillin-clavulanic acid or cephalaxin are good first choices and are safe for nursing neonates. Other measures that may be useful in the management of mastitis include warm compresses, hydrotherapy, and frequent milk stripping. If a fluctuant abscess pocket is identified on palpation, early lancing and flushing may limit the degree of skin necrosis that follows. Large, ruptured mammary abscesses may be successfully managed as open wounds with warm compresses, hydrotherapy, and systemic antibiotics, but in these cases mastectomy may provide a more rapid and cosmetic resolution of the problem.

**Endometritis**

Endometritis is a bacterial infection of the uterus that is generally seen within the first three days (up to one week) after whelping, though it may develop during pregnancy as well. Potential causes include retained fetuses or placentas, abortions, uterine trauma secondary to dystocia or obstetrical manipulation, and ascending infection from the vaginal canal. Typical signs include fever, lethargy, anorexia, vomiting, diarrhea, poor lactation, neglect of offspring, and foul-smelling vaginal discharge. Just as in the non-pregnant dog, any purulent vaginal discharge noted during or after pregnancy is abnormal and should prompt investigation.

Labwork abnormalities consistent with sepsis may be seen, including leukocytosis with a left shift or leukopenia, thrombocytopenia, elevated liver values, and hypoalbuminemia. Coagulation testing should be performed to rule out disseminated
intravascular coagulation. Radiographs or ultrasound are indicated to evaluate for fetal death, retained placentas, or evidence of uterine enlargement. Cytology of vaginal discharge typically shows degenerate neutrophils and macrophages with intracellular bacteria. The most common organisms associated with uterine infections include Staphylococci, Streptococci, E. coli, Salmonella, Campylobacter, and Chlamydia.

An animal suspected of having septic metritis should be treated aggressively with IV fluids. Broad spectrum antibiotic combinations such as ampicillin-enrofloxacin, ampicillin-aminoglycoside, or cefazolin-aminoglycoside-metronidazole, should be administered. Following stabilization, ovariohysterectomy is the treatment of choice for metritis. If the animal is not showing signs of sepsis and the owner wishes to use her for breeding purposes in the future, evacuation of the uterine contents using PGF₂α (Lutalyse) may also be attempted in conjunction with broad spectrum antibiotics. PGF₂α is typically administered at doses of 0.1-0.25 mg/kg SQ once daily for 5 days. If initial dosing does not result in adequate expulsion of uterine contents, the author generally increases treatment frequency to twice daily. Potential complications of PGF₂α include vomiting, abdominal discomfort, uterine rupture, and septic peritonitis. Because PGF₂α treatment may require several days to achieve a good effect, animals that are severely ill should always be treated with ovariohysterectomy. Ovariohysterectomy is also the best choice when the animal is not intended for future breeding or if the health of the dam is a higher priority than possible future breedings.

**Eclampsia**

Eclampsia or puerperal tetany is a life threatening condition that results from the development of hypocalcemia in the periparturient period. It is one of the more common complaints noted following parturition, accounting for roughly 1/4 of periparturient emergencies. Eclampsia is results from the loss of calcium through lactation and fetal skeletal mineralization, in excess of that entering the extracellular fluid through gastrointestinal absorption and bone resorption. Other factors such as inadequate diet or parathyroid atrophy resulting from oversupplementation of calcium may also contribute, though diet in affected animals has not been reported to be significantly different from non-affected animals. Increasing litter size to maternal body weight ratio has also been identified as a significant factor in the development of periparturient hypocalcemia.

Eclampsia is most commonly seen in small dogs, first-time whelpings, and dogs with large litter sizes. It typically develops 2-4 weeks after parturition but is occasionally seen in late gestation. Clinical signs in dogs most commonly include stiff gait, trembling, twitching, seizures, tachycardia, panting, and hyperthermia, but some dogs may present with atypical signs such as whining, vomiting, diarrhea, and behavior changes. If untreated, death may result from respiratory impairment, or from hyperthermia and cerebral edema. Cats may present with clinical signs similar to dogs, but unlike dogs, are more prone to hypothermia, and may present with hyperexcitability, hypersensitivity, or flaccid paralysis in place of clonic-tonic muscle spasms.

Diagnosis of eclampsia is made on the basis of history and physical exam findings in conjunction with low total or ionized calcium levels. Ionized calcium represents the physiologically active portion of calcium within the body, and is involved in muscular contraction, as well as neurologic and cardiovascular function. Ionized calcium levels are therefore believed to be a more sensitive indicator of extracellular calcium levels than total calcium, and typically fall below 0.8 mmol/L in dogs with eclampsia (reference range: 1.2-1.4 mmol/L). However, total calcium levels have been found to be decreased in all dogs with eclampsia, suggesting that total calcium levels may provide sufficient information in this disease if ionized calcium measurement is not available.

Animals presenting with eclampsia should have an IV catheter placed and intravenous fluids administered to address fever, dehydration, and tachycardia. Calcium gluconate (10%) should immediately be administered intravenously slowly to effect. Most animals will have tremors controlled at doses ranging from 0.5 to 1.5 ml/kg. An ECG should be monitored during calcium administration and the infusion stopped if bradycardia or arrhythmias develop. Ionized calcium levels should be rechecked post administration to make sure that ionized calcium levels remain within the normal range. Temperature should be carefully monitored in animals presenting with tremors, and active cooling measures (cool fluids, alcohol applied to footpads) should be instituted for patients with severe hyperthermia. Body temperature generally falls quickly once tremors are controlled, so active cooling measures should be discontinued once the temperatures falls below 103°F. Oral calcium carbonate (Tums) supplementation should be continued at a dose of 100 mg/kg/day throughout lactation. Up to 20% of dogs may have recurrence of eclampsia despite supplementation if puppies are allowed to nurse, so bottle feeding and early weaning of the puppies is recommended.

Supplementation of calcium prior to whelping is not recommended, as this may downregulate parathyroid hormone secretion, decreasing intestinal calcium absorption and increasing the risk of eclampsia during lactation. Instead, calcium administration (100 mg/kg/day divided) should be instituted following whelping in dogs at risk and dogs with a previous history of eclampsia.

**References**


Managing Difficult Urethral Obstructions
Ari Jutkowitz, VMD, DACVECC
Michigan State University
East Lansing, MI

Overview and pathophysiology
Feline urethral obstruction is one of the most common emergency presentations in the cat, accounting for approximately 9% of feline emergency admissions. While there are many factors that may play into the development of lower urinary tract diseases in the cat, matrix-crystalline plugs and urolithiasis are the most common causes of obstruction. Cats with urethral obstruction may have signs localized to the lower urinary tract including dysuria, stranguria, pollakiuria, hematuria, vocalizing, and pain, or they may show signs of systemic illness such as vomiting, lethargy, or collapse. Cats with obstructive urinary tract diseases may or may not have demonstrated preceding signs of lower urinary tract disease.

Following the development of urethral obstruction, clinical signs of uremia typically develop within 24 hours. Dehydration occurs due to decreased water intake and ongoing fluid losses secondary to vomiting. Acid-base (metabolic acidosis) and electrolyte disturbances (hyperkalemia and hyperphosphatemia) develop due to impaired excretion. Accumulation of metabolic wastes leads to post renal azotemia. Bladder capacity is reached, leading to rising intravesicular pressure and subsequently falling glomerular filtration rate (GFR). Prolonged obstruction may result in intrinsic renal failure. Damage to the urothelium and detrusor muscle may also develop during this time. If left untreated, death secondary to cardiopulmonary failure or hyperkalemia may occur within 3-6 days. Damage to bladder mucosa or urethra may shorten survival times.

Diagnosis of urethral obstruction
Diagnosis of urethral obstruction is generally made on the basis of history and physical exam findings. Abdominal palpation typically reveals a turgid, painful bladder, though in rare cases, the bladder may be moderate in size if the cat is presented to the veterinarian shortly after clinical signs develop. Blood and/or crystalline debris may be visualized at the urethral orifice. The presence of bradycardia frequently indicates hyperkalemia, and severe systemic signs in conjunction with free abdominal fluid should prompt consideration of bladder leakage or rupture. In contrast, cats that present with stranguria but appear systemically healthy and have palpably small bladders typically have non-obstructive lower urinary tract disease.

At the time of presentation, a peripheral IV catheter is placed and blood is collected for complete blood count, serum biochemistry panel, and venous blood gas/electrolyte panel. The blood gas/electrolyte panel is particularly helpful as it provides rapid information on parameters such as potassium concentration (as well as acid-base status and renal values) that may affect initial interventions. Electrocardiography can also be helpful in the initial evaluation of the patient with urethral obstruction. Early ECG changes suggestive of hyperkalemia include bradycardia, dampened P-waves, tented T-waves, and prolongation of the P-R interval. As hyperkalemia worsens, loss of P-waves (atrial standstill) and widening of the QRS complex may develop. Electrocardiographic changes typically do not develop until potassium levels are greater than 7 mEq/L, but there is a great deal of individual variation in terms of patient response to hyperkalemia. Metabolic acidosis, hyponatremia, and hypocalcemia may contribute to the likelihood of hyperkalemic cardiotoxicity.

Once the animal has been medically stabilized and deobstructed, urine is submitted for urinalysis and culture. Because crystalline and cellular composition of the urine may change over time, evaluation of a fresh, undiluted sample is preferred. Diagnostic imaging should be performed to rule out cystic or urethral calculi. If a urolith or crystalline-matrix plug is retrieved at the time of deobstruction, composition should be determined as this may impact future therapies.

If free abdominal fluid is identified, fluid chemistry may be helpful in determining whether urinary tract rupture has occurred. An abdominal fluid:serum creatinine ratio of 2:1, or abdominal fluid:serum potassium ratio of 1.9:1 (cat) or 1.4:1 (dog) is predictive of uroperitoneum. Cytology of the fluid sample should also be performed to rule out urosepsis. Contrast cystourethrography is used to determine location and severity of the rupture.

Treatment of urethral obstruction
Fluid therapy
Initial management of urethral obstruction in the cat should focus on correction of hypovolemia, hyperkalemia, and other acid-base and electrolyte disturbances. In most cases, appropriate fluid therapy followed by restoration of urine flow will effectively correct these abnormalities. A peripheral IV catheter should be placed and fluid therapy instituted immediately using 0.9% sodium chloride or balanced electrolyte solution such as lactated Ringer’s solution (LRS). A shock rate of fluids (66 ml/kg/hour in the cat) is calculated and then administered to effect in increments of approximately ¼ of the calculated dose, reassessing major body systems after each bolus. For example, the calculated shock rate in a 5 kg cat is approximately 330 ml, and should be administered in individual boluses of 50-100 ml every 10-15 minutes until cardiovascular status is restored. The goal of fluid therapy should be normalization of vital signs such as heart rate, level of consciousness, pulse quality, blood pressure, and capillary refill time. The specific type of intravenous
Techniques for urethral deobstruction

During the initial exam, the urethra may be gently massaged, followed by careful palpation of the bladder to potentially dislodge superficial plugs. Extreme care should be taken to avoid accidental bladder rupture. While this technique is rarely effective, it is a simple extension of the initial physical exam and therefore may be worth trying in less severely affected cats prior to catheter deobstruction.

Although severely depressed patients may be deobstructed without the need for chemical restraint, sedation/analgesia is employed in the majority of “blocked” cats to improve patient comfort, facilitate deobstruction, and avoid urethral or bladder trauma secondary to patient struggling. Ketamine (100 mg/ml) may be combined with diazepam (5 mg/ml) in equal parts by volume and given at a dose of 1 ml/10 kg of the 50:50 mix. However, this combination should be avoided in cats with known or suspected hypertrophic cardiomyopathy, or when an undiagnosed murmur or gallop rhythm is present. In these cases, hydromorphone (0.05 mg/kg) in combination with diazepam (0.2 mg/kg) may provide a safer option.

Following sedation, the cat is positioned in dorsal recumbency with the legs pulled forward over the head. In this position, the prepuce may be retracted and the penis extruded by simply pushing the prepuce downward towards the anus. A further advantage to this technique is that it allows the urethra to be maximally straightened to facilitate deobstruction. The author’s preferred technique for deobstruction uses an olive tip catheter (FUS needle 21 g x 1”, Jorgensen Laboratories, Loveland, CO). This is a metal, bulb-tipped catheter that can be used to flush the urethra and either break down matrix-crystalline plugs or hydropulse them atraumatically into the bladder. Initially, the olive tip catheter is lubricated and inserted gently into the urethra to the site of the obstruction, approximately 1-2 cm. A 3 cc syringe is then used to lavage and break down the plug. Bits of the plug will often be seen emerging from the urethral orifice during the lavage. When the catheter is withdrawn, a strong stream of urine will frequently force the remainder of the plug from the urethra. Gentle bladder palpation may be used at this point to assist in the expulsion of the plug. To avoid urethral trauma, the catheter should not be forced past the obstruction. Instead, the lavage solution should be allowed to do the work. Additionally, acidic solutions should not be used for lavage as these have not been shown to be effective at plug dissolution and may further traumatize the urethral mucosa. If lavage alone is not successful at dislodging the urethral plug, the tip of the urethra can be pinched around the bulb tip of the catheter and hydropulsion used to push the plug back into the bladder.

Many clinicians use polypropylene “tomcat” catheters for the purposes of unblocking cats. These have the potential to cause additional trauma to the urethra when the rigid catheter is forced past the site of obstruction. If used, a number of steps may help to minimize iatrogenic urethral damage and maximize chances of success. (1) Completely straighten the urethra by pushing the prepuce dorsally towards to anus until the penis is parallel to the spine. (2) Use copious amounts of lubrication. (3) Hydropulse with sterile saline prior to advancing the catheter to assist in dislodging the plug. (4) Use a very light touch when advancing the catheter. Hold the catheter between index finger and thumb and twirl gently while advancing. Think about “picking a lock” when attempting to advance the catheter. Use finesse instead of force. (5) Once the catheter is well seated in the urethra, the penis may be allowed to retract into the prepuce. The prepuce may then be pulled caudally (toward tail tip) to further straighten the urethra while the catheter is advanced.

Some experienced clinicians advocate the use of cystocentesis prior to deobstruction to decompress the bladder and to potentially facilitate hydropulsion of urethral plugs. The author prefers to reserve this technique for use only as a last resort due to the number of
cats presenting to the emergency service with uroperitoneum and apical bladder tears following cystocentesis of overdistended bladders. However, it should be noted that our institution may see a biased population of more severely affected animals.

Cats that are critically ill, and those demonstrating large amounts of “sandy” crystalline debris in the urine, blood clots, uroliths, plugs hydropulsed into the bladder, bladder atony, or urethral narrowing are particularly at risk for reobstruction post-unblocking. For this reason, a soft, indwelling, 3.5-5 French red rubber catheter is placed following deobstruction to facilitate urine drainage overnight and to assist in quantitation of urine output. Indwelling catheters should be placed using liberal clipping and scrubbing of the perineum and aseptic technique to minimize risk of catheter-induced urinary tract infection. The tip of the catheter should sit just past the bladder neck to reduce risk of kinking or knotting. The catheter should then be connected to a sterile, closed collection system. To decrease the likelihood of premature catheter removal, careful attention should be given to suture placement. A piece of butterfly tape is placed around the catheter and appositional sutures are placed at the margin of the butterfly tape to prevent kinking of the catheter. The catheter body is then taped to the tail. An Elizabethan collar should be placed prior to anesthetic recovery.

**Hospital management**

**Fluid therapy**

Following initial stabilization and correction of hypovolemia, fluid rates should be adjusted to account for remaining fluid deficits, daily maintenance requirements, and ongoing losses. Deficits can be estimated as follows based upon clinical signs of dehydration: mild (5-6%), moderate (7-8%), and severe (8-10%). Multiplying the estimated percent dehydration by body weight gives the fluid deficit, which may then be replaced over the next 24 hours. For example, a 5 kg cat estimated to be 8% dehydrated would have an estimated deficit of 400 ml. To this value must be added maintenance needs (approximately 60 ml/kg/day) and ongoing losses. Ongoing losses following “unblocking” result from post-obstructive diuresis and can be estimated most easily by quantitating urine output. Normal urine output is approximately 1-2 ml/kg/hour (5-10 ml/hour in the average 5 kg cat). Urine output in excess of this amount typically results from post-obstructive diuresis. During the first 24 hours of therapy, a fluid rate should be selected that accounts for these ongoing losses. In other words, the intravenous fluids administered should slightly exceed measured urinary losses.

Urine output is quantified every four hours. Inadequate urine production (<1 ml/kg/hr) indicates inadequate fluid administration or urinary catheter occlusion with debris. After troubleshooting the catheter, a fluid bolus followed by an increase in fluid rate is indicated if urine output remains low.

Fluid therapy is typically tapered over the next 24-36 hours. Daily monitoring of electrolytes and renal values should be performed to ensure that azotemia resolves and electrolytes normalize. Potassium supplementation may be required during post-obstructive diuresis should hypokalemia develop.

**Urinary catheter care**

Indwelling urinary catheters and tubing should be cleaned externally once daily with a dilute chlorhexidine solution. Gloves should be worn and aseptic technique used when handling the catheters to avoid nosocomial infection. Bladder palpation should be performed every 4-6 hours to ensure that the bladder remains decompressed. When moving the patient, the urine collection system tubing should be clamped and the bag held below the level of the patient to prevent retrograde flow of urine into the bladder.

To minimize likelihood of catheter-induced urethral irritation or urinary tract infection, catheters should be removed as soon as possible. For most cats, the catheter is removed within 48 hours, but the presence of excessive crystalline debris or blood clots in the urine may necessitate longer indwelling catheter duration to avoid reobstruction. Use of antibiotics during hospitalization is not recommended as this is unlikely to prevent catheter-related infection, but may contribute to antibiotic resistance of organisms protected by the catheter biofilm. Culture should be performed prior to catheter removal, with antibiotic therapy initiated as indicated based upon results of culture and sensitivity.

Following catheter removal, patients should be monitored for an additional 12-24 hours to ensure that the urethra remains patent. Cats will typically urinate small volumes frequently following catheter removal due to irritation resulting from obstruction and catheterization. Although they may appear to strain in the litterbox, the bladder should remain small on palpation. A progressively distending bladder post-catheter removal typically indicates reobstruction (firm bladder, difficult to express) or bladder atony (large, flaccid, expressible). Cats with suspected urethral spasm post catheter removal may benefit from a smooth muscle relaxant following catheter removal (prazocin 0.5 mg/cat q24h).

**Pain management**

Urinary obstruction and initial management are frequently associated with significant discomfort. In our practice, butorphanol (0.01 mg/kg IV q6h) is commonly used to provide analgesia for the first 24-48 hours.

**Long term management**

Strategies for long-term prevention of recurrence focus primarily on environmental modification and dietary changes. Occasionally, pharmacologic intervention may be warranted. An ample number of litterboxes should be provided, particularly in multi-cat households, and litterboxes should be cleaned regularly to encourage more frequent use. Canned or moistened food may decrease frequency of lower urinary tract episodes by promoting a more dilute urine and increasing frequency of urination. Fresh water should
be available at all times. In cases where obstruction was caused by struvite-matrix plugs, an acidifying diet may be of benefit. Antibiotics, anti-inflammatories, and antispasmodics have not been associated with reduction in frequency of episodes and their routine use is not recommended.

**Perineal urethrostomy**

Perineal urethrostomy may be considered in cases where frequency of urethral obstruction is unacceptable despite appropriate medical management or when irreversible changes in the urethra (striction, scarring, urolithiasis) cause recurrent or persistent obstruction. Perineal urethrostomy has been associated with significant short and long term complications including recurrent urinary tract infection and stricture, and as such should not be considered a first line recommendation for cats with urethral obstruction.

**References**


Pericardial effusion is defined as the accumulation of fluid within the pericardial space. As the pressure within the pericardial space increases, right sided cardiac filling is impaired, resulting in decreased stroke volume with subsequent decreases in cardiac output and ultimately decreased oxygen delivery to the tissues (shock). These manifestations of pericardial effusion are referred to as cardiac tamponade. Successful emergency management of dogs with life threatening pericardial effusion depends on early triage, a thorough physical examination, point of care diagnostic imaging techniques, and subsequent pericardiocentesis or placement of an indwelling pericardial drain.

Key etiologic and pathophysiologic points

Pericardial fluid accumulation and cardiac tamponade in the dog most often occurs secondary to a neoplastic process. Hemangiosarcoma (HSA) is most commonly identified in the region of the right atrium or right atrial appendage while chemodectoma (common in brachycephalic breeds) is most often identified at the heart base. Mesothelioma and any metastatic tumor are additional neoplastic causes. Although location and breed are frequently suggestive of tumor type, definitive diagnosis is dependent on a biopsy specimen.

Idiopathic pericardial effusion tends to be an inflammatory process and is frequently recognized in similar breeds to those that frequently develop HSA. Significant efforts in recent years have been directed towards developing diagnostic tests to help differentiate malignant from benign pericardial effusion (idiopathic). Pericardial fluid pH was initially thought to aid in making this differentiation, however, pericardial fluid pH has now been clearly shown to be of little diagnostic value. Recent evidence suggests that blood concentrations of cardiac troponin I (cTnI) are significantly higher in dogs with masses consistent with HSA than in dogs without evidence of an underlying cause (idiopathic). Vitamin K1 antagonists (anticoagulant rodenticides and coumadin) can also result in pericardial effusion. Therefore; it is the authors’ practice to always perform an ACT or other point-of-care coagulation assessment at the cage side prior to pericardiocentesis. If significant coagulopathy is present and patient condition permits, correction of coagulopathy with blood products (fresh frozen plasma or fresh whole blood) is indicated prior to pericardiocentesis. Subsequent institution of Vitamin K1 therapy for 4 weeks is indicated.

Left atrial tear is an uncommon consequence of chronic mitral regurgitation and left atrial dilatation, however, it has been recognized as a cause of acute pericardial effusion in the dog. An infectious cause of pericardial effusion is fungal disease (coccidiomycosis). Bacterial pericarditis and pericardial effusion secondary to trauma also occur, but are uncommon.

Numerous additional conditions such as congestive heart failure, uremia, decreased oncotic pressure, and a host of systemic inflammatory processes frequently result in small volume pericardial effusion accumulations without evidence of cardiac tamponade.

Key clinical diagnostic points

Triage and physical examination in pericardial effusion

The most common presenting complaints from the owners of dogs with pericardial effusion and cardiac tamponade are lethargy, anorexia, collapse or syncope, abdominal distention, and dyspnea. Major body systems assessment of the dog with pericardial effusion will likely reveal compromise to one or all of the major body systems. Assessment of the cardiovascular system may frequently reveal the following:

- Pale mucous membranes: due to vasoconstriction and poor peripheral perfusion
- Slow CRT: due to decreases in cardiac output
- Increased heart rate: due to compensatory activation of the sympathetic nervous system
- Poor pulse quality: due to decreased stroke volumes and low blood pressure

Assessment of the respiratory system will frequently reveal increased respiratory rate and effort.

Assessment of the central nervous system will frequently reveal a decreased level of consciousness secondary to decreased oxygen delivery to the brain. Any one or combination of these findings should necessitate movement to the treatment area for further assessment including full physical examination, measurement of blood pressure, oxygen saturation, cardiac rhythm (ECG), and placement of an intravenous catheter from which a small blood sample for PCV / TS / Blood Glucose +/- Venous Blood Gas and Electrolytes can be rapidly acquired. If possible, blood for CBC, serum biochemical profile, and coagulation profile or ACT should also be collected. Concurrently, a second team member will be able to collect a full medical history.

Physical examination should still be centered on the major body systems, but subtle findings supportive of pericardial effusion may be noted including:

- Jugular venous distention: due to right sided congestive heart failure.
• Muffled heart sounds normal lung sounds: unlike pleural effusion which will frequently cause decreased heart and lung sounds, pericardial effusion will frequently only cause decreased heart sounds.

• Abdominal distention: ascites and hepatic engorgement may result from longstanding (days) pericardial effusion due to right sided congestive heart failure. Abdominocentesis will frequently reveal a relatively clear fluid will low cellularity and a protein concentration greater than 2.5g/dL but less than 3.5g/dL most consistent with a modified transudate.

• Pulsus paradoxus: An inspiratory fall of arterial systolic blood pressure of more than 10mmHg resulting in variation in pulse intensity with respiratory cycle due to increased venous return during inspiration, increased right sided filling, shifting of the interventricular septum to the left with decreased left sided diastolic filling and subsequent decreased left sided stroke volume.²

• Other physical examination findings specific to the underlying cause of the effusion such as fever in septic or fungal pericarditis.

Pericardial effusion causing cardiac tamponade should be HIGHLY suspected based on signalment, history, and physical examination findings, supported by diagnostic testing such as abdominocentesis and electrocardiography (+/- radiography) and confirmed through point of care diagnostic imaging techniques.

Diagnosis techniques

Abdominocentesis
See above.

Electrocardiography
Assessment of ECG in patients with pericardial effusion may reveal sinus tachycardia +/- ventricular arrhythmias. Ventricular arrhythmias may result from decreased myocardial oxygen delivery or aberrant conduction associated with the underlying cause of the effusion. QRS complexes <1mV in amplitude and the presence of electrical alternans (regular or irregular variation in QRS complex amplitude associated with the heart moving within the pericardium to and from the positive pole of lead II) are supportive of pericardial effusion.⁴

Echocardiogram
Echocardiogram is the diagnostic test of choice for confirmation of the presence of pericardial effusion in the dog. Many dogs with pericardial effusion have SEVERE cardiovascular compromise and can be on the verge of death. The stresses associated with radiographic imaging may put cause these patients to decompensate. Consequently, in the ideal world, radiographic imaging should be avoided initially. The authors have found that the presence of a small, portable ultrasound machine with a mid-range frequency transducer placed at the primary treatment station in the emergency room / treatment area to be of great utility for identifying conditions like pericardial effusion, pleural effusion, and to assess patients with acute abdomen for the presence of abdominal fluid. Echocardiographically, pericardial effusion appears as a hypoechoic space located between the hyperechoic pericardium and the right ventricular wall when viewed through the right cardiac notch. The presence of pericardial effusion provides excellent contrast to aid in the diagnosis of cardiac masses, however, pericardiocentesis should NOT be delayed in a patient with signs of shock simply to aid the diagnosis.

Thoracic radiography
As previously mentioned, thoracic radiography can be an extremely stressful procedure for dogs with cardiac tamponade. However, not all practices are equipped with ultrasound capabilities. If thoracic radiography is performed in dogs with suspected pericardial effusion, ventrodorsal positioning should be avoided. A dorsoventral projection can be acquired with minimal stress. Lateral thoracic radiographs may also be performed. Supportive radiographic findings include an enlarged, globoid cardiac silhouette. Acute effusions may not cause severe enlargement of the cardiac silhouette because the pericardium has not had time to stretch. Concurrent pleural effusion may be present. The other primary differential for a globoid heart is dilated cardiomyopathy (DCM) or other underlying cardiac disease. Key findings to try to differentiate DCM from pericardial effusion include:

• Heart sounds: Heart sounds in dogs with DCM are frequently normal in contrast to the decreased heart sounds seen in pericardial effusion. A systolic murmur may be noted in dogs with DCM and is uncommon in dogs with pericardial effusion.

• ECG: Atrial fibrillation is common in dogs with DCM. Atrial fibrillation is uncommon in dogs with pericardial effusion. Electrical alternans may be seen in dogs with pericardial effusion.⁴

• Cardiac Silhouette: The silhouette of the heart on thoracic radiographs of dogs with pericardial effusion tends to be extremely round with sharp borders. The silhouette of the heart in dogs with cardiomyopathy can be round, but often, there are still some dimples or “waist” associated with the divisions between the chambers and the borders of the cardiac silhouette tend not to be as sharp because of motion artifact.

• Pulmonary infiltrate: Pulmonary edema is common in DCM and uncommon in pericardial effusion.

• Pulsus paradoxus: Pulmonary edema is common in DCM and uncommon in pericardial effusion.

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Key therapeutic points
Pericardiocentesis
Pericardiocentesis can be a stressful procedure. Use of cardiovascularly sparing sedatives (narcotics and benzodiazepines) may alleviate patient stress and facilitate safe pericardiocentesis. Numerous techniques have been described for pericardiocentesis in the dog including, but not limited to the use of a large-gauge over-the-needle catheter, through the needle catheter, and catheters placed using the Seldinger technique. Numerous commercial pericardiocentesis trays / kits are also available. The authors prefer to use a 14-16g, 5.5” over-the-needle catheter (Abbocath T, Hospira Inc. Lake Forest, IL) with two additional small side-holes or a commercial multi-lumen intravenous catheter placed using the Seldinger technique (Arrow Triple Lumen Central Venous Catheter, Arrow International, Reading, PA). The former is much less expensive while the latter may be left in place for ongoing drainage.

ECG should be monitored during and after pericardiocentesis for the presence of arrhythmias induced by catheter-associated irritation of the epicardium and decreased myocardial oxygen delivery experienced during cardiac tamponade. Lidocaine should be readily available, as should a defibrillator.

Pericardiocentesis is most often performed from the right hemithorax because injury to the left coronary artery is unlikely, and the cardiac notch is slightly larger. The patient can be positioned in sternal recumbency (preferred by most) or laterally. Full surgical preparation should be performed between the 2nd to the 8th ribs and from the mid-thorax to the level of the sternum. A fenestrated drape should be placed. Aseptic technique should be practiced at all times. The apex beat of the heart should be palpated (most often between the 4th and 5th ribs just above the costochondral junction) and lidocaine should be infiltrated locally off of the cranial edge of the rib (to avoid the intercostal neurovascular bundle). Ultrasound guidance can also be used to identify the optimal location for pericardiocentesis. A small skin incision (<5mm) should be made in the proposed insertion site and the catheter advanced through this incision (off the cranial edge of the rib). Upon the appearance of fluid in the flash chamber, the catheter and stylet should be advanced together for 2-3mm and the catheter fed over the stylet into the pericardium. Initially, a small fluid sample should be placed in an ACT or clot tube. A sample retrieved from the ventricle should clot (unless the underlying condition is anticoagulant rodenticide intoxication) while one that has been in the pericardial space for any appreciable period of time should not. A fluid sample should be saved for cytologic analysis and culture and the pericardium should be evacuated.

Monitoring
Patient response to decompression of significant pericardial effusion is often very rapid and very gratifying as vital signs and physical examination findings improve dramatically. Monitoring for recurrence of fluid accumulation by frequent reassessment of major body systems, physical examination and echocardiography is useful. Placement of a central venous catheter and monitoring of central venous pressure can also be a useful technique in that re-accumulation of pericardial fluid will result in a rise in central venous pressure.

Key prognostic points
Prognosis for dogs with pericardial effusion will depend on the underlying cause of the disease. Surgical removal of a mass on the right atrial appendage will at least temporarily alleviate signs of recurrent pericardial effusion. Surgical removal of right atrial / appendage HSA followed by chemotherapy will prolong life in dogs with pericardial effusion.9 Pericardecotomy will temporarily palliate clinical signs of pericardial effusion for most neoplastic processes, and will most often be curative for idiopathic pericardial effusion. Thoracoscopic pericardecotomy or creation of a pericardial window may have similar effects.10-12 Treatment with fresh frozen plasma, vitamin K1, and pericardiocentesis will be curative for dogs with anticoagulant rodenticide intoxication. Culture and sensitivity based antimicrobial therapy +/- surgical debridement is indicated for the management of infectious pericarditis. Dogs with left atrial tear secondary to chronic mitral valve regurgitation and left atrial dilation carry a guarded prognosis. Surgical repair of such a lesion has been described.13

Summary
Triage and careful attention to physical examination findings supported by ancillary diagnostic tests and point-of-care diagnostic imaging are the keys to the rapid identification of pericardial effusion in the dog. Rapid identification of problems and institution of treatment will maximize the likelihood of a positive outcome.

References/suggested reading
Hypoadrenocorticism is an uncommon disease in small animals, with relatively non-specific clinical signs and laboratory changes that may mimic other disease processes. Characteristic alterations in sodium and potassium are often present, but electrolyte concentrations may be normal in dogs with secondary or atypical primary hypoadrenocorticism. Early diagnosis may therefore pose a challenge, particularly when atypical signs are present.

**Etiology and pathophysiology**

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

Adrenocortical insufficiency results from atrophy or destruction of the adrenal cortex and may be classified as either primary or secondary. Primary hypoadrenocorticism results from bilateral destruction of the adrenal cortices. Most cases are presumed to have an immune-mediated basis, though other causes include infections (fungal or mycobacterial), infarctions, neoplasia, surgical trauma, and amyloidosis. Iatrogenic destruction may also result from lysodren, ketoconazole, or megestrol acetate therapy. Secondary hypoadrenocorticism results from lack of adrenal stimulation via CRH or ACTH. Most cases of secondary hypoadrenocorticism are caused by inflammation, tumors, trauma, or congenital abnormalities of the hypothalamus or pituitary gland. Exogenous steroid administration may also suppress ACTH release, resulting in adrenal atrophy. Most dogs and cats have normal ACTH stimulation tests within 2 weeks of steroid withdrawal, but this is dependent on chronicity of treatment.

Most reports of dogs with hypoadrenocorticism have noted an increased prevalence in young to middle aged female dogs. Some of the breeds reported to be at greater risk include Standard Poodles, Leonbergers, Portugese Water Dogs, Labrador Retrievers, Bearded Collies, Old English Sheepdogs, Standard Schnauzers, Soft Coated Wheaten Terriers, Basset Hounds, English Springer Spaniels, German Shorthaired Pointers, Nova Scotia Duck Tolling Retrievers, Great Danes, German Shepherds, West Highland White Terriers, and Rottweilers. Primary hypoadrenocorticism is considered rare in cats.

The pathophysiologic changes seen with hypoadrenocorticism are a direct result of glucocorticoid and mineralocorticoid deficiency. Glucocorticoids have effects on nearly every tissue in the body. Some of these effects include the provision of a sense of well-being, stimulation of appetite, modulation of white blood cell function, and maintenance of blood pressure. Glucocorticoids also maintain fasting blood sugar levels by promoting gluconeogenesis, impairing uptake by peripheral tissues, and augmenting lipolysis. They are involved in maintaining calcium balance by augmenting renal excretion, reducing GI absorption, and decreasing resorption of calcium from bone. Decreased cortisol levels consequently lead to signs of lethargy, inappetance, vomiting, diarrhea, depression, and weight loss. Decreased sensitivity of the vasculature to catecholamines may contribute to hypotension. Hypoglycemia and hypercalcemia may develop. Failure to mount a stress leukogram or the presence of a “reverse stress leukogram” characterized by neutropenia, lymphocytosis, and/or eosinophilia may also result from cortisol deficiency.

Aldosterone is critically involved in the maintenance of sodium and water balance, acting primarily upon the distal nephron to promote reabsorption of sodium and chloride and excretion of potassium and hydrogen ions. Inability to release aldosterone therefore results in a number of adverse effects. Loss of sodium and chloride results in severe water losses, leading to polydipsia, polyuria, isosthenuria, and decreased effective circulating volume. Antidiuretic hormone (ADH) release is initially enhanced in response to hypovolemia, exacerbating hyponatremia by promoting free water retention. Ultimately, further ADH secretion may become impaired as the osmotic stimulus for ADH release is removed by the severity of hyponatremia. As salt and water losses continue, dehydation, hypovolemia, and azotemia become progressively more severe.

Failure to secrete potassium in exchange for sodium results in hyperkalemia. This is worsened by decreased renal perfusion (further impairing potassium excretion) and accompanying metabolic acidosis (promotes extracellular potassium shifts). Hyperkalemia causes signs of muscle weakness, bradycardia, hypotension, and ultimately death. A profound metabolic acidosis is also frequently seen, and results from loss of aldosterone stimulation of H+ secretion by the intercalated cells of the distal nephron, as well as by decreased perfusion & azotemia.

**Diagnosis of hypoadrenocorticism**

A tentative diagnosis of hypoadrenocorticism can frequently be made on the basis of history and physical examination findings. Common presenting complaints may include vomiting, diarrhea, abdominal pain, lethargy, weakness, and weight loss. Polyuria, polydipsia, and shaking or shivering are also frequently reported. Gastrointestinal ulceration is sometimes seen, and is believed to result from insufficient cortisol for normal maintenance of the gastric mucosa, coupled with poor gastrointestinal perfusion secondary...
to hypovolemia. Severe gastrointestinal ulceration may result in signs of hematemesis, melena, and profound anemia. In some animals, symptoms of hypoadrenocorticism may develop acutely without prior signs of illness, or following a period of stress such as boarding at a kennel. In others, symptoms may have been present chronically, waxing and waning in intensity.

Patients presenting in Addisonian crisis typically manifest signs of shock. They frequently appear markedly dehydrated, hypothermic, depressed, and weak. Prolonged capillary refill time and weak pulses may be noted. Rapid respiratory rate may be seen in compensation for severe metabolic acidosis. One of the most striking findings, however, is the presence of a relative bradycardia, rather than tachycardia, in the face of signs of circulatory shock.

Electrocardiography can be helpful in the initial evaluation of the Addisonian patient. Early ECG changes suggestive of hyperkalemia include bradycardia, dampened P-waves, tented T-waves, and prolongation of the P-R interval. As hyperkalemia worsens, loss of P-waves (atrial standstill) and widening of the QRS complex may develop. Electrocardiographic changes typically do not develop until potassium levels are greater than 7 mEq/L, but there is a great deal of individual variation in terms of patient response to hyperkalemia.

Results of complete blood counts, serum biochemical profiles, and urinalysis may further support a diagnosis of hypoadrenocorticism. The typical Addisonian has hyperkalemia and hyponatremia, with a Na:K ratio that is <27:1. It should be remembered that a number of other diseases may result in altered sodium:potassium ratios including oliguric or anuric renal failure, urinary obstruction, uroabdomen, pregnancy, chylothorax, and primary gastrointestinal diseases such as trichuriasis, duodenal ulceration, and salmonellosis.5-12

Other common laboratory abnormalities include azotemia (present in over 80%), hypochloremia, hyperphosphatemia, and metabolic acidosis. Even in the face of significant prerenal azotemia, isosthenuria is a frequent finding and results from osmotic diuresis secondary to sodium losses.1 Hypoglycemia has been reported in 16-33% of dogs with hypoadrenocorticism, and may be the only clinical finding in dogs with atypical Addison’s disease.1-3 Hypercalcemia occurs in approximately 30% of dogs.1 Hypoalbuminemia is also common, though the mechanism by which this occurs is unclear. Gastrointestinal protein losses, anorexia, and loss of glucocorticoid stimulation of hepatic synthesis are speculated to play a role.1-3 Hypercholesterolemia may also develop secondary to decreased fat metabolism as a result of hypocortisolemia. Elevations in ALT, AST, and alkaline phosphatase are reported in 20-30% of cases and may result from hypoperfusion or cholestasis.1-3

Hematologic changes reported in Addisonian dogs include homoconcentration, normocytic-normochromic non-regenerative anemia, failure to mount a stress leukogram, and the presence of a reverse stress leukogram (characterized by an absolute lymphocytosis or eosinophilia). The anemia in animals with hypoadrenocorticism is multifactorial, resulting from a combination of chronic disease and the development of gastrointestinal ulceration. Glucocorticoids are also believed to have a facilitatory role in the responsiveness of the bone marrow to erythropoietin.13 The anemia is frequently more severe than initially suspected based on packed cell volume (PCV) due to concurrent homoconcentration. Reverse stress leukogram has been reported in 10-20% of dogs with hypoadrenocorticism and results from withdrawal of the effects of cortisol on maturation (glucocorticoids are known to stimulate neutrophil progenitors and inhibit eosinophil progenitors) and sequestration of leukocytes.14

Thoracic radiographs, if taken, may reveal changes consistent with hypovolemia such as microcardia and attenuation of the pulmonary vasculature and caudal vena cava. A small percentage of patients with hypoadrenocorticism may also have megaesophagus, and aspiration pneumonia may be present in these dogs due to regurgitation.1,3

Definitive diagnosis of hypoadrenocorticism is made on the basis of ACTH stimulation test. Venous blood is collected in either a heparinized or serum separator tube (depending on the laboratory) for baseline cortisol determination. Cortrosyn (synthetic cosyntropin) 5 μg/kg is given intravenously and a second blood sample is collected 60 minutes later. Minimal or absent cortisol secretion in response to ACTH is consistent with hypoadrenocorticism.

Other diagnostics that may be warranted in the atypical Addisonian include aldosterone levels and endogenous ACTH levels. Aldosterone levels may be useful in differentiating primary from secondary hypoadrenocorticism as dogs with secondary disease generally have normal aldosterone levels. Dogs with primary hypoadrenocorticism can also initially have glucocorticoid deficiency without mineralocorticoid deficiency. In these dogs, it is assumed that mineralocorticoid deficiency may develop in the future as adrenal atrophy progresses. Aldosterone levels may therefore be helpful in these cases to detect occult mineralocorticoid deficiency. However, whether aldosterone levels may be used to predict the development of mineralocorticoid deficiency in atypical Addison’s disease is not known at this time. Endogenous ACTH levels are the most reliable means of differentiating primary from secondary disease. Animals with primary hypoadrenocorticism will have endogenous ACTH concentrations that are very high, while animals with secondary (ie hypothalamic or pituitary) forms of hypoadrenocorticism will have low ACTH levels.

**Treatment of the Addisonian crisis**

Treatment of patients suspected to be experiencing an Addisonian crisis should be instituted immediately with intravenous fluid therapy. 0.9% sodium chloride is the fluid of choice and should be administered *to effect* through a large bore intravenous catheter. A shock rate of fluids in the dog may be calculated at 90 ml/kg over the first hour. This is typically given in increments of one-quarter to
one-third of the calculated dose, reassessing every 10-15 minutes. The goal of fluid therapy should be normalization of vital signs such as heart rate, level of consciousness, pulse quality, blood pressure, capillary refill time, body temperature, and urine output, rather than the administration of an arbitrary fluid volume. Care should be taken in patients with severe hyponatremia not to correct sodium levels too quickly as neurological deficits may result. An increase in sodium concentration of 12-14 mEq/L per 24 hours is generally accepted to be a safe rate of correction.

Relative or absolute bradycardia should be immediately investigated by monitoring electrocardiography and serum electrolyte concentrations. The presence of severe echocardiographic changes such as atrial standstill, widened QRS complexes, or sine wave formation provide strong indication for the administration of calcium gluconate. Calcium gluconate (10%) is given slowly at a dose of 0.5-1.5 mL/kg IV while carefully watching the patient’s ECG for arrhythmias. Although calcium gluconate does not lower the serum potassium level, it has the immediate effect of buffering the myocardium from the toxic effects of hyperkalemia by restoring the normal difference between resting and threshold membrane potentials. Other intermediate to long-term interventions for hyperkalemia include the administration of regular insulin/dextrose and sodium bicarbonate. Regular insulin is given intravenously at a dose of 0.1 unit/kg to promote potassium uptake by the cells through stimulation of sodium-potassium pumps. It is typically followed by a 50% dextrose “chaser” of 2 gm per unit of insulin to avoid hypoglycemia related to insulin administration. Blood glucose should then be monitored carefully for several hours afterward. Sodium bicarbonate is given at a dose of 1 mEq/kg intravenously to effect intracellular potassium shifting in exchange for hydrogen ions.

Glucocorticoid therapy should be rapidly initiated in the Addisonian crisis patient. Dexamethasone sodium phosphate is preferred in the emergency setting, because unlike prednisone, it will not interfere with cortisol assays. Dexamethasone may be administered before or during the ACTH stimulation testing at a dose of 0.2-0.5 mg/kg. Remember that dexamethasone is 6-7 times as potent as prednisone, so this dose is substantially above the physiologic range. Subsequent glucocorticoid requirements may be fulfilled using prednisone at a dose of 0.2-0.5 mg/kg twice daily for the remainder of the hospital stay.

Although electrolyte abnormalities can generally be corrected with fluid therapy alone, mineralocorticoid supplementation should be initiated once stable to maintain sodium reabsorption and potassium excretion. Fludrocortisone acetate (Florinef) is most commonly used pending results of the ACTH stimulation test, and can be given orally at a starting dose of 0.01-0.02 mg/kg every 12 hours. Because of the inconvenience of multiple daily pill administrations, many patients are switched to desoxycorticosterone pivalate (DOCP) at the time of discharge. DOCP is labeled for use at a dose of 2.2 mg/kg subcutaneously every 25 days though clinical experience suggests that much lower doses are actually needed, particularly in larger dogs. Most dogs are successfully treated with a monthly dose of no greater than 1 mL of DOCP (25 mg/mL) with giant breed dogs receiving 1.5 mL. Electrolytes are typically measured at 12 and 25 days after each of first 2-3 treatments and the dosage adjusted downward in 10% increments or the interval between doses extended by 48 hr increments.

Outcome

The prognosis for dogs and cats with primary hypoadrenocorticism is generally good, and a normal quality of life can be expected as long as hormone replacement therapy is provided. This can generally be accomplished with either daily florinef or monthly DOCP as described above. Physiologic doses of prednisone are often required, though many dogs will tolerate dose reduction, requiring approximately 0.2 mg/kg every 24-48 hours. Prednisone supplementation may not be required in dogs receiving florinef as this drug possesses some glucocorticoid effects. Owners should be advised to increase the prednisone dose during boarding, hospitalization for non-adrenal illness, travel, other stressful situations, or if symptoms consistent with hypoadrenocorticism are noted.

References


Cardiopulmonary cerebral resuscitation (CPCR) refers to the re-establishment of circulation and preservation of neurologic function following an arrest. Since its inception in the late 1800's, CPCR has saved the lives of countless human and veterinary patients. However, low overall survival rates following CPCR indicate that there is still much room for improvement in these practices. This session reviews current practices and updates on CPCR in the veterinary patient with an emphasis on evidence-based guidelines derived from the RECOVER initiative.

**Basic life support**

Basic life support refers to the process of establishing an airway, initiating positive pressure ventilation, and performing chest compressions. Because cardiopulmonary arrest (CPA) in veterinary patients is frequently initiated by respiratory arrest, an ABC approach is generally taken as described below. In recent years, there has been a paradigm shift prioritizing chest compressions above all other measures (CAB approach).

**Circulation**

Chest compressions are initiated at a rate of 100-120 per minute, compressing the circumference of the chest by approximately 30-50%. The patient should be in lateral recumbency during compressions. In smaller dogs, where the cardiac pump theory is believed to predominate, hands should be placed over the ventral third of the chest just behind the point of the elbow, corresponding to a position directly over the heart. In larger dogs, the thoracic pump theory is believed to be most important in generating blood flow, and hands should therefore be placed over the widest part of the thorax to create a maximal rise in intrathoracic pressure.

**Airway**

Orotracheal intubation is easily achieved in dogs, as the larynx can be directly visualized by retracting the tongue. The head and neck should be gently extended and a laryngoscope may be used to improve visualization of the larynx. In cases where hemorrhage, saliva, or gastric contents interfere with visualization, suction may be helpful. Alternately, the glottis may be palpated with one finger used to guide tube placement. Once tube placement is verified, the tube should be secured by tying to the nose or around the back of the head. The cuff should be inflated, and assisted ventilation provided. If chest wall excursion is not seen, lung sounds are absent, or abdominal distension is noted, tube placement should be reconfirmed by direct visualization and the cuff should be reinflated. Improper tube placement and tube dislodgement are common causes of CPCR failure.

**Breathing**

Once an endotracheal tube is in place, breathing is initiated at a rate of 10 breaths per minute with 100% oxygen to a tidal volume of approximately 10 ml/kg. An ambu bag with attached oxygen line is ideal for this purpose. If only one person is available to perform CPR, 2 breaths should be given for each 30 chest compressions. If several trained personnel are available, then breaths may be delivered independent of compressions. Chest wall excursion should be seen with each delivered breath. Airway pressures Ideally should not exceed 20-30 cm H2O. High airway pressures or inadequate chest wall excursion should prompt a search for pleural space disease, tube malposition, or tube occlusion.

A number of alternative techniques have been investigated that may help to augment blood flow during CPCR. Those that are directly applicable in veterinary patients include circumferential chest compression and interposed abdominal compressions. Circumferential chest compression is most commonly performed in cats and small dogs by encircling the chest with both hands to maximize the rise in intrathoracic pressure during chest compression. In larger animals, interposed abdominal compression may be implemented by having an additional person perform abdominal compressions during the relaxation phase between chest compressions. Interposed abdominal compressions increase venous return to the heart, leading to greater stroke volumes and cardiac output, and have been associated with increased survival to discharge in human patients.

**Advanced life support**

Advanced life support consists of drug administration, determination of cardiac electrical activity, and application of electrical defibrillation if indicated. These techniques build upon basic life support to increase the likelihood of successful resuscitation.

**Drugs**

Establishing vascular access is one of the first priorities during advanced life support. While central lines are preferable for rapid distribution of drugs, peripheral catheters are acceptable, and drug delivery may be facilitated by following drug administration with a 10-20 ml IV fluid “chaser”. If vascular access is not immediately obtained, surgical cutdown or intraosseous techniques should be considered. The intratracheal route may also be used initially to deliver drugs. Epinephrine, atropine, vasopressin, lidocaine, and
naloxone may all be given in this way by administering twice the normal dose of the drug (or using the “high” dose for epinephrine) and administering several large breaths to disperse the drug.

Drugs administered during CPCR include intravenous fluids, narcotic reversal agents, vasopressors, vagolytics, antiarrhythmics, and potentially sodium bicarbonate. Shock doses of intravenous fluids should be provided in cases where hypovolemia is believed to have played a role in the arrest. Moderate fluid rates should be used in euvolemic patients or patients with underlying heart disease, as rapid administration in these cases may excessively elevate right atrial pressure and consequently decrease myocardial and cerebral perfusion pressure.

Patients who have received narcotic pain relievers or other sedative/anesthetic drugs prior to arrest should immediately be given the reversal agent for that drug. Naloxone may be used to reverse most narcotics at a dose that is isovolumetric to the dose of the original narcotic, or at 0.02-0.04 mg/kg IV if the original dose is unknown. Flumazenil (0.02 mg/kg IV) may be used to reverse benzodiazepines, and yohimbine (0.1 mg/kg) or atipamazole (0.2 mg/kg or isovolumetric) may be used to reverse xylazine and medetomidine respectively. Any anesthetic gases, if still in use, should be discontinued and the anesthetic circuit flushed with fresh oxygen.

Vasopressors are commonly used during CPCR to increase blood pressure and redistribute blood flow to vital organs like the brain and heart. Epinephrine continues to be the vasopressor of choice during CPCR in veterinary patients, though its use is largely extrapolated from clinical studies in human patients. Both low dose and high dose epinephrine protocols are described in human medicine. While high dose epinephrine has been associated with increases in early return of spontaneous circulation, no long-term benefits have been identified. High dose epinephrine has additionally been associated with increased myocardial oxygen demand and worse neurologic outcomes. For these reasons, it is recommended that low dose epinephrine initially be administered every 3-5 minutes during CPCR, switching to the high dose only if there is a lack of response to the lower doses. Epinephrine dosing may be rapidly calculated according to the following rule of thumb: 0.1 ml per 20 lb of the 1:1,000 formulation for low dose, or 1 ml per 20 lb for high dose.

Vasopressin is another potent vasoconstrictor that is increasingly used in resuscitation of human patients. Unlike epinephrine, it does not increase myocardial workload, and its effect is not blunted by acidosis. Although clinical data in veterinary patients is currently lacking, animal models and human clinical trials suggest that vasopressin may be as effective as epinephrine.3 Vasopressin (0.8 units/kg IV) may therefore be considered as an alternative to epinephrine in dogs.4

Atropine is another drug frequently administered during CPCR to reverse parasympathetic contribution to the arrest or to treat sinus bradycardia. Atropine is administered at a dose of approximately 1 ml per 20 lb (0.04 mg/kg) for asystole or pulseless electrical activity. When treating sinus bradycardia, only half this dose is needed.

Sodium bicarbonate use in CPCR is controversial, as it has been associated with numerous adverse effects including hypernatremia, paradoxical CNS acidosis, and decreased resuscitation rates in people. However, its use should still be considered during long duration (>10 minutes) arrests, as control of acidosis may improve response to catecholamines as well as post-arrest neurologic outcomes. Bicarbonate is typically given only after 10 minutes of CPCR at a dose of 1 mEq/kg and is repeated every 5 minutes thereafter.

**Electrical activity**

ECG leads should be attached as soon as feasible to assess electrical activity. Connecting the leads to the skin of the lower forelimbs and hindlimbs will help to minimize motion artifact associated with resuscitation efforts. Four rhythms are commonly seen during cardiopulmonary arrest in dogs. Asystole and pulseless electrical activity are the initial arrest rhythms most commonly seen in dogs, followed by ventricular fibrillation and sinus bradycardia.5,6 Accurate ECG diagnosis is vital to a successful code. The presence of sinus bradycardia or suspicion of a vagal arrest should prompt administration of atropine. Asystole should be confirmed in more than one lead, to rule out the possibility of artifact related to poor contact. While some dogs in asystole will convert directly to sinus rhythm following resuscitation, many develop ventricular fibrillation and require electrical shock for conversion. Once ventricular fibrillation is identified, electrical defibrillation should immediately be administered, temporarily bypassing all other resuscitation measures. The greater the time that a dog spends in fibrillation, the lower the likelihood of successful conversion.

**Defibrillation**

Early application of electrical shock is the only effective method for converting VF to sinus rhythm. VF is a form of disorganized electrical activity with various portions of the heart muscle firing at different times. Electrical shock essentially "resets" the cardiac cells so that organized activity can resume. Practically speaking, applied current must pass through at least 30% of cardiac myocytes to effectively convert VF.

To accomplish defibrillation, the dog is flipped into dorsal recumbency immediately preceding defibrillation and handheld paddles are placed on either side of the chest directly over the heart. Ample conducting gel should be applied to the paddles to ensure good contact and prevent dispersion of current. The chest should be compressed between the paddles, minimizing impedance by narrowing the distance between paddles. If using a monophasic defibrillator, the energy for the first shock should be set at 3-5 J/kg. If defibrillation is not successful, CPCR is resumed for 60-90 seconds and a subsequent shock should then be given at the same energy.
setting. Electrical shock is discontinued once the rhythm converts from VF. Lower energy biphasic shock waveforms have been shown to be as effective as higher energy monophasic waveforms and exclusively used at this time in human patients. If using a biphasic defibrillator, the pediatric settings should be used (2-4 J/kg). 7

For shock-refractory VF, a search should be undertaken to identify problems such as improper paddle position, inadequate contact, insufficient conduction gel, or the presence of pleural space disease that may increase impedance. Drug-shock techniques may then be considered, administering epinephrine or amiodarone (5 mg/kg IV) prior to shock to lower defibrillation threshold. Lidocaine was previously used for this purpose as well, but has been reclassified as a therapy of indeterminate benefit in the most recent ACLS guidelines.7

Open chest CPCR
There are a number of absolute indications for open chest CPCR. These include cardiac arrest caused by or associated with pleural space disease (pneumothorax, pleural effusion, diaphragmatic hernia), pericardial effusion, or penetrating injury resulted in cardiac arrest. However, debate exists in veterinary medicine as to other indications for performing open chest CPCR. Some advocate open chest CPCR immediately in large breed dogs because of the limited success of restoring adequate circulation with external compressions while others prefer to perform external CPCR for 5 minutes and then open the chest if there is little or no evidence of effective circulation. Open chest CPCR has the advantage of allowing the clinician to directly compress the heart and improve stroke volume. In addition, opening the chest makes assessment of ventricular filling feasible aiding in the decision of volume delivery.

When opening the chest, it is critical to auscult the chest just prior to the incision to rule out ECG dysfunction as the cause of asystole. The left chest should be crudely clipped of hair at the left 5th-6th intercostals space and a chlorhexidine based antisepic solution should be briskly applied. An incision should be made through the skin and subcutaneous tissues from just below the spinal musculature to the level of the costochondral junction. Between positive pressure breaths, mayo scissors should be used to poke through the intercostal musculature and the pleura and the chest is opened by sliding the mayo scissors dorsally and ventrally along the cranial border of the rib (to avoid the neurovascular bundle). The pericardium is opened at the pericardio-diaphragmatic ligament and the heart is compressed from the apex to the base. In large dogs, the heart can be compressed against the opposite chest wall.

In the event of return of spontaneous circulation, antibiotics should be instituted immediately, the chest should be lavaged with copious amounts of warm saline, and should be closed using sterile technique over a chest tube.

ICU care
Following a successful code, a search for underlying causes or complications should be performed and any problems corrected. Blood gases, hematocrit and total solids, blood pressure, and oxygen saturation are carefully monitored and optimized during this time. This tends to be the most challenging phase of arrest management, as complications and recurrence of CPA are common. Neurologic recovery is promoted by maintaining arterial blood pressure and oxygen saturation. Because elevation in carbon dioxide levels leads to cerebral vasodilation and consequently increased intracranial pressure, hypercarbia should be prevented by employing mechanical ventilation if needed. Once cardiovascually stable, mannitol (0.25-0.5 g/kg IV over 20 minutes) may also be indicated to treat cerebral edema and resultant elevations in intracranial pressure. Corticosteroids are associated with potentially deleterious hyperglycemia in post-arrest patients, and current protocols do not support their use.7

Prognosis
Recurrence of CPA in the post-arrest period is common, occurring in up to 70% of successfully resuscitated dogs. Intensive care and monitoring during this time is therefore essential. Survival to discharge following cardiopulmonary arrest has been reported in 4-11% of cases.5,6,8 Transient blindness, seizures, circling, ataxia, and decreased level of consciousness are common for some period of time following CPA, but the majority of survivors have a good prognosis for functional recovery.6

References
2005 American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2005;112(suppl.)
Clinical Approach to Anemia
Ari Jutkowitz, VMD, DACVECC
Michigan State University
East Lansing, MI

Anemia is commonly seen in veterinary emergency and critical care medicine. Patients may be brought in with the presenting complaint of anemia or may develop anemia during hospitalization as a result of their underlying disease or treatment. Anemia may contribute to patient morbidity, cost of treatment, and length of stay, frequently necessitating expensive interventions such as blood transfusion while the underlying disease is being treated.

Classification of anemia
Anemia seen in veterinary patients may be classified into three broad categories that relate to cause; blood loss, hemolysis, and decreased production. Classification of anemia in this way is not merely academic but is crucial to the workup of anemic patients. Because regenerative and non-regenerative anemia have different sets of differentials and diagnostics, this classification will guide further testing and provide useful prognostic information.

Three simple, in-house diagnostic tests can be performed in all anemic patients to help classify their anemia. These tests are inexpensive, easy to perform, and will frequently provide a great deal of information about the cause of the anemia. They can all be performed in approximately 5 minutes, allowing the clinician to classify the anemia and provide an appropriate diagnostic plan while the owner is still present at the hospital during the initial exam.

The first test is the packed cell volume (PCV) and total solids (TS). The importance of interpreting the PCV in conjunction with the total solids cannot be overemphasized. If the PCV and TS are both low, acute blood loss should be suspected. In contrast, a low PCV with normal total solids would be consistent with hemolysis or decreased red blood cell production. To differentiate these two clinical entities, the plasma of the spun sample should be carefully evaluated for the presence of hemoglobin or bilirubin that may suggest intravascular or extravascular hemolysis respectively.

The second test that should be performed is the blood smear. Blood smears are useful for differentiating hemolysis from decreased production anemia as the presence of significant polychromasia and anisocytosis may indicate the presence of a regenerative response. Blood smears should also be evaluated for blood parasites and telltale alterations in red blood cell morphology. Heinz bodies are characterized by bulging of the red blood cell membranes and indicate oxidative red blood cell damage secondary to toxins such as onions, garlic, or propylene glycol. Spherocytes are small, round erythrocytes with loss of central pallor that result when antibodies bound to red blood cell membranes lead to a portion of the membrane being phagocytized or “pinched off” by macrophages. Large numbers of these cells are typically seen in dogs with immune-mediated hemolysis. Schistocytes are erythrocytes that have become fragmented as a result of passage through narrowed microvasculature. Schistocytes typically reflect microangiopathic causes of hemolysis such as caval syndrome, disseminated intravascular coagulation, hemangiosarcoma, or splenic torsion. Acanthocytes are red blood cells with long spiny projections that are frequently seen in patients with hepatic or splenic neoplasia, though they may also be seen in animals with disorders of lipid metabolism as well.

Finally, a slide agglutination test should be performed when hemolysis is suspected. In this test, a drop of anticoagulated blood from a purple top tube or capillary tube is mixed with several drops of saline. Autoagglutination may be evidenced by the observation of obvious flecks within the drop of blood. The saline is used to disperse rouleaux that may mimic agglutination. Autoagglutination is caused by cross-linking of antibodies bound to the erythrocyte membranes, and as such is diagnostic for an immune-mediated component to the hemolysis.

Formulating a list of differential diagnoses
Once the anemia has been classified as blood loss, hemolysis, or decreased production, a list of differentials may be formulated. (see table 1)

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<th>Hemolysis</th>
<th>Decreased Production</th>
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Table 1. Some common differentials for anemia
**Acute blood loss**

Diagnosing acute blood loss is simple when an external source of bleeding is present. However, cavity bleeding, gastrointestinal losses, coagulopathies, and chronic blood loss may be more challenging clinical entities. History and physical exam are usually the key elements in identifying a source of acute blood loss. Additional diagnostic testing generally includes minimum database (complete blood count, serum biochemistry panel, and urinalysis) and imaging studies such as radiographs and/or ultrasound to identify the source of bleeding. Platelet counts and coagulation testing should be performed any time hemostatic defects are suspected.

Gastrointestinal blood loss should be suspected when external or cavitary bleeding is not identified. Significant gastrointestinal blood loss may occur before signs of melena, hematemesis, or hematochezia are noted.

**Hemolysis**

In patients presenting with hemolysis, CBC, chemistry, and urinalysis should be run as part of a minimum database. The presence of hemoglobinemia/hemoglobinuria or bilirubinemia/bilirubinuria may suggest intravascular or extravascular hemolysis, respectively. Leukocytosis is frequently noted on the CBC from patients with IMHA and may result from non-specific “gearing up” of the bone marrow, or more likely, from tissue damage secondary to hypoxia and thrombosis. White blood cell counts in excess of 40,000/µl have been associated with a poorer prognosis in dogs with IMHA. Platelet counts should also be evaluated. Moderate thrombocytopenias may suggest consumptive coagulopathy or tick-borne illness, while severe thrombocytopenias (<50,000/µl) should prompt consideration of a concurrent immune-mediated thrombocytopenia. A Coombs test is indicated if hemolysis is suspected but autoagglutination is not present. The Coombs test, or direct antiglobulin test, is essentially a test for the presence of antibodies or complement bound to erythrocyte membranes. It is performed by adding anti-dog antibodies (immunoglobulins directed against canine IgG, IgM, or complement) to a sample of the patient’s red blood cells. If autoantibodies are present on the patient’s blood cells, the antiserum binds to them and cross-linking occurs. Because the end (positive) result of this test is agglutination, the Coombs test need not be run if the patient is already autoagglutinating. Note also that the Coombs test is not highly sensitive. Review of cases seen at our hospital (unpublished data) identified a sensitivity of only 66%, comparable to other reports in the veterinary literature.

A search should also be conducted for possible trigger factors. History taking should include questioning about recent vaccinations or medications. Recent vaccination (ie. within 4 weeks) has been associated with the development of IMHA in retrospective studies. Sulfur drugs, penicillins, and cephalosporins have also been associated with IMHA by acting as haptens, substances that become adsorbed to erythrocyte membranes and subsequently are able to stimulate an immune response. Neoplastic processes such as hemangiosarcoma, lymphoma, leukemia, and histiocytic sarcoma are another common trigger factor, and chest radiographs and abdominal ultrasound are rule out these entities. Testing should also be performed for vector-borne illnesses such as Ehrlichiosis, Babesiosis, Bartonellosis, and Hemoplasmosis. FeLV and FIV testing should not be overlooked in the cat.

Non-immunologic causes for hemolysis should also be considered. In addition to the oxidative toxins such as onions, ingestion of zinc may result in fulminant intravascular hemolysis, hemoglobinuria, multi-organ dysfunction, and DIC. Hereditary diseases such as phosphofructokinase deficiency seen in English Springer Spaniels may result in episodic hemolytic anemia, easily confused with IMHA because of its “apparent” response to steroids.

Reticulocyte count should always be performed to assess regenerative response. Hemolytic anemias are typically strongly regenerative, though it may take three days for regenerative response to be noted. The presence of a non-regenerative anemia (absolute reticulocyte count < 60,000/µl) should prompt suspicion of non-regenerative immune-mediated anemia (NRIMA), bone marrow disease, or other forms of decreased production anemia described below.

### Other Red Blood Cell Disorders

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Immunosuppressive therapies like prednisone ideally should not be initiated until neoplastic and non-immunologic causes of hemolysis have been ruled out, as the use of these drugs may interfere with accurate diagnosis and subsequent therapies. However, in cases where IMHA is strongly suspected and clinical signs are severe, prednisone is typically started pending labwork to avoid excessive delays in therapy.

**Decreased production**

An anemia should be considered non-regenerative when the reticulocyte count is less than 60,000/µL (corresponding to a corrected reticulocyte count of less than 1%). However, it should be noted that a regenerative response usually becomes apparent after a minimum of 2-3 days, so acute blood loss or hemolysis may initially appear to be non-regenerative. Once a non-regenerative anemia is identified, bone marrow aspiration is generally indicated. Differentials for decreased production anemia may be grouped according to bone marrow histopathology. Some diseases cause a selective hypoplasia of red cell lines, while others affect all cell lines within the bone marrow (see table 1 above).

**Selective erythroid hypoplasia**

Anemia of chronic disease, also termed anemia of inflammation, is immune driven. Cytokines and cells of the reticuloendothelial system (RES) induce changes in iron homeostasis, erythrocyte lifespan, production of erythropoietin, and proliferation of erythroid lines. Iron is diverted from circulation to storage sites within the RES, limiting availability for erythroid progenitors. Inflammatory mediators (TNF, IL-1, IFN) suppress activity of erythroid precursors and decrease their responsiveness to erythropoietin. Release of erythropoietin is also inhibited. Finally, erythrophagocytosis and free radical mediated erythrocyte damage shorten RBC survival. In contrast to iron deficiency anemia, anemia of chronic disease tends to be normocytic, normochromic, rather than microcytic, hypochromic. Serum iron tends to be low, but bone marrow iron stores are adequate. Anemia of chronic disease is generally mild to moderate unless complicated by other factors such as blood loss or hemolysis. Treatment is therefore directed at correcting the underlying disease. Transfusion may be considered if anemia is associated with clinical signs. Iron supplementation for anemia of chronic disease is controversial, and indications for its use in veterinary patients with chronic disease is unclear.

Chronic renal failure is typically associated with mild to moderate anemia. Because of the gradual and chronic nature of this type of anemia, it tends to be well compensated until very advanced stages. Anemia in renal failure is multifactorial and results from decreased erythropoietin production by the kidney, impaired responsiveness of bone marrow precursors, shortened RBC lifespan due to uremia, and GI blood loss resulting from uremic ulcers. Anemia of renal failure is typically well compensated, though transfusions may be indicated in the event of concurrent losses or surgery. Human recombinant erythropoietin has been used to stimulate RBC production in veterinary patients with renal failure, but is increasingly being used only as a “last ditch effort” as antibody production against epogen may lead to antibodies being directed against the patient’s own erythropoietin as well. Canine recombinant erythropoietin has not been associated with antibody production in dogs with renal failure, but is unfortunately not commercially available. Anabolic steroids (Winstrol-V) have been used in patients with renal failure based on the observations that they increase RBC mass in healthy animals. A benefit in these cases has not been clearly identified.

Pure red cell aplasia (PRCA) and precursor-targeted immune mediated anemia (PIMA) are immune mediated diseases directed against erythrocyte precursors. The anemia is non-regenerative, normocytic-normochromic, with normal leukocyte and platelet counts. Animals tend to present with marked anemia, as the progression of the disease is typically slow and there is adequate time to mount a compensatory response. Diagnosis is made on the basis of bone marrow aspiration or biopsy, with few to no erythroid precursors seen in PRCA. In cases of PIMA, left shifted erythroid hyperplasia is frequently seen, with maturation arrest at the level of the metarubricytes or rubricytes. Some animals with PIMA will also have immune mediated destruction of mature erythrocytes, resulting in concurrent hemolysis. Cats with PRCA should always have PCR or IFA performed on the bone marrow to rule out feline leukemia C associated attack on erythroid progenitors, as this form of PRCA is typically fatal. Treatment for immune-mediated PRCA and PIMA relies on immunosuppressive therapies similarly to IMHA. Periodic transfusions may be needed until regeneration occurs. This may take weeks to several months. Clinical signs and progression tend to be less severe than IMHA, as the anemia results from decreased production, rather than hemolysis.

Endocrine diseases such as hypothyroidism and hypoadrenocorticism may also result in a decreased production anemia. Both cortisol and thyroid hormone have a permissive role in the response of red blood cell precursors to erythropoietin. These forms of anemia are generally mild unless complicated by concurrent blood loss and resolve with hormone replacement therapy.

**Generalized bone marrow hypoplasia**

Generalized bone marrow hypoplasia may result from radiation, toxic, or infectious insults to the bone marrow. Common toxins include estrogen, chloramphenicol, phenylbutazone, antifungals, and chemotherapeutic drugs. Infectious diseases resulting in bone marrow hypoplasia include feline leukemia and chronic Ehrlichiosis. Generalized bone marrow hypoplasia may also result from the crowding out of normal bone marrow precursors by neoplastic cells, a process termed myelophthisis. The most common neoplastic causes are the hematopoietic and lymphoid neoplasms including lymphosarcoma, granulocytic leukemia, and lymphoid leukemias. Myelofibrosis, the replacement of marrow spaces by connective/scar tissue, usually represents the endpoint of previous severe marrow 146
injury (as in the case of estrogen toxicity and ionizing radiation) or it may occur spontaneously. Peripheral blood features of myelofibrosis usually include severe nonregenerative anemia, severe leukopenia, and a variable platelet response. Confirmation of the diagnosis depends on marrow core biopsy with a demonstration of connective tissue filling the marrow space.

**Normal to hypercellular bone marrow**

Iron deficiency anemia results from chronic blood loss. In young animals, parasitic infection is the primary ruleout for iron deficiency anemia, while in older animals, gastrointestinal masses or ulcers are generally implicated. Chronic blood loss leads to depletion of bone marrow iron stores over time, resulting in inability to form hemoglobin. Nuclear maturation of RBC precursors is normal however. Precursors continue to divide, getting smaller in size because they never acquire a complete amount of hemoglobin. This results in a hypercellular bone marrow with a build up of metarubricytes. Diagnosis is based on the presence of microcytic, hypochromic anemia, thrombocytosis, source of blood loss, and a bone marrow smear containing no stainable iron. Low serum iron is not diagnostic as it may rapidly decrease with inflammatory disease as a result of tissue sequestration. Treatment is aimed at removal of the source of blood loss. Ferrous sulfate may be administered at a dose of 100-300 mg per day in dogs and 50-100 mg per day in cats if needed. Note that this dose refers to ferrous sulfate, not elemental iron. Reticulocytosis should develop within 3-4 days of supplementation.

Myelodysplasia refers to a poorly understood group of diseases characterized by non-regenerative anemia or pancytopenia and prominent dysplastic changes in the bone marrow. Abnormal erythrocytes are generally unable to completely differentiate and early cell death results. Myelodysplasia may result from idiopathic (primary), neoplastic, toxic, immune-mediated, or infectious (FeLV) causes. The myelodysplasias tend to carry a very guarded prognosis, with treatment aimed at immunosuppression, chemotherapy, and/or erythropoietin depending on the suspected cause.

**General comments on the treatment of non-regenerative anemias**

Treatment of decreased production anemia is best aimed at identifying and eliminating any underlying disease processes or myelosuppressive drugs. Once this is done, clinical experience suggests that the most important thing we can do for patient is to buy time for the bone marrow to repopulate with normal precursor cells. Blood transfusions should be provided as needed until the patient is able to mount a regenerative response of their own. Most patients with non-regenerative anemia require transfusions every 4-6 weeks until their disease is well controlled. Broad-spectrum antibiotics are indicated in the event of severe neutropenia to prevent secondary infections. Immunosuppressive agents may be indicated if an immune-mediated disease (eg. red cell aplasia) is identified or strongly suspected. Myeloproliferative diseases and myelodysplasia tend to carry a poor prognosis, but other forms of decreased production anemia may respond well if the underlying disease or insult is eliminated and adequate time is provided for recovery.

**Conclusion**

A variety of diseases, both immunologic and non-immunologic in nature, may result in anemia and/or hemolysis in veterinary patients. Successful management relies upon accurate diagnosis and treatment of the underlying disease process. Simple test to help classify a patient’s anemia as blood loss, hemolysis, or decreased production may facilitate correct diagnosis. Initiation of immunosuppressive therapy prior to performing a methodical search for infectious, neoplastic, or other causes of anemia may result in therapeutic “missteps” and treatment failure.

**References**


Immune-Mediated Hemolytic Anemia: Current Perspectives
Ari Jutkowitz, VMD, DACVECC
Michigan State University
East Lansing, MI

Immune-mediated hemolytic anemia (IMHA) is one of the most common hematologic diseases seen in dogs with a reported mortality rate that ranges between 29% and 77% in the veterinary literature. Hemolysis results from the binding of immunoglobulins to red blood cell surface antigens, causing those cells to be lysed by complement intravascularly or removed from circulation by mononuclear phagocytes. IMHA may be a particularly frustrating disease for both owners and clinicians because of its waxing and waning clinical course, the potential for sudden complications, and the expense associated with treatment.

Immune-mediated red blood cell destruction may be classified in a number of ways. Primary or idiopathic autoimmune hemolytic anemia (AIHA) refers to immune-mediated hemolysis in the absence of an identifiable trigger factor, whereas secondary IMHA results from an underlying process such as neoplasia, infectious disease, or drug reaction. IMHA may also be categorized based on whether it results in intravascular or extravascular hemolysis. Intravascular hemolysis results from the lysis of red blood cells by complement within the vasculature, and may be identified by the presence of free hemoglobin within the plasma and urine. Extravascular hemolysis results when there are insufficient antibodies present to cause complement fixation, and antibody-labeled red blood cells are removed by the reticuloendothelial system within the spleen and liver. Extravascular hemolysis tends to be a more gradual process and may be identified by the presence of bilirubin, rather than hemoglobin within the plasma and urine. IMHA may also be classified based on the presence or absence of autoagglutination. Autoagglutination is the spontaneous clumping of red blood cells and results from the cross-linking of erythrocytes by large numbers of antibodies. We have noted autoagglutination in approximately 70% of dogs treated for IMHA.

IMHA is typically a disease of middle-aged to older pets. As with other types of immune-mediated disease, a female gender predisposition has been reported. At Michigan State University in the past eight years, approximately 2/3 of IMHA cases were seen in female dogs. Although any breed may develop IMHA, a number of breed predispositions have also been reported and include Cocker Spaniels, Poodles, Shih Tzus, Lhasas, Old English Sheepdogs, Border Collies, and Springer Spaniels. A seasonal predilection has also been suspected, as some studies have observed a larger number of cases presenting in spring and summer months. This may be a result of increased exposure to outdoor allergens or antigenic stimulation, or may simply reflect the overall increase in patient admissions seen during these months.

Clinical signs of IMHA may be acute or chronic, depending upon the rate of hemolysis. With chronic disease, symptoms such as lethargy, weakness, inappetance, vomiting, diarrhea, and pigmenturia are most commonly reported, whereas with more rapid hemolysis, acute collapse may be the first symptom noted. It is not uncommon for dogs to be brought in for “possible urinary tract infection” because the owners have noted discoloration of the urine with hemoglobin or bilirubin. Symptoms related to anemia, including tachycardia, tachypnea, and systolic ejection murmurs may also be noted on physical exam. Hepatosplenomegaly is not unusual as these organs are common sites for extramedullary hematopoiesis as well as clearance of antibody-labeled erythrocytes. Fevers are frequently seen as a result of release of endogenous pyrogens like IL-1 and IL-8. Reactive lymphadenopathies may also be seen.

Initial in-house diagnostics should include PCV/TS, blood smear, and slide agglutination test, as these are inexpensive, easy to perform, and will frequently provide a great deal of information about the cause of the anemia. The importance of interpreting the PCV in conjunction with the total solids (TS) cannot be overemphasized. If the PCV and TS are both low, blood loss (rather than hemolysis) should be suspected. In contrast, a low PCV with a normal TS would be consistent with hemolysis or decreased red blood cell production. To differentiate these two clinical entities, the plasma of the spun sample should be carefully evaluated for the presence of hemoglobin or bilirubin that may suggest hemolysis. Blood smears may also be useful in differentiating hemolysis from decreased production anemia, as the presence of significant polychromasia and anisocytosis indicates the presence of a regenerative response. Blood smears should also be evaluated for blood parasites and telltale alterations in red blood cell morphology. Spherocytes are small, round erythrocytes with loss of central pallor, that result when antibodies bound to red blood cell membranes lead to a portion of the membrane being phagocytized or “pinched off” by macrophages. Large numbers of these cells are typically seen in dogs with immune-mediated hemolysis. Ghost cells, which appear as “empty” cell membranes may be seen with intravascular hemolysis. Finally, a slide agglutination test should be performed when hemolysis is suspected. In this test, a drop of anticoagulated blood from a purple top tube or capillary tube is mixed with several drops of saline. Autoagglutination may be evidenced by the development of obvious flecks within the drop of blood. Autoagglutination is caused by cross-linking of antibodies bound to the erythrocyte membranes, and as such is diagnostic for an immune-mediated component to the hemolysis.

A number of other diagnostics should be considered in the evaluation of animals suspected to have IMHA. CBC, chemistry, and urinalysis should be run as part of a minimum database. The presence of hemoglobinemia/hemoglobinuria or
bilineubinemia/bilirubinuria may suggest intravascular or extravascular hemolysis, respectively. Leukocytosis is frequently noted on the CBC from patients with IMHA and may result from non-specific “gearing up” of the bone marrow, or from tissue damage secondary to hypoxia and thrombosis. White blood cell counts in excess of 45,000/µl have been associated with a more guarded prognosis. Platelet counts should also be evaluated. Moderate thrombocytopenias may suggest consumptive coagulopathy or tick-borne illness, while severe thrombocytopenias (<50,000/µl) should prompt consideration of a concurrent immune-mediated thrombocytopenia. Reticulocyte count should always be performed to assess regenerative response. Immune-mediated hemolytic anemias are typically strongly regenerative, though it may take three days for regenerative response to be noted. Non-regenerative anemias should prompt suspicion of red cell aplasia, precursor-directed immune-mediated anemia (PIMA), or other form of decreased production anemia. A Coombs test is indicated if hemolysis is suspected but autoagglutination is not present. The Coombs test, or direct antiglobulin test, is essentially a test for the presence of antibodies or complement bound to erythrocyte membranes. It is performed by adding anti-dog antibodies (immunglobulins directed against canine IgG, IgM, or complement) to a sample of the patient’s red blood cells. If autoantibodies are present on the patient’s blood cells, the antiserum binds to them and cross-linking occurs. Because the end (positive) result of this test is agglutination, the Coombs test need not be run if the patient is already autoagglutinating.

A search should also be conducted for possible trigger factors. History taking should include questioning about recent vaccinations or medications. Recent vaccination (ie. within 4 weeks) has been associated with the development of IMHA. Sulfa drugs, penicillins, and cephalosporins may also cause IMHA by acting as haptons, substances that become adsorbed to erythrocyte membranes. If these haptons are targeted by the immune system, the entire red blood cell may be destroyed. Neoplasias such as hemangiosarcoma, lymphoma, myeloproliferative diseases, and hemophagic histiocytosis are another common trigger factor, and chest radiographs and abdominal ultrasound are frequently performed to rule out these entities. Testing should also be performed for tick-borne illnesses such as Ehrlichiosis and Babesiosis.

Treatment of IMHA consists of improving tissue oxygen delivery, suppressing the immune response, preventing some of the major complications of IMHA (such as thromboembolic disease), and hopefully preventing future recurrence. In the emergent patient, tissue oxygenation may be improved greatly by the administration of intravenous fluids. Although some clinicians worry about “diluting” an already anemic patient with IV fluids, in actuality, fluids will improve tissue oxygen delivery in the hypovolemic patient by maximizing cardiac output. However, the majority of dogs with IMHA will also require blood transfusion or oxyglobin during the course of their hospitalization, as immunosuppressive therapies are not rapidly effective in stopping the hemolytic process. The decision to transfuse is based on a number of factors, including hematocrit values, clinical signs, and the chronicity of the anemia. Clinical signs of anemia such as reluctance to eat, tachycardia unresponsive to fluids, tachypnea, dyspnea, lethargy, and altered mentation should prompt consideration for transfusion.

A number of drugs may be considered for the purpose of immunosuppression. Prednisone is the mainstay of therapy in dogs with IMHA, and at this time no other drug has been proven to work better than prednisone alone. Clinical experience suggests that 2 mg/kg/day in dogs provides adequate immunosuppression in the dog. Higher doses are not necessarily more immunosuppressive but may be associated with an increased risk of gastrointestinal complications. Prednisolone, rather than prednisone, should be used in cats as the bioavailability of prednisone is limited in this species. Additionally, cats may require higher doses than dogs, and the author typically uses 4 mg/kg/day in cats. A growing number of retrospective studies have suggested that azathioprine may improve long-term survival, and that cyclophosphamide may be associated with a poorer outcome. Caution should be used in interpreting these studies as inherent bias may be present due to their retrospective or small scale nature. In our clinic, we frequently use azathioprine (2 mg/kg q24h for 7 days then q48h), cyclosporine (Atopica 5-10 mg/kg/day divided), or mycophenolate (10 mg/kg PO q12h) as adjunct therapies and to facilitate prednisone weaning later in the course of treatment. Intravenous immunoglobulin (IVIG) is also occasionally used in patients who are slow to respond to conventional therapy. IVIG is essentially purified IgG antibodies collected from the pooled plasma of over 2000 human donors. It is believed to act primarily by blocking macrophage Fc-receptors, thereby decreasing phagocytosis of red blood cells. Downregulation of antibody production, enhanced catabolism of antibodies, and suppression of cytokine release are other possible mechanisms of action. Although one small prospective study did not demonstrate more rapid response times in IMHA patients receiving IVIG at presentation, clinical experience and a number of retrospective studies have demonstrated its utility as a rescue therapy in individual patients. IVIG is typically dosed at 0.5-1 mg/kg given over 6 hours. Side effects include vomiting, fever, potential for anaphylaxis, and possible increased risk of thrombosis.

Thromboembolic disease (TE) is a frequent complication of IMHA. In studies of dogs with IMHA that underwent necropsy, TE was identified in 60-80% of cases. Sites most commonly affected were the pulmonary and splenic vasculature. Although exact mechanisms for the prothrombotic state have not been elucidated, increased concentrations of procoagulant factors, decreased concentrations of anticoagulant and fibrinolytic factors, vasculitis, enhanced platelet reactivity, the presence of antiphospholipid antibodies, liberation of RBC stroma, blood transfusion, and administration of steroids have all been hypothesized to play a role in the development of TE. Changes in primary hemostasis are also thought to play a role in the development of a pro-thrombotic state. Weiss & Brazzell demonstrated increased platelet P-selectin expression in dogs with IMHA, supporting the hypothesis that platelets circulate in an activated state. Documentation of the pro-thrombotic state remains challenging in clinical cases and has traditionally
been based upon detection of increased fibrinolysis (increased fibrin degradation products (FDPs) and D-dimers) and decreased endogenous anticoagulants (antithrombin) rather than rate of clot formation. Recently, our group has documented hypercoagulability as assessed by thromboelastography in this patient population. 26/26 dogs with idiopathic IMHA enrolled in this study all had an MA (maximal amplitude; a reflection of clot strength) that was significantly greater than normal.

Though antemortem identification of thromboembolic events can be challenging, data from 110 dogs treated for IMHA at the Michigan State University Veterinary Teaching Hospital between 2004 and 2007 showed that 34% were suspected to have developed TE during their hospital stay. Of the dogs with suspected TE, 51% had pulmonary thromboembolism (PTE) alone, 8% had portal venous thrombosis (PVT) alone, and 41% had both PTE and PVT. The development of TE appears to significantly contribute to the morbidity and mortality of IMHA. In our data, survival to discharge in dogs with TE was significantly lower than in dogs without TE (49% vs 81%) and median duration of hospitalization was longer (7 days vs 4 days). Of note however, 7 of 7 dogs with PVT identified on ultrasound whose owners opted for aggressive therapy all survived, suggesting that early identification and management of this problem may improve outcome. We are currently evaluating CT angiography as a technique for definitive identification and monitoring of pulmonary and portal clots.

Because hemostatic abnormalities are common in dogs with IMHA, obtaining baseline coagulation testing at the time of admission is strongly recommended. In our critical care unit, dogs with IMHA are then treated with heparin sodium at a loading dose of 150 units/kg IV followed by a continuous infusion of 30-60 units/kg/hour. The heparin dose is adjusted daily to prolong the activated partial thromboplastin time (aPTT) to 1.5-2 times the baseline value. Twenty-six dogs prospectively enrolled in a coagulation study and heparinized based upon this protocol all survived to discharge and serial evaluation of thromboelastography showed normalization of parameters related to clot formation by 30 days, once hemolysis was no longer taking place. Low dose aspirin (0.5 mg/kg PO BID)\(^2\) may also be started during hospitalization, particularly in cases where there is failure to achieve a target aPTT. Plavix (2 mg/kg q24h) or aspirin (0.5 mg/kg q12h) are frequently started at the time of discharge to prevent rebound hypercoagulation associated with heparin withdrawal.

Gastrointestinal protectants, such as pepcid (0.5 mg/kg q24h) or sucralfate, are used by many clinicians in hopes of preventing GI ulceration. At this time there is no evidence to suggest that these medications are effective in preventing ulcers, and in our hospital, they are typically administered only once ulceration is suspected to have occurred. Gastric ulceration should be suspected if melena, vomiting, or reluctance to eat develop, or if serum total protein begins to fall in conjunction with the hematocrit. It is important to recognize the development of GI blood loss, because the resulting drop in hematocrit can otherwise be easily confused with treatment failure.

Dogs with idiopathic IMHA are at risk for recurrence of disease, and care should be taken not to wean the immunosuppressive drugs too quickly. Prednisone is typically maintained within the immunosuppressive range for at least one month following hospital discharge, and then may be decreased by approximately 20-25% each month, provided that the hematocrit remains stable. If azathioprine or other adjunctive agent is being administered in conjunction with the prednisone, it may be discontinued one month after discontinuing prednisone. In total, the weaning process should span at least 4-6 months. Labwork should be rechecked one week after each decrease in drug dosage to make sure that the change is tolerated. If relapse occurs during the weaning process, immunosuppressive dose prednisone should be re instituted, then gradually weaned back to the lowest effective dose. Following weaning, it is frequently recommended that vaccines be avoided, though the association between vaccines and IMHA development is still unknown. Splenectomy may be considered for dogs with recurrent or refractory disease.

References


Immune-mediated hemolytic anemia (IMHA) is one of the most common hematologic disorders seen in dogs. Hemolysis results from the binding of immunoglobulins to red blood cell surface antigens, causing cell lysis by complement intravascularly or phagocytosis within the liver or spleen. IMHA carries a guarded prognosis with mortality rates that have ranged between 29% and 77%.1-8

Thromboembolic disease (TE) is a frequent complication of IMHA and appears to be a major contributor to morbidity and mortality.3,10 In studies of dogs with IMHA that underwent necropsy, TE was identified in 60-80% of cases.8-17 Sites most commonly affected were the pulmonary, portal, and splenic vasculature, with many dogs having thrombi in multiple organs.3,9-12 Although exact mechanisms for the prothrombotic state have not been elucidated, increased concentrations of procoagulant factors such as tissue factor and fibrinogen, decreased concentrations of antithrombin and fibrinolytic factors, vasculitis, enhanced platelet reactivity, the presence of antiphospholipid antibodies, liberation of RBC stroma, circulating microparticles, blood transfusion, and administration of steroids have all been hypothesized to play a role in the development of TE.9-15

The antemortem prevalence of thromboembolic disease in dogs with IMHA has not been well described due to difficulties involved in making an accurate diagnosis. However, preliminary data from 110 dogs treated for IMHA at the Michigan State University Veterinary Teaching Hospital between 2004 and 2007 suggested that 34% had a clinical diagnosis of TE during their hospital stay.16 Clinical diagnosis of pulmonary thromboembolism (PTE) was made on the basis of hypoxemia, thoracic radiographs excluding other respiratory diseases, and laboratory evidence of a prothrombotic state. Clinical diagnosis of portal vein thrombosis (PVT) was based upon the presence of ascites, vomiting or diarrhea, ultrasonographic findings consistent with altered portal blood flow or visualized thrombus, and laboratory evidence of a prothrombotic state. Of these dogs with a clinical diagnosis of TE, 51% had pulmonary thromboembolism (PTE) alone, 8% had portal venous thrombosis (PVT) alone, and 41% had both PTE and PVT.

The development of TE appears to significantly contribute to the morbidity and mortality of IMHA.5,9,10,12 In our data, survival to discharge in dogs with TE was significantly lower than in dogs without TE (49% vs 81%) and median duration of hospitalization was longer (7 days vs 4 days).16 However, 5 of 5 dogs with suspected PTE and confirmed PVT (by ultrasound or CT angiography) that were treated with thrombolytic therapy and thromboprophylaxis with warfarin survived > 1 year from the time of diagnosis. This suggests that accurate and prompt identification and treatment of TE may result in improved survival in this compromised patient population.

Definitive diagnosis of PTE remains challenging. Radiographic changes suggestive of PTE may include interstitial or alveolar infiltrates, small volume pleural effusion, regional oligemia resulting from reduced pulmonary blood flow distal to the thrombus, wedge-shaped pulmonary opacities, and enlarged or truncated pulmonary arteries. Unfortunately, radiographic changes associated with PTE in dogs are neither sensitive nor specific and may be absent in some cases.12,17 Ventilation-perfusion (V-Q) scintigraphy has been evaluated in experimental PTE in dogs and was reported to be helpful in supporting a diagnosis of PTE in one dog with IMHA.10 However, V-Q scanning is not widely available and the need for a 24 hour isolation period at a nuclear medicine holding facility makes this technique unfeasible in animals requiring oxygen therapy and critical care monitoring. In human medicine, CT angiography (CTA) is considered the test of choice for diagnosing pulmonary embolism. Recently, we developed pulmonary and portal angiographic techniques using a 16 slice multidetector CT unit that have been successfully used to detect PTE and/or PVT in dogs with IMHA.

Documentation of the pro-thrombotic state that is responsible for the TE disease is also challenging, and has traditionally been based upon detection of increased fibrinolysis (increased FDPs and D-dimers) and decreased endogenous anticoagulants (antithrombin) rather than rate of clot formation. Thromboelastography (TEG) has shown promise in demonstrating hypercoagulability in dogs with IMHA.18,19 Recently, our group has documented hypercoagulability as assessed by TEG in this population and demonstrated resolution of the prothrombotic state once hemolysis has ceased. (unpublished data)

Because of the close association between TE and mortality in dogs with IMHA, thromboprophylaxis is commonly instituted. Low-dose aspirin, clopidogrel (Plavix), and parenteral unfractionated heparin are the drugs most frequently used in veterinary patients at this time. Despite the frequent use of these drugs in dogs with IMHA, no large scale prospective randomized clinical studies exist and the use of various drug and dosing regimens remains controversial.

Aspirin irreversibly inhibits the formation of thromboxane A2 thus inhibiting platelet aggregation. It has traditionally been used in disease states associated with arterial thromboembolic disease such as heart disease, as the composition of arterial thrombi tend to be more “platelet-rich” than venous thrombi. However, several human studies have reported a decrease in the occurrence of TE in patients at high risk for venous TE when they have been prescribed low dose aspirin in addition to other antithrombotic drugs. Additionally, inhibition of platelet aggregation with aspirin appeared to be beneficial in one retrospective study evaluating treatment protocols in 151 dogs with IMHA.7 The optimal aspirin dose in dogs with IMHA has not been determined. However, a comparison of
aspirin doses in normal dogs showed that 0.5 mg/kg q12 hours was more effective than 0.5 mg/kg q24 hours or 10 mg/kg q24 hours at inhibiting platelet aggregation.20

Clopidogrel inhibits ADP receptors P2Y12 on the platelet membrane, offering a different mechanism for platelet inhibition than does aspirin. A daily dose of 1 mg/kg was shown to effectively inhibit platelet aggregation in normal dogs.21 Clopidogrel was also evaluated in a small prospective study and resulted in similar short-term survival rates when compared to low dose aspirin.22 Whether clopidogrel offers additional benefits over aspirin in dogs with IMHA remains to be determined.

Heparin inhibits secondary hemostasis through activation of antithrombin (AT) and subsequent inhibition of the proteases (factors II, IX, X, XI, XII) necessary for the formation of a clot. Because fibrin-rich pulmonary and venous thrombi are most common in IMHA, drugs like heparin that target coagulation would appear to be the most logical choice for clot prevention. However, unfractionated heparin was not shown to be beneficial in retrospective studies23,24 although the dose administered (75-125 U/kg subcutaneously every six to eight hours) in these studies was lower than that shown to prolong activated partial thromboplastin time (aPTT) in healthy dogs.25 In dogs with IMHA, heparin doses of 300 U/kg every 6 hours were insufficient to achieve therapeutic anti-Xa activity (>0.35 u/ml) in a majority of clinical cases, suggesting that significantly higher doses may be required in this patient population.24 It is also clear that titration of heparin to a therapeutic endpoint is most appropriate due to variations in individual response to heparinization. Dogs with IMHA that had their heparin doses individually adjusted based upon anti-Xa activity demonstrated significantly longer survival times when compared with dogs on fixed dose heparin.25 However, the optimal test for monitoring heparinization and the appropriate therapeutic endpoints that should be employed are not well established in dogs. Anti-Xa activity appears to be a likely candidate, but is not widely available at most institutions and therapeutic endpoints are currently extrapolated from human patients. Activated partial thromboplastin time (aPTT) is readily available, but has shown questionable correlation with anti-Xa activity in dogs. Further studies comparing therapeutic endpoints and outcome in clinical patients are necessary.

The current protocol at our institution is a 150 U/kg intravenous bolus of unfractionated heparin followed by 30-60 U/kg/hr constant rate intravenous infusion. The heparin dose is then adjusted daily in 10 U/kg/hr increments, to achieve target prolongation of aPTT (1.5-2.5x upper limit of reference interval). Aspirin (0.5 mg/kg q12h) is added in the event of failure to achieve a target aPTT by day 2. In a pilot study, 26 consecutive dogs with IMHA were admitted to the hospital and treated with this heparin protocol before being transitioned to oral low-dose aspirin before discharge. In this population, no significant TE or bleeding complications were reported and 60 day survival was 100%. (unpublished data)

Conclusion

Despite the frequency with which TE is suspected in dogs with IMHA, definitive diagnosis is rare. Consequently, effective preventative and therapeutic options may be withheld due to concerns about side effects such as bleeding. However, current evidence suggests that thromboprophylaxis is an important consideration in the management of dogs with IMHA. Further studies are required to better define the optimal drugs, dosages, and monitoring strategies in this patient population.

References


Trauma is one of the most common emergencies seen in the busy emergency room. Examples of common veterinary trauma presentations include motor vehicle accidents (i.e. hit by car) interaction with other animals, interaction with humans, fall from heights, and penetrating trauma such as gunshot wounds, knife wounds, and impalement by sticks.

Trauma may affect only one body system or it may affect multiple organ systems. For this reason, the initial approach to the trauma patient must be rapid, thorough, and detailed to decrease further morbidity and mortality.

The initial triage evaluation should be rapid, developing a problem list outlining life-threatening conditions. The goals of the initial triage examination are to:

1. Assess / evaluate the ABCD’s of triage medicine:
   a. Airway: Does the patient have a patent airway? Upper airway or lower airway abnormalities?
   b. Breathing: Does the patient have an abnormal breathing pattern? Is the patient dyspneic? Is there a rapid, shallow breathing pattern? Is there a slow, labored breathing pattern? Is there increased stertor or stridor?
   c. Circulation: Is there an abnormal heart rate? Are the mucous membranes an abnormal color with evidence of internal or external hemorrhage? Are the pulses weak? Are the extremities cold?
   d. Disability: Is there evidence of head trauma or other neurological injury?

2. Specifically regarding thoracic trauma, the goal is to rapidly determine if there are respiratory abnormalities. If present, the goal is to localize the cause for respiratory distress to best provide treatment:
   a. Inspiratory wheezes: associated with narrowing of the upper airways by inflammation, hemorrhage, mucosal edema, or mucus.
   b. Expiratory wheezes: associated with narrowing of the lower airways by inflammation, hemorrhage, mucosal edema, or mucus.
   c. Crackles: fluid present within the lower airways / alveoli (e.g. edema, hemorrhage)
   d. Stridor or stertor: indicates an upper airway respiratory abnormality
   e. Short / shallow pattern: may indicate pleural space disease such as pneumothorax, pleural effusion, or diaphragmatic hernia
   f. Paradoxical breathing: recognized by a lack of synchronous movement of the chest and abdominal walls.

Initial therapy chosen will be based on the degree and location of injury. Common therapies include oxygen supplementation, intravenous fluid therapy, and analgesia. Procedures such as a thoracocentesis may also be required, which can be both diagnostic and therapeutic.

Oxygen supplementation is one of the mainstays of therapy for a patient with respiratory difficulty. Initially, oxygen is often provided by facemask or flow-by to permit the clinician to perform the initial assessment. While oxygen cages may allow a higher percentage of oxygen to be delivered, it is difficult to assess the patient once in the closed oxygen cage, and therefore placement into the oxygen cage is often delayed until after initial assessment has been performed.

### Oxygen supplementation techniques.

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

Nasotracheal catheter | 25-50 ml/kg/min | 80-90%

Intravenous access for fluid therapy and drug administration is also important. Initially, intravenous access is preferred via the use of by peripheral veins, notably the cephalic or saphenous veins. Other sites such as the jugular vein, while available, are not preferred as placement is not only more technically challenging but requires increased restraint which can be distressing to the dyspneic patient. Moreover, the use of the jugular vein should be avoided if there is a concern for head trauma and increased intracranial pressure. If patient stability allows, when placing the catheter it is advised to pull blood for anticipated testing including a minimum database (packed cell volume (PCV), total protein (TP), Azostick / dipstick BUN, and blood glucose concentration). A complete blood count, chemistry panel, and coagulation panel can also be drawn at that time if patient stability allows.

Once intravenous access is obtained, fluid therapy for resuscitation can be initiated. The goal of fluid resuscitation is to restore tissue perfusion and oxygenation. The type, volume, and rate of fluid administration are determined based on the patient assessment and underlying injuries. The two most common fluid choices for the resuscitation phase are isotonic crystalloids and synthetic colloids. Examples of isotonic crystalloid replacement fluids are 0.9% saline, lactated Ringer's solution, Normosol-R or Plasmalyte-A. Typically smaller doses of fluids are administered (10-30ml/kg in the dog, 5-10ml/kg in the cat) with frequent re-assessment rather than large volumes at once with the risk of worsening respiratory distress. Colloids are larger molecular weight fluids considered intravascular volume expanders. Examples of synthetic colloids include Hetastarch and Vetstarch. Typically smaller doses of fluids are administered (2-5ml/kg in the dog, 1-3ml/kg in the cat) with frequent re-assessment rather than large volumes at once with the risk of worsening respiratory distress. Once initial evaluation, treatment, and stabilization have started, the clinician can further evaluate the patient with a more thorough general examination to assess other complications of thoracic trauma.

Airway trauma
Trauma to the major airways may be seen with penetrating wounds or blunt trauma to the neck and chest. Clinical signs of upper airway trauma include abnormal upper airway noise on inspiration and expiration. Respiratory changes may result from traumatic inflammation, edema, hemorrhage, or even tracheal rupture or avulsion.

Subcutaneous emphysema may also be noted on examination, prompting a thorough airway integrity assessment. Pneumomediastinum and pneumothorax are more severe complications of airway trauma. While subcutaneous emphysema and pneumothorax may be easily found on examination alone, the diagnosis of pneumomediastinum is made radiographically by increased contrast with the mediastinal structures resulting in a clear visualization of the thoracic vena cava, aorta and esophagus.

Pneumothorax
Pneumothorax is defined as the abnormal accumulation of air in the pleural space. Air accumulation is most commonly bilateral but unilateral pneumothorax can occur. It is the most common complication of blunt trauma to the chest. Studies have shown that animals hit by car with fractures had evidence of pneumothorax 47.1% of the time. Furthermore, 36% of dogs and 63% of cats that fell from high rises that had evidence of pneumothorax on examination. Pneumothorax can be further classified as closed, open, and tension pneumothorax.

- Closed pneumothorax is seen following trauma due increased intra-thoracic pressure against a closed glottis causing rupture of alveoli or small airways, laceration of lung by fractured rib, iatrogenic, and airway or esophageal rupture causing pneumomediastinum which has progressed to pneumothorax.
- Open pneumothorax may result from gunshots, dog bites, knife wounds, and stick impalement.
- Tension pneumothorax is the third type, resulting when an air leak acts as one-way valve increasing intrathoracic pressure, compressing the lungs and decreasing venous return to the heart.

The astute clinician often makes the diagnosis of a pneumothorax based on history and examination alone. Common examination abnormalities include an increased respiratory rate and effort characterized by a short and shallow breathing pattern, dull lung sounds dorsally, and muffled heart sounds. Less specific examination abnormalities may include pale or cyanotic mucous membranes, poor pulses, and an abnormal posture with the head and neck extended and elbows abducted. While useful in the diagnosis of a pneumothorax, thoracic radiographs risk increased stress on the compromised patient. Radiographic signs of pneumothorax include elevation of the cardiac silhouette from the sternum, collapse of the lung lobes, and absence of vascular markings out to the periphery of the thorax.

Recently, the use of ultrasound has been documented for rapid detection of pleural space disease, specifically the "TFAST" (thoracic focused assessment with sonography for trauma) procedure. It does, however require practice to be competent in its use.
When radiographs are not suitable, the unstable patient may benefit from thoracocentesis, which can be both diagnostic and therapeutic. The equipment needed for this procedure includes clippers, scrub, sterile gloves, a 10-60ml syringe, 3-way stop-cock, butterfly catheter or needle and extension tubing. The site preparation and eventual needle placement for a patient suspected of a pneumothorax is on the dorsal 1/3 of the thorax between the 7th-10th intercostal spaces. The needle is inserted cranial to the rib to avoid the intercostal artery, vein, and nerve located caudal to each rib. Air is aspirated until negative pressure is obtained.

A chest tube is indicated when thoracocentesis needs to be repeated multiple times over a short period of time or when the clinician cannot achieve negative pressure on simple thoracocentesis. Large bore chest tubes require sedation or general anesthesia. Smaller bore chest tubes are also available, placed via the modified seldinger technique with the patient awake or receiving local analgesia. Equipment required for chest tube placement includes clippers, surgical scrub, surgical blade, local analgesia, suture material, the thoracostomy tube, 3-way stop-cock and syringes for initial aspiration. The chest tube can be used intermittently or attached to a suction device for continuous suction. The technique for chest tube placement will depend on the type of tube used, including surgical and trocar methods for the larger bore tubes or the modified seldinger technique for the smaller bore tubes. Similar to the thoracocentesis, surgical preparation of the site between the 7th-10th intercostal spaces is recommended.

### Pulmonary contusions

Pulmonary contusions result from blunt or crushing trauma and are one of the most common problems associated with thoracic trauma, seen in approximately 50% of all thoracic injuries. Thoracic trauma leads to blood within the alveoli, ventilation/perfusion mismatch, increased pulmonary shunt fraction, and loss of lung compliance. Hypoxemia, increased work of breathing, and hypercarbia, are the physiologic results.

Physical examination findings may include tachypnea, hemoptysis, increased respiratory effort, and harsh lung sounds or crackles on auscultation. Radiographically, there may not be evidence of pulmonary contusions on presentation, delayed anywhere from 12 to 48 hours following trauma. When present, contusions appear radiographically as dense patchy, interstitial to alveolar disease.

As discussed above, initial fluid resuscitation must be started with caution as large volumes of rapidly administered fluid can exacerbate the fluid within the alveolar space with increased vascular permeability, worsening the hypoxemia. If radiographs have evidence of pulmonary contusions, the astute clinician should carefully look for concurrent abnormalities including pneumothorax and/or rib fractures. Additional diagnostic findings may include hypoxemia on pulse oximetry or arterial blood and an increased A-a gradient.

There is no specific medication or reversal therapy for pulmonary contusions. Common supportive care measures include oxygen supplementation, judicious IV fluid therapy, and analgesics. Although evidence is lacking, low dose diuretic therapy has been described anecdotally (furosemide, 0.5 to 1 mg/kg IV intermittently or CRI) in the treatment of pulmonary contusions.

### Fractured ribs

Rib fractures result in discomfort and reduced diaphragmatic and chest wall motion. More specifically, the reduced chest wall motion and pulmonary expansion results in decreased oxygenation, ventilation, and atelectasis of the lungs. Rib fractures should be a clue to the astute clinician that severe thoracic trauma occurred prompting careful evaluation for additional injuries such as pulmonary contusions or a pneumothorax. Physical examination findings may include an increased respiratory rate with shallow respirations, subcutaneous emphysema, palpation of crepitus over the fracture site, and/or conformational changes of the chest wall.

Treatment of rib fractures consists of treating concurrent injuries such as pulmonary contusions, oxygen therapy if hypoxemia exists, and pain management with local or systemic analgesia.

### Flail chest

A flail chest is a more severe manifestation of the simple rib fracture. A flail segment occurs when 2 or more ribs are fractured at the junction of ribs and the sternum producing a paradoxical movement of the flail segment. On inspiration, the chest wall normally expands. With a flail segment, the negative intrapleural pressure causes the flail segment to collapse inward during inspiration. On expiration, the chest wall normally collapses. With a flail segment, the intrapleural pressure becomes less negative and the flail segment moves outward on expiration. Abnormal chest movement and the accompanying pain from the fractures themselves result in decreased oxygenation, ventilation, and pulmonary atelectasis.

Treatment consists of placing the patient in lateral recumbency with the flail side down, minimizing movement of the flail segment and reducing the associated fracture discomfort. Pain management includes local nerve blocks and systemic opioid analgesia. Surgical stabilization of the flail segment may also be indicated.

### Hemothorax

A hemothorax is defined as an accumulation of blood in the pleural space. This is uncommon following trauma. If present, the amount of blood loss into the pleural space is usually minimal and does not contribute significantly to respiratory compromise. If a large amount of hemorrhage into the pleural space is documented, there should be an increased suspicion for rupture of a large vessel.
More common causes for a hemodynamically insignificant hemothorax include laceration of pulmonary or intercostal vessels and/or lung laceration by a fractured rib.

The diagnosis of hemothorax is often be made on physical examination with signs including dyspnea, tachypnea, dull lung sounds ventrally, muffled heart sounds, and signs of hypovolemic or hemorrhagic shock. Thoracentesis confirms the diagnosis when hemorrhagic fluid is obtained during aspiration with a PCV and TP of the effusion similar to that of the PCV and TP of the peripheral blood.

Treatment of a traumatic hemothorax may include diagnostic and therapeutic thoracentesis, intravenous crystalloid or synthetic colloid therapy and blood products, notably whole blood or packed red blood cell transfusions. Autotransfusion can be considered if blood products are not available.

**Diaphragmatic hernia**

Diaphragmatic hernia is defined as disruption of the diaphragm, allowing displacement of abdominal organs into the thoracic cavity. Diaphragmatic hernia occurs most often as a result of blunt trauma where intra-abdominal pressure is suddenly increased causing rupture of the diaphragm. The resulting herniation of abdominal contents can range from a single organ or component of an organ (such as a single liver lobe) to almost all the abdominal contents moving cranially through the diaphragmatic rent into the chest cavity. The result is restriction of lung expansion and respiratory distress.

The diagnosis of diaphragmatic hernia can be made with physical examination findings and radiographic abnormalities. Clinical signs of diaphragmatic hernia depend upon the type and number of organs within the chest cavity as well as associated abnormalities such as fluid in the pleural space or pulmonary contusions. Examination findings may be mild and include a slight tachypnea or may result in severe dyspnea, dull lung sounds, muffled heart sounds, borborygmi from the stomach or intestines ausculted in the thorax, abnormal percussion, and a tucked/empty abdomen on palpation. Thoracic radiographs are often diagnostic with the presence of abdominal organs in the thorax.

Treatment for diaphragmatic herniation will depend on the clinical signs of the patient with surgical repair being the definitive therapy. Although there are no recent studies which outline the recommended time from stabilization to surgical correction, worsening respiratory distress or compromised blood supply to the displaced organs and ischemia would warrant a more rapid surgical correction.

**Summary**

Thoracic trauma is common in small animal medicine. Most patients respond well to rapid and aggressive support therapy. Concurrent injuries are common and the clinician should carefully evaluate their patients to address each specific medical condition to reduce patient morbidity and mortality.

**References**


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

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

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

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

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

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

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

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

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

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

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

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

Normal is approximately 17–24 mEq/L.

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

\[
\text{Osmolality} = 2[\text{sodium (mEq/L)} + \text{potassium (mEq/L)}] + \text{BUN (mg/dl)/2.8} + \text{glucose (mg/dl)/18}. \text{Normal osmolality is approximately 290–310 mOsm/kg.}
\]

Additional diagnostics performed in DKA patients to help formulate the most appropriate treatment plan include:
- Complete blood count
- Chemistry profile
- Serum electrolyte profile
- Urinalysis
- Urine Culture and Sensitivity
- Thoracic radiographs
- Abdominal radiographs
- Abdominal ultrasound
- Coagulation profile

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

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

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

Electrolyte abnormalities are common, notably abnormalities of potassium, phosphorus, and magnesium. DKA patients often have a total body depletion of potassium as a result of decreased intake (anorexia) and increased losses through the gastrointestinal tract (vomiting and diarrhea), and osmotic diuresis. Although a total body potassium depletion is present, initial bloodwork often shows a normal or low-normal potassium level due to shifting of potassium. To maintain electroneutrality to balance the concurrent acidosis, potassium is shifted from the intracellular space to the extracellular space and hydrogen ions are shifted from the extracellular space into the intracellular space, giving a false sense of a normal potassium level although a total body depletion exists. With treatment, notably fluid and insulin therapy, potassium and glucose are shifted back intracellularly resulting in hypokalemia. The hypokalemia, which can be significant, can result in muscle weakness, cervical ventroflexion, cardiac arrhythmias, and respiratory muscle failure.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Electrolyte supplementation

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

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

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

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

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

$$\text{Base deficit} \times \text{body weight (kg)} \times 0.3 \text{ (ECF volume).}$$

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

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

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

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

References

| 2.5–2.9 | 40 |
| 2.0–2.4 | 60 |
| <2.0 | 80 |
Emergency Management of Hepatic Lipidosis
Garret Pachtinger, DVM, DACVECC
Veterinary Specialty and Emergency Center
Levittown, PA

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

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

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

**Pathogenesis**

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

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

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

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

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

**Presenting complaint**

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

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

**Clinical signs- examination**

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

**Bloodwork**

**Complete blood count**

Complete blood count (CBC) findings often include a nonregenerative anemia and a stress leukogram. The nonregenerative anemia may result from chronic disease, phlebotomy, or hypophosphatemia. Stress leukograms have a characteristic white blood cell count differential including a mature neutrophilia, lymphopenia, and eosinopenia. A monocytosis is variable in cats. Neutrophilia is due to decreased adherence to the vascular endothelium, which prolongs circulating time and increased bone marrow release of neutrophils. Lymphopenia is due to redistribution or lysis of lymphocytes. Poikilocytosis is common and may reflect altered red blood cell membrane lipids or oxidative stress to the red blood cells affecting cell membrane stability and flexibility. Heinz bodies are also common, and may reflect oxidation as a result of medications, inflammation, or the underlying disease\(^5\).
Serum biochemistry
Serum biochemical changes primarily reflect cholestasis. Cholestasis, is a term used to describe any condition in which there is impaired flow of bile from the bile duct preventing bile from entering into the intestines. Cholestasis may result from a variety of diseases related to the gallbladder, liver, and pancreas.

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

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

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

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

Coagulation profile
Evaluation of Prothrombin time (PT) and Partial Thromboplastin Time (PTT) is an essential part of the diagnostic evaluation in feline hepatic lipidosis. In less than 7 days cats can become vitamin K deficient and over 50% of cats with hepatic lipidosis have coagulation test abnormalities. Coagulation profile abnormalities are not uncommon as the liver plays a primary role in clotting factor synthesis, notably the Vitamin K dependent clotting factors II, VII, IX and X, Protein C and Protein S.10 The importance of checking clotting factors and treating coagulopathies cannot be over emphasized in patients that may require the placement of large bore feeding tubes, liver aspirates or biopsies, or jugular venipuncture.

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

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

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

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

In health, the liver is isoechoic to the falciform fat and to the cortex of the right kidney, and hypoechoic to the spleen. With hepatic lipidosis, the liver is characteristically large (hepatomegaly) with diffuse hyperechoic parenchyma, hyperechoic to the falciform fat and renal cortex, and isoechoic to the spleen.6

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

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

**Therapy**

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

**Enteral feeding**

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

1. RER = 70 x (current bodyweight in kilograms)^0.75 (for > 5 kg)
2. RER = 30 x BWkg + 70 (for < 5 kg)
3. RER = 60kcal x BWkg

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

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

- 5kg x 60kcal/kg = 300kcal over a 24 hour period.
- Day 1 RER = 300kcal * 25% = 75kcal total, or approximately 19kcal every 6 hours.
- Day 2 RER = 300kcal * 50% = 150kcal total, or approximately 38kcal every 6 hours.
- Day 1 RER = 300kcal * 100% = 300kcal total, or approximately 75kcal every 6 hours.

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

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

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

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

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

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

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

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

Fluid and electrolyte therapy is essential for rehydration, maintenance, and correction of electrolyte abnormalities primarily resulting from a lack of nutritional intake. A balanced electrolyte solution is recommended. Due to decreased hepatic lactate metabolism, hyperlactatemia may already be present. For this reason, some clinicians avoid lactate-containing solutions such as

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Lactated Ringer's solution (LRS). With that said, more often we are concerned about improving overall hydration and intravascular volume replacement, and the use of one specific crystalloid as compared to another is more academic than clinical. Dextrose supplementation is also avoided unless hypoglycemia is documented, as many patients are already showing some degree of hyperglycemia as a result of glucose intolerance.

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

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

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

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

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

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

Supplementation with L-carnitine has demonstrated improved fat metabolism and clinical survival. L-carnitine transports long chain fatty acids across the mitochondrial membrane for Beta oxidation, and is an essential cofactor for fatty acid oxidation.

S-adenosyl-L-methionine (SAMe), an essential methyl donor and important for glutathione (GSH) synthesis may also aid in hepatic recovery.

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

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

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

<table>
<thead>
<tr>
<th>Pet Food Brand</th>
<th>Caloric Density</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hill’s A/D TM</td>
<td>1.2 kcal/ml</td>
</tr>
<tr>
<td>Rebound TM</td>
<td>1 kcal/ml</td>
</tr>
<tr>
<td>Clinicare TM</td>
<td>1 kcal/ml</td>
</tr>
<tr>
<td>Royal Canin/MediCal Recovery TM</td>
<td>1.23 kcal/ml</td>
</tr>
<tr>
<td>Eukanuba Maximum Calorie TM</td>
<td>2.1 kcal/ml</td>
</tr>
</tbody>
</table>
## Drugs used for vomiting

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

## Drugs used for appetite stimulation

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

## Drugs used for hepatic support

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

## Drugs used for electrolyte support

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

Hemoabdomen is defined as free blood in the peritoneal or retroperitoneal space. It is most commonly categorized into nontraumatic and traumatic causes with non-traumatic causes being further categorized into coagulopathic and non coagulopathic (spontaneous). Patients can present with internal hemorrhage that is mild and self-limiting. Patients can also present with rapid and severe hemorrhage, which is ultimately fatal without rapid intervention. It is up to the clinician to perform a rapid assessment and provide emergency treatment to reduce further morbidity and mortality.

Signalment & history
Breed, age, and history can be extremely helpful when evaluating a patient with hemoabdomen. Trauma is often a presenting complaint, offered as information by the owner and part of the immediate triage history directing further patient assessment and treatment. If the history is unknown, clinical examination findings (see below) can provide some important clues regarding the possibility of trauma. If there is no history or evidence of trauma, signalment can help form a differential diagnosis and treatment plan. For example a spontaneous hemoabdomen in a 2 year old dog is more likely from rodenticide exposure whereas a 14 year old large breed dog with a spontaneous hemoabdomen is more likely to have a neoplastic cause.

Physical examination
Most animals presenting with a hemoabdomen will have historical clues lethargy, collapse, exercise intolerance, and weakness. Physical examination abnormalities include pale mucous membranes, prolonged capillary refill time, snappy (short and narrow) femoral pulses, tachycardia, and tachypnea. Evidence of traumatic causes of hemoabdomen may include bruising, abrasions, lacerations, fractures, and/or road rash. Whether the bleeding is traumatic or non traumatic, the abdominal cavity is the most common place for clinically significant internal hemorrhage. Dependent on the amount and the speed of blood loss signs may range from mild anemia to hemorrhagic shock. Surface bleeding of the skin and mucosa such as petechia, ecchymoses, epistaxis, gingival bleeding, melena, hematochezia, and/or hematuria are more likely to be seen with a primary hemostatic disorder (thrombocytopenia or thrombocytopathia) and are less common with coagulation defects that cause cavity bleeding.

Diagnostic testing
A minimum database of a bleeding patient includes a packed cell volume (PCV), total protein (TP), blood urea nitrogen (BUN) and blood glucose (BG). Further emergency database information includes blood gas analysis, lactate, and electrolytes. Blood pressure and ECG should also be obtained. Common Findings consistent with hemoabdomen include decreased PCV, decreased TP, and increased lactate. Additionally, hypotension (low blood pressure) and a sinus tachycardia (on ECG) are common. A blood smear is useful to provide a platelet estimate, to evaluate RBC morphology, and to perform a differential blood count. Each platelet per high power oil emersion field represents approximately 15–20,000 platelets/µl blood. The feathered edge of the slide should be carefully evaluated as white blood cells and platelet clumping may be found there, notably platelet clumping which can explain a lower than expected platelet count in the monolayer when attempting to calculate an estimated platelet count.

Imaging studies can also be a valuable diagnostic tool for patients presented with hemoabdomen. Radiographs may show decreased serosal detail, organ enlargement, abdominal masses, diaphragmatic and/or body wall hernia. Decreased serosal detail may indicate free peritoneal fluid. Alternatively, many are now using ultrasound as a more detailed diagnostic tool. Specifically, ultrasound is used in combination with the FAST (focused assessment sonography trauma) protocol. The FAST protocol is the quickest and most sensitive way to detect a hemoabdomen. If ultrasound is not readily available, a four quadrant abdominocentesis can be performed to obtain free abdominal fluid. Obtaining non-clotting hemorrhagic fluid via this technique supports a diagnosis of free abdominal fluid unless a coagulopathy is present. If grossly hemorrhagic, then PCV and TP of the fluid should be evaluated. Acute hemorrhage tends to have PCV and TP that is similar to peripheral blood. A cytological evaluation should be performed on the fluid to assess for inflammation, bacteria or neoplastic cells.

Finding hemorrhagic fluid in the abdominal cavity confirms the diagnosis of hemoabdomen. Other diagnostics on the effusion that can be considered depending on the clinical presentation includes:

- Measurement of potassium and creatinine if urinary bladder rupture is suspected.
- Measurement of bilirubin if gall bladder rupture is suspected.
- Let’s discuss more the general categories of hemoabdomen:
Coagulopathic hemoabdomen

Hemorrhage as a result of coagulopathy is most commonly caused by disorders of the secondary hemostatic system. Disorders of the primary hemostatic system (platelets) less commonly cause cavity bleeding.

One of the most common coagulopathic causes of hemoabdomen is toxicity, specifically vitamin K deficiency due to anticoagulant rodenticide poisoning. While this can happen at any age, it is the most common cause for spontaneous (non traumatic) hemoabdomen in young patients. If anticoagulant rodenticide toxicosis is suspected, the goals are to prevent further hemorrhage and reverse coagulopathy by administration of vitamin K1. Treatment for the coagulopathic patient may also include transfusion medicine including whole blood, packed red blood cells, and/or fresh (frozen) plasma.

Traumatic hemoabdomen

Treatment of the patient that presents with a hemoabdomen as a result of trauma will depend on the severity of bleeding, resulting anemia, and concurrent injuries. Regarding traumatic causes of hemoabdomen, ultimately, there is a lack of evidence to support immediate surgery versus medical therapy. In the author’s opinion, most traumatic hemoabdomen cases can be managed with nonsurgical measures. If stabilization fails, the clinician should be prepared to perform surgery. While surgical intervention can often be avoided, these patients may require immediate and intensive care including intravenous fluid therapy and blood transfusions.

If hypovolemia is present, intravenous fluid resuscitation is warranted. Choices for fluid therapy include isotonic crystalloid therapy, hypertonic crystalloid therapy, or synthetic colloid therapy.

- Isotonic crystalloid 10-30 ml/kg IV bolus
- Synthetic colloid 2-5 ml/kg IV bolus
- Hypertonic saline (7.5%) 2-4 ml/kg IV

Regardless of the fluid choice, careful monitoring is warranted due to the risk of abrupt increases in systemic blood pressure and the concern for increased hemorrhage. With severe acute blood loss, blood transfusions or blood substitutes are indicated. The blood product used (packed RBCs, whole blood) depends on the availability and on the type of the hemostatic disorder.

Specific variables to monitor to help direct further therapy and case management include:

- Blood pressure
- Heart rate
- PCV and TP
- Lactate

A specific resuscitation therapy reported for traumatic conditions such as this is hypotensive resuscitation. This technique employs small volumes of fluid rather than large rapid volumes with the goal of increasing perfusion but tolerating slight hypotension with a Doppler blood pressure of 80-100mmHg. This method has been shown to reduce mortality in human patients with abdominal bleeds after trauma. The theory is that there is less likelihood of disrupting blood clots that are forming, and that bleeding will stop. Additional supportive measures include external abdominal counterpressure, strict cage rest, analgesia, and careful handling.

Measuring intra-abdominal pressure can be done if you have a urinary catheter in place. It is just like measuring central venous pressure and can be done easily with a stopcock and water manometer. Pressures above 25cm H2O are associated with decreased organ perfusion.

Spontaneous hemoabdomen

This category is distinct from other common causes of a hemoabdomen. Obtaining a thorough history and point-of-care diagnostics can quickly decrease the suspicion of a traumatic hemoabdomen or coagulopathy. Often with a traumatic hemoabdomen, the patient presents with a recent history of trauma, such as vehicular trauma. Physical examination findings can also increase the suspicion for an unwitnessed trauma, such as bruising, fractured ribs, or skin abrasions or lacerations. Point of care diagnostics such as a PT clotting test (prothrombin time), can also be very helpful. A PT test that is normal or slightly elevated in the presence of a hemoabdomen would decrease the suspicion of the primary cause being a coagulopathy, as clinical experience would require a PT test to be out of range (or close to out of range) to increase the suspicion of the primary cause being a coagulopathy to result in a hemoabdomen. A slight elevation often can be considered a consumptive coagulopathy.

Once trauma and coagulopathic causes have been ruled out, especially in an older, and often large breed dog (although there are no studies to say smaller breed dogs are any different), the term spontaneous (or non-traumatic, non-coagulopathic) hemoabdomen can be used.

There are several studies that have evaluated the spontaneous (non-traumatic, non-coagulopathic) hemoabdomen. These studies indicate an overwhelming likelihood neoplasia as an underlying cause, most commonly a ruptured splenic hemangiosarcoma (65-85%). Other causes do exist, both benign (ruptured hematoma) and malignant (e.g. mesothelioma, carcinoma, pheochromocytoma, lymphoma), but unfortunately the overwhelming likelihood is that a spontaneous hemoabdomen in an older dog will result from a splenic hemangiosarcoma.
Often these patients present in shock, specifically hypovolemic shock. Physical examination findings may include tachycardia, poor pulses, pale mucous membranes, increased respiratory rate and effort, and a distended abdomen with a palpable fluid wave. As in other causes of hemoabdomen, the first priority should be stabilization (e.g. intravenous catheter placement, fluid therapy, oxygen therapy, etc). Based on the patient's state of illness, fluid therapy options to debate would include isotonic crystalloids, hypertonic saline, colloids, and even blood products.

Following diagnosis and stabilization, as these are often older dogs with a primary concern for a neoplastic process, diagnostics that to considered should include:

- **Bloodwork (CBC and Chemistry Screen)** – to check for cell counts, organ values, electrolytes, and overall assess for metabolic or electrolyte derangements which would need correction
- **Coagulation testing (specifically a prothrombin time – PT)** – this should have been performed in the initial diagnostics on presentation to place the patient in this specific category (non-traumatic, non coagulopathic) – but if not, should be performed pre-operatively.
- **Thoracic X-Rays** – While helpful to assess cardiac size and shape, often the primary reason to recommend thoracic x-rays is to identify pulmonary metastasis. The presence of pulmonary metastasis would worsen the prognosis substantially and likely make this patient a poor candidate for surgery and anesthesia.
- **Abdominal Ultrasound** - My personal experience with an abdominal ultrasound and interpretation for clients falls in 1 of 3 scenarios:
  1. There is a solitary mass (spleen, liver, etc) that can be identified. Often radiologists are reluctant (and refuse) to note their impression of malignancy and while not helpful in differentiating between a benign or malignant tumor for the owners in their decision, a solitary mass present would hopefully lead one to assume this patient is a better surgical candidate in the absence of diffuse disease. The owner must also understand that there is a possibility that microscopic disease exists (not able to be seen on ultrasound) which may be identified during the exploratory procedure.
  2. There are multiple masses present (not just on one organ). While malignancy cannot be confirmed, the presence of multiple masses throughout the abdomen would give the impression that malignancy is more likely and this patient is likely a worse surgical candidate than the previous patient with one solitary mass.
  3. No masses/lesions have been identified. At that time further investigation is warranted (unwitnessed trauma?) and further stabilization may be needed to note progression.

Does every patient need an ultrasound? I have clients that would like to save their pet regardless of the ultrasound findings. Are the ultrasound findings then academic in nature? If the client understands the risk that diffuse disease may be present and identified during surgery, resulting in a phone call to discuss humane euthanasia on the table, wouldn't it then be reasonable to save the $400-$600 on the ultrasound and proceed directly to surgery following stabilization and additional diagnostics? Ultimately, once stabilized to the best of the clinician’s ability, an exploratory laparotomy is needed.

**Hemoabdomen in cats**

Hemoabdomen in cats is relatively rare compared to dogs. In a study that evaluated hemoabdomen in cats, 46% had abdominal neoplasia and 56% had non-neoplastic causes. Hemangiosarcoma was diagnosed in 60% of the cats with neoplasia with the spleen being the most common site. Unfortunately, only 12% of the 65 cats survived to discharge suggesting that the overall prognosis of hemoabdomen in cats is poor.

**Summary**

In theory, surgery is a consideration for every non-coagulopathic hemoabdomen patient, especially in patients that do not stabilize medically. Specifically regarding the traumatic hemoabdomen, the author believes that most patients can be stabilized with medical therapy within two hours of presentation. If appropriate resuscitation efforts are not successful and do not achieve cardiovascular stabilization within 2 hours, it is unlikely further medical therapy will be successful. Traumatic hemoabdomen patients that do not stabilize with aggressive and appropriate medical therapy should be considered surgical candidates. Surgery will not only achieve hemostasis but also provide an underlying diagnosis.

**References**


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

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

Total body water (TBW) fluid compartments

<table>
<thead>
<tr>
<th>ECF (33% TBW)</th>
<th>ICF (66% TBW)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interstitial Fluid</td>
<td>Intracellular Fluid</td>
</tr>
<tr>
<td>Plasma (25% ECF)</td>
<td></td>
</tr>
</tbody>
</table>

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Complications of fluid therapy

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

- Pulmonary edema
  - Volume overload
  - Increased vascular permeability
- Rapid sodium shifts
  - Neurologic signs
  - Obtundation
  - Cerebral edema
  - Seizures
- Phlebitis
  - Use of hyperosmotic agents

Conclusions

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

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

In vitro

### Table: Common crystalloids and their chemical properties.

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

### References


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

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

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

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

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

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

Approximate injection volumes of sterile saline include:

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

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

To perform a transtracheal wash, the ventral neck is clipped and scrubbed, notably between two rings of cartilage 3-4 rings below the larynx. Along with manual restraint, chemical restraint can reduce stress and anxiety during the procedure. A local block combined with an opioid or benzodiazepine is considered for mild sedation. When inserting the sampling catheter, the bevel of the needle should be faced downward. The needle is advanced through the skin on the midline of the neck through two cartilage rings, perpendicular to the trachea into the tracheal lumen. As you enter the trachea, you will feel a pop. Once seated within the tracheal lumen, the needle is advanced 2-3 mm further to ensure appropriate positioning. The sampling catheter is advanced through the needle completely into the tracheal lumen. Once the catheter is completely advanced, the needle is pulled back until it is no longer in contact with the catheter. Once the sample is obtained, the catheter is removed and the tracheal lumen is closed with a clip.
Thoracocentesis

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

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

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

Thoracostomy tube placement

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

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

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

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

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

Pericardiocentesis

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

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

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

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

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

The central venous catheter is placed with the patient in lateral recumbency with the assistance of chemical restraint. Similar to other critically ill patients, this can often be easily accomplished with the use of a local block combined with an opioid or benzodiazepine. The lateral cervical area is clipped and aseptically prepared from the ventral ramus of the mandible caudally to the thoracic inlet and dorsally and ventrally to the respective midlines. Sterile drapes are then placed over the aseptically prepared area. The assistant extends the head and neck with the front legs pulled caudally. If available, a second assistant or the clinician occludes the jugular vein for visualization. Once the site is prepped, the provided 18 - 20 gauge over-the-needle catheter is inserted into the jugular vein. Once seated within the jugular vein, the stylet is removed and the catheter is connected to the extension tubing along with a three-way stopcock. Using a 10-20ml syringe, the fluid is aspirated. A sample of the aspirated fluid is to placed into a red top tube and a lavender top tube for further analysis. Specifically, the red top tube is monitored for clotting. A clot within the red top tube is a concern for trauma to the heart via the catheter and the catheter should be removed from the pericardial space. The amount of fluid obtained will vary but may be as much as 1/2 to 1 liter in a large breed dog. As they are often tachycardic on presentation, the clinician should notice a fairly dramatic decrease in heart rate within a few minutes of successful pericardiocentesis.

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

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

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

Potential risks of central venous catheter placement include hemorrhage, thrombus formation, emboli, and infection.
Intraosseous catheter placement

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

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

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

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

Nasal and nasopharyngeal oxygen catheter placement

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

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

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

Temporary tracheostomy tube placement

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

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

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


Suter PF. Trauma to the thorax and cervical airways. Thoracic Radiology of the Dog and Cat. Switzerland: PF Suter, 1984;130-151.

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


Emergency Management of Cardiac Disease
Garret Pachtiner, DVM, DACVECC
Veterinary Specialty and Emergency Center
Levittown, PA

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

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

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

Oxygen supplementation techniques

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

Once initial patient assessment is made, a more thorough physical examination is essential in the diagnosis and management of emergency cardiac patient.

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

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

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

While thoracic radiographs often confirm the diagnosis of CHF, thoracic ultrasound is an upcoming diagnostic in the ER. Along with the TFAST and AFAST, a new term, “Vet Blue” has recently been discussed. Using these ultrasound techniques, lung pathology is assessed based on the distinction between wet (ultrasound lung rockets (ULRs) vs. dry lung (A-lines with a glide sign). The goal of using this technique is to provide rapid, point-of-care global evaluation of the emergent patient with minimal restraint and risk of decompensation. For the TFAST (Vet Blue) procedure, the patient is placed in either right lateral recumbency and/or sternal recumbency. Dorsal recumbency is not recommended as it has not been validated for VetBlue and it also may increase patient stress.

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

In cases of low output failure (weak pulses, pale membranes, slow CRT, weakness, hypothermia, azotemia), dobutamine is a synthetic beta-adrenergic agonist is considered. This is commonly used in patients with DCM. Dobutamine has a dose range of 2–20 mcg/kg/minute At lower doses, dobutamine improves cardiac contractility with minimal effects on chronotropy or heart rate. At higher doses, however, dobutamine can be pro-arrhythmogenic.

Pimobendan (0.25 mg/kg PO BID) has been used with success in dogs with CHF secondary to DCM and mitral valve insufficiency. Pimobendan is a phosphodiesterase-III inhibitor that sensitizes the myocardium to calcium, and improves inotropic activity in addition to causing arteriolar and venous dilation. In addition to its use as a long-term inodilator in the treatment of dogs with CHF, Pimobendan is also recommended for use in emergency therapy of CHF, as it can have an onset of effects within one hour.

### Cardiac tamponade

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

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

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

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

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

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

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

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

**Life-threatening arrhythmias**

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

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

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

**Summary**

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

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

### References


I recently asked a veterinary student what a minimum database (MDB) for a patient should be…They responded, “Complete Blood Count, Serum Chemistry Panel, Chest X-rays, and Abdominal Ultrasound.” I don’t know about you, but in my day, this “MDB” was not my “MDB!” What ever happened to the PCV (packed cell volume), TP (total protein), Azo (Azo stick®), and BG (blood glucose)? These rapid, cheap, and readily available tests provide clinically useful information in a short period of time. For only a few dollars you even have a chance to make the diagnosis! If not a diagnosis, they help with patient assessment and can guide not only therapy, but determine the best way to proceed with more advanced diagnostics. Especially as the cost of veterinary care increases, using the financial resources of the owner to the best of our ability will ultimately allow for better patient care.

Let’s dive a little deeper into the specific components of the MDB:

**PCV/TP**
There is a current Ford car company commercial that has the punch line, AND is better. Who wants a PCV or TP? Having a PCV without the TP is like having to choose a Bed or Breakfast rather than a Bed and breakfast. Evaluation of the PCV and TP together can help diagnose or fine-tune your differential diagnoses.

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

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

<table>
<thead>
<tr>
<th>Packed cell volume and total protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCV/TP</td>
</tr>
<tr>
<td>↓ PCV/ Normal TP</td>
</tr>
<tr>
<td>↓ PCV/ ↓TP</td>
</tr>
<tr>
<td>Normal PCV/ ↓TP</td>
</tr>
</tbody>
</table>

| Hemoconcentration                  | - Hemolytic anemia | - Blood loss |
| - Anemia of chronic disease        | - GI               |
| - Pure red blood cell aplasia      | - Body cavity (abdominal, thoracic, etc) |
|                                    | - Protein Losing Enteropathy (PLE) |
|                                    | - Protein losing nephropathy (PLN) |
|                                    | - Acute blood loss |
|                                    | - Liver disease / failure |

Before breaking the hematocrit tube for evaluation on the refractometer, make sure to use all of the tools available to you! Evaluation of the color of the serum in the hematocrit tube can also help. A yellow discoloration of the serum within the hematocrit tube can indicate icterus or hemolysis. If the patient has a low packed cell volume with yellow serum, for example your 5-year-old Cocker Spaniel, immune mediated hemolytic anemia would be a concern. If the patient is not anemic, but is a 5 year old obese domestic shorthair cat that has been anorexic for 5 days, hepatic lipidosis would be a concern when there is icteric serum. Other abnormalities when evaluating the hematocrit tube include a buffy coat for a gross assessment of a white blood cell count elevation and lipemia, which can be seen in sick patients with severe hypothyroidism or pancreatitis.

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

<table>
<thead>
<tr>
<th>Hypoglycemia</th>
<th>Euglycemia</th>
<th>Hyperglycemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Insulin overdose</td>
<td>- Normal</td>
<td>- Stress (cat &gt; dog)</td>
</tr>
<tr>
<td>- Hepatic failure</td>
<td>- Regulated diabetic</td>
<td>- Diabetes Mellitus</td>
</tr>
<tr>
<td>- Sepsis</td>
<td></td>
<td>- “Death Glucose”</td>
</tr>
<tr>
<td>- Insulinoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Paraneoplastic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Pediatric hypoglycemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Portosystemic shunt</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Adrenal insufficiency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Toxicity (xylitol)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

### AZO / BUN

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

### Ancillary testing

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

### Learning on the run

In the ever-growing number of hours we work and the ever-growing number of resources that we are supposed to study, memorize, and put to use in practice, having information at your fingertips may be life saving for patients. There are now numerous options we have to carry medical references digitally. These include Amazon Kindle, Google Play, and Apple resources to download textbooks and Apps to your mobile devices.

We all know what books are, but what are “apps”? “Apps” are applications, otherwise known as computer programs built to run on a mobile devices, such as your iOs or Android based device. Many apps are now available to the veterinary professional to enhance your education, patient assessment and patient treatment. While there are numerous apps out there, just to exemplify how these apps can be useful in practice, here are 2 examples:

- A common app for the small animal practitioner is the Target app. This is veterinary specific antibiotic reference published by the North American Compendiums and developed by veterinary clinical pathologist Dr. David Aucoin. This allows the practitioner to evaluate specific, common antibiotics and their likelihood of efficacy based on the species and body system affected.

- Another app many find useful in practice is a fluid therapy app from Abbott Animal Health. This is an app for veterinarians and technicians to assist with small animal crystalloid fluid therapy. Abbott’s app allows the practitioner to develop a fluid therapy following assessment including patient weight, percent dehydration and any ongoing losses.

### Get your head out of the clouds…or should you?

Finally, don’t forget to use the “cloud” to your advantage as a busy clinician. What is the “cloud”? The cloud is a term used to indicate that data is stored on another hard drive, on another server, not your own hard drive.

Especially as laptop hard drives get smaller and smaller, it is becoming impossible to store all the data on your computer. But even better, cloud storage allows the user to access files on multiple devices (e.g. home computer, work computer, Smartphone, tablet, etc), knowing that they are available and backed up.

There are numerous options for cloud storage, many of which have free options, allowing the user to see if they like the service, with the ability to upgrade to larger data plans. Examples include Google Drive, Dropbox, Copy, and many more.

### More specifically, how do I use cloud storage in practice?

#### 1. I have created my own digital medical library

In my searchable Google Drive account, I have a personal library that includes:

- Journal articles
- Lecture notes from veterinary school
• Quick references
  o Hospital phone list
  o Favorite treatment and protocols
  o Reference lab information
  o Personal notes of drugs, devices, therapies, patient treatment options.
  o Consensus Statements and Guidelines

In my Dropbox account I have:
• Conference proceedings and notes
• Pictures of cases, normals, abnormals
• Client Handouts

Using cloud storage I am also able to easily share documents, upload and download documents, and even collaborate on documents, at home, at work, or even at a local coffee shop.

Summary
Ultimately, the condition of your emergency patient can be rapidly assessed with rapid and cost effective bedside diagnostics included in the MDB. Along with the history and physical examination this more objective information can help you maximize additional testing and treatment and make a large difference in your quality of care.

References