Cardiogenic Shock:
Hold the Fluids

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

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

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

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

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

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

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

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

Treatment of CS differs fundamentally from all other forms of shock in which volume expansion and vasopressor therapy are the mainstays of treatment. In fact, volume expansion and vasopressor therapies are often contraindicated for CS patients and their use could lead to worsening of the patient's condition up to and including death. Cardiogenic shock must therefore be managed primarily by improving cardiac performance first and altering vasomotor tone second. Oxygen should be administered at the highest FiO2 possible immediately upon recognition of a patient in shock to maximize arterial oxygen content as much as possible. Animals with confirmed or suspected pulmonary edema should be administered a loop diuretic (furosemide 2 mg/kg IV or IM) and the dose should be repeated every 30-60 minutes until the clinical signs improve. The use of mild sedatives such as butorphanol (0.2 mg/kg IV or IM) may facilitate placement of an intravenous catheter to optimize medication administration.

Positive inotropes should be administered to patients with known or highly suspected systolic dysfunction. Due to the critical state of these patients these medications are almost invariably administered as constant rate IV infusions. Dobutamine can be administered at a starting dose of 2.5 mcg/kg/min and can be titrated up every 5-10 minutes until clinical signs improve or a rate of 20 mcg/kg/min is reached. In some instances it is possible to use pimobendan to improve cardiac contractility. Unfortunately, in the U.S. pimobendan is only available as an oral medication making its use limited. An injectable form of pimobendan is available for use in Europe with a recommended starting dose of 0.15 mg/kg IV.

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

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

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

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

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

Unfortunately, published mortality rates for veterinary patients with CS are lacking but human literature suggests mortality rates of 50-80%. In most cases, CS develops secondary to severe underlying cardiac disease. For this reason the short and long-term prognosis for these patient should be considered guarded to poor. Dogs receiving pacemaker implantation for 3rd degree AV block or surgery for caval syndrome provide two notable exceptions.

CPR Update:
What is RECOVER?

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In 2010 the American College of Veterinary Emergency and Critical Care undertook the task of developing cohesive, veterinary specific recommendations for the performance of cardiopulmonary resuscitation utilizing an evidence-based approach. The result of this effort was the publication of the Reassessment Campaign on Veterinary Resuscitation (RECOVER) in 2011, developed through the participation of over 100 veterinary specialists and are composed of 101 clinical recommendations spanning 6 categories.

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

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

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

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

can improve outcomes but comes with difficulties inherent with thoracotomies. Attempts at open chest CPR should only be attempted if the means for managing a patient post-resuscitation are readily available.

In order to optimize CPR it is necessary that patients be monitored for signs of success or failure. All patients with suspected CPA should be instrumented with ECG. Analysis of ECG may help to rule out CPA or may identify rhythms that are amenable to a specific treatment (i.e. defibrillation). In the ideal setting all patients undergoing CPR will have end-tidal CO_2 monitoring performed. While useful for confirming correct placement of endotracheal tubes within the tracheal lumen in non-CPA animal, E_TCO_2 should not be used as the sole confirmation of endotracheal intubation in CPA animals. Since E_TCO_2 is linearly associated with cardiac output in CPA it can be a useful monitoring tool to gauge effectiveness of chest compressions and can be the earliest indicator of return of spontaneous circulation. When performing chest compressions and maintaining the minute ventilation at a constant level, E_TCO_2 levels can predict the likelihood of success with E_TCO_2 level of less than 15 mmHg and 20 mmHg suggesting worse prognosis in dogs and cats respectively. Once ROSC occurs E_TCO_2 will undergo a rapid and sustained rise as the heart more efficiently delivers CO_2 laden blood from the periphery to the pulmonary circulation. If ROSC is achieved then post-resuscitative monitoring should be tailored to the individual patient's needs.

The care of an arrest patient does not end with ROSC; rather this is when the true care of the patient begins. In the immediate post-resuscitation phase every effort should be made to maintain arterial oxygen content within the normal range. No evidence exists that supra-physiologic oxygen levels are beneficial and theoretical detrimental effects exist. While post-arrest therapeutic hypothermia has become the standard of care in human medicine it is still beyond the capabilities of most veterinary facilities. However, if hypothermia occurs during the course of the arrest then rewarming efforts should not be vigorous and the patient should be allowed to return to normothermia at a gradual rate. Hyperthermia should be avoided if at all possible. There is no evidence in support of the routine use of corticosteroids, hypertonic fluids (mannitol or hypertonic saline), or prophylactic treatment with anti-seizure medications. If patients exhibit signs of intracranial hypertension then hypertonic saline or mannitol can be considered. Finally, referral to a comprehensive care facility with 24-hour capabilities should be considered for ongoing care of the post-arrest patient.

Crotalid Envenomation:
Avoid Being Snake-Bitten by Snakebites

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North America is home to 2 different families of venomous snakes, the elapinae (coral snakes) and the crotalinae (pit vipers). Of the two, the crotalinae are much more clinically significant due to their less reclusive behavior and wide range. In fact, pit-vipers can be found throughout North, Central and South America and include all rattlesnakes, the cottonmouth/water moccasin, and the copperhead. Pit-vipers are characterized by having moveable, hollow fangs through which venom is injected into the victim. Mature pit-vipers are capable of metering the dose of venom that they inject in order to preserve venom for the purpose of obtaining food. Juvenile snakes are incapable of dose adjustment and envenomate in an all-or-nothing fashion.

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

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

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

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

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

The dosing of antivenom is based on the amount of venom injected not on the size of the patient. Typically dogs receive between 1-5 vials of antivenom. Paradoxically, smaller dogs often require higher doses of antivenom than large dogs due to the smaller volume of distribution of the venom. Administration usually begins slowly to allow for monitoring of possible reactions but is ideally completed within 30-60 minutes. Treatment with antivenom is indicated if progression of venom effects is seen including progressive

swelling and pain, coagulopathy or systemic effects such as hypotension or altered mental status. When administered antivenom binds to venom components and can reverse some of the clinical signs while preventing or slowing progression of signs. Ideally, antivenom should be used early following envenomation (i.e. within 4 hours) but may be beneficial up to 24 hours after a bite. Two different types of antivenom are currently available, a whole molecule antibody derived from immunized horses and a partial molecule antibody (Fab) derived from immunized horses or sheep. Both types of antivenom can lead to immune reactions including anaphylactoid dose related reactions and delayed type III hypersensitivity reactions.

Comprehensive management of envenomated patients includes effective analgesia. Liberal administration of opioids should be considered provided the patient's neurological status does not preclude their use. Adjunctive analgesic medications to consider would include ketamine, lidocaine or dexmedetomidine. Limbs should be maintained in a functional position. Areas of necrosis should be cleaned and covered with a sterile bandage and managed as any other open wound would be. The prophylactic use of antibiotics in pit viper envenomation is controversial but both the recent human and veterinary literature suggests that prophylactic antibiotic use is not beneficial. Rather, antibiotics should be reserved for treatment of a known or suspected infection. Administration of steroids is not beneficial and should be avoided. Non-steroidal anti-inflammatory drugs are useful for treating associated inflammation but their use should be restricted to patients that are hemodynamically stable. NSAID medications should not be given to patients with impaired renal function or myoglobinuria. Since the coagulopathy associated with pit viper envenomation is due to the presence of circulating venom molecules, the mainstay of treatment is antivenom administration. Fresh frozen plasma is not expected to be beneficial so long as circulating venom is present and when used should be given after all intended antivenom doses.

Dystocia:
Is it Time?

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Recognition of dystocia is based on an understanding of normal parturition. The average gestational length for the dog is 65 days (57-72) and for the cat is 66 days (56-70). Once the fetuses are full term parturition begins. Normal labor can be divided into 3 stages. Stage 1 includes restlessness, nesting and panting. No detectable uterine contractions occur during stage 1 labor which usually lasts for 6-12 hours but may last as long as 24 hours. Stage 2 begins with strong abdominal contractions and clear discharge and is complete when a fetus is expelled. The first fetus should be expected 2-4 hours after the onset of stage 2 labor with subsequent fetuses being delivered every 30 minutes to 4 hours. This stage of labor may last as long as 12-24 hours in dog. Cats have the unique ability to voluntarily interrupt stage 2 labor if they are feeling distressed and stage 2 may last for up to 48 hours. Stage 3 labor consists of placental expulsion which should occur 5-10 minutes after delivery of a fetus. Generally stage 2 and stage 3 labor alternate between fetuses but it is possible for 2 fetuses to be delivered consecutively followed by 2 placentae, one from each uterine horn.

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

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

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

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

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

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

repeated up to 3 times. If this fails to result in delivery of a fetus the patient should proceed to surgery for caesarean section. Only 1/3 of dogs with dystocia will respond to administration of oxytocin alone.

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

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

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

Following delivery neonatal resuscitation begins. Ideally, each neonate will have a dedicated assistant responsible for its resuscitation. The umbilical cord should be clamped if this has not already been done and the face and mouth should be cleared of placental membranes. The neonates should be rubbed vigorously with a clean, dry towel focusing on the face, thorax, genitals and umbilicus. The mouth and nostrils can be cleared by using a bulb syringe. Neonates should not be swung in an attempt to clear airways as this could lead to significant injury. Neonates should have a respiratory rate between 10-18 brpm. Manual, mouth to snout ventilations can be used and supplemental oxygen should be provided. Aggressive needling of the acupuncture point GV26 (at the philtrum of the nose) with a 25-gauge needle can stimulate ventilation in an apneic neonate. As long as a heartbeat is detectable resuscitation should continue even in the presence of prolonged apnea. In neonates that are responding poorly to resuscitative efforts, reversal of anesthetic/analgesic drugs can be attempted. Naloxone can be administered at 0.1 mg/kg. Dosing can be intravenous, intramuscular, intraosseous or sublingual. Vascular access can be obtained at the jugular vein or the umbilical vein. The administration of doxapram is not recommended due to the risk of cerebral acidosis in an apneic patient. Once resuscitation is complete and the neonates are vocalizing and vigorous, they should be evaluated for the presence of cleft palate, atresia ani and umbilical hernia prior to being placed in a warm environment. The dam and surviving neonates should be discharged from the hospital when anesthetic recovery is complete and milk letdown has been confirmed and all neonates have been

Hypernatremia:
What to Do with the Salty Dog

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Under normal conditions total body water is divided into the intracellular and extracellular fluid compartments with 66% of total body water being located within the intracellular space. The remaining 33% of total body water is distributed between the intravascular and extravascular spaces. Sodium is the most ubiquitous electrolyte in the extracellular compartment. It is responsible for maintaining plasma osmolarity between fluid compartments and regulating water movement between the intracellular and extracellular spaces. Serum sodium and subsequently serum osmolarity is closely regulated by the kidneys and brain under the influence of several hormones the most important of which is antidiuretic hormone (ADH). Changes in serum osmolarity by as little as 1-2 percent are detected at osmoreceptors located in the hypothalamus. Stimulation of these receptors results an increase in thirst and the release of ADH from the posterior pituitary gland. Once released, ADH causes aquaporin-2 channels into the collecting tubules of the kidney resulting in free water retention and restoration of plasma osmolarity. Serum sodium is therefore a better reflection of total body water content than total body sodium content.

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

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

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

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

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

Once correction of hypernatremia has been started serum sodium should be monitored every 4 hours and the rate of correction adjusted to maintain an appropriate drop of sodium without exceeding the established maximum rate of correction (0.5 to 1 mEq/hr). It is typical for correction of severe hypernatremia to require 24 to 48 hours to correct. In addition to serum sodium concentration, the patient should be monitored for neurological signs associated with too rapid a correction including mentation changes (obtundation, stupor, coma), head pressing confusion or seizures. When hypernatremia is corrected too rapidly the intracellular osmolarity exceeds

blood osmolarity and water moves back into the cells down its osmotic gradient. This cellular swelling manifests as cerebral edema and increased intracranial pressure.

If a patient develops any signs associated with cerebral edema the serum sodium should be immediately measured to determine if the sodium concentration has gone down. If the serum sodium is lower, even if the rate of correction has not been excessive, cerebral edema should be considered. If cerebral edema is suspected treatment with 7.2% hypertonic saline at a dose of 3-5 mL/kg over 20 minutes should be started. If hypertonic saline is not available, mannitol can be considered at a dose of 0.5 to 1 g/kg IV over 20 minutes.

Hyponatremia:
Not Enough Salt?

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Under normal conditions total body water is divided into the intracellular and extracellular fluid compartments with 66% of total body water being located within the intracellular space. The remaining 33% of total body water is distributed between the intravascular and extravascular spaces. Sodium is the most ubiquitous electrolyte in the extracellular compartment. It is responsible for maintaining plasma osmolarity between fluid compartments and regulating water movement between the intracellular and extracellular spaces. Serum sodium and subsequently serum osmolarity is closely regulated by the kidneys and brain under the influence of several hormones the most important of which is antidiuretic hormone (ADH). Changes in serum osmolarity by as little as 1-2 percent are detected at osmoreceptors located in the hypothalamus. Stimulation of these receptors results an increase in thirst and the release of ADH from the posterior pituitary gland. Once released, ADH causes aquaporin-2 channels into the collecting tubules of the kidney resulting in free water retention and restoration of plasma osmolarity. Serum sodium is therefore a better reflection of total body water content than total body sodium content. Consequently hyponatremia reflects free water excess. Whether this free water excess represents absolute fluid excess depends on the underlying cause.

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

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

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

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

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

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

Hypovolemic hypoosomolar hyponatremia is the most common category of hyponatremia encountered in daily practice. This type of hyponatremia develops due to volume depletion from either renal or non-renal causes. When volume is lost from the vascular space through the gastrointestinal tract (vomiting or diarrhea) or through loss into a third space (pleural effusion or peritoneal effusion) the

body again attempts to preserve volume at the expense of tonicity through release of ADH and free water retention. Renal fluid and sodium loss occurs from administration of loop diuretics or mineralocorticoid deficiency (hypoadrenocorticism).

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

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

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

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

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

Regardless of the cause of hyponatremia, when correction is begun the patient should have serum sodium levels monitored every 4-6 hours targeting a return to normal of 0.5-1 mEq/hr. The late onset of clinical signs associated with correction of chronic or profound hyponatremia means that a more conservative correction rate is often preferred. Due to the delayed presentation of neurological signs owners should be instructed to monitor the patient carefully and to return if the patient seems to be declining in any way. The best way to manage central demyelination is to be aware of the possibility and to prevent it from occurring. Once demyelination occurs treatment becomes entirely supportive.

Managing Trauma Patients:
The First Critical Hour…and Beyond

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Trauma is a common cause of veterinary emergency room visits. Common causes of trauma include bite wounds and blunt injuries suffered from encounters with vehicles or falls from heights. Due to the high degree of variability possible in the injuries associated with different mechanisms of trauma a systematic approach at the time of presentation followed by a deliberate medical or surgical plan is essential. The initial evaluation of a trauma patient begins with a brief history in which the owner or agent conveys what transpired if known. This initial history should be kept brief and should focus on important facts about the traumatic event including mechanism of injury (fall, hit by car, bite wound), loss of consciousness during or after the event, time from event to presentation and voluntary ambulation post event.

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

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

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

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

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

Orthopedic and cutaneous wounds are finally identified. Patients should be evaluated for puncture wounds, abrasions, lacerations or bruises. Any areas of possible cutaneous injury should be gently clipped to allow for complete evaluation. Long bones and joints are assessed for fractures or disarticulations.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Refeeding Syndrome:
When Good Food Goes Bad

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

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

The physiology of starvation can be divided into an acute response (that occurring within the first 2 weeks of starvation) and a delayed response (that occurring greater than 10 days after the onset of starvation). During the initial phase of starvation or anorexia there is a voluntary reduction in physical work and an involuntary reduction in basal metabolic rate. The decreased metabolic rate is characterized by a decrease in protein turnover, and decreased levels of thyroid hormones and somatomedins. If these changes do not result in equilibrium of energy demand and energy intake then endogenous fuels (adipose tissue, muscle) are used to balance the equation. As mentioned above, triglycerides are the major form of fuel storage in mammals and the body's fat supply is the major determinant of the length of survival under starvation conditions. Carbohydrates are not stored in any significant amount and are therefore of little value with liver and muscle glycogen stores accounting for less than one days worth of caloric needs. While body protein could account for up to 2 weeks of caloric requirements, its depletion would have profound adverse effects due to its role in essential non-fuel functions. The metabolic alteration that occurs under starvation conditions is mediated by changes in circulating hormone levels with decreases in insulin and triiodothyronine (t_3) and increases in glucagon, growth hormone, catecholamines and plasma cortisol. The end result of these hormonal alterations is enhanced hepatic glycogenolysis, gluconeogenesis and skeletal muscle proteolysis thereby facilitating lipolysis. The brain is subsisting on glucose generated from protein catabolism and gluconeogenesis in the liver. During the delayed response to starvation there is a major shift from glucose to fat as the main energy source. Gluconeogenesis is reduced during this period and protein catabolism is minimized. Ketone bodies from hepatic oxidation of fatty acids are used by most of the tissues for energy. At this stage the brain is reliant on ketone bodies as an energy substrate. It is also during this delayed response to starvation that the kidneys begin to assume the role of an important glucose-producing organ.

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

The metabolic response to refeeding following starvation or lengthy malnourishment results from a major shift back to the use of carbohydrates as the primary energy substrate instead of fat. When patients are fed with carbohydrate and protein there is an immediate increase in circulating insulin levels. This insulin secretion inhibits glycogenolysis, gluconeogenesis and fatty acid mobilization by reducing glucagon levels. The same insulin secretion results in enhancement of cellular uptake of glucose, potassium, phosphorus and magnesium. Abrupt cessation of natriuresis occurs causing rapid expansion of the extracellular fluid compartment.

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When the diet used for refeeding is composed of a high proportion of carbohydrate the cessation of natriuresis is abrupt and can lead to the development of peripheral edema and fluid overload. Refeeding with fat or protein alone will allow natriuresis to continue and may prevent fluid overload or edema formation from occurring.

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

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

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

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

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

initiation of nutritional support. Because of the risk for fluid overload, the administration of intravenous fluids should be judicious and should be geared at correcting dehydration and meeting maintenance needs.

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

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

Perhaps the most difficult aspect of preventing and treating refeeding syndrome is convincing the owners/rescuers and hospital staff that a slow and methodical approach to refeeding is in the patient's best interest. A frank discussion with staff members is recommended immediately upon recognition of an at risk patient to ensure that a thoughtful nutritional plan can be formulated. Although it is with the best intentions that rescuers and staff offer food to dogs and cats rescued from starving conditions, that approach can have dire consequences. Veterinarians who are in a position to evaluate patients rescued from hoarding conditions or those responding to natural disasters should brief support personnel prior to seizure of pets to ensure proper introduction of nutrition. Once hospitalized, the best way to prevent overzealous feeding is to delegate responsibility of feeding to one person within the hospital. The attending veterinarian should continue to update staff on the expectations and possible consequences of refeeding syndrome ensuring the staff that they have the patient's best interest at heart to prevent staff members, family or rescuers from sneaking food to the animal. Ultimately, the best treatment for refeeding syndrome is prevention, and prevention begins with awareness.

Resuscitation Endpoints in Critical Care:
It's Not about the Journey, it's about the Destination

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The use of targeted endpoints to guide resuscitation from shock has been in routine use in human medicine for the last 10 years with most evidence directed at resuscitation from septic shock. The utility of endpoints lies in their ability to alert the clinician to the possibility of incomplete resuscitation or occult shock. By evaluating specific endpoints the treating veterinarian is able to determine if a different approach to shock resuscitation is necessary or if addition of vasoactive agents in indicated. The ease with which endpoints are reached may also provide the veterinarian with prognostic information.

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

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

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

Central venous pressure measures the amount of hydrostatic pressure within the cranial vena cava and provides a direct assessment of vascular volume and an indirect assessment of cardiac pre-load. Measurement and normalization of central venous blood pressure allows for the exclusion of hypovolemia as the cause of shock. When CVP is utilized as a resuscitation endpoint the goal should be to achieve a CVP of 8-12 mmHg (11-16 cm H20). Utilization of CVP as a resuscitation endpoint can help prevent morbidity associated with over-resuscitation. Although relatively easy to perform, measurement of CVP requires a water manometer or multiparameter monitor capable of measuring invasive pressures. A moderate to high skill level is needed to successfully and correctly place a central venous catheter in a patient in shock and is often a limiting factor in the use of CVP in veterinary medicine.

Assuming that no co-morbidities exist and that the patient was euhydrated at the onset of shock, urine output is a good resuscitation target. Normal urine production of greater than 1 mL/kg/hr indicates that renal perfusion is adequate to maintain normal GFR making it unlikely that a significant perfusion derangement exists. To be utilized however, the patient must have an indwelling catheter placed and a closed collection system attached. Although this is easy to achieve in most male dogs, it is much more difficult in female dogs and cats of both sexes making its use infrequent.

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

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

Cases from the Trenches:
What We Learn When Things Get Ugly

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This lecture highlights some interesting cases and novel therapies, along with lessons learned along the way. A brief summary of some of the topics touched upon is provided here.

High rise syndrome

High rise syndrome occurs when a patient falls from a height and sustains injuries consistent with the fall. Injuries during these falls are unique due to the body's reaction to free fall. Cats are especially interesting in this regard due to their small body mass and their ability to right themselves so that they can land in a feet-first position. As cats fall, they are able to rotate themselves so that their limbs are extended and directed downwards, and as they reach their maximum speed (terminal velocity, approximately 60 mph in a cat), stimulation of the vestibular system causes them to relax and extend their limbs into a "parachute" like position. Upon landing, the limbs all hit the ground simultaneously, distributing the force evenly to the thorax, limbs, and chin. Thus, common injuries in cats suffering from high rise syndrome include orofacial injuries (epistaxis, hard palate fractures, mandibular fractures), thoracic injuries (pneumothorax, pulmonary contusions), and less commonly, abdominal injuries, pancreatitis and limb fractures.

Cocaine toxicity

Cocaine is an illicit drug with sympathomimetic effects that occur due to inhibition of dopamine, serotonin and norepinephrine re-uptake into presynaptic neurons, resulting in increased concentrations in the synaptic cleft. While cardiovascular signs may occur (tachycardia, hypertension, vasoconstriction), neurologic signs are also commonly reported in veterinary patients.[1] These signs may be secondary to sympathetic stimulation or concurrent intoxication. Even though patients may present with severe neurologic or cardiovascular signs initially, they generally respond well to supportive care including fluids, sedatives or anxiolytics if needed.

Anticoagulant rodenticide toxicity

Anticoagulant rodenticides inhibit the enzyme vitamin K epoxide reductase, preventing the reactivation of vitamin K, which is integral to the formation of clotting factors II, VII, IX and X. Prolongation of the prothrombin time (PT) generally occurs within 36-48 hours of ingestion, with clinical bleeding occurring within 3-5 days. Body cavity bleeding in most common (peritoneum, retroperitoneum, pleural space, lungs) but patients can also hemorrhage into their joints or trachea. Treatment of anticoagulant rodenticide toxicity includes supportive care, plasma transfusion, red blood cell transfusion if necessary, and vitamin K1 (phytonadione) therapy.

Massive transfusion

Massive transfusion is defined as the administration of one blood volume (approximately 80-90 ml/kg in dogs, 40-60 ml/kg in cats) within 24 hours, 50% of one blood volume within three hours, 150% of one blood volume regardless of time, or 1.5 ml/kg/min of blood products for 20 minutes. Complications of massive transfusion include electrolyte abnormalities, hypothermia, acid-base abnormalities and transfusion reactions. Given these potential complications, close monitoring of these patients is recommended. To improve outcomes, newer transfusion strategies, including component blood product therapy in a 1:1:1 ratio of plasma, platelets and red blood cells, as well as additional therapies may be recommended.

Subcutaneous emphysema

Subcutaneous (SQ) emphysema involves the extravasation of air into the interstitial and SQ tissues which dissects between soft tissue planes and causes swelling and tissue compression. In cases of disruption of the trachea causing SQ emphysema, air leakage may also occur into the mediastinum and retroperitoneum. Varying degrees of respiratory distress may occur, and patients can benefit from oxygen therapy, which will help to speed the resolution of the emphysema by facilitating resorption of nitrogen from the distended tissues.

References

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Head Trauma without the Headaches

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Causes of traumatic brain injury (TBI) in veterinary patients most commonly include motor vehicle accidents and animal interactions (bite wounds), with less common causes including things like blunt trauma, falls from heights, gunshot wounds and malicious human activity. Having a basic understanding of the pathophysiology of TBI and remembering to focus on both intracranial and extracranial injuries can help to ensure positive outcomes in these often severely affected patients.

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

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

$$V_{intracranial} = V_{brain} + V_{CSF} + V_{blood} + V_{mass\ lesion},$$

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

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

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

Radiographs of the skull are rarely useful in cases of head trauma as they are insensitive and difficult to interpret. In patients that require imaging, CT is preferred due to its superior ability over MRI to assess bone and areas of edema or acute hemorrhage.

Advanced imaging should be considered in patients with severe abnormalities on presentation, failure to improve over the course of treatment or worsening of signs during treatment, or lateralizing signs.

Two major treatments for intracranial hypertension exist – mannitol and hypertonic saline. Mannitol is an osmotic diuretic that has two main effects: (1) within minutes, it reduces blood viscosity through plasma expansion, improving cerebral blood flow and oxygen delivery to the brain (rheologic effects), and (2) reducing brain water content after 15-30 minutes through a delayed osmotic effect that establishes a gradient between plasma and cells and pulls water out of the brain parenchyma and into the vasculature; this effect lasts anywhere from 1-6 hours. Mannitol has been shown to have a beneficial effect on neurologic outcomes in human patients with head injury, and decreases ICP, and increases CPP and cerebral blood flow. It may also have some free radical scavenging properties. Because mannitol is a diuretic, patients who are hypovolemic should be volume resuscitated prior to administration, and urine output should be carefully monitored. Monitoring of serum osmolality is also recommended in patients receiving repeated boluses, since osmolality > 320 mOsm/L has been associated with acute renal failure in human patients; for this reason and because it could leak into the brain parenchyma and worsen edema, use of mannitol as a CRI in patients with head trauma is not recommended. In veterinary patients, mannitol is usually administered at 0.5-1.5 g/kg over 15-20 minutes through a fluid line filter since the medication can crystallize. Treatment should be followed by crystalloid fluids to maintain intravascular volume.

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

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

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

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

References available upon request.

Medical Management of Thoracic Trauma

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

Pulmonary contusions

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

Pneumothorax

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

Management of a patient with pneumothorax includes continued and careful monitoring and may require repeat thoracocentesis, tube thoracostomy, continuous evacuation, or, rarely, thoracotomy. Generally, surgical placement of a chest tube should be considered when negative pressure cannot be obtained during thoracocentesis or when multiple taps are performed in a short period of time ("three strikes" rule). Two types of indwelling thoracostomy tubes can be placed: larger tubes with a sharp stylet or trochar to allow surgical placement, or smaller long flexible catheters that can be placed using the Seldinger technique under light sedation (Guidewire Inserted Chest Tube, MILA International, KY). Surgical placement of a standard chest tube should be performed under anesthesia with the patient intubated. Necessary materials include clippers, surgical scrub, a sterile blade, small surgical pack, suture, a 12-24 French trocar thoracostomy tube, catheter adapter, 3-way stopcock, injection caps and a continuous drainage device if indicated. Following surgical preparation of the 7th to 11th intercostal space, an assistant pulls the skin cranially about two intercostal spaces, and a skin incision is made over the 8th to10th intercostal space. Hemostats are used to bluntly dissect into the pleural space, and then opened to allow the tube and trocar to fit through the opening. The tube is directed cranially and ventrally and the stylet is removed as the tube is clamped. Releasing the skin allows a subcutaneous tunnel to form around the tube, reducing air leakage. The tube is then connected to a closed system and sutured into place using a purse-string suture around the entry site and a finger trap pattern to secure the tube. Alternatively, the Seldinger technique can be used to place a smaller, long, fenestrated catheter under light sedation and/or local block. Similar surgical preparation is performed following patient sedation, and a local block with 2% lidocaine can be used in the dermis and intercostal muscle. The provided short catheter is used to enter the thoracic cavity between the 7th to 8th intercostal space, and the stylet is then removed to allow for feeding of the wire. The short catheter is then removed, dilation performed, and the long fenestrated catheter placed over the wire. Following removal of the wire, the catheter can be secured with suture and bandaging. In both cases, placement should be checked with radiographs, and 24-hour monitoring is essential.

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

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

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

Penetrating thoracic trauma

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

Other thoracic injuries

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

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

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

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

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

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

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

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

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

Fluid balance should be closely monitored using physical examination, body weight, urine output, and indices of hydration. CVP monitoring may be helpful in guiding fluid therapy (N = 0-5 cm H_2O), although more recent evidence suggests that compounding factors may make these values difficult to interpret. It is important that oliguric or anuric animals not become volume overloaded as this can be life threatening, causing pulmonary/organ edema and failure.

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

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

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

Recovery from acute kidney injury may follow a prolonged clinical course during which afflicted animals are hypercatabolic and suffer from a variety of chemical and acid-base disturbances. Although precise nutritional requirements for small animals with acute kidney injury are unknown, a high-energy, moderate protein, potassium and phosphate deficient diet is a logical choice. In animals that are reluctant to eat appropriate amounts of food, enteral feeding with a nasoesophageal, esophageal, or gastrostomy tube is indicated. If vomiting and nausea preclude enteral feeding, total or partial parenteral nutrition may be administered. Phosphate binders

such as aluminum hydroxide or aluminum carbonate (30-90 mg/kg/day divided with feedings) should be used in conjunction with dietary phosphorous restriction in hyperphosphatemic animals.

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

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

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

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

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

Hypertonic crystalloids (most often 7.2-7.5% hypertonic saline) have an osmolality approximately eight times that of plasma (2400 mOsm/L) and can be used for rapid intravascular volume expansion – up to 3.5 times greater than the volume infused. By creating an osmotic gradient from the intracellular to extracellular space, hypertonic saline decreases intracellular volume and increases vascular volume. As a crystalloid, the effects are short lived, but rapid volume expansion can be helpful in cases of hypovolemic shock, especially for small volume resuscitation or in large patients. Hypertonic saline is commonly used for resuscitation of the head trauma patient or in those with intracranial hypertension. It has also been shown to decrease endothelial swelling, improve cardiac contractility, and improve cardiac output and tissue perfusion by decreasing afterload and increasing preload. Immunomodulatory effects (inhibition of neutrophil respiratory burst activity and cytotoxic effects) have also been reported. Since hypertonic saline is usually provided as a 23.4% solution, it must be diluted before use. This can be done by mixing it with an isotonic crystalloid in a 1:2 ratio (hypertonic saline: crystalloid). Typical doses are 3-5 ml/kg over 5-10 minutes. Too rapid administration (> 1 ml/kg/min) can result in hypotension and bradycardia.   Synthetic colloids are isotonic crystalloids to which large molecules have been added. Administration results in increases in COP and a pull of fluid into the vascular space from the interstitial space. Colloids also increase the intravascular volume greater than the volume infused (about 1.4-1.5 times). In addition to volume expansion, synthetic colloids

remain in the intravascular space longer than crystalloids and provide a more sustained effect. The synthetic colloids most commonly used in the United States include VetStarch, a 6% tetrastarch veterinary product, and 6% hetastarch. Colloids can adversely affect coagulation via dilutional effects and interference with platelet function, von Willebrand's factor and factor VIII, with prolongation of bleeding times seen at doses higher than 20 ml/kg/day. In human patients, concern regarding the occurrence of acute kidney injury has led to the removal of synthetic colloids from the market in some countries. In veterinary medicine, recent studies have shown mixed results correlating the use of synthetic colloids to acute kidney injury. Based on these concerns and a number of large human trials that have shown no clear benefit to using synthetic colloids, the author no longer uses these products.

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

References available upon request.

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Using Acid-Base Analysis in Your Practice:
It Can Save a Life!

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In the emergency setting and during monitoring of critical patients, alterations in acid/base and respiratory function are common, and obtaining and interpreting blood gases can be a rapid and relatively inexpensive means of evaluating a patient and narrowing a list of differentials. With a little bit of practice and regular analysis, acid-base interpretation becomes quick and easy. Acid-base imbalances are important to recognize, as they can lead to impaired cardiovascular function, changes in neurologic status, respiratory impairment and altered response to drug therapies.

It is important to understand several definitions before interpreting acid/base abnormalities. An acid is a molecule that donates a hydrogen ion, and a base accepts one. Buffers are weak acids or bases that help to protect against large changes in pH. The primary extracellular buffer is bicarbonate. pH is a measure of acidity or alkalinity, and is equal to the negative log of the hydrogen ion concentration. Acidemia occurs when the blood pH is less than 7.35, and alkalemia occurs when the blood pH is greater than 7.45. Acidosis and alkalosis refer to the processes causing the pH disturbance. There are four basic types of disturbances: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. PaO_2 is the partial pressure of oxygen dissolved in arterial blood and indicates oxygenation, not ventilation. $PaCO_2$ is the partial pressure of carbon dioxide dissolved in the arterial blood, and is an indicator of the patient's ability to ventilate. The base excess/deficit (BE) is a calculated value that reflects the metabolic portion of the acid/base balance, and is an estimate of how much base needs to be added or taken away from the system to achieve a normal pH at a normal body temperature. Cats have been reported to have slightly lower values for their PCO_2 and a wider range of normal BE than for dogs.

Venous or arterial samples can be obtained, both of which will allow for evaluation of metabolic abnormalities. A venous sample, however, will provide evaluation of only ventilation, while an arterial sample will also provide information about oxygenation. If an arterial sample cannot be obtained, pulse oximetry can be used to assess oxygen saturation in conjunction with a venous sample. Either sample can be used to evaluate overall acid/base status with the exception of severe shock and post-arrest situations which may result in large discrepancies between arterial and venous samples. Whether arterial or venous, samples should be drawn into heparinized syringes with 0.05-0.10 ml (1000 unit/ml) of unfractionated heparin per 1 ml of blood. Less than 1 ml of patient blood is needed for many bedside analyzers. Samples should be capped to avoid exposure to air, and run within 10 minutes (or within 1 hour if kept on ice). Prolonged exposure will result in artefactual changes in CO_2 and O_2.

Once a sample is obtained, there are five steps to interpret the acid/base status. The first step is to assess the pH, determining whether the patient is acidemic (pH < 7.35) or alkalemic (pH 7.45). If the pH is normal, that does not mean that the patient does not have an acid base disturbance – it could be compensated or mixed, so further evaluation is still necessary. The second step is to evaluate acidemia if present. If an acidemia is present, it should be categorized as respiratory ($PaCO_2$ > 45 mmHg) or metabolic (BE < -4 mmol/L or HCO_3^- < 21 mmol/L). Third, if an alkalemia is present, it should be categorized as respiratory ($PaCO2$ < 35 mmHg) or metabolic (BE > 2 mmol/L or HCO_3^- > 25 mmol/L). The fourth step is to determine compensation. A change in the respiratory or metabolic component of the acid-base status normally induces an opposite compensatory response in an effort to normalize the pH. For example, if a primary metabolic acidosis is present, a compensatory respiratory alkalosis may also exist. While the lungs can compensate quickly by adjusting minute ventilation in a matter of minutes, the kidneys compensate more slowly, taking anywhere from a few hours to a maximum of 4 to 5 days. The absence or presence and degree of compensation can provide some information about the chronicity of the disturbance. Overcompensation does not occur, so a mixed disturbance should be suspected if this appears to be the case. Lastly, oxygenation should be assessed. Normal PaO_2 is 90-100 mmHg. PaO_2 of less than 80 mmHg is considered hypoxemia, and less than 60 mmHg represents a severe compromise to tissue oxygenation. If the patient is on supplemental oxygen, the PaO_2 should equal approximately 5 times the FiO_2. Once the sample has been evaluated and the primary disturbance determined, differentials can be considered for metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. If hypoxemia is present, differentials for hypoxemia should also be considered.

Several additional equations may be helpful in acid/base analysis. The Henderson-Hasselbalch equation, derived as:

$$HCO_3^- + H^+ \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2,$$
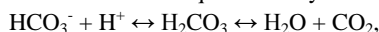
Is important to keep in mind for two reasons. First, the amount of carbon dioxide will affect the bicarbonate measurement. For example, in a patient with a severe respiratory acidosis and elevated PCO_2, the HCO_3^- measurement will be increased, which could be misinterpreted as compensation. Using the BE as a reflection of the metabolic side of the acid/base disturbance is more appropriate because it is independent of CO_2. Second, some acid/base disturbances may require administration of sodium bicarbonate, and this equation is a reminder that sodium bicarbonate will cause an increase in CO_2 and potentially a respiratory acidosis if the patient cannot ventilate appropriately. Another equation, the A-a gradient, can be useful in blood gas analysis when determining lung function in

patients that are hypoxemic. It can be measured by calculating the alveolar PO_2 (P_AO_2) and subtracting the measured arterial PaO_2 from the blood gas. The P_AO_2 can be calculated by:

$P_AO_2 = [PB - PH_2O] FiO_2 - (PaCO_2/R)$, where:

- PB = atmospheric pressure
- PH_2O = partial pressure of water
- FiO_2 = concentration of oxygen in inspired air
- R = respiratory quotient (0.8)
- This equation can be simplified for patients breathing room air at sea level to:

$$P_AO_2 = 150 - 1.1(PaCO_2)$$

After calculating the P_AO_2, the A-a gradient is calculated as:

A-a gradient = $P_AO_2 - PaO_2$

Normal values for the A-a gradient are 5-15 mm Hg, and values greater than 20-25 mm Hg are considered abnormal and indicate ventilation/perfusion mismatch in the lungs. Lastly, calculation of the anion gap (AG) can also be helpful when interpreting acid/base abnormalities. It is the difference between the sums of the commonly measured cations and anions:

$AG = (Na^+ + K^+) - (Cl^- + HCO3^-)$

In the patient, there is no true anion gap because electroneutrality is maintained by unmeasured anions and cations. The majority of unmeasured anions are plasma proteins such as albumin. The unmeasured cations include calcium and magnesium. Decreases in AG are seen commonly with hypoalbuminemia. Increases in AG are seen with some causes of metabolic acidosis such as ethylene glycol toxicity, ketoacidosis, lactic acidosis and uremia. Normal anion gap acidosis is referred to as a hyperchloremic acidosis since the AG is maintained by increases in Cl^- when HCO_3^- is decreased. This can be seen with diarrhea and renal tubular acidosis.

As mentioned, there are four primary acid/base disturbances: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. Once the disturbance has been characterized, differentials should be considered and treatment implemented. Metabolic acidosis is one of the most common disturbances seen in patients in the emergency setting, and severe acidosis can be life-threatening due to effects on the cardiovascular system including vasodilation, hypotension, arrhythmias and decreased cardiac contractility. There are five major differentials for a metabolic acidosis. They are: lactic acidosis, diabetic ketoacidosis, uremic acidosis, exogenous acids (ethylene glycol, salicylates), and loss of bicarbonate. Treatment of each should be directed at the underlying cause. Lactic acidosis is caused by hyperlactatemia, usually due to impaired oxygen delivery or utilization by the tissues and subsequent anaerobic metabolism. Diabetic ketoacidosis is caused by an accumulation of ketones such as the anions of β-hydroxybutyrate and acetoacetate. Uremic acidosis is caused by retained organic and inorganic acids that the damaged kidneys are unable to excrete. With kidney failure, the kidneys are unable to excrete hydrogen ions and cannot resorb bicarbonate. Exogenous acids cause a metabolic acidosis via accumulation of acids from outside of the body such as the metabolites of ethylene glycol. The loss of bicarbonate can occur via the urinary system or gastrointestinal tract. Treatment of metabolic acidosis generally involves fluid therapy, and for causes other than lactate accumulation, may also include treatment with sodium bicarbonate in cases of severe acidosis (pH < 7.0) that persist despite fluid resuscitation.

Metabolic alkalosis is caused by an accumulation of bicarbonate. Major differentials include a high gastrointestinal obstruction with loss of hydrogen, potassium and chloride in the vomitus, or diuretic (commonly furosemide) administration. In patients with gastrointestinal loss of chloride, fluid therapy with 0.9% NaCl may be ideal because of its higher chloride concentration. If the alkalosis is due to diuretic administration, it will usually correct itself once therapy is discontinued or reduced and the patient is eating.

Respiratory acidosis is caused by an accumulation of CO_2 due to hypoventilation. Common causes include neurologic impairment affecting the respiratory center or cervical spinal cord, upper airway obstruction, pleural space disease, chest wall disruption, neuromuscular dysfunction, pain, stress and excitement. Iatrogenic causes such as sedation should also be considered. The treatment for respiratory acidosis includes oxygen supplementation, treating the cause of the hypoventilation (for example, reversing sedatives, performing thoracocentesis to a patient with pleural space disease) and possibly intubation and mechanical ventilation if indicated.

Respiratory alkalosis is caused by hyperventilation. Pathologic respiratory alkalosis is usually caused by hypoxemia, pulmonary disease that stimulates stretch receptors and nociceptors independent of hypoxemia, heart failure and baroreceptor stimulation, pain and anxiety, excessive mechanical ventilation, and multiple factors that stimulate centrally mediated hyperventilation (CNS disease). Therapy is directed at treatment of the underlying cause.

There are five major categories of differentials for hypoxemia. They are (1) decreased partial pressure of inspired oxygen, (2) hypoventilation, (3) ventilation-perfusion (V/Q) mismatch, (4) shunt, and (5) diffusion impairment. A decreased partial pressure of oxygen can occur at high altitudes, but is seen more commonly in veterinary patients due to iatrogenic causes such as a compromised oxygen source for a patient under anesthesia. The differentials for hypoventilation are discussed above. Ventilation-perfusion (V/Q) mismatch occurs when ventilation and blood flow to the alveoli are not closely matched, resulting in inefficient gas exchange. A shunt is an extreme example of V/Q mismatch in which areas of the lung are perfused but not ventilated, resulting in venous blood that mixes with oxygenated arterial blood. Diffusion impairment occurs when the movement of oxygen across the alveolar-arterial

membrane is impaired. Treatment of hypoxemia should be directed at the underlying cause, and emergency stabilization may require oxygen supplementation, intubation and mechanical ventilation.

Table 1: Normal values for canine and feline arterial and venous blood gases (From Middleton DJ and DiBartola SP)

	Arterial Values	Venous Values
Dogs		
pH	7.395 ± 0.03	7.352 ± 0.02
PO2 (mm Hg)	102.1 ± 6.8	55 ± 9.6
PCO2 (mm Hg)	36 ± 2.7	42.1 ± 4.4
HCO3- (mmol/L)	21.4 ± 1.6	22.1 ± 2
BE (mmol/L)	-1.8 ± 1.6	-2.1 ± 1.7
Cats		
pH	7.34 ± 0.1	7.30 ± 0.08
PO2 (mm Hg)	102.9 ± 15	38.6 ± 11
PCO2 (mm Hg)	33.6 ± 7	41.8 ± 9
HCO3- (mmol/L)	17.5 ± 3	19.4 ± 4
BE (mmol/L)	-6.4 ± 5	-5.7 ± 5

Table 2: Expected compensatory responses (from DiBartola SP)

Disorder	Changes	Compensatory Response
Metabolic acidosis	$\downarrow HCO_3^-$	0.7 mm Hg *decrease* in $PaCO_2$ for each 1 mEq/L *decrease* in HCO_3^-
Metabolic alkalosis	$\uparrow HCO_3^-$	0.7 mm Hg *increase* in $PaCO_2$ for each 1mEq/L *increase* in HCO_3^-
Acute respiratory acidosis	$\uparrow PaCO_2$	1.5 mEq/L *increase* in HCO_3^- for each 10 mm Hg *increase* in $PaCO_2$
Chronic respiratory acidosis	$\uparrow PaCO_2$	3.5 mEq/L *increase* in HCO_3^- for each 10 mm Hg *increase* in $PaCO_2$
Acute respiratory alkalosis	$\downarrow PaCO_2$	2.5 mEq/L *decrease* in HCO_3^- for each 10 mm Hg *decrease* in $PaCO_2$
Chronic respiratory alkalosis	$\downarrow PaCO_2$	5.5 mEq/L *decrease* in HCO_3^- for each 10 mm Hg *decrease* in PaCO2

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