Optimal Surgical Timing for Traumatized Patients
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Veterinary patients requiring surgery can be broadly classified into 3 categories as follows: patients requiring immediate surgery to prevent imminent death, patients with varying degrees of hemodynamic stability with injuries that require surgical intervention and patients that are stable and can undergo surgery on a semi-elective basis. Patients within the first and last category require little consideration with regards to timing, however patients within the second category present a dilemma. These patients have often suffered severe trauma and may require surgery for injuries such as long bone fractures, pelvic fractures, severe soft tissue wounds or ballistic injuries. The dilemma arises due to the fact that these patients often develop an inflammatory state similar to sepsis that may not be identified early in their evaluation. In its worst incarnation, patients that undergo inappropriately timed surgical intervention are at risk for developing multiple organ failure due to the addition of surgical trauma/inflammation to the already primed immune response.

Human trauma surgery evolved from an early belief that all surgical procedures should be delayed due to the patient’s being “too sick” to an “early total care” approach in which every patient received definitive surgery as soon as possible. While this approach improved outcomes for many patients a small subset were found to be adversely affected. The most current recommendations for human trauma management focus on a “damage control surgery” (DCS) approach in which a patient is taken to early surgery but the procedure may not be definitive. Patients undergoing a DCS approach require re-operation once they are deemed stable enough to receive definitive care. The DCS approach minimizes surgical trauma while maximizing the benefit (i.e. preventing ongoing contamination or hemorrhage).

The pathophysiology of trauma is complex. The immune recognition of tissue damage is necessary to initiate healing and this recognition occurs trough activation of the immune system. When trauma is severe, this local inflammatory response may become systemic and result in a condition that is virtually indistinguishable from severe sepsis or septic shock. Following a traumatic event two main determinates dictate the host defense response. The first and most important is direct tissue damage from the trauma; the second is sequelae of the inflammatory response. Direct tissue injury is determined by the force and impact severity of the trauma and is commonly referred to as the “first hit”. This first hit in turn dictate the severity of the secondary response or “second hit”. Second hits can be considered endogenous (complications arising from the initial injury; i.e. shock, necrosis, etc.) or exogenous (resulting from attempts to treat the initial injury; i.e. surgical trauma, anesthesia, transfusions, etc.).

Intra-abdominal or intra-thoracic organ damage is the most common clinical problem associated with blunt trauma. Hemorrhage associated with damage to the liver, spleen, kidneys or large blood vessels can be significant. Blunt crushing injuries to intra-abdominal organs including the GI tract can be particularly severe due to the amount of mechanical injury that occurs at the time of trauma. Thoracic injuries such as rib fractures, pulmonary contusions and pneumothorax are often accompanied by shock and hypoxemia.

Long bone fractures are generally associated with a large amount of soft tissue injury in addition to the bony injury. When shock is present the extremities are temporarily sacrificed as precious blood flow is diverted to the core and vital organs. This added hypoxia might exacerbate crushing or shearing injuries and increase the possibility of ischemia and reperfusion injury and secondary infections. Humeral, femoral and pelvic fractures can result in large amounts of blood loss further contributing to the duration and severity of shock. In veterinary medicine blunt torso trauma or crushing bite wounds are the most likely types of trauma to result in a systemic inflammatory response syndrome.

Systemic inflammation secondary to trauma results from stimulation of the innate immune system, the branch of the immune system evolved to respond to novel microbes and to mediate both non-infectious and infectious inflammation. When the inflammatory response is stronger than necessary organ damage and dysfunction can result. The classic example is acute respiratory distress syndrome (ARDS) in which pulmonary capillary permeability is increased leading to the accumulation of pulmonary edema. Necessary activation of the coagulation system occurs in parallel with activation of the immune system at the time of trauma to minimize blood loss. Due to the intricate interactions between these two systems, pro-coagulant states lead to further stimulation of the immune system. Ultimately, this stimulation can result in the development of disseminated intravascular coagulation.

Veterinary guidelines are currently lacking, however extrapolating from the human literature it would appear that the worst timing for surgery of polytrauma patients is between days 2-4 post injury. During this time the systemic inflammatory response and immunologic changes are sustained and the immune system is primed to respond to any additional trauma load. In some cases the very need for surgical intervention is being questioned with a growing movement in human trauma management toward non-operative
management of many blunt injuries. Ultimately, the ideal timing of surgical intervention is likely to be highly patient dependent with the clinician using their judgment to find the balance between adequate resuscitation and prevention of complications associated with delayed surgical intervention (i.e. sepsis, SIRS, MODS).

**Specific disease categories**

Blunt abdominal trauma can result in many injuries including diaphragmatic hernia, hemoperitoneum and axial skeletal fractures. The forces created during these injuries can cause devastating injuries and death with an overall mortality rate of 10-12%. Unfortunately, human and veterinary studies show physical exam findings and blood work results are unreliable for evaluating the severity of abdominal trauma. Diagnostic tests that may impact the decision of surgical intervention have been investigated in human and veterinary studies. Currently in people suffering blunt abdominal trauma, focused assessment with sonography for trauma (FAST) is the preferred imaging modality compared to CT scan for penetrating injuries. The average time to perform this test is 6 minutes with free abdominal fluid being found in up to 45% of patients.

The most common etiology of blunt trauma is motor vehicle accidents, accounting for up to 90% of cases. One large, retrospective study combined all blunt trauma cases, analyzing data from 200 dogs and found 50% of these patients required some type of surgical intervention, with 8% requiring multiple surgeries (Simpson et al). Polytrauma was seen in 72% of these cases illustrating how difficult it is to characterize these patients into only one trauma subtype (open fracture vs. hemoperitoneum, etc). The most common surgical procedures were orthopedic (63.5%) followed by soft tissue procedures (36.5%). Hemoperitoneum was present in 23% of cases and hernias were present in 5% with only 5% of dogs with hemoperitoneum requiring emergency surgery. The mean number of days from admission to surgery in this study was 2.2 days (+/- 1.7 days). Unfortunately, the timing of surgery in relation to the time of trauma or admission was not analyzed for outcome. There were no significant associations with mortality and the need for surgical intervention, length of surgery, length of anesthesia or postoperative temperature.

Diaphragmatic hernia (DH) is the only specific injury that has been evaluated in the veterinary literature with regards to surgical timing and outcome. The overall mortality rate for DH ranges from 6.3-20%. Early evidence suggested that early intervention (less than 24 hours) was associated with an increased mortality rate. A more recent study specifically designed to evaluate surgical timing and outcome found that 42.6% of patients went to surgery within 24 hours of injury with a survival rate of 89.7% (Gibson). These findings suggest that surgical intervention within 24 hours of DH may not have an adverse effect on survival, contradicting earlier recommendations. Importantly, most of the patients in this study were stabilized at a primary care practice before being referred to a specialty center for surgery. Paradoxically, it may be necessary in some patients to perform surgery to achieve hemodynamic stabilization.

Blunt abdominal trauma may result in the rupture of any component of the urinary system; in fact blunt trauma is responsible for 85% of uroperitoneum (UP) cases. This injury can be difficult to discover since up to 69% of animals may urinate or have a palpable bladder at the time of the exam. Surgery is generally recommended for UP but reports of conservative catheter management exist although length of hospitalization is extended (2-4 weeks). Urinary diversion via cystotomy tube, urinary catheter and intra-abdominal drainage is generally recommended prior to attempting definitive surgical repair due the high risk of anesthetic complications associated with the acid-base and electrolyte derangements these patients often have. One large retrospective study in cats failed to find a correlation between time of injury or time of presentation on outcome (Aumann). Mortality rates from 38.5% to 42% have been reported for dogs and cats and delay of diagnosis is associated with an increase in mortality in dogs.

Hemoperitoneum is diagnosed with abdominal effusion has a PCV within 25% of a patient’s peripheral PCV. Many patients likely expire from life-threatening hemorrhage before stabilization can be attempted leading to the paucity of information in veterinary medicine. In one review, arresting ongoing hemorrhage is fourth on a list of initial stabilization goals and can in some cases be managed without the need for surgery (abdominal counter pressure techniques). If a patient cannot be stabilized with volume expansion, blood products and counter pressure, then emergency surgery is warranted. The timing of surgical intervention with regards to traumatic hemoabdomen has not been analyzed, perhaps because the need for surgical intervention appears to be rare. Surgical readiness not only of the patient but also of the facility is vitality important in cases of hemoperitoneum, especially in trauma patients. Specific staffing needs for a decompensating patient (anesthetist, primary surgeon, assistant) must be considered and met before attempting surgical resolution of a hemoperitoneum. Blood products should be available in the operating suite and operative times kept short. This may include rapid clip and prep if catastrophic hemorrhage is occurring. All emergency and pain-related drug dosages should be calculated before induction and the need for ventilation, blood pressure support, and intensive anesthetic monitoring assumed.

Penetrating trauma, especially bite wounds, are common in veterinary medicine with reported survival rates ranging from 38% to 100%. Evaluating surgical recommendations for penetrating trauma is made more difficult due to the vast difference in severity of trauma and mechanism of trauma. Abdominal impalement injuries may warrant special consideration for emergency laparotomy as extent of injuries, particularly with sticks, may not be immediately evident due to the potential for these foreign bodies to migrate. Human patients with gunshot wounds to the abdomen are often managed operatively but can be successfully managed non-
operatively. Recent work suggests that an initial non-operative approach to patients meeting specific criteria leads to fewer non-therapeutic surgeries without affecting outcome.

Trauma associated specifically with bite wounds has been well reported with survival rates ranging from 73-83%. The degree of tissue damage, especially with bite wounds, is commonly underappreciated by visual exam and traditional radiography. It is recommended to take patients to surgery based on finding injuries to the thorax or abdomen because of the high potential for intra-cavitary trauma with early exploratory surgery recommended to rule out more serious internal injury. With regards to thoracic trauma, some recommend exploratory surgery for any patient with a flail chest, rib fractures, lung contusion or pneumothorax but the optimal timing of surgery for these potentially unstable patients is unknown; however many clinicians treat these injuries conservatively with great success and one report showed no significant difference in outcome between cases of flail chest stabilized surgically versus those that were not.

Very little clinical work has been done regarding spinal trauma in veterinary medicine. This may be due to actual or perceived poor outcomes causing many owners to opt for euthanasia in this subset of patients. The most common region affected in dogs is the T3-L3 region (20-55%) with up to 45% of patients having concomitant injuries. Approximately 1/3 of spinal trauma patients are euthanized without treatment while 1/3 receive surgical care and 1/3 are managed conservatively. The necessity of surgical intervention is determined by: spinal instability, compression of the spinal cord, continued pain past 48-72 hours of medical treatment, and deterioration in neurologic status.

Traumatic long bone and pelvic fractures from motor vehicle accidents are one of the most commonly encountered problems in blunt trauma patients yet despite a large amount of research in this area, the optimal timing of definitive surgical stabilization is controversial. In veterinary medicine, no definitive guidelines with respect to fracture management in the traumatized patient have been written, leaving it to the surgeon to determine when the patient is sufficiently stable for a potentially long anesthetic procedure. Up to 59-72% of cats with long bone fractures will have concurrent injuries and that identification and management of these potentially life-threatening problems should be the initial focus. Emergency treatments that must be performed on open fractures include: sedation/aesthesia for patient comfort, removal of gross debris, clipping, flushing of wounds, culture procurement, administration of antibiotics, and coverage of the wounds with a sterile dressing. Unfortunately no specific guidelines as to when these fractures should be definitively repaired are in existence.

Ultimately, the application of DCS principles to veterinary patients is an area that has not been explored. While there may be a subset of patients that may benefit from a DCS approach, this is far from proven, and if attempted this limitation should be kept in mind. Whether DCS has been performed or not, the optimal time to operate a trauma patient that required aggressive hemodynamic stabilization is not known.
Oxygen and carbon dioxide are the two major gases present in the blood in times of health. Each molecule is transported by a unique method and the presence of type of molecule affects the transport of the other. Oxygen is transported in the blood in either a dissolved form or bound to hemoglobin. The amount of oxygen that is transported as dissolved gas is extremely small accounting for 0.3% of total blood oxygen, but it is this form of blood oxygen that is utilized to assess pulmonary function. Carbon dioxide can be transported in three different ways. Approximately 7-10% of CO2 is transported as gas dissolved in plasma. A further 20% is transported bound to hemoglobin as carbaminohemoglobin. The remainder is transported in plasma as bicarbonate after being converted from CO2 in the red blood cells.

Based on these principles blood gas samples may be utilized to evaluate patients for pulmonary failure (hypoxemia), ventilatory failure (hypoventilation) or perfusion derangements. Arterial blood samples are collected from peripheral arteries and are utilized to evaluate patients for hypoxemia or ventilatory failure. These samples must be collected and handled anaerobically and processed promptly since exposure to air can generate several artifacts. Venous blood gas samples can be used to evaluate acid/base status, electrolyte profiles and possible perfusion abnormalities.

Analysis of an arterial blood gas sample begins with the arterial CO2 content (PaCO2). This value is an indication of how well the body is able to move gas from the environment into the alveolus and does not reflect lung function. Normal PaCO2 is between 35-45 mmHg. Values below 35 mmHg indicate hyperventilation, which may be due to many causes including: pain, anxiety, metabolic acidosis or hypoxemia. Hypoventilation on the other hand is diagnosed when the PaCO2 is greater than 45 mmHg. Common causes of hypoventilation include: pleural space disease (pneumothorax, pleural effusion, etc.), thoracic wall disease (flail chest, constrictive bandage), primary neurological disease, drug administration (opioids, dexmedetomidine, etc.), upper airway obstruction (laryngeal paralysis, tracheal foreign body) or severe abdominal distension (GDV, ascites, obesity).

Arterial oxygen content (PaO2) on the other hand reflects how well oxygen is able to move into the blood from the alveolus. Normal PaO2 values range from 80-100 mmHg on room air at sea level. A value below 80 mmHg is defined as hypoxemia. There are 5 general causes of hypoxemia: low partial pressure of inspired oxygen (PiO2), hypoventilation, right to left pulmonary shunt, ventilation/perfusion mismatch (V/Q mismatch) and diffusion impairment. Decreased PiO2 is uncommon in veterinary medicine but may result from a malfunctioning anesthetic machine (no fresh gas flow or exhausted CO2 adsorbent) or from living at high altitude. Hypoventilation leads to hypoxemia by reducing the amount of oxygen in the alveolus and subsequently the arterial blood. Right to left shunting occurs when venous blood bypasses the gas exchange regions of the lung and enters the arterial circulation. This mechanism of hypoxemia is seen with structural cardiac diseases (atrial or ventricular septal defects, reverse patent ductus arteriosis), arteriovenous fistula, pulmonary hypertension or atelectic lung tissue. The most common cause of hypoxemia is V/Q mismatch. This is almost always caused by a specific pulmonary parenchymal pathology (pneumonia, pulmonary edema, PTE, hemorrhage). Although similar V/Q mismatch is not the same as right to left shunting since blood is being exposed to gas exchange surfaces and a small amount of exchange is still occurring. For instance, a single pulmonary capillary may flow past several alveoli, some which are ventilated and others which are not. Diffusion impairment is an extremely rare cause of hypoxemia in veterinary patients but can be caused by any combination of the following three factors: defect in the tissue that inhibits gas diffusion (thickened membranes), decreased equilibration time (fast blood flow) or decreased surface area for diffusion (emphysema).

The A-a gradient is an assessment of pulmonary gas exchange that removes the effect of ventilation and is a useful way to evaluate ventilation and perfusion mismatch. Normal A-a gradient is approximately 5-15 mmHg when breathing room air. This difference is primarily due to normal small amounts of right to left shunting (pulmonary blood vessels, i.e. bronchial, mediastinal etc.) and gravity dependent irregularities of blood flow in the lung. The A/a gradient is determined by subtracting the partial pressure of arterial oxygen from the partial pressure of alveolar oxygen. Alveolar oxygen is determined using the following formula: P AO2 = [(Barometric pressure – 50 mmHg) x FiO2] - (P,CO2/0.8) where barometric pressure at sea level = 760 mmHg and fraction of inspired oxygen (FiO2) ranges from 0.21 (room air) to 1.0 (100% oxygen). It is normal for the A-a gradient to increase as patients age. The A-a gradient can increase to approximately 150 mmHg when a patient is breathing 100% oxygen.

Since the A-a gradient becomes less reliable when oxygen is supplemented the PaO2/FiO2 ratio is used to quantify pulmonary function in this setting. This is possible because when FiO2 is greater than 0.50 the PaCO2 exerts a negligible effect on PaO2 and can therefore be ignored. Normal values for PaO2/FiO2 ratio are greater than 500. Values between 300-500 represent mild oxygenating inefficiency; values between 200-300 represent moderate inefficiency and values less than 200 represent severe venous admixture. A rough approximation of the PaO2/FiO2 ratio can be made by using the “5 times rule” in which the PaO2 should be roughly 5 times the inspired oxygen percentage assuming normal ventilation.
Pain Management of the Critically Injured Patient  
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The importance of effective pain management goes beyond the ethical consideration of ameliorating pain. The presence of pain causes increases in sympathetic tone and catecholamine secretion leading to increased cardiac output, increased peripheral vascular resistance and increased myocardial oxygen consumption. While important for the fight or flight response these changes, if chronic, further impair oxygen delivery to tissues. In addition to the cardiovascular effects of pain, a neuroendocrine response occurs resulting in elevated levels of catabolic hormones including cortisol while a concurrent decrease in the anabolic hormones insulin and testosterone is present. The net result of the neuroendocrine response is the development of a catabolic state characterized by hyperglycemia, type B hyperlactatemia and ketogenesis. Treatment of pain reduces or eliminates these responses, facilitates patient evaluation, improves patient quality of life and leads to more rapid healing and reduced morbidity.

Patients that suffer severe trauma or burns should be considered to be very painful and analgesia should be provided liberally. The best method of pain management in traumatized or burned patients utilizes a multimodal approach. Pure opioid agonists are the mainstay of pain management and should be provided early in the course of treatment. Opioid medications have no ceiling effect meaning that there is no maximum dose at which further administration would not be expected to affect a result. In most cases administration should be continued throughout the duration of hospitalization. While opioids are capable of depressing respiratory drive in veterinary patients this is rarely clinically relevant. If there is concern over a patient’s ventilatory drive then the dose of opioid can be reduced or broken into aliquots and administered to effect.

The dissociative agent ketamine has some analgesic properties, particularly for superficial pain. More importantly perhaps are the n-methyl D-aspartate (NMDA) antagonistic properties of ketamine that may alter spinal modulation and prevent or ameliorate central sensitization (wind-up). In order to derive the full benefit of ketamine in regards to sensitization prevention it should be administered for at least 24 hours as a constant rate infusion (5-15 mcg/kg/min). The use of ketamine alone is not effective for deep or visceral pain and can result in behavioral changes, therefore ketamine should almost always be administered in conjunction with an opioid. When discontinuing ketamine for patients that have been on a CRI for longer than 24 hours a gradual reduction of the infusion rate over several hours is used to prevent behavioral changes associated with abrupt discontinuation.

Clinically, the α2-agonists are most commonly used for sedation however their use for pain management is becoming more common. Their short half-lives and reversibility make them theoretically attractive options in many trauma patients but careful consideration should be taken prior to their use since clinically relevant cardiovascular side effects are possible. Animals in shock or with significant heart disease (especially diseases causing decreased systolic function) should not be administered α2-agonists. A constant rate infusion is often employed to most effectively use this class of drugs as a component of the pain management protocol. When used at rates of 0.5 to 2 mcg/kg/hr, dexmedetomidine can result in anxiolysis and analgesia without significant cardiovascular side effects.

Regional or local anesthesia should be considered as an adjunct to traditional, systemic analgesic methods. The use of regional or local anesthesia often requires coordination with the surgical team since these interventions often require general anesthesia or sedation to be performed. Placement of diffusion catheters for repeated administration of local anesthetic medication should be considered at the time of surgery if applicable; their use in contaminated wounds (bite wounds) should be avoided however. Epidural administration or placement of an epidural catheter can provide excellent pain management for animals with pelvic limb or pelvic ring trauma. If sacral fracture or sacro-iliac luxation is present or the landmarks used for epidural administration are disrupted then epidural administration should not be performed. Local anesthetic drugs can also be used systemically with IV infusion of lidocaine being useful as both an analgesic adjunct and a pro-motility agent for management of ileus.

Both blunt trauma and burn injury are capable of inducing a very pronounced inflammatory state. The use of non-steroidal anti-inflammatory drugs can be very beneficial in these patients however, animals that are severely traumatized or are in shock should not be administered NSAIDs until all tissue perfusion has normalized and is not anticipated to change abruptly. Patients with severe soft tissue injury or crush injury are at risk for development of myoglobinuria and renal failure. Any animal that has pigmenturia or evidence of renal insufficiency should not receive NSAIDs. The decision to use NSAID medications should be deliberate and if case selection is appropriate then NSAIDs may prove to be a very useful analgesic adjunct. Due to the risk of systemic side effects including further impairment of the already compromised immune system in these patients, animals that have suffered severe trauma or burn injury should not receive corticosteroid medications unless specifically indicated.

Many animals that suffer severe soft tissue injury require repeated sedation events on concurrent days or even multiple sedation events within the same 24-hour period. Patients that are going to require repeated sedation should be identified early in the course of hospitalization and measures should be taken to coordinate their care. Following repeated sedation with the same anesthetic medications it is common to see recovery times become more prolonged and patients require larger doses of drugs to achieve the same
level of sedation due to the development of tolerance. It is good practice to alter the sedation protocol every few days to prevent development of tolerance or toxicity with repeated dosing such as reported with propofol administration in cats. Additionally, animals that are sedated serially are at risk for nutritional deficiency due to repeated fasting. Efforts should be made to sedate the animal at the same time every day to allow for the most opportunities to provide nutrition.
Historically, hemostasis has been divided into two categories: primary hemostasis, which is platelet mediated and secondary hemostasis that is mediated by soluble clotting factors. Normal hemostasis begins with damage to the endothelium leading to exposure of tissue factor and collagen. When this tissue injury occurs local vasoconstriction takes place decreasing blood flow to the damaged vessel. Platelets become activated and bind to subendothelial collagen and von Willebrand’s Factor (vWF). These platelets change shape and release granules to attract other platelets resulting in the formation of a temporary platelet plug. Concurrent with the formation of the platelet plug, soluble factors are activated primarily via the tissue factor pathway (extrinsic) with the ultimate formation of a fibrin meshwork that stabilizes the platelet plug and provides lasting hemostasis.

Testing the hemodynamic system in a bleeding patient begins with obtaining a comprehensive history. Specifically, any individual history or lineage history of bleeding that required interventions should be elucidated; this includes need for blood product transfusion, bandaging or surgery. A thorough physical exam should be performed with extra attention paid to perfusion parameters such as mucous membrane color, pulse quality, capillary refill time and respiratory rate. The site of bleeding should be evaluated to determine if the cause is not due to a hematicstic defect (i.e. loss of blood vessel integrity). Primary hemostatic disorders often result in development of petechia, ecchymoses or gastrointestinal bleeding whereas disorders of secondary hemostasis often lead to hematoma formation or cavitory bleeding (hemothorax, hemoabdomen, pericardial effusion).

Readily available methods to evaluate primary hemostasis include platelet count and buccal mucosal bleeding time (BMBT). Thrombocytopenia can be caused by decreased production, increased consumption or destruction. Automated platelet counts are generated by in house CBC analyzers however, agglutination (platelet clumping), poor sample quality, giant platelets and red or white blood cell fragments may all cause erroneous results. Manual platelet estimates can be performed by evaluating a direct blood smear on 100x magnification in the red cell monolayer. Each platelet observed at this magnification is equal to approximately 15 x 10^3/ul platelets. The number of platelets in several high-powered fields (HPF) is averaged and the total multiplied by 15 x 10^3/ul to obtain an estimate. When performing a manual platelet estimate it is imperative that the feathered edge be carefully evaluated for the presence of platelet clumps. Normal platelet counts range from 170 – 400 x 10^3/ul.

Buccal mucosal bleeding times are performed by on patients with a known normal platelet count and coagulation profile. In theory BMBT evaluates platelet function and platelet interaction with subendothelial components such as vWF and is not affected by disorders of secondary hemostasis. Unfortunately the BMBT test is highly operator dependent, poorly reproducible and insensitive for detection of mild bleeding disorders. For the best results a commercially available spring loaded device is used for incising the oral mucosa of the upper lip. The patient is gently restrained and the lip is inverted to expose the buccal mucosa. The test device is placed flat against the mucosa and deployed resulting in 2 incisions of equal length and depth. Blood is gently blotted away using a circular filter paper taking care not to disturb the forming clot. The final time is measured from the time of device deployment until cessation of bleeding. Normal BMBT time is 2-4 minutes.

Secondary hemostasis is typically evaluated by measuring the prothrombin time (PT) and the activated partial thromboplastin time (aPTT). The prothrombin time evaluated the tissue factor (extrinsic) and common pathways (factors VII, X, V, prothrombin, fibrinogen) and is unaffected by platelets or intrinsic factors. The test is run on citrated blood that is exposed to thromboplastin, phospholipids and calcium. Since factor VII has the shortest half-life of the vitamin K dependent coagulation factors (II, VII, IX, X), PT is particularly useful as a screening test for rodenticide exposure and should be performed 2-3 days post exposure. Importantly, a normal PT does not definitively rule out factor deficiency since prolongation typically does not occur until greater than 75% of factor activity is lost.

Activated partial thromboplastin time evaluates the intrinsic and common pathways (XII, XI, IX, VIII, V, X, prothrombin, fibrinogen). The aPTT is also run on a citrated blood sample exposed to phospholipids and calcium. In combination with a normal PT the aPTT can be useful as a screening test for hemophilia A (VIII) or B (IX). Unfortunately, prolongation of the aPTT with normal PT also occurs with factor XII deficiency which does not lead to clinical bleeding and is relatively common in cats. Activated partial thromboplastin time is subject to the same limitation as PT in that 75% of factor activity must be lost before significant prolongation of the test time occurs.

Ultimately, definitive diagnosis of either an inherited or acquired factor deficiency must be made by specific factor assays in which individual factor activity levels are measured and compared to a standardized normal. Specific factor assays exist for most soluble coagulation factors and von Willebrand’s factor. Perhaps the most commonly assessed soluble factor is fibrinogen, which can be measured by several different desktop analyzers. Hyperfibrinogenemia can occur with inflammation, stress, infection or disseminated neoplasia. Hypofibrinogenemia may be congenital, acquired (hemodilution, consumption, DIC, sepsis), or due to decreased hepatic synthesis.
Fibrinolysis is more difficult to evaluate directly without performing viscoelastic testing which has limited availability and is therefore not very practical. Indirect tests of fibrinolysis include measurement of fibrin degradation products (FDP) and D-dimers. Fibrin degradation products are produced when fibrinogen or fibrin is cleaved by plasmin. Elevation of FDPs may be an indication that the fibrinolytic system is active although elevated FDP may occur in conditions with hyperfibrinoginemia. D-dimers are a unique type of FDP that is specific for mature clot breakdown. Elevation of D-dimers is therefore indicative of degradation of a mature clot.
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 oversuscitation 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 cavitory 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 a 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 dexametomidine are also useful analgesic drugs at doses lower than those used for sedation. Dexametomidine is typically used as a CRI at a rate of 0.5 to 2 mcg/kg/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 immunocompromized patient with potential pathogens such as methicillin resistant staphylococcus species.

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

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

Determination of prognosis can be difficult due to the confounding effect of euthanasia. Factors that have been associated with poorer outcomes include: head trauma, vertebral fractures, 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%.
Veterinary CPR Update
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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 CPA is most likely to be performed. In most veterinary practices one of these locations will be in the vicinity of the operating room or areas where anesthesia is most frequently performed. In addition to crash carts, an easily visible and legible poster or individually tailored form with pre-calculated doses of common CPR drugs should be available. The veterinary team should make efforts to develop leadership and communications skills for members of the staff to improve their effectiveness during CPR. Communication skills can be further honed, and performing a debriefing following each CPR event can reveal important learning points. During this debriefing the team should critically evaluate their performance and determine what aspects of the CPR went well and which areas need to be targeted to improve future performance. The adage “practice makes perfect” or at least “practice makes better” in the case of CPR holds true. Therefore, the use of high fidelity mannequins or veterinary CPR training dummies and regular training events is recommended as their use can improve effectiveness of CPR by developing psychomotor skills allowing the clinician to slow down the event and think in a more clear fashion.

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

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

In order to optimize CPR it is necessary that patients be monitored for signs of success or failure. All patients with suspected CPA should be instrumented with ECG. Analysis of ECG may help to rule out CPA or may identify rhythms that are amenable to a specific treatment (i.e. defibrillation). In the ideal setting all patients undergoing CPR will have end-tidal CO₂ monitoring performed. While useful for confirming correct placement of endotracheal tubes within the tracheal lumen in non-CPA animal, EₜCO₂ should not be used as the sole confirmation of endotracheal intubation in CPA animals. Since EₜCO₂ 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, EₜCO₂ levels can predict the likelihood of success with EₜCO₂ level of less than 15 mmHg and 20 mmHg suggesting worse prognosis in dogs and cats respectively. Once ROSC occurs EₜCO₂ will undergo a rapid and sustained rise as the heart more efficiently delivers CO₂ laden blood from the periphery to the pulmonary circulation. If ROSC is achieved then post-resuscitative monitoring should be tailored to the individual patient’s needs.

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