TRIAGE STAT! EMERGENCY APPROACH TO THE TRAUMATIZED SMALL ANIMAL PATIENT

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Initial Assessment and Stabilization: Remember the "ABC's"

One of the most important concepts to remember when approaching any critically ill patient is to routinely perform a rapid primary survey, keeping in mind the ABC's of evaluation and resuscitation.

"**A**": **A**irway and **A**rterial Bleeding. Observe the patient from a distance. Take note of the patient's respiratory rate and character. Rapid, shallow, restrictive respirations can be associated with a variety of conditions of the thoracic cage, pulmonary parenchyma, or pleural space, including the pain associated with rib fractures, flail chest, pulmonary contusions, diaphragmatic hernia, pneumothorax, or hemothorax. Any arterial bleeding should have a compression bandage or rapid ligature placed to prevent exsanguination. Definitive repair of lacerations can occur once the patient's overall status has been assessed and the clinical condition is determined to be stable.

"B": Breathing: What is the color of the mucous membranes? Watch the character of the patient's respirations. Slow deep respiration with inspiratory stridor is often associated with an upper airway obstruction. Careful auscultation of the upper airways and thorax can aid in the diagnosis of the primary problem. Harsh sounds that are the loudest over the arytenoid area is likely associated with an upper airway obstruction, whereas, harsh pulmonary crackles after a traumatic event are most likely associated with pulmonary contusions. Decreased lung sounds dorsally with a restrictive respiratory pattern may be associated with pneumothorax or the presence of a diaphragmatic hernia. Decreased lung sounds more ventrally may be associated with pleural effusion including hemothorax or a diaphragmatic hernia, depending on the location of the rent in the diaphragm, and the abdominal contents now within the pleural space.

"**C**": **C**irculation Assess the patient's perfusion status. What is the heart rate and rhythm? What is the ECG? What is the blood pressure? What is the pulse quality? What is the capillary refill time? Is there any evidence of external hemorrhage, or do you suspect internal bleeding? When clinical signs of hypovolemic shock are present, fluids must be replaced in an emergency phase of fluid resuscitation.

"**D**": **D**isability Is the patient ambulatory? What is the patient's mental status? Is it the same as on presentation or is the patient becoming more mentally dull or obtunded. Are the pupils equal in size or is there any anisocoria? Is the patient laterally recumbent with rigid forelimbs and flaccid paralyzed hind limbs suggestive of a Schiff-Sherrington with a spinal cord lesion somewhere between T3 to L3? If so, that patient should be placed immediately on a backboard to prevent further neurologic injury. Does the patient have evidence of fractures? Are there any open wounds that should be covered to prevent infection with nosocomial organisms? If there is blood on the patient, always wear gloves, as sometimes human caretakers get bitten during the process of transporting the injured animal. You might not be sure whether the blood on the animal is human or non-human animal in origin.

Treatment of Shock

Shock is defined as inadequate circulating blood flow such that oxygen delivery is insufficient to meet cellular energy and substrate demands. After sustaining a traumatic injury, shock is usually associated with some form of hypovolemia and inadequate circulating blood volume secondary to internal or external hemorrhage. Treatment of shock largely consists of re-establishing adequate circulating blood volume without exacerbating further hemorrhage.

Ideally, the administration of isotonic crystalloids fluids and natural and synthetic colloids during the treatment of hypovolemic shock should be based on constant assessment and reassessment of the patient's cardiovascular status and perfusion parameters. In dogs, shock volume of fluid is related to the patient's intravascular blood volume, 90 ml/kg. In cats, shock volume of fluid is calculated at 44 – 45 ml/kg. Typically, I start with administering ¼ of the calculated shock volume as rapidly as possible, then reassess the patient to evaluate if heart rate is decreasing, if blood pressure is rising, and if the patient's capillary refill time and mucous membrane color is improving. Fluid resuscitation to reach supraphysiologic blood pressures should be avoided. First, hypertension can cause clots that have formed to become unplugged, exacerbating further hemorrhage. Secondly, overzealous fluid administration of isotonic crystalloid fluids can contribute to the diffusion impairment and interstitial and alveolar flooding observed with pulmonary contusions. Finally, dilutional coagulopathies can occur with fluid replacement without administration of coagulation factors. Ideally, fluid therapy should be titrated to a systolic blood pressure of 100 mm Hg, diastolic blood pressure above 40 mm Hg, and mean arterial blood pressure above 60 mm Hg. Pulse pressure and quality alone are poor methods of assessing an accurate blood pressure in the traumatized patient, and thus, direct or indirect methods should be obtained, whenever available.

To avoid iatrogenic worsening of pulmonary edema and dilutional coagulopathies, administration of synthetic colloids such as Hydroxyethyl starch (5 ml/kg IV) can be administered as a bolus. Although some authors feel that administration of a colloid to a patient with pulmonary contusions can worsen pulmonary pathology and diffusion impairment, the risks of colloid administration are largely outweighed by the benefits of small volume resuscitation and decreased alveolar flooding with isotonic fluids. Hypertonic saline (7.5%) can be administered as a bolus (5 – 7 ml/kg IV in dogs, 2 – 4 ml/kg IV in cats) along with a colloid (5 – 10 ml/kg IV) in a hypovolemic traumatized patient. Hypertonic saline draws fluid from the intracellular and interstitial spaces into the intravascular compartment to restore circulating fluid volume and oxygen delivery. The effect of hypertonic saline is short-lived, and lasts just 20 – 30 minutes without further colloid or crystalloid administration.

Finally, in some cases, shock remains unresponsive to fluid administration due to continued patient pain and discomfort. The judicious and appropriate use of analgesic drugs is necessary as one of the most important treatments of any trauma patient.

Analgesia for the Traumatized Patient

No patient should ever be painful. Depending on the nature of the patient's injuries, however, analgesic choices should be considered carefully to prevent iatrogenic exacerbation of injuries and impaired oxygen delivery. In cases of head or ocular injury, for example, ketamine should be avoided due to the risk of increasing intracranial and intraocular pressure. No patient should receive any α-2 receptor agonist due to the inherent properties of decreased cardiac

output, and increased systemic vascular resistance even at minutely small doses. Instead, the best drugs available for veterinarians to use are opioids that cause minimal cardiovascular and respiratory depression and can readily be reversed with naloxone if difficulty arises. Opioids are classified based on their potency relative to morphine. Fentanyl (2 mcg/kg as an IV bolus, followed by 2 – 7 mcg/kg/hour IV CRI) is the most potent drug we have available in our analgesic armamentarium. Fentanyl has a potency 100 times that of morphine, and is extremely safe to use in patients with severe trauma. Hydromorphone, too, is a safe and potent alternative (0.1 – 0.2 mg/kg IV, SQ, IM). Partial agonists such as buprenorphine, or agonist-antagonist drugs such as butorphanol can never reach the same efficacy of analgesia as the pure mu-agonists, and therefore, are not ideal to use in any painful patient. Both buprenorphine and butorphanol bind avidly to opioid receptors. Because of this pharmacokinetic property, it may be difficult to reverse any adverse side effects that may occur, and they may also inhibit the efficacy of more potent analgesics used later. In specific circumstances such as rib fractures and flail chest, local anesthetic blocks can greatly assist in pain management and improve ventilatory function. Nonsteroidal anti-inflammatory drugs should not be administered until the patient's intravascular volume status and blood pressure have been normalized, due to the risks of renal and GI hypoperfusion.

Thoracic Trauma

Many patients with thoracic trauma and associated injuries have a rapid, shallow, restrictive respiratory pattern, often with a pronounced expiratory effort. Trauma to the thorax is first characterized as open versus closed thoracic trauma. Injuries can occur that involve the pleural space, pulmonary parenchyma, thoracic wall, and tracheobronchial tree. Finally, injuries to the thorax can also damage or irritate the underlying myocardium and lead to cardiac dysrhythmias and impaired cardiac output. The four most common injuries associated with trauma to the thoracic cage include pulmonary contusions, pneumothorax, rib fractures or flail chest, and a diaphragmatic hernia. In many cases of thoracic trauma, any or all of these injuries may be observed, depending on the severity of the trauma. Thoracic radiographs should be performed only after initial stabilization with oxygen, therapeutic (relieve respiratory distress) and diagnostic (confirm pneumothorax) thoracocentesis and alleviation of respiratory distress.

Abdominal Trauma

Any penetrating traumatic injury to the abdomen requires surgical exploration. A negative exploratory laparotomy is much better than waiting for septic peritonitis to manifest itself as leakage from bowel or biliary perforation occurs. In some cases, injuries such as hemo- or uroabdomen are obvious at the time of initial injury. In other cases, however, mesenteric thrombosis or bile peritonitis may take days to weeks to become apparent. Diagnosis of abdominal trauma is usually based on index of suspicion, abdominal radiographs, ultrasonography, and abdominal paracentesis or diagnostic peritoneal lavage. Placing an abdominal compression bandage around the patient's abdomen with careful titration of intravenous fluid support is usually sufficient to tamponade any hemorrhage. Most recently, human trauma surgeons and criticalists have learned from what veterinary criticalists have known for years, and are becoming more conservative in their approach, as well. Ruptured urinary bladder, avulsed kidneys, avulsed ureters, and traumatic injury to the urethra can cause life-threatening metabolic complications, but are rarely a surgical emergency, provided that aggressive fluid and medical management are performed. Abdominal fluid creatinine should be compared with peripheral creatinine to rule out a uroabdomen in any case of traumatic injury to the abdomen. If abdominal fluid creatinine is greater than that in the periphery, a diagnosis of uroabdomen is made. If creatinine is not available on an emergent basis, a simple azostick comparison or potassium will also suffice. The urea nitrogen and potassium in the abdominal

fluid will be greater than that in the periphery if urine is present. Placement of a drainage catheter into the abdominal cavity under local anesthesia, then connecting the drainage catheter to a closed collection system is usually sufficient to remove urine from the abdominal cavity until the patient can be stabilized medically and become a more suitable candidate for anesthesia and definitive surgical repair of the urinary tract trauma. In such cases, the presence of an inappropriate bradycardia can signify atrial standstill secondary to hyperkalemia. Every effort should be made to decrease serum potassium to less than 7 mmol/L before any anesthesia is induced. Treatment protocols include administering calcium gluconate (0.5 – 1.0 ml/kg 10% solution IV), regular insulin (0.25 units/kg IV) with dextrose (2 gm dextrose IV per unit of insulin, followed by 2.5 – 5% dextrose CRI to prevent hypoglycemia), or intravenous sodium bicarbonate (0.25 – 1.0 mEq/kg).

Neurologic Trauma

The patient should be assessed carefully for mentation, the presence of nystagmus, miosis, stupor, coma, seizures, or abnormal postures such as Schiff-Sherrington. Worsening mentation or coma after a head injury should rapidly be treated with mannitol (0.5 – 1 g/kg IV) followed 20 minutes later by furosemide (1 mg/kg IV). Although there is a potential risk of worsening intracranial hemorrhage, patient's that are dying before your eyes can benefit from this aggressive therapy. If spinal trauma is suspected, the patient should be stabilized immediately on a flat stable surface to prevent worsening of a potentially correctable injury. The absence of deep pain perception indicates a very poor prognosis for return to function. It is important to attempt to elicit some degree of conscious perception of a painful stimulus, rather than a local withdrawl reflex alone, when making the decision to pursue further aggressive therapy in cases of spinal trauma. If concurrent cerebral injuries are present, it may be difficult to accurately assess spinal cord function until the patient is more alert. The administration of glucocorticosteroids in the treatment of head trauma or any other form of shock is not indicated unless the patient has severe head injuries that is causing swelling of the oropharynx and obstruction to adequate ventilation. Glucocorticosteroids have not been shown to definitively improve neurologic outcome in cases of head injury. Additionally, glucocorticosteroids influence negative nitrogen balance, delay wound healing, impair glucose homeostasis, and suppress immune function. Hyperglycemia and decreased cerebral oxygen delivery can contribute to intracranial and intracellular acidosis that can worsen neurologic outcome