Stabilizing the Trauma Patient

Harold Davis, BA, RVT, VTS (ECC)
William R. Pritchard Veterinary Medical Teaching Hospital
University of California, Davis CA

Trauma is a wound or injury occurring as a result of an animal's body striking or being struck by an object, from environmental, chemical and thermal insults, or injuries inflicted by other animals. Motor vehicle, animal interactions and trauma of unknown cause comprised 70 to 80% of all injuries in dogs and cats in one study.

The most frequent sources of trauma are mechanical forces. These forces produce injuries that can be explained by the physical laws governing acceleration, inertia, momentum, and the absorption and dissipation of energy. Trauma results in disruption and / or impairment of tissues, cellular damage, and organ dysfunction. In addition, mild to severe or even, fatal hemorrhage can occur.

Trauma patients may have multisystemic problems, which require rapid and accurate assessment, and treatment. This discussion will address pathophysiology, assessment, management and monitoring of the thoracic, head, abdominal, and musculoskeletal trauma patient. In addition, hypovolemia will be discussed.

**Primary Survey and Resuscitation**

When presented with a trauma patient a primary survey is performed. The primary survey is the initial, brief assessment, which addresses the ABCDEs of emergency care (A) airway, B) breathing, C) circulation D) for dysfunction or disability of the central nervous system, and E) for examination). When a life-threatening problem is identified during the primary survey then resuscitative action should be instituted immediately.

**Secondary Survey**

Following the primary survey a secondary survey is performed. The secondary survey is the timely, systematic, and directed evaluation of each body system for injury. Injuries of a lower priority are addressed following initial stabilization. A thorough head to tail physical examination and history are completed. Finally, a comprehensive plan of diagnostics and monitoring is developed and carried out.

**Thoracic Trauma**

Trauma of the thorax can result in pleural filling defects such as pneumothorax, hemothorax and diaphragmatic hernia. Other problems include rib fractures / flail chest, pulmonary and myocardial contusions.

**Pathophysiology**

A simple pneumothorax is caused by the presence of air in the pleural space. The air comes from the outside through the chest wall, from a defect in the lung or both. A tension pneumothorax is caused by air entering the pleural space via a one-way valve flap without the ability to escape. Intrapleural pressure exceeds atmospheric pressure.
The accumulation of air permits the separation of the visceral and parietal pleural surfaces. As the separation expands the lung collapses. Hypoxemia ensues because of a ventilation perfusion mismatch. Venous return may decrease owing to the loss of negative pleural pressure.

Blood in the pleural space constitutes a hemothorax. Blood comes from several sources such as the lung, intercostal vessels, or systemic arterial vessels. Small amounts of blood do not usually cause detectable signs, while blood loss sufficient to cause hypovolemia (30 – 60 ml/kg) can affect ventilation.

Penetrating and / or blunt trauma can cause diaphragmatic hernias. A tear occurs in the diaphragm allowing abdominal contents to enter the thorax. The severity of respiratory distress is dependent on the amount of abdominal organs in the pleural cavity. The space occupied by the organs can restrict lung expansion and reduces ventilation.

Rib fractures may cause pain, hemorrhage (owing to laceration or tear of intercostal arteries), and injury to the pulmonary parenchyma. When three or more adjacent ribs are fractured this is termed a “fail chest”. These patients display a paradoxical breathing pattern (flail segment collapses in on inspiration and expands out during exhalation). It is thought that the flail chest causes minimal respiratory compromise and that it is the presence of pulmonary contusions that is the major contributor to hypoxemia in these patients.

Pulmonary contusions are caused by the transmission of mechanical forces to the thoracic cage resulting in increased tissue pressure and tearing of tissues. Direct laceration may occur from displacement of rib fractures or chest wall compression causing interstitial and alveolar bleeding. Ventilation perfusion mismatch and loss of lung compliance may occur as a result of the pulmonary parenchymal damage. The end result is hypoxemia, increased work of breathing, and with severe lesions, hypercarbia.

The heart lies between the left and right thoracic wall. Myocardial contusions result from the compression of the heart, between the ribs and the bruising of the myocardium. The injury can cause alteration in the heart’s electrical conduction system leading to arrhythmias, decreased cardiac output and poor oxygen delivery.

**Patient Assessment**

First, patency of airway and adequacy of ventilation should be assessed. This is done by visualization, auscultation, and palpation. When looking at the animal, you can determine if the animal is tachypneic or having difficulty breathing. Some animals with respiratory distress may assume a posture with the head and neck extended with abducted elbows. Additional signs include absent chest wall motion, exaggerated ventilatory effort, flaring of the nares, open mouth breathing and paradoxical breathing. Cyanosis may be seen, indicating hypoxemia. Animals with small and large airway problems may have noisy breathing, either stertor / sonorous or wheezes which is
suggestive of partial airway obstruction or bronchoconstriction respectively. Patients with absent or diminished breath sounds and a rapid shallow breathing pattern are suggestive of pleural filling defects (Pneumo/hemo thorax and / or diaphragmatic hernia). The chest wall may be palpated to assess chest wall integrity. Crepitus about the body may be due to subcutaneous emphysema, which can be caused by tracheal tears, or chest wall defects.

Radiographic imaging can be helpful in making a diagnosis of pleural filling defects, rib fractures and pulmonary parenchymal injury. Contrast agents may be required to diagnose a diaphragmatic hernia. Ultrasound may also be helpful in the diagnosis of diaphragmatic hernia.

The end result of pulmonary related trauma is hypoxemia. Arterial blood gases and / or pulse oximetry can aid in the diagnosis of hypoxemia. Hypoxemia is defined as a PaO$_2$ of ≤ 80 mm Hg or an SpO$_2$ ≤ 91%.

**Patient Management**

Oxygenation supplementation should be provided until it is proven that it is not required.

Thoracentesis is performed in patients with signs suggestive of pleural filling defects. If the thoracentesis has to be repeated frequently or if a pleural effusion is present a thoracostomy tube and intermittent or continuous chest drainage may be warranted. If the patient is hypovolemic secondary to a hemothorax, fluid resuscitation will be required.

Treatment for diaphragmatic hernia should be as for any patient in respiratory distress. Surgical reduction and repair is the definitive therapy. Some debate exists whether surgical repair should be performed as soon as possible or can be delayed until the patient is stable. Immediate surgery is commonly recommended when the stomach is in the chest. Patients with acute diaphragmatic hernia should be monitored continuously for respiratory compromise and clinical deterioration.

Rarely is surgical intervention required for flail chest. Oxygen therapy and pain control are essential. In those cases where the flail chest hinders ventilation mechanical ventilation will be required.

Therapy for pulmonary contusions is supportive in nature with the goal of maintaining adequate ventilation and oxygenation. Fluid therapy should be administered to correct perfusion deficits and over hydration should be avoided. Monitoring with central venous pressure measurements and careful and frequent auscultation of the chest may help in tailoring fluid therapy requirements. Mechanical ventilation may be required in some patients with severe pulmonary contusions and respiratory compromise.

Ventricular arrhythmias are a sequela of myocardial contusions. Anti-arrhythmics such as lidocaine are administered if the patient is cardiovascularly compromised. In
addition, the patient is assessed and treated for extracardiac influences on cardiac conduction disturbances (acidosis, hypovolemia, hypoxemia, hypokalemia and pain).

**Monitoring**

Several questions are asked when monitoring the respiratory system. Is the rate and tidal volume adequate; is the breathing effort smooth and easy or labored; is the breathing pattern regular? Can normal breath sounds be ausculted? Abnormal breath sounds could be described as crackles, wheezes, squeaks, muffled and quiet. Is the patient able to meet its ventilation and oxygenation requirements? Arterial blood gases (ABG) are an excellent way to assess ventilation and oxygenation. PaCO$_2$ tell how well the patient is ventilating. PaO$_2$ tells how well the patient is oxygenating. Pulse oximetry, is a non-invasive technique that continuously measures arterial oxygen saturation of the blood. In some instances repeated imaging will be required to assess the status of the thoracic injury.

**Hypovolemia**

Trauma has multisystemic effects, one of which is hypovolemic shock. Hypovolemic shock is a decreased circulating blood volume.

**Pathophysiology**

Usually some initiating cause (blood loss, severe dehydration or maldistribution of body fluids) results in a decreased circulating volume. The initiating cause results in a decreased venous return, which leads to a decreased stroke volume, decreased cardiac output and finally a decreased blood pressure and oxygen delivery. In response the body activates the sympathetic nervous system (SNS) causing a release of epinephrine and nor -epinephrine, the end result is an increase in cardiac output. In addition to the activation of the SNS stimulation the body releases rennin /angiotensin and aldosterone as well as vasopressin. These hormonal responses cause increased retention of fluids. Fluids also shift from the interstitium in an effort to increase circulating intravascular volume. The end result is increased cardiac output, increased circulating blood volume and increased tissue perfusion and oxygen delivery.

**Patient Assessment**

Does the history or physical findings support evidence of blood loss? Many of the signs that we see suggestive of hypovolemic shock are a result of a compensatory sympathetic reflex. Clinical signs include: tachycardia, pale or grey mucous membranes, prolonged capillary refill, poor pulse quality, decreased mentation, cool extremities, and decreased urine production.

**Patient Management**

Oxygen therapy is initiated, hemorrhage is controlled and fluid therapy is initiated.

Fluid resuscitation improves inadequate tissue perfusion. Options for fluid resuscitation include crystalloids (Lactated Ringers, Normosol R, Plasmalyte 148, and Normal Saline), and Colloids (Plasma, Dextran 70, Hetastarch, and whole blood). Initially,
crystalloids are the fluid of choice in the treatment of hypoperfusion. The dose is 80 - 90 ml/kg and 50 - 55 ml/kg in the dog and cat respectively (equivalent to one blood volume). It may be necessary to use 0.5 - 1.5 times the blood volume to resuscitate the patient. 90 % of the crystalloids shift from the intravascular space into the interstitial space in about thirty minutes.

Hypertonic saline (7.5% Sodium Chloride Injection USP: Sanofi Animal Health) has been recommended for use in hypovolemic shock therapy in cases where it is difficult to administer large volumes of fluids rapidly enough to resuscitate the patient. Hypertonic saline causes fluid shifts from the intracellular space to the extracellular (including intravascular) space resulting in improved venous return and cardiac output. Hypertonic saline also causes vasodilation and improves tissue perfusion. The recommended dose range is 4 - 6 ml/kg over five minutes. Dextran 70 has been added to hypertonic saline to potentiate and sustain vascular augmentation.

Colloids are classified as either synthetic (Dextran 70, Hetastarch or Oxyglobin® or naturally occurring (Plasma, whole blood or packed RBCs). Colloids are better blood volume expanders, 50 - 80% of the infused volume remains in the intravascular space. Colloids should be administered when crystalloids are not improving or maintaining blood volume restoration. Colloids should be administered when the total protein or albumin are decreased below 4.0 g/dl or 1.5 g/dl respectively. Plasma provides albumin, immunoglobulins platelets and clotting factors. Large volumes of plasma may be required to affect total protein or albumin concentrations. The approximate dose of plasma is 10 - 40 mL/kg; however, it should be administered to effect. Dextran and hetastarch may be given as a bolus of 10 - 40 mL/kg to effect. Because the synthetic colloids only replace intravascular volume, crystalloids still must be given to replace interstitial fluid deficits. Crystalloids are given at fifty percent of the dose had crystalloids been used alone.

Hemoglobin must be available in sufficient concentrations to insure adequate oxygen content. If hemoglobin decreases from 15 gm/dl to 10 gm/dl, oxygen content is reduced by one third; cardiac output will need to increase in order to maintain adequate oxygen delivery. In the absence of hemoglobin measurements, hemoglobin can be estimated from the micro-hematocrit. The hemoglobin is usually about one third the hematocrit value. The hematocrit should be maintained around twenty-five percent. Oxygen delivery is limited when the hematocrit decreases below twenty percent. Whole blood and packed red blood cells are administered at 10 - 30 ml/kg and 5 - 15 ml/kg, respectively; again, this will need to be administered to effect. These doses will increase the hematocrit approximately five to fifteen percent. Oxyglobin® solution is an alternative to whole blood or packed red blood cells. Oxyglobin® is a hemoglobin-based oxygen carrying solution. It contains 13 g/dl bovine polymerized hemoglobin in a modified Lactated Ringer’s solution. Due to its molecular size it exerts a colloid oncotic pressure effect, which may be beneficial. The manufacturer recommended one time dose range is 10 - 30 ml/kg not to exceed a rate of 10 ml/kg/hr. The patient’s condition should be monitored and the fluid given to effect. Administration of hemoglobin (either whole blood, packed RBCs or Oxyglobin®) improves O2 content and ultimately oxygen
delivery.

Sympathomimetcs, such as dopamine and dobutamine are indicated when the patient is unresponsive to vigorous fluid therapy and arterial blood pressure, vasomotor tone, and tissue perfusion have not returned to acceptable levels. These drugs support myocardial contractility and blood pressure with minimal vasoconstriction. If these drugs are used blood pressure monitoring is necessary. Sympathomimetics should not be a substitute for adequate volume restoration.

**Monitoring**

Physical exams are repeated as frequently as dictated by the patient’s condition. Assessment of the cardiovascular system may begin with the heart rate. The heart rate is a nonspecific parameter; there are several reasons for tachycardia and bradycardia. If arrhythmias are auscultated then an ECG is indicated. The ECG measures electrical activity; it does not measure mechanical activity. Indicators of peripheral perfusion include: heart rate, pulse quality, mentation, mucous membrane color, capillary refill time, urine output and appendage temperature. A full strong pulse indicates a good pulse pressure and stroke volume. Arterial blood pressure is the product of cardiac output and systemic vascular resistance. Normal systolic, diastolic, and mean blood pressure are approximately 100-160, 60-100, 80-120 mm Hg respectively. Central venous pressure (CVP) measures the relative ability of the heart to pump the quantity of blood returned to it. CVP is also an estimate of the relationship between blood volume and blood volume capacity. It can be an indirect indicator of preload. Normal CVP ranges 0-10 cm H$_2$O. CVP measurements require the placement of a jugular catheter, which lies in the anterior vena cava and attachment to a water manometer or transducer. A CVP of < 0 Cm H$_2$O suggest vasodilation or hypovolemia.

**Head Trauma**

**Pathophysiology**

Primary brain trauma is the physical disruption of intracranial structures that occurs immediately at the time of the traumatic event. The injury may be surface mechanical and / or shear injury. Once the injury has occurred nothing can be done to change it. Secondary brain injury is the activation of multiple inflammatory cascades. Activation of inflammatory cascades may result in edema, hemorrhage, increased intracranial pressure (ICP), decreased tissue perfusion, and / or neuronal cell death.

ICP is the pressure exerted by the brain, blood and CSF in the cranial vault. An increase in any one volume must be accompanied by a decrease in another so as to minimize any increase in intracranial pressure. This is called the Monro-Kellie doctrine.

Cerebral perfusion pressure (CPP) is the principle determinate of cerebral blood flow, brain oxygenation and nutritional support. It is calculated by CPP = MAP - ICP. One of the major contributors to CCP is mean arterial blood pressure (MABP). As MABP rises, cerebral vasoconstriction prevents an increase in ICP. As MABP falls, vasodilation
occurs, vasodilation prevents ICP decrease. This is called pressure autoregulation. CPP compromise results in ischemic death of brain tissue

Chemical autoregulation refers to the responsiveness of cerebral vasculature to \( \text{PaCO}_2 \); increased \( \text{PaCO}_2 \) causes vasodilation and decreased \( \text{PaCO}_2 \) causes vasoconstriction. Cerebral vasodilation contributes to increased cerebral blood flow and increased ICP.

**Patient Assessment**

The physical examination will focus on neurological parameters. The following parameters suggest neurological deficits: decreased levels of consciousness (Obtunded, stuporous and comatose), pupillary light reflex (Pupils that are poor to non-responsive with unilateral or bilateral mydriasis, bilateral miosis, or pupils that are miotic and become mydriatic), abnormal posture (Shiff Sherrington, decerebellate, and decerebrate); and neurologic breathing patterns (Biots, Cheyne stokes, central neurogenic hyperventilation and apneustic), inappropriate response to painful stimuli.

If the facility has the capabilities, imaging is beneficial once the patient is stable. Computed tomography is superior to magnetic resonance imaging for examining bone and identifying areas of acute hemorrhage and edema. Radiography rarely provides useful information except when depressed skull fractures are present.

**Patient Management**

The patient should be treated for hypovolemia if present, fluid therapy should be conservative but adequate. Osmotic therapy such as mannitol (0.25 – 0.5 g/kg (over 10 – 20 minutes)) should be administered if indicated. Events (increased blood pressure, hypercapnia, and hypoxemia) or drugs (alpha 2-agonist, ketamine, and inhalational anesthetics) that increase intracranial blood flow should be avoided. Events that decrease intracranial outflow (Jugular vein obstruction, coughing, aggressive ventilation, and a head-down position) are avoided. Maintain normal temperature and control seizures.

**Monitoring**

The same neurological parameters that are used in the initial assessment are continued in the monitoring phase. The patient is observed for signs suggestive of increasing intracranial pressure. The signs include: deterioration of level of conciseness, change in resting pupil size and loss of pupillary light reflex and the sudden appearance of dilated unresponsive pupils. Intracranial pressure monitoring may be used if available. Other physiological parameters are assessed such as blood pressure, arterial \( \text{PaO}_2 \) or \( \text{SPO}_2 \) and \( \text{PaCO}_2 \) or end-tidal \( \text{CO}_2 \). Electrolytes and osmolality are evaluated with repeated mannitol administration.

**Abdominal Trauma**

Hemo and uroperitoneum are perhaps the more common intra-abdominal injuries. Bilary tract rupture is rare.
Pathophysiology
Injuries to the abdomen are primarily penetrating or blunt trauma. Penetrating injuries such as projectiles or stab are more readily visible. The path of the injury is frequently visible and can help localize the possible injured organs. Penetrating injuries may cause bleeding from major vessels or solid organs or perforation of a segment of bowel.

Blunt trauma to the abdomen results in compression and shear injuries. Compression injuries occur when the abdomen is crushed between solid objects. Shear injuries occur when there is the rupture of solid organs or vessels in the cavity because of the shearing or tearing forces exerted against their stabilizing ligaments or vessels. Pelvic fractures can cause bladder or urethral injuries.

Large hemorrhage into the abdomen can be the major reason for the development of hypovolemic shock. Perforation of the gastrointestinal tract can result into the development of peritonitis.

Assessment
Knowledge of the mechanism of injury is helpful in the assessment of intra-abdominal trauma. Palpation can reveal abdominal tenderness, splinting and/or distension. It is helpful to clip the fur over the abdomen in order to visualize abdominal bruising. The abdomen should be observed for distension, contusion, abrasion, penetration, evisceration, impaled objects and/or obvious bleeding. These are all signs suggestive of underlying bleeding. Intra-abdominal hemorrhage should be considered when there is an unexplained reason for hypovolemic shock.

Abdominal centesis or diagnostic peritoneal lavage can aid in the diagnosis of intra-abdominal bleeding, peritonitis, bladder rupture, and abdominal contamination. If this diagnostic tool is to be utilized it should be done following imaging so as not to introduce gas into the abdomen. Analysis of fluid recovered from a diagnostic peritoneal lavage will aid in the diagnosis of uroperitoneum. Serum and lavage fluid, creatinine/potassium levels are compared. If the lavage sample exceeds the serum sample that is diagnostic for uroperitoneum.

Imaging can be extremely helpful in diagnosing intra-abdominal trauma. Radiographs with or without contrast are beneficial in diagnosing displaced or ruptured organs, masses, peritoneal effusion, and free gas. Once uroperitoneum is diagnosed, positive contrast studies of the urinary tract are warranted. Excretory urograms will assess the integrity of the kidneys and ureters while positive contrast studies of the urethra and urinary bladder provide more detailed evaluation of the lower urinary tract.

Ultrasound, computed tomography and magnetic resonance have all been used to assess abdominal trauma.

Patient Management
Hemoperitoneum is one of the more common serious complications from blunt trauma to the abdomen. Hemoperitoneum should be considered in any trauma patient that
presents in shock without signs of external hemorrhage or thoracic cavity abnormalities. Severe bleeding usually occurs from the spleen or liver although avulsion of the kidney and other organs may cause substantial hemorrhage. As in all hypovolemia cases fluid therapy is the treatment of choice. Fluids should be given in sufficient quantities to support and maintain tissue perfusion but not create a hypertensive state. The fear being that the hypertension could aggravate the abdominal bleeding. Abdominal counter pressure may be used to arrest or control hemorrhage. If this technique is employed care and consideration should be given not to interfere with breathing owing to the increased abdominal pressure on the diaphragm. Abdominal wraps are contraindicated in the diaphragmatic hernia patient; there is the potential that more abdominal organs may be forced into the chest. The decision to go to surgery is difficult. In those cases where free gas is evident on radiographs immediate surgery is necessary. Surgical repair and generous lavage is the treatment of choice when uroperitoneum is diagnosed.

**Monitoring**

Because hypovolemia is one of the more common problems encountered with abdominal trauma, those parameters that are monitored during resuscitation should continue. A urinary catheter may be placed to give a better idea of urine production. Abdominal girth should be observed. The patient should be observed for signs suggestive of pain. Depending upon the type of abdominal trauma, the patient may be at risk for the development of a systemic inflammatory response (SIRS). SIRS requires a high index of suspicion and therefore should be a consideration in the monitoring of the patient. CBC, serum chemistry, electrolytes and blood gases are repeated as needed.

**Musculoskeletal**

**Pathophysiology**

The musculoskeletal system is a very large system and prone to many types of trauma related injuries. There are perhaps 5 major injuries that can involve the musculoskeletal system. These include hemorrhage, instability (fractures and dislocations), loss of tissue (avulsion and amputation), simple lacerations, and interruption of blood supply.

**Assessment**

Initial examination of the musculoskeletal system entails observation and palpation. Deformity, bleeding, guarding, and pain may be indicators of musculoskeletal injury. Spinal injuries can be causes of neurological deficits. A neurological examination will need to be performed to localize lesions. Care should be exercised so as not to cause further trauma when assessing the patient.

Radiographic imaging will provide a definitive diagnosis of skeletal problems.
Management
The basic premise of musculoskeletal management in the emergency patient comprises the immobilization of the spine or limb to prevent further injury and the appropriate treatment of open wounds.

Those patients that are unable to rise following trauma or are displaying opisthotonic postures should be treated as if they are spinal trauma patients. If possible they should be immobilized on a backboard until the status spinal status is known. Where possible, temporary immobilization of the extremity fractures is undertaken to: prevent motion of the bone fragments that could damage muscles, nerves and blood vessels, or cause skin laceration (allowing a closed fracture to become open); control excessive bleeding; and minimize pain.

Until definitive care can be provided, a sterile dressing is applied to any open wounds. The wound should remain covered in the hospital environment; this procedure decreases the risk of bacterial contamination and hospital-acquired infection. Further wound treatment is undertaken after patient stabilization.

Monitoring
The patient is assessed and treated for pain. If an immobilization device is utilized on an extremity the patient’s toes should be checked for warmth and swelling. Abnormal smells emanating from bandages or splints are investigated. Soft tissue injuries should be observed for bleeding, swelling, and / or redness.