HOW I TREAT THORACIC TRAUMA

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Thoracic injury in dogs and cats is a common cause of death following trauma. Among dogs involved in motor vehicle accidents, 31 to 39% sustain thoracic injuries, whereas thoracic trauma occurs in 39% of cats. Other types of trauma associated with thoracic injuries include gunshot wounds, bite wounds, and falling from heights. Common thoracic injuries include pleural space lesions (pneumothorax, hemothorax), chest wall injuries (rib fractures, flail chest), diaphragmatic hernia, mediastinal injuries, cardiac injury, and pulmonary injury. Early recognition of thoracic trauma through clinical signs, radiography, and blood gas analysis will help animals receive appropriate therapy.

PNEUMOTHORAX

Pneumothorax occurs in 13 to 50% of all traumatic thoracic injuries. Traumatic pneumothorax primarily occurs from blunt force injury that suddenly increases intrathoracic pressure and ruptures pulmonary parenchyma. Less commonly, penetrating injury or fractured ribs will lacerate the lung, trachea, or bronchi.

Pneumothorax can be classified as open, closed, or tension. Open pneumothorax has a communication between the pleural space and the external environment. Closed pneumothorax occurs from leakage through the pulmonary tissues. Tension pneumothorax results from injured soft tissues of the thoracic wall or lung creating a one-way valve that traps air in the pleural space. Increasing intrathoracic pressure severely compromises ventilation and venous return, quickly resulting in shock then death.

The degree of respiratory compromise depends on the amount of lung collapse and concurrent pulmonary injury. Pneumothorax is usually tolerated if there is less that 50% collapse and no other underlying injury or pulmonary pathology. The typical breathing pattern is rapid and shallow. Physical examination reveals dorsally decreased respiratory sounds and muted heart sounds.

Emergency treatment of pneumothorax is initiated on animals in respiratory distress without radiographic confirmation. Radiographs are performed once the animal is stable. Typical radiographic signs of pneumothorax on lateral views include elevation of the cardiac silhouette away from the sternum, visualization and retraction of visceral pleural margins away from the thoracic wall, and increased lung lobe opacity due to atelectasis.

The goal of treatment of traumatic pneumothorax is to remove the extrapulmonary, intrapleural air to allow reexpansion of the lungs. Thoracocentesis is typically performed initially. This is performed by using a 20- to 22-gauge needle that is connected to an extension set, three-way stopcock, and a 60 mL syringe. The needle is inserted cranial to the 8th rib at a 45-degree angle with the bevel facing the thoracic wall. Both sides of the thorax are aspirated. If air retrieval is continuous or if the animal requires multiple aspirations to alleviate clinical signs, a thoracostomy tube is indicated.

Thoracostomy tubes are utilized for drainage of pneumothorax or pleural effusion, or tubes are placed following thoracic surgery to reestablish negative pressure within the thoracic cavity. Thoracostomy tubes can also be used to lavage the pleural cavity in cases of pyothorax. Local analgesics can be administered through the tubes to provide postoperative pain relief following thoracotomy. Either red rubber feeding tubes or commercially available tubes with trocar stylets can be utilized for thoracic drainage. The size of the tube is dependent on the size of the animal. Typically the tube diameter approximates the size of the mainstem bronchus. Tubes may be placed unilaterally or bilaterally depending on the underlying cause.

Tubes placed for pneumothorax can be removed once negative pressure has been established and maintained for 12 to 24 hours. Complications of thoracostomy tubes include kinking, clogging, inadvertent removal, and risk of ascending nosocomial infections.

Technique:

1. Place the animal under general anesthesia. If the animal is too unstable to tolerate anesthesia, heavy sedation and local analgesia may be used.
2. Perform a standard clip and prep of the lateral thorax.
3. Place additional holes in the tube to facilitate drainage. The holes should not be larger than 1/3 of the circumference of the tube. The last hole should be through the radio-opaque line found on most commercial tubes.
4. Slide the skin on the lateral thorax cranially and then make a small skin incision in the dorsal one-third of the thorax at the level of the 9th to 11th intercostal rib space.
5. If using a trocar tube, use the trocar to make a subcutaneous tunnel in a cranioventral direction spanning at least two intercostal spaces.
    a. The trocar tube is grasped with one hand close to the body wall. The other hand gently pops the tube into the pleural cavity using gentle even pressure.
    b. The trocar is slightly retracted to avoid any injury to underlying structures, but it is used as a stylet to facilitate proper positioning of the tube along the ventral aspect of the pleural cavity.
6. If using a red rubber tube, the subcutaneous tunnel is made using a curved Rochester-Carmalt forceps.
    a. Clamp the red rubber feeding tube with the jaws of the forceps and use the tips of the forceps to puncture into the pleural cavity. Feed the tube into the pleural cavity.
7. The tube is secured using a finger-trap suture (see below). A horizontal mattress suture can be placed under the tube in the subcutaneous tunnel to minimize leakage.
8. Connect to the tube to a Christmas tree adapter and then to a three-way stopcock. These connections can be secured using wire or quick-ties.
9. Thoracic radiographs should be performed to confirm proper placement.

Securing Indwelling tubes

The most common method to secure indwelling tubes to the skin is to use a finger trap suture. This suture is may also be called a Roman sandal suture. Examples of such tubes that could be secured in this manner include esophagostomy tubes, gastrostomy tubes, jejunostomy tubes, thoracostomy tubes, peritoneal dialysis catheters, closed suction drain tubes, and urinary catheters.

Technique:

1. Use a large gauge monofilament nonabsorbable suture on a cutting needle (e.g., 2-0 polybutester).
2. Anchor the suture to the skin using a simple interrupted suture or a purse-string suture around the exit site of the tube. Be sure to leave the free ends of the suture long.

3. Wrap the suture around the tube and place a surgeon's throw. Cinch the suture tightly down on the upside of the tube. Alternatively, place a square knot instead of a surgeon's throw, particularly if the tube is quite stiff.
4. Pass and cross the ends of the suture underneath the tube and then place another surgeon's throw as described above.
5. Repeat steps 3 and 4 at least four times.
6. Once finished, complete the finger-trap by throwing an additional three throws.

Thoracostomy Tube Alternatives

For animals with chronic effusion, there are alternative to traditional large-bore trocar or red rubber thoracostomy tubes. The use of small-bore wire-guided chest tubes in veterinary patients has been described. In human medicine, the use of smaller tubes is associated with less insertional and infectious complications. These tubes are placed using a modified Seldinger technique.

Technique:

1. Animals are sedated or placed under general anesthesia, depending on the temperament of the animal and severity of respiratory compromise.
2. A small stab incision is made in the skin at the dorsal level of the 9^{th} or 10^{th} intercostal space.
3. An introducer catheter (18 or 20 gauge) is tunneled from the skin incision through the subcutaneous tissue cranially, and inserted into the thoracic cavity at the 7^{th} or 8^{th} intercostal space.
4. A J-wire is inserted through the catheter and advanced into the thoracic cavity in a cranioventral direction.
5. The catheter is removed, leaving the wire in place.
6. A 14-g chest drain (Mila International, Inc.) is inserted into the thoracic cavity over the guide wire, and the guide wire is removed.
7. The drain is secured to the skin using the preplaced suture holes on the end of the chest drain.

The use of modified vascular access ports (VAP) in conjunction with multifenestrated drain tubing (e.g., Jackson-Pratt drain) has also been described as an alternative method for managing chronic pleural effusion. More recently, a commercial device has become available (PleuralPort ™, Norfolk Vet Products, Skokie, IL) for management of pleural effusion in both dogs and cats. The device consists of a multifenestrated radio-opaque silicone thoracostomy tube that is connected to a drainage hub that is placed outside the thorax in the subcutaneous tissue. The hub is accessed percutaneously using a Huber point (non-coring) needle to allow for repeated thoracic drainage. Reported advantages of this device include increased comfort, decreased risk of infection, decreased risk of complications associated with repeated thoracocentesis, decreased risk of accidental removal, and potential management of thoracic effusion in a home environment.

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HEMOTHORAX

Hemorrhagic pleural effusion occurs in approximately 10% of animals sustaining thoracic trauma and can occur with both blunt and penetrating trauma. This is a result of lung laceration or injury to major vascular structures. Hemothorax may also occur due to erosion of large vessels wall secondary to local tumor invasion. Considerable hemorrhage into the thoracic cavity usually results in death, as the animal will bleed out before receiving emergency care.

Lung laceration hemorrhage is usually clinically insignificant as the hemorrhage is self-limiting. However, damage to large arteries or veins, particularly the intercostal vessels, causes profound hemorrhage. These animals will present in hypovolemic shock with tachycardia, pale mucous membranes, and decreased capillary refill time. Circulatory compromise precedes respiratory compromise from hemothorax, as ventilation impairment will not occur until 30 ml of blood per kilogram has accumulated in the pleural space. Hypovolemic shock will have occurred by this point.

Auscultation of patients with hemothorax reveals muffled heart and lung sounds. A fluid-gas interface may be identified if concurrent pneumothorax is also present. Thoracocentesis is the diagnostic tool of choice as these animals are often too unstable to allow radiographic examination. Blood in the thoracic cavity is quickly defibrinated by the constant motion of the lungs and therefore will not clot after aspiration; although subacute massive hemorrhage may clot as the blood is accumulating too rapidly. The packed cell volume of the aspirated fluid is typically equal to or above the animal's peripheral packed cell volume.

Small amounts of thoracic hemorrhage are typically clinically insignificant. However, more significant hemorrhage requires aggressive treatment. Drainage of the pleural cavity is performed through thoracostomy tube placement as needle aspiration will not drain the area fast enough and the blood may clot the needle if there is peracute hemorrhage. Volume replacement must also be instituted to address the hypovolemic shock. Transfusion is indicated if greater than 20 to 30 ml of blood per kilogram is retrieved from the chest and if the packed cell drops acutely below 20%. If hemothorax is a result of trauma, autotransfusion is a reasonable treatment option, especially if blood products are in short supply or not readily available. Although there are several methods of autotransfusion, direct aspiration and reinfusion is most appropriate in an emergency situation. Blood is aspirated though IV tubing and 3-way stopcock to a 60 ml syringe. The blood is then pushed into a blood collection bag and returned to the patient through a blood filter set. Complications of autotransfusion include hemolysis, coagulopathy, and sepsis.

Exploratory thoracotomy is rarely indicated for hemothorax as the source of hemorrhage is rarely found or the animal is too unstable to survive the procedure. However, if the animal is not responsive to replacement therapy or if intrathoracic blood loss is continuous, surgery may be the only treatment option left.

DIAPHRAGMATIC HERNIAS

Diaphragmatic hernias can be congenital or traumatic in origin, and acute or chronic in duration. The degree of respiratory compromise depends on the amount of abdominal viscera displacement, and the presence and severity of other thoracic injuries or conditions. Animals may display respiratory distress, tachypnea, tachycardia, and cyanosis. Cardiac dysrrhythmias are common due to the irritation of the heart by abdominal viscera. Dogs and cats with congenital or chronic diaphragmatic herniation may not be clinical or have only mild increases in respiratory rate and effort.

A large percentage of injured animals do not outwardly demonstrate the clinical signs connected with thoracic injury. A potentially devastating mistake is to presume there is no thoracic injury following trauma based on the lack of clinical signs and presence of injury only in caudal half of the body. The thoracic wall is an inherently resistant structure and it is often spared from significant injury even if there are significant injuries to the internal thoracic structures. Also, there is no correlation between the pattern of appendicular injury and the existence of thoracic trauma.

The creation of an abdominal-thoracic pressure gradient across the diaphragm at the time of impact dictates the nature of the injury to the thorax. If abdominal pressure is greater than thoracic, a diaphragmatic tear results. It has been classically thought that herniation occurs if the glottis is open at impact. A more balanced gradient across the diaphragm with a closed glottis places stress on the lung parenchyma resulting in a pneumothorax. However, both pneumothorax and diaphragmatic herniation can occur simultaneously. Diaphragmatic tears typically occur through the muscular portion, which is the weakest part of the diaphragm. The location of the diaphragmatic tear depends on the location of the viscera at the time of injury; the area least protected by abdominal organs is typically the area that ruptures. Abdominal organ displacement into the thoracic cavity depends on the location and size of the tear. Almost any organ can herniate, although the liver is the most frequent followed by the small intestine, stomach, and spleen.

Diagnosis

Diaphragmatic hernias may go undetected following trauma and may not be diagnosed until days to weeks after injury. Animals with respiratory compromise may display dyspnea, tachypnea, tachycardia, and cyanosis. Cardiac dysrrhythmias are common due to the irritation of the heart by abdominal viscera.

Thoracic radiography may demonstrate obvious organ herniation in the thoracic cavity. Radiographic signs include loss of diaphragmatic outline and cardiac silhouette, displacement of lung fields, presence of gas filled viscera, and pleural effusion. Effusion is usually associated with liver entrapment and venous occlusion. Abdominal radiographs may demonstrate cranial displacement of abdominal organs. Identification of stomach or intestines within the thoracic cavity makes the diagnosis of diaphragmatic hernia uncomplicated. However, if there is a large amount of pleural fluid or if the soft-tissue parenchymal organs are herniated the diagnosis of diaphragmatic hernia may be less obvious. Repeating radiographs following thoracocentesis may identify an underlying cause that was not apparent before. Performing all radiographic views (right lateral, left lateral, ventrodorsal, and dorsoventral) may shift herniated viscera and allow better visualization.

Additional diagnostic imaging procedures can be used to aid confirmation of a diaphragmatic hernia. Unconventional radiographic imaging when compared with routine survey radiographs may provide valuable information. Such views include horizontal beam projection and standing ventrodorsal projections. Upper gastrointestinal positive contrast studies using orally administered barium sulfate will show the location of the stomach and intestines. However if a large amount of pleural fluid is present, water-soluble iodinated contrast media is suggested to potentially avoid barium contamination of the pleural or peritoneal cavity due to possible gastrointestinal perforation.

Ultrasonography can also be used to diagnose diaphragmatic hernia. The diaphragm itself is not readily visualized even in normal animals. Rather, the interface between air-filled lung and hyperechoic liver that identifies the location of the diaphragm. A straightforward diagnosis occurs when there is identification of abdominal viscera next to the cardiac silhouette. Disruption of the lung-liver interface may contribute to information gathered from survey radiography to help diagnose a subtle diaphragmatic hernia. A common normal ultrasonographic artifact when imaging the diaphragm through the liver is the appearance of liver on both sides of the diaphragmatic interface. This mirror image occurs due to the extremely reflective nature of the lung-liver interface.

Positive contrast peritoneography can also be used if suspicion of diaphragmatic herniation is high, but cannot be confirmed from the previously described studies. Water-soluble iodinated contrast media is injected into the peritoneal cavity. Translocation of the contrast into the thoracic cavity confirms disruption of the diaphragm.

Emergency Treatment

Landmark retrospective studies found mortality associated with diaphragmatic hernia is higher when surgery is performed less than 24 hours or more than 1-year after injury. However, more recent studies did not find an effect of the duration of the hernia on overall outcome. Anesthesia and surgery should ideally be delayed until the animal can be adequately stabilized. However, emergency surgery is indicated is there is gastric herniation or if the animal respiratory status deteriorates. Gastric herniation is a surgical emergency as these animals are at risk for acute gastric distension, severe respiratory compromise, and death.

Surgical Treatment

The goals of surgery are to reduce the herniated organs back into the abdominal cavity, examine the organs for any vascular compromise or perforation, and repair the diaphragmatic defect. Occasionally the diaphragmatic tear needs to be enlarged or the incision extended cranially through the sternum to allow reduction of abdominal contents and improve visualization. Tears are sutured from dorsal to ventral using an absorbable or nonabsorbable monofilament suture in a simple continuous pattern. Use care when suturing near the caval, esophageal, or aortic foramina. If the diaphragm has been avulsed from its thoracic wall insertions, incorporate the ribs into the closure. Defects too large to close are rarely encountered; however, if faced with this situation autogenous flaps or synthetic implants may be used. Autogenous flaps include a sliding transverse abdominus muscle flap or an omental pedicle flap. Examples of synthetic materials include polypropylene mesh and silastic sheeting. Following repair of the diaphragm, air is removed from the thoracic cavity by needle thoracocentesis or placement of a thoracostomy tube.

Prognosis

If the animal survives for the first 12 to 24 hours after surgery, the prognosis is excellent. Reported survival rates range from 80 to 90% following surgical correction. Deaths are most often due to concurrent injury. Re-expansion pulmonary edema is an uncommon but very serious complication. It occurs in animals with chronic herniation where the atelectatic lungs are rapid reinflated. Pulmonary edema occurs as capillary integrity has been altered due to an anoxic environment in the atelectatic lung. Reperfusion of damaged vessels directs fluid in to the interstitium. Although this is an uncommon complication, it most often occurs in cats with chronic herniation. Ways to prevent this complication is to avoid aggressive positive pressure lung expansion during anesthesia once the hernia has been reduced and to avoid aggressive removal of air through the thoracostomy tube in the postoperative period. The animal should be allowed to gradually reinflate their lungs on their own. Other major complications include pneumothorax, hemothorax, liver lobe necrosis, gastrointestinal vascular compromise, and reherniation.

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