“Thoracic Trauma? What Could Possibly Go Wrong!”

With Dr Philip Judge

BVSc MVS PG Cert Vet Stud MACVSc (Veterinary Emergency and Critical Care; Medicine of Dogs)

July 2013

Vet Education is proudly supported by Hill’s Pet Nutrition (Australia) and Merial Animal Health
Injuries to the thorax and thoracic structures are very common in traumatised dogs and cats. Because of the vital importance that normal thoracic cage, pulmonary and cardiovascular systems play in the well-being of the patient, it is not surprising that thoracic trauma is a significant cause of both morbidity and mortality in the traumatised patient. It is therefore critical that thoracic injuries be managed appropriately, and in a timely manner, to reduce the potential for serious consequences in the patient.

**What Happens in Thoracic Trauma?**

To fully understand the potential for injury to the thorax following trauma, it is important to consider the forces and mechanics involved in the injury itself. To do this, we must review three things – kinetic energy, Newton’s first law of motion, and the law of conservation of energy. Let’s consider these individually...

1. **Kinetic energy.** Kinetic energy is the energy of motion. Whenever two objects collide, each of them will have an amount of energy, determined by the formula kinetic energy \( K = 0.5 \times \text{object mass} \times v^2 \).
   
   From this equation, we can tell two things about the energy involved in collisions in thoracic trauma. Firstly, the larger the mass is that is colliding with the patient, the greater the energy. Secondly, the faster the masses collide, the greater the energy — i.e. speed kills.

2. **Newton’s First Law of Motion:** “A body in motion stays in motion unless acted on by an outside force; a body at rest stays at rest unless acted on by an outside force”.
   
   This law is self-explanatory in trauma — our patients do not suffer trauma unless acted on by an external force e.g. a car, a dogs’ jaw etc. In addition, for a dog, for example that is hit by a car, the motion in the patient induced by that car will continue until the patient lands on the ground or comes to rest.

3. **The Law of Conservation of Energy:** The law of energy conservation states that “energy cannot be created or destroyed – it can only be changed from one form to another”.
   
   This law is critical to our patients. It states that the energy will be transferred from one object to another, following a collision — and that the energy transferred will be in the form of motion, absorption into tissues, sound and propulsion, among other forms.

**Why does this have significance for our patients?**

- When a moving body (the patient) is acted on by an outside object (car, dog jaws, bullet etc.) that changes its motion, the kinetic energy involved in that collision must change to some other form of energy.
- If the moving body is a dog or cat, and the energy transfer of a collision occurs to rapidly, tissues absorbing the energy of the collision become traumatised, and tissue disruption and injury results.
Thoracic trauma is often characterised as being either blunt or penetrating.

- **Blunt thoracic trauma** is the most common type of thoracic trauma, and most often occurs as a result of vehicular trauma. However, animal-animal interactions, human-animal interactions and falls from a height are other common causes of blunt thoracic injury.
- **Penetrating thoracic injury** is somewhat less common, and is usually the result of animal-animal interactions, projectile injury (bullets, arrows etc.) and impalements.

Most serious thoracic injuries are suspected, or identified during the physical examination – making the physical assessment of a patient with known or suspected trauma extremely important. Most thoracic injury will result in some sort of respiratory embarrassment or compromise, making observation of respiratory rate, effort and character vital both in patient assessment, and in helping direct further diagnostic and therapeutic intervention.

Patients with breathing difficulty are frequently distressed, and usually will have the following general signs...

- Tachypnoea (rapid respiration)
- Anxiety
- Restlessness
- Cyanosis, and/or pale mucous membranes
- Mouth breathing
- Coughing

In addition, patients with breathing difficulty often have an exaggeration of inspiratory effort, expiratory effort, or both.

**Appearance of the Patient with Inspiratory Dyspnoea**

The patient with inspiratory dyspnoea typically has the following characteristics

- Lips are frequently drawn back
- The neck is frequently extended. Breathing often becomes worse when the neck is flexed
- Costal margins protrude – meaning that it is easier to see the ribs, especially the caudal ribs (ribs 7-13).
- **Inspiration is extended in time**
- There is frequently increased noise during inspiration. With a stethoscope, this noise will be louder when listening over the trachea than when listening over the chest wall

**Physiology** – these symptoms are most commonly caused by a partial physical obstruction of the airway, or a decreased ability of the patient to expand their lungs due to disease (decreased lung compliance). This reduces lung expansion and results in a small lung volume. Animals try to compensate for reduced airflow into the lungs by increasing respiratory rate, and by increasing inspiratory effort. These efforts generally lead to a worsening of symptoms, as the animal struggles to breathe through a small or obstructed airway.

Note that partial airway obstruction may not give rise to clinical signs until **over 75% of the airway is compromised** – meaning if you see signs of airway obstruction or inspiratory dyspnoea, it is generally pretty serious, and demands immediate action on the part of the veterinary team to prevent patient decompensation.
Appearance of the Patient with Expiratory Dyspnoea

Expiratory dyspnoea is characterized by the following
- Abdominal effort is increased in expiration
- **Expiration extended in time**
- Active expiratory effort required
- Rapid, shallow respiration, with an expiratory grunt is suggestive of pleural space diseases such as pleural fluid accumulation with blood, exudate, chyle, or pleural air.

Appearance of the patient with both Inspiratory and Expiratory Dyspnoea

Inspiratory and expiratory exaggeration has the following characteristics
- A combination of the clinical signs listed above for inspiratory and expiratory dyspnoea
- The presence of an increase in both inspiratory and expiratory effort is suggestive of diseases involving the lung parenchyma, cardiovascular systems, and blood disorders influencing the amount of oxygen bound to haemoglobin in blood. Pleural place disease, lung oedema, central nervous system disease, pneumonia (including aspiration pneumonia), and haemoglobin abnormalities also may result in both inspiratory and expiratory dyspnoea

What about Cyanosis?

Cyanosis – Cyanosis, the presence of a bluish hue to the mucous membranes of the mouth, conjunctival or reproductive tract. **Cyanosis is a late and unreliable sign of hypoxia.** However, the presence of cyanosis is never a good sign, and demands immediate action to secure the patient airway and restore ventilation.
Differential Diagnosis of Respiratory Distress with Thoracic Trauma

The following table summarizes many of the common causes of dyspnoea that may be encountered with, or in association with thoracic trauma, according to the presence of inspiratory dyspnoea, expiratory dyspnoea, or both.

Table 1: Causes of Dyspnoea in the Dog and Cat Found in Association with Thoracic Trauma

<table>
<thead>
<tr>
<th>Inspiratory Dyspnoea</th>
<th>Expiratory Dyspnoea</th>
<th>Inspiratory and Expiratory Dyspnoea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper Airway Obstruction</td>
<td>Pulmonary Parenchymal Disease (diseases of the lower airways and lung tissue)</td>
<td>Pulmonary Parenchymal Disease – all diseases causing expiratory dyspnoea can cause inspiratory difficulty as well</td>
</tr>
<tr>
<td>• Stenotic (narrow) nares</td>
<td>• Pneumonia</td>
<td>Central Nervous System Disease – can alter respiratory pattern, and respiratory drive</td>
</tr>
<tr>
<td>• Elongated soft palate</td>
<td>• Bacterial</td>
<td>• Head trauma</td>
</tr>
<tr>
<td>• Laryngeal paralysis</td>
<td>• Protozoal (rare)</td>
<td>• Seizures</td>
</tr>
<tr>
<td>• Everted laryngeal sacculae</td>
<td>• Fungal</td>
<td>• Drugs/medications e.g. opiates such as morphine</td>
</tr>
<tr>
<td>• Tracheal collapse</td>
<td>• Parasitic</td>
<td>• Toxicities – e.g. snail bait</td>
</tr>
<tr>
<td>• Hypoplastic trachea</td>
<td>• Neoplasia</td>
<td>• Brain tumours</td>
</tr>
<tr>
<td>• Severe bronchitis</td>
<td>• Primary lung neoplasia</td>
<td>• Meningitis</td>
</tr>
<tr>
<td>• Upper Airway Trauma</td>
<td>• Secondary (metastatic) neoplasia</td>
<td>Disorders of the nerve-muscle junction</td>
</tr>
<tr>
<td>o Pharyngeal trauma</td>
<td>• Inflammatory lung disease</td>
<td>in the chest wall</td>
</tr>
<tr>
<td>o Pharyngeal hematoma</td>
<td>• Feline allergic lung disease (feline asthma)</td>
<td>• Snake bite</td>
</tr>
<tr>
<td>o Fractures at the base of the skull (basilar skull fractures)</td>
<td>• Chronic bronchitis</td>
<td>• Tick paralysis</td>
</tr>
<tr>
<td>o Hypoid cartilage fractures</td>
<td>• Allergic bronchitis</td>
<td>• Botulism</td>
</tr>
<tr>
<td>o Laryngeal trauma causing bronchospasm</td>
<td>• Pulmonary infiltrates with eosinophils (PIE)</td>
<td>• Muscle relaxants</td>
</tr>
<tr>
<td>o Tracheal trauma causing bleeding into the airways</td>
<td>• Trauma to the lung – pulmonary contusions</td>
<td>• Neuro-muscular diseases such as myasthenia gravis, and polyradiculoneuropathy</td>
</tr>
<tr>
<td>• Pharyngeal oedema</td>
<td>• Aspiration pneumonia</td>
<td>Pleural Space Diseases</td>
</tr>
<tr>
<td>o secondary to trauma</td>
<td>• Pulmonary thromboembolism</td>
<td>• Pneumothorax</td>
</tr>
<tr>
<td>o insect bites or stings</td>
<td>• Pulmonary oedema</td>
<td>• Pyothorax</td>
</tr>
<tr>
<td>o swelling from increased breathing effort</td>
<td>• Secondary to heart disease (cardiogenic)</td>
<td>• Chylothorax</td>
</tr>
<tr>
<td>• Laryngeal oedema – see causes of pharyngeal oedema</td>
<td>• Secondary to neurological disease e.g. seizures, electrocution</td>
<td>• Haemothorax</td>
</tr>
<tr>
<td>• Infection – infectious nasal cavity disease (“snuffles”), abscesses, fungal disease</td>
<td>• Acute respiratory distress syndrome – present in severe illness such as pancreatitis</td>
<td>• Pleural space neoplasia</td>
</tr>
<tr>
<td>• Neoplasia</td>
<td>Pulmonary Space Diseases – as for inspiratory and expiratory dyspnoea</td>
<td>• Cardiac disease (esp. cats)</td>
</tr>
<tr>
<td>• Foreign body</td>
<td>Rib Fractures, Flail Chest</td>
<td>• Diaphragmatic hernia</td>
</tr>
<tr>
<td>• Neurological disease – results in decreased clearance of mucus, vomit etc. from the airways.</td>
<td>Diseases causing inspiratory and expiratory dyspnoea also cause inspiratory dyspnoea</td>
<td></td>
</tr>
</tbody>
</table>

Regardless of the diagnosis, what you have to do to stabilise these patients is a matter of following five (5) simple steps...
1. Assess Respiratory Rate, Pattern, and Effort
2. Assess and Establish Airway Patency
3. Assess the Evacuate the Pleural Space
4. Assess Effectiveness of Breathing Efforts
5. Provide Effective Respiration

© Vet Education Pty Ltd 2013
A brief summary of how thoracic auscultation can provide a framework for patient diagnostic and therapeutic intervention following thoracic trauma is outlined below...

**Auscultation Algorithm for Acute Thoracic Injuries with Signs of Respiratory Compromise**

- **Thoracic Trauma**
  - Characterise lung sounds as increased or decreased
  - **Decreased lung sounds - suspect pleural space disease**
    - **Pneumothorax**
      - *diagnostic/therapeutic thoracocentesis*
    - **Haemothorax**
      - *Supportive care*
      - *diagnostic/therapeutic thoracocentesis*
    - **Diaphragmatic hernia**
      - *supportive care*
      - *radiograph when stable*
  - **Increased lung sounds - suspect pulmonary contusions/injury**
    - **Evaluate for upper airway/referred airway sounds**
      - *oxygen therapy*
      - *clear upper airways*
      - *Endotracheal intubation*
    - **Suspect pulmonary contusions**
      - *oxygen therapy*
      - *supportive care*

Adapted from Beal MW “Trauma-Associated Thoracic Injury” in Manual of Trauma Management in the Dog and Cat; Drobotz, Beal, Syring (Ed) Wiley-Blackwell (2011)
The Injuries in Thoracic Trauma

A number of thoracic injuries may be encountered in thoracic trauma. We will consider the following injuries specifically in this presentation...

1. Pulmonary Contusions
2. Diaphragmatic Hernia
3. Pneumothorax
4. Rib Fracture and Flail Chest

Pulmonary Contusions

Introduction

Pulmonary contusion is a serious anatomical and physiological lesion of the lung following non-penetrative, compression-decompression injury to the chest wall. The disruption of alveolar-endothelial integrity results in the pathology of haemorrhage and oedema (Roch et al., 2011). A study of humans with pulmonary contusions determined that 80% of human patients with pulmonary contusions also suffered non-thoracic injuries (Fulton 1970) necessitating the clinician conduct a thorough patient evaluation and consideration of pulmonary contusions in patients presenting with injuries following trauma.

Aetiology

Pulmonary contusions result from compressive-decompressive injury to the chest wall. They may be encountered in many different types of trauma, including road traffic trauma impact, dog attack injuries, incidents where the owners stand on a young or small animal, or falling injuries, and other types of injuries.

Pathophysiology

- Pulmonary contusions result in intra, and extra pulmonary haemorrhage. Haemorrhage into the alveoli causes interference with the gas exchange unit, and results in hypoxia, and reflex-mediated increased ventilatory rate and effort mediated via central nervous system chemoreceptors and the respiratory centre in the brain.

- Bronchospasm occurs due to pulmonary trauma. In addition, the presence of fluid in airways reduces airflow within the larger airways and bronchioles. Lung surfactant is diluted, reducing lung compliance, increasing the work of breathing, and leading to atelectasis - further interfering with alveolar ventilation and gas exchange.

- Concurrent traumatic injury to the myocardium, the presence of circulatory shock, and intra-pleural diseases (haemorrhage, effusion, pneumothorax, fractured ribs, diaphragmatic hernia, and flail chest) may also interfere with gas exchange and respiration

- A secondary inflammatory reaction occurs in response to extravasation of blood into the interstitium and alveoli, from concussive trauma (and subsequent damage) to the cells of the lungs, and hypoxia-induced lung damage. It is important to note that extravasation of fluid and cells into pulmonary interstitial and alveolar spaces may result in progressive impairment in gas exchange and related increase in respiratory distress for up to 12-18 hours following trauma.

© Vet Education Pty Ltd 2013
Treatment

- **Oxygen therapy**
  - Oxygen should be supplemented initially by fly-by or oxygen hood or cage, but should be transitioned to intra-nasal oxygen within the first few hours of admission to hospital (except in cases of head trauma or increased intra-cranial pressure) to avoid the potential for excessive inhaled oxygen concentration and oxygen toxicity.
  - Failure to improve oxygen saturation in a patient receiving oxygen supplementation may be an indication for provision of ventilation therapy.

- **Management of Pleural Space Disorders**
  - Drainage of pleural fluid and/or air will reduce lung atelectasis, reduce the work of breathing, and may improve gas exchange in the lung.
  - Stabilisation of fractured ribs, and/or flail chest using external stabilization techniques will reduce the effort required for the patient to breathe. Provision of local anaesthesia in the form of local nerve blocks to intercostal nerves in patients with rib fractures/displacement can ease discomfort of breathing in affected patients.

- **Ventilation assessment and therapy** – Patients with pulmonary contusions may require ventilation assistance if supplementation with oxygen therapy, and other supportive measures do not result in a return to normal SpO₂ or PaO₂. Given that respiratory function may deteriorate over the first 24 hours or so following trauma (owing to the development of an inflammatory reaction in response to tissue trauma within the pulmonary parenchyma), close monitoring of patients during this period is essential in order to detect deteriorating respiratory effectiveness and function, so that timely intervention is possible.

- **Intravenous fluid therapy**
  - Damage to the pulmonary capillary walls results in haemorrhage and extravasation of fluid into the interstitial fluid compartment in the lung, and into the alveoli. In addition, pulmonary tissue damage results in leukocyte chemotaxis and cytokine-mediated inflammatory response, which further increases pulmonary capillary permeability.
  - Administration of appropriate intravenous fluid therapy to patients with pulmonary contusions has long been an area of controversy, owing to concerns about excessive fluid administration raising pulmonary capillary pressures and contributing to worsening of pulmonary oedema. At present, scientific studies offer conflicting evidence regarding appropriate fluid administration. As a result, a case-by-case assessment must be made, following some general guiding principles (outlined below).
  - Clinicians must therefore achieve a balance between limiting pulmonary pressures and providing adequate fluid resuscitation to avoid hypoperfusion complications of other organ systems.
• Administration of large volumes of isotonic crystalloids e.g. lactated Ringer’s solution should be avoided, as they are associated with excessive lung water accumulation and a deterioration of respiratory function and gas exchange.

• Several studies have showing the benefit to mild fluid restriction in patients with pulmonary contusions, although the studies are flawed in many respects, through exclusion of key patient subsets, and treatment criteria.

• A study in 2009 explored the concept of biphasic (early and late) fluid management of patients suffering septic shock complicated by acute lung injury – which has many facets similar to those observed in pulmonary contusions – including the presence of high pulmonary capillary permeability and inflammation. Their study evaluated the relationship between adequate initial fluid resuscitation (AIFR), where patients received an initial fluid bolus corresponding to a positive fluid balance, and conservative late fluid management (CLFM), defined as an even-to-negative fluid balance measurement during the first 7 days after lung injury. The results revealed both AIFR and CLFM to have lower mortality rates if used separately when compared to not being used at all; however mortality rates were lowest if used in combination suggesting an additive effect of both fluid strategies (Murphy el al., 2009).

• Given many patients with pulmonary contusions have traumatic injury to other organ systems (such as head trauma, fractures, open wounds etc.) – all of which require positive fluid balance to ensure adequate tissue oxygen delivery for optimal healing to take place, a strategy of fluid resuscitation to restore cardiac output and tissue oxygen delivery in acute resuscitation, followed by a more conservative fluid administration protocol seems appropriate for most patients with pulmonary contusions.

  o Fluid type for administration to patients with pulmonary contusions...

  • Most studies demonstrate little difference in patient survival, requirement for ventilation therapy, or lung function when isotonic crystalloid fluids, colloid fluids or hypertonic crystalloid fluids are used for patient resuscitation. However, one study in humans, and one in pigs, demonstrated lower lung water volumes in patients with acute lung injury in sepsis that received synthetic colloids (hydroxy-ethyl starch) than those that did not, suggesting there may be benefit in providing colloids in some patients with severe inflammatory lung disease

  o Current recommendations – it is difficult to provide recommendations regarding fluid resuscitation in pulmonary contusions for all patients, as all patients are different, and require individual assessment. However, the following may be used as a guide...

  • For acute patient resuscitation

    o Lactated Ringers’ solution 10 ml/kg IV over 10 minutes, and repeated until clinical signs of shock have resolved
One bolus of lactated Ringer’s solution may be substituted for a single bolus of hydroxy-ethyl starch (HES; Voluven) @ 3-5 ml/kg given over 10 minutes if desired to prolong the effectiveness of lactated Ringer’s solution.

- Following acute volume resuscitation

  - Lactated Ringer’s solution or other buffered poly-ionic isotonic solution should be administered at 2-4 ml/kg/hr to provide for maintenance fluid requirements.
  
  - Hydroxy-ethyl starch may be administered at 10-20 ml/kg/day if significant protein loss is expected due to other injuries e.g. severe cutaneous wounds caused by road traffic trauma or dog attack) to assist in maintaining colloid oncotic pressure and tissue perfusion in other injured tissues. Crystalloid fluid rates should probably be reduced to 2 ml/kg/hr in cases in which synthetic colloids are administered to avoid the potential for fluid overload in pulmonary tissues.

- **Monitor the patient** – regular assessment of cardiovascular status (heart rate, pulse quality, mucus membrane color) and respiratory function (respiratory rate, effort, pulse oximetry) is essential to aid in determining the progress of the patient, and the requirement for further intervention e.g. mechanical ventilation. Blood gas analysis and venous blood lactate can also be used as a monitoring tool to evaluate global tissue perfusion adequacy, and the effectiveness of pulmonary gas exchange.

- **Analgesia** – pulmonary injury is painful, especially if it occurs with concurrent pleural or thoracic cage injury. Analgesia should be provided with an opioid analgesic, +/- ketamine. Anti-anxiety medications such as midazolam may also be considered to reduce stress associated with respiratory difficulty.

  - **Opioid medications**: fentanyl constant rate infusion is considered the agent of choice in most patients with acute pulmonary contusions and/or thoracic wall injury, owing to its short-acting pharmacodynamics, and minimal cardiovascular and respiratory depressant effects. Morphine and methadone, particularly if administered at dose rates exceeding 0.1 mg/kg, can produce profound depression in the compromised patient, along with respiratory depression, both of which are undesirable in the acute trauma patient with breathing difficulty. Pain caused by mild injury may be successfully managed with butorphanol, which, like fentanyl, is associated with minimal respiratory depression.

  - **Adjunct medications**: Ketamine constant rate infusions may be employed alongside fentanyl or butorphanol constant rate infusions if patients remain painful despite opioid medication. Likewise, stressed patients may benefit from mild sedation with ketamine or midazolam constant rate infusions. Lidocaine intercostal local nerve blocks administered to patients with fractured ribs can provide useful analgesia in combination with the constant rate infusions outlined above.
**Pneumothorax**

**Aetiology**

Pneumothorax is one of the most common emergency respiratory presentations following traumatic injury to dogs and cats. It results primarily from traumatic chest wall compression, with rupture of alveoli secondary to increase in intra-thoracic pressure against a closed glottis, direct penetration of thoracic wall (sharp objects, rib fractures), or rupture of major airway.

Note that rupture of the trachea or mainstem bronchi will typically cause pneumo-mediastinum +/- pneumothorax.

**Pathophysiology**

The pleural space is normally at sub-atmospheric pressure, with small amount of fluid forming a cohesive bond between the lungs and parietal pleura. If air enters the pleural space, the cohesion is lost and the lungs collapse. The initial response of the patient is tachypnoea, leading to decrease in pCO$_2$, and increasing pH, tachypnoea due to Herring Breuer reflex (triggered by pulmonary deflation) or hypoxia (triggered by aortic and carotid chemoreceptors which relay to the respiratory centre. Hyperventilation decreases physiologic dead space, and increases efficiency of gas exchange BUT does increase energy needs, and compounds cellular hypoxia. Note- dogs can increase the degree of chest wall expansion by 2.5-3.5 times normal during compromised pulmonary function – but this does come at a cost of increased energy expenditure. It is estimated that the energy of breathing can increase by up to 600% in times of severe dyspnoea, and this energy expenditure, in the face of decreasing tissue oxygen delivery usually cannot be sustained. As the pneumothorax becomes worse, compensatory mechanisms fail, and the patient will develop hypercapnoea, severe metabolic and respiratory acidosis and death.

The collapsed lung lobes in pneumothorax result in ventilation/perfusion deficits, and right to left shunting of blood without gas exchange, as blood circulates through damaged and collapsed pulmonary parenchyma, resulting in decreased pO$_2$, which contributes to shock.

**Clinical signs**

Clinical signs of pneumothorax are typical of other pleural space diseases, and include

- Tachypnoea
- Anxiety
- Restlessness
- Cyanosis
- Pale mucous membranes
- Mouth breathing
- Barrel shaped thorax
- Increased inspiratory +/- expiratory effort

© Vet Education Pty Ltd 2013
The Approach to the Patient with Pneumothorax

Following initial patient evaluation (history, physical examination), thoracocentesis is recommended – prior to radiography – to provide both diagnostic confirmation, and therapeutic relief from lung collapse. Note that in many trauma patients, a respiratory rate of 45-60 breaths per minute, together with findings on auscultation supportive of diminished lung sounds, indicates thoracocentesis is required.

Respiratory distress from pleural effusion is caused by the inability of the lungs to expand – therefore immediate oxygen supplementation and thoracocentesis is indicated to stabilize these patients. Thoracocentesis is also useful for diagnostic purposes as well, as fluid collected during initial patient stabilization can be submitted for microbial culture and sensitivity, cytology, and biochemical analysis.

Patients suspected of having pneumothorax should be managed in the following manner

1. Provide supplemental oxygen using fly-by oxygen, nasal oxygen catheter or oxygen cage to improve oxygen saturation prior to handling the patient
2. Diagnostic thoracocentesis (also a therapeutic procedure)
   a. Thoracocentesis requires gentle patient restraint only in most cases. However, in fractious or anxious patients, sedation +/- local anesthesia at thoracocentesis sites may be indicated.
   b. An elevated respiratory rate, or the presence of supportive clinical signs, such as exaggerated chest wall movement during respiration, dull or absent lung sounds, +/- cyanosis, indicates thoracocentesis is required.
   c. Thoracocentesis - NOTE: be prepared to evacuate the entire pleural space at the first attempt at thoracocentesis.

**Thoracocentesis - The Procedure**

1. **Clip and prep intercostal spaces 5-11 on both sides of the chest**
2. **Infiltrate lignocaine into intercostal space 6-7.**
3. **Insert a 22 g needle or butterfly catheter attached to 3-way stopcock extension set and 10ml - 20ml syringe, into chest cavity, at the level of the rib 6-7 intercostal spaces.**
4. **If air is suspected in the pleural cavity, the needle may be inserted at the junction of upper and middle 1/3 of the rib cage; if fluid is suspected in the pleural space, the needle may be inserted at the junction of middle and lower 1/3 of the rib cage (just above the costo-chondral junctions).**
d. **ALWAYS DRAIN BOTH SIDES** of the chest cavity, due to compartmentalization of the thoracic cavity with mediastinal anatomy in dogs and cats.

e. Remove as much fluid from the pleural space as possible (except in animals with acute haemothorax, in which case, enough fluid is removed to improve respiratory function, and decrease respiratory distress)

3. Cardiovascular stabilization – many patients with pleural effusion require support of their cardiovascular system to manage shock, haemorrhage, dehydration, and ongoing fluid losses. Intravenous fluid support with crystalloids, colloids and blood products should be administered as required

Thoracocentesis should be repeated every 30-60 minutes following patient presentation (or more frequently if the patient deteriorates), with the volume of air removed recorded. The frequency of thoracocentesis can be reduced to 60-120 minutes if the volume of air being aspirated from the chest cavity is falling within 1-2 hrs of patient presentation. If the volume of air is increasing, or not diminishing within the first 6-8 hours of presentation, a chest drain should be placed, and attached to a continuous suction apparatus to prevent excessive pneumothorax development, patient decompensation and death. Other indications for chest drain placement with continuous suction include...

- Patients status is not significantly improving
- Vacuum cannot be established using simple aspiration

Patient progress and pneumothorax resolution can be checked by intermittently (every 2-4 hours) turning off continuous chest suction drainage, and monitoring the patient for 30-60 minutes (unless the patient deteriorates, in which case continuous suction is resumed), and performing a thoracocentesis or suction from the chest drain via syringe to determine the volume of air accumulating in the thoracic cavity within a 30-60 minute time period. Most small pulmonary tears show some sign of resolving – evidenced by decreasing volumes of air suctioned off the chest cavity every 2-4 hrs – over the first 24-48 hours of hospitalization.

If no negative pressure can be established despite thoracocentesis or continuous suction, an exploratory thoracotomy may be recommended to isolate damaged airways and effect surgical repair

Note that patient with pneumothorax ma also present with severe pulmonary contusions and other injuries. Failure to establish normal respiratory pattern may be an indication for anaesthesia, endotracheal intubation, intermittent positive pressure ventilation (IPPV), followed by thoracocentesis or immediate percutaneous thoracotomy to relieve intra-thoracic pressure, to allow for an improvement in venous return, diastolic function and cardiac output. Follow-up with thoracotomy and/or chest drain placement.
**Flail Chest**

**Definition**

Flail chest is the fracture of two or more adjacent ribs in more than two places (ventral and dorsal generally), resulting in a segment of thoracic wall that has lost continuity from the rest of the thoracic wall. This isolated segment of chest wall therefore moves independently of the remaining thoracic wall. This independent movement during respiration has been described as paradoxical movement.

**Pathophysiology of Respiratory Abnormalities in Flail Chest**

Paradoxical movement of the thoracic wall in flail chest results from changes in intra-pleural pressures. On inspiration, intra-pleural pressure decreases, as the thoracic cage expands. Because the flail segment is unstable, and is only (usually) attached by muscle, and not attached by ribs to the remainder of the chest wall, it does not move outward with the chest wall, but is rather drawn inward during inspiration, due to the negative pressure in the thoracic cavity.

During expiration, intra-pleural pressure rises as the chest wall relaxes, and this increase in pressure forces the flail segment to move outwards, while the remainder of the chest wall is moving inwards.

Flail chest is almost always accompanied by concurrent thoracic injury, and frequently is found with injuries elsewhere in the body. The most common thoracic injuries present include pulmonary contusions, myocardial contusions, and occasionally pneumothorax or haemothorax. Pain is a major complicating factor in flail chest also, and can contribute to alterations in respiratory rate and tidal volume associated with flail chest.

Respiratory abnormalities observed in patients with flail chest can be severe, and may result from the presence of the flail segment, but more importantly, the presence of concurrent thoracic injury producing the following:

- Decreased vital lung capacity, and functional residual capacity – due to
  - pulmonary contusions
  - intra-pulmonary haemorrhage
  - increased airway resistance
    - airway obstruction – with haemorrhage and pulmonary secretions
  - loss of lung compliance
    - due to dilution of surfactant with pulmonary haemorrhage
    - due to loss of cohesion of visceral and parietal pleura in patients with pleural space disease (haemothorax, pneumothorax
  - pain

- Increased work of breathing – due to
  - Decreased lung compliance
  - Increased airway resistance
  - The presence of reduced lung vital capacity and functional residual capacity
  - Hypoxia
Therapeutic Interventions in Flail Chest

Traditionally, the clinical signs and respiratory distress associated with flail chest were thought to be due, in large part, to the paradoxical movement of the flailing section. Therapeutic efforts have traditionally, therefore been primarily directed at stabilizing the unstable section as soon as possible. As a result, there are many published techniques for stabilization, from procedures that place and maintain traction on ribs in the unstable section to internal fixation of the fractures in an effort to restore synchronous motion of the thoracic wall.

Improved understanding of flail chest pathophysiology has revealed that while thoracic wall paradoxical motion may produce a transient, minimum volume of pendulous airflow as well as mechanical disruption of thoracic movement, these problems alone seem to have little effect on ventilation in the majority of patients. However, there is much evidence to suggest that associated or concurrent pulmonary injury – such as the presence of pulmonary contusions, airway obstruction, pleural space disease (pneumothorax, pleural fluid accumulation), pain and hypoventilation are responsible for most of the clinical signs, respiratory incompetence, and hypoxia associated with flail chest. To restore respiratory competence, treatment should therefore be directed at resolving these respiratory abnormalities.

Isolated flail chest may be successfully managed with conservative management, which includes the following:

- Oxygen supplementation
- Placement of the patient in such a manner that the flail segment is facing down can reduce the degree of paradoxical movement of the flail segment, and may reduce pain associated with the flail segment movement.
- Bandaging the chest – with a light bandage may reduce paradoxical flail segment movement in a similar manner to facing the flail segment ventrally/downwards in patients resisting postural manipulation
- Provision of continuous positive airway pressure (CPAP)
- Analgesia – parenteral opiate administration is recommended; along with local intercostal nerve blocks dorsally and ventrally to the rib fractures in the flail segment.
- Non-surgical flail segment immobilization – in humans, mechanical ventilation is the treatment of choice for pulmonary contusions and flail chest, as the flail segment paradoxical movement is abolished through abolishing negative intra-pleural pressure during positive pressure ventilation

Stabilization of the flail segment is advantageous in that it may prevent further damage to intra-thoracic structures, improve pulmonary ventilation, and may decrease pain associated with movement of flail fragments. Surgical stabilization is also associated with a faster ventilator wean, shorter ICU time, less hospital cost, and faster recovery of pulmonary function in patients with mild to moderate pulmonary injury. However, it is worthy of note, that in patients who have severe pulmonary contusion, there appears to be little role for flail segment stabilization as supportive therapy and pneumatic stabilization (ventilation therapy and CPAP) is the recommended approach for these patients.
**Surgical Stabilization in Flail Chest - Introduction**

In numerous human and animal model studies, the presence of concurrent thoracic injuries and pain appear to contribute to much of the hypoxia and hypoventilation observed in flail chest, rather than the presence of the flail segment itself. However, on occasion, flail chest segments have cause serious intra-thoracic injury, such as aortic laceration, lung laceration, and can worsen or exacerbate the concurrent thoracic injury.

In addition, reduction and fixation of unstable chest wall segments may be associated with a reduction in the duration of ventilatory support, pain, mortality and pneumonia in patients with mild to moderate pulmonary contusion compared to those in which chest wall fixation is not performed. The best indication for early operative chest wall stabilization is flail chest without pulmonary contusion, because this will lead to a significant reduction in the duration of ventilatory support required. Secondary stabilization is recommended in patients with pulmonary contusion showing paradoxical movement of the chest wall during weaning from the respirator.

The optimum position for the flail segment to be fixed is in the external position, as opposed to the internal position – that is, the flail segment should be fixed so that it is in approximate confluence with the remainder of the chest wall, and not in the internally deviated position. The reason for this recommendation is that respiratory rate, tidal volume and minute volume are all found to be superior when the fixation is in the external (abducted) position ventilatory status improves after FEP, because the thoracic cage tends to recover its normal volume and stabilizes the paradoxical movement.

**Surgical Stabilization in Flail Chest - Indications**

Given the aforementioned discussion, surgical stabilization of flail chest is generally recommended to be performed in the following circumstances

1. Following initial patient stabilization
2. When there is imminent risk of further trauma to thoracic organs due to motion of the flail section.
3. When flail chest has resulted in severe tissue disruption, open pneumothorax, or fracture fragments that have or may lacerate thoracic organs
4. The presence of flail chest without pulmonary contusion
5. To stabilize the flail chest segment prior to weaning from ventilation therapy

**Surgical Stabilization in Flail Chest - Methods**

1. **Repair of rib fractures** – rib fracture repair can be undertaken with appropriately sized orthopedic pins and wire or plates and screws. It is equally important to re-establish soft tissue integrity such that negative pleural space pressure can restored.

2. **Repair of soft tissues** –

   a. When one intercostal space has been disrupted can be accomplished in a manner similar to closure of an intercostal thoracotomy following adequate debridement of devitalized tissues. This is achieved by pre-placing 4-8 heavy monofilament absorbable (or non-absorbable) sutures round the ribs adjacent to the affected intercostal space. Approximate the ribs and tie the sutures to close the intercostal space defect.
b. When the soft tissue integrity of multiple intercostal spaces has been disrupted it may be necessary to place a series of staggered overlapping circum-costal sutures incorporating all of the affected ribs and one rib cranial and caudal. This creates a "basket weave" pattern and can act as a support for soft tissues mobilized to cover the defect such as the latissimus dorsi or external abdominal oblique muscles or a flap created from the greater omentum. Placement of a thoracostomy tube will facilitate reestablishment of negative intra-pleural pressure.

3. **Flail section stabilization** involve the percutaneous placement of sutures that encircle ribs within the flail section, applying traction with those sutures and attaching them to some form of external brace that uses the adjacent intact thoracic wall to provide counter traction for stabilization.

   a. Administer intercostal nerve blocks with long acting local anesthesia to facilitate placement of the external brace.
   
   b. The external brace most commonly used is in the form of heat sensitive plastic or fiberglass casting material (e.g. orthoplast) that has been molded to fit the thoracic wall over the area of the flail section. It is important that the material extends well beyond the borders of the flail section so that it rests across non-fractured ribs.
   
   c. Drill two holes through the material in locations that will correspond to each fractured rib in the flail section. It is important to place holes sufficient for passage of two sutures per fractured rib, dorsally and ventrally positioned. This will prevent pivoting of the ribs that may occur with only one point of fixation.
   
   d. Clip and clean the area for suture placement, and perform aseptic surgical preparation.
   
   e. Place sutures using monofilament non-absorbable sutures around each rib, dorsally and ventrally, so that the suture ends can be passed through the corresponding holes placed through the bracing material. In order to avoid interference of the brace with proper suture placement it is important to pre-place all of the sutures prior to securing the brace. When passing the suture it is prudent that as the needle passes around the rib it remains adjacent to the bone especially along the caudal and medial borders. This will minimize the potential of encircling the neurovascular bundle caudally and lacerating pulmonary parenchyma.

**Post-operative Management**

Management of patients with flail chest following stabilization of their flail segment should continue as for any patient with traumatic respiratory disease, which will include

   a. Oxygen supplementation
   
   b. Evacuation and management of any pleural space disorder – pneumothorax, haemothorax etc.
   
   c. Ventilation assistance – CPAP or intermittent positive pressure ventilation for patients with pulmonary contusions
   
   d. Analgesia – local intercostal nerve blocks, combined with parenteral narcotic analgesia is recommended in most patients
   
   e. Intravenous fluid support
   
   f. Management of concurrent injuries and illness
   
   g. General nursing care
Prognosis

The prognosis for animals with flail chest or thoracic wall trauma is generally dependent on the amount and degree of concurrent pulmonary or cardiac trauma, and is also influenced by the intensity and rapidity of therapeutic intervention. In general, most rib fractures do not require internal fixation, and the prognosis for non-flail thoracic wall injuries is generally good. A flail chest segment per se does not warrant a poor prognosis. However, the presence of severe pulmonary contusions, or a failure of improvement in patient respiratory function despite appropriate medical and surgical intervention warrants a more guarded outlook.

Diaphragmatic Hernia

Incidence

Diaphragmatic hernia was reported in 5.8% of dogs sustaining fractures as a result of motor vehicle accidents.

Pathophysiology

The pathophysiology surrounding diaphragmatic hernia in the dog and cat is thought to centre on an acute rise in intra-abdominal pressure with the major energy dissipation directed cranially toward the diaphragm.

Diagnosis

The diagnosis of diaphragmatic hernia is based on a thorough assessment of the respiratory system based on physical examination, and is aided by a variety of imaging techniques.

Clinical Signs

Clinical findings in dogs and cats with diaphragmatic hernia are varied, and depend to some extent on which organs are herniated

- Stomach
- Liver
- Kidneys
- Small intestine
- Omentum

Clinical signs also depend on the presence of concurrent injuries...

- 38% have concurrent thoracic injuries
- 48% have no other clinical signs

Furthermore, clinical signs depend on the acuteness of the injuries. Acutely, signs of cardiovascular (shock) and respiratory compromise (tachypnoea, dyspnoea, cyanosis, decreased lung sounds, and borborygmus on thoracic auscultation) will predominate.
Diagnostics

Imaging techniques available include...

- survey radiography
- positive contrast celiography/peritoneography
- upper gastrointestinal contrast study
- Diagnostic ultrasound of the thoracic cavity

Diagnostics

Radiographic findings include gross evidence of liver lobes, bowel loops, or spleen or stomach within the pleural space.

In addition, abdominal radiographs may be helpful to identify organs "missing" from the abdomen.

It is important to note that often in cases of diaphragmatic hernia; concurrent pneumo/haemo thorax is often present. The presence of concurrent pulmonary contusion may also be noted making definitive diagnosis challenging.

Surgery Controversies

Some debate exists as to the optimal time for surgical intervention in cases of diaphragmatic hernia. One study found increased mortality in cases in which surgery was performed within 24 hours of the inciting trauma as well as after one year following trauma.

As a result of this study, it is recommended surgical management of patients with diaphragmatic hernia should be delayed if possible until patient stability is achieved.

Immediate Surgery

Indications for immediate surgery include...

- Patients that cannot be stabilized medically (recognizing that surgical intervention may not improve the patient’s condition)
- Evidence of strangulation of abdominal viscera
- Ongoing haemorrhage
- Concurrent injury for which emergency surgery is necessary
- The presence of a distended stomach within the thoracic cavity that cannot be decompressed via tube or trocar catheter.

Regardless of the timing of surgery, mean reported mortality in cases of diaphragmatic hernia is approximately 14-20%.
**Surgical Anatomy**

- The diaphragm projects into the thoracic cavity like a dome
- It attaches to the lumbar vertebrae, ribs, and sternum.
- It has an extensive muscular periphery, and a small V-shaped tendonous centre.
- Muscle fibres arise on lumbar vertebrae, ribs and sternum and radiate towards the tendonous centre.
- The diaphragm is composed of only one layer of muscle and two layers of tendon and therefore is weaker than the multi-layered abdominal wall.
- The central tendon of the diaphragm of the cat is relatively small. In its tendonous portion, transverse fibres course from one side to the other as a reinforcing apparatus.

The muscular part of the diaphragm is divided into three parts – depending on its attachments

- Lumbar part - The pars lumbalis of the diaphragmatic musculature is formed by the right and left diaphragmatic crura. Seen from the abdominal cavity each crus of the diaphragm is a triangular muscular plate whose borders give rise to the tendonous portions. The musculature of the crus mediale is the thickest (3–4 mm).
- Costal part – The Pars costalis consists of fibres radiating from the costal wall to the tendonous centre.
- Sternal part – The pars sternalis arises from the dorsal surface of the sternum, cranial to the xiphoid

The diaphragm domes far into the thoracic cavity, and its costal part lies on the medial surface of the last few ribs and costal arch (when tears occur here, the costal arch can be used in the repair). The stomach and liver attach by ligaments to the concave peritoneal surface of the diaphragm.

**Pre-operative Assessment**

Immediate surgical intervention for the repair of a diaphragmatic hernia is rarely indicated. Emergency surgery should not be undertaken unless the surgeon and anaesthesiologist are prepared to handle any complications and are confident they can maintain the animal's essential requirements while the animal is anesthetized.

Prompt surgical repair is indicated in

- acutely injured animals with severe dyspnoea, cyanosis, and respiratory distress who demonstrate massive herniation
- In patients that present with an air filled stomach in the thoracic cavity (these patients can develop life threatening dyspnoea if enough swallowed air enters the stomach).

**Anaesthesia**

Patient stress must be kept to a minimum during the anaesthetic induction phase as any exertion by the animal can be disastrous. Use anaesthetic agents that produce minimal cardiovascular depression, and minimal respiratory depression, that are reversible, and that facilitate rapid intubation.
Acceptable agents may include an opiate-benzodiazepine-ketamine combination given as a constant rate infusion, or an opiate-alfaxolone combination.

Avoid hypotensive drugs; and avoid respiratory depressant drugs such as alpha-2 agonists and isoflurane (use isoflurane at the lowest effective dose)

**Surgical Approaches**

A midline abdominal celiotomy (xiphoid to pubis) is the easiest and most versatile approach. Positioning the patient's head toward the top of the table and tilting the table at a 30° to 40° angle will facilitate gravitation of abdominal viscera out of the thorax. Rarely is it necessary to extend the incision into the thorax via a median sternotomy however the animal should be prepared in case this becomes necessary.

**The Surgery**

- An incision is made from xiphoid to pubis.
- Once the peritoneal cavity is opened, the diaphragm is exposed and the situation evaluated.
- Some hernias, especially in the area of the dorsal attachments of the crura and the aortic hiatus are not easily visualized; therefore, this area should be carefully inspected even when another laceration is present.
- The herniated contents are replaced in their proper position and inspected for damage.
  - Torsion of one or more liver lobes
  - Ruptured viscous
  - Intussusception
  - Costal abdominal hernia
- If adhesions exist, they should be broken down using blunt dissection so as to avoid excess haemorrhage and inadvertent damage to a vital structure.
- Using large sponges or laparotomy pads moistened with warm saline, the liver and bowel are retracted caudally.
- The diaphragmatic tear is now more easily visualized so that a careful examination of the thorax can be done both visually and manually.
- All thoracic fluid should be aspirated.
- The lungs should be SLOWLY expanded over several, slowly increasing tidal volumes (up to 10 l/kg only) over several minutes to remove atelectasis and to inspect for pulmonary tears and persistent areas of collapse.
  - THIS MUST BE DONE SLOWLY
  - TAKE SEVERAL MINUTES TO GRADUALLY INFLATE LUNGS
  - RAPID OR OVER-ZEALOUS EXPANSION IS ASSOCIATED WITH LUNG REPERFUSION INJURY AND DEATH
- If the hernia is more than 48 hours old, the edges of the tear should be debrided, by incising the hernia edge instead of trimming a piece of the diaphragm off.
- It is recommended to suture the hernia from dorsal to ventral, as it is much easier to visualize the dorsal structures (vena cava, aorta, oesophagus) when suturing.
- The hernia is closed with a single layer, simple continuous suture pattern using synthetic monofilament absorbable suture material e.g. PDS; Maxon, with suture size recommended in cats being 3-0; and in dogs 2-0 – 3-0
• Pre-place the most dorsal sutures for better visualization of the tear during suturing. It is also helpful to reconstruct the tear with several simple interrupted sutures to facilitate visualization of the rent.
• When tears near the caval hiatus are sutured, care is taken to avoid constriction of the vena cava by placing sutures too close to the cava.

Evacuating the Pleural Space

Pleural space evacuation should be carried out slowly, to minimise the risk of re-expansion pulmonary oedema. Maximal reinflation of the lungs prior to diaphragm closure is no longer recommended and should not be performed. Air can be evacuated from the chest using several techniques.

• Thoracocentesis
  o Through the diaphragm
  o Through the chest wall
• Abdominal Chest Tube
  o A 12–14 French feeding tube
• Chest Tube
  o A 12–14 French diameter chest tube can be placed

Post-Operative Care

• All patients are monitored carefully for the next six to eight hours. If signs of respiratory abnormalities arise (dyspnoea, tachypnoea, etc.), the right and left hemi-thorax should be tapped with a needle and syringe.
• Post-surgical care involves use of systemic antibiotics and careful monitoring of the patient's breathing, temperature, and colour.
• Analgesics may be used to relieve patient discomfort; however care should be taken to monitor the effects of various analgesic drugs on respiratory effort.

Conclusion

Successful repair of a diaphragmatic hernia depends on careful preoperative and postoperative care of the patient. During the surgical repair, the surgeon must work quickly and effectively to complete the procedure as efficiently as possible. In addition, anaesthetic management is vital to patient survival.
Thoracic Trauma – What Could Go Wrong?

The patient with thoracic trauma represents a potentially severely unwell patient with critical illness in not only the respiratory tract and pleural space, but also frequently involving the cardiovascular system, and other organ systems. Close attention must be paid to the patients airway, respiratory effort, cardiovascular status and neurological function to ensure that optimal conditions are provided for adequate tissue oxygen delivery to body tissues – as without this, organs elsewhere in the body, such as the kidneys, and gastrointestinal tract, with suffer oxygen debt, with organ dysfunction and/or failure resulting. In addition, management of other respiratory conditions associated with trauma, such as open wounds associated with pneumothorax, is critical to ensuring patient stabilisation and appropriate tissue healing whilst minimising opportunities for complications such as pyothorax or systemic infection and/or inflammation.

References

Pulmonary Contusions:
Flail Chest


5. Gyhra, Alberto, Torres, Patricio; Pino, Jorge; Palacios, Silvia; Cid, Luis; Experimental flail chest: Ventilatory function with fixation of flail segment in internal and external position Journal of Trauma. 40 (6). 1996. 977-979

6. Chang, YS; Lin, PJ; Chang, CH; Blunt trauma with flail chest and penetrating aortic injury; European Journal of Cardio-thoracic Surgery 16 (3): 374-377 SEP 1999

Diaphragmatic Hernia


© Vet Education Pty Ltd 2013