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Thoracic trauma and respiratory distress
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As one of the major body systems, examination of the respiratory system should always be a top priority in any patient presenting as an emergency. Tachypnoea and dyspnoea following trauma is common and may be a cause of death. Concurrent thoracic injury is common with other injuries. In one study, approximately 60% of patients with humeral fractures and 40% with pelvic fractures had significant thoracic injuries, which delayed the timing of anaesthesia.

In patient with trauma the commonest injuries are (in approximate order of frequency):
- Pulmonary contusions (crackles or harsh lung sounds).
- Pneumothorax (dull lung sounds esp dorsally).
- Thoracic wall injury (e.g. rib fractures).
- Diaphragmatic rupture.
- Haemothorax (rare for this to be clinically significant).
- Damage to the major airways.

Ideally many of these injuries can be suspected from the physical examination. Although radiography is sometimes necessary for confirmation of pathology and is warranted at some point in all traumatised patients, it should be delayed until the patient is stable. Certainly a dyspnoeic traumatised patient with dull lung sounds should have thoracocentesis performed prior to radiography – this is both diagnostic and therapeutic.

Examining the respiratory system in the traumatised patient
The patient presenting with dyspnoea post trauma represents both a diagnostic and therapeutic challenge. These patients are very fragile and the restraint necessary to perform diagnostic tests or administer therapy may precipitate sudden deterioration or even death. Both diagnostic and therapeutic interventions must be carefully considered in terms of their potential risk:benefit for any individual patient. There are some therapies that will be of benefit whatever the underlying cause of the dyspnoea and others that are specific to certain diagnoses. Especially with severely dyspnoeic patients where diagnostic tests such as radiography may be fatal, the aim should be to achieve as much information as possible from a careful physical examination.

The goal of the physical examination should be to assess the severity of dyspnoea and to make a judgement as to the anatomical source of the respiratory distress. This is vitally important in severely dyspnoeic patients as it guides empirical stabilisation measures. The physical examination should include:
- Observation
  - Rate
  - Pattern – is the dyspnoea inspiratory, expiratory or mixed?
  - Is there any audible noise?
    - Stridor – whistling/squeaking inspiratory noise
    - Stertor – snoring noise
- Auscultation
  - Both sides of the chest should be listened to in multiple sites. Dependent on the size of the patient the chest wall should be split into a “noughts and crosses” board and the pattern of abnormal sounds noted. In a normal animal, lung sounds are bilaterally symmetrical and a little louder cranio-ventrally as there is greater lung mass there.
  - Sounds should be classified as
    - Decreased
    - Increased
      - Harsh – increased noise without specific crackles or wheezes
      - Wheezes – whistling noises typically heard on expiration and suggestive of lower airway disease
• Crackles – “popping” noises typically heard on inspiration and typical of alveolar disease
  o When deciding whether sounds are increased or decreased, consideration must be given to the level of breathing effort the animal is making. For example a normal dog breathing hard (eg post exercise) will have louder lung sounds than it will at rest; a dog with a significant pneumothorax breathing hard may have a similar loudness of lung sounds as a normal dog breathing quietly.
  o Referred upper airway noise can be distinguished from lower airway noise by listening over the trachea.
  o You may need to find a quiet room to listen properly!

Severity of dyspnoea

The following findings can all be used to evaluate the severity of dyspnoea
• The animal may adopt abnormal postures to ease breathing (orthopnoea). These include extension of the head and neck, a preference for a standing or sitting position rather than lying down (especially in dogs), and abduction of the elbows. Nostril flaring on inspiration may be seen.
• Open-mouthed breathing in cats is often a sign of severe respiratory distress. Any attempt to move the patient from the desired position causes anxiety. If a severely dyspnoeic patient chooses lateral recumbency, it is usually a sign of impending arrest, especially in a cat. There usually are large excursions of the chest wall and diaphragm unless the patient cannot move air into the lungs, such as with airway obstruction, or the lungs cannot be expanded, such as with pleural space (pneumothorax) or chest wall injuries.
• Paradoxical breathing represents marked inspiratory effort. During paradoxical breathing the chest wall and abdomen move in opposite directions. During normal inspiration, the chest moves outward as the intercostal muscles contract and the abdomen also moves outwards as the diaphragm flattens. During paradoxical breathing with marked increased inspiratory effort, the chest still moves outwards during inspiration but the force of contraction of the intercostals is such that the diaphragm is actually sucked forwards and the abdomen moves in.
• Cyanosis may be detectable on examination of the mucous membranes. Detection of cyanosis requires a capillary deoxygenated haemoglobin level of approximately 5 g/dL. Considering dogs with normal haematocrits of 45% have a haemoglobin level of around 15g/dL, roughly one third of a dog’s haemoglobin must be deoxygenated before cyanosis is detectable, thus it always represents severe hypoxia.

Following a careful examination, it should be possible to localise the origin of the dyspnoea to an anatomical region of the respiratory tract. This helps determine what treatment and stabilisation measures are most appropriate. The major anatomical regions are and the pathology following trauma that is associated with each of them is des cribed in the table below:

<table>
<thead>
<tr>
<th>Site</th>
<th>Respiratory pattern</th>
<th>Audible noise</th>
<th>Auscultation findings</th>
<th>Disease Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper airway</td>
<td>Inspiratory effort; long and slow inspiratory phase</td>
<td>Yes</td>
<td>Referred upper airway noise</td>
<td>Trauma to the face and nose; nasal or pharyngeal bleeding or swelling</td>
</tr>
<tr>
<td>Pulmonary parenchyma</td>
<td>Mainly inspiratory but mixed patterns possible</td>
<td>No</td>
<td>Harsh sounds and/or crackles</td>
<td>Pulmonary contusions</td>
</tr>
<tr>
<td>Pleural space</td>
<td>Short shallow inspiration</td>
<td>No</td>
<td>Dull lung sounds</td>
<td>Pneumothorax (common)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Haemothorax (rare to be clinically significant)</td>
</tr>
<tr>
<td>Thoracic wall/rib cage</td>
<td>Short shallow respiration May be areas of asynchronous movement</td>
<td>No</td>
<td>Variable; may be dull SQ emphysema and crepitus also common</td>
<td>Rib fractures Intercostal muscle tears</td>
</tr>
<tr>
<td>Diaphragm</td>
<td>Paradoxical movement</td>
<td>No</td>
<td>Variable; borborygmi occasionally heard</td>
<td>Rupture</td>
</tr>
</tbody>
</table>
Depression or injury to the respiratory centre in the CNS or neuromuscular injury causing failure of respiratory muscular function may also occur and results in decreased breathing effort; excursions of the chest wall and diaphragm are reduced and the rate is usually decreased. Further evaluation of the severity of the problem should include pulse oximetry and ideally arterial blood gas analysis. Imaging techniques are helpful in determining aetiology but are unfortunately of little use in assessing the functional status of the lung.

Pulse oximetry is widely available but has several limitations. Firstly, a good pulse wave is required for an accurate reading and many patients with severe dyspnoea post trauma have concurrent hypovolaemic shock meaning a strong, regular pulse wave is not present in the peripheral circulation. Secondly, due to the sigmoid nature of the oxyhaemoglobin dissociation curve, a relatively comforting pulse oximetry reading of 90-93% corresponds to an arterial oxygen partial pressure (PaO$_2$) of around 60mmHg where a relatively small further drop in the PaO$_2$ will result in a rapid and precipitous drop in haemoglobin saturation and the pulse oximetry reading. And finally, many of our patients either have pigmented skin or are uncooperative which makes it difficult to achieve an accurate value.

Arterial blood gas analysis is the most accurate way of assessing arterial oxygen levels. Arterial blood is usually obtained from the dorsal metatarsal artery in patients over 5kg and from the femoral artery in patients weighing less than this. The arterial partial pressure of oxygen (PaO$_2$) should be approximately five times the fractional inspired oxygen, thus on room air (21% oxygen), a normal animal should have a PaO$_2$ of 85-110 mmHg. Values less than 60mmHg are cause for severe concern. Arterial blood gas also allows measurement of arterial CO$_2$ which allows assessment of ventilation as well as oxygenation status.

**Treatment and stabilisation of the dyspnoeic trauma patient**

There are several management and treatment strategies that should be adopted whatever the cause of the dyspnoea.

**Minimal stress** Animals with severe hypoxaemia will limit their movement and therefore have sufficient oxygen delivery to support major organ systems. Any increase in non-essential tissue oxygen consumption may prove life threatening. Most especially, increased skeletal muscle activity, such as may occur with restraint or agitation, may precipitate cardiorespiratory arrest. All patients with severe lung disease should have their activity restricted and any stressful procedures such as catheter placement or radiography should be carried out cautiously and incrementally, allowing time for recovery between steps.

**Oxygen supplementation** All animals with severe lung disease will benefit from oxygen supplementation. Oxygen can be supplemented in a variety of ways including flow-by, nasal cannulation, and oxygen cages. Care must be taken that the method of oxygen supplementation does not itself lead to increased stress. Oxygen cages are ideal but may not be available especially for larger patients. Improvised oxygen cages or hoods can be created using normal kennels or Elizabethan collars with cling film or similar over the front. The increase in fractional inspired oxygen (FiO$_2$) achieved with improvised cages or nasal supplementation is variable but it is possible that an FiO$_2$ of 60% may be reached. Ultimately the animal can be intubated and 100% oxygen delivered – although it is possible to do this long term it is not practical in most practice situations.

**Positioning** Body position can significantly affect arterial oxygen concentration and there is a growing body of evidence from human clinical and animal experimental studies that prone positioning (sternal recumbency) can significantly improve PaO$_2$ in some patients. Placing or propping the animal in sternal recumbency may therefore be a simple yet effective way of increasing oxygenation.$^3$ In animals with lateralisng alveolar disease, placing them with their “good side” down should be avoided.

**Specific therapy**

**Upper airway:** In patients with dyspnoea secondary to upper airway obstruction following trauma, there has typically been trauma to the head. The oro- and naso-pharynx can be suctioned if the patient allows. Rarely placement of a tracheostomy tube is required. In contrast to patients with the more typical causes of upper airway obstruction such as BOAS and laryngeal paralysis, sedation is rarely indicated and may be detrimental if the animals has concurrent hypovolaemia.

**Pleural space disease:** Thoracocentesis is the recommended treatment for patients where pleural space disease is suspected and can provide a rapid improvement in the level of dyspnoea. It will also yield a sample that confirms the diagnosis. Thoracocentesis is usually performed using a butterfly catheter in cats and small dogs and a needle or over-the-needle intravenous catheter in larger dogs.
The precise site for needle introduction depends on the physical examination and where the clinician identifies areas of dullness, however the 7-10th intercostals rib spaces are commonly used. The needle should always be introduced off the cranial aspect of a rib to avoid traumatising the intercostals vessels and nerve. Most patients tolerate the procedure conscious although sedation or local anaesthesia may be necessary in more fractious animals. The effectiveness of thoracocentesis can be determined principally by an improvement in the signs of dyspnoea and confirmed by radiography. If the pneumothorax re-develops over a short timeframe, after a total of 3 productive thoracocentesis procedures, then an indwelling chest drain should be placed.

Open pneumothorax occurs very rarely but needs urgent action when it is seen. These patients have a wound that creates an open communication between the exterior and the pleural space; some patients may present in respiratory arrest. To stabilize the patient the wound should immediately be covered with a water soluble gel such as K-Y jelly and an occlusive dressing providing a temporary airtight seal. The pleural space should then be evacuated by needle thoracocentesis. The animals should then have concurrent signs (especially shock) stabilised and should then be managed surgically.

Parenchymal disease: Contusions (pulmonary bruising) is a very common injury post trauma and can range in severity form very mild to rapidly life threatening. Treatment is difficult and the most severely affected patients may require mechanical ventilation. Avoiding worsening of contusions is a key management strategy. The rationale for intravenous fluid therapy should be carefully considered in patients with severe and developing contusions. Fluid therapy will tend to increase pulmonary capillary hydrostatic pressure. As this is the major determinant of pulmonary fluid extravasation, fluid therapy has the potential to worsen dyspnoea. Some patients will have concurrent indications for intravenous fluid therapy such as hypovolaemia. The rate and amount of fluids administered should be carefully tailored to both the cardiovascular and respiratory needs of the patient. This may mean giving fluids cautiously and at lower rates and volumes than might otherwise be chosen.

In severe cases where mechanical ventilation is not available drug therapy may be tried but is unlikely to have a major impact. Furosemide is clearly the initial drug of choice for patients with cardiogenic oedema, however there is also increasing evidence that furosemide may be of benefit in patients with other forms of oedema including the permeability oedema seen with pulmonary contusions. Although its principal action is diuresis, it also has beneficial vasoactive effects including venodilation, especially of the pulmonary veins, and it may increase perfusion to ventilated areas of the lung. An area of experimental investigation is the use of agents which speed alveolar fluid reabsorption. Drugs under investigation for this purpose include the β2 agonists (e.g. dobutamine, salmeterol, terbutaline) and cAMP phosphodiesterase inhibitors. Finally, considering how detrimental stress can be, sedation may be indicated in these patients. Morphine represents a good choice of sedative agent as it also has venodilating properties and the respiratory depressant effects are minimal in veterinary small animal patients.

Rib fractures are often associated with pulmonary contusions or lacerations of intercostal blood vessels. Although pain, pneumo/hemothorax, and inadequate movement of the affected segment of the chest wall all contribute to decreased ventilation and hypoxia, often it is the concurrent pulmonary contusions which impair oxygenation to the greatest degree. Rib fractures are generally managed conservatively with oxygen supplementation and analgesia. Systemic opioids are generally required but intercostal nerve blocks can also be considered to reduce pain and may improve ventilatory efforts.

Diaphragmatic rupture: Ultimately these require surgical management. Ideally surgery should be delayed for 24-48 hours while other injuries are stabilised; however occasionally the rupture is so severe that urgent surgery is required.

In summary when presented with a dyspnoeic trauma patient the aim should be to use the history and physical examination to identify the most likely anatomical location for the source of the dyspnoea. Urgent stabilisation measures can then be instituted alongside support of other injuries.

References