Approaches to diagnosis and treatment of pneumothorax

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Pneumothorax, a frequent cause of dyspnoea in dogs and cats, is defined as an accumulation of air in the pleural space. The air can originate from the respiratory tract or oesophagus, or can enter the thoracic cavity via a penetrating wound through the thoracic wall. Any pneumothorax is usually bilateral in dogs and cats because they have a delicate and usually fenestrated mediastinum.

Classification and underlying causes

Panel 1. Causes of spontaneous pneumothorax

Frequent causes of spontaneous pneumothorax
Ruptured pulmonary bullae
Ruptured subpleural blebs
Foreign body migration from the airways
Penetrating trauma of the oesophagus or trachea
Ruptured pulmonary abscess
Ruptured pulmonary neoplasia
Pneumonia (bacterial, viral, fungal)
Lungworm infestation: Angiostrongylus vasorum infection (dogs), Aelurostrongylus abstrusus infection (cats)
Eosinophilic small airways inflammation (cats mainly)
Less frequent but reported cases
Pleural infection with gas-forming bacteria
Pulmonary thromboembolism
Unfrequent causes in the UK

Heartworm disease (*Dirofilaria immitis*)
Paragonimus kellicotti infection
Ruptured pulmonary fungal granuloma

Causes of pneumothorax can be classified as traumatic, iatrogenic, or spontaneous.

Traumatic pneumothorax is the most common cause of pneumothorax in dogs and probably in cats, and occurs in approximately half of all significant traumatic chest injuries in dogs. The pneumothorax is classified as open when a direct communication exists between the pleural space and the atmosphere via a thoracic wall wound, usually as a result of a penetrating trauma of the thoracic wall, such as a bite or stab wound, shearing injuries or lacerations, or as a result of rib fracture with perforation of the thoracic wall.

The pneumothorax is classified as closed when there is no direct communication between the atmosphere and the pleural space. A traumatic closed pneumothorax can occur with internal damage to the airways or pulmonary parenchyma, such as with fractured ribs damaging the lung parenchyma or airways, blunt trauma to the thorax creating a sudden increase in thoracic pressure leading to pulmonary or bronchial rupture, or traumatic perforation of the oesophagus or airways by a foreign body.

Iatrogenic pneumothorax can be a complication of thoracocentesis, thoracostomy tube, thoracic surgery or percutaneous pulmonary fine needle aspiration. It can also result from damage to the airways during bronchoscopy, tracheal rupture associated with intubation, oesophageal rupture following foreign body removal or oesophageal stricture dilation, or from barotrauma due to excessive airway pressure during positive pressure ventilation.

Spontaneous pneumothorax is diagnosed in the absence of a history or physical evidence of trauma or possible iatrogenic cause. Spontaneous pneumothorax is always closed.

Primary spontaneous pneumothorax is diagnosed when no obvious underlying cause can be found on investigation. It is most often caused by pulmonary blebs or bullae (38% to 68% of dogs in studies). These derive from air-filled lesions in the pleura or pulmonary parenchyma, which rupture to create a pneumothorax.

Pulmonary blebs are accumulations of air escaped from the pulmonary parenchyma, where the air gets trapped between the internal and external layers of the visceral pleura. Pulmonary bullae are air-filled spaces that develop between the pleura and lung surface or within the lung parenchyma, following destruction and confluence of adjacent alveoli.

Grossly, both blebs and bullae usually appear as “blisterlike” lesions of variable size, most commonly located on the apical margins of the affected lung lobes. They usually don’t have a detectable cause and their pathogenesis is unclear; in dogs they probably represent a primary disease process. They occur most commonly in middle-aged, large breed, deep-chested dogs without a previous history of respiratory disease, but they can occur in any breed and at any age. The Siberian husky breed was overrepresented in studies and may be predisposed.

Various underlying conditions can cause a secondary spontaneous pneumothorax (**Panel 1**).

A tension pneumothorax is present when the lesion leaking air in the pleural space acts as a one-way valve, allowing air to enter the pleural space at inspiration, but not to exit at expiration. This can occur with either traumatic or spontaneous pneumothorax. Tension pneumothorax rapidly leads to a high pressure in the thoracic cavity, and can cause rapid deterioration and death of the patient.

Physiopathology

The pneumothorax causes an increase in intrathoracic pressure and collapse of the lungs. Tidal volume is decreased and a ventilation/ perfusion mismatch develops, which leads to hypoxaemia.

If the pleural pressure exceeds central venous and pulmonary artery and venous pressures (as in a tension pneumothorax), there is al s o dec reased venous return to the heart, leading to decreased cardiac output. Myocardial ischaemia may also contribute to the decreased cardiac output.

History and physical examination

The most common clinical signs of pneumothorax are tachypnoea and dyspnoea, exercise intolerance, lethargy and anorexia. A cough may be present. In traumatic pneumothorax, the signs are acute and the cause is generally obvious from the history. With spontaneous pneumothorax the clinical signs can be acute or can develop more gradually if the air leak into the pleural cavity is slow. Once the venous return to the heart is decreased it leads to tachycardia, systemic hypotension and shock. This can occur rapidly, particularly with a tension pneumothorax.

Auscultation often reveals muffled lung sounds dorsally, and muffled cardiac sounds in severe pneumothorax. Hyperresonance on percussion of the thoracic wall may be present. Particular attention should be paid at external examination of the thoracic wall, to determine if any wounds or signs of trauma are present, and clipping may be required to detect small wounds.

Stabilisation in dyspnoeic patients

In severely dyspnoeic patients complete clinical examination and further diagnostic tests, such as

radiographs, should be delayed until stabilisation has been achieved, as stress may compromise survival.

It is useful to obtain a SpO2 measurement (haemoglobin oxygen saturation) with a pulse oximeter to rapidly estimate the severity of the hypoxaemia and the need for oxygen supplementation.

Supplemental oxygen should be provided via an oxygen cage or mask, or by flow-by initially, as is best tolerated by the patient. The aim of oxygen therapy is to maintain the SpO2 above 95% (equivalent to a PaO2 above 80mmHg).

Stress should be minimised. If the patient is distressed or anxious, mild sedation with acepromazine and/or butorphanol can be administered intravenously or intramuscularly, starting at a low dose to avoid deep sedation, hypotension and respiratory depression.

Opioid analgesia should be provided as ventilation will improve when the pain associated with breathing decreases, with care to avoid respiratory depression; it is also beneficial for subsequent thoracocentesis.

Thoracocentesis and pleural air drainage should be performed as an emergency measure in dyspnoeic patients with a suspicion of pneumothorax. Most of the time this can be performed in the conscious or lightly sedated patient. Pneumothorax is most commonly bilateral, so the thoracocentesis can be performed on either side.

Open chest wounds should be covered immediately with an occlusive sterile dressing. Thorough wound debridement and closure under anaesthesia can be delayed until the patient has been stabilised.

Diagnosis and further investigation

Diagnostic thoracocentesis can lead to confirmation of pneumothorax while stabilising the patient.

In stable patients, thoracic radiographs can be performed before thoracic drainage; a pneumothorax creates an elevation of the heart from the sternum on lateral views due to collapse of the lung lobes visualised as retraction from the chest wall, and a radiolucent area of free air in which no pulmonary vascular structures are visible. This is most evident in the caudal thorax on lateral views or in the lateral thorax on dorsoventral views. In severe cases the diaphragm may appear flattened or caudally displaced.

Radiographs should also be performed after pneumothorax drainage to assess pulmonary re-inflation and the pulmonary parenchyma. In cases of traumatic pneumothorax, the radiographs should be assessed for rib fractures, haemorrhagic pleural effusion, and pulmonary contusions. In spontaneous pneumothorax they should be assessed for foreign bodies and underlying airway or

pulmonary disease. In spontaneous pneumothorax without an underlying cause detected on pulmonary radiographs, the cause is highly likely to be pulmonary blebs or bullae. These may appear on radiographs as circular, lucent cavities with thin walls at the margins of lung lobes.

Unfortunately, they are rarely identified on radiography unless they become very large or develop thickened walls; in one study, bullae were seen on radiography in only 25% of affected dogs, and blebs were not visualised. CT scan is much more sensitive than thoracic radiographs in detecting blebs and bullae, but is rarely readily available in general practice.

In spontaneous pneumothorax, further investigation should include haematology, biochemistry, and faecal flotation to detect Angiostrongylus vasorum larvae (dogs) or Aelurostrongylus abstrusus larvae (cats).

Treatment

The pneumothorax can be drained via thoracocentesis or via a thoracostomy tube. Thoracostomy tube placement is recommended in patients requiring more than two thoracocenteses per 24 hours, and in all cases with a tension pneumothorax.

Re-expansion pulmonary oedema is a rare complication following drainage of pneumothorax. It occurs when the pneumothorax is drained rapidly in animals with chronic pulmonary atelectasis and can be fatal. The onset is rapid, usually immediate to two hours postthoracic drainage. The exact aetiology of re-expansion pulmonary oedema is not known, but increased pulmonary vasculature permeability appears to play an important role.

Thoracostomy tube

Thoracostomy tube placement usually requires general anaesthesia. Stabilisation prior to anaesthesia is essential, and the patient should receive oxygen and be ventilated adequately during anaesthesia. Care should be paid not to apply excessive airway pressure during ventilation to avoid barotrauma to the airways and increasing the size of the respiratory air leaks. Nasal oxygen catheters can be placed prior to anaesthesia recovery if the patient is likely to require continuous oxygen therapy.

Types of thoracostomy tubes include classical large tubes with stylets, and low-profile tubes placed with the Seldinger method using a guidewire. Low-profile tubes are preferred as they are less traumatic and easier to place, and have a sufficient diameter to allow drainage of air.

Frequent drainage of the thorax should be performed (at least every one to two hours initially, and more frequently if required). The frequency of drainage can then be adjusted as a function of air production.

Alternatively, when very frequent drainage is needed, and if a large thoracostomy tube was placed, a Heimlich unidirectional valve can be connected to the tube and will allow spontaneous air evacuation. However, it is not recommended if pleural fluid is also present as it may impair air evacuation. It also prevents monitoring of the amount of air drained.

If the air leakage in the thoracic cavity is very severe, continuous suction may be required, but this is more difficult to set up and requires specific equipment and very close patient monitoring. It is rarely practical in general practice settings, although fortunately, it is rarely required.

Frequent monitoring of the patient, particularly respiratory rate and pattern and of SpO2, is extremely important, as well as frequent verification of the tube connections to avoid disconnection and creation of an open pneumothorax.

Intrapleural local analgesia can be provided with bupivacaine. Dilute 0.5% bupivacaine (0.2ml/kg) with 0.9% saline (3ml in cats and small dogs and 6ml to 12ml for medium and large dogs) to allow a sufficient total volume and diffusion in the thoracic cavity. This solution is instilled twice daily into the pleural space via the thoracostomy tube, being careful to use aseptic technique, then the tube is flushed with saline (3ml to 12ml or a volume equal to thoracostomy tube volume) to ensure that the bupivacaine solution reaches the pleural space.

The bupivacaine solution is acidic and can initially cause pain immediately after administration, but this can be reduced by adding sodium bicarbonate to the solution (in a ratio of 0.05mmol of sodium bicarbonate per ml of bupivacaine). Onset of analgesia occurs within 20 minutes.

If the pneumothorax resolves with no more air drained from the tube, radiographs should be taken to confirm resolution of the pneumothorax (by opposition to tube obstruction), and the thoracic drain should be clamped for 24 hours. The thorax can then be drained one last time to confirm complete resolution of the pneumothorax prior to tube removal.

Surgery

The prognosis for resolution of a closed pneumothorax with conservative management with thoracocentesis or thoracostomy tube drainage is guarded.

Closed, traumatic pneumothorax without evidence of oesophageal or large airways perforation is often self-limiting and conservative management is frequently successful, as healthy pulmonary parenchyma can heal. Larger pulmonary leaks or lesions in larger airways may, however, fail to heal spontaneously and may require surgical correction.

Conservative management frequently fails to resolve spontaneous pneumothorax, and even when the pneumothorax resolves, the risk of recurrence is high (approximately 50% of dogs). One study suggested early surgical intervention may be associated with a better outcome and significantly

less recurrence of pneumothorax than conservative management in dogs with spontaneous pneumothorax. Surgery (thoracotomy or thoracoscopy) may, therefore, be a good initial approach in cases with focal or without pulmonary lesions when owners are keen for more aggressive treatment, and should also be considered in cases managed conservatively when the amount of air drained remains significant and does not decrease after three to five days. Advanced imaging with CT scanning prior to surgery allows confirmation of the location and distribution of lesions, and should be performed when possible.

In cases with pulmonary lesions on radiographs affecting multiple lung lobes, the prognosis should be considered guarded to poor even with surgery, although air leakage may not be occurring from all lesions and surgery to remove the leaking lesions may lead to a good prognosis if the underlying disease can then be treated.

Pulmonary bullae and blebs are most frequently found in spontaneous pneumothorax when previous thoracic radiographs were unremarkable. A median sternotomy approach is recommended for thoracotomy in these cases so all lung lobes can be thoroughly examined. It is frequent to find multiple lesions in a patient, and several lung lobes can be affected. If possible, partial or complete lobectomy of all affected lung lobes is recommended with blebs or bullae, and any excised lesions should be sent for histology. If lesions are present in multiple lung lobes and cannot all be resected, the prognosis is guarded and risk of recurrence is high. Pleurodesis can be considered in patients with recurrent disease, but rarely appears successful in the few clinical studies published.

Other treatments

Systemic analgesia is required in cases of trauma, painful underlying conditions and for animals with a thoracostomy tube. Careful use of opioids is recommended to avoid respiratory depression.

Cage rest is very important, with sedation if required. Acepromazine is usually sufficient; deeper sedation, such as dexmedetomidine constant rate infusion, may be needed in very excitable dogs.

Other treatment measures will involve supplemental oxy gen as needed, intravenous fluids, antibiotics in case of open wounds or pulmonary contusions, and drainage of pleural effusion if required. Any pleural haemorrhagic effusion should be drained if the patient is dyspnoeic, but the volume of blood removed should be as small as possible, as blood in body cavities will be reabsorbed.

Treatment for potential Angiostrongylus vasorum infection in dogs and Aelurostrongylus abstrusus in cats is recommended in cases with spontaneous pneumothorax without a determined cause, even if the faecal flotation is negative, as intermittent faecal excretion of larvae is possible.

In cases of secondary spontaneous pneumothorax the treatment and prognosis will depend on the

underlying disease.

Prognosis

Traumatic pneumothorax usually carries a good prognosis in the absence of other complications from trauma (such as pulmonary haemorrhage) or associated with thoracostomy tube placement or surgery when required (such as secondary infections).

The prognosis for secondary spontaneous pneumothorax depends on the underlying cause and the amount of pulmonary damage.

The prognosis for primary spontaneous pneumothorax due to pulmonary blebs or bullae is guarded and owners should be warned of the risk of recurrence, particularly in cases where the pneumothorax resolves with conservative management and surgery is not performed (approximately 50% recurrence rate in dogs), or if multiple bullae are present at surgery and all cannot be excised. Dogs with a single bulla or bleb removed surgically via lung lobectomy usually have a good long-term prognosis, with long-term recurrence rates reported to be less than 13%. However, owners should be warned that other bullae may form.

The overall mortality rate for dogs with spontaneous pneumothorax in a study was 12% for dogs treated surgically and 53% for dogs treated conservatively.

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Further Reading

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