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MANAGEMENT OF RESPIRATORY EMERGENCIES

Pneumothorax

Pneumothorax is an accumulation of air or gas within the pleural space. Traumatic pneumothorax may be classified either as "open" or "closed". Traumatic pneumothorax is the most frequent type of pneumothorax in dogs. It most often occurs due to blunt trauma (i.e., vehicular accidents, being kicked by a horse), which causes parenchymal pulmonary damage to the lung and a closed pneumothorax. When the thorax is forcefully compressed against a closed glottis, rupture of the lung or bronchial tree may occur. Alternately, pulmonary parenchyma may be torn due to shearing forces on the lung. Pulmonary trauma occasionally results in subpleural bleb formation, similar to those seen with spontaneous pneumothorax. Open pneumothorax occurs less commonly, but is also frequently due to trauma (i.e., gun shot, bite or stab wounds, lacerations secondary to rib fractures). Some penetrating injuries are called "sucking chest wounds" because large defects in the chest wall allow an influx of air into the pleural space when the animal inspires. These large, open chest wounds may allow enough air to enter the pleural space that lung collapse and marked reduction in ventilation occur. There is a rapid equilibration of atmospheric and intrapleural pressure through the defect, interfering with normal mechanical function of the thoracic bellows which normally provides the necessary pressure gradient for air exchange. Pneumomediastinum may be associated with pneumothorax, tracheal, bronchial, or esophageal defects, or may be due to subcutaneous air migration along fascial planes at the thoracic inlet.

Spontaneous pneumothorax occurs in previously healthy animals without antecedent trauma and may be primary (i.e., an absence of underlying pulmonary disease) or secondary (underlying disease such as pulmonary abscesses, neoplasia, chronic granulomatous infections, pulmonary parasites such as *Paragonimus*, or pneumonia are present). Based on the histologic appearance of the pulmonary lesion, both cysts and bullae have been reported in dogs. Primary spontaneous pneumothorax in dogs may be due to rupture of subpleural blebs; the remaining lung tissue may appear normal. These blebs are most commonly located in the apices of the lungs. Secondary spontaneous pneumothorax is more common in dogs than the primary form. In these animals, the subpleural blebs are associated with diffuse emphysema or other pulmonary lesions. It has been shown that volume strain from expansive pressure within the lung increases disproportionately at the apex as height increases. A majority of affected people are cigarette smokers, suggesting that the underlying pulmonary disease could be a result of interference of the normal function of alpha-1-antitrypsin in inhibiting elastase. It is believed that alpha 1 antitrypsin is inactivated in people who smoke, allowing increased elastase-induced destruction of pulmonary parenchyma.

Surgical Treatment

Surgical therapy of animals with traumatic pneumothorax is seldom necessary. However, non-surgical management of spontaneous pneumothorax usually results in a less than satisfactory outcome. Mechanical pleurodesis of the lungs may decrease the recurrence of pneumothorax in animals operated for spontaneous pneumothorax. Mechanical pleurodesis damages the pleura such that healing results in adherence of the visceral and parietal pleural. Postoperative pneumothorax or pleural effusion must then be prevented as they will result in separation of the parietal and visceral pleura, precluding adhesion formation.

Prognosis

With appropriate monitoring and care, the prognosis is excellent for animals with traumatic pneumothorax in which therapy is initiated prior to extreme dyspnea or respiratory arrest. In a recent study of dogs with spontaneous pneumothorax, 100% of those treated with needle thoracentesis alone and 81% of those managed with chest tubes had recurrence of pneumothorax. The times until recurrence varied from 3 days to 30 months. Three of 12 dogs (25%) undergoing thoracotomy had recurrence; only 1 of these had intraoperative pleural abrasion performed.

Lung lobe torsion

General Considerations and Clinically Relevant Pathophysiology
Any mechanism that increases mobility of a lung lobe seems to favor development of a torsion. Partial collapse of the lung (i.e., with pulmonary disease or trauma) frees it from its normal spatial relationships with the thoracic wall, mediastinum, and adjacent lung lobes. They may enhance mobility. Pleural effusion or pneumothorax, along

with subsequent atelectasis of lung lobes, can allow increased movement of a lobe, predisposing to torsion. Although LLT has been reported to cause chylothorax in dogs, it is more likely that the chylothorax caused the LLT. LLT has been reported secondary to previous thoracic surgery, where lung lobes are manipulated and may remain partially collapsed after thoracic closure.

Torsion of a lung lobe results in venous congestion of the affected lobe; however, the arteries remain at least partially patent, allowing blood to enter. As fluid and blood enter the alveoli, lung consolidation occurs and the lobe becomes dark-colored and firm, similar in shade to the liver. The shape of the affected lobe is often altered and it may appear displaced from its normal location within the thorax radiographically. Pleural fluid usually accumulates due to continued venous congestion.

Surgical Treatment
Spontaneous correction of a torsed lung lobe is uncommon due to swelling of the lobe and rapid formation of adhesions. The treatment of choice for LLT is lobectomy of the affected lobe. Unless LLT is diagnosed very quickly (i.e., immediately after a surgical procedure), damage to the pulmonary parenchyma is generally severe enough that attempts to salvage the lobe are not warranted. Recurrence has been reported following surgical correction where lobectomy was not performed.

Surgical Technique
Clamp the affected pedicle with a noncrushing forceps to prevent release of toxins into the bloodstream, prior to attempting to derotate it. Untwisting the lobe before its removal may help facilitate identification of the vascular structures and bronchus for ligation; however, in some cases, the lobe cannot be easily returned to its normal position due to extensive adhesions. Check the remaining lobes for position and normal expansion. Culture pulmonary parenchyma following removal of the lobe. Submit excised tissue for histologic examination to help determine underlying causes (i.e., pneumonia, neoplasia). Place a chest tube before closing the thoracic cavity.