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CHAPTER 28

PLEURAL SPACE DISEASE

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KEY POINTS

- Abnormalities within the pleural space may include pleural effusion, pneumothorax, or space-occupying soft tissue structures (diaphragmatic hernia, neoplasia).
- A diagnostic thoracocentesis may also prove therapeutic in severely affected patients.
- Fluid analysis and cytologic evaluation should always be performed on aspirates from a patient with newly diagnosed pleural effusion of unconfirmed etiology.
- Aerobic and anaerobic culture and susceptibility testing of suppurative effusions are imperative.
- Comparison of pleural fluid and serum triglyceride levels and cholesterol concentrations are necessary to confirm the diagnosis of chylothorax.
- Clinical evidence of cardiovascular shock often precedes dyspnea in patients with hemothorax.
- Tension pneumothorax, regardless of its origin, rapidly may be fatal. Immediate drainage via thoracocentesis or thoracostomy tube placement is required before taking thoracic radiographs.
- Clinical signs of a traumatic diaphragmatic hernia may be delayed; however, early detection and correction are important because perioperative outcome is worse in chronically affected patients.
- Tools such as ultrasonography, computed tomography (CT), and thoracoscopy are becoming increasingly available to aid in the diagnostic evaluation and treatment of pleural space disease.

PLEURAL SPACE

The pleural space is a potential space formed by the parietal and visceral pleura. It normally contains a minimal amount (few milliliters) of serous fluid to facilitate motion of the lungs in relation to the thoracic cavity and to each other, as well as force distribution during normal breathing.[1,2] The pleura is a thin epithelium formed of mesothelial cells overlying a thin basal membrane. The partition between the right and left hemithoraces is incomplete in small animals, but unilateral or unevenly distributed disease is common, especially when copious fibrin is present within the pleural space.[3]

Physiologic fluid flux in the pleural space is governed by Starling's law (Box 28-1), the degree of mesothelial and endothelial permeability, and the lymphatic drainage.[2] The visceral pleura assumes a larger role in determining the net pressure and favors reabsorption of fluid from the pleural space, where a greater vascular supply and lower hydrostatic pressure exist. Pleural lymphatic vessels are also an important component of fluid and cell reabsorption from the thorax.[2,3]

There is an average pleural pressure of −5 cm H_2O, representing the difference between the elastic recoil properties of the lung and the thoracic cavity expanding forces at rest.[4] Air, fluid, or soft tissue within the pleural space can cause the lungs to collapse and the chest wall to expand outward by increasing the pressure within the thorax.[5] Pleural pathologic conditions such as these subsequently lead to a decrease in tidal volume, total vital capacity, and functional residual capacity.[6] The resulting atelectasis can lead to both hypoxemia and hypoventilation.

BOX 28-1 ***Modified Starling's Law Applied to the Pleural Cavity***[3]

$$\text{Net filtration} = K\{[(P_{c\ \text{parietal}} - P_{c\ \text{visceral}}) - P_{if}] - (\pi_c - \pi_{if})\}$$

P_c: capillary hydrostatic pressure of the visceral and parietal pleura
P_{if}: intrapleural hydrostatic pressure
π_c: plasma oncotic pressure
π_{if}: intrapleural oncotic pressure

CLINICAL EVALUATION

Clinical signs of pleural disease may include tachypnea, open-mouth breathing, coughing, extended head and neck, crouched sternal recumbency with elbow abduction (orthopnea), cyanosis, and short, shallow breathing with an increased abdominal component. Paradoxical breathing has been strongly associated with pleural space disease, particularly in cats.[7] The degree of dyspnea varies depending on the amount of fluid/air/soft tissue, rate of fluid/air/soft tissue accumulation, and concurrent respiratory and metabolic disturbances. Auscultation reveals muffled breath sounds ventrally (fluid or tissue) or dorsally (air). The heart sounds may be muffled by fluid or tissue or abnormally loud or displaced with unilateral or focal disease.

Thoracic radiographs are extremely helpful in diagnosing and quantifying pleural space disease and other intrathoracic pathology. Repeat radiographs after thoracocentesis can be of diagnostic utility to assess improvement and better visualize intrathoracic structures (Figure 28-1). Horizontal beam thoracic radiographs have higher sensitivity for detection of small-volume pneumothorax and pleural effusion in human patients. It has been shown that the lateral recumbency horizontal beam (VD) thoracic radiograph with the standard left lateral view (vertical beam) have the highest detection rate for small volume pneumothorax and allows better severity assessment in traumatized pets or pets suspected of having a pneumothorax.[8]

Ultrasonographic examination is very helpful for rapid identification of pleural fluid in the emergency setting and guiding thoracocentesis. Furthermore, thoracic focused assessment with sonography for trauma (TFAST) ultrasound examination is becoming current practice in the emergency room to evaluate for presence and severity of pneumothoraces as well as other thoracic anomalies (see Chapter 189).[9,10] A pneumothorax is identified by the absence of "lung sliding" or "glide sign," which is the motion of the lung margins sliding against the chest wall surface during normal respiratory movement.[9,11] The transition zone (lung point) is the location where the lung sliding reappears, which helps determine grossly the quantity of

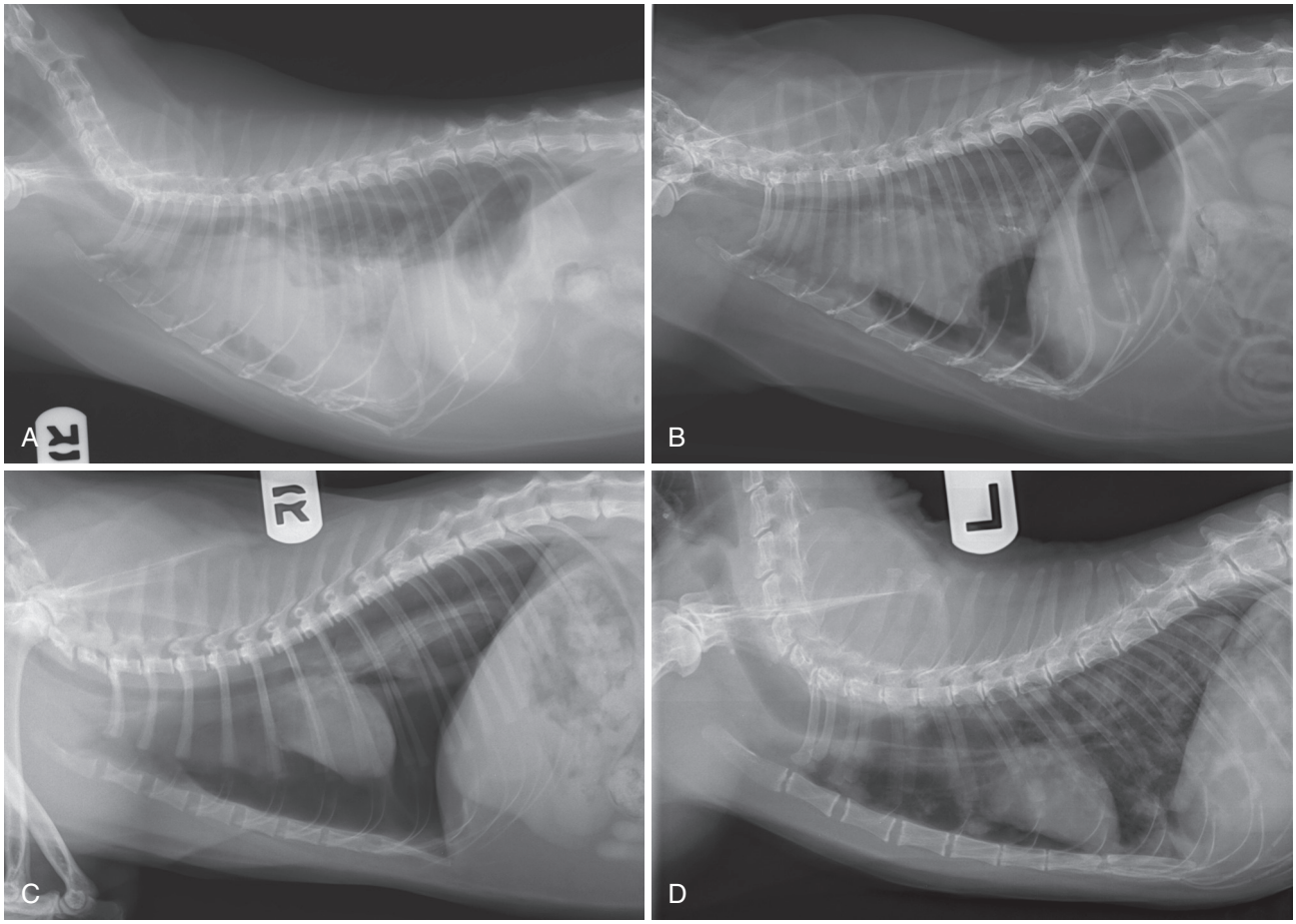

FIGURE 28-1 Cats with pleural space disease. **A,** Moderate volume of malignant effusion secondary to bronchogenic adenocarcinoma. **B,** Pneumothorax after thoracocentesis in the patient shown in **A. C,** Traumatic pneumothorax from high-rise syndrome. **D,** Spontaneous pneumothorax from diffuse pulmonary metastasis of salivary gland adenocarcinoma.

air present in the pleural space. Complete thoracic ultrasonography may reveal underlying pathology such as a diaphragmatic hernia, neoplastic process, thoracic wall disease, or lung lobe torsion.[12,13] Echocardiography will assist in the diagnosis of cardiac disease, heart base tumor, and pericardial disease.

Computed tomography is increasingly used to identify and characterize pleural and pulmonary lesions.[12,14] Thoracoscopy is another useful diagnostic and therapeutic tool in patients with pleural effusion and other intrathoracic pathology, allowing good visualization of the thoracic structures and acquisition of adequate biopsy samples.[15-18]

Thoracocentesis is an invaluable diagnostic, and often therapeutic, tool (see Chapter 198). Indications include (1) the presence of any undiagnosed pleural effusion and (2) therapeutic thoracocentesis to relieve respiratory signs caused by large amounts of air or fluid. However, if the cause of the effusion is known and the patient is not dyspneic, the procedure may be delayed and the clinical signs followed.[19, 20] Fluid analysis has great diagnostic utility in patients with pleural effusion of an undetermined etiology.[19-21]

PLEURAL EFFUSION

Pure Transudates and Modified Transudates

Transudative pleural effusion, or hydrothorax, is the result of variations in the Starling forces that govern pleural fluid flux (see Box 28-1). Pure transudates are characterized by a low total protein and total nucleated cell count (Table 28-1) and generally develop secondary to decreased oncotic pressure (e.g., hypoalbuminemia) within the vasculature. It may also originate from presinusoidal or sinusoidal increased in hydrostatic pressure (e.g., portal hypertension, lymphatic obstruction). Modified transudates are associated with an increased posthepatic hydrostatic pressure (i.e., heart failure) or vascular permeability (e.g., vasculitis, lung lobe torsion, diaphragmatic hernia) causing leakage of a higher protein ultrafiltrate.[8,21] However, in animals with chronic effusion, irritation of the pleura may cause an increased nucleated cell count and water can be reabsorbed in

Table 28-1 Fluid Type and Characteristics[21,22]

Fluid Type	Classic Fluid Characteristics	New Proposed Criteria (Cats)
Pure transudate	TP < 2.5 g/dl TNCC <1500/µl	TP < 3.5 g/dl TPr < 0.56 TNCC < 5900/µl LDHp < 226 IU/L
Modified transudate	TP 2.5 to 7.5 g/dl TNCC 1000 to 7000/µl	
Exudate	TP >3.0 g/dl TNCC >7000/µl	TP > 3.5 g/dl TPr > 0.56 TNCC > 5900/µl LDHp > 226 IU/L

TP, Total protein; *TPr,* total protein ratio; *TNCC,* total nucleated cell count; *LDHp,* pleural lactate dehydrogenase.
Note that the new proposed criteria in cats do not differentiate between a pure transudate and a modified transudate.

excess of protein and cells, thus increasing the cell count and protein concentration.[6] Translocation of abdominal effusion, neoplastic effusion, and chylothorax are other causes of transudates or modified transudates.

Exudates

Exudative effusions are the result of alterations in the permeability of the capillaries.[22] Degenerate neutrophils usually will predominate with an infectious process (e.g., pyothorax).[21] Bacteria may originate from hematogenous or lymphatic spread, penetrating insults (iatrogenic, inhaled or external foreign body, bite wounds, trauma), or spread from infected organs (lung, gastrointestinal).[21] Aerobic and anaerobic cultures are recommended for all exudates. *Nocardia* spp, *Actinomyces* spp, and *Fusobacterium* spp are filamentous rods that are difficult to grow on culture media or identify with culture, cytologic, or histologic examination.[21,23] Other types of organisms, such as fungi, protozoa, and rickettsiae, may also cause septic pleural exudates.[21]

In aseptic exudates, the predominant cell type may vary to include nondegenerate neutrophils (inflammation), small lymphocytes (chylothorax), or neoplastic cells. Potential causes of an aseptic exudate include pneumonia and other well-circumscribed infections (e.g., abscess), generalized sepsis, pancreatitis, or necrosis of intracavitary neoplasia.[21]

Other fluid parameters are gaining interest in veterinary patients in order to classify effusions and help determine the etiology of pleural effusion. Among the markers studied in cats, pleural fluid lactate and total protein, as well as the ratio between the pleural and serum values, have higher capacity to distinguish between transudates and exudates[22] (see Table 28-1).

Feline Infectious Peritonitis

Feline infectious peritonitis (FIP), caused by a coronavirus (feline infectious peritonitis virus [FIPV] or feline coronavirus [FCoV]), is a common cause of aseptic pleural exudative effusion in cats, but it may also cause a modified transudate. Abdominal and pericardial effusion can be concomitant. The effusive form is a more acute disease process but may be present at the onset of the disease or terminally in animals with noneffusive FIP.[23,24] Deposition of infected macrophages forming pyogranulomas adjacent to small venules in the affected tissues and the inflammatory response associated with this cause a severe vasculitis associated with effusion formation.[23]

The diagnosis of FIP should be based on cumulative information rather than one diagnostic test. Pleural or peritoneal fluid typically will be viscous, straw-colored, and have a high protein concentration (>3.5 g/dl) with a relatively low nucleated cell count (<5000 cells/µl, although up to 25,000 cells/µl has been reported).[23,24] Nondegenerate neutrophils predominate in the fluid, with or without macrophages and lymphocytes.[23] A high serum antibody titer range (≥1:1600) is strongly suggestive of the disease.[25] Reverse transcriptase polymerase chain reaction (RT-PCR) on the effusion has shown good results at demonstrating the disease, although false positive results are possible.[24,25] Immunohistochemistry can be performed on the cells of the effusion; alternatively, examination of formalin fixed tissues for viral antigens will permit a definitive diagnosis.[24]

Pyothorax

A pyothorax is an accumulation of purulent exudate within the thoracic cavity. Bacterial infection within a feline thorax was previously attributed to bite wounds.[26] However, increasing evidence now suggests that the extension of pulmonary infections is a common cause, possibly secondary to aspiration of oropharyngeal flora.[27-29] Migrating inhaled foreign bodies and traumatic thoracic penetration are more common in dogs.[30-33] Other bacterial sources reported include pneumonia, pleuropneumonia, lung abscess, aberrant migration of *Cuterebra* larvae or grass awns, hematogenous or lymphatic dissemination, esophageal or tracheal perforations, lung parasites, diskospondylitis, neoplasia with abscess formation, and iatrogenic causes.[26,30,31] Septic suppurative effusion typically is diagnosed when intracellular organisms are present on cytologic examination and the presence of intracellular organisms. Culture and susceptibility testing should be performed on the fluid and antibiotic therapy initiated. Anaerobic bacteria are found commonly,[33-35] and infections with multiple organisms are highly prevalent.[26,34] In cats, nonenteric bacteria are most common and *Pasteurella* spp is most often isolated.[26,35] In dogs, *Escherichia coli* and other members of the family Enterobacteriaceae are isolated most often.[35,36] *Actinomyces* spp and *Nocardia* spp infections have been associated with intrathoracic pyogranulomatous infections in dogs.[37]

Hospitalization for appropriate supportive care and intravenous antibiotics is recommended. Pending culture and susceptibility testing results, broad-spectrum intravenous antibiotic therapy, such as enrofloxacin for gram-negative bacteria and ampicillin with sulbactam or ticarcillin with clavulanate for gram-positive and anaerobic infections,[38] should be instituted as soon as possible. However, an increasing resistance of *E. coli* to enrofloxacin has been documented and amikacin and ceftizoxime have shown to have better efficacy against this organism.[35] Clindamycin is also effective against many of the offending organisms in cats. Medical management with thoracostomy tubes (bilateral in most cases) is recommended, and sterile lavage with warm physiologic saline (10 to 20 ml/kg q6-12h daily) may be used initially if the effusion is thick and flocculent (see Chapter 199). Absorbed lavage solution by the inflamed pleura can lead to fluid overload, so close monitoring of fluid "ins and outs" is recommended. The use of pleural lavage with heparin may improve outcome in dogs with pyothorax treated with thoracostomy tubes by decreasing adhesion within the pleural space.[33] Intermittent thoracocentesis is not a recommended means of drainage and is associated with high mortality in both cats and dogs.[16,33] Tubes will often be necessary for 4 to 6 days [28,34,36] and removal is based on daily fluid reevaluation and the quantity of fluid produced (<2.2 ml/kg per tube q24h, although this can vary depending on the severity of pleuritis).[30] Thoracic radiographs or ultrasonographic examination should be used to monitor the efficacy of drainage. A thoracotomy with or without pneumonectomy should be performed if compartmentalized fluid, lung or pleural abscess, foreign body, perforated esophagus, thoracic wall lesion, or neoplasia is suspected or if medical management is failing.[33,39] Thoracoscopy is an alternative to thoracotomy in certain cases and should be consider as it is associated with lower morbidity and complication rate.[32]

Overall survival rate in small animals with pyothorax is good (63% to 66.1%).[26,33] In cats, success rates have been found to be up to 95% in cats treated with thoracostomy tubes.[28] Medical management is reported to fail in a minority of cats (5% to 9%),[26,28] but cats requiring thoracotomy maintain an excellent prognosis.[26,39,40] In dogs, surgical treatment has been associated with a better outcome.[36] In animals treated with thoracostomy tubes or thoracotomy, the prognosis was significantly better, 71% to 77.6%. The need for surgical exploration has not been associated with poorer outcome.[26,33]

Chylothorax

Chylous effusion is opaque and white or pink. Small lymphocytes usually predominate; however, nondegenerate neutrophils may become predominant after repeated thoracocenteses or with chronic disease.[21] The triglyceride concentration within the effusion is higher than the concentration in the serum, whereas the cholesterol level is equal to or lower than that of the serum. Causes of chylothorax include heart disease (e.g., cardiomyopathy, congestive heart failure,

pericardial disease), thoracic duct obstruction (e.g., intraluminal or extraluminal neoplasia or granuloma), traumatic rupture of the thoracic duct, cranial mediastinal mass (e.g., thymoma, lymphosarcoma, aortic body tumor), lung lobe torsion, diaphragmatic or peritoneopericardial hernia, pacemaker implantation in cats, heartworm disease, congenital malformations, cranial vena caval thromboembolism, ligation of the left brachiocephalic vein, and idiopathic diseases.[41,42]

Idiopathic chylous effusion is diagnosed by exclusion in most animals with true chylothorax. [41,43] Medical management consists of intermittent thoracocentesis, a reduced-fat diet, medium-chain triglycerides, and rutin. Rutin is a benzopyrone nutraceutical that stimulates macrophage breakdown of protein in lymph, accelerating its reabsorption.[44] Thoracostomy tubes are indicated in animals with a traumatic chylothorax, if thoracocentesis is required several times weekly, or following surgery.[6] Surgical intervention is recommended if the medical management is unsuccessful at providing good quality of life to the animal. Multiple surgical interventions have been described; however, a combination of thoracic duct ligation with subphrenic pericardectomy has become the most successful procedure and is often performed via thoracotomy or thoracoscopy.[43, 45,46] Long-term recovery rates vary from 73% to 100%.[45-47] The placement of pleural access ports at the time of surgery enable aspiration of pleural fluid by veterinary staff and owners after surgery and can allow animals with slowly resolving effusion to go home for continued postoperative care. Complications of chylous effusion and its drainage include weight loss, electrolyte abnormalities (pseudoaddisonian changes), lymphopenia, hypoproteinemia, dehydration, and fibrosing pleuritis.[41] Rarely, spontaneous resolution of idiopathic effusion occurs. This is expected in most animals suffering from traumatic thoracic duct rupture.

Hemothorax

A hemothorax is defined as a pleural space effusion with a hematocrit greater than 10%.[8] A lack of gross clotting and evidence of erythrophagocytosis and absence of platelets on cytologic examination differentiate iatrogenic hemorrhage from a true hemorrhagic effusion (unless peracute). Hemorrhage within the pleural cavity can be caused by a severe coagulopathy, often associated with ingestion of an anticoagulant rodenticide (see Chapter 111). Blunt or penetrating trauma, diaphragmatic hernia, hiatal hernia, thymic hemorrhage, neoplasia, pulmonary thromboembolism, lung lobe torsion, *Spirocerca lupi*, pancreatitis, and dirofilariasis are other reported causes. The most common cause of spontaneous hemothorax in dogs with a normal coagulation profile is neoplasia.[48] Finally, iatrogenic hemorrhage may be caused by venipuncture, jugular catheter placement, Swan-Ganz catheter placement, thoracocentesis, intrathoracic biopsy, and intrathoracic fine-needle aspiration, and may occur after thoracotomy or herniorrhaphy.

Cardiovascular shock often precedes respiratory compromise because as much as 30 to 60 ml/kg (dogs) or 20 ml/kg (cats) of pleural effusion is required to impair ventilation in those animals with normal lungs.[49,50] Therefore treatment includes appropriate fluid resuscitation and blood transfusions as needed. Only sufficient blood should be retrieved from the pleural space to relieve dyspnea and allow adequate oxygenation because the red blood cells that remain will be reabsorbed over the ensuing several days. Autotransfusion should be considered in trauma patients if more than 10 ml/kg of effusion is present.[49] Thoracostomy tube placement should be considered if the animal cannot be stabilized with thoracocentesis and the hemorrhage is ongoing (see Chapter 199). Surgery is rarely indicated in animals with a traumatic hemothorax unless a penetrating injury or uncontrollable hemorrhage is present but is often necessary for noncoagulopathic spontaneous hemothoraces.

Neoplastic Effusions and Pleural Neoplasia

Intrathoracic neoplasia may result in transudates or exudates by causing increased vascular permeability, obstruction of pleural and pulmonary lymphatic vessels or veins, shedding of necrotic material at the pleural surface (increasing oncotic pressure within pleural space), and obstruction or perforation of the thoracic duct.[51] Hemorrhage and pneumothorax may also result from neoplasia. Common primary thoracic cancers include mesothelioma, pulmonary carcinomas, and lymphosarcoma, but metastatic disease can also result in pleural abnormalities. Fluid analysis and cytologic studies are informative, but thoracic ultrasonography, computed tomography, thoracotomy, or thoracoscopy with fine-needle aspiration or biopsy will often be necessary to obtain a definitive diagnosis. In addition to treating the underlying neoplasia, long-term and palliative management of neoplastic effusions can be achieved in some patients by surgically creating a drainage system, such as placement of vascular access ports with intrathoracic drains or thoracic omentalization.[52,53] In human medicine, chemical pleurodesis is often also performed palliatively.[54] Intracavitary chemotherapy may also prove beneficial in some cases.

Fibrosing Pleuritis

Fibrosing pleuritis is a chronic condition in which the visceral pleura becomes thickened and restricts lung expansion as a result of inflammation within the thoracic cavity. Causes of this condition in humans include chylothorax, hemothorax, pleural infection, drugs, neoplasia, asbestosis, rheumatoid pleurisy, coronary bypass surgery, and uremia.[51] In veterinary medicine, this pathology is most often associated with chylous effusion.[55] Development of fibrosis depends on the degree of mesothelial cell and basement membrane damage and regeneration.[55] Radiographs show rounded, retracted lung lobes that will not expand after thoracocentesis. Pulmonary edema and interstitial fibrosis may contribute to dyspnea.[56] Decortication is the only successful therapy in humans and should be considered early for better outcome, while pulmonary changes are minimal. Pneumothorax is a common complication, and reexpansion pulmonary edema is also possible. The prognosis is guarded with diffuse disease.[56]

PNEUMOTHORAX

A pneumothorax is open if it results from an insult to the thoracic wall, such as a penetrating thoracic trauma. In patients with a closed pneumothorax, the thoracic cavity is intact and the air originates from a lesion within the lung parenchyma, trachea, airways, esophagus, mediastinum, or diaphragm. A tension pneumothorax develops if the site of air leakage creates a one-way valve during inspiration and results in a rapidly increasing pleural pressure that exceeds atmospheric pressure.

Traumatic pneumothorax is a common sequela of motor vehicular accidents and was found concurrently in 47% of dogs with pulmonary contusions.[57] It has also been reported in most (63%) cats with high-rise syndrome.[58] External wounds, such as a projectile injury, bite wounds, and penetrating sharp objects to the thorax and cervical spine, are also common causes. Iatrogenic pneumothorax after thoracocentesis is common, with an incidence of 3% to 20% in humans, with approximately 20% of those patients requiring thoracostomy tube placement.[20] Other common iatrogenic causes include leakage after lung lobectomy or respiratory tract surgery, thoracostomy tubes, fine-needle lung aspiration, barotrauma during positive pressure ventilation, and tracheal tears. Spontaneous pneumothorax is most often associated with pulmonary bullous emphysema in dogs, with the Siberian Husky being overrepresented.[59] Multiple other pathologic conditions can lead to a spontaneous pneumothorax:

neoplasia, feline asthma, pulmonary abscess, heartworm disease and other parasitic infections, foreign body migration, subpleural blebs, and pneumonia.[6] Finally, an infectious pneumothorax can be created by gas-forming bacteria within the thoracic cavity.

A tension pneumothorax can rapidly become life threatening, and immediate thoracocentesis is indicated in animals suspected to have this condition. If the pneumothorax is not easily relieved with thoracocentesis, an emergency mini-thoracotomy or rapid placement of a thoracostomy tube with intubation and mechanical ventilation may prove lifesaving. Decreased venous return to the thorax in animals with a tension pneumothorax can be associated with cardiovascular collapse and shock. The thorax may become barrel shaped, and limited chest expansion is noted despite significant respiratory effort. However, animals with subclinical air accumulation may not require thoracocentesis and the animal's progression should be followed closely because the air will be reabsorbed over days to weeks. A small amount of air in animals with severe pulmonary pathology may contribute significantly to dyspnea and should be relieved. Most patients with a closed traumatic or iatrogenic pneumothorax require thoracocentesis only once or twice.

Animals should be monitored closely after thoracocentesis for return of dyspnea, and cage rest is recommended for 2 weeks. The indications for a thoracostomy tube vary according to the clinical situation, but a tube should be placed in patients requiring more than two thoracocenteses within 6 to 12 hours (see Chapter 199). Other indications include patients with a tension pneumothorax and those with a pneumothorax that require mechanical ventilation.

Constant negative pressure applied within the pleural cavity is recommended using a two-chambered or three-chambered continuous suction device or commercially available Pleur-evac. Alternatively, a Heimlich valve may be used in medium and large breed dogs (although caution should be exercised if fluid accumulation is also present within the pleural space).

An exploratory thoracostomy is indicated if a closed traumatic pneumothorax does not resolve after 3 to 5 days of drainage. If an open pneumothorax is caused by a penetrating injury, the injury should be covered with an occlusive bandage and thoracocentesis performed; surgical repair is required as soon as the patient is stable. A spontaneous pneumothorax in dogs is best treated with surgical exploration, leading to a higher survival rate and decreased recurrence.[59] Thoracoscopic lobectomy has also been described in these patients.[60] Overall prognosis is good, with an 86% survival rate for treated dogs and cats with various causes of air accumulation.[61]

SPACE-OCCUPYING LESIONS

Space-occupying lesions within the pleural space may occur secondary to benign or malignant masses within the mediastinum or chest wall. These typically are diagnosed with thoracic radiographs or computed tomography. Further details on these diseases are beyond the scope of this chapter.

DIAPHRAGMATIC HERNIA

Acquired diaphragmatic hernias are usually the result of blunt trauma associated with vehicular trauma, high-rise syndrome, or dog fighting or attacks but may also be iatrogenic. Congenital diaphragmatic hernias are a result of aberrant embryogenesis and may be pleuroperitoneal, peritoneopericardial, or hiatal. These hernias are rare and beyond the scope of this chapter.

Clinical signs may occur immediately after the traumatic event but are considered chronic if present for more than 2 weeks.[62,63] Dyspnea varies from none to severe according to the organ herniated, resulting pleural effusion, and concomitant thoracic injuries. The organs most often involved are the liver, stomach, and small intestine; the omentum and spleen are also commonly herniated.[62-64] On physical examination, borborygmus over the chest or asymmetrically quiet heart or lung sounds may be ausculted. The abdomen may be further tucked in or palpated "empty," with failure to distinguish certain organs. Thoracic radiographs may reveal gas-filled abdominal organs within the thorax, an incomplete diaphragmatic border, pleural effusion, or cranially displaced abdominal organs. Additional radiographic views, ultrasonography, positive contrast celiography, and an upper gastrointestinal contrast study may aid in the diagnosis.

Thoracocentesis and gastrocentesis may relieve the dyspnea before surgery. Cardiovascular stabilization before surgery is also important. Indications for immediate surgical intervention include herniated stomach, strangulated bowel or organ, inability to oxygenate properly after medical intervention, and ruptured viscera. Most data suggest that early surgical intervention (within 24 hours of admission) provides an excellent prognosis for acute cases.[63]

Postoperative complications include pneumothorax, hemorrhage, aspiration pneumonia, sepsis, arrhythmias, and death.[62-64] Reexpansion pulmonary edema (RPE) is a rare complication after surgery. It results from release of endotoxins and oxygen free radicals, decreased surfactant concentrations, negative interstitial pressures, or chronic hypoxia causing increased vascular permeability and protein-rich pulmonary edema. Increased incidence of RPE has been associated with a longer duration of collapsed lung (≥72 hours). Care should be given to keep peak airway pressure below 20 cm H_2O to avoid positive end-expiratory pressure, and pleural air should be slowly evacuated postoperatively (>12 hours).[65] Prognosis for full recovery is excellent for acute cases (survival rate 94%).[63] Perioperative survival rate is lower (82% to 89%) when chronic acquired cases are included in the statistical analysis.[62-64] In some studies, dyspnea did not affect prognosis,[63] but older age, lower respiratory rate, and concurrent multiple injuries were associated with higher mortality in cats.[64]

REFERENCES

1. Dyce KM, Sack WO, Wensing CJC, editors: Textbook of veterinary anatomy, ed 3, St Louis, 2002, Saunders.
2. Dempsey SM, Ewing PJ: A review of the pathophysiology, classification and analysis of canine and feline cavitary effusions, J Am Anim Hosp Assoc 47:1, 2011.
3. Noone KE: Pleural effusion and diseases of the pleura, Vet Clin North Am Small Anim Pract 15:1069, 1985.
4. West JB: Respiratory physiology, ed 4, Baltimore, 1990, Williams & Wilkins.
5. West JB: Pulmonary pathophysiology: the essentials, ed 5, Philadelphia, 1995, Lippincott Williams & Wilkins.
6. King LG, editor: Textbook of respiratory disease in dogs and cats, St Louis, 2004, Saunders.
7. Le Boedec K, Arnaud C, Chetboul V: Relationship between paradoxical breathing and pleural diseases in dyspneic dogs and cats: 389 cases (2011-2009), J Am Vet Med Assoc 240(9):1095, 2012.
8. Lynch KC, Oliveira CR, Matheson JS, et al: Detection of pneumothorax and pleural effusion with horizontal beam radiography, Vet Radiol Ultrasound 53(1):38, 2012.
9. Lisciandro GR, Lagutchik MS, Mann KA: Evaluation of thoracic focused assessment for trauma (TFAST) protocols to detect pneumothorax and concurrent thoracic injury in 145 traumatized dogs, J Vet Emerg Crit Care 18(3):258, 2008.
10. Lischiandro GR: Abdominal and thoracic focused assessment with sonography for trauma, triage, and monitoring in small animals, J Vet Emerg Crit Care 21(2):104, 2011.
11. Lichtenstein DA, Menu Y: A bedside ultrasound sign ruling out pneumothorax in the critically ill. Lung sliding, Chest 108:1345, 1995.
12. Schwarz LA, Tidwell AS: Alternative imaging of the lung, Clin Tech Small Anim Pract 14:187, 1999.

13. Larson MM: Ultrasound of the thorax (noncardiac), Vet Clin North Am Small Anim Pract 39(4):733, 2009.
14. Reetz JA, Buza EL, Krick EL: CT features of pleural masses and nodules, Vet Radiol Ultrasound 53(2):121, 2012.
15. Kovak JR, Bergman PJ, Baer KE: Use of thoracoscopy to determine the etiology of pleural effusion in dogs and cats: 18 cases (1998-2001), J Am Vet Med Assoc 221(7):990, 2002.
16. Radslinsky MG: Complications and need for conversion from thoracoscopy to thoracotomy in small animals, Vet Clin North Am Small Anim Pract 39(5):977, 2009.
17. Schmiedt C: Small animal exploratory thoracoscopy, Vet Clin North Am Small Anim Pract 39(5):953, 2009.
18. Monet E: Interventional thoracoscopy in small animals, Vet Clin North Am Small Anim Pract 39(5): 965-975, 2009.
19. American Thoracic Society: Guidelines for thoracentesis and needle biopsy of the pleura, Am Rev Respir Dis 140:257, 1989.
20. Collins TR, Sahn SA: Thoracentesis: clinical value, complications, technical problems, and patient experience, Chest 121:178, 2002.
21. Cowell RL, Tyler RD, Meinkoth JH: Diagnostic cytology and hematology of the dog and cat, ed 2, St Louis, Mosby, 1999.
22. Zoia A, Slater LA, Heller J: A new approach to pleural effusion in cats: markers for distinguishing transudates from exudates, J Feline Med Surg 11(10):847, 2009.
23. Greene C: Infectious diseases of the dog and cat, ed 3, St Louis, 2006, Saunders.
24. Peterson NC: A review of feline infectious peritonitis virus infection: 1963-2008, J Feline Med Surg 11:225, 2009.
25. Hartmann K, Binder C, Hirschberger J: Comparison of different tests to diagnose feline infectious peritonitis, J Vet Intern Med 17:781, 2005.
26. Waddell LS, Brady CA, Drobatz KJ: Risk factors, prognostic indicators, and outcome of pyothorax in cats: 80 cases (1986-1999), J Am Vet Med Assoc 221:819, 2002.
27. Barrs VR, Beatty JA: Feline pyothorax—new insights into an old problem. Part 1. Aetiopathogenesis and diagnostic investigation, Vet J 179:163, 2009.
28. Barrs VR: Feline pyothorax: a retrospective study of 27 cases in Australia, J Feline Med Surg 7(4):211, 2005.
29. Anastasio J, Sharp C, Needle D: Histopathology of lung lobes in cats with pyothorax: 17 cases (1987-2010). In Small Animal IVECCS Abstracts 2012, presented at 18th International Veterinary Emergency & Critical Care Symposium, San Antonio, TX, Sept 8-12, 2012.
30. Demetriou JL, Foale RD, Ladlow J et al: Canine and feline pyothorax: a retrospective study of 50 cases in the UK and Ireland, J Small Anim Pract 43:388, 2002.
31. Scott JA, Macintire DK: Canine pyothorax: pleural anatomy and pathophysiology, Compendium 25:172, 2003.
32. Jimanez Pelaiez M, Jolliffe C: Thoracoscopic foreign body removal and right middle lung lobectomy to treat pyothorax in a dog, J Small Anim Pract 53(4):240, 2012.
33. Boothe HW, Howe LM, Boothe DM: Evaluation of outcomes in dogs treated for pyothorax: 46 cases (1983-2001), J Am Vet Med Assoc 236(6):657, 2010.
34. Scott JA, Macintire DK: Canine pyothorax: clinical presentation, diagnosis and treatment, Compendium 25:180, 2003.
35. Walker AL, Spencer JS, Hirsh DC: Bacteria associated with pyothorax of dogs and cats: 98 cases (1989-1998), J Am Vet Med Assoc 216:359, 2000.
36. Rooney MB, Monnet E: Medical and surgical treatment of pyothorax in dogs: 26 cases (1991-2001), J Am Vet Med Assoc 221:86, 2002.
37. Dovie JL, Kuipers RG, Worth AJ: Intra-thoracic pyogranulomatous disease in four working dogs, N Z Vet J 57(6):346, 2009.
38. Jang SS, Breher JE, Dabaco LA, et al: Organisms from dogs and cats with anaerobic infections and susceptibility to selected antimicrobial agents, J Vet Intern Med 210:1610, 1997.
39. Crawford AH, Halfacree ZJ, Lee KCL: Clinical outcome following pneumonectomy for management of chronic pyothorax in four cats, J Feline Med Surg 13(10):762, 2011.
40. Barrs VR, Beatty JA: Feline pyothorax—new insights into an old problem. Part 2. Treatment recommendations and prophylaxis, Vet J 179:171, 2009.
41. Birchard SJ, Smeak DD, McLoughlin MA: Treatment of idiopathic chylothorax in dogs and cats, J Am Vet Med Assoc 212:652, 1998.
42. Greenberg MJ, Weisse CW: Spontaneous resolution of chylothorax in a cat, J Am Vet Med Assoc 226:1667, 2005.
43. Fossum TW, Mertens MM, Miller MW: Thoracic duct ligation and pericardectomy for treatment of idiopathic chylothorax, J Vet Intern Med 18:307, 2004.
44. Thompson MS, Cohn LA, Jordan RC: Use of rutin for medical treatment of idiopathic chylothorax in four cats, J Am Vet Med Assoc 3:345, 1999.
45. Radlinsky MG, Mason DE, Biller DS, et al: Thoracoscopic visualization and ligation of the thoracic duct in dogs, Vet Surg 31:138, 2002.
46. Mayhew PD, Culp WTN, Mayhew KN: Minimally invasive treatment of idiopathic chylothorax in dogs by thoracoscopic thoracic duct ligation and subphrenic pericardectomy: 6 cases (2007-2010), J Am Vet Med Assoc 241(7):904, 2012.
47. da Silva CA, Monnet E: Long-term outcome of dogs treated surgically for idiopathic chylothorax: 11 cases (1995-2009), J Am Vet Med Assoc 239(1):107, 2011.
48. Nakamura RK, Rozanski EA, Rush JE: Non-coagulopathic spontaneous hemothorax in dogs, J Vet Emerg Crit Care 18(3):292, 2008.
49. Ludwig LL: Surgical emergencies of the respiratory system, Vet Clin North Am Small Anim Pract 30:531, 2000.
50. Cockshutt JR: Treatment of fracture-associated thoracic trauma, Vet Clin North Am Small Anim Pract 25:1031, 1995.
51. Taubert J: Treatment of malignant pleural effusion, Nurs Clin North Am 36:665, 2001.
52. Talavera J, Agut A, del Palacio JF: Thoracic omentalization for long-term management of neoplastic pleural effusion in a cat, J Am Vet Med Assoc 234(10):1299, 2009.
53. Cahalane AK, Flanders JA, Steffey MA: Use of vascular access ports with intrathoracic drains for treatment of pleural effusion in three dogs, J Am Vet Med Assoc 230(4):527, 2007.
54. American Thoracic Society: Management of malignant pleural effusion, Am J Respir Crit Care Med 162:1987, 2000.
55. Huggins JT, Sahn SA: Causes and treatment of pleural fibrosis, Respirology 9:441, 2004.
56. Fossum TW, Evering WN, Miller MW, et al: Severe bilateral fibrosing pleuritis associated with chronic chylothorax in five cats and two dogs, J Am Vet Med Assoc 201:317, 1992.
57. Powell LL, Rozanski EA, Tidwell AS, et al: A retrospective analysis of pulmonary contusion secondary to motor vehicular accidents in 143 dogs: 1994-1997, J Vet Emerg Crit Care 9:127, 1999.
58. Kapatkin AS, Matthiesen DT: Feline high-rise syndrome, Comp Cont Educ Vet Pract 13:1389, 1991.
59. Puerto DA, Brockman DJ, Lindquist C, et al: Surgical and nonsurgical treatment of and selected risk factors for spontaneous pneumothorax in dogs: 64 cases (1986-1999), J Am Vet Med Assoc 220:1670, 2002.
60. Brissot HN, Dupre GP, Bouvy MW et al: Thoracoscopic treatment of bullous emphysema in three dogs, Vet Surg 32:524, 2003.
61. Krahwinkel DJ, Rohrbach BW, Hollis BA: Factors associated with survival in dogs and cats with pneumothorax, J Vet Emerg Crit Care 9:7, 1999.
62. Minihan AC, Berg J, Evans KL: Chronic diaphragmatic hernia in 34 dogs and 16 cats, J Am Anim Hosp Assoc 40:51, 2004.
63. Gibson TWG, Brisson BA, Sears W: Perioperative survival rates after surgery for diaphragmatic hernia in dogs and cats: 92 cases (1990-2002), J Am Vet Med Assoc 227:105, 2005.
64. Schmiedt CW, Tobias KM, Stevenson MA: Traumatic diaphragmatic hernia in cats: 34 cases (1991-2001), J Am Vet Med Assoc 222:1237, 2003.
65. Stampley AR, Waldron DR: Reexpansion pulmonary edema after surgery to repair a diaphragmatic hernia in a cat, J Am Vet Med Assoc 203:1699, 1993.