# FELINE PLEURAL DISEASE
## Diagnosis and Treatment

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## INTRODUCTION

Diseases of the pleural space, including pneumothorax, pleural effusion, and space occupying mass lesions (tumor, diaphragmatic hernia) are relatively common disorders in small animal clinical practice. The abnormal presence of air, fluid and/or tissue within the pleural cavity does not generally reflect disease of the pleura per se; but instead represents a disorder of the airways and/or lung parenchyma, or the development of a primary systemic illness.

Regardless of the cause(s), a significant collection of air or fluid within the pleural cavity can represent a potentially life threatening condition and this must be the first priority of the clinician. This manuscript will discuss common causes of the most frequently seen pleural space disorders, and outline general treatment plans that can be used as guidelines when treating actual clinical cases.

## SPECIFIC DISORDERS

### *Pneumothorax*

Trauma is the most commonly recognized cause of pneumothorax. Blunt chest trauma, such as being hit by a car, is the most frequently seen condition that cause pneumothorax. Less commonly, pneumothorax may result from penetrating chest injuries, rupture of lung lesions associated with infection (pneumonia) tumor (primary or metastatic) or congenital blebs or bullae (unusual). When trauma creates a "one-way" flap valve from a portion of the injured chest wall, air flows into the chest cavity on inspiration only. This results in pressure within the chest that exceeds atmospheric pressure, and is known as a "tension pneumothorax". This condition is rapidly fatal if not treated promptly and aggressively.

*Clinical signs* of pneumothorax may be subtle or dramatic, and progress from rapid shallow breathing to open mouth panting as the condition worsens. If there is a slow accumulation of air, the early signs of respiratory difficulty may be on inspiration only. Rapid shallow breathing is a vagal reflex, and is not generally due to low oxygen tension or a significant acid base disorder. In fact, if an animal does not have underlying parenchymal lung disease, 60 cc/kg of air must be present within the pleural cavity before *significant* labored breathing occurs; 30 cc/kg of free pleural air is required to cause subtle clinical signs.

*Diagnosis* of pneumothorax is usually made on the basis of rapid shallow breathing and the classic radiographic appearance of an "elevated" heart, retraction of the lung from the chest wall and increased density of collapsed long lobes. If the chest radiograph is hard to interpret, the disorder can be confirmed if thoracocentesis produces free pleural air.

*Treatment*: Because the most common cause of pneumothorax is trauma, the author emphasizes the importance of pain relief for these patients. Not only is it the humane and right thing to do, but the painful animal will avoid deep breathing. This reluctance to make a full inspiratory effort will worsen the hypoventilatory state.

It is important to recognize that only about 10-15 cc/kg of air needs be removed for clinical signs to be greatly relieved, at least in the short term:

1. Air should be removed slowly from the pleural cavity, using a 60 cc syringe and a 3-way stopcock. Rapid removal of air using an electric suction device is ill advised. This is because rapid expansion of a collapsed lung lobe is associated with a phenomenon known as "re-expansion pulmonary edema". In this setting micro capillaries within the rapidly expanding lobe rupture and leak their fluid contents into the parenchyma of the lung. Rapid expansion of a lung lobe may also result in displacement of a recently formed fibrin seal that might have formed over the original source of the air leak.
2. Animals with pneumothorax should be treated with 100% oxygen as soon as possible. When the animal inhales 100% oxygen the dissolved gas pressure drops within the pulmonary blood vessels. The dissolved gas pressure of the free air in the pleural space is higher than the recently lowered pressure in the pleural vessels. Thus, by administering 100% oxygen you have established a pressure gradient that drives the free pleural air into the pleural blood vessels, this gradient can increase the speed of absorption of free air by a factor of 500%.
3. The decision to place one or more chest tubes instead of frequent chest tapping with a needle and syringe apparatus is often a practical one, and there are no controlled studies documenting the "proper" time to place chest tubes in animals. In some cases, air can most efficiently be removed using a “Heimlich” valve. This device was created one half century ago to treat traumatic pneumothorax in wartime conditions, and is based on the principle that positive pressure from within the chest will cause air to leave through the open rubber flutter valve during expiration. Conversely, the flutter valve collapses as negative pressure is created during inspiration, and no additional air will enter the chest.
4. I routinely place a single chest tube when the clinical signs associated with a pneumothorax recur more than once after needle drainage. This chest tube, ideally, should be attached to a constant drainage device such as a commercially available pleurovac. A two or three bottle system drainage device is equally effective, less costly, and can be created from bottles and tubing found in any veterinary practice.

***Solid Tissue Mass***

The abnormal presence of solid tissue within the pleural cavity is almost always the result of a cranial mediastinal mass (CMM) or displaced abdominal organs through a diaphragmatic hernia (DH). Mediastinal tumors are quite rare and are usually malignant. Diaphragmatic hernia may be congenital or may result from abdominal trauma at a time when the glottis is closed. The resulting pressure differential across the diaphragm can lead to rupture and herniation of abdominal viscera into the thorax. Interestingly, cats seem predisposed to right sided herniation.

*Clinical signs* are the result of the space occupying mass, and include rapid shallow breathing and occasional asymmetry of chest wall motion.

*Diagnosis* of CMM or DH can often be made during the physical examination. Palpation may reveal a non-compliant thoracic cage or displacement of the cardiac impulse from its characteristic location at the 5th intercostal space in the left hemithorax (see article in this edition on diagnostic techniques). Auscultation may reveal displacement of this maximal cardiac impulse, dull cardiac tones, or borborygmus if a bowel loop is present in the field of auscultation. Occult diaphragmatic hernia or small CMM may be diagnosed during routine thoracic radiography. When it is not clear if the diaphragm is intact, a diagnosis of DH may be made by ultrasonography. If this is not available, a dilute contrast agent placed in the abdominal cavity may be visible radiographically in the thorax within one hour.

*Treatment* of CMM is based on current recommendations of veterinary oncologists. Diaphragmatic hernia may be present for months or years without causing clinical signs. The author strongly believes that DH should be surgically repaired at the time of diagnosis, whether or not clinical signs of DH are present. There is no advantage to waiting until signs appear, because this serves to increase the peri- and post-operative mortality associated with the surgical repair.

***Pleural Effusion and Hemothorax***

The pleural cavity is composed of two potential spaces separated by a fenestrated mediastinum. The pleural cavity in the dog usually contains less than 5 cc of fluid, and a similar amount in the healthy cat is assumed. Excess fluid accumulation may be the result of increased production (systemic hypertension, decreased colloid pressure, increased capillary permeability) or decreased drainage (venous hypertension, lymphatic obstruction).

*Clinical signs* of pleural effusion are similar to those associated with pneumothorax, and include rapid shallow breathing. In the absence of lung parenchymal disease, clinical signs of serious respiratory embarrassment are not evident until at least 30-60 cc/kg body weight of pleural fluid has accumulated. (see notes on pneumothorax)

*Diagnosis* of pleural effusion often begins with recognition that the pet has respiratory difficulty with a rapid shallow breathing pattern. Auscultation of the heart may reveal muffling of heart tones. Percussion of the chest (of limited value in diagnosis of feline chest disease) may result in dull percussive notes. If this is recognized it can be used to distinguish pneumothorax from pleural effusion. Chest radiographs classically reveal retraction of lobar borders from the thoracic walls, thickening of interlobar fissures, and blunted cardiophrenic angles. Confirmation of pleural effusion ideally requires aspiration of free pleural fluid, although loculated fluid may be difficult to aspirate. To increase the opportunity to aspirate smaller amounts of fluid, the patient may be "tilted" forward, resting on the forelegs. Alternatively, the pet may be placed onto its back so that the fluid accumulates in the "V" shaped space between the chest wall and the thoracic contents. This is the same principle that is used when a VD projection is suggested for radiography.. If the presence of free pleural fluid is in doubt, ultrasonography is a particularly sensitive method of confirming pleural effusion.

If pleural effusion is confirmed, determining the cause of the effusion usually requires cytologic and biochemical analysis of the pleural fluid. Descriptions of pleural fluid have traditionally been based upon the distinction between transudates (low specific density, low protein content, poorly cellular), exudates (high specific gravity, high protein content, highly cellular) and modified transudates (moderate cellularity, more protein than transudates). The large number of disorders that may produce modified transudative fluid, a category that does not point to any particular disorder, limits this somewhat outdated classification scheme. We have reported a classification scheme for analysis of **feline** pleural fluid that is based on the method used to classify human pleural effusion. (This system is not applicable to pleural fluid from canine patients). In this system, the first step is to determine if the pleural fluid is a transudate or an exudate. Transudates contain ≤ 200 IU/L of the enzyme lactic dehydrogenase (LDH). *Importantly, if a transudate is found, further fluid analysis including cell content and differential, specific gravity measurement, culture, etc. is not necessary.*

1. Transudates in cats are caused almost exclusively by congestive heart failure (right or left sided), hypoproteinemia or excessive intravenous fluid administration. In the case of hyproteinemia, transudates do not generally form unless albumin is less than 1.5 g/dl. In these cases there is usually fluid found in other potential spaces, or in the abdomen.
2. Fluid with an LDH content > 200 IU/L is classified as an exudate.
3. Exudative fluid with a ph of 6.9 or less is always due to pyothorax. Therefore, if pleural fluid analysis confirms that the fluid is an exudate and has a ph of 6.9 or less, broad spectrum antibiotic therapy is indicated before culture results confirm infection. Additionally, infected pleural fluid almost always contains less than 50 mg/dl glucose and > 85% neutrophils.
4. In contrast, pleural effusions associated with malignancy are also exudative, but have a normal or high pH (7.4 or greater), glucose usually >10 <80 and <30% neutrophils on average. Thus, exudative pleural fluid effusions with low/normal pH, low glucose and low neutrophils counts are most often associated with malignancy.
5. In the absence of trauma, an additional indicator of pleural effusion associated with malignancy is a red blood cell count > 50,000/ul.
6. 

*Treatment* of pleural effusion must first address the immediate presence of fluid and then later the cause of the fluid accumulation. The presence of infected pleural fluid is a medical emergency and will be discussed separately below. Animals with non-infected pleural effusion should be approached on a case-by case basis, with consideration to the changing clinical status of the patient. Thus, a slowly developing effusion may require only intermittent needle drainage, while a rapidly accumulating and large effusion may best be treated with continuous drainage through bilateral chest tubes.

***Pyothorax*** is defined as the presence of infected fluid within the thoracic cavity. The etiology of most cases of pyothorax in dogs is presumed to be inhalation of plant material. In the cat this is less well established. Culture results obtained from purulent thoracic fluid in cats (Pasteurella sp. and Bacteroides sp.) implies that animal bites may be a very common cause of feline pyothorax. In dogs, actinomyces and nocardia are commonly found organisms, reinforcing the need to treat both species for presumed anaerobic bacteria with or without the benefit of anaerobic culture testing. Occasionally, an infected lung may leak material into the chest cavity.

*Diagnosis* is presumed by the characteristic gross appearance of purulent material and signs of systemic infection, although culture results should be used for confirmation. As *previously cited, an exudative effusion with a pH less than 6.9 is also an excellent marker of infected material.*

*Successful treatment* of pyothorax requires early, continual drainage of the infected fluid. Although the mediastinum is fenestrated, the inflammation associated with pyothorax effectively seals this fenestration, resulting in the formation of two separate pleural cavities. If radiographic evidence of bilateral infection is present, the author recommends placement of bilateral chest tubes. Even with very sick animals, by using local anesthesia and minimal anesthesia/tranquilization the experienced clinician can insert chest tubes in a minimum of time. The particular type of chest tube that is used is basically irrelevant and should be based on the prior successful experience of the clinician.

Following initial drainage of infected material, the author recommends instillation of warmed saline through a chest tube at a dose of 10 cc/kg into one side of the chest. This fluid can be left for 3-5 minutes, while gently rotating the animal to permit optimal mixing of the saline with the infected fluid. The amount of fluid aspirated should be recorded and the fluid discarded. This procedure should then be repeated for the other side of the chest. The author routinely continues this practice 2-4 times daily for 2-5 days, as dictated by the clinical signs of the patient, radiographic appearance of the chest, and the gross and microscopic appearance of the fluid. An additional sign of decreasing infection is a rise in pH of the aspirated fluid. Systemic antibiotics are indicated, based upon culture and sensitivity data. Anaerobic infection should be assumed to be present regardless of culture results. Most anaerobes are susceptible to simple penicillin treatment. However, in practice *Bacteroides* species are generally resistant to most antibiotics but susceptible to metronidazole. For this reason, the author frequently uses a combination of a fluoroquinilone (with a simple penicillin to cover strep species) and clindamycin until culture results are obtained.

There is no evidence that instillation of antibiotics into the chest has any positive effect on morbidity or mortality in these cases.

***Chylothorax*** is most easily demonstrated in the dog by triglyceride measurement of greater than 140 mg/dl within pleural fluid. In the cat, the data is less convincing. However, in general, triglyceride values greater than 95 mg/dl or a pleural fluid/serum triglyceride ratio >10 is consistent with the diagnosis of chylothorax. Chyle is bacteriostatic, and infection associated with chylothorax is uncommon. Additionally, chyle does not usually cause a significant inflammatory response within the thorax unless it is present in large quantities for long periods of time.

*Causes* of chylothorax include mediastinal lymphoma, heart failure including heart worm infestion, pulmonary fungal infection, thromboembolic phenomena, thoracic duct rupture and thoracic lymphangectasia. Unfortunately, the cause of chylous effusions are frequently not recognized in dogs or cats.

*Treatment* of chylothorax follows the same principles as treatment of intestinal lymphangiectasia, including the use of medium chain triglycerides to bypass the production of chylomicrons. Because it may be difficult to demonstrate a specific leak in the thoracic lymph system, classical surgical treatment to ligate the thoracic duct is only sometimes successful. A surgical approach to rechannel the thoracic chyle into the abdomen is sometimes used, and is probably the surgical method of choice in dogs with no identified underlying disease.

The drug Rutin has been reported to increase reabsorption and decrease production of chyle. I have not had a positive experience with this approach in cats.

Pleurodesis has been used to obliterate the pleural space in an effort to "cure" chylothorax. The author has no primary experience using this technique.

**Most recently a surgical approach has been used by Dr Fossum at Texas A and M Veterinary School. The author refers the reader directly to Dr Fossum for this procedure.**

***Hemothorax*** refers to pleural fluid with a hematocrit of either 10%, 25% or 50% compared to whole blood, depending on the reference cited. This is different from a hemorrhagic pleural effusion, that need contain only 1-5% RBC's to impart a distinctly "bloody" cast to the appearance of the fluid. Centrifugation and measurement of hematocrit of the effusion can easily distinguish bloody fluid from hemorrhagic fluid.

*Treatment* The distinction between a hemothorax and a hemorrhagic pleural effusion is critical when deciding how to treat the two conditions. The initial treatment for a hemorrhagic pleural effusion involves drainage of as much of the fluid as can be obtained. Hemothorax however, involves an active bleed into the thorax. Because it takes 30 cc/kg to cause *any* signs and 60 cc/kg of fluid to cause *serious* clinical signs, and because the blood volume of dogs and cats is only 65-90 cc/kg, the clinical signs associated with hemothorax cannot be due to the presence of blood in the thoracic cage unless the animal bled an entire blood volume into its pleural space. Instead, clinical signs associated with hemothorax are due to decreased oxygen carrying capacity and increased sympathetic tone (associated with blood loss and vascular collapse) rather than the restrictive presence of blood in the thorax. Additionally, as much as 90% of the RBC's within the thorax can be resorbed within 4 days if left untapped, although the RBC lifespan within this fluid is less than normal. For these reasons complete removal of the hemorrhagic fluid is generally not indicated. Instead, the primary indication for complete removal of pleural fluid associated with a hemothorax is when a transfusion is planned. As noted above, a pleural fluid RBC count that exceeds 50,000/ul, in the absence of trauma is most commonly associated with neoplasia.

*Pulmonary embolism* is an infrequently recognized cause of respiratory distress. The true incidence of this condition in dogs and cats is not known, although the more we look for it the more frequently we find it. Pulmonary embolism frequently causes pleural effusion in humans, and has been identified by the author as the cause of pleural effusion in cats. Infarcted lung tissue will cause an exudative effusion. Atelectasis secondary to a pulmonary embolus causes a decrease in intrapleural pressure at the site of the collapsed lobe, leading to an accumulation of transudative fluid. While this effusion can be appreciated radiographically, it is more helpful as a clue to the diagnosis of pulmonary embolus than as a signal to perform thoracocentesis.

## CONCLUSIONS

There are many reasons why air, fluid or tissue may accumulate in the pleural cavity. In all cases, this leads to a restricted ability to breathe in, and often results in hypoventilatory respiratory failure. The presence of abnormal fluid or air is often quickly recognized on physical exam or after review of chest radiographs, and early removal of relatively small amounts of free air or fluid can be life saving. If the clinician is not sure if there is free air or fluid in the chest, the author strongly believes in performing a diagnostic needle thoracentesis in virtually all cases.

In cats, a new system of classifying pleural fluid may allow the practitioner to more quickly recognize the cause(s) of abnormal pleural fluid, and begin appropriate treatment much sooner than was previously possible.