**Dealing with Pleural space disease in the ER**

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The pleural space is a potential space bounded by the parietal and visceral pleura, within which there is normally only a small amount of serous fluid (1–5 ml) to allow motion of the lungs during respiration. The pleura is a serosal surface composed of a thin epithelium of mesothelial cells overlying a basal membrane, along with a superficial network of blood vessels, lymphatic ducts and few nerves. The parietal pleura, which lines the thoracic walls, mediastinum and diaphragm, is rich in lymphatics and receives its blood supply from the systemic circulation. The visceral pleural covers the serosal surface of the lungs, and its blood supply is provided by the pulmonary circulation. In normal animals, there is continuous production of a small amount of pleural fluid from the capillaries of the parietal pleura, and this is absorbed by the visceral pleural capillaries and parietal lymphatic network. In small animals, even though the partition between the right and left hemithoraces is incomplete, disease can sometimes be unevenly distributed or unilateral.

# Pathophysiology

In the normal thoracic cavity, hydrostatic pressure favors fluid flux into the pleural space (hydrostatic pressure in the systemic capillaries to the parietal pleura is around 30 cm H2O while that of the pleural space is about –5 cm H2O) and oncotic pressure favors reabsorption of fluid from the pleura (the colloid osmotic pressure of the pleural space is 2–3 cm H2O in dogs, compared to 24.5 to 27 cm H2O in the vascular space). As part of the systemic circulation, the parietal pleura has a greater filtration capacity than the visceral pleural (pulmonary circulation). The visceral pleural plays a larger role in determining the net pressure and favors reabsorption of fluid from the pleural space. Pleural lymphatic vessels, along with mesothelial and endothelial permeability, also play a role in fluid distribution. Lymphatic flow is stimulated by normal respiratory movements, along with movement of the diaphragm and thoracic musculature. The parietal pleural lymphatics, which are more abundant than those of the visceral pleural, absorb pleural fluid and protein from the pleural space. In disease states that cause a high-protein pleural effusion, this becomes even more important since an increase in COP of the pleural fluid decreases absorption of fluid by the pulmonary capillaries. The normal average pressure within the pleural space is negative and subatmospheric (average –5 cm H2O). Pleural space disease is caused by an accumulation of air, fluid or soft tissue within the pleural cavity and causes respiratory signs due to impairment of the ability to expand the lungs. The presence of air, fluid or soft tissue will decrease the maximal tidal volume that can be achieved at peak inspiration, reducing lung vital capacity and inspiratory reserve volume. The same is true at end expiration, thus functional residual capacity is reduced as well as expiratory reserve volume. These changes predispose the lungs to collapse and atelectasis, resulting in hypoxemia and hypoventilation. Prompt recognition of pleural space anomalies and rapid treatment are of utmost importance to ensure patient stability for further diagnostics and treatment.

# Clinical presentation

Physical exam findings suggestive of pleural space disease may include a restrictive breathing pattern (fast and shallow breathing), tachypnea, open-mouth breathing, an increased abdominal component, orthopnea or reluctance to lie down, and cyanosis. Restrictive breathing and asynchronous breathing (outward movement of the chest and inward movement of the abdomen during inspiration) patterns have classically been described as signs of pleural space disease. Because pleural space disease reduces functional residual capacity, the lungs must operate on a less compliant area of the compliance curve. Hypothetically, a patient could compensate for this change by increasing the rate of breathing, resulting in a restrictive pattern. Additionally, when the inspiratory intercostal muscles have to work harder against increased intrapleural pressure, the ribs are elevated during inspiration and the abdominal contents move toward and into thorax, resulting in asynchronous breathing. Two recent veterinary studies have evaluated the association of respiratory patterns with underlying disease. In one study, short and shallow respiration was not associated with pleural space disease and was actually more common in cats without respiratory disease, possibly secondary to stress. In that same study, pleural space disease was significantly associated with a costoabdominal breathing pattern (increased abdominal effort) and asynchronous breathing in both dogs and cats. Another study also found that asynchronous breathing was strongly associated with pleural space diseases in both dogs and cats. While these respiratory patterns should prompt one to look further for evidence of pleural space disease, many patients may not exhibit them, and a lack of such patterns should not rule out pathology in the pleural space. The cats also often exhibit an inspiratory respiratory distress, but compared to upper airway obstruction, pleural space disease respiratory distress is not associated with inspiratory noises.

The degree of respiratory distress may also vary depending on the amount of air, fluid or soft tissue within the pleural space, as well as the rate of accumulation. Concomitant disease processes may also contribute to a patient's respiratory signs. Other signs of pleural space disease include muffled or dull breath sounds on auscultation unilaterally or bilaterally, and muffled or displaced heart sounds with focal or unilateral disease. A lack of chest compressibility of the cranial thorax in cats may also be suggestive of pleural effusion or the presence of a mediastinal mass.

# Initial stabilization

As every other cause of respiratory distress, first stabilization relies on oxygen supplementation and sedation. If tolerated by the patient (and only if tolerated), an IV catheter should be placed to allow for sedation if needed and vascular access if the patient arrests during handling.

Ultrasonography is very useful for pleural space evaluation. A quick scan of the thorax should be done at admission, in order of rule in or rule out pleural space disease. Thoracic radiographs are not necessary if there is a strong clinical suspicion of pleural effusion or effusion can be

confirmed by ultrasound, and radiography may pose a significant risk to a patient in severe respiratory distress. Radiographs after thoracocentesis may show an underlying cause that would have been obscured by the presence of fluid and collapse of the lung lobes.

# Thoracocentesis and chest tube

Once pleural effusion is confirmed by ultrasound, thoracocentesis should be performed promptly. To perform thoracocentesis, a needle, butterfly catheter or peripheral catheter can be used to access the pleural space. Other necessary supplies include clippers, scrub, sterile gloves, an extension set, a 3-way stopcock and a collection syringe (10 to 60 ml, depending on patient size). The patient should be positioned in sternal recumbency or the most comfortable position to minimize stress. Blind thoracocentesis can be performed at the 7th to 9th intercostal spaces (dorsally for air, ventrally for fluid), but it is safer to find the best location (zone of greater fluid accumulation) with the ultrasound. The needle should be inserted cranial to the rib to avoid the nerves and vessels that run caudally and perpendicular to the chest wall. The needle is then advanced slowly through the skin and into the intrathoracic space while aspirating gently;

The collected liquid should be quantified and analyzed to determine the most likely cause. The air should be quantified during aspiration.

Generally, placement of a chest tube is considered when negative pressure cannot be obtained during thoracocentesis or when multiple taps are performed in a short period of time (more than 2 thoracocentesis in 24 hours) in cases of pneumothorax, or for management of pyothorax, large-volume effusions, or postoperative thoracotomies.

# Fluid analysis

The maximum amount of fluid or air present in the pleural space should be removed, except in case of hemothorax. In this particular case, the minimum amount to improve the respiratory distress will be removed.

Once the fluid is removed, attention should be paid on macroscopic aspect of the fluid: brown and smelly fluid will be consistent with pyothorax, white fluid with chylothorax…

Fluid can be classified as pure transudate, modified transudate and exudate based on total protein and total nucleated cell counts (TNCC).

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| Fluid type | Characteristics | Etiology |
| Pure transudate | TP < 2.5 g/dl  TNCC < 1500/µl | Heart failure  Hypoalbuminemia  Pericardial disease |
| Modified transudate | TP 2.5 to 7.5 g/dl  TNCC 1000 to 7000/µl | Chronic transudate |
| Exudate | TP > 30 g/dl  TNCC > 7000/µl | FIP  Neoplasia  Lung lobe torsion  Pyothorax  Hemothorax |

Other complementary exams that should be done are:

* Confirming an hemothorax: the hematocrit of the fluid is at least equal to 25% of the blood hematocrit
* Confirming a chylothorax: The concentration of triglycerides in the effusion will be higher than that of blood, and that of cholesterol equal to less than that of blood. The main cause of chylothorax in the cat is heart failure.
* Confirming a pyothorax: the cytological observation of degenerate neutrophils and bacteria in intracellular position. Differential between blood glucose and effusion (difference of 0.2 g/L) and lactate (difference of 2 mmol/l) could help to raise suspicion of pyothorax.

References are available on request: [celine.pouzotnevoret@vetagro-sup.fr](mailto:celine.pouzotnevoret@vetagro-sup.fr)