Canine Pyothorax: Pleural Anatomy and Pathophysiology*

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ABSTRACT: Lymphatics, arteries, veins, capillaries, and an elastic connective tissue layer are responsible for normal physiologic functions of the pleural linings and space. Movement of pleural fluid adheres to Starling’s forces, and abnormal collections of fluid within the pleural space result from alterations in these homeostatic forces. The route of infection in most cases of pyothorax is unknown.1 Retrieval of grass awns from other parts of the respiratory tract coupled with a high number of affected sporting breeds lends support to grass awn aspiration and migration as a frequent cause of pyothorax.

Pyothorax is defined as an accumulation of septic purulent material within the pleural space.1 Other less common terms include purulent pleuritis, empyema, and thoracic empyema. Recognized over 2000 years ago in humans as a sequela to respiratory infections or penetrating chest trauma, pyothorax in veterinary medicine remains a disease with an insidious course and a delayed presentation that requires prompt diagnosis and treatment for a successful outcome.2 There have been several recent reports of canine pyothorax.3–6 This article discusses normal pleural anatomy and physiology and covers the pathophysiology of pleural infection, including sources of infection and the role of grass awns.

NORMAL PLEURAL ANATOMY AND PHYSIOLOGY

The pleurae are serous membranes that line the entire thoracic cavity, lungs, and mediastinum.7 The surface consists of a single layer of mesothelial cells, whereas a deeper layer contains a network of elastic fibers and sparse smooth muscle cells, which allow stretching of the pleurae during normal respiratory movements.7,8 Lymphatics, arteries, veins, and capillaries course through this connective tissue layer and are responsible for normal physiologic functions of the pleural space. The pleurae are subdivided into the parietal pleura, which lines the internal surface of the ribs, diaphragm, and mediastinum, and the vis-

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Cervical pleura, which covers the serosal surface of the lungs and reflects into the interlobar surfaces as a continuous sheet. The pleurae form two separate sacs (cavities). In normal animals, the pleural space is only a potential cavity containing 2 to 3 ml of transudative fluid. This fluid lubricates the pleural surfaces, allowing a sliding motion during respiration that prevents friction. The parietal pleura normally lies in direct contact with the visceral pleura; a true cavity forms only when gas or excess fluid collects in the space and separates the two layers.

In healthy dogs and cats, the mediastinal pleura is fenestrated (incomplete), allowing air and most effusions to pass freely between the right and left pleural cavities. With inflammatory conditions affecting the mediastinal pleura, it is possible for the fenestrations to become plugged, thereby trapping fluid unilaterally. However, this phenomenon is an uncommon clinical finding, and reported cases of unilateral effusions are rare in dogs and cats. The mediastinal tissue is delicate and offers little resistance to the spread of disease from one hemithorax to the other. Bilateral effusions, therefore, are more common clinically.

NORMAL PLEURAL FLUID DYNAMICS

Pleural fluid production and absorption are continual and dynamic processes dependent on a net gradient of pressures, surface area of the pleural membranes, and several drainage mechanisms. The amount of fluid present in the pleural space stays constant because of an equilibrium between fluid produced and fluid absorbed. In humans, 5 to 10 L of fluid traverses the pleural space per day. Fluid formation and movement through the pleural space adheres to Starling’s forces, including hydrostatic pressure, colloidal osmotic pressure, capillary permeability, and lymphatic function. Starling’s law is defined as follows:

\[
\text{Fluid movement} = k \cdot [(\text{HP}_c - \text{HP}_f) - (\text{COP}_c - \text{COP}_f)]
\]

where:
- \(k\) = filtration coefficient (a measure of the permeability of the capillary wall)
- \(\text{HP}_c\) = capillary hydrostatic pressure (parietal and visceral capillary networks)
- \(\text{HP}_f\) = pericapillary (pleural space) hydrostatic pressure
- \(\text{COP}_c\) = plasma colloid osmotic pressure
- \(\text{COP}_f\) = pericapillary (pleural space) colloid osmotic pressure

Normally, fluid leaves the arterial capillaries of the parietal pleura, moves across the pleural space, and is subsequently reabsorbed by the venous capillaries and lymphatic system of the visceral pleura.

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**Figure 1**—Schematic representation of pressures (cm H₂O) involved in pleural fluid formation and absorption. \(\text{HP}\) = hydrostatic pressure; \(\text{OP}\) = colloid osmotic pressure; \(P\) = pressure. (From Forrester SD, Troy GC, Fossum TW: Pleural effusions: Pathophysiology and diagnostic considerations. *Compend Contin Educ Pract Vet* 10(2):121–136, 1988; with permission.)
Parietal pleural capillaries, which arise from the systemic circulation, have a higher hydrostatic pressure than the visceral pleural capillaries, which arise from the pulmonary circulation. This difference sets up a gradient that forces the pleural fluid across the pleural cavity, which itself has a low hydrostatic pressure (Figure 1). It is thought that the visceral pleura has a greater role in fluid absorption because of the lower hydrostatic pressure and a greater vascularity.

Visceral and parietal capillary oncotic pressures are equal and are greater than those of the intrapleural cavity (Figure 1). This favors fluid absorption from the pleural cavity to the parietal and visceral capillaries.

Permeability of capillary membranes affects the movement of fluid, cells, solutes, and protein between the vascular and pleural spaces. It also determines the size of molecules that may leave the capillary and their speed. Although not clearly defined, the ultrastructure of pleural capillaries is similar to that of other capillaries within the body (i.e., capillary membranes are composed of a continuous endothelial lining). Therefore, movement of electrolytes and other solutes occurs across the cell membrane, whereas cells pass through intercellular junctions. Protein passage occurs at intercellular junctions or by pinocytosis and depends on size and shape.

Small globulins and proteins smaller than albumin pass freely, while larger globulins and lipoproteins remain in the blood. Additionally, the parietal mesothelium can also be viewed as having some filtering capability in that it is a membrane with few but large pores and low permeability to solutes. This makes it possible to sieve proteins very efficiently, thereby keeping the protein concentration of pleural fluid normally low.

Although not originally identified in Starling’s equation of fluid movement, lymphatics play a primary role in pleural fluid dynamics. Removal of cells and protein occurs via the lower mediastinal and costal pleural lymphatics. Recent evidence suggests that approximately 75% of pleural fluid is drained via the lymphatics, particularly through the parietal pleural lymphatics. Lymphatic flow is enhanced through intrinsic (smooth muscle contractions of lymphatic walls) and extrinsic (respiratory movements) influences. Furthermore, flow rate through lymphatics can increase with a greater fluid filtration by the parietal pleura. This relationship is extremely efficient; a 10-fold increase in fluid filtration results in only a 15% increase in pleural fluid volume. However, the system can become saturated, and an increase in fluid filtration beyond the maximum lymph flow results in pleural effusion.

**PRESSURE GRADIENTS**

The gradient of forces involved in pleural fluid movement is represented in Figure 1. A net pressure of 9 cm H$_2$O at the level of the parietal pleura drives fluid into the pleural space. The amount of fluid entering the space depends on the permeability of capillaries and the surface area of the parietal pleura. Absorption of fluid is favored at the level of the visceral pleura. A net pressure of 10 cm H$_2$O moves fluid from the pleural space into the visceral pleura. The increased vascularity of the visceral pleura lowers the resistance to flow of fluid. This tends to enhance net fluid absorption through the visceral pleura.

Stretching of the pleural membranes between parietal and visceral pleurae contact points prevents complete absorption of fluid from the pleural space. The contact points form as pleural fluid is absorbed. The inherent elastic action of the pleura lowers the already subatmospheric hydrostatic pressure in the pleural space. This increased negative pressure develops between the pleural contact points and decreases the overall absorption pressure. This prevents total absorption of pleural fluid.

**PATHOPHYSIOLOGY OF PLEURAL INFECTION**

Abnormal collections of fluid within the pleural space result from alterations in the homeostatic forces responsible for normal fluid movement. Specifically, increases in hydrostatic pressure, decreases in colloid osmotic pressure of pleural capillaries or increases in colloid osmotic pressure of the pleural cavity, increases in capillary permeability, and lymphatic outflow impairment can result in pleural fluid accumulation.

Of particular importance to the development of pyothorax is changes in capillary permeability and impairment of lymphatic function. Through the action of mediators such as kinins and histamines and an increased body temperature, inflammatory conditions of the pleura damage the endothelial lining of capillaries. These increase the permeability and filtration coefficient of pleural capillaries. The end result is an increased influx of fluids, proteins, cells, and macromolecules into the pleural space. Additionally, increases in local blood flow associated with inflammation can increase capillary hydrostatic pressure and further favor fluid movement into the pleural space.

Lymphatic outflow obstruction alters pleural fluid dynamics in two ways:

- Drainage of pleural fluid can be impeded when inflammation, edema, and fibrin deposition lead to thickening of the costal parietal pleura, a major lymphatic drainage point in dogs and cats.

- Decreased resorption of protein can occur when the lymphatic drainage system is blocked. Because protein
can only leave the pleural space via the lymphatics, protein concentration is normally kept low. Increased levels of protein in the pleural space lead to increases in pleural space oncotic pressure.\textsuperscript{14} Subsequently, the osmotic pressure gradient is altered so that it favors fluid movement into the pleural space.\textsuperscript{14}

**SOURCES OF INFECTION**

The route of infection in most dogs with pyothorax is usually not found\textsuperscript{17–19}; however, suspected routes include penetrating wounds to the chest, neck, or mediastinum (e.g., bites, foreign objects); esophageal perforations (e.g., *Spirocerca lupi*); lung parasites; direct extension from bacterial pneumonia; hematogenous or lymphatic spread from septic foci; spread from cervical or lumbar infections (diskospondylitis); neoplasia and abscission; iatrogenic causes from thoracocentesis or thoracic surgery; and migrating foreign bodies (grass awn).\textsuperscript{1,3,8,17,20–24} Immunosuppression has not yet been proven to be a predisposing factor in canine pyothorax.\textsuperscript{1,25}

Most investigators believe that aspiration of plant parts, colonized by commensal organisms within the oral cavity, is a common etiology of pleural infections in dogs.\textsuperscript{1,3,8,17,18,20,21,26,27} Grass awn retrieval from other portions of the respiratory tract (including the trachea, mainstem bronchi, or lungs) or from thoracic wall swellings is not uncommon.\textsuperscript{21,26,28–31} Additionally, perforation of the lung by a grass floret with subsequent infection has also been documented.\textsuperscript{27} These findings, combined with a seemingly high prevalence of infection in sporting dogs, lend support to the theory that grass awn aspiration and migration causes pyothorax. However, there are very few reported cases in which the patient actually has foreign bodies isolated in the pleural cavity either at surgery or the postmortem examination.\textsuperscript{2,3,6,28,29,32} Possible explanations include failure to visualize plant material during exploration because of small size or location, migration to other parts of the body or out of the body with continued thoracic signs, or resorption of the plant part.\textsuperscript{3,17} It has been proposed that microscopic foreign bodies of plant origin, detectable only through histopathologic examination, may serve as a continued nidus of infection that can be refractory to host immune responses and/or antibiotics.\textsuperscript{3} This theory could help explain the chronic nature and treatment resistance of pyothorax. These mechanisms should be considered when formulating a treatment plan and monitoring the response.

Species of grasses implicated vary by region of the country. In the western United States, *Hordeum* spp, commonly known as foxtail, is the principal species of importance\textsuperscript{21,32} (Figure 2). Within the Southeast, *Stipa* and *Setaria* spp, commonly known as speargrasses, buzzard grases, and feathergrasses, are reported.\textsuperscript{20,21} Other species of note include *Avena fatua* and *Bromus avenaceus*.\textsuperscript{26} The smaller size of the *Stipa* and *Setaria* spp (10 to 12 mm) facilitates easier inhalation into the respiratory tree and may account for an apparently higher number of aspiration cases when compared with the larger *Hordeum* spp (20 to 50 mm).\textsuperscript{21} Passive acquisition of grass awns, in which the awn is trapped in an animal’s haircoat and migrates into the visceral compartments, is common in California.\textsuperscript{21}

Grass awns gain access to the oral cavity when an animal is open-mouth breathing during exercise or training.\textsuperscript{20} During these times, dogs run with their mouths open, tongues extended, and upper airway (including the glottis, larynx, trachea, and bronchi) maximally dilated.\textsuperscript{26} The speed of air entering the respiratory tract is thought to be fast enough to propel the lightweight grass awns to deep portions of the respiratory tree.\textsuperscript{26} In
the southeastern United States, some species of grasses flower in early fall; thus the florets are readily available when training for hunting begins. In the western United States, Hordeum spp are a problem in late spring and summer. Furthermore, the distance between the head of the dog and the height of the grass may facilitate aspiration.

Barbs on the grass floret prevent retrograde movement once inhalation occurs (Figure 3). Active respiratory movements of the dog only further promote caudal movement of the floret down the respiratory tract. The awn becomes trapped in the narrower portions of the bronchial tree, finally migrating through the lung and settling near the dorsal attachment of the diaphragm and lodging in the thoracic wall or sublumbar region. Here commensal organisms carried from the initial exposure to the oropharyngeal mucosa are thought to initiate an infection. Coughing is usually not productive in removing the awn.

**SUMMARY**

Pyothorax is the accumulation of purulent fluid within the pleural space. In dogs, the cause of pyothorax is often unknown, but migrating grass awns are thought to be an initiating factor in many cases, especially in outdoor working dogs subjected to running in tall grasses. Other possible causes include penetrating wounds or systemic spread of other infections. Alterations in Starling's forces, such as increases in hydrostatic pressure and capillary permeability or decreases in lymphatic drainage and colloid osmotic pressure, can result in an increased tendency toward pleural fluid accumulation, contributing to the morbidity associated with canine pyothorax.

**REFERENCES**


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1. Which of the following does the parietal pleura not cover?
   a. internal surface of the ribs
   b. mediastinum
   c. serosal surface of the lungs
   d. diaphragm

2. Parietal pleura capillaries arise from the ___________ circulation and thus have a ___________ hydrostatic pressure than the visceral pleura capillaries.
   a. pulmonary; lower
   b. systemic; lower
   c. systemic; higher
   d. pulmonary; higher

3. Protein concentration of pleural fluid is normally kept low partly by
   a. normal respiratory movements.
   b. low permeability of parietal mesothelium.
   c. increased vascularity of visceral pleura.
   d. high oncotic pressure.

4. ___________ are responsible for removing cells and protein from the pleural cavity.
   a. Capillaries
   b. Lymphatics
   c. Parietal pleura veins
   d. Visceral pleura arteries

5. Fluid movement into the pleural space is favored by
   a. inflammatory mediators.
   b. increased body temperature.
   c. increases in local blood flow.
   d. all of the above

6. Which of the following is not a suspected route of infection in canine pyothorax?
   a. extension from bacterial pneumonia
   b. diskospondylitis
   c. abdominal surgery
   d. esophageal perforations

7. Which of the following is the most likely reason for a seemingly higher number of grass awn aspiration cases in the southeastern United States?
   a. longer hunting season
   b. smaller size of grass
   c. warmer summers
   d. spring flowering season

8. What percentage of pleural fluid is drained by lymphatics from the pleural space?
   a. 65%
   b. 25%
   c. 98%
   d. 75%

9. Which of the following statements regarding canine pyothorax is true?
   a. Bilateral accumulations of fluid are more common.
   b. It has been clinically proven that immunosuppressed dogs are predisposed to developing pleural infections.
   c. Coughing is effective in removing potential aspirated grass awns.
   d. Most cases have foreign bodies isolated in the thoracic cavity at surgery or postmortem examination.

10. Pleural fluid can accumulate with all of the following except
    a. lymphatic outflow obstructions.
    b. increases in capillary permeability.
    c. decreases in pleural cavity colloid osmotic pressure.
    d. decreases in pleural capillary colloid osmotic pressure.