Introduction
Bacterial pleuropneumonia, frequently referred to as pleuritis, is a common and often severe disorder of horses [1-8]. The condition involves bacterial colonization of the pulmonary parenchyma, development of pneumonia and/or pulmonary abscesses, and subsequent extension to the visceral pleura and pleural space. In humans it is noted that up to 40% of patients with bacterial pneumonia have accompanying pleural effusions [9]. While similar data is not available for the horse, the increased use of thoracic ultrasound has documented that pleural effusion is not uncommon in any horse with pneumonia and is not restricted to those horses with severe pleuropneumonia [10].

Pathogenesis
The first stage of bacterial pleuropneumonia is an exudative stage characterized by rapid outpouring of sterile pleural fluid into the pleural space in response to inflammation of the pleura. The associated pneumonic process is usually contiguous with the visceral pleura and results in increased permeability of the capillaries in the visceral pleura. If appropriate antimicrobial therapy is initiated at this stage the pleural effusion may progress no further. With progression the bacteria invade the pleural fluid from the contiguous pneumonic process and the second, fibropurulent, stage evolves. This stage is characterized by the accumulation of large amounts of pleural fluid with many neutrophils, bacteria, and cellular debris. Fibrin is deposited in a continuous sheet covering both the visceral and parietal pleural in the involved area. As this stage progresses, the tendency is for loculation and the formation of limiting membranes. These loculations prevent extension of the empyema, but make drainage of the pleural space with chest tubes increasingly difficult.

The last stage is the organization stage in which fibroblasts grow into the exudate from both the visceral and parietal pleura surfaces and produce a inelastic membrane called the pleural peel (Fig. 1). This inelastic pleural peel encases the lung and renders it virtually functionless. At this stage the exudate is thick.

Although pleuropneumonia can occur spontaneously, it is often associated with a stressful event such as transportation over an extended distance [1] or recent illness from acute viral disease. It is most commonly seen in Thoroughbred and Standardbred racehorses. Aspiration of pharyngeal secretions may play a significant role in the etiology of pleuropneumonia, as suggested by the bacterial populations responsible for pleuropneumonia. Transportation of horses usually involves an elevation in environmental temperature and relative humidity and an increase in the number bacterial organisms in the air. These changes, combined with the stress of transportation, may predispose the animal to development of lower respiratory disease. The aerobic bacteria most commonly involved in equine pleuropneumonia include β-hemolytic Streptococcus spp., Pasteurella spp., Actinobacillus spp., E. coli and Klebsiella pneumoniae. The majority of the horses have a mixed infection, with both aerobic and anaerobic bacteria. Commonly isolated anaerobes include Bacteroides spp. and Clostridium spp. A wide variety of other anaerobes are commonly found in these horses [2].

Clinical Signs
Clinical signs include fever, anorexia, depression, cough, respiratory distress, stiff gait, weight loss, sternal or limb edema and colic. In the acute stage of pleuritis, pain in the thorax may be elicited by palpation over the thoracic wall. Pain is demonstrated by grunts, intercostal muscle spasm, or even escape maneuvers by the patient. Horses may abduct their elbows and have a "catch" to inspiration. As more fluid accumulates in the pleural space and the disease becomes...
chronic, pain is less evident. Auscultation of a horse with pleuropneumonia reveals a normal lung sound in the dorsal lung field with no sounds or only bronchial tracheal sounds heard ventrally. Pleural friction rubs are often not heard because they are present only in the acute stage of the disease. If they are heard, friction rubs are present predominantly at the end of inspiration and the early part of expiration. They disappear as inflammation decreases or as pleural fluids accumulate. Cardiac sounds are often heard over a wider area of the chest than normal, probably as a result of enhanced conduction of sound through the pleural fluid. Thoracic percussion frequently confirms the impression gained from auscultation. Pleural effusion causes a dullness of the ventral aspects of the lung field and is often delineated by a horizontal line (Fig. 2).

![Figure 2. Horse with pleural effusion secondary to pleuropneumonia. Horizontal line marked by tape indicates fluid level in thorax detected by auscultation and percussion. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

**Diagnostic Procedures**

**Thoracic Ultrasonography** - Thoracic ultrasonography [10-14] is currently regarded as the preferred method to diagnose pleuropneumonia in the horse. While the value of the art of thoracic auscultation and percussion should not be undermined, clinicians managing horses with thoracic disease recognize the limitations of these tools. With the widespread use of thoracic ultrasound, the equine practitioner currently has the ability to determine not only the presence of pleuropneumonia, but also the location and the extent of the disease. Although sector scanners are superior (preferably 3.5 - 5.0 MHz transducers), linear probes can also be used to evaluate the thorax in practice. Thoracic ultrasonography in horses with pleuropneumonia allows the clinician to characterize the pleural fluid and to evaluate the severity of the underlying pulmonary disease [14]. The appearance of the pleural fluid may range from anechoic to hypoechoic, depending on the relative cellularity (Fig. 3). This fluid is usually found in the most ventral portion of the thorax and causes compression of normal healthy lung parenchyma with retraction of the lung toward the pulmonary hilus. The larger the effusion, the greater the amount of compression atelectasis and lung retraction that occurs.

![Figure 3. Sonographic appearance of large volume of anechoic pleural fluid. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

The presence of adhesions, pleural thickening, pulmonary necrosis and compression atelectasis can also be detected [14]. Fibrin has a filmy to filamentous or frond-like appearance and is usually hypoechoic (Fig. 4).

![Figure 4. Sonographic appearance of fibrin on visceral and parietal pleura in horse with severe pleuropneumonia. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)

Fibrin is deposited in layers or in web-like filamentous strands on surfaces of the lung, diaphragm, pericardium, and inner thoracic wall limit pleural fluid drainage [14]. Dimpling of the normally smooth pleural surface results in the appearance of "comet-tail" artifacts, created by small accumulations of exudate, blood, mucus, or edema fluid. Pulmonary consolidation varies from dimpling of the pleural surface to large, wedge-shaped areas of sonoluent lung. Atelectic lung is sonolucent and appears as a wedge of tissue floating in the pleural fluid. Necrotic lung appears gelatinous and lacks architectural integrity. Peripheral lung abscesses are identified ultrasonographically by their cavitated appearance and the absence of any normal pulmonary structures (vessels or bronchi) detected within. While detection of a pneumothorax may be easy for the experienced ultrasonographer it is not as easy for the less experienced. The gas-fluid interface can be imaged moving simultaneously in a dorsal to ventral direction with respiration, the "curtain sign" reproducing the movements of the diaphragm (Fig. 5).

![Figure 5. Sonographic appearance of pneumothorax. - To view this image in full size go to the IVIS website at www.ivis.org. -](image)
The dorsal air echo moves ventrally during inspiration, similar to the lowering of a curtain, gradually masking the underlying structures. A pneumothorax without pleural effusion is even more difficult to detect ultrasonographically. While free bright gas echoes within the pleural fluid can occur following thoracocentesis, they are more often seen with anaerobic infections or when sufficient necrosis has occurred in a segment of parenchyma to erode into an airway and form a bronchopleural fistula (Fig. 6). The absence of gas echoes in pleural fluid does not rule out the possibility that anaerobic infection may be present.

Ultrasonography is a valuable diagnostic aid in the evaluation of the pleura, lung, and mediastinum of horses with pleuropneumonia. The detection and further characterization of the above abnormalities improve the clinician's ability to form a more accurate prognosis. Adhesions can be detected which ultimately may affect the horse's return to his previous performance level. Horses with compression atelectasis and a non-fibrinous pleuritis have an excellent prognosis for survival and return to performance. The detection of areas of consolidation, pulmonary necrosis, or abscesses all increase the probable treatment and recovery time and the prognosis for survival decreases as they become more extensive. Ultrasonography can be used as a guide to sample or drain the area with a large fluid accumulation or the least loculation. These patients often benefit from progressive scanning to assess response to treatment and the need for drainage.

Thoracocentesis - If pleural effusion is suspected, thoracocentesis should be considered. In the acute stages of pleuropneumonia with small volumes of pleural effusion, thoracocentesis is not necessary if the horse is improving or is not showing signs of respiratory distress. Moderate amounts of pleural effusion may be resorbed quite readily. However, if fluid accumulates rapidly, if the horse is in respiratory distress, or if its condition deteriorates, thoracocentesis should be performed. The preferred site is the sixth or seventh intercostal space just dorsal to the palpable costochondral junction. Choosing a site farther caudal may provide a sample but does not allow adequate drainage of the chest. When attempting to aspirate pleural fluid from a horse with a minimum amount of effusion, one should choose a space no farther back than the sixth or seventh intercostal space. Thoracic ultrasound aids the site selection. If the procedure has caused some trauma, the first fluid obtained may be blood tinged, but this clears as more fluid is withdrawn. If the pleural fluid is blood tinged because of the underlying disease process, the red coloration persists throughout the entire procedure. An aliquot of pleural fluid is transferred from the syringe into tubes containing anticoagulant solution (EDTA) so that appropriate laboratory evaluation may be performed. Part of the fluid should be saved in sterile containers with transport media for subsequent Gram stain and culture. Fluid should be removed as long as it flows freely. Both sides of the thorax should be tapped.

Examination of Pleural Fluid - The color, turbidity, viscosity and odor should be noted (Fig. 7). Normal pleural fluid is clear and yellow; cloudiness reflects an increased number of white blood cells (WBC). Putrid-smelling pleural fluid is a hallmark of anaerobic infection; however, the absence of odor does not exclude anaerobic infection. In addition to the odor of the pleural fluid, the odor of the horse's breath should be noted, particularly after coughing. The majority of horses with anaerobic infections have a putrid odor associated with the pleural fluid or breath. These horses have a low survival rate.

The WBC count of normal pleural fluid is generally less than 10,000/ul. The WBC count of pleural fluid in pleuropneumonia can range from 1600 to 300,000 cells/ul, varying in the same pleural fluid sample between the beginning and the end of the thoracocentesis. There is no association between the WBC count in pleural fluid and survival. Pleural fluid protein is greater than 3 g/dl in horses with pleuropneumonia, but this is also not a prognostic indicator. Pleural fluid should be Gram stained and cultured for bacteria. The Gram stain may provide tentative identification until culture results are obtained. Both aerobic and anaerobic cultures should be performed. Anaerobes occur in 46% of
horses with pleuropneumonia. The pleural fluid used for anaerobic cultures should be transferred to the laboratory immediately after collection in a manner that prevents or minimizes exposure to air. Anaerobic transport media is commercially available and should be routinely used. Specimens submitted for isolation of anaerobes should not be refrigerated, since many anaerobes are intolerant to cold. Isolation of anaerobic bacteria from either the pleural fluid or tracheobronchial aspirate provides a poor prognosis.

While pleuropneumonia is the most common cause of pleural effusion in the horse, the second most common cause is neoplasia. Differentiating between the two conditions is a challenge for the equine clinician as there are similarities in the clinical signs and physical examination findings.

Pleuropneumonia effusions are more likely to have abnormal nucleated cell count greater than 10,000/ul (usually greater than 20,000) with greater than 70% neutrophils. Bacteria are frequently seen both intra and extracellularly. A putrid odor may be present. Neoplastic effusions have variable nucleated cell count. If caused by lymphosarcoma, there may be a predominance of abnormal lymphocytes. However, neoplastic cells are often not readily apparent and a definitive diagnosis may be difficult. Rarely do neoplastic effusions have a putrid odor nor have bacteria seen cytologically.

Pleural Drainage - Following selecting of an appropriate anti-microbial agent, the next decision to be made is whether to drain the pleural space [15]. Ideally the decision is based on an examination of the pleural fluid. If the pleural fluid is thick pus, drainage using a chest tube should be initiated. If the pleural fluid is not thick pus, but the Gram stain is positive and WBC counts are elevated, pleural drainage is recommended. Another indication for therapeutic thoracocentesis is the relief of respiratory distress secondary to a pleural effusion.

There are many options for thoracic drainage including the following: intermittent chest drainage, indwelling chest tube (Fig. 8), pleural lavage, pleuroscopy and debridement, open chest drainage/debridement - no rib resection (standing), open chest drainage/debridement - rib resection (standing), open chest drainage and debridement (general anesthesia), and lung resection (general anesthesia).

Figure 8. Chest tube drainage of pleural fluid in horse with severe pleuropneumonia. - To view this image in full size go to the IVIS website at www.ivis.org . -

Drainage of a pleural effusion can be accomplished by (1) using a cannula, (2) indwelling chest tubes or (3) thoracostomy. Thoracostomy [16] is reserved for severe abscessation of the pleural space. Thoracocentesis is easily accomplished in the field and may not need to be repeated unless considerable pleural effusion re-accumulates. Indwelling chest tubes are indicated when continued pleural fluid accumulation makes intermittent thoracocentesis impractical. If properly placed and managed, they provide a method for frequent fluid removal and do not exacerbate the underlying pleuropneumonia or increase the production of pleural effusion. The chest entry site and end of the drainage tube must be maintained aseptically. A one-way flutter valve may be attached to allow for continuous drainage without leakage of air into the thorax. If a chest tube is placed aseptically and managed correctly, it can be maintained for several weeks. It should be removed as soon as it is no longer functional. Heparinization of tubing after drainage helps maintain patency. Local cellulitis may occur at the site of entry into the chest, but is considered a minor complication. Bilateral pleural fluid accumulation requires bilateral drainage in most horses. Open drainage or thoracostomy may be considered when tube drainage is inadequate. It is important not to begin open drainage too early in the disease. An incision is made in the intercostal space exposing the pleural cavity and causing a pneumothorax, unless the visceral and parietal pleura adjacent to the drainage site have not been fused by the inflammatory process. The wound is kept open for several weeks while the pleural space is flushed and treated as an open draining abscess.

Pleural Lavage - Pleural lavage may be helpful to dilute fluid and remove fibrin, debris and necrotic tissue. Lavage appears to be most effective in sub acute stages before loculae develop; however, pleural lavage may help break down fibrous adhesions and establish communication between loculæ. Care must be exercised that infused fluid is communicating with the drainage tube. Lavage can be performed by infusing fluid through a dorsally positioned tube and draining it through a ventrally positioned tube. Ten liters of sterile, warm lactated Ringer’s solution is infused into each affected hemithorax by gravity flow. After infusion, the ventrally placed chest tube is opened and the lavage fluid is allowed to drain. Pleural lavage is probably contraindicated in horses with bronchopleural communications because it
may result in spread of septic debris up the airways. Coughing and drainage of lavage fluid from the nares during infusion suggests the presence of a bronchopleural communication.

Tracheobronchial aspirate provides an excellent specimen for Gram stain and bacterial culture.

Thoracic radiography is often limited by the availability of facilities. Lateral thoracic radiographs can often show small amounts of pleural effusion not detectable by either auscultation or percussion.

Pleuroscopy, a procedure in which a flexible or rigid endoscope is introduced into the pleural space, is rarely indicated in bacterial pleuropneumonia. This technique is better reserved for equine patients with pleural effusion of undiagnosed etiology.

Hematologic findings in horses with pleuritis are usually nonspecific and do not predict the outcome of the case. A low hematocrit (less than 30%) usually reflects an anemia of chronic disease, whereas elevated total plasma proteins (greater than 8 g/dl) are probably caused by hypergammaglobulinemia. Both these findings suggest that the pleuropneumonia is chronic. White blood cell counts can be misleading, since not all affected horses have leukocytosis. Plasma fibrinogen appears to be a more sensitive indicator of inflammation because it is elevated in almost all cases of pleuropneumonia.

Management
The primary goals of managing a horse with pleuropneumonia are to stop the underlying bacterial infection, remove excess inflammatory exudate from the pleural cavity, and provide supportive care. Ideally an etiologic agent is identified from either the tracheobronchial aspirate or pleural fluid, and antimicrobial sensitivity determined. Without bacterial culture results, broad-spectrum antibiotics should be used because many horses have mixed infections of both gram-positive and gram-negative aerobic and anaerobic organisms. Commonly used therapy is penicillin combined with an aminoglycoside such as gentamicin, enrofloxacin, trimethoprim and sulfamethoxazole, or chloramphenicol. Because of the need for long-term therapy, initial intravenous or intramuscular antimicrobials may need to be followed by oral antimicrobials. Preferably the oral antimicrobials are not administered until the horse's condition is stable and improving because blood levels obtained by this route are not as high as those achieved following intramuscular or intravenous administration.

Treatment of anaerobic pleuropneumonia is usually empiric, since antimicrobial susceptibility testing of anaerobes is difficult because of their fastidious nutritive and atmospheric requirements. Thus familiarity with antimicrobial susceptibility patterns is helpful in formulating the treatment regimen when an anaerobe is suspected. The majority of anaerobic isolates are sensitive to relatively low concentrations of aqueous penicillin (22,000 IU/kg bwt, IV four times per day). Bacteroides fragilis is the only frequently encountered anaerobe that is routinely resistant to penicillin, although other members of the Bacteroides family are known to produce β-lactamases and are potentially penicillin-resistant. Chloramphenicol (50 mg/kg, orally four times per day) is effective against most aerobes and anaerobes that cause equine pleuropneumonia. However, because of human health concerns, the availability of chloramphenicol may decrease. Metronidazole has in vitro activity against a variety of obligate anaerobes including B. fragilis. Pharmacokinetic studies indicate a dose of 15 mg/kg intravenously or orally, four times a day, is necessary to maintain adequate serum levels. Oral administration rapidly results in adequate serum levels and thus is an acceptable route of administration for horses with pleuropneumonia. Metronidazole is not effective against aerobes and therefore should always be used in combination therapy with the above mentioned drugs for aerobes, at a dose of 15 mg/kg three or four times per day. It is important to recognize the side effects of metronidazole, including loss of appetite and lethargy, and to stop the use of the drug when these signs are observed. Aminoglycosides and enrofloxacin should not be considered for the treatment of pleuropneumonia caused by an anaerobe unless it is used in combination therapy (i.e., with penicillin).

Other Therapies
Anti-inflammatory agents help reduce pain and may decrease the production of pleural fluid. This in turn may encourage the horse to eat and maintain body weight. Flunixin meglumine (500 mg once or twice daily) or phenylbutazone (1 to 2 g twice a day) is commonly used for this purpose. The author believes that corticosteroids are contraindicated in bacterial pleuropneumonia. Rest and the provision of an adequate diet are important components of the treatment of pleuropneumonia. Because the disease course and period of treatment are usually prolonged, attempts should be made to encourage eating. Intravenous fluids may be indicated in the acute stages of the disease to treat dehydration from anorexia and third-space losses into the thorax.
References


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