Inflammatory conditions of the pleura may be dry, serofibrinous, pyogranulomatous, or purulent. *Dry pleuritis* often precedes inflammatory pleural effusions. Dry pleuritis may be caused by bacteria, viruses, or trauma. A diagnosis of dry pleuritis is suggested by clinical findings of a rapid and shallow respiratory pattern, obscure thoracic pain, nonproductive cough, and auscultation of a pleural friction rub. *Serofibrinous pleuritis* is reported with canine hepatitis, canine leptospirosis, canine distemper, canine and feline upper respiratory viruses, and parasitic diseases such as *Aelurostrongylus* in cats and *Spirocerca lupi* in dogs. Bile and tuberculosis are unusual causes of severe serofibrinous pleuritis. *Pyogranulomatous pleuritis* is associated with feline infectious peritonitis. The effusion is secondary to virus-induced vasculitis affecting all serous membranes.

*Purulent pleuritis*, also referred to as *pyothorax* or *empyema*, is invariably the result of bacterial or fungal sepsis of the pleural space.

**ETIOLOGY**

Sources of bacterial contamination include penetrating thoracic wounds, extension of bacterial pneumonia, migrating foreign bodies, esophageal perforations, extension of cervical, lumbar or mediastinal infections, and hematogenous spread. Thoracic bite wounds are frequently implicated in feline pyothorax. Inhalation and migration of a grass awn often is suspected in field dogs with pyothorax. Anaerobic bacteria and *Nocardia asteroides* are isolated most often from dogs with pyothorax. *Nocardia* and *Actinomyces* are very commonly associated to a foreign body. Pleural infections are almost always polymicrobial in nature. There is a high incidence of obligate anaerobic bacteria as sole pathogens or in combination with aerobic-facultative and anaerobic bacteria. Obligate anaerobic bacteria (*Bacteroides, Fusobacterium*) and Gram positive filamentous organism such as *Nocardia* and *Actinomyces* are most commonly isolated from dogs with pyothorax. *Pasteurella multocida* and anaerobes are the most prevalent isolates in cats.

**PLEURAL SPACE**

The thoracic cavity is lined entirely by a serous membrane known as pleura. The pleura is divided into *visceral pleura* which covers the lungs and *parietal pleura* which covers the remaining thoracic cavity. The pleura is composed of a single layer of mesothelial cells supported by a delicate network of elastic connective tissue. The visceral and parietal pleura contain a rich capillary network that originates from the pulmonary and systemic circulations, respectively. In addition, the parietal pleura contains a rich lymphatic network responsible for lymphatic drainage of the pleural space. Under normal conditions, the pleural space is only a potential cavity. The visceral and parietal pleura are separated by a thin layer of pleural fluid, the average volume of which is 2.4 ml in a 10 kg dog. Liquid coupling between the thoracic wall and lungs provides instantaneous transmission of thoracic volume changes to the lungs, and yet allows low friction sliding between the pleural surfaces.

Because high pleural permeability causes the pleural space to be continuous with the interstitial fluid of the thoracic wall, the dynamics of pleural fluid formation and absorption are controlled by Starling's forces. Since hydrostatic pressure in the systemic capillaries that supply the parietal pleura is a 30 cm of water and hydrostatic pressure of the pulmonary capillaries that supply the visceral pleura is approximately 11 cm of water, one theory suggests pleural fluid is
formed by the parietal pleura and absorbed by the visceral pleura under physiologic conditions. More recent evidence suggests that pleural fluid filters through the parietal pleura and is drained by parietal lymphatics.

CLINICAL FINDINGS

Pleuritis and pyothorax frequently have an insidious course and presentation may be delayed. Pyothorax occurs most commonly in young adult, male, non-purebred cats and adult large breed dogs. Clinical signs result from restrictive disease, including increased respiratory rate, shallow respiration, dyspnea and orthopnea. Other clinical signs include exercise intolerance, lethargy, anorexia, and fever. Physical examination reveals muffled heart sounds, decreased lung sounds, and hypersonant (dull) percussion sounds, especially over the ventral portions of the thorax. Chronic or severe infection result in a patient in septic shock with dehydration debilitation or hypothermia.

DIAGNOSIS

A diagnosis of pyothorax is confirmed by cytologic evaluation and culture of the pleural fluid. Neutrophilic leucocytosis with or without a left shift is the most common hematologic finding. Leukogram results do not correlate with the severity of the underlying infection. Radiographic signs of free pleural fluid include hazy density of the lung fields which obscures the cardiac silhouette, retraction of the lung lobes from the chest wall, visibility of the interlobular fissures and rounding of the costophrenic angles.

Cytologic evaluation of the pleural is consistent with a septic or a nonseptic exudate. Degenerate neutrophils and mixed population of bacteria are usually seen. If degenerative neutrophils are observed an anaerobic-anaerobic culture of the pleural fluid should be performed. Inflammatory exudates typically exhibit a total protein greater than 3.0 gm/dl, a specific gravity greater than 1.018, and a total cell count greater than 30 X 10^3 cells/ul. Inflammatory exudates may be nonseptic or septic. Nonseptic exudates usually have a serofibrinous or serosanguineous appearance. Feline infectious peritonitis produces a nonseptic exudative pleural effusion that appears yellow, translucent, and viscous on gross examination. Total protein values will approach serum levels ranging from 4 to 8 gm/dl. Electrophoresis will reveal an elevated gamma globulin fraction. The predominant cell types present in nonseptic exudates are non-degenerative neutrophils and macrophages. Total cell counts are generally not high, ranging from 5 to 15 X 10^3 cells/ul. Septic exudates are characteristic of pyothorax. The fluid is viscous, opaque, and varies in color from white or yellow to green or red. The fluid may clot or exhibit fibrinous debris, and often produces a foul odor. Cell counts range from 30 to 200 X10^3 cells/ul, although accurate cell counts are difficult due to extensive cellular degeneration. Degenerate neutrophils predominate and bacteria are often visualized. Gram stains may give an early indication of the types of bacteria present. Fluid should be cultured for aerobic and anaerobic bacteria. Macrophages and plasma cells increase as an exudative process becomes longstanding.

TREATMENT

Treatment of pyothorax must be prompt and aggressive. The prognosis is guarded but not hopeless. Supportive care with intravenous fluids is necessary to correct dehydration, acid-base and electrolyte imbalance. The initial goals of therapy are to relieve respiratory embarrassment by thoracocentesis, preferably under minimal restraint with the patient sternal or standing and to administer antibiotics.

Systemic antibiotic therapy should be initiated immediately, and then adjusted based on culture and sensitivity results if necessary. A broad spectrum antibiotic regimen should be instituted. We recommend a combination of Ampicillin sorium (20 mg/kg IV three times a day) Enrofloxacin (5 mg/kg two times a day IV). Clindamycin (11 mg/kg two times a day) has a good activity against anaerobic bacteria including Bacillus Fragilis. Clindamycin needs to be associated with Enrofloxacin to treat Gram negative organisms. Success in culturing anaerobic bacteria is dependent on sample handling. Samples must be submitted in a capped syringe within one hour or in appropriate transport media within 24 hours. Due to the high incidence of anaerobic infections, antibiotics with an anaerobic spectrum should be started upon diagnosis of pyothorax and continued throughout the course of disease. Many animals with pyothorax will have bacteremia or septicemia so intravenous antibiotics are indicated in the initial treatment period. Oral antibiotic therapy should be continued for at least four to six weeks after diagnosis.

Once the patient is stable, a thoracostomy tube should be placed utilizing local anesthesia and sedation. The biggest tube should be used to try to prevent occlusion with fibrin plugs. The thoracic cavity should be evacuated either by intermittent hand aspiration (every 3 to 4 hours) or by continuous suction. Continuous suction does not necessarily decreased the time required to manage pyothorax. The system should be checked regularly for leakage, kinks and clogging. Continuous suction offers the advantage of maximal drainage, whereas intermittent suction is more simple logistically. It is important to monitor drainage of the thoracic cavity by frequent
auscultation and percussion of the thoracic cavity. Thoracic radiographs will also evaluate
drainage and placement of the thoracostomy drain. Bilateral thoracostomy tubes often will be
necessary if both hemithorax are not draining adequately.

After complete evacuation of the pleural space, pleural lavage is initiated. The pleural space
should be lavaged twice daily with approximately 20 ml/kg of warmed 0.9% saline or Ringer’s
solution. The lavage fluid should be instilled slowly and discontinued if respiratory distress
occurs. The lavage fluid should remain in the pleural space for one hour if respiratory distress
does not occur. Approximately 25% of the initial lavage volume will be absorbed by the patient.
Efficacy of treatment is monitored by clinical findings, thoracic radiographs, and cytology of the
pleural effusion. Most animals with successful treatment will have a decrease in fever and
improvement in general attitude within the first 48 hours. Cytology of pleural fluid can be used to
assess the response to therapy. Neutrophils, both total number and percentage of degenerate
cells, and bacteria should gradually subside over three to five days. The combination of
thoracostomy tube, pleural lavage, and antibiotic therapy have been reported to resolve 50 to
60% of cases of pyothorax in small animals.

Lack of significant clinical improvement within 48 to 72 hours or radiographic demonstration
of undrained encapsulated fluid are indications to surgically explore the thoracic cavity.
Radiographic evidence of lung lobe consolidation and pneumothorax suggest the possibility of a
ruptured pulmonary abscess and is a relative indication for surgery. Exploratory thoracotomy
should be undertaken by medium sternotomy which gives access to both hemithoraces.
Adhesions and loculated pockets of fluid should be broken down carefully during surgery.
Mediastinectomy often is necessary since the ventral mediastinum is invariably thickened and
filled with small abscesses. The pericardium also may require excision if it is thickened and
abscessed. Consolidated lung lobes which cannot be inflated should be excised by partial or
complete lobectomy. Large lung lacerations created by adhesion breakdown must be repaired or
the damaged tissue excised. Before closure, the thoracic cavity is vigorously lavaged with copious
amounts of warm isotonic crystalloid solution. Closed pleural lavage should be continued
postoperatively at least two to three days. The probability of success with surgical
management of refractory pyothorax is better for dogs than for cats.

Constrictive pleuritis is a serious sequela to longstanding pyothorax that is suggested by an
inability to re-expand the lungs following resolution of the pyothorax. If the constriction is diffuse,
then surgical decortication of the fibrous peel from the visceral pleura is necessary. Decortication
in small animals is a difficult surgical procedure, but it can be successfully accomplished with
careful technique. The procedure often results in numerous pulmonary lacerations that should be
treated with continuous pleural drainage postoperatively. Decortication should be attempted as
soon as possible after it is recognized.