Acute Respiratory Distress in Cattle

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Acute respiratory distress is a common problem to the bovine practitioner. Acute respiratory distress can be categorized into upper respiratory disease or lower respiratory disease. Since dyspnea is the primary clinical sign in cases of acute respiratory distress, any condition that can cause dyspnea can cause clinical signs suggestive of respiratory disease. There are also nonrespiratory causes of apparent acute respiratory disease in cattle.

Inadequate perfusion of the lungs, inadequate supply of oxygen to the tissues and the reduction in effective circulating blood volume can cause dyspnea and lead the clinician to suspect an acute respiratory disease. Cardiovascular and diaphramatic disease that can lead to inadequate perfusion of the lung would include pericarditis, selenium deficency and high mountain disease. Hemogloblin or red blood cell disorders can lead to an inadequate supply of oxygen to the tissues. Anaplasmosis and the resulting anemia is an example of a red blood cell disorder causing dyspnea and signs that could lead one to think that a respiratory disease was present. The altered hemogloblin found with nitrate toxicity is also an example of a nonrespiratory cause of apparent acute respiratory disease. Animals in shock and animals with endotoxemia will appear to have respiratory problems because of the reduction in effective circulating blood volume. Acidiotic animals are also an example of a nonrespiratory cause of apparent acute respiratory disease, in that CO_2 stimulates the respiratory center and dyspnea will occur.

Upper respiratory disease is caused by conditions that compromise the nasal pharynx by swelling or proliferation of the peripheral tissues. Upper respiratory disease is characterized clinically by inspiratory dyspnea. Loud noises such as snoring, strider, honking, coughing are present with upper respiratory disease. There are auscultable inspiratory wheezes. Animals with acute upper respiratory disease are in obvious distress and are mouth breathing. Manipulation of their head or forced exercise can often lead to fatal asphyxiation. Nasal discharges seen in these animals can vary from a small amount of serosanguineous to large quantities of purulent exudate. Signs are usually consistently present, however, they may be intermittent in conditions such as a pedunculated growth in the pharynx. Animals with upper respiratory disease can be hypoxic and show anxiety and aggression. Nasal obstructions are not common in cattle, however, granulomas sometimes are found in the upper respiratory tract of cattle. Granulomas may be caused by *Fusiformis necrophorus, Actinobacillus* and *Rhinosporidium.* Acute rhinitis, either allergic or infectious, can also cause upper respiratory disease in bovine.

Pharyngeal problems are more common than nasal problems in the bovine animal. Acute pharyngitis will cause a significant inspiratory dyspnea. Cellulitis resulting from injuries caused by balling guns and other foreign objects lead to edema, phlegmon, and eventual abscessation.

Allergies, inhalation of irritant substances, and infectious agents such as IBR, MCF and diphtheria can compromise the laryngeal region.

Acute lower respiratory diseases are mostly infectious and mild to rapidly fatal. Most of these conditions are characterized by expiratory dyspnea with grunting, and frothing at the mouth. Loud auscultable crackles are heard. There may or may not be subcutaneous emphysema present. On post mortem, edema and emphysema are seen grossly. Conditions that cause acute lower respiratory disease would include anaphylaxis, lung worms, atypical interstitial penumonia and Bovine Respiratory Syncitial Virus. Although a sensitizing substance cannot always be isolated, anaphylaxis in the bovine animal is generally thought to be due to sensitization to a protein substance followed by a second exposure to that substance. However, anaphylaxis can occur with no known prior exposure. Iatrogenic anaphylaxis is caused through the use of biologicals and antibiotics. Bacterins, vaccines, and blood transfusions are examples of iatrogenic causes of anaphylaxis. Two more common examples would be *Brucella abortus* strain 19 and salmonella bacterins. Penicillin, of course, is a classical example of an antibiotic causing anaphylaxis in some cows. Milk allergies can also cause anaphylaxis with signs attributable to the lower respiratory tract present. Milk allergies in cattle occur most commonly at the time of drying off or bagging for show. When grubs are killed in the subcutaneous tissues of cattle, anaphylaxis may result from breakdown products of the larva. Pasture and feedlot cattle sometimes exhibit anaphylaxis attributable feed antigens and the ingested offending protein is seldom identified.

The signs of anaphylaxis usually occur within minutes of exposure to the antigen. Most signs will occur within 10-20 minutes. The animals will either die in minutes or recover in hours. Most aniamls that are going to recover will do so in two hours. Animals affected with anaphylaxis are very responsive to epinephrine administered intramuscu-

larly. The response is often immediate. Corticosteroids will potentiate the epinephrine effects. Often steroids are administered just after the epinephrine. Antihistamines give variable results when treating anaphylaxis. Most likely the variable results are due to presence of mediators other than histamines. Catecholamines, kinins, and prostaglandins are mediators that are often present in the bovine animal in cases of anaphylaxis. Atropine is of little value when treating anaphylaxis in cattle. The major target organs of anaphylaxis are the respiratory tract, the alimentary tract, and the skin. Systemic hypotension with marked pulmonary venous constriction and pulmonary artery hypotension are present in cattle anaphylaxis. There is an increase in the mesenteric venous pressure and mesenteric vascular resistance causes considerable pooling of blood on the venous side of mesentery vessels. Changes in vascular tone coupled with increase capillary permeability, cause an increased secretion by the mucus glands and bronchospasms. Death is due to anoxia. Necropsy lesions of cattle with anaphylaxis are confined to the lungs. There is a severe pulmonary edema and vascular engorgement. Emphysema is present. Generally the lesions involve the entire lung whereas in cases of acute pneumonia, the changes are more marked in the ventral lung field. Histopathology of the lung should reveal a lack of syncitial cells, hyaline membranes and epithelial hyperplasia.

Acute lower respiratory disease can also be seen with *Dictyocaulus viviparus,* the cattle lung worm. This condition is seen when there is massive reinfestation and also post treatment. Larvae migrating through alveoli and bronchioles initate an inflammatory response that may result in blockage with exudate and result in collapse of alveoli and the development of edema. Cattle can become sensitized to lung worm tissue and develop a hypersensitivity reaction, especially with massive reinfestation. These cattle can exhibit signs resembling acute atypical pneumonia.

Lung worm reactions can easily be confused with bacterial pneumonia, atypical pneumonia, and viral pneumonias. Tracheal wash is one procedure that can be done to help diagnose lung worm reactions. The tracheal lavage should result in a fluid high in eosinophils and possibly larvae can be identified in the tracheal lavage fluid. Since the prepatent period is three weeks, fecal examinations could be negative in animals with lung worm reactions. Hispathologic examination of the lungs of cattle with lung worm reactions should reveal the presence of pulmonary edema, eosinophils, hyaline membranes along with alveolar epithelialization. Lung worms, larva or eggs can also be identified.

Lung worms in cattle are most frequently treated with ivermectin, fenbendazole, albendazole or lavamisole. If the lung worm infestation has been massive and caused alot of extensive damage, treatment may not result in clinical improvement. However, if these animals are treated early, clinical improvement can be dramatic. In cattle sensitized to lung worm tissue, treatment of massive infestations may make the clinical signs worse and even progress to fatal termination. Consequently, anti-inflammatory drugs can be included in the therapy.

Atypical interstitial pneumonia is a pneumonintoxication usually seen in adult cattle in the fall when they are moved from dryer, poorer pastures to more lush pastures. Recognized causes of atypical interstitial pneumonia include L-tryptophan present in some plants being changed to 3-methylindole in the rumen. The 3-methylindole is absorbed in the blood stream from the rumen and metabolized by a mixed function oxidase system in the lung to produce a pneumotoxicity. Bronchiolar and alveolar epithelial cells along with endothelial cells are damaged resulting in edema, hyaline membranes, alveolar epithelial cell hyperplasia and interstitial emphysema.

A typical animal with atypical interstitial pneumonia is an older animal, over 2 years of age, that has undergone change of feed or pasture in the last 3-7 days. The animal is frothing at the mouth, has a normal to slight increase in temperature. No coughing is present. These animals stand still and are reluctant to move. There is a low to moderate morbidity perhaps up to 50%, with a high mortality. Sudden death is often found in cases of atypical interstitial pneumonia.

Treatment is generally unrewarding. The important consideration in treatment is to subject the animals to minimal stress. Often these animals will die when being treated. Drugs that have been promoted in the treatment of an atypical interstitial pneumonia include corticosteroids, atrophine, epinephrine, dieurtics, and acetylsalicylic acid.

Animals with atypical interstitial pneumonia should be removed from the pasture as quietly as possible.

Management should avoid sudden introductions of hungry cattle to better pastures. This can be difficult to accomplish, however, there are some things to try. Pregrazing the pasture with less susceptible animals or introducing the animals to the pasture before lush growth appears may be effective. Continuous strip grazing may also help. Some people feed hay or concentrate during the period animals are adapting to the lush pasture.

The addition of ionophores to the diet at 200 mg per head per day will alter ruminal metabolism inhibiting or lowering the production of 3-methyindole may be the most effective practice to prevent clinical signs. The ionophores should be fed for 7-10 days after introduction to the pasture. Since ionophores only lower the production of 3-methylindole they are of no benefit in treating animals after signs are present.

Bovine respiratory syncytial virus is associated with varying degrees of bovine respiratory disease in cattle of all ages, including both dairy and beef.

Clinical signs range from subclinical to very severe including sudden death. Affected animals have elevated

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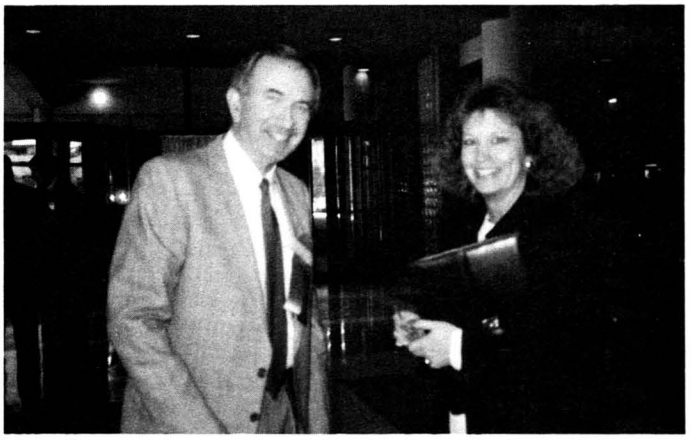
temperatures, hyperpnea, and an increased harshness of lung sounds. Coughing can be spontaneous or easily induced. Nasal discharge is often present in varying degrees. Hypersalivation is also characteristic. Subcutanous emphysema can be seen, usually later in the course of the disease.

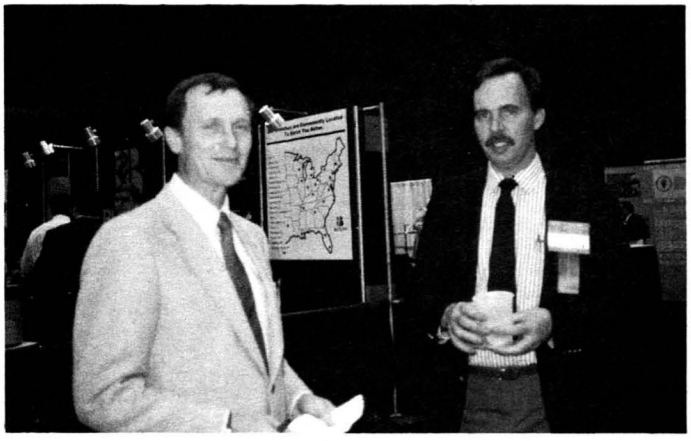
There is evidence that a often fatal severe respiratory disease resembling a typical interstitial pneumonia often follows a mild BRSV infection.

BRSV is difficult to isolate. Isolation attempts are most successful from samples taken in the initial stages of the disease. Demonstration of a rise in serum antibodies is often used as a diagnostic tool. Immunofluorescent technique on material taken from nasal swabs or lung lavage can also be beneficial in diagnosing BRSV. Often syncytial cells can be identified in samples taken via lung lavage.

Treatment and prevention of BRSV is controversial. Antihistamines, corticosteroids, antibiotics and supportive therapy are all promoted in the therapy. Removing feed to reduce exposure to pneumotoxins and reduce hypersensitivity reactions has also been encouraged.

Vaccination may be of value, perhaps more in reducing the severity of the disease rather than eliminating it.





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