

ENZOOTIC PNEUMONIA OF CALVES AND SHIPPING FEVER PNEUMONIA

- Enzootic pneumonia of calves refers to infectious respiratory disease in calves. The term “viral pneumonia of calves” is sometimes used but is not preferred based on the current understanding of etiology and pathogenesis.
- Enzootic pneumonia is primarily a problem in calves <6 mo old with peak occurrence from 2–10 wk, but it may be seen in calves up to 1 yr of age. It is more common in dairy than in beef calves and is a common problem in veal calves.
- It is also more common in housed dairy calves than in those raised outside in hutches. Peak incidence of disease may coincide with decline of passively acquired immunity. Morbidity rates may approach 100%; case fatality rates vary but can reach 20%.

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- The etiology is similar to that for BRD complex in general. The pathogenesis involves environmental and management stressors and possibly an initial respiratory viral infection followed by a secondary bacterial infection of the lower respiratory tract.
 - Stress results from environmental and management factors, including inadequate ventilation, mixing by adding calves to an established group, crowding, and nutritional factors such as poor-quality milk replacers.
 - Partial or complete failure of passive transfer of maternal antibodies is an important host factor related to development of disease.

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- Any of several viruses may be involved, and a variety of bacteria may be isolated from affected calves.
 - Mycoplasmal and bacterial agents, including *Pasteurella multocida*, *Mannheimia haemolytica*, and *Mycoplasma bovis*, represent the most frequently isolated pathogenic organisms.
 - The individual viral and bacterial etiologies, clinical signs, lesions, and treatment are discussed under Viral Respiratory Tract Infections in Cattle and Bacterial Pneumonia in Cattle.


CONTROL AND PREVENTION:

- When calves of varying ages are placed in communal pens, control of enzootic pneumonia is difficult. The severity of the pneumonia may be decreased by improved husbandry, proper housing, adequate ventilation, and good nursing care. Prevention begins with vaccinating the cows against specific respiratory viruses and bacteria 3–4 wk prepartum to improve the quality of colostrum antibodies.
- Calves should receive good-quality colostrum at 8%–10% of body wt in the first 6 hr after birth. Newborn dairy calves should be housed individually in hutches or stalls and fed whole milk or a high-quality milk replacer with a fiber content of <0.25% until 8–12 wk old.

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- The use of calf hutches in dairy herds is the preferred standard for calf housing and has been shown to significantly improve calf respiratory health. However, delivering milk to a large number of hutches in cold weather presents a significant challenge. Single calf housing in naturally ventilated calf barns is the next best alternative.
 - Calves should be vaccinated against respiratory viruses 3–4 wk before the first grouping, although in some situations, the presence of passive immunity may interfere with an active immune response. Calves should be of similar age when assembled into groups, and a group should be limited to ≤ 10 .

SHIPPING FEVER PNEUMONIA

- Shipping fever pneumonia, or undifferentiated fever, is a respiratory disease of cattle of multifactorial etiology with *Mannheimia haemolytica* and, less commonly, *Pasteurella multocida* or *Histophilus somni* (see [Histophilosis](#)) being the important bacterial agents involved

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- Shipping fever pneumonia is associated with the assembly into feedlots of large groups of calves from diverse geographic, nutritional, and genetic backgrounds.
 - Morbidity in feeder calves often peaks within 7–10 days after assembly in a feedlot. Morbidity can approach 35%–50%, and case fatality is 5%–10%; however, the level of morbidity and mortality strongly depends on the array of risk factors present in the cattle being fed.


ETIOLOGY:

- The pathogenesis of shipping fever pneumonia involves stress factors, with or without viral infection, interacting to suppress host defense mechanisms, which allows the proliferation of commensal bacteria in the upper respiratory tract
- Subsequently, these bacteria colonize the lower respiratory tract and cause a bronchopneumonia with a cranioventral distribution in the lung. Multiple stress factors are believed to contribute to suppression of host defense mechanisms.


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- Weaning is a significant stressor, and the incidence of this disease is highest in recently weaned calves. Transportation over long distances serves as a stressor; it may be associated with exhaustion, starvation, dehydration, chilling and overheating depending on weather conditions, and exposure to vehicle exhaust fumes
 - Additional stressors include passage through auction markets; commingling, processing, and surgical procedures on arrival at the feedlot; dusty environmental conditions; and nutritional stress associated with a change to high-energy rations in the feedlot.

CONTROL AND PREVENTION:

- Prevention of shipping fever pneumonia should focus on reducing the stressors that contribute to development of the disease.
- Cattle should be assembled rapidly into groups, and new animals should not be introduced to established groups.
- Mixing of cattle from different sources should be avoided if possible; however, in the North American beef industry, this risk factor is almost unavoidable for large intensive feedlots.

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- Transport time should be minimized, and rest periods, with access to feed and water, should be provided during prolonged transport.
 - Calves should ideally be weaned 2–3 wk before shipment, and surgical procedures should be performed in advance of transport; however, the availability of these “preconditioned” calves is quite limited.
 - Cattle should be processed within 48 hr after arrival at the feedlot. Adaptation to high-energy rations should be gradual, because acidosis, indigestion, and anorexia may inhibit the immune response. Vitamin and mineral deficiencies should be corrected. Dust control measures should be used.

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- Metaphylaxis with long-acting antibiotics such as oxytetracycline, tilmicosin, florfenicol, gamithromycin, tildipirosin, or tulathromycin has been widely adopted as a control measure given “on arrival” to cattle at high risk of developing shipping fever pneumonia.
 - Metaphylaxis on arrival has been shown to significantly reduce morbidity and improve rate of gain and, in some cases, reduce mortality.
 - Mass medication in feed or water is of limited value because sick animals do not eat or drink enough to achieve inhibitory blood levels of the antibiotic, and many of these oral antibiotics are poorly absorbed in ruminants.

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- On arrival, processing usually involves administration of modified-live vaccines for viral antigens and for bacterial components of shipping fever pneumonia.
 - Because most cases of pneumonia occur during the first 2 wk after arrival, these on-arrival vaccines may not have adequate time to stimulate immunity.
 - When possible, vaccinations for the viral and bacterial components of shipping fever pneumonia should be given 2–3 wk before transport or earlier and can be repeated on entry to the feedlot.


MANNHEIMIA HAEMOLYTICA-ASSOCIATED BOVINE RESPIRATORY DISEASE



ETIOLOGY:


- *Mannheimia haemolytica* serotype 1 is the bacterium most frequently isolated from the lungs of cattle with BRD.
- Although less frequently cultured, *Pasteurella multocida* is also an important cause of bacterial pneumonia. *Histophilus somni* is being increasingly recognized as an important pathogen in BRD; these bacteria are normal inhabitants of the nasopharynx of cattle (see [Histophilosis](#)). When pulmonary abscessation occurs, generally in association with chronic pneumonia, *Trueperella pyogenes* is frequently isolated.

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- Under normal conditions, *M haemolytica* remains confined to the upper respiratory tract, in particular the tonsillar crypts, and is difficult to culture from healthy cattle.
 - After stress or viral infection, the replication rate of *M haemolytica* in the upper respiratory tract increases rapidly, as does the likelihood of culturing the bacterium.
 - The increased bacterial growth rate in the upper respiratory tract, followed by inhalation and colonization of the lungs, may occur because of suppression of the host's defense mechanism related to environmental stressors or viral infections. It is during this log phase of growth of the organism in the lungs that virulence factors are elaborated by *M haemolytica*, such as an exotoxin that has been referred to as leukotoxin.

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- The interaction between the virulence factors of the bacteria and host defenses results in tissue damage with characteristic necrosis, thrombosis, and exudation, and in the development of pneumonia.
 - The pathogenesis of pneumonia caused by *P multocida* is poorly understood. This organism may opportunistically colonize lungs with chronically damaged respiratory defenses, such as occurs with enzootic calf pneumonia or existing lung lesions of feedlot cattle, and cause a purulent bronchopneumonia.
 - *H somni* may invade the lung and cause pneumonia after damage to the respiratory defenses. This organism is capable of systemic spread from the lung to the brain, myocardium, synovium, and pleural and pericardial surfaces; often, death can occur later in the feeding period (40–60 days after arrival) from involvement of these additional organ systems.

CLINICAL FINDINGS:

- Clinical signs of bacterial pneumonia are often preceded by signs of viral infection of the respiratory tract. With the onset of bacterial pneumonia, clinical signs increase in severity and are characterized by depression and toxemia.
- A combination of clinical signs of depression and fever (104°–106°F [40°–41°C]), without any signs attributable to other body systems, are the classic components of a case definition for early cases of BRD.

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- Serous to mucopurulent nasal discharge; moist cough; and a rapid, shallow respiratory rate may be noted. Auscultation of the cranioventral lung field reveals increased bronchial sounds, crackles, and wheezes.
 - In severe cases, pleurisy may develop, characterized by an irregular breathing pattern and grunting on expiration. The animal will become unthrifty in appearance if the pneumonia becomes chronic, which is usually associated with formation of pulmonary abscesses.


LESIONS:

- *M haemolytica* causes a severe, acute, hemorrhagic fibrinonecrotic pneumonia. The pneumonia has a bronchopneumonic pattern. Grossly, there are extensive reddish black to grayish brown cranioventral regions of consolidation with gelatinous thickening of interlobular septa and fibrinous pleuritis.
- There are extensive thromboses, foci of lung necrosis, and limited evidence of bronchitis and bronchiolitis.

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- *P multocida* is associated with a less fulminating fibrinous to fibrinopurulent bronchopneumonia. In contrast to *M haemolytica*, *P multocida* is associated with only small amounts of fibrin exudation, some thromboses, limited lung necrosis, and suppurative bronchitis and bronchiolitis.
 - *H somni* infection of the lungs results in purulent bronchopneumonia that may be followed by septicemia and infection of multiple organs. *H somni* is associated with extensive fibrinous pleuritis in feedlot calves.


DIAGNOSIS:

- Generally, neither serologic testing nor direct bacterial detection are performed, and diagnosis relies on gross necropsy findings and bacterial culture.
- Because the bacteria involved are normal inhabitants of the upper respiratory tract, the specificity of culture can be increased by collecting antemortem specimens from the lower respiratory tract by tracheal swab, transtracheal wash, or bronchoalveolar lavage.

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- Lung specimens can be collected for culture at necropsy. If possible, specimens for culture should be collected from animals that have not been treated with antibiotics to permit determination of antimicrobial sensitivity patterns. A multiplex PCR has been used to identify a number of bacterial agents implicated with bovine respiratory disease, including *M hemolytica*.


TREATMENT:


- Early recognition by trained personnel skilled at detecting the early clinical signs of disease followed by treatment with antibiotics is essential for successful therapy.
- Treatment protocols should be established so the producer has a standardized approach to identifying and treating cases.
- Long-acting antimicrobials such as tulathromycin, tilmicosin, florfenicol, and enrofloxacin have label claims to treat BRD and are commonly used as first- or second-line treatment options in feedlot calves.

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- These long-acting antimicrobials allow the feedlot producer to avoid commingling sick animals in a hospital pen, and treated animals can return directly to the home pen.
 - NSAIDs have been shown to be a beneficial ancillary therapy in controlling fever in cases of BRD, but data are lacking in terms of effect on relapse and mortality outcomes. If selection for treatment is late and pulmonary abscessation has occurred, it is difficult to achieve resolution with antimicrobials, and use of a convalescent pen or culling of the animal should be considered.

MYCOPLASMAL PNEUMONIA

- *Mycoplasma bovis* is an emerging cause of respiratory disease and arthritis in feedlot cattle and in young dairy and veal calves.
- Experimental infections usually result in inapparent to mild signs of respiratory disease, but virulent strains have been identified that cause severe lung disease in calves.
- However, this does not preclude a synergistic role for mycoplasmas in conjunction with viruses and bacteria in BRD. Mycoplasmas can be isolated from the respiratory tract of nonpneumonic calves, but the frequency of isolation is greater in those with respiratory tract disease

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- *M bovis* has been associated with otitis media in young calves and a syndrome involving chronic pneumonia and polyarthritis in feedlot cattle.
 - These cattle invariably have a pneumonic lesion, and 40%–60% may also develop a polyarthritis and tenosynovitis that causes severe chronic lameness.
 - The condition results in a chronic disease that does not respond to antimicrobial therapy. A significant proportion of these animals are euthanized because of the chronic nature of the disease.

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- Lesions include chronic bronchopneumonia with caseous and coagulative necrosis. In severe cases, >80% of the lung tissue may be involved.
 - Culture of these organisms requires special media and conditions; growth of the organisms may take up to a week. PCR tests are now available that can detect the mycoplasma within hours, thus greatly speeding up diagnosis.
 - Immunohistochemical tests can also be done on fixed tissue that link the mycoplasma antigen directly with the lung lesion. Vaccines are commercially available for *M bovis*, but their efficacy has not been demonstrated conclusively.

CHLAMYDIAL PNEUMONIA

- Chlamydial agents have been implicated in a number of diseases of cattle, including pneumonia. Clinical signs and lesions of bronchopneumonia have been produced by experimental infections. A synergism between *Chlamydia* and *Mannheimia haemolytica* has been demonstrated experimentally.
- Because this pathogen is infrequently tested for, its overall importance remains undetermined. The organism can be tested for by staining sections of lung lesions with Gimenez stain or by fluorescent antibody. Isolation requires inoculation of yolk sacs of embryonating chicks. Chlamydial agents are sensitive to tetracyclines