Diseases of the Lower Respiratory Tract and Thorax

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DISEASES OF THE LOWER RESPIRATORY TRACT

A. Pneumonia in young animals

1. Pneumonia in neonates. Clinical signs of pneumonia in all species generally include fever, tachypnea or dyspnea, anorexia, and, particularly with bacterial involvement, depression. Accompanying these signs are a productive cough and a bilateral mucoid to mucopurulent nasal discharge. On chest auscultation, loud bronchial tones over the trachea and lungs are audible, with adventitious sounds (wheezes, crackles) over the lung fields.

2. Pneumonia in foals. Respiratory disease of foals can have a variety of causes, both infectious and noninfectious. Of these causes, the more important infectious diseases are discussed in this chapter.

a. Overview

(1) Viral pneumonia

(a) Patient profile and etiology. The viral agents that result in signs of pneumonia in foals include the equine herpesviruses (EHV-1, EHV-4), influenza virus, and adenovirus. Pure viral pneumonia in foals is associated with immunocompromised, debilitated foals (e.g., failure of passive transfer, combined immunodeficiency, steroid therapy, poorly nourished foals). The main problem with these infections is the potential to predispose to more clinically serious secondary bacterial pneumonia.

(b) Clinical findings are similar to those described for neonatal pneumonia because they are seldom specific for any one viral agent. In foals, the severity of pneumonia is often poorly correlated to chest auscultation findings. For the pathogenesis, diagnostic plans, and therapeutic plans for these agents, refer to Chapter 6.

(2) Bacterial pneumonia

(a) Patient profile and etiology. A variety of bacteria can be associated with pneumonia in foals.

(i) Streptococcus zooepidemicus is the most common [see I A 2 b (1)].

(ii) Actinobacillus equuli can cause pneumonia in older foals, whereas this organism causes septicemia and nephritis in very young foals.

(iii) Klebsiella pneumoniae causes a severe pneumonia in septicemic foals, as do the Salmonella species in foals 2–3 weeks of age, in which other signs include diarrhea and arthritis.

(iv) Escherichia coli can be a cause of embolic pneumonia secondary to septicemia.

(v) Bordetella bronchiseptica has been a cause of bacterial pneumonia in foals, though it is usually not a primary pathogen.

(vi) Rhodococcus equi is a specific pathogen for foals, with lung abscessation as the primary lesion [see I A 2 b (2)].

(b) Risk factors

(i) In the early stages of life, foals with septicemia usually have an associated bacterial pneumonia. Clearly, the elements that predispose to septicemia (e.g., problems with sufficient passive transfer) are key factors.

(ii) Older foals are at risk to secondary bacterial pneumonia when they experience respiratory viral infection. In these cases, there is usually stress of transport and overcrowding, as may occur accompanying transport of the mare to be bred.
(iii) Inadequate ventilation (excessive dust levels, poorly cleaned stalls), poor nutrition, and parasite migration are thought to contribute to foals being predisposed to developing pneumonia, although these are not sufficient causes on their own.

b. Specific conditions

(1) Streptococcus zoopneumonia
   (a) Patient profile and history is variable because the organism is an opportunistic pathogen in horses of all ages and ubiquitous in their environment.
   (b) Clinical findings. Signs include tachypnea, anorexia, depression, fever, abnormal lung sounds, and possibly a cough. None of these signs are specific for S. zoopneumonia infection. However, this organism can cause lymph node abscessation, in contrast to the other opportunistic bacteria, and could resemble S. equi infection in the early stages before signs of pulmonary involvement.

(c) Etiology and pathogenesis
   (i) Etiology. S. zooepidemicus is a normal inhabitant of the equine upper respiratory tract and does not normally invade intact mucous membranes. Any of several stressors (e.g., poor air quality, inadequate ventilation, damage to respiratory epithelium by viral infections) to the normal respiratory defenses may predispose this organism to spread to the lungs.
   (ii) Pathogenesis. Acute infections result in severe fibrinopurulent bronchopneumonia with hemorrhage, whereas less fulminant infections cause abscesses in the lungs, lymph nodes, and occasionally in the pleura (pyogranulomatous pneumonia).

(d) Diagnostic plan and laboratory tests
   (i) A tracheal aspirate aids diagnosis and rapidly directs specific treatment. Typically, there are gram-positive cocci noted in the fluid recovered, which is evidence of Streptococcus infection. Culture and sensitivity tests are then performed to establish an etiologic diagnosis for appropriate treatment and advice on prophylaxis.
   (ii) Thoracic radiographs (used for foals only because these are not usually available for adult horses) can confirm the presence of pneumonia and may detect lung abscesses, which require far more prolonged treatment.
   (iii) Hematology usually reveals leukocytosis and increased fibrinogen, but these changes are not specific for this infection.

(e) Therapeutic plan
   (i) Penicillin is the drug of choice for S. zooepidemicus infections. If the clinical signs are marked (indicating fulminant infection), foals should be started on intravenous medications, such as sodium or potassium penicillin (20,000 IU/kg intravenously every 6 hours). For milder infections, affected foals (and adult horses) respond well to procaine penicillin (20,000 IU/kg intramuscularly every 12 hours).
   (ii) An alternative treatment is trimethoprim-sulfadiazine (30 mg/kg orally every 12 hours). Treatment should continue well beyond resolution of clinical signs. Treatment effectiveness should be monitored using blood work and serial chest radiographs.

(f) Prevention. Because there are 13 serotypes of S. zooepidemicus, there is no vaccine available. Therefore, preventive measures include attention to hygiene and reduction of stress, particularly in the neonatal period. For older foals and even adult horses, adequate rest and a stress-free environment following respiratory viral infection should be ensured for at least several weeks.

(2) Rhodococcus equi pneumonia
   (a) Patient profile and history. This is an infectious respiratory disease of foals usually 4 months or younger that is usually sporadic in occurrence on any one farm. R. equi is an opportunistic bacterium that appears to infect the foal when maternal antibody levels are waning and viral infections impair defense mechanisms. Affected foals can have a prolonged course of chronic respiratory signs or exhibit a sudden onset of severe respiratory disease that can be rapidly fatal.

(b) Epidemiology
   (i) Foals between the ages of 1 and 3 months are most commonly affected, but the onset of signs can range from age 2 weeks to 6 months. The disease is rare in adult horses and occurs only in immunocompromised animals.
   (ii) There appears to be an increased incidence during the hot, dry months of summer, which may be attributable to the increased spread of soil- and fecal-born organisms in dusty conditions. During these dry conditions, the risk of infection can be reduced if the foals are put onto grass pasture. Morbidity is low, but the case fatality rate is high if left untreated.

(c) Clinical findings. The signs of disease appear to vary with the age at which foals become affected.
   (i) Young foals affected after 1 month of age often have acute signs of illness with fever, respiratory distress, anorexia, and, in some cases, swollen joints.
   (ii) Older foals can develop serious lesions in the absence of marked clinical abnormalities, which then begin to manifest as a persistent cough and a progressive increase in respiratory effort at rest, with crackles and wheezes audible over the chest. These foals often succumb normally and have no fever during such disease progression, but they become emaciated. In some foals, diarrhea may follow or accompany the respiratory signs, but nasal discharge and lymph node enlargement in the throat region are absent.
   (iii) Other clinical signs can include arthritis in one or several joints and uveitis, both presumed to be immune mediated. In foals with these signs, the slow development of apparent pneumonia masks the severity of underlying pulmonary disease; therefore, it is prudent to suspect R. equi infection in a foal with nonresponsive pneumonia.

(d) Diagnostic plan and laboratory tests. Although clinical signs are usually suggestive of pneumonia, it is important to establish an etiologic diagnosis in the case of R. equi infection for appropriate treatment and advice on prognosis. Tracheal aspirate cytology and culture, along with thoracic radiology, are the most valuable diagnostic tests.
   (i) On tracheal wash, there are usually large numbers of neutrophils, and the bacteria (often intracellular) can have a characteristic “Chinese letter” appearance. Culture of the sample should be positive for R. equi, but in those foals that have been treated with antibiotics, there may be no bacterial growth.
   (ii) Radiography. Classically, “cotton-wool abscesses” of the lungs are present on chest radiographs, although the radiographic image can also consist of diffuse pulmonary infiltration and air bronchograms with hilar lymphadenopathy.
   (iii) Blood work. A complete blood cell count (CBC) and fibrinogen level, although not definitive for R. equi, are useful for monitoring the course of the inflammatory process. Affected foals usually show a neutrophilic leukocytosis with hyperfibrinogenemia, accompanied by anemia of chronic disease. The fibrinogen response can also be used as a guideline for response to treatment.
   (iv) Serologic tests have been of limited value because although foals with pulmonary infection caused by R. equi show seroconversion to this organism, normal foals in the first few months of life can also show seroconversion as a result of intestinal colonization by the organism.

(e) Etiology and pathogenesis
1. **Enzootic pneumonia in calves**

   a. Patient profile and history. This disease occurs almost exclusively in calves raised indoors. Therefore, enzootic pneumonia is found mainly in dairy herds. Although this disorder can occur as early as the first week of life, this disease is most common in calves between 2 and 5 months and up to 1 year of age. Fall and winter are the times it is most often observed. Poor air quality is a contributing factor to infection.

   b. Clinical finding

   (1) Affected calves in the acute stage have a moderate fever (40°C–40.5°C) and a harsh, hacking cough that is easily induced by pinching the trachea.

   (2) Tachypnea and dyspnea are often present, with increased bronchial tones audible over the cranial lung fields suggestive of lung consolidation. Crackles and wheezes at the periphery of areas of consolidation may also be audible, suggesting bronchiolitis.

   (3) Calves are usually alert unless there is a significant bacterial component to the pneumonia, in which case the fever may be higher.

   c. Etiology and pathogenesis. At approximately 2 months of age, calves’ immunity to respiratory infection (a combination of waning colostral immunity and the slow development of an independent response) is at its lowest point. This may be a key reason that most of calf pneumonia problems caused by infections begin to appear at this age.

   (1) **Infection** may begin as a viral respiratory infection, which may resolve or become complicated by a variety of bacteria, mycoplasmal organisms, or both (multifactorial).

   a. Viral agents include parainfluenza-3 (PI-3), bovine respiratory syncytial virus (BRSV), and bovine viral diarrhea (BVD), of which PI-3 and BRSV appear to be the most significant. BRSV is increasingly implicated as a major factor in the genesis of enzootic pneumonia. Unless there is secondary complication by bacteria, the disease is generally an interstitial pneumonia affecting the cranial lung lobes. Other viruses of cattle (e.g., rhinoviruses, adenoviruses, reoviruses) are not considered important.

   b. Mycoplasmal organisms, including Mycoplasma bovis, M. dispar, M. boviphilus, and Ureaplasma, also are found in many cases of enzootic pneumonia.

   (i) These organisms inhabit bronchiolar epithelium; thus, they cause ciliary destruction and changes in mucus composition as goblet cells proliferate and mucus hypersecretion occurs.

   (ii) The classic pathology attributed to mycoplasmal infection in these calves is peribronchial lymphocytic cuffing of the bronchioles. It appears that the adult cattle act as mycoplasma carriers and transfer the organisms by aerosol up to several meters’ distance to calves.

   c. Bacterial agents involved in this pneumonia include many of the organisms that can be found in the pharyngeal region of normal calves, including Pasteurella haemolytica, P. multocida, and Actinomycetes pyogenes. Chlamydia species also have been involved in some mixed infections. Involvement of these secondary agents results in purulent secretions accumulating in the airways and eventually the formation of microabscesses, bronchiolitis, or both. The pathology that occurs is a consolidation of cranial, middle, and accessory lobes with bronchiolitis and alveolitis, with varying degrees of consolidation and suppuration depending on the numbers and type of bacterial involvement.

   (2) Environmental factors

   a. The infectious agents are seldom able to induce pneumonia in calves with infectious pathology, mainly those that are housed in ventilated barns and those that are exposed to outdoors, especially during the fall and winter months.
out the added stress of poor or absent ventilation in the housing areas for the calves.

(b) Colder weather often precipitates problems when the producers try to prevent the calf housing temperature from dropping by reducing fresh air circulation. Consequently, there is increased humidity in the ambient air of the calves, which allows pathogen multiplication. This is compounded by a high pathogen density developing with the lack of fresh air to dilute the ambient air.

(c) Another factor of adverse air quality is the increased exposure to noxious gases (e.g., ammonia) that can adversely affect mucociliary clearance.

(3) Management factors include the crowding and mixing of various age groups that frequently occurs with indoor housing, particularly in older barns.

d. Diagnostic plan and laboratory tests
(1) Clinical signs of pneumonia are the main mode of diagnosis. Attempts can be made to isolate the virus using serology, but these efforts are often unrewarding and unlikely to change the methods of management of the problem.

(2) Laboratory studies. For the individual calf, a transtracheal aspirate can be performed. Cytology findings of increased numbers of degenerate neutrophils with numerous intracellular bacteria implicate secondary bacterial involvement. In these cases, bacterial culture and sensitivity may aid in guiding antibiotic treatment of the calf. A CBC indicates changes of leukocytosis and hyperfibrinogenemia where bacteria are involved.

(3) Radiography. For calves that have a prolonged course of pneumonia and respiratory distress, chest roentгенograms may be performed. Radiographic findings of significant Consolidation of the lung parenchyma (e.g., pneumonia) or new infiltrates (e.g., fibrin deposits) may indicate bacterial infections. However, these findings may also be seen in acute, nonbacterial pulmonary edema or in cases of atelectasis, pneumothorax, or pleural effusion. In these cases, bacterial culture and sensitivity may aid in guiding antibiotic therapy.

e. Therapeutic plan. Acutely affected calves often respond well to antibiotics, unless the infection is purely viral, as can occur in some BRSV infections.

(1) Although many of the commonly available antibiotics for cattle can be used, drugs that are effective against the mycoplasmas, such as tetracycline (10 mg/kg intramuscularly daily for 3–4 days) or oxytetracycline (10 mg/kg intramuscularly daily for 3–4 days), provide appropriate coverage against the spectrum of possible organisms involved.

(2) More recently, flunixin meglumine (2.2 mg/kg intravenously every 24 hours for 2–3 days) also has been advocated as an adjunct treatment because it may reduce lung inflammation in these calves.

f. Prognosis. In chronically debilitated calves that may have extensive lung damage, including abscessation, the prognosis for full recovery is poor, and therapy may not be cost-effective.

g. Prevention
(1) Vaccines. The commonly available vaccines against the viral pathogens are of questionable value, as colostral immunity usually interferes with adequate response at the time of administration.

(2) Management strategies
(a) For producers with major problems, individual outdoor calf hutch areas are highly recommended before the provision of ventilation and a level of isolation from older cattle.

(b) In addition, producers with less severe problems can reduce the incidence of enzootic pneumonia with attention to adequate ventilation in the calf barn, which removes pathogens, noxious gases, and excess moisture.

(c) Adequate colostrum feeding also may prevent enzootic pneumonia because survival of calves with respiratory disease has been directly correlated with serum immunoglobulin G levels.

4. Pneumonia in lambs and kids is similar to that in adult sheep and goats (see I B 3).

5. Pneumonia in piglets
a. Etiology. Pneumonia in piglets can be caused by viruses, including pseudorabies, swine influenza (see Chapter 6), and the recently described porcine reproductive and respiratory syndrome (PRRS).

(1) PRRS, also known as porcine endemic abortion and respiratory syndrome, blue-eared pig disease, swine infertility and respiratory syndrome, and originally "mystery disease of swine," was first recorded in the United States in 1987 and is thought to be caused by the Lelystad virus in Europe and a simi-
lar, if not identical virus, in North America. Since being reported in the United States, this virus has been recorded in Canada and several countries in Europe.

(2) Transmission appears to be airborne because even well-managed and isolated herds can become infected.

b. Clinical findings
(1) Adults. Signs include anorexia and fever lasting several days, after which there may be mid- to late-term abortions with large mummified fetuses or partially autolyzed fetuses, increased percentages of stillbirths, and weak piglets. This period of reproductive problems is followed or accompanied by severe respiratory disease in nursing or weaned piglets.

(2) Piglets. Signs include dyspnea, polydipsia, and decreased growth. The abdo-
men, ears, or vulva may appear bluish (hence the name blue-eared piglets). In a litter, up to 30% may have anemia, 30% show respiratory distress, 10% have fever, and only 5% show cyanosis. Morbidity in weanling pigs can be as high as 30%, with mortality of 5%–10%.

c. Diagnostic plan and laboratory tests. There are several serologic tests available to detect seroconversion to the virus, but because of the possible widespread infection, the serodiagnosis should be based on a herd rather than on a sample from an individual animal. There are no characteristic lesions in the aborted or stillborn pig-
lets or in the sows. Piglets will have a proliferative and necrotizing pneumonia and, in some cases, a purulent bronchopneumonia.

d. Differential diagnoses. This disease needs to be differentiated from other condi-
tions that cause abortion, stillbirths, and weak piglets, such as pseudorabies, par-vovirus, leptospirosis, and the encephalomyocarditis or hog cholera viruses.

e. Therapeutic plan and prevention. Because there is no specific treatment available, control is the main focus. There is not sufficient data available for understanding the methods of transmission, but restricting the movement of pigs from affected to unaffected areas has slowed the spread.

B. Pneumonia in growing and mature animals

1. Equine bacterial pneumonia
a. Patient profile and etiology. Pneumonia in the adult horse is not common and is usually secondary to upper respiratory virus infection (see I A 2 b (1)) or occurs after stressful events (e.g., shipping), in which case it often develops into pleuritis (see I E).

b. Diagnostic and therapeutic plans. The guidelines given for foals are used, with the exception that chest radiographs are not obtained because they are not readily available for monitoring progress in the adult horse.

2. Bovine respiratory disease complex (shipping fever, pneumonia pasteurellosis)

a. Patient profile and history. This disease can affect cattle of any age, breed, or sex. However, because of current management practices, this condition occurs primarily in young cattle between the ages of 6 and 18 months. Characteristically, the disorder follows a stressful event (e.g., shipping, mixing in sales), occurring 2–14 days later, and is one of the major causes of morbidity and mortality in feedlot cattle in North America.

b. Clinical findings
(1) Affected cattle are depressed, usually anorectic, and have rapid shallow respirations with a weak, productive cough. Early signs of the depression may be sim-
ply a failure to stretch freely when encouraged to rise. Fever is often as high as 41°C, and examination of the penes may reveal fever in animals that otherwise appear clinically normal.

(2) In the early stages of the disease, lung sounds only consist of increased breath sounds. In the advanced stages, cracks and wheezes become readily audible, and there may be pleural friction rubs in some animals. A mucopurulent
nasal discharge, crusty nose, and ocular discharge may appear, along with a
gaunt appearance to the abdomen after several days of not eating.

c. Etiology and pathogenesis
(1) Etiology
(a) This disease is a fibrinous pneumonia caused by Pasteurella haemolytica
(biocotype A, serotype I) and, less commonly, Pasteurella multocida or Hem-
ophilus somnus. Experimentally, these organisms alone do not produce dis-
ease without massive doses or manipulation of the host with viral patho-
gens or severe stressors.
(b) In naturally occurring disease, the associated stressful events are numer-
ous and include weaning, transportation, sudden changes in climatic con-
tions (e.g., temperature, humidity), inadequate or irregular feeding, water
depprivation, crowding, dehorning, castration, and vaccination. Respiratory
viral infection can be a factor, but in many outbreaks of naturally occur-
rising disease, there is no preceding viral infection.
(2) Pathogenesis. P. haemolytica and P. multocida are normal inhabitants of the
bovine upper respiratory tract. With the stressors, these organisms are able to
 colonize and proliferate in the lower respiratory tract. P. haemolytica stressors
are associated with increased numbers and virulence of the strain found in the
pharynx of cattle. Because this organism appears as the predominant agent
in this disease syndrome, most investigation into pathogenesis has focused on
P. haemolytica.
(a) P. haemolytica has four virulence factors that interact to produce disease:
fimbrae, polysaccharide capsule, endotoxin, and leukotoxin.
(i) The bacterial fimbrae enhance the colonization and proliferation of
bacteria in the upper respiratory tract.
(ii) The polysaccharide capsule inhibits complement-mediated serum kill-
ing as well as phagocytosis and intracellular killing of the organism.
(iii) Endotoxin is directly toxic to the bovine endothelium and greatly
modifies cardiopulmonary hemodynamics. Also, it can alter bovine
leukocyte functions.
(iv) Leukotoxin is a species-specific cytotoxin against ruminant leukocytes
and platelets, acting as a pore-forming cytolytin.
(b) Transmission. Cattle either have the bacterial organisms in their na-
sopharynx or become infected by aerosol from other cattle. However, the disease
does not appear to be contagious in the way it affects the individual.
(c) Route of infection
(i) With increased proliferation of the bacteria in the nasopharyngeal re-
 gion and increases in virulence type, P. haemolytica is allowed to
 colonize the airways of the lung. The presence of P. haemolytica lipopolysaccharide
(neutrophils to the lung as a normal defense mechanism.
(ii) Although local immunity such as immunoglobulin can be protective,
some forms of systemic immunity can actually enhance intrapulmon-
 ary pathology of Pasteurella infections, possibly because of opsoniza-
tion (immunoglobulin G) effect.
(iii) P. haemolytica produces an exotoxin during log phase growth that is
cytotoxic for ruminant neutrophils and macrophages. Therefore, the
increased bacterial phagocytosis induced by opsonization may merely
cause increased cell death of the very cells (neutrophils and macro-
phages) that are intended for defense. Also, release or "spilling" of
the cytotoxic contents of these cells can contribute to lung damage.
(iv) The pathology that results is a severe fibrinous pneumonia with in-
creased capillary permeability, thrombosis, and interstitial edema.
Bacterial cell death, which releases endotoxin (lipopolysaccharide),
probably contributes to these changes. In contrast to earlier theory,
viral involvement is not an essential component of this disease.

d. Diagnostic plan and laboratory tests
(1) Clinical findings of rapid onset of high fever, depression, and rapid, shallow,

(2) Laboratory analysis is seldom warranted with individual cases and is not diag-
nostic for shipping fever.
(a) Blood work. Predictably, there is a leukocytosis on the CBC, but there
also can be leukopenia if endotoxia is a major component. Hyperfibri-
ngenemia is usually present (greater than 7 g/dL).
(b) A transtracheal aspirate yields septic, degenerate tracheal exudate contain-
ing gram-negative rods and often Pasteurella species on culture.
(c) If nasopharyngeal swabs are taken, P. haemolytica can be cultured, but
there appears to be poor correlation of antimicrobial sensitivities between
isolates from the nasopharynx versus the lung. Therefore, these samples
should not be used to guide the choice of antibiotic for the treatment of
acute cases.

e. Therapeutic plan
(1) Antimicrobial treatment. Early treatment of acutely affected cattle with almost
any of the commonly available antimicrobials (e.g., oxytetracycline, trimetho-
prim-sulfadoxine, penicillin, sulfonamides) is highly effective.
(a) Surprisingly, a single-dose treatment in early stages is often sufficient,
but severely affected cattle should be treated daily for 3–5 days.
(b) Delayed or irregular treatment, inappropriate dose, or premature termina-
tion of therapy in some cattle can result in increased mortality. Cattle
with more than 50%–60% pulmonary consolidation generally respond
poorly and frequently relapse. These animals may be individuals that were
not detected in the acute stage or were not adequately treated early in the
course of the disease.
(2) In addition to antimicrobial treatment, sick cattle should receive adequate shel-
ter and good nutrition, particularly when the management system in place en-
courages crowding and competition for feed and water space.
(3) Response to treatment
(a) Antimicrobial-resistant strains of P. haemolytica have been appearing
and, with resistance being plasmid mediated, may significantly increase
the incidence of poor response to treatment.
(b) To assess adequate response to treatment, the rectal temperature should
be used as a guide. A positive response is the abatement of fever within
48 hours of treatment.

f. Prevention. Because this disease is so clearly linked to stressful events, a great
deal of the prevention can be managed by avoiding or lessening the impact of
known and preventable stressors.
(1) Preconditioning. The best example of stressful events is the practice of wean-
ing calves simultaneously with castration, dehorning, and vaccination. By per-
forming some of these procedures at a separate time, such massive stressors can
be lessened. This management method, called preconditioning or back-
grounding, vaccinates and processes (castrates, dehorns, implants) calves be-
fore weaning, all of which should occur 3 weeks to 1 month before shipping.
There are many variations of the timing and order of these preventive meas-
ures. However, the financial benefit to the producer is less clear, and unless a
 premium is paid for these calves at sale, the extra cost and effort may not be
worthwhile.
(2) Vaccination against shipping fever has been a main focus of prevention but
does not have been particularly effective until recently.
(a) Where respiratory viral agents (e.g., IBD, PI-3, BRSV) are involved, the
use of such antigens in vaccines may be beneficial when they are adminis-
tered at least several weeks before weaning and shipping. Also, there has
been evidence that vaccination with live products at the time of stress
may increase morbidity or mortality.
(b) Progress has been made in creating vaccines against the pathogenic effects
of P. haemolytica. Recently, a vaccine was produced to stimulate immu-
nity against the exotoxin or leukotoxin that is produced as a major part of
the pathogenicity of *P. haemolytica*. This vaccine appears to protect against pulmonary disease in experimental models of disease but is less effective in field use. Current evidence from field trials suggests that this vaccine at best only marginally reduces the morbidity and mortality.

3. **Chemoprophylaxis.** Another fact taken to reduce the morbidity and mortality of shipping fever is the administration of antibiotics at the time of stresses or upon arrival at a feedlot.

(a) **Drugs used** include long-acting oxytetracycline (LA200, 20 mg/kg) or tilmicosin (micotil, 10 mg/kg subcutaneously). Such treatment is presumed to be effective by postponing the bacterial invasion of the lung, so that the infection is usually less severe and less likely to result in relapses.

(b) **Medication in feed or water** has been used also, but both these approaches can give a false sense of security, reducing scrutiny of the cattle’s health and leading to more advanced cases occurring before diagnosis. Also, with the current consumer concern regarding drug use in animals that are intended for human consumption, such practices can create problems for the image of the industry.

3. **Ovine and caprine bacterial pneumonia**

a. **Pasteurellosis**

(i) **Patient profile and history.** Outbreaks are often associated with sudden changes in environment or climate (e.g., when a flock of sheep is exposed to inclement weather shortly after shearing).

(ii) **Etiology and pathogenesis.** As in cattle, *Pasteurella haemolytica* is the most common cause of bacterial infections of the respiratory tract in sheep and goats. However, the resulting diseases are not the same as those in cattle. If detected early, affected lambs or kids appear dull and become prostrate before death in a matter of hours. Sick animals are often unobserved until the final stages of the disease.

(b) **Predisposing factors.** Although *P. haemolytica* can be a primary pathogen in young lambs, older animals require predisposing factors for the occurrence of disease.

(i) **Virulent strains** occur in newborn lambs but has met with equivocal results.

(ii) **Adenovirus** has also been incriminated as predisposing to naturally occurring pneumonia. The organism can usually be isolated in large numbers from tonsils, lung, and subpleural areas of the neck and thorax, with edematous lungs and subpleural ecchymosis, but pneumonia is not a feature. There is ulceration and necrosis of the pharynx and esophagus, as well as the occurrence of small necrotic areas on the tips of the abomasal mucosal folds.

(c) **Prophylactic antibiotics**

(i) **Vaccination with autogenous and commercial bacterins** has not been effective in controlling disease. Specific serotypes or “protective” antigens may be absent from these products.

(ii) **Sulfonamides** have been recommended as a preventive for baby lamb pneumonia by intermittent medication of the drinking water of ewes before lambing.

(iii) **Management strategies**

(a) **Avoid overcrowding** by providing a minimum of 14 square feet per pregnant ewe and a minimum of 4–5 square feet for each lamb.

(b) **Provide fresh water** and shelter at lambing, which has an advantage in that housed animals with access to the outdoors are not carrying a high moisture content in their fleeces; thus, the humidity of the barn is lower. Shorn ewes will also seek shelter at lambing to reduce the risk of hypothermia due to exposure in newborn lambs. However, adequate shelter from wind and rain does need to be provided for the shorn ewes.

(iv) **Avoiding cold wet weather** approaching.

3. **Other bacterial causes of pneumonia in sheep and goats**

(a) **P. multocida**, *Streptococcus* species, *Escherichia coli*, and *Haemophilus* species may be present in ovine and caprine pneumonia. These bacteria arc
usually secondary invaders. *S. typhimurium* and *S. dublin* cause diarrhea and abortion in sheep and goats. Respiratory signs may be seen initially.

(2) *Franciscella tularensis* infection can result in acute illness manifested by high fever, prostration, diarrhea, and respiratory signs, including nasal discharge and coughing. Diagnosis is confirmed by the isolation of the causative agent, the presence of Dermacentor andersoni, and a die-off of rodents in the area.

4. Porcine pneumonia. Although individual pigs can be affected with pneumonia on a sporadic basis, there are two forms of infectious respiratory problems that plague the swine industry: enzootic pneumonia and pleuropneumonia.

a. **Enzootic pneumonia**

(1) Economic implications. Enzootic pneumonia consistently ranks as the most economically important disease in finishing pigs in North America. Enzootic pneumonia occurs worldwide and has a particularly high incidence in intensive rearing operations. In infected herds, the morbidity rate is highest during the growing period, but the case fatality rate is low. The main adverse effects of this disease include an increase in the need to treat clinical illness caused by secondary bacterial pneumonia and the reduction in feed efficiency and average daily gain for getting the pigs to market.

(2) Clinical findings. The most common form of the disease, as observed in enzootically infected herds, begins at 3–10 weeks of age and is insidious in onset.

(a) Initially, the only clinical abnormality is cough in a small proportion of the piglets. This increases such that most animals in the pen show persistent cough, which is particularly obvious at times of activity, such as feeding, and can continue through the entire growing period. The cough is dry and hacking, but signs of respiratory embarrassment are rare, and there is no fever or diminished appetite.

(b) Those pigs that develop signs of pneumonia usually have secondary invasion of the lungs by Pasteurella species or other bacteria. Clinical signs of disease become less obvious with increasing age.

(3) Etiology and pathogenesis

(a) Etiology. The disease is caused by the primary initiator *Mycoplasma hyopneumoniae* (*or suipneumonia*) with *Pasteurella multocida* as a common secondary invader of the lung. *M. hyopneumonia* appears to be the primary cause of enzootic pneumonia in pigs.

(b) Pathogenesis

(i) Piglets are infected by the mycoplasmal organism early in life, likely from the sow, but also possibly from airborne particles from other pigs.

(ii) *M. hyopneumoniae* causes peribronchiolar lymphoid hyperplasia and mononuclear accumulation in the lamina propria, resulting in obliteration of the bronchial lumens. Also, the bronchial mucous glands undergo hypertrophy, and there is hypertrophy of the type II alveolar epithelial cells and progressive loss of cilia on the bronchial mucosa, decreasing the defenses against secondary bacterial infection.

(iii) These damages heal on their own, but if secondary bacterial invasion occurs, more severe pathology occurs, including bronchopneumonia and pleuritis. These complications also cause decrease in feed efficiency and average daily gain.

(4) Diagnostic plan and laboratory tests. The gross and microscopic findings on the lungs of pigs affected with enzootic pneumonia are not pathognomonic; thus, a positive diagnosis requires culture of *M. hyopneumoniae* from tissues. However, a negative result can often occur because the organism is difficult to culture and is readily overgrown by other nonsignificant organisms, such as *M. hyorhinis*. A fluorescent antibody test or an enzyme-linked immunoperoxidase technique may help demonstrate the organism in tissues.

(5) Therapeutic plan. Treatment is usually restricted to individual pigs showing acute respiratory distress, which, being of a secondary bacterial nature, should respond to most broad-spectrum antibiotics.

(a) There is no effective treatment that eliminates infection by *M. hyopneumoniae*. However, antibiotics such as tyllosin tartrate (50 mg/kg) and tiamulin (10 mg/kg) orally for 10 days can reduce pulmonary lesions.

(b) Other antibiotics show activity against this mycoplasma, including tetracyclines and the newer fluoroquinolones, such as ciprofloxacin.

(6) Prevention. Because *M. hyopneumoniae* infects only pigs and transmission requires close pig-to-pig contact, its spread can be limited or even eradicated from a herd.

(a) Eradication is the most satisfactory method of control but requires depopulation of the herd, followed by repopulation with pigs from specific pathogen-free (SPF) herds. Such pigs are commercially available, having been raised in special units populated with the progeny of Caesarian-derived piglets.

(b) Separation. Less successful methods involve isolating the farrowing area for sows believed free of infection from the rest of the herd, and raising the piglets separately. Alternatively, newborn piglets can be treated with antibiotics effective against Mycoplasma species and removed to isolated premises, with subsequent serologic testing of the breeding herd and culling of seropositive animals. These techniques are far less successful than complete repopulation with disease-free pigs.

b. **Contagious pleuropneumonia**

(1) Economic implication. This disease is of major economic importance, is worldwide in occurrence, and appears to be increasing in prevalence with the increase in intensive swine operations.

(2) Patient profile and history. The disease is predominately found in growing pigs, from ages 2 to 6 months.

(3) Clinical findings

(a) The disease is characterized by rapid onset and a short course of severe dyspnea, the passage of blood-stained foam from the mouth, and a high case-fatality rate.

(b) Clinical course. The disease can be peracute, acute, or chronic, depending on the immune status, and each form is reasonably well defined. The clinical course of the disease in a herd can last several weeks, with new acute cases occurring as chronically affected animals develop a generally unthrifty appearance.

(i) In peracute cases, the only sign is sudden death in pigs that may be close to market weight.

(ii) In acute cases, severe respiratory distress is observed along with an exaggerated abdominal component (thumps), a reluctance to move, anorexia, and a fever up to 41°C.

(iii) In more chronic cases, there is fever and anorexia, but the respiratory distress is less severe and a persistent cough develops.

(4) Etiology and pathogenesis

(a) Etiology. The condition is caused by *Actinobacillus pleuropneumoniae* (formerly known as *Hemophilus pleuropneumoniae*), a highly contagious organism that is not isolated from normal porcine respiratory tissues but persists in chronic lesions in the lungs of recovered and apparently healthy pigs, which provide the source for continued infection.

(b) Pathogenesis

(i) It is thought that natural transmission occurs by the aeroogenous route, with the source of infection being a subclinically infected or recovered pig. Outbreaks appear to occur in pigs that lack immunity and are overcrowded or subjected to recent stressors, such as large...
fluctuations in temperature, recent transportation, or problems with ventilation in the barn.

When the organism begins to multiply in the lung (within hours of infection), there is rapid development of pulmonary edema and diffuse neutrophilic bronchiolitis and alveolitis. There are also marked vascular effects, which result in infarcts in the lung, thrombosis, and hemorrhage. A hemorrhage, in turn, may result in the pleural inflammation. Pigs appear to die of septic shock.

Diagnostic plan and laboratory tests. The clinical signs in growing pigs of rapid onset and sudden death associated with respiratory signs provides a presumptive diagnosis. Culture at necropsy confirms the infection, and there is a reliable serological test to assess recent infection in the live animals.

Therapeutic plan. Anthelmintics, such as fenbendazole (30 mg/kg or ivermectin (200 µg/kg orally), are effective treatment. Animals may initially worsen with treatment because of the death of larvae causing an intense inflammatory response. Therefore, affected horses may benefit from concurrent treatment with anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), or a glucocorticoid.

Prevention. Donkeys should be treated with appropriate anthelmintics, and pastures should be rotated. In temperate regions, the parasites are unable to survive during winter on pasture. Most routine broad-spectrum anthelmintic regimens for intestinal parasites also control lungworms in horses.

Parasitic pneumonia

1. Equine parasitic pneumonia

   a. Lungworms

      (1) Patient profile and history. The donkey is the natural host of the equine lungworm (Dictyocaulus amfieli). Horses become infected when they graze pastures with infected donkeys or previously contaminated pastures. The common complaints include a persistent cough, increased respiratory rate, and forced expiration in horses. Donkeys usually show no signs even with heavy infestation.

      (2) Clinical findings. The predominant clinical sign is a chronic cough. Horses are afibrile, and their appetites are unaffected.

      (3) Etiology and pathogenesis. The larvae migrate through the gut wall and are carried hematomogenously to the lungs.

          (a) In the donkey, the larvae mature in the bronchi and lay eggs. The eggs are coughed up and swallowed.

          (b) In the horse, the maturation of the larvae in the airways is retarded, and the worms remain immature; thus, the infection does not become patent. Lung pathology is limited to the caudal lobes. There is epithelial hyperplasia with an increase in size and number of goblet cells. Grossly, there are areas of overinflation, mucous exudate, and coiled worms.

      (4) Diagnostic plan and laboratory tests. Clinical signs of lungworm infestation must be differentiated from chronic obstructive pulmonary disease (COPD).

          (a) Clinical suspicion. Exposure to donkeys on pasture is suggestive of lungworm infestation, and although this infestation is not patent in horses and is fecal Baerman-negative, it may be useful to perform fecal sedimentation on any closely housed donkeys.

(b) A transtracheal aspirate shows neutrophils and eosinophils, and bronchoalveolar lavage may recover intact lungworms.

   (5) Therapeutic plan. Anthelmintics, such as fenbendazole (30 mg/kg) or ivermectin (200 µg/kg orally), are effective treatment. Animals may initially worsen with treatment because of the death of larvae causing an intense inflammatory response. Therefore, affected horses may benefit from concurrent treatment with anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), or a glucocorticoid.

   (6) Prevention. Donkeys should be treated with appropriate anthelmintics, and pastures should be rotated. In temperate regions, the parasites are unable to survive during winter on pasture. Most routine broad-spectrum anthelmintic regimens for intestinal parasites also control lungworms in horses.

b. Parastcard migration in foals

   (1) Patient profile and history. This disease occurs in foals and weanlings, with cough as the main complaint.

   (2) Clinical findings. Signs consist of a transient episode of coughing accompanied by a mucopurulent nasal discharge. Throughout the episode, the rectal temperature remains normal, and there is seldom sufficient damage in the lungs to cause a noticeable increase in the respiratory rate or depth.

   (3) Etiology and pathogenesis. The disease is caused by the larval Parasarcis equorum in the course of its development as an intestinal parasite of foals. When foals are infected with P. equorum eggs, through ingestion, the larvae penetrate the gut wall and undergo a hepatic–bronchial migration. The larvae are then carried by a hematogenous route and then migrate up the airways and return to the intestine to mature. Their presence in the airways stimulates mucous production. Signs are usually transient.

   (4) Diagnostic plan and laboratory tests. As in lungworm infections, a transtracheal aspirate or bronchoalveolar lavage may show eosinophils present in the cytology. However, as the clinical signs are usually mild and the clinical course transient, such diagnostic procedures are seldom performed.

   (5) Differential diagnoses. This infection must be differentiated from other causes of coughing in foals, particularly the far more prevalent viral respiratory infections.

   (6) Therapeutic plan. Most of the commonly used anthelmintics for horses are effective against adult P. equorum, with ivermectin also being effective against the larval stages.

2. Bovine parasitic pneumonia

   a. Patient profile and history. This disease occurs most commonly in dairy calves that are younger than 1 year of age in the summer and fall of the first season at pasture. Clinical disease may also occur in adult cattle that have minimal prior exposure to the parasite and have recently moved onto heavily contaminated pasture.

   b. Clinical findings

      (1) Acute form. In acute cases, severe verminous pneumonia occurs. Acute cases progress quickly, and death from progressive respiratory failure can occur in 3–14 days. This form of the disease occurs in calves 1–2 weeks after being moved to heavily contaminated pasture, and many calves are affected simultaneously.

          (a) There is a sudden onset of rapid shallow breathing with a marked abdominal component. Accompanying this is a frequent deep cough and a fever that may reach 41°C.

          (b) On auscultation, all portions of the lung are affected with increased bronchial tones and fine crackles. The animals can remain reasonably bright and active and attempt to eat, although severe respiratory distress may prevent eating.

      (2) Subacute form. The more common form of the disease is a subacute verminous pneumonia, which has a prolonged clinical course of 3–4 weeks.

          (a) In these cases, the onset is also sudden, with an increased respiratory rate...
(2) Necropsy. In areas of outbreaks, deaths are also frequent enough to allow post-mortem diagnosis, with the adult worms readily observed in the airways by the naked eye.

(3) Laboratory studies
(a) A transtracheal aspirate or bronchoaveolar lavage shows greatly increased numbers of eosinophils and possibly ova or larvae in the sample.
(b) Fecal sedimentation of affected animals should be examined by the Baer-
man technique for the presence of lungworm larvae.

e. Therapeutic plan
(1) Most modern, broad-spectrum anthelmintics [e.g., albendazole at 7.5 mg/kg orally, levamisole (13.6%) at 8 mg/kg subcutaneously, fenbendazole at 5 mg/kg orally] are active against all stages of D. viviparous, Ivermectin (0.2 mg/kg subcutaneously) is particularly effective against the immature and mature stages, even at one-fourth of the recommended dose. With the full recom-
mended dose, residual protection is provided for up to 28 days.

(2) The topical formulations of both levamisole and ivermectin are also effective. However, killing the parasite does not resolve the damage already present in the lungs.

f. Prevention
(1) Management strategies. Much of the prevention rests on pasture management, such as preventing overcrowding and avoiding continuous use of the same past-
ture for young stock. Wet, swampy pastures allow maximal larval develop-
ment and, therefore, should be used for grazing only the adult (immune) ani-
imals.

(2) Deworming. After winter housing, yearlings should be dewormed before re-
lease onto pasture so that they do not begin a new cycle of pasture contamin-
ation.

(3) Vaccination. In Europe, a vaccine is available that consists of irradiated larvae that cannot mature. This vaccine is administered orally to calves before they are turned out to pasture. This primes the immune response in advance of any exposure to natural infection.

3. Ovine and caprine parasitic pneumonia
a. Etiology. Lungworms that affect sheep and goats include Dictyocaulus filaria, Pro-
tostrongylus rufescens, and Muellerius capillaris. Each organism is capable of causing varying degrees of verminous pneumonia, but the former two appear to be of greater clinical significance.

(1) D. filaria is usually found in the posterior dorsal region of the diaphragm

(2) P. rufescens invades small bronchioles and may lead to secondary pneumonia and pleuritis. Although it is pri-\n
(3) M. capillaris infections in adult sheep usually result in nodular or diffuse le-

(4) Clinical findings
(a) D. filaria infection produces a cough that results from bronchial irritation,

(5) F. rufescens infestations cause clinical signs similar to those of D. filaria, with

(6) Infection with M. capillaris is relatively innocuous clinically but may consti-
tute a limiting factor in the production of choice lambs.
I

Aspiration pneumonia can occur in any species, and the etiology of this disease is variable. In some cases, aspiration pneumonia can occur as a result of misdirection of a stomach tube and subsequent introduction of fluid destined for the intestinal tract. Horses are also at risk for this disease, particularly when they are exercised, resulting from the parasitic bronchitis. In severe cases, pneumonia, poor growth, and debilitation can occur, but minimal clinical signs are apparent after experimental disease.

Pathogenesis. The parasites lays eggs in the lungs of the pig. The eggs are then coughed up, swallowed, and passed in the manure. The embryonated eggs or larvae are eaten by earthworms and develop successively to second- and third-stage larvae. Reinfestation occurs when the earthworm is eaten by other pigs.

d. Diagnostic plan. Necropsy is usually the method of diagnosis.

e. Therapeutic plan. Many of the broad-spectrum anthelmintics are effective, including levamisole and ivermectin, which are effective against the adult forms. Efficacy against all forms of the parasite requires treatment with products such as fenbendazole or albendazole in the feed for 2 weeks.

f. Prevention. Preventing parasitic pneumonia by pasture rotation is difficult. Larvae of *D. filaria* can withstand long periods of freezing. Snails, which are the intermediate hosts of *P. rufescens* and *M. capillaris*, are particularly prevalent in poorly drained pastures. Fencing of wet areas of pastures may be beneficial.

4. Parasitic pneumonia in swine

a. Patient profile and etiology. Parasitic pneumonia occurs in pigs that are raised in management systems that allow access to earthworms and is most prevalent in pigs ages 4–6 months. Lungworms that infest pigs include *Metastrongylus* (most common), *M. salmi*, or *M. pudendotectus*, with mixed infections possible.

b. Clinical findings. Clinical cases show a barking cough that is easily induced by exercise, resulting from the parasitic bronchitis. In severe cases, pneumonia, poor growth, and debilitation can occur, but minimal clinical signs are apparent after experimental disease.

c. Pathogenesis. The parasites lays eggs in the lungs of the pig. The eggs are then coughed up, swallowed, and passed in the manure. The embryonated eggs or larvae are eaten by earthworms and develop successively to second- and third-stage larvae. Reinfestation occurs when the earthworm is eaten by other pigs.

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3. Clinical findings
   a. Pain
      (1) Animals with acute pleuritis show chest pain and are sensitive to touch over the thorax.
      (2) Other signs of pain include abduction of the elbows, reluctance to lie down, and a splinted abdomen. In horses, these signs are sometimes misinterpreted as a sign of colic.
   b. Accompanying signs can include the presence of guarded, shallow respiration with a shallow cough and a nasal discharge, which may have a fetid odor. Anorexia, depression, and fever are usually present. Ventral edema in the brisket area, when present, is a hallmark of the effects of the pleural inflammatory process.
   c. In chronic cases, the signs may be less obvious, with weight loss, anorexia, depression, or ventral edema being the only significant clinical findings.
      (1) On auscultation of the lungs, there is usually an absence of lung sounds ventrally, which is accompanied by widely radiating heart sounds.
      (2) Pleural friction rubs may be detected but are surprisingly not a consistent finding in pleuritis.
      (3) There is ventral dullness on percussion of the chest.
   d. In acute cases in the horse, ventral dullness is often associated with a horizontal fluid line. Because the disease in cattle is most often a component of diffuse fibrinous pleuropneumonia, no discrete horizontal line is to be expected on chest percussion.

4. Etiology
   a. Equine pleuritis results from the stress of transport, trauma of perforating thoracic wounds, esophageal perforation, or, less commonly, lymphosarcoma or accompanying equine infectious anemia. Solely infectious causes also are sporadically reported and include infections by Mycoplasma felis or Nocardia species.
   b. Bovine pleuritis is usually secondary to Pasteurella pneumonia or traumatic reticuloperitonitis. An infectious cause of bovine pleuritis, contagious bovine pleuropneumonia (Mycoplasma mycoides var mycoides), caused large losses in the North American cattle population when the continent was being settled, but this has been eradicated from the continent.
   c. Porcine pleuritis. Pigs have pleuritis and pleural effusion as part of actinobacillosis and in polyserositis of Glasser's disease caused by Hemophilus suis or parasuis infection, which also results in acute arthritis, peritonitis, and pericarditis (see Chapters 8 and 13).

5. Diagnostic plan and laboratory tests
   a. The clinical signs of systemic illness, accompanied by chest pain and shallow breathing, are highly suggestive of pleuritis. Thoracic percussion in the acute phase is suggestive of fluid within the chest.
   b. Of the laboratory tests available, both hematology and routine biochemistry are nonspecific. A neutrophilia with or without left shift and possibly anemia of chronic infection may be noted on hematology, and low albumin accompanied by elevations in fibrinogen and globulins are observed on biochemistry.
   c. More definitive tests for diagnosis include radiology, ultrasonography, and thoracentesis.
      (1) On chest radiographs, there is a pleural effusion line and often signs of an accompanying pneumonia, pulmonary abscess, or both. With adult horses and cows, this finding is often restricted in value because the large chest size reduces the diagnostic quality of the radiographs.
      (2) Chest ultrasound is highly sensitive for the detection of pleural fluid and can provide information regarding pleural thickening, loculation of pleural fluid, and the presence of fibrin. Also, hyperchoic echoes in the pleural fluid can suggest the presence of gas bubbles caused by anaerobic bacteria. These findings dictate highly specific therapy and an unfavorable prognosis.
      (3) Thoracentesis is often key in assisting in diagnosis, therapy, and prognosis.

6. Therapeutic plan. Chest drainage, appropriate antibiotics, and supportive nursing care are required for horses with pleuritis. The other large animal species seldom receive similar intensive care; thus, this discussion focuses on the horse. Should treatment be required, similar methods can be used for specific treatment of pleuritis in other species.
   a. Drainage of pleural fluid is important if the process in the chest is highly purulent.
      (1) Continuous drainage can be managed by using indwelling chest drains with a one-way Heimlich valve, but this is a labor-intensive venture and may not be practical, particularly in many stable environments. This method is associated with complications, such as celluria and possible pneumothorax.
      (2) Alternatively, repeated thoracocentesis can be performed every several days until the pleural inflammation subsides with treatment. In chronic pleuritis, fibrinous adhesions often impair complete drainage.
   b. Antibiotics. Selection of the appropriate antibiotic for treatment is initially based on Gram-stain results.
      (1) In horses, sodium penicillin (20,000 IU/kg intravenously every 6 hours) and gentamicin (2.2 mg/kg intravenously every 8 hours) are commonly chosen because this combination is effective against most gram-positive, gram-negative, and non-Bacillus fragilis anaerobic bacteria.
      (2) In circumstances in which B. fragilis is involved, (penicillin producers) metronidazole should be added to the regimen (25 mg/kg orally every 12 hours).
      (3) Alternatively in horses, chloramphenicol (25 mg/kg orally every 6 hours) can be used as a sole treatment. Chloramphenicol should not be used in food-producing animals.
   c. Supportive care. Stress should be minimized. Rest and fluids should be provided as required. Pain relief using NSAIDs (e.g., phenylbutazone, flunixin meglumine) may help the animal regain its appetite and increase its comfort.
   d. Additional treatment
      (1) Heparin (40 IU/kg subcutaneously every 12 hours) may decrease adhesion formation.
      (2) For chronic, one-sided pleuropneumonia, rib resection and thoracotomy to drain purulent material has been a successful salvage procedure.

7. Prognosis. On initial assessment, the prognosis is often poor, particularly for return to performance. Also, pleuritis is very expensive to treat. However, there is a wide range in recovery, from respiratory cripples to apparently complete resolution. The prognosis is guarded in anaerobic and gram-negative infections with accompanying pneumonia, pulmonary abscess, or both. Conversely, some horses with mild pleural effusion appear to have full recovery. Unfortunately, no one laboratory parameter accurately determines the prognosis.

F. Noninfectious respiratory diseases
1. Metastatic pneumonia (also called veno cava syndrome)
   a. Patient profile and history. This disease occurs in cattle older than 1 year and can
Chronic obstructive pulmonary disease (COPD)

c. Etiology and pathogenesis

(1) This disease is thought to develop from an initial rumenitis secondary to lactic acidosis (see Chapter 3 1 A 2). As a result of the chemical damage to the rumen epithelium, bacteria (e.g., Fusobacterium necrophorum, Actinobacillus pyogenes) are able to penetrate the rumen epithelium to be transported to the liver portal drainage system, where they are filtered and cause liver abscesses.

If an abscess is located adjacent to the caudal vena cava, it may result in the development of septic emboli within the caval vein. This condition then shows the lungs and causes pulmonary arterial thrombosis and pulmonary abscessation, along with pulmonary hypertension and aneurysm formation.

(2) The eventual hemoptysis that is so clinically distinctive is the result of erosion of a pulmonary abscess into an arterial wall, rupture of a pulmonary aneurysm, or both. Other signs, such as anemia, hemic murmur, melena, and widespread wheezes are directly related to the massive lung bleeding.

d. Diagnostic plan and laboratory tests

(1) The CBC shows anemia and a neutrophilic leukocytosis. Serum chemistry may have hyperglobulinemia and liver enzyme changes (elevated aspartate aminotransferase, \( \gamma \)-glutamyl transferase) merely passive reflection of massive congestion.

(2) Chest radiography usually shows only an irregular increase in lung density, but in some cases, there may be more definitive changes, including small dense shadows indicative of embolic infarction and collapse or large spherical densities with cavitating nodules and gas or fluid interfaces.

e. Therapeutic plan

(1) The case fatality rate is usually 100%. Thus, when the diagnosis is established, treatment is rarely indicated.

(2) In valuable animals, supportive treatment can be undertaken, such as blood transfusion in the acute stage, along with furosemide (0.4–1.1 mg/kg intravenously) and flunixin meglumine (0.3–1.1 mg/kg intravenously or intramuscularly; given one to three times daily) as needed for the dyspnea. For the organisms usually involved, penicillin is the drug of choice, with a dose of 22,000 U/kg intramuscularly twice daily for extended periods (weeks to months).

f. Prevention. Because the initiating basis of this disease is rumenitis with subsequent liver abscess formation, measures to reduce the possibility of developing a pulmonary abscess syndrome include:

(1) Slowing the introduction of high-energy rations to the cattle

(2) Feeding antibiotics during the periods of increase in concentrate feed

2. Chronic obstructive pulmonary disease (COPD)

a. Patient profile and history

(1) This is a worldwide disease of horses that are usually more than 5 years of age and is seen more frequently in stabled animals.

(2) Clinical signs associated with COPD (also known as chronic airway disease, heaves, broken wind, emphysema, chronic bronchitis, or recurrent airway obstruction) are usually exacerbated by poor environmental conditions, such as poor ventilation, overcrowding, dusty stables, or breathing mists from stored feeds.

b. Clinical findings

(1) The main complaint is a chronic cough and sometimes an associated exercise intolerance. The cough is usually worse when the horse is stabled. Other common signs include bilateral mucopurulent nasal discharge, dyspnea characterized by nostril flaring, an abdominal lift to the expiration, abdominal muscle hypotrophy (heave line), and pumping of the ars on respiration.

(2) On examination of the lung fields, there are often crackles, wheezes, or both on auscultation, and expanded caudal lung borders with hyperresonance on chest percussion.

(3) Affected horses are alebrile and usually maintain an excellent appetite and demeanor, unless the increased effort of breathing is severe enough to interfere with eating. In such circumstances, weight loss may also occur.

c. Etiology and pathogenesis

(1) Etiology. Proposed instigating factors for horses developing COPD include previous respiratory viral infection, allergies to dust and fungal spores, dietary factors such as ingestion of 3-methylindole, and, in some horses, genetic predisposition.

(2) Pathogenesis

(a) Irrespective of the initiating cause, the affected horse develops excessive pulmonary reactivity to ill-defined airborne allergens, which, when present in sufficient concentration in the ambient air, induce clinical signs. Conversely, affected horses can be relatively free of any clinical signs when not exposed to these allergens and in dust-free settings.

(b) The structural changes seen in the lungs of affected horses with these clinical signs vary from chronic bronchitis with diffuse epithelial hyperplasia and mucus plugs to acinar overinflation and peribronchial fibrosis and cellular infiltration.

d. Diagnostic plan and laboratory tests

(1) Clinical signs of chronic recurrent cough in an older horse associated with stabling and no systemic sign of illness is usually sufficient for establishing a diagnosis.

(2) Laboratory studies. The CBC is almost invariably normal. Transtracheal aspirate or bronchoalveolar lavage cytology reveals large numbers of non-degenerate neutrophils, which are present in the lower airways because of immune stimulation rather than bacterial infection.

(3) Atropine challenge. Horses can be tested pharmacologically for the presence of airway spasm by administering atropine (0.022 mg/kg intravenously). Resolution of clinical signs of dyspnea and improvement of lung sounds indicates a positive response. These results also determine the proportion of the clinical signs caused by reversible bronchospasm, which can be treated with bronchodilator treatment.

(4) Arterial blood gas determination and pulmonary function testing. A resting arterial oxygen tension (\( PaO_2 \)) of less than 83 mm Hg and a maximum change in intrapulmonary pressure with a tidal breath of greater than 6 mm Hg are suggestive.

(5) Chest radiographs may demonstrate prominent bronchial and interstitial markings but seldom contribute substantially to diagnosis or disease management.

e. Differential diagnoses. The differential diagnoses can include bacterial pneumonia, pulmonary neoplasia, and diffuse restrictive diseases. Radiography and lung biopsy are the primary tools for these differentiations.

f. Therapeutic plan

(1) Management strategies

(a) Improvement of air quality in the horse's environment is the most important factor in managing the clinical signs and decreasing the progression of the disease. The horse should spend as much time as possible outside on pasture.
3. Hypersensitivity

**Pneumonitis** (extrinsic *alveolitis*, bovine farmers' lung)

(a) Patient profile and history. This disease occurs almost exclusively in housed adult cattle (particularly dairy cattle) that are fed stored roughage feed. Although the disease occurs sporadically, it appears mainly in the fall, winter, and early spring, when cattle are confined.

(b) Clinical findings

(1) The main complaint is usually the increasingly progressive respiratory distress and coughing, along with anorexia and decreased milk production in an individual cow.

(2) On clinical examination, the affected animal shows tachypnea, expiratory dyspnea and a dry, nonproductive cough. There may be a thick nasal discharge, and on chest auscultation, there are increased bronchial sounds and crackles. Fever may also be present but it is usually transient.

c. Etiology and pathogenesis. The respiratory distress is attributable to a hypersensitivity reaction to thermophylic molds (Microspora faeni and Thermoactinomyces vulgaris) that contaminate roughage feeds. Antigen exposure in sensitive individuals activates cellular and humoral immune responses. The pulmonary inflammation results from complement activation, histamine release, and polymorphonuclear neutrophil and macrophage recruitment. The resulting pathology is interstitial infiltration by lymphocytes, plasma cells, macrophages, and granuloma formation. Clinical signs include dyspnea, cyanosis, and evidence of respiratory distress.

d. Diagnostic plan and laboratory tests

(1) On transtracheal aspirate, there are neutrophils and macrophages in the cytology but no bacteria unless there is a secondary bacterial pneumonia. It is the absence of these signs of infection that suggests consideration of this immune mediated disease.

(2) Suspect cattle can have serum samples analyzed for the presence of precipitins to the antigens of *M. faeni* and *T. vulgaris*. Also, the *M. faeni* antigen can be administered intradermally, and a positive reaction may be noted as a local Anthus reaction after 4--8 hours.

e. Therapeutic plan. Antihistamines and corticosteroids may provide some relief for individual animals, but when the respiratory signs are noticed, the pulmonary damage is usually irreversible.

f. Prevention. When the producer recognizes the problem, only the change in management (if the cattle must be housed for extended periods) is to minimize antigen exposure by feeding hay outside or by feeding silage.

4. Acute respiratory distress syndrome (ARDS, acute bovine pulmonary edema (ABPE), foal fever)

(a) Patient profile and history. This severe type of respiratory distress occurs in late summer and fall mainly in adult beef cattle (ages 3--8 years) that have a history of having been moved from poor to lush pasture in the previous 1--2 weeks. Geographically, this disease appears to be most prevalent in the western part of North America.

(b) Clinical findings

(1) The main complaint is usually an acute onset of severe dyspnea or open-mouth breathing, expiratory grunt, and tachypnea in mature cattle, often with several to many in a herd affected simultaneously. Although the heart rates are usually elevated, fever (suggestive of an infectious cause) is not usually a prominent feature.

(2) The mucous membranes can become cyanotic as respiratory embarrassment advances, and subcutaneous emphysema over the shoulders and thoracic inlet may develop as a consequence of the severe respiratory effort. There may even be some cattle found dead with few premortem signs.

(3) In acute cases, there are usually increased bronchial tones over the ventral aspects of the lungs, but the dorsal portions of the lungs are surprisingly silent, with a relative absence of breath sounds despite the obvious respiratory distress. Nursing calves running with the cows are usually completely unaffected.

c. Etiology and pathogenesis

(1) This disease is an interstitial pneumonia with lung changes of pulmonary edema and interstitial emphysema, which is the result of ruminal transformation of dietary L-tryptophan to more toxic 3-methylindole (3-MI).

(a) A sudden feed change to lush pasture or brassica plants (e.g., rape, canola, tops of turnips) results in the overgrowth of ruminal lactobacillus, which produces toxic levels of 3-MI from dietary L-tryptophan. The ruminally produced 3-MI is then absorbed into the circulation and is metabolized by microsomal enzymes in the Clara cells of the lung to pneumotoxic metabolites that injure the alveolar and capillary epithelium.

(b) Consequently, there is plasma transudation in the exudative stage. If the animal survives, there is a proliferation of type II pneumocytes, alveolar epithelialization, and irreversible fibrosis. At necropsy, there is subpleural and interstitial emphysema, alveolar edema, epithelial hyperplasia, and hyaline membrane formation.

(2) There are several other causes of ARDS, with no clinical and pathologic pictures being indistinguishable from the mechanism described in 1 F 4 c (1). These other causes include:

(a) Pulmonary damage by mixed function oxidase metabolism of other...
5. Exercise-induced pulmonary hemorrhage (EIPH)

a. Patient profile and history. This disorder occurs in horses that undertake strenuous exercise such as racing. There is no relation of its occurrence to gender or finishing position, but its incidence may increase with age. The main complaint is either epistaxis at exercise or, more commonly, poor athletic performance without an offending site, such as new pasture or feed source.

b. Clinical findings. In addition to epistaxis, clinical findings are likely to include problems related to athletic performance. Owners may report suspected EIPH horses as losing speed near the end of the race, after which they may take longer to "cool out." Affected horses may be observed to swallow more frequently during this cooling-off period.

c. Etiology and pathogenesis. The bleeding occurs mainly in the dorsal portions of the lung and appears to be related to events during strenuous exercise. Various theories have been offered to explain EIPH. Rupture of blood vessels is likely necessary for hemorrhage to occur.

(1) An older theory proposes that pulmonary hypertension and edema are the mechanisms of action. This was the rationale for treating the problems with furosemide. Furosemide therapy remains a popular treatment for "bleeders" on the race track, but there is no clinical evidence showing improvement of the lung bleeding. Recently, furosemide has been shown to have bronchodilating effects.

(2) A more recent theory is that EIPH occurs in horses that have subclinical lung disease, causing some degree of bronchial obstruction. This obstruction may be sufficient to prevent the filling of the alveoli distal to them when the respiratory rate is increased.

d. Diagnostic plan. Diagnosis usually rests on the clinical history and risk factors for the possible causes, coupled with the signs of an acute onset of severe respiratory distress without fever or tachycardia.

e. Differential diagnoses. Additional causes of acute respiratory distress in cattle that appear to be similar in clinical appearance to ARDS include acute immune reactions such as systemic anaphylaxis, milk allergy, or massive pulmonary migration of parasites.

f. Therapeutic plan. There is nothing specific that can be done to reverse the lung damage of affected cattle. Therefore, the main treatment goal is to reduce the adverse effects of the cell damage and inflammation as well as pulmonary edema.

(1) Furosemide at 2.2 mg/kg intravenously every 12 hours (extra-label use) is useful for its anti-inflammatory effects and furosemide at 0.4–1 mg/kg intravenously or intramuscularly every 12 hours reduces pulmonary edema.

(2) Equally important in management of these animals is the avoidance of stress or exercise in hypoxic animals and removal of affected animals from any offending site, such as new pasture or feed source.

g. Prognosis. The prognosis is grave in severely affected animals, for which slaughter and salvage is often the most preferred option. For ARDS associated with 3-MI, clients should be cautioned that removal from pasture may not stop further clinical cases from developing in the short term.

h. Prevention. In ARDS associated with 3-MI, the herd should have limited access to pastures with pathogenic potential, such as alfalfa, kale, rape, turnips, or rapidly growing lush pasture. Where the problem is common and not readily avoidable by appropriate pasture management, monensin or lasalocid (700 mg/kg) beens will effectively prevent the disease because these products inhibit overgrowth of ruminal lactobacilli that convert the dietary L-tryptophan to 3-MI.

6. Exercise-induced pulmonary hemorrhage (EIPH)

DISEASES OF THE THORAX

A. Diaphragmatic hernia

1. Patient profile and history. The type of animal affected with a diaphragmatic hernia varies. Often the history includes a previous trauma, such as dystocia, breeding, or foaling trauma, or severe physical exertion. Specific to cattle, traumatic reticuloperitoneal hernia (TRP) is a main cause of this uncommon problem.

2. Clinical findings. Depending on the extent of the damage to the diaphragm and the amount of abdominal content herniated, the signs can range from mild colic and dyspnea to acute severe colic with tachypnea and obvious dyspnea.

a. Gastrointestinal sounds may be heard over the thorax, and lung sounds can be reduced or even absent over one side of the chest.
b. If the small intestine becomes strangulated in the hernia, there may be gastric re-flux. In cattle with herniation associated with TRP, forestomach stasis can occur.

3. Diagnostic plan
   a. Radiography. When diaphragmatic hernia is suspected, chest radiography is usually diagnostic. Findings include loss of the diaphragmatic shadow and multiple fluid lines in the thorax that are associated with intestines in the thoracic cavity.
   b. Thoracocentesis generally yields a serosanguinous fluid, but this is in no way definitive for this specific diagnosis.
   c. Electrocardiography. Animals with diaphragmatic hernias usually have decreased amplitude of QRS complexes on an electrocardiogram (ECG).

4. Therapeutic plan and prognosis. Surgical closure of the diaphragmatic defect is the treatment of choice. Given the size of the large animal patients, the prognosis for success is only fair to guarded even if surgery is performed.

**Pneumothorax**

1. Patient profile and history. As in diaphragmatic hernia, all types of animals can be affected with pneumothorax. Usually there is some history of chest trauma. Undergoing an invasive technique, such as thoracocentesis or lung biopsy, may also be part of the recent history.

2. Clinical findings
   a. The main clinical sign with pneumothorax is a variable degree of dyspnea, the degree depending largely on the amount of air in the chest and subsequent lung collapse. If pneumothorax is associated with an open chest wound, this is usually obvious.
   b. On chest auscultation, there is an absence of lung sounds over the affected side because of collapse of lung parenchyma away from the chest wall. In some cases, the air also is found under the skin over the chest in the form of subcutaneous emphysema.

3. Etiology. Trauma to chest from external means, such as a penetrating wound, can result in pneumothorax. Alternatively, air leakage through the visceral pleura of the lung from lung damage, such as in rib fracture or ruptured emphysematous bulla, can also result in pneumothorax.

4. Diagnostic plan. Chest percussion often demonstrates a drum-like resonance with the presence of pneumothorax. Aspiration of air freely from the chest cavity by thoracocentesis confirms the diagnosis. With mild cases, chest radiographs may be the only diagnostic method, with retraction of the lung margins from the dorsal-most part of the thorax.

5. Therapeutic plan. The main goals in treatment of pneumothorax are to remove a sufficient amount of air from the chest cavity to resolve signs of dyspnea and to treat, if possible, the underlying cause.
   a. Any penetrating chest wound needs closure, at which time routine wound prophylaxis of antibiotics and administration of tetanus antitoxin (for horses) can be performed.
   b. To evacuate the chest of air, a teat canula can be placed into the pleural space and continuous suction applied. Medical suction devices are available for such purposes, but it is also possible to perform this in an ambulatory setting on dairy farms by using the suction from the milk line.

**Study Questions**

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the ONE numbered answer or completion that is BEST in each case.

1. Which one of the following statements regarding Rhodococcus pneumonia is true?
   (1) The treatment of choice is oral rifampin combined with a macrolide, such as lincomycin.
   (2) Foals up to 4 months of age can be acutely infected from this soilborne organism; thus, a foal with clinical *R. equi* should be isolated from other foals to prevent the spread of infection.
   (3) In addition to pulmonary abscessation, other sequelae to infection can include uveitis, nonseptic arthritis, ulcerative colitis, and vertebral body abscesses.
   (4) This organism is able to live and grow within macrophages because it can prevent the fusion of phagosomes to lysosomes and has an antiphagocytic capsule (M protein) that prevents phagocytosis by granulocytes.
   (5) Serocconversion to *R. equi* in conjunction with signs of pneumonia and a leukocytosis is the preferred method of diagnosis.

2. Which statement regarding enzootic pneumonia in calves is true?
   (1) This disease is associated with bacterial pneumonia [secondary to bovine respiratory syncytial virus (BRSV) or parainfluenza-3 (PI-3) infection] or mycoplasmas, such as Mycoplasma mycoides.
   (2) Enzootic pneumonia can occur in beef calves between ages 2 months and 5 months that are housed indoors in the fall and winter.
   (3) Vaccination against viral agents such as BRSV or PI-3 in the first several weeks of life can help prevent this disease.
   (4) Cold weather can precipitate this disease when producers leave housing ventilation open, exposing calves to cold outside air.
   (5) The pathology is usually a consolidation of cranial, middle, and accessory lung lobes, with mycoplasmal involvement classically resulting in alveolitis and bronchietasis.

3. Regarding pasteurellosis in ruminants, which one of the following statements is true?
   (1) In sheep and goats, pasteurellosis due to *Pasteurella haemolytica* can occur in two forms: rapidly fatal septicaemia with biotype T and pneumonia with biotype A.
   (2) In cattle, shipping fever pneumonia due to *Pasteurella* usually peaks several days after the stressful event.
   (3) Vaccination against pasteurellosis in cattle should include the leukotoxin of *P. haemolytica* because it increases specific immunoglobulin A production in the lungs.
   (4) Vaccination against pasteurellosis in cattle should include the respiratory viruses [infectious bovine rhinotracheitis (IBR), parainfluenza-3 (PI-3), bovine respiratory syncytial virus (BRSV)] because prior viral infection that accompanies the stress of handling is usually required for development of this disease.
   (5) Pathogenic *P. haemolytica* strains can be part of normal pharyngeal flora in cattle, whereas the strains in sheep and goats spread from subclinical carrier animals.
4. Which one of the following statements regarding lungworms in large animals is true?

(1) The lungworm Dictyocaulus arnfieldi causes a patent infection in donkeys and results in a persistent cough in these infected animals.

(2) Metastrongylus species can cause lungworm in pigs raised outdoors, and though minimal clinical disease is found in experimental infection, the infection can result in exercise-induced cough and poor growth in field situations.

(3) Reinfection syndrome of D. viviparus in mature cattle causes disease because effective immunity is short-lived and has waned.

(4) D. filaria and Protostrongylus rufescens in sheep cause similar clinical signs, but because P. rufescens has a direct life cycle, it can cause more severe disease in adults.

(5) Although it causes a low-grade productive cough in growing kids and lambs, Mecistocirrus capillaris can be effectively treated with a single dose of fenbendazole.

5. Which one of the following statements regarding pleural disease of large animals is true?

(1) Clinical signs of shallow breathing and chest pain are caused by the inflammation of pain receptors on the visceral pleural surfaces.

(2) Pleural fluid accumulation is usually readily apparent in both horses and cattle with pleuritis.

(3) Unilateral thoracic disease is possible in ruminants because of the intact mediastinum, but disease can spread bilaterally in horses and pigs because of the thinner, often perforated mediastinum.

(4) Normal pleural fluid has a cell count of less than 10,000/μl white blood cells, but it is usually difficult to obtain in the clinically normal large animal.

(5) A foul odor of the pleural fluid in cases of pleuritis is suggestive of Escherichia coli growth, warranting inclusion of aminoglycoside treatment.

6. A herd of cattle is moved from sparse, meager pasture to a lush grazing of turnip tops. The adult cows develop severe dyspnea, open-mouthed breathing, and in some, subcutaneous emphysema. Which one of the following statements best applies to this problem?

(1) The new pasture was likely higher in 3-methylindole (3-MI), which caused the direct lung damage by acting on the Clara cells.

(2) The lung damage from preformed toxin results in eventual hyaline membrane formation and irreversible fibrosis.

(3) Pretreatment of this herd with monensin or lasalocid before pasture change can prevent such outbreaks.

(4) Nursing calves are less severely affected, with signs of mild expiratory dyspnea and cough.

(5) Associated with the severe respiratory distress, lung sounds are typically harsh with crackles and wheezes over the entire lung field.

7. A horse shows blood at the nostrils following intense exercise. Which one of the following statements is true?

(1) The blood likely originated from the caudal lung lobes as a result of left heart failure and fluid overload of the lungs.

(2) The medication furosemide is allowable for this condition in certain racing jurisdictions.

(3) The blood most likely originated from the ethmoid region or guttural pouch because this is an uncommon clinical finding in race horses.

(4) The bleeding clearly indicates a performance-limiting problem.

(5) Treatment with procoagulants, such as aminocaproic acid or vitamin K<sub>1</sub>, is indicated.

8. Which one of the following statements regarding thoracic disease in large animals is true?

(1) Diaphragmatic hernia in horses is most often caused by trauma, such as dystocia, whereas in cattle it has been linked to traumatic reticuloperitonitis.

(2) Signs of diaphragmatic hernia in horses can be mild to moderate colic and dyspnea, whereas cattle have occult herniation because the rumen is too large to herniate into the thorax.

(3) Gastrointestinal sounds (borborygmi) heard over the ventral chest of the horse are highly suggestive of diaphragmatic hernia.

(4) In cattle with pneumothorax, there is a drum-like resonance to percussion over the chest and very harsh lung sound ventrally because of the collapse of the lung.

(5) Sometimes uncovered incidentally, finding decreased amplitude of QRS and high spikes on the T wave of an electrocardiogram (ECG) of a horse are suggestive of diaphragmatic hernia.
1. The answer is 3 [I A 2 b (2)]. The presently accepted and specific treatment for *Rhzodococcus* equi pneumonia of foals is a combination of rifampin and erythromycin estolate. In horses, the use of oral lincomycin can induce fatal colitis. There is little benefit in isolating foals with *R. equi* pneumonia. Foals are resistant at 5 weeks of age; therefore, clinically affected foals pose little danger to most foals on the premises. The organism can prevent fusion of phagosome to lysosome but does not have an M protein capsule. Seroconversion is not a measure of disease because many foals seroconvert because of exposure to *R. equi* without ever developing *R. equi* pneumonia.

2. The answer is 2 [I A 3 a]. The disease can occur in any group of calves (not only dairy) that are between 2 months and 5 months of age and housed indoors, particularly when exposed to older cattle. Bacterial pneumonia, which is the end result of the enzootic pneumonia complex, may be secondary to infection by Mycoplasma species, but M. *mycoides* has not been described as a precursor agent. Prevention of enzootic pneumonia involves changing management practices. Vaccination has not proven beneficial because levels of colostral immunity interfere with the timing of vaccination. Cold outside air does not precipitate the disease. The buildup of noxious gases in poorly ventilated spaces has the negative impact on mucociliary clearance. The pathologic description in the last choice is accurate; however, this is the result of bacterial not mycoplasmal involvement.

3. The answer is 1 [I B 3 a]. Pasteurellosis in sheep and goats may appear as an acute septicaemia as well as a primary pneumonia. The clinical signs of shipping fever in cattle may not occur until 2 weeks after the stressful event (most commonly co-mingling cattle). Viruses are not essential precursors for the disease. Vaccination is not known to produce protective levels of immunoglobulin A antibodies. Pathogenic *Pasteurella haemolytica* strains can be part of the normal pharyngeal flora in cattle, sheep, and goats.

4. The answer is 2 [I C 4 a]. *Dictyocaulus* arnfieldiodes does not cause clinical signs in its natural host, the donkey. Re-infection syndrome of *D. viviparatus* is experienced in cattle with immunity (i.e., this is an immune-mediated reaction to re-infection with massive numbers of infection larvae). *Protostrongylus rufescens* has an indirect life cycle, with the land snail as the intermediate host. *Mijellarius capillaris* is difficult to treat, requiring treatment with fenbendazole over a 2-week period.

5. The answer is 4 [I E 5 c (3)]. The visceral pleura lacks pain receptors and is not the site of pain with pleuritis. In swine, the mediastinum is intact similar to ruminants. Pleuritis may not be obvious on clinical examination, particularly in cattle where the pleuritis is often more diffuse and a component of fibrinous pleuropneumonia. A necrotic foul odor to the breath or pleural fluid indicates the possibility of an anaerobic infection, suggesting treatment with metronidazole.

6. The answer is 3 [I F 4 a, b, h]. The clinical description best fits a diagnosis of acute respiratory distress syndrome in the bovine. In this condition, 3-methylindole (3-MI) is not ingested preformed but as dietary L-tryptophan. Clara cells metabolize the ruminally manufactured 3-MI to pneumotoxic metabolites, which injure the alveolar and capillary epithelium. Nursing calves (prematures) are not affected because the source of L-tryptophan is the pasture. A very important clinical feature of this condition is the absence of breath sounds over the dorsal portions of the lung fields despite the obvious respiratory distress.

7. The answer is 2 [I F 5 b, f]. Blood at the nostrils following intense exercise of a horse is most likely exertion-induced pulmonary hemorrhage (EIPH). The present accepted theory of pathogenesis is that EIPH results from subclinical lung disease. Asynchrony in movement of various groups of alveoli in the dorsal (distal) airways results in the tearing of lung parenchyma and small vessel hemorrhage. This is a common condition in racehorses but has not been identified as performance limiting. Treatment with *furosemide* has shown the best clinical response.

8. The answer is 1 [II A 1]. Signs of diaphragmatic herniation in horses can be mild to severe colic. However, borborygmi over the ventral chest is not diagnostic of diaphragmatic herniation because the diaphragm is extremely concave (when viewed from the rear) in the horse, and normal abdominal sounds can be transmitted through the chest cavity. Although decreased QRS amplitude may be found in horses with diaphragmatic hernia, the high spike T wave is not a feature. In cattle with pneumothorax, there is a drum-like resonance but a complete absence of lung sounds over the affected chest.