Clinical Classification of Pneumonias in Cattle

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More beef cattle develop and subsequently die from respiratory diseases than from all other causes. In cattle under close confinement respiratory diseases cause 75% of the illnesses and 64% of the deaths.[1] Pneumonias constitute 75% of the diagnoses of respiratory diseases.

Epizootics of respiratory disease are recognized by cattle owners who request and expect accurate diagnosis, but veterinarians experience difficulties making definitive diagnoses because of the similarity of signs in sick cattle and the many viral and bacterial pathogens recovered at necropsy.

Since a specific clinical diagnosis may be difficult to make in the field, the purpose of this paper is to simplify diagnoses by recommending a classification of pneumonias based on pathogenesis where major signs in each pneumonia are different and distinguishable.

To clarify this classification, each pneumonia will be discussed in relationship to etiology, pathogenesis, pathology, and clinical signs.

Review of Anatomy and Physiology of the Lung

To more fully comprehend the pathogenesis of pneumonias, a brief review of the anatomy and physiology of the bovine lung is indicated.

The lung is situated within the thoracic cavity on an oblique angle so that the dorsal border of the lobes slopes dorsocaudad (Figure 1). When cattle stand, the trachea slopes caudoventrad from the larynx into the thoracic inlet, then remains level to the bifurcation where the primary bronchi slope dorsocaudad toward the caudal lobes. When exudate is present in the lung, it gravitates into the cranioventral parts of the lung (Figures 2, 13 and 17).

Although the trachea bifurcates medial to the middle lobe of the right lung, the first or apical bronchus emerges from the right side of the trachea at the level of the third rib and passes laterad to enter and ventilate the cranial lobe.[2] In exudative pneumonia, therefore, auscultation of the right cranial lobe for abnormal lung sounds is necessary.

The basic structural feature of the lung is the lobule (Figure 3) which is clearly defined in the bovine organ by prominent interlobular septa and a thick pleura that is supplied by branches of the bronchial artery.[3] Each lung is composed of thousands of lobules which contain the terminal parts of the bronchial tree. Within each lobule, primary bronchioles branch into secondary bronchioles which, in turn, branch into terminal bronchioles. The lining epithelium becomes lower and the number of ciliated cells and goblet cells fewer in distal progression through these airways. Goblet cells are not present in the simple cuboidal epithelium of the terminal bronchioles. Smooth muscle also decreases progressively in the bronchiolar walls. Most of the terminal bronchioles branch into alveolar ducts.[3] Rarely, a terminal bronchiole may continue into a very short respiratory bronchiole. Alveolar ducts receive alveolar sacs and alveoli (Figure 3).

A complex of blood vessels ramifies throughout the lobule. The branches of the pulmonary artery break-up into a thick meshwork of capillaries surrounding the alveolar sacs and alveoli (Figure 3). When pasteurellas are the pathogens for pneumonia, their endotoxins can cause thromboses in the capillary plexus and in pulmonary blood and lymphatic vessels resulting in ischemic necrosis of parts of lobules or many lobules (Figures 2 and 4).

There are two sets of interconnecting lymphatic vessels in the abundant connective tissue of the bovine lung—a pleural plexus and a deep (peribronchovascular and septal) plexus.

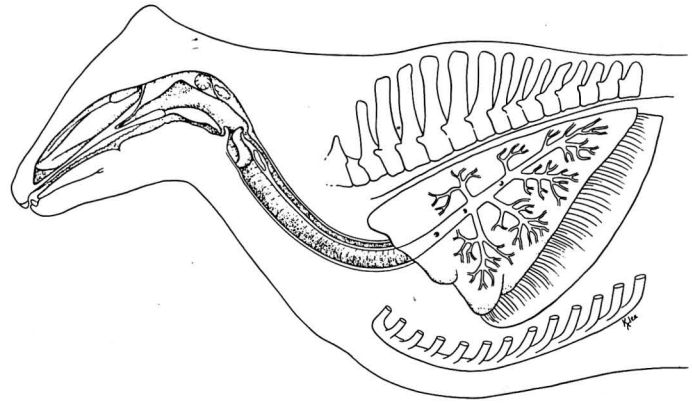

Figure 1: Black and white drawing of anatomical position of respiratory system.

The most distal branches are the juxta-alveolar lymphatic vessels in the connective tissue adjacent to peripheral alveolar walls. Lymphatic vessels do not occur within interalveolar septa.[4] Lymph drains toward tracheobronchial or caudal mediastinal lymph nodes. Septal lymphatic vessels, which carry lymph toward the pleural plexus, are particularly prominent when dilated with fibrinous exudate as a consequence of bronchial pneumonia (Figure 17).

The main function of the lung is the exchange of oxygen and carbon dioxide between air and blood across the alveolar-capillary membrane. This delicate membrane has an absorptive surface 25-30 times that of the body surface and is constantly exposed to irritants and potential pathogens. Diffusion of oxygen across the air/blood barrier is less efficient in the bovine lung than it is in the lungs of most mammals. When pneumonia is developing, the lobule is the primary target for injury; consequently, the interface for transfer of gases between the air and blood can be damaged or completely destroyed. If many lobules are damaged, the animal compensates either by breathing faster and deeper or by increasing the heart rate and force to push the blood through a damaged alveolar capillary bed. Prolonged pneumonia leads to hypoxia and right heart failure.

Other functions of the lung are compromised during inflammation of the organ. Alterations in the regulation of body temperature, acid/base balance, blood pressure (due to decrease in converting enzyme which catalyzes to activation of angiotensin I to angiotensin II), the production of histamine, prostaglandins E and F, serotonin, norepinephrine and bradykinin exert subtle to profound systemic changes.[5]

Classification of Pneumonias

Pneumonias can be classified according to: (1) morphology (lobular, lobar); (2) character of exudate (catarrhal, fibrinous, suppurative); (3) histologic changes (edema, emphysema); (4) etiology (viral, pasteurellas, aspiration); (5) radiology; and (6) disease (shipping fever, enzootic calf pneumonia). None of these classifications satisfies all requirements and each may contribute to the confusion.

A classification that will aid clinical skills is based on the route of the entry of the pathogen (pathogenesis) and includes three types of pneumonia: bronchial, interstitial, and metastatic. Ninety percent or more of the pneumonias in cattle fit into this classification.

In a recent survey into the causes of death of feedlot yearlings,[6] over 83% of the pneumonias were bronchial, 12% interstitial, and 4% metastatic.

A. **Bronchial Pneumonia.** Shipping fever is an example of bronchial pneumonia. Although the exact etiology and pathogenesis have not been fully established, some facts about shipping fever are presented below.

1. **Etiology of Bronchial Pneumonia.** Shipping fever is caused by a sequential interaction of stressors, viruses, and bacteria. Stressors include weaning, transportation, processing, and exposure to changeable weather. Viral agents include infectious bovine rhinotracheitis, para-influenza-3 viruses and possibly others. Pathogenic agents are various *Pasteurella spp., Mycoplasma spp.,* and *Hemophilus spp.*

2. **Pathogenesis of Bronchial Pneumonia.** Jensen[2] and others proposed the following hypothesis for the pathogenesis of shipping fever: (1) Chilling of the mucous membranes of the head and/or infections in combination with viruses cause tissue changes that predispose the nasopharynx to colonization by pasteurellas and the development of rhinitis. In early inflammation, ciliary action is increased, but later it decreases. (2) Pathogens, especially pasteurellas and possibly mycoplasmas, transfer from the nasopharynx to the lungs: (a) by gravitational drainage of infected nasal secretions along the tracheal floor into ventral bronchi; and; (b) by inhalation of infective droplets. Because of higher ventilation and gravitational settling, both fluids and droplets inoculate the cranial (apical), middle (cardiac), and lower parts of the caudal (diaphragmatic) lobes and compromise the function of the lobules (Figure 2). (3) *Pasteurella spp.* and possibly *Mycoplasma spp.* colonize the ventral parts of the lungs and produce pneumonia. (4) *Pasteurella* endotoxin causes clotting and thrombosis in lymphatic vessels, capillaries, and veins. The ischemia results in necrosis (Figures 2 and 4). (5) Necrotic tissue may sequester an abscess (Figure 4). (6) Death results from hypoxemia, endotoxemia, pulmonary necrosis, shock, and heart failure.

3. **Pathology of Bronchial Pneumonia.** Figure 5 illustrates the sequential pathogenesis of lobular necrosis in the same section of lung. Figure 4-1 is a transverse section of a middle lobe with beginning bronchial pneumonia, showing exudate consisting of plasma protein rich in fibrin which has gravitated into the ventral respiratory bronchioles and alveolar sacs resulting in consolidation of the lobules. Lobules are in different stages of pneumonia from hyperemia (stage I) to red and grey hepatization (stage II and III). Figure 4-2 is a more advanced bronchial pneumonia with all ventral lobules consolidated. The interlobular lymphatic vessels are dilatated and contain fibrin clots, a result of *Pasteurella* endotoxins. In Figure 4-3 the pneumonia is more advanced with early infarction caused by a thrombus in a large pulmonary vein. The resulting necrotic tissue involves many lobules, and the affected tissues are seen as pale brown surrounded by a grey line (leukocytes and bacteria). Figure 4-4 demonstrates coagulative necrosis and early sequestration. Sections 4-5 and 4-6 are more advanced. They depict fibrosis of interlobular tissue with connective tissue forming capsules around foci of necrosis and abscesses.

4. **Clinical Signs of Bronchial Pneumonia.** From the onset of shipping fever, before pneumonia is present and until recovery or death of the animal, depression is a major

clinical sign. Depression, characterized by lowered head (Figure 5) and ears, separation from the other animals and a certain reluctance to move, is probably a result of the *Pasteurella* endotoxins. Other signs include anorexia, pyrexia, shallow abdominal breathing, and suppressed cough due to pleuritis. In more advanced cases, there is dehydration, dry muzzle, and occasional profuse diarrhea. Upon auscultation of the lung, referred bronchial sounds are heard. Discharge from the nose is minimal. Death results from hypoxemia, endotoxemia, pulmonary necrosis, shock, and heart failure. Complications of bronchial pneumonia include multilobular and even lobar necrosis, abscessation of lobules, adhesions, bronchiectasis, cor pulmonale and a permanently unthrifty animal.

B. **Interstitial Pneumonia.** Interstitial pneumonia is less common than bronchial pneumonia. It includes several distinct entities, and confusion arises from terminology. In feedlot cattle intestitial pneumonia has been referred to as atypical intestitial pneumonia and pulmonary adenomatosis; in pastured cattle as acute pulmonary emphysema or fog fever; in housed cattle as bovine farmer's lung; and in recently weaned calves as pulmonary emphysema. Acute respiratory distress syndrome (ARDS) has been recommended as a term inclusive for this group of pneumonias.[7]

1. **Etiology.** All causes of interstitial pneumonia (ARDS) are not known, but two conjectured causes—dietary tryptophan and hyper-sensitivity—are gaining acceptance. Recently a bovine respiratory syncytial (BRS) virus has been identified as a possible cause.[5]

Since several distinct entities fit into this classification, they will be discussed separately under their common names.

a. **Acute bovine pulmonary emphysema** (ABPE), fog fever. ABPE occurs principally in adult cattle shortly after a change from dry to lush pasture in the autumn. The disease is caused by the ingestion of grass containing the amino acid, L-tryptophan, which is converted by the ruminal microorganisms to the highly toxic compound, 3-methyl-indole[3] (Figure 6).

b. **Atypical interstitial pneumonia** (pulmonary adenomatosis, farmer's lung). This group of syndromes performs as an allergic respiratory disease which develops after exposure to moldy hay or silage containing the spores and metabolic products of *Mycopolyspori faeni* and other thermophilic actinomycetes. Feedlot cattle may become sensitized to the fungus ingested from the feedbunks (Figure 7). Later they become re-exposed to the fungus and then develop an allergic reaction.[7]

c. **Pulmonary emphysema of recently weaned calves.** The etiology of this syndrome may be multiple and complex with features similar to bronchopneumonia.[6] One possible cause for this is the BRS virus. The cause may also be a hypersensitivity reaction due to preconditioning.

2. **Pathology of Interstitial Pneumonia** (ARDS). Interstitial pneumonia usually involves both lungs with combinations of the following changes: alveolar emphysema, interstitial edema, hyperplasia of alveolar epithelium, and hyaline membrane development. The enlarged, firm, red-purple lungs fail to collapse. Affected lobules and lobes are red-purple rather than the salmon-pink of normal lobules. The caudal lobes are more commonly affected than other lobes. A small amount of fluid exudes from the cut surface of the lung, and the bronchial mucous membranes are congested and sometimes hemorrhagic. Neither suppuration nor pleuritis occurs (Figure 10).

3. **Pathogenesis of ARDS.** Since several specific syndromes are included in this classification, the pathogenesis differs. For more details refer to recent publications.[7,8,9,10]

4. **Clinical Signs of Interstitial Pneumonia** (ARDS). The term "acute respiratory distress" describes the general attitude of an animal suffering from interstitial pneumonia. Affected cattle stand with heads extended, nostrils dilated, mouths open, and tongues protruded. Respirations are accelerated and labored; the pulse is rapid and weak; and the temperature is normal or slightly elevated. Any exertion may cause death (Figures 8 and 9).

C. **Metastatic Pneumonia with Hemoptysis.**

1. **Causes.** Causes include: phlebitis, endocarditis, and thrombosis associated with diseases such as metritis and mastritis. In feedlot cattle, caudal vena caval thrombosis secondary to the rumenitis-liver abscess complex is the most common cause.[11]

2. **Pathogenesis - Caudal Vena Caval Trombosis** (Figures 11 and 12). Pathogenesis starts with a rumenitis associated with a sudden change in feed from roughage to concentrate. *Fusabaterium necrophorum,* a rumen inhabitant, gains entrance through the portal vein and showers the liver with abscess-forming bacteria (Figure 11). If an abscess develops adjacent to the caudal vena cava or a large hepatic vein, vasculitis and, subsequently, a caval thrombus develop. Septic emboli detach from the thrombus and lodge in and occlude pulmonary vessels, weakening their walls and leading to saccular aneurysms. Blood pressure ruptures the saccule and opens adjacent bronchi (Figure 16). Extavasated blood dissects the tunica adventitia, forms hematomas, pours into a bronchus, and is expelled from the larynx. Death is a result of exsanguination and pulmonary incapacitation.

3. **Pathology.** The erosion of hepatic abscesses into the caudal vena cava often result in septic pulmonary embolization.[12] Affected lungs contain regions of pneumonia and multiple abscesses. Lungs with ruptured aneurysms are large, firm, and fail to collapse. One or more aneurysms with clotted blood surround the ruptured aneurysm and fill bronchi, bronchioles and alveolar sacs with blood (Figure 12 and 16).

4. **Clinical Signs of Metastatic Pneumonia and Hemoptysis.** Since signs of metastatic pneumonia may resemble either bronchial pneumonia or acute respiratory distress, it may be difficult to diagnose. Sudden elevation of temperature followed by rapid, shallow breathing may

suggest showering of the lung with septic emboli, especially if the history indicates associated metritis, mastitis, or similar problems. Hemoptysis (bright foamy blood around nose and mouth), respiratory distress with head and neck extended, groaning on expiration, pale membranes, generalized weakness, and pulmonary rales over a large area during auscultation are signs suggestive of a ruptured pulmonary aneurysm associated with metastatic pneumonia (Figure 12).

Summary

Clients expect and whenever possible deserve a diagnosis of their animal's disease. However, in cases of respiratory disease, diagnosis may be extremely difficult. If pneumonia is present, a careful evaluation of the history and major clinical signs may allow the veterinarian to classify pneumonia into one of three different types: bronchial, interstitial, or metastatic. With this classification, the diagnosis is specific, the prognosis accurate, and the treatment regime likely to succeed.

Bronchial pneumonia, the most common type, is frequently a result of viral and bacterial pathogens inhaled into the lung where they colonize the mucous membrane and produce exudate which fills the incapacitated lobules (Figures 13 and 17). Endotoxins, produced by bacteria such as *Pasteurella spp,* cause vasculitis and thromboses of lymphatic vessels, capillaries and veins resulting in lobular necrosis and abscesses (Figure 4). Major signs of bronchial pneumonia include depression characterized by lowered head and ears, pyrexia, coughing, and shallow breathing (Figures 5 and 14). Beginning cases respond to therapy.

Interstitial pneumonia is often due to hypersensitivity reactions, dietary causes, or viruses. The reaction in the lung causes combinations of edema, emphysema, hyaline membranes, and hyperplasia of alveolar septal cells, all of which incapacitate the lobule (Figure 15). This reaction is more acute than in bronchial pneumonia, and is characterized clinically by acute respiratory distress with extended head and open mouth breathing (Figure 14). Response to treatment is discouraging.

Metastatic pneumonia, frequent sequel to caudal vena caval thrombosis, may predispose the lung to pulmonary embolic aneurysms. Rupture of an aneurysm results in hemoptysis and fatal exsanguination. Unless hemoptysis is present, metastatic pneumonia may be difficult to diagnose. Treatment, if started early, may prevent the aneurysm.

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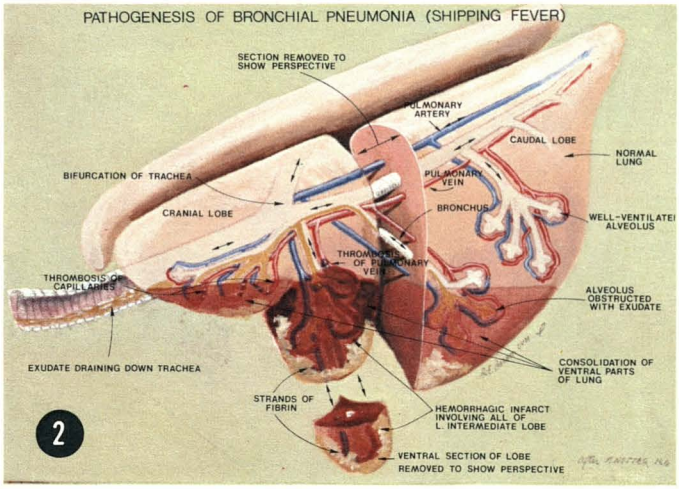
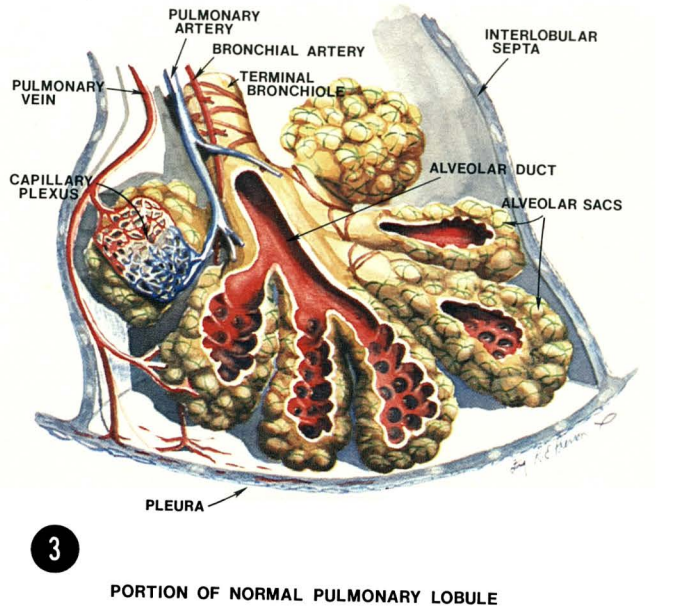
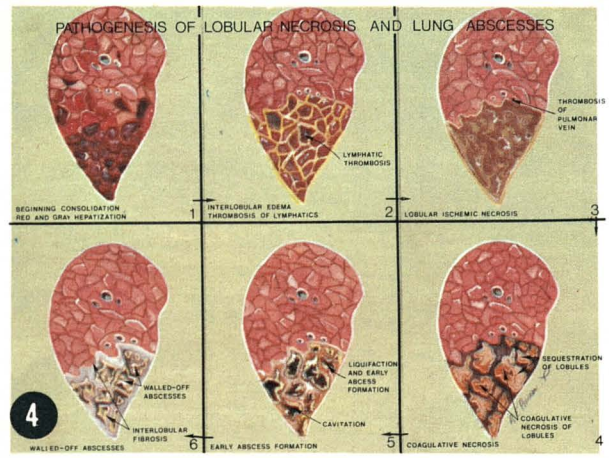
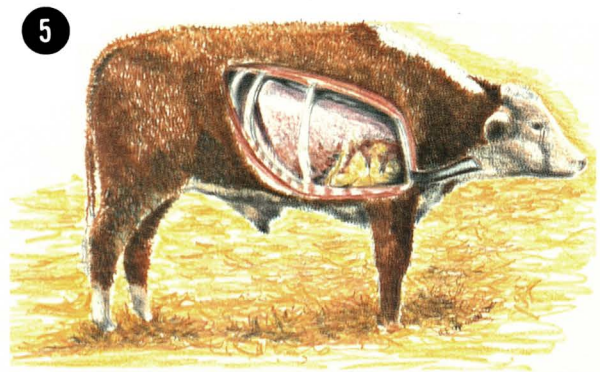
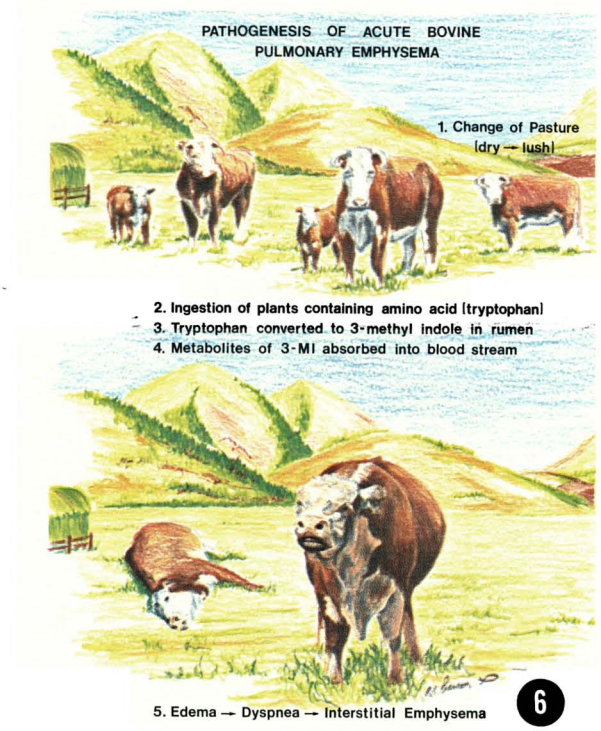




PORTION OF NORMAL PULMONARY LOBULE







CAUSES & PATHOGENESIS OF ACUTE RESPIRATORY DISTRESS SYNDROME

1. HYPERSENSITIVITY REACTION
   a. moldy hay - spores of micropolyspora faeni
   b. preconditioning of calves

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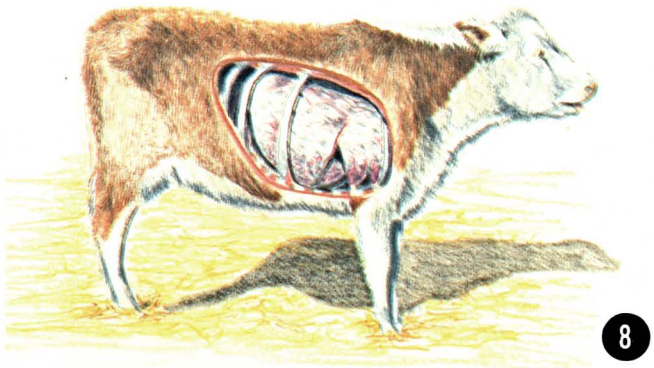
2. VIRUS
   a. bovine respiratory syncytial virus (BRS)

ACUTE ALVEOLAR EMPHYSEMA OF CALVES



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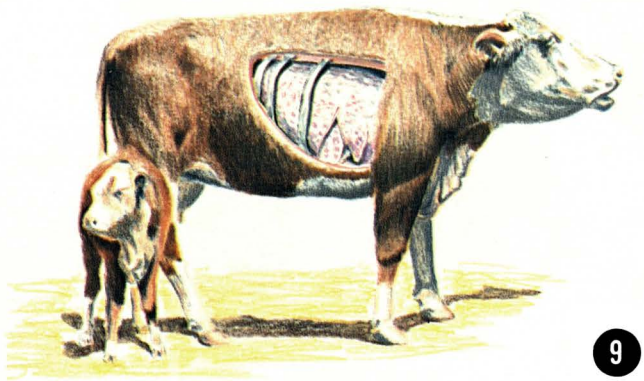
ACUTE BOVINE PULMONARY EMPHYSEMA



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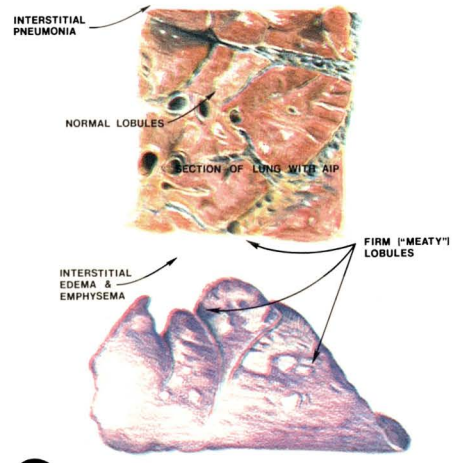
ATYPICAL INTERSTIAL PNEUMONIA (AIP)



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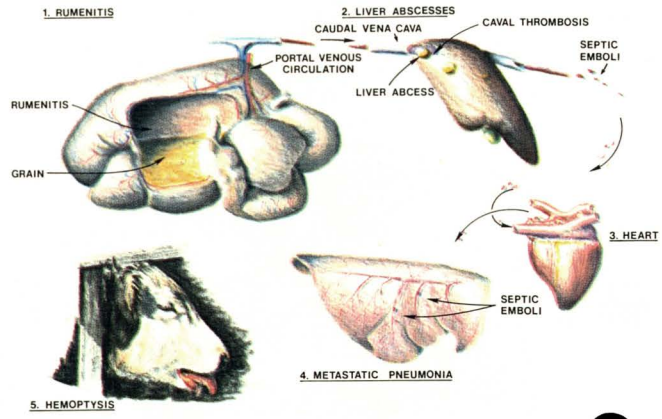

PATHOGENESIS OF SEPTIC PULMONARY EMBOLISM (METASTATIC PNEUMONIA)

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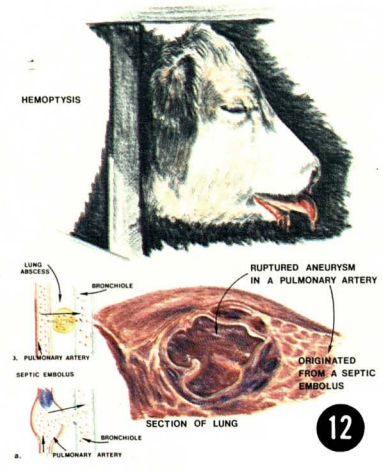
PATHOGENESIS OF HEMOPTYSIS



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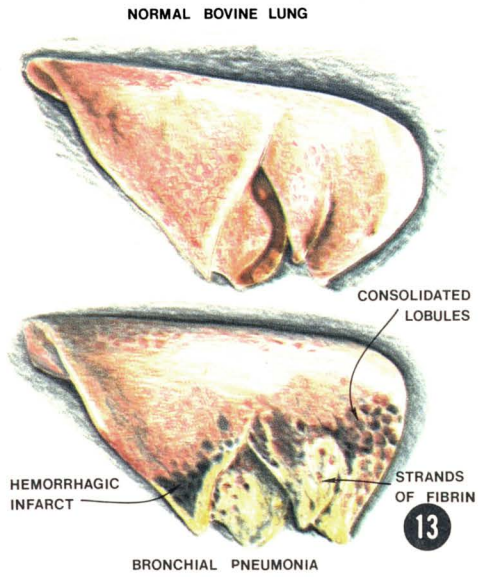


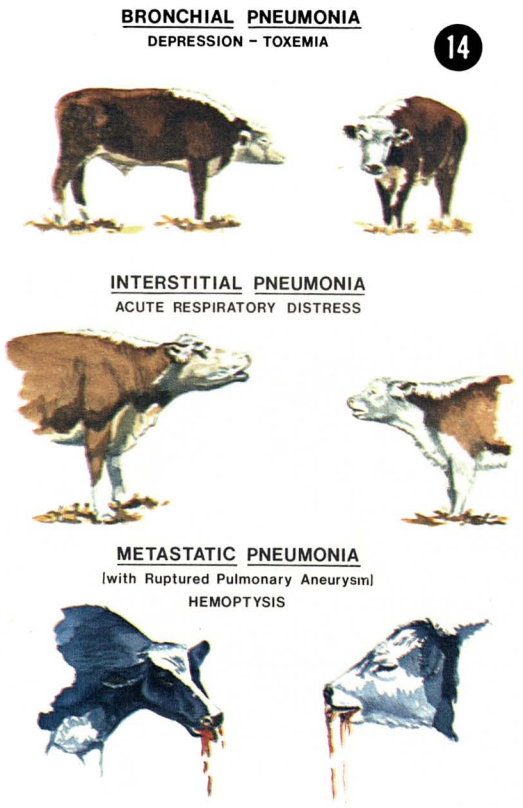
Figure 13: Normal bovine lung and bovine lung with bronchial pneumonia.

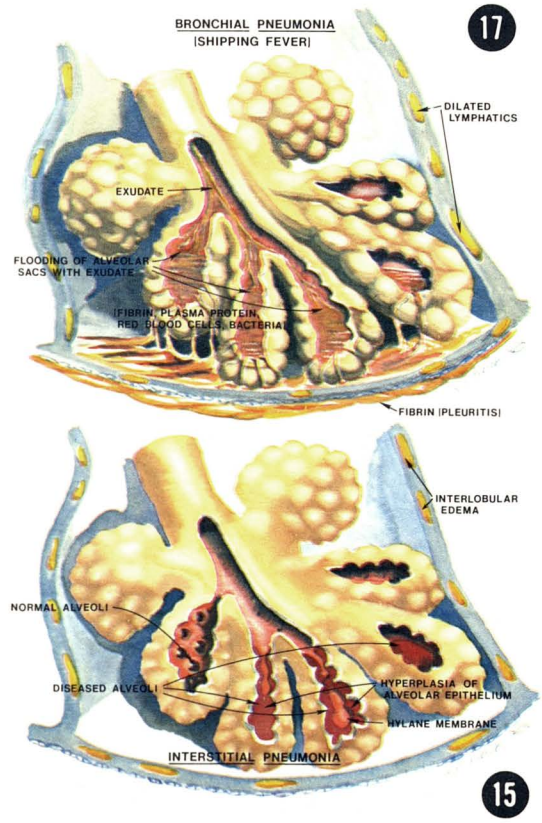
Figure 14: Clinical attitude of bovine with bronchial and interstitial pneumonia.

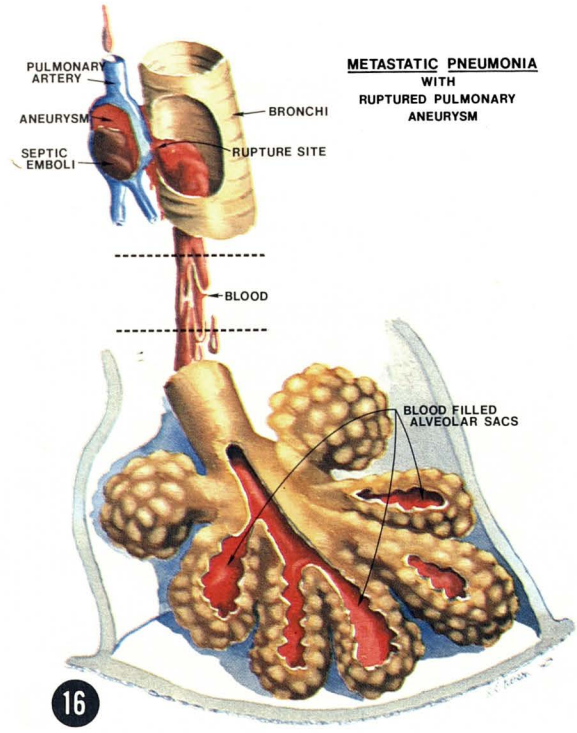
Figure 15: Schematic drawing of lobule with interstitial pneumonia.

Figure 16: Schematic drawing of lobule with blood filled alveoli from a ruptured aneurysm.

Figure 17: Schematic drawing of lobule showing flooding of alveoli with exudate with bronchial pneumonia.







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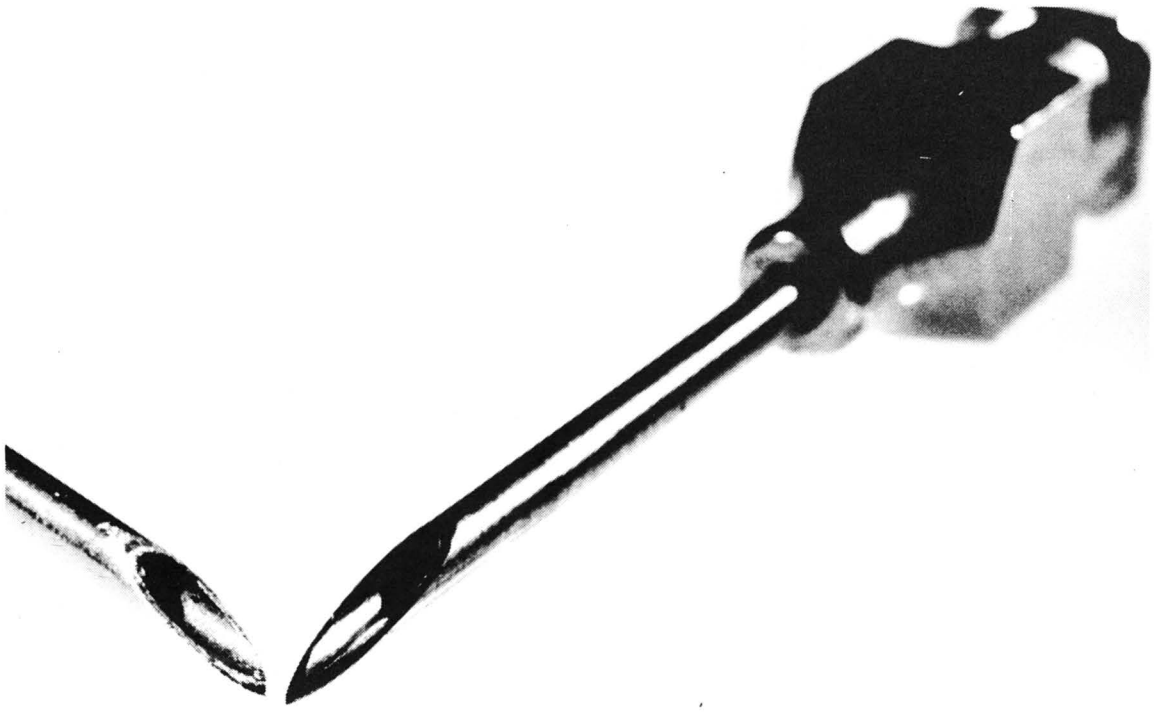

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