Pathology of Respiratory System

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Objectives

- At the end of the session students will able to explain and determine
  - General consideration of respiratory system.
  - Pathology of nasal cavity, larynx, trachea and bronchi.
  - Deferent the anomalies of the respiratory system

Pre test for respiratory system

- Discuss in group the important anatomic features of the respiratory apparatus as well as their function(s) in respiration, beginning with the nasal mucosa and ending with the alveolar walls?

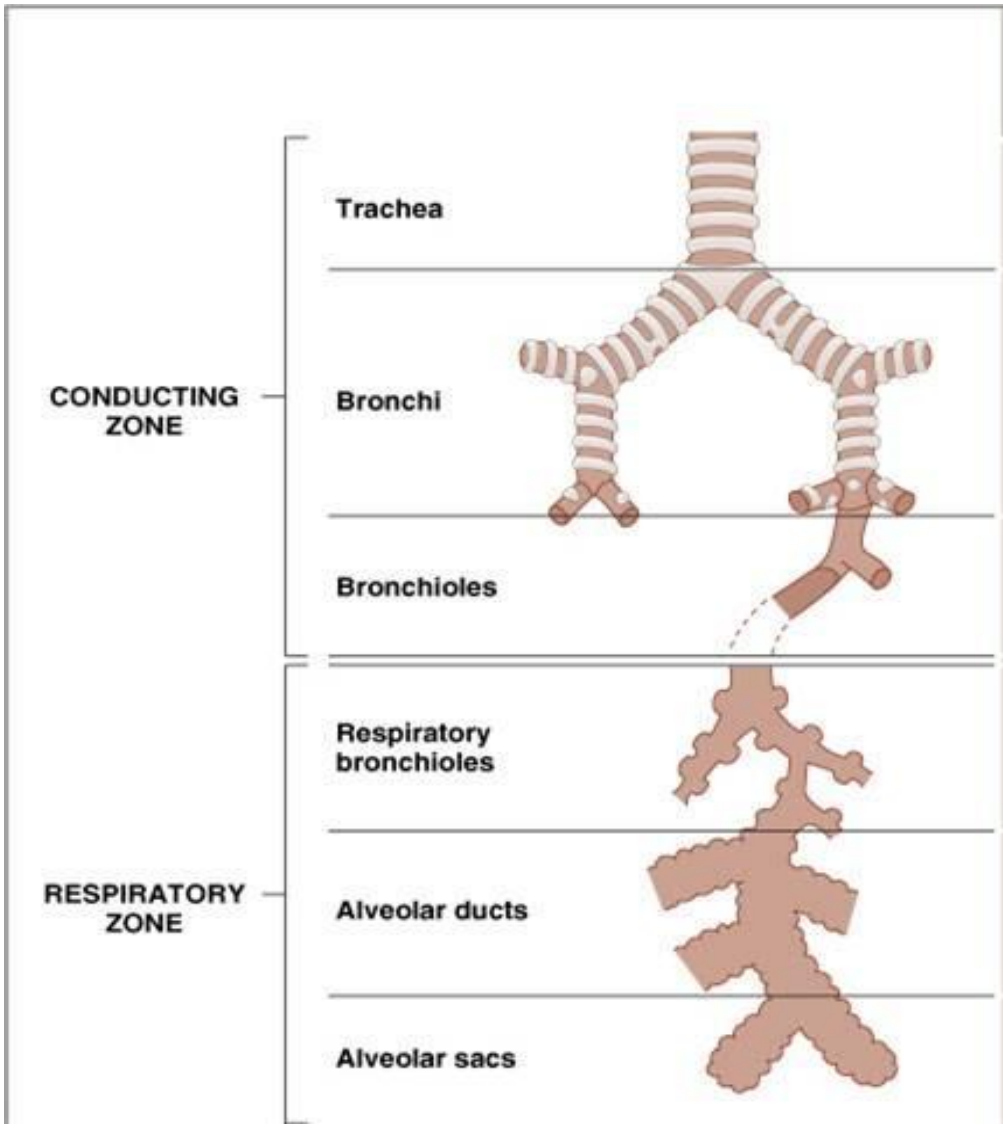
- Animal's or human's life is highly dependent on oxygen.
- Oxygen is vital for normal oxidative metabolism of body cells.
- Carbon dioxide is major end product of cellular respiration, which if not removed, accumulate & poisons cells.
- The respiratory system is well equipped system to mediate exchange of these gases between the body & the atmosphere.

- The respiratory tract is arbitrarily divided into three continuous systems

1. **Conducting system:** The conducting system includes the nasal cavity, sinuses, larynx, trachea and bronchi.
   - The mucosa of the conducting system is lined primarily by ciliated epithelium and goblet cells.

2. **Transitional system:** The transitional system is formed by the bronchioles that are lined by a specialized mucosa containing several types of ciliated and secretory cells such as Clara cells.
   - Unlike the conducting system, the normal bronchiolar mucosa contains no goblet cells.

3. **Exchange system:** This system is composed of the alveoli that are lined externally by epithelial cells called pneumonocytes.
   - The type I (membranous) pneumonocytes are thin cells and together with the capillary endothelium and basement membrane constitute the air-blood barrier.
   - Type II pneumonocytes are cuboidal and produce surfactant.



	Number	Cilia	Smooth Muscle	Cartilage
Trachea	1	Yes	Yes	Yes
Bronchi	2 4 8	Yes	Yes	Patchy
Bronchioles	–	Yes	Yes	No
Respiratory bronchioles	–	Some	Some	No
Alveolar ducts	–	No	Some	No
Alveolar sacs	6×10^8	No	No	No

Pathology of Nasal Cavities and Sinuses

- Inflammatory diseases are the most common disorders affecting the nose and paranasal sinuses.

1. Rhinitis

- Rhinitis is inflammation of the nasal cavities.
- The condition may be acute or chronic, and the exudate may be serous, catarrhal, purulent, pseudome membranous.

- The cause of rhinitis is based on the interplay of viruses, bacteria, and allergens.
- Acute rhinitis is usually initiated by viruses which commonly evoke a profuse catarrhal discharge. Also, allergens may initiate an acute rhinitis, but they are important only in cattle.

- **Pathogenesis of viral rhinitis / tracheitis / bronchitis**
- Virus in air or saliva → virus replication in epithelial cells → cell degeneration → loss cellular attachment → cell exfoliation → ulceration →exudation (fluid and cellular) → cell mitosis →repair.
  - Complete repair occurs in approximately 14 days.

- **Grossly:** During the initial acute stage of rhinitis, the nasal mucosa is swollen, edematous, and pale grey to red (depending on the degree of hyperemia)**.**

- The mucosal surfaces are covered by a thin watery to mucoid discharge which is relatively clear.

- When such acute reactions persist for a few days, bacterial infection modifies the character of the discharge and produces a mucopurulent or suppurative exudate.

- **Microscopically**, edema is the most prominent feature; the lamina propria is sparsely infiltrated by inflammatory cells.

- Swelling of the mucous membrane may cause mild respiratory discomfort.

- **Chronic rhinitis** is characterized by the presence of fibrous connective tissue scarring; the epithelium becomes atrophic, foci of squamous metaplasia may develop and there is progressive atrophy of the mucous secreting glands.

2. Amyloidosis:

- Amyloid is sometimes deposited in the nasal submucosa of horses.
- The deposition is not part of a generalized amyloidosis and the cause is unknown.
- The amyloid is deposited in the anterior portion of the nasal cavity and stenosis can be severe enough to cause clinical signs of nasal obstruction.

3. Epistaxis:

- The term epistaxis refers to nasal hemorrhage.
- Hemorrhage from the nose may be due to traumatic, Bacterial (Anthrax), Parasites (*Eimeria canis in dogs; Oestrous ovis in sheep*).
- Blood-stained foam emitted from the nose of carcasses, especially sheep is indicative of severe pulmonary congestion and edema with seepage of blood from the congested alveolar walls.

4. Nasal Polyps:

- Nasal polyps are inflammatory new growths which resemble true neoplasms.
- Polyps are non neoplastic masses that resemble tumours.
- They represent focal accumulations of edematous fluid accompanied by hyperplasia of submucosa connective tissue and inflammatory cells **(**neutrophils, lymphocytes, plasma cells).
- Older polyps may contain considerable fibrous connective tissue.

5. Nasal Parasites:

- The larvae of a number of flies of the **family *Oestridae*** are parasites of the nasal cavities of domestic animals.
- However, ***Oestrus ovis*** **(nasal bot of sheep)** is most commonly encountered.
- The adult flies are relatively harmless from a pathologic point of view; however, they do cause considerable annoyance to their host.

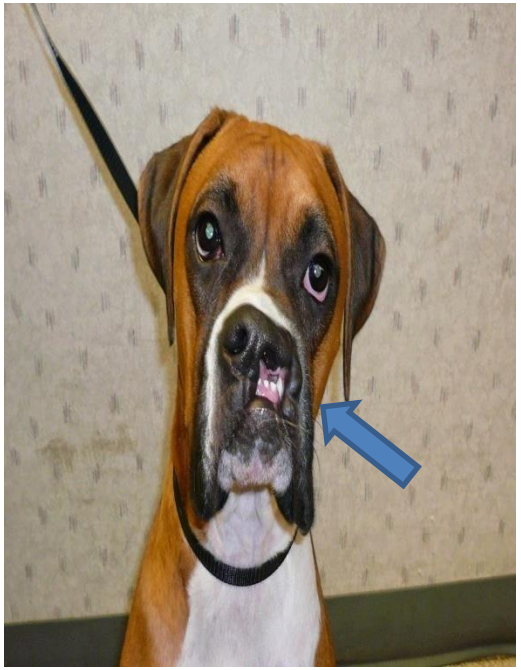
6. Nasal Neoplasms:

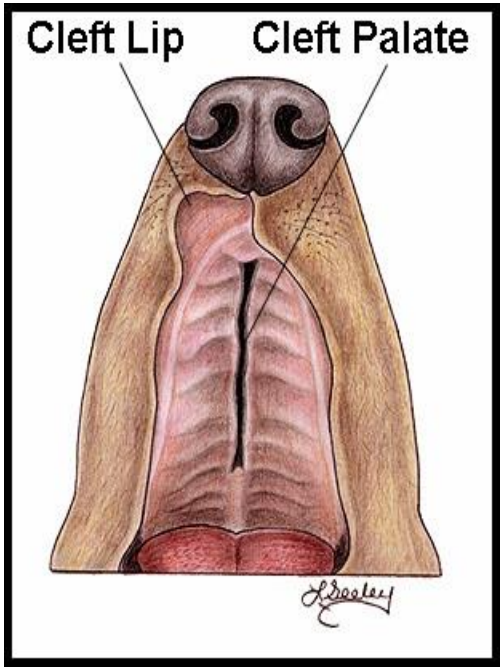
- Neoplasms of the nasal cavity are uncommon; however, a wide histogenic variety may be found **(benign and malignant).**
- **Fibrosarcomas** are the most common of the mesenchymal neoplasms and **squamous cell carcinomas** are the predominate neoplasm of epithelial cell origin.

Benign	Malignant
Osteoma	Osteosarcoma
Chondroma	Chondrosarcoma
Myxoma	Myxosarcoma
Fibroma	-
Angioma	-
-	Adenocarcinoma
-	Squamous cell carcinoma

7. Congenital Anomalies:

- The most common congenital anomalies of the nose and pharynx are cleft lip and palate





8. Sinusitis:

- Sinusitis is Inflammation of the paranasal sinuses.
- Sinusitis is common in sheep as a response to the larvae of *Oestrus ovis*. And it is most significant in the horse, because this species has the largest and most complex sinus structure, coupled with the poorest drainage.
- In addition, in horses, periodontitis often extends into the sinuses.

Specific Diseases of the Nasal Cavities and Related Structures

✓ Strangles of Horses

- Strangles is an acute contagious disease of horses caused by ***Streptococcus equi*** and characterized by **inflammation of the upper respiratory tract** and **abscessation in the regional lymph nodes**.

- Clinicaly indicated by fever, a slight cough, and bilateral nasal discharge **(which changes from serous to catarrhal and then purulent).**

- In the nasal cavities, a purulent rhinitis develops and large amounts of creamy yellow pus collect in the folds of turbinates. The nasal mucosa is edematous and hyperemic and occasionally small ulcers may develop.

✓ **Necrotic Rhinitis of Swine**

- **("Bull Nose," Rhinohyperplasia)** is characterized by irregular proliferation of fibrous connective tissue and bone over the snout region.

- Apparently, a variety of infectious agents can initiate the condition. Such agents usually enter through defects in the gums.

✓ Glanders

- Glanders is a disease of horses caused by *Burkholderia mallei*.
- The bacteria enter through the nasal cavity and cause big caseo-necrotizing tracts through the sinuses, within the subcutis, and in the lung.
- When the lesions occur in the subcutis of the skin, the disease is called “farcy.”

Pharynx And Guttural Pouches

- The pharynx is common to both the respiratory and digestive systems and shares the misfortunes of both.
- Guttural pouches are diverticula of the eustachian tubes and they are found only in the horse.
- Occasionally, the guttural pouches become infected by extension of infections from the eustachian tubes. Such infections are entirely comparable with those of the paranasal sinuses.

Larynx and Trachea

Laryngitis/Tracheitis:

- Inflammation is the most common and important disorder affecting the larynx and trachea.
- Because of their location, these structures frequently become inflamed as a part of inflammatory diseases of either the upper or lower parts of the respiratory tract.
- Thus, laryngitis and, to a lesser extent, tracheitis is expected to accompany rhinitis; whereas tracheitis and, to a lesser extent, laryngitis, is expected to accompany acute pneumonias.

- Inflammations of the air passages usually involve all levels and they are characterized by coughing, noisy inspiration and some degree of inspiratory embarrassment.

- Diseases in which *laryngitis and tracheitis* are a prominent feature include *infectious bovine rhinotracheitis*, *calf diphtheria*, *equine viral rhinopneumonitis*, *strangles*, *swine influenza*, *feline infectious respiratory diseases*, etc.

- Grossly: Mucosa of larynx is swollen. The mucosa is haemorrhagic and dry at first, later becomes coated with mucus or mucopurulent discharge.
- Tracheal mucosa is congested and the lumen contains mucus or blood tinged mucus containing red worms.
- Microscopically: Mucosa is covered with exudate - mucus, blood or necrotic material Lamina propria is infiltrated with leucocytes.
- Lesions may vary from catarrhal to chronic granulomatous involvement, depending on the duration and severity of infection.

Hemorrhages:

- Laryngeal and tracheal hemorrhages may occur as a result of infection, trauma, violent coughing, etc.
- Laryngeal hemorrhages occur in many septicemic diseases **(hog cholera, salmonellosis, etc.).**
- In the trachea, agonal hemorrhages are often times associated with severe dyspnea and hypoxia.

Edema:

- Edema of the larynx is usually inflammatory and part of the picture of acute respiratory infection.
- laryngeal edema may be associated with allergic reactions, inhalation of irritants, insertion of tracheal tubes, etc.
- In the tracheal lumen, foamy fluid is commonly observed. Such foamy fluid is associated with severe pulmonary edema. The foams are actually formed in the alveoli.

Reading assignments

- **Specific Diseases Involving The Larynx And Trachea**
  - **Calf Diphtheria**
  - **Infectious Bovine Rhinotracheitis**
  - **Feline Upper Respiratory Disease**
  - **Feline Viral Rhinotracheitis**
  - **Feline Pneumonitis**
  - **Infectious Laryngotracheitis**
  - **Syngamus Trachea Infection**

Bronchi and Bronchioles

- The bronchi and bronchioles have a hybrid nature. Their function is conducting air.
- However, they are embedded within the lung parenchyma, which is predominantly focused on gaseous exchange.

1. Bronchitis and Bronchiolitis

- The consequences of inflammation of the large bronchi may be quite different from those of inflammation of smaller bronchi and bronchioles.

- The larger bronchi, for example, lie in interstitial tissue outside of the lung lobules. The epithelium is pseudostratified and well supplied with mucous-secreting and ciliated cell and the peribronchial connective tissue is abundant. The lumen is large enough to remain patent even in the presence of copious exudate and such exudate can be expelled by an effective cough reflex.

- By contrast, the small bronchi and bronchioles lie within the lung parenchyma. The epithelium is simple and the ciliated and mucous-secreting cells decrease in number and disappear from the smallest branches.

- The walls are thin and the small lumens are easily occluded by exudate which may be too far distal for the cough reflex to be particularly effective.

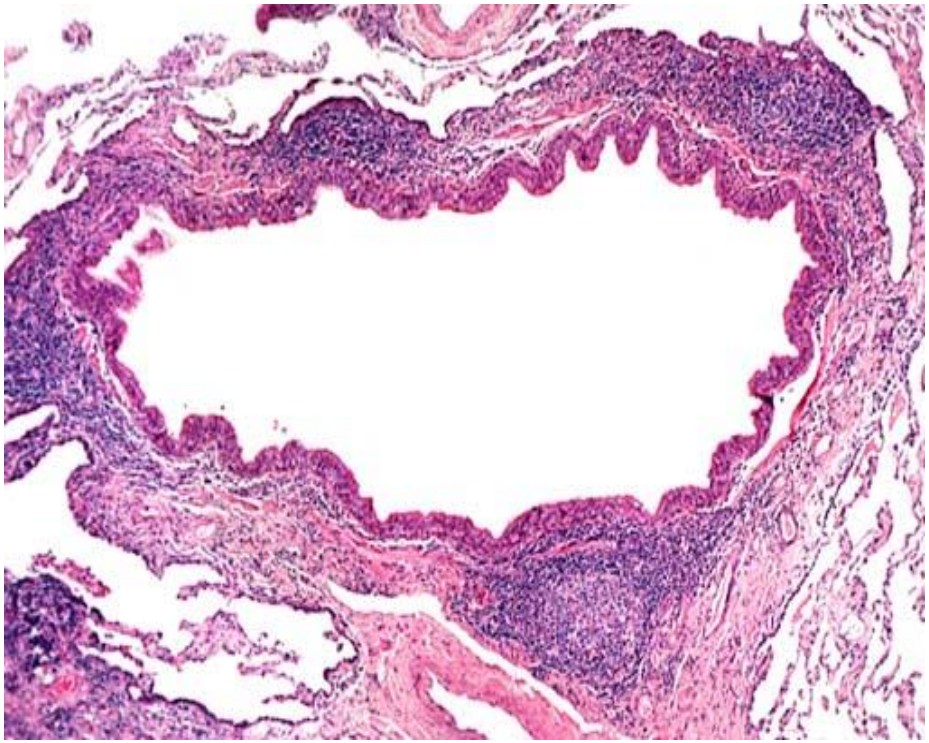
- Therefore, inflammation of larger bronchi may or may not have significant consequences for the lung, but inflammation of small bronchi and bronchioles almost inevitably leads to occlusion with extension of inflammation to the lung parenchyma.

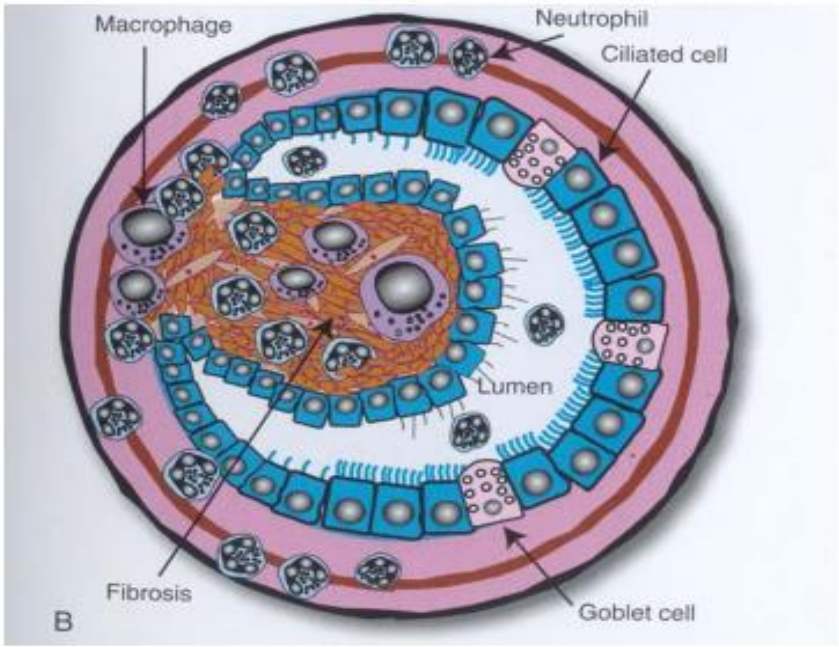
- The causes of bronchitis/bronchiolitis are chemical agents, bacteria, viruses, helminths.

- The condition may be acute or chronic, focal or generalized and characterized by a catarrhal, purulent, fibrinous or pseudomembranous exudation

- **Grossly,** bronchitis/bronchiolitis are usually accompanied by hyperemia of the mucosa and exudation of materials into the lumen.
- If fibrin is present, this mass of material may solidify and form a **"cast"** within the lumen.

- **Microscopically**, Inflammation of bronchi bronchioles should be diagnosed only if inflammatory cells are found in their wall. If the inflammatory cells are in the lumen but not in the wall, it is not a bronchitis or bronchiolitis





Chronic follicular bronchiolitis

2. Stenosis of Bronchi/Bronchioles

- Stenosis is narrowing of the lumen**.** It can be caused inflammation, parasites, Aspiration of foreign bodies, peribronchial pressure, Accumulation of exudates and infiltration etc..

- There may be partial or complete closure of the bronchial lumen and this results increased resistance in the airways.

✓ If there is partial obstruction, greater effort is required to force air into the alveoli on inspiration. During this difficult and slow passage of air, the alveolar walls are momentarily stretched.

- If this process is repeated over a prolonged period, this continued over-stretching of the alveolar walls eventually causes them to break resulting in a condition referred to as emphysema.

- Then upon expiration, not all of the air is expelled. This results in incomplete exchange of gases.
  - Over-inflated alveoli without rupture is also referred to as emphysema

- ✓ If complete stenosis/obstruction of the smaller bronchioles occurs, at first the air is trapped in the alveoli because it cannot escape during expiration.

- Eventually, the body will absorb the air. As air is absorbed from the alveoli, they collapse and this area of the lung is no longer functional

  - This process leads to partial stasis of blood in the alveolar capillaries, and mild edema
  - This is a choice place for infection to start if bacteria gain entrance to this part of the lung.

3. Bronchiectasis

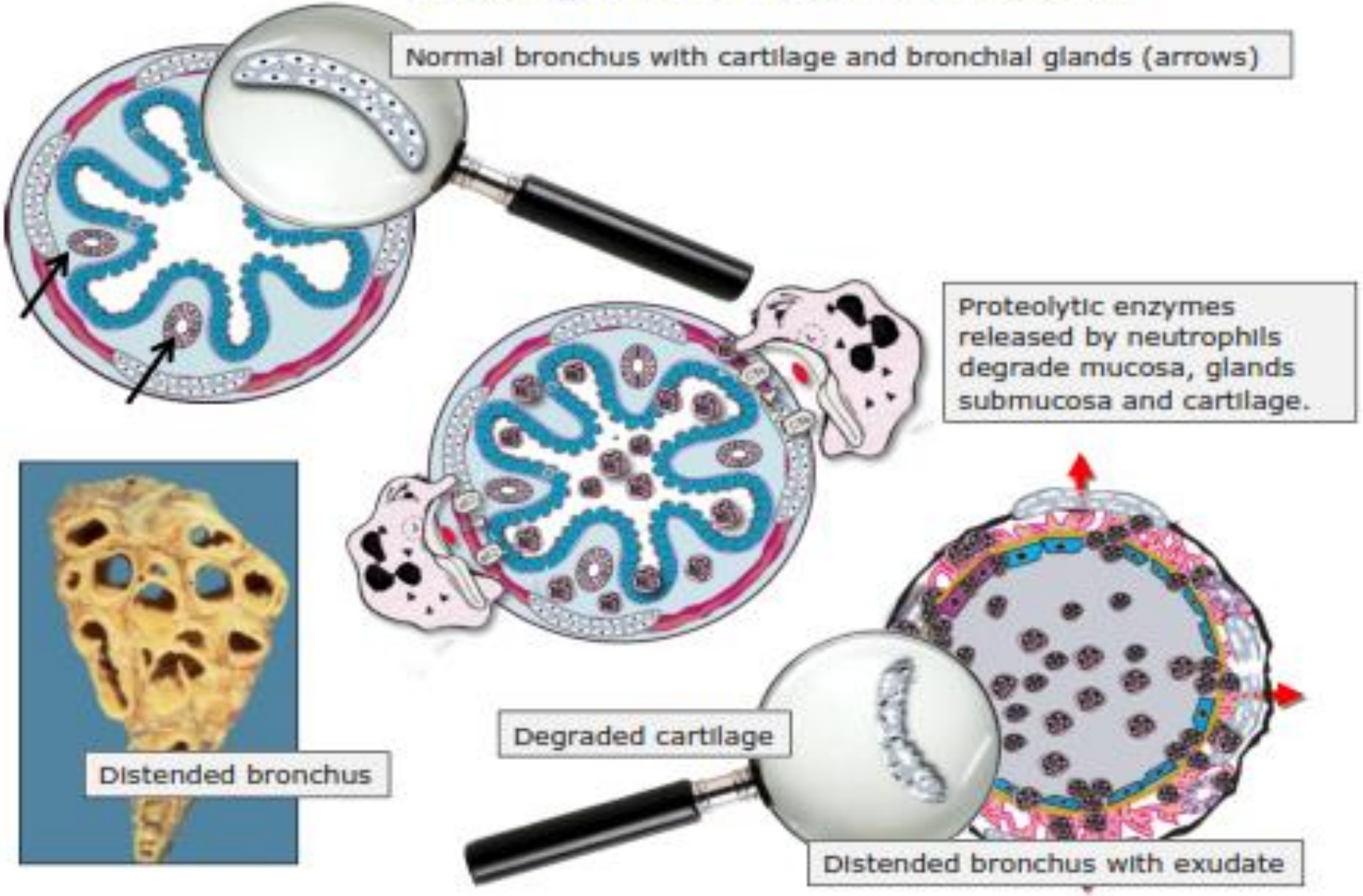
- Bronchiectasis refers to dilatation of bronchi and bronchioles.
- Mainy due to chronic inflammation (chronic bronchitis) there is repeated coughing.
- Repeated coughing results in continuous over-stretching of the alveolar walls as well as over-dilatation of the bronchioles as they stretch to accommodate the cough.

- As this over-distention and over-stretching is repeated over long periods of time, the smooth muscle in the wall of these bronchioles loses its tone and undergoes degeneration.

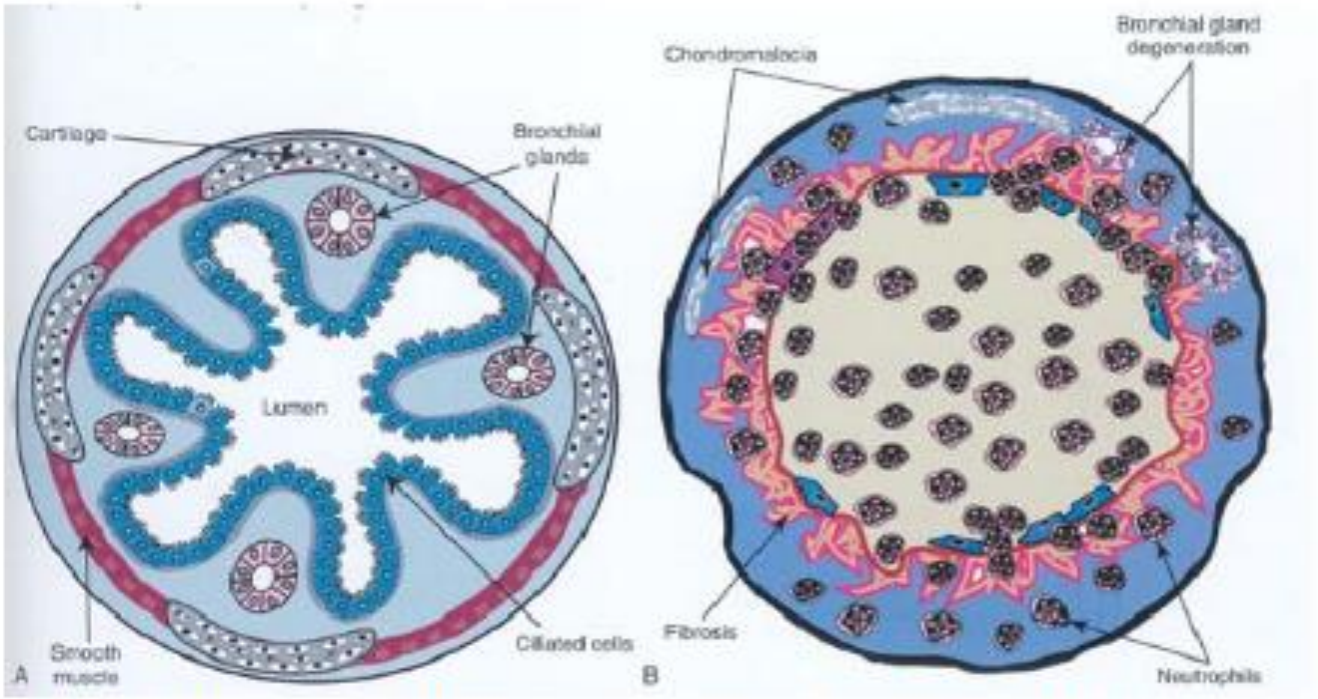
- Eventually, the lumens remain open instead of contracting as they do when they are relaxed and not being used.

- The mucosa gradually becomes thinner and the walls are gradually destroyed and replaced by fibrous connective tissue. This connective tissue helps to tie the walls of these bronchioles to the surrounding tissue, which in turn aids in keeping them over-dilated.

Pathogenesis of Bronchiectasis



- Grossly: Cylindrical form of bronchiectasis is more common in cattle and Saccular form is less common
- Microscopically: Destruction and disappearance of the elastic tissue, lamina propria infiltrated with mononuclear cells

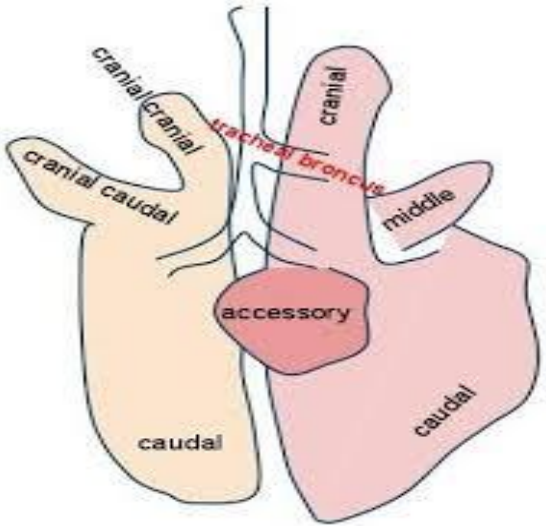


Normal bronchiole

Bronchiectasis - held open permanently

The Lungs

- There are notable differences in lung morphology among animal species; *i.e., equine* poorly defined pulmonary lobes; *bovine and canine* well defined lobes.
- Pulmonary lobes are subdivided into lobules by interlobular septa.
- Lobules are prominent in bovines and pigs, poorly defined in horses and man, and absent in dogs and cats.



Inflation Disturbances of the Lung

1. Atelectasis of the Lungs

- Atelectasis is imperfect or incomplete expansion.
- The lungs contain no air and nothing else.
- Atelectasis may be focal or involve entire lobes.
- It may be classified as fetal or acquired.
- Atelectasis occurs in all species except birds where the rigid structure of the parabronchi prevents collapse of the alveoli.

a. Fetal Atelectasis

- The lung is dark-red, liver-like, and will not float in water.
- If the lungs of a newborn are not inflated, it can be assumed that it was born dead**.**
- However, a newborn animal may take a few breaths and then die. In this case, areas of the lungs will be partially inflated while other areas remain atelectatic.
  - Fetal atelectasis is common in newborn foals .why? (***reading assignment***)

b. Acquired Atelectasis

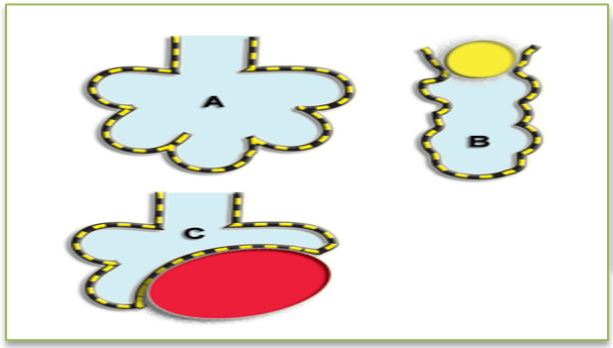
- May be caused by either compression or obstruction.
- Compression atelectasis is due to pressure on the lungs due to fluid, air, neoplasms, etc., which drives air out of the lungs resulting in collapse of the walls of alveoli.

- **Grossly,** the lung tissue is deflated, depressed, dark red, and will not float in water**.**

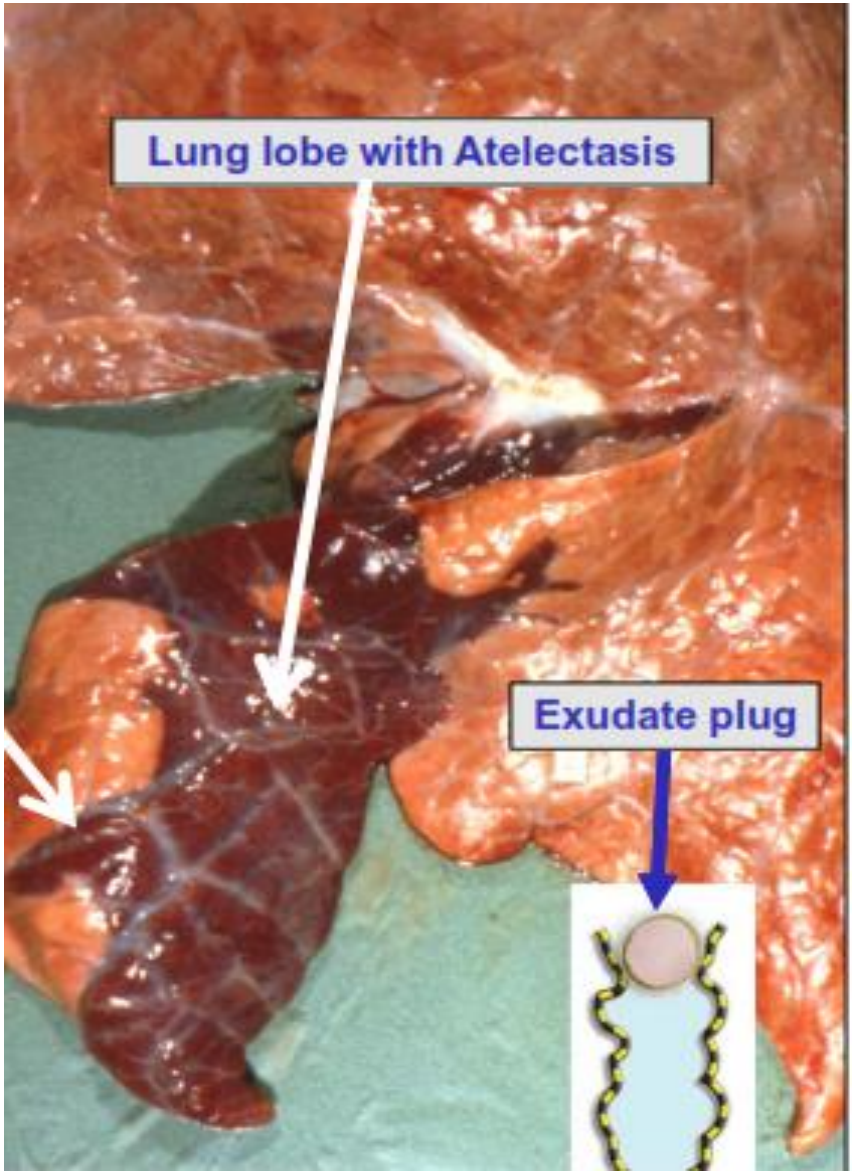
- If the cause is removed, the lung tissue will reinflate and no damage will follow.

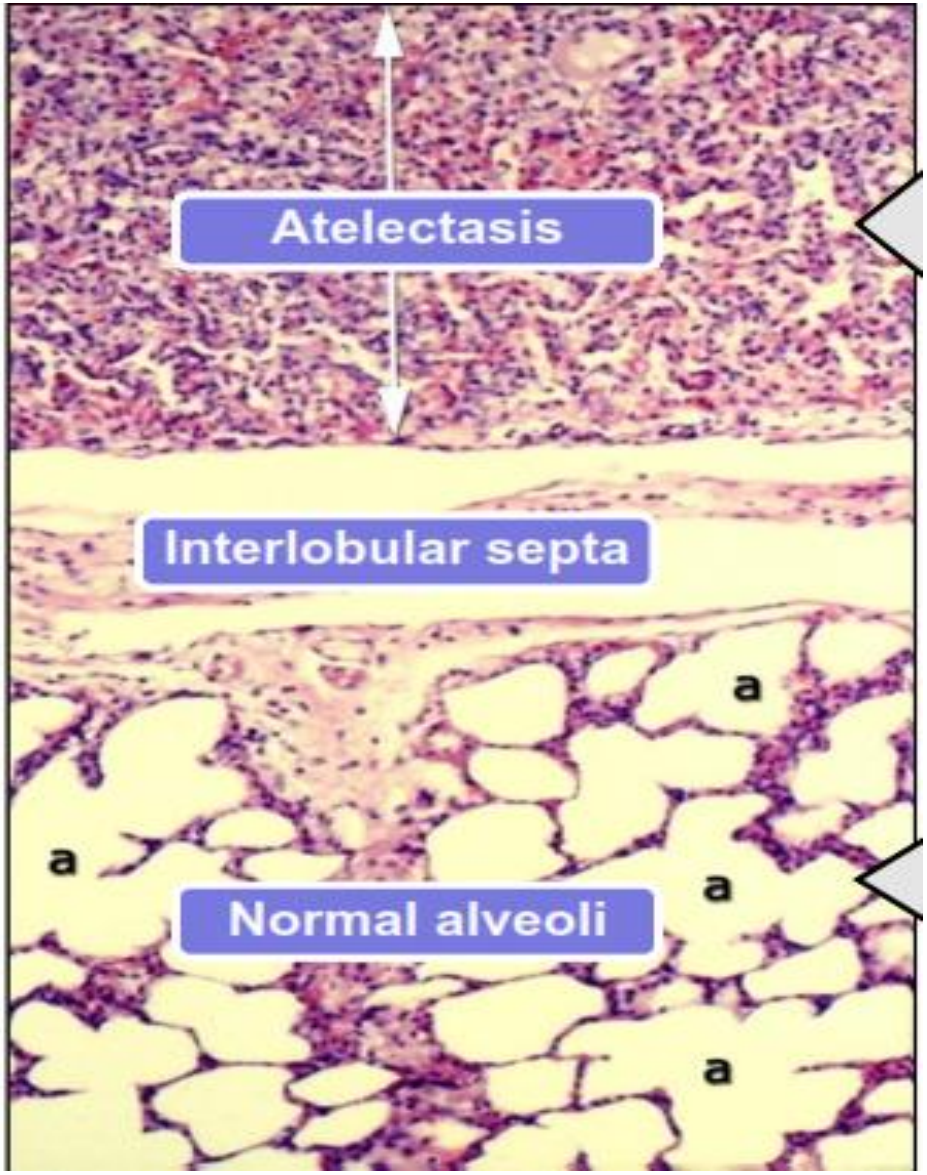
- Respiratory distress is not noticeable unless a considerable portion of the lung tissue is involved.

- Obstructive atelectasis is due to an accumulation of material in the bronchioles.
- If the airway is completely obstructed, air cannot enter the alveolus and it collapses.
- The effect on the body depends on the size of the affected area.
- Histologically, Atelectatic lung showed collapse of the alveolar space and devoid of air with increased thickness of the septa. Alveoli appeared as small or elongated cleft. The blood vessel of the affected part was congested



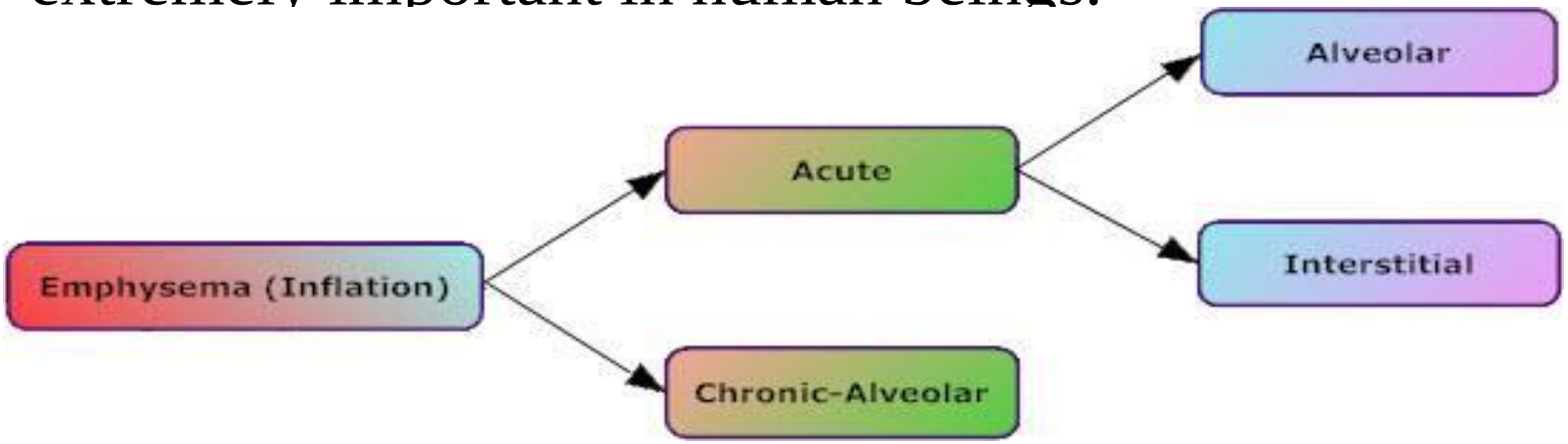
- **A. Normal alveolus**
- **B. Obstructive atelectasis:** Airway obstruction such as exudate, parasites, food particles.
- **C. Compressive atelectasis:** Large intra-thoracic masses such as abscesses, tumors, pericarditis, and external trauma.


Lung lobe with Atelectasis
Exudate plug


Atelectasis
Interlobular septa
a
a
a
a
Normal alveoli

2. Emphysema of the Lungs

- Emphysema is Over-inflated alveoli without rupture.
- Emphysema is an abnormal and permanent enlargement of airspaces distal to terminal bronchioles with destruction of their alveolar walls.
- Primary lung emphysema is rare in animals but extremely important in human beings.



2.1. Acute Pulmonary Alveolar Emphysema

- Caused by
  - Physical: Over exertion during struggling leads to over ventilation.
  - Nutritional: Feeding on lush green pastures.
  - Pathological
    - Over exertion during coughing (Bronchitis/ Bronchopneumonia).
    - Compensatory at areas adjacent to pneumonia.
    - Loss of elasticity of wall of alveoli.
    - Allergy: Feeding mouldy forage/potato
    - Poisons: Parathion poisoning.

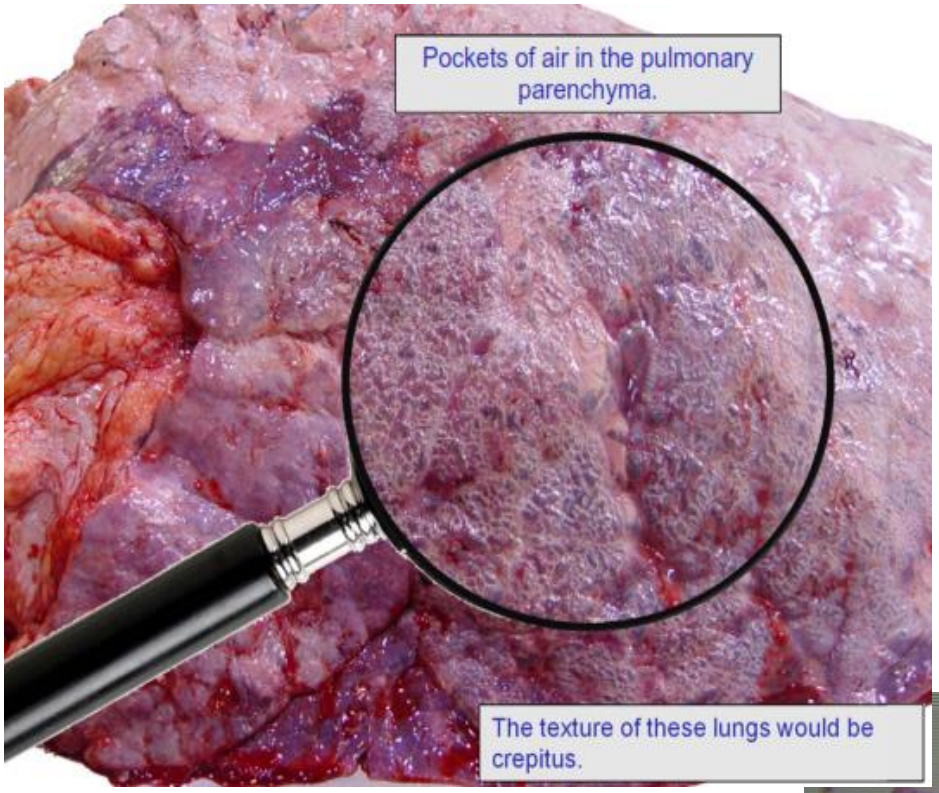
- Gross pathology
  - Certain areas of lung are unduly distended with air.
  - Pale or white areas project above surrounding tissue.
  - Cut surface is dry.

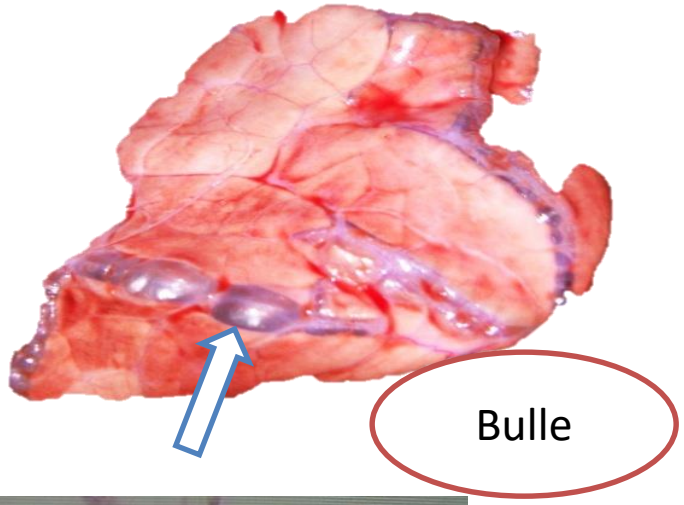
- Histopathology
  - Alveolar size greatly distended (Giant alveoli).
  - Wall of the lung : Sometimes ruptured combined to form bullae.

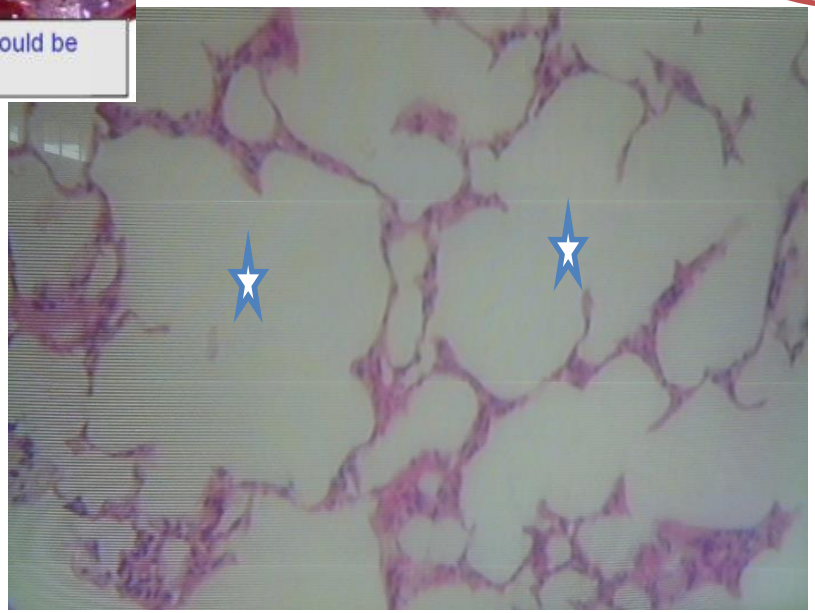
2.2. **Acute Interstitial Emphysema**

- In this condition air collects in the interlobular septa beneath the pleura and wherever there is interstitial tissue in the lungs.
- Often it accompanies with acute alveolar emphysema.
- More often seen in cattle and sheep.
- Caused by :
  - Violent respiratory effort (struggling) during death from loss of blood.
  - Perforation of lung by mechanical means- foreign body through rumen and reticulum.
  - Excessive bellowing in estrum or when separated from calf.
  - Forced breathing as in old hunting dogs.
  - Pulmonary strongylosis.

- Gross pathology
  - Air may escape through thoracic inlet into the subcutis of neck.
  - In Severe case,
    - Air collects beneath the pleura and other interstitial tissue of lung.
    - Air accumulate along the spine – from pole to base of tail.
  - Interlobular septa are thickened and seen as criss cross straight lines usually at wider intervals.
  - Interlobular septum: shiny, well outlined and filled with large and small air bubbles.
- Histopathology
  - Lung interstitial tissue is widely separated.
- Chronic alveolar emphysema **(**Broken Wind/Heaves) ***reading assignment.***


Pockets of air in the pulmonary parenchyma.
The texture of these lungs would be crepitus.


Bulle



Circulatory disturbance of the lung

1. Congestion of The Lungs

- Pulmonary congestion and edema develop acutely in a variety of diseases.

- Chronic congestion and edema are usually attributable to some functional defect in the left heart (the left ventricle and atrium are prevented from clearing the blood that comes from the lungs).

- Hypostatic congestion develops due to the effects of gravity on blood flow through the lungs.

- If an animal lies on one side for a long period of time blood accumulates in the lower lung. The affected lung will be nonfunctional, extremely heavy, dark-red, and firm to the touch.

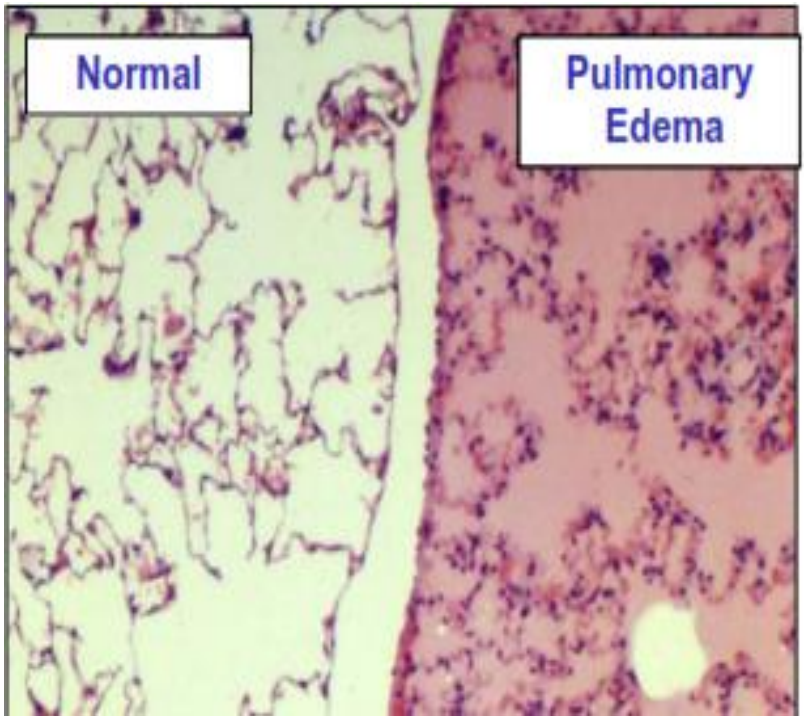
- In contrast, the upper lung will be pale, inflated, and light in weight.

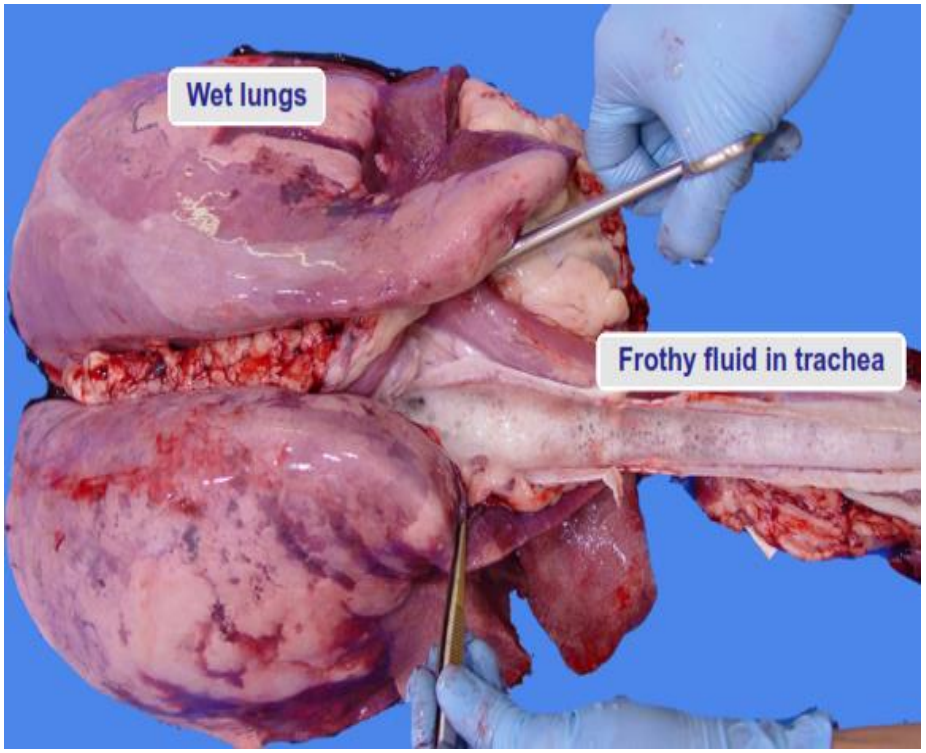
2. Edema of the Lungs

- Pulmonary edema is the accumulation of fluid **(**plasma protein filtrate**)** in alveoli. This is due to:
  - Altered hemodynamics - (disturbance of normal fluid exchange in the lungs such as in cardiac failure backup of blood, and consequently increased hydrostatic pressure in the alveoli.
  - Sudden diffuse and direct damage to capillary endothelium - (usually the peracute stage of inflammation).

- **Grossly,** the lungs are doughy, heavy, firm, and foamy. Fluid flows from the cut surface.

- **Microscopically**: Bronchi and alveolar lumen contain a pink stained homogeneous material proportional to the amount of protein present.





- **Clinically**: the head is held low, the nose is flecked with froth, dyspnea and gasping are prominent, and wheezing, bubbly sounds of respiration are characteristic.

- Here are two clues when doing the post mortem:
  - Look in the trachea – if there is foam, that is a good indication that there was pulmonary edema.
  - Remove the lungs from the thorax and set them in a dry spot. If, after 5 minutes, the lungs are sitting in a puddle of fluid, that is evidence of pulmonary edema.

Inflammation of The Lungs

- Pneumonitis is the correct term for inflammation of the lungs.

- However, pneumonia is the conventional term for lung inflammation (correctly, the term pneumonia refers to **filling of alveoli with cellular exudate**).

- The causes of pneumonia are numerous and may include bacteria, viruses, aspirated foreign matter, fungi, parasites, etc.

CLASSIFICATION OF PNEUMONIAS

Controversial topics in veterinary medicine

- There are numerous classifications such as :
  - **Etiological:** Viral pneumonia, Mannheimiosis, Histophilosis pneumonia, distemper pneumonia, allergic pneumonia, etc.
  - **Epidemiological:** Enzootic pneumonia, contagious bovine pleuropneumonia, etc.
  - **Exudate:** Suppurative, fibrinous, or granulomatous pneumonias.
  - **Topographical (distribution):** Lobar, lobular, diffuse, interstitial, focal, etc.
  - **Miscellaneous:** Progressive pneumonia, proliferative pneumonia, atypical pneumonia, pneumonitis, etc.

❑ Stages of Pneumonia:

– Traditionally, bronchopneumonias caused by bacterial agents have been divided into four successive stages:

1. Stage of congestion,
2. Stage of red hepatization.
3. Stage of grey hepatization, and
4. Stage of resolution.

- **Stage of congestion** represents the developing bacterial infection and lasts for approximately 24 hr.

  – Microscopically, it is characterized by hyperemia**,** fluid within the alveoli (edema), a few neutrophils, and often by the presence of numerous bacteria.

  – **Grossly,** the involved lung is heavy, boggy, and red (the affected areas are not consolidated).

- **Stage of red hepatization** is characterized by increasing numbers of neutrophils and precipitation of fibrin to fill the alveolar spaces.

- Affected areas of the lungs are consolidated (liver-like in consistency). Completely consolidated lung tissue sinks in water.

  – Grossly, involved lung is distinctly red, firm, and airless, with a liver-like consistency.

- **Stage of gray hepatization** is characterized by a continuing accumulation of fibrin, associated with progressive disinte-gration of neutrophils and erythrocytes.

- The hyperemia is decreased and erythrocytes have disappeared from the alveolar contents. Mononuclear cells and fibrin predominate, with fewer neutrophils.

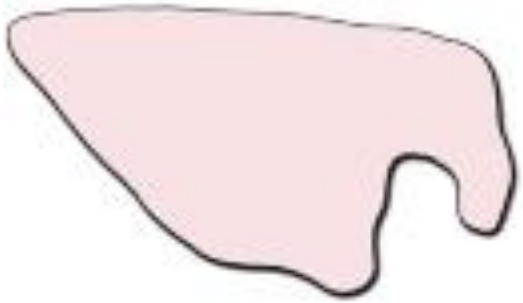
- **Grossly**, the affected lung tissue appears grayish **(**or less red). Tissues are still consolidated (liver-like) and will sink in water.

- **Stage of resolution** is the final stage. this stage may take in 7 to 10 days after the onset of pneumonia and a favorable outcome may follow.

- During this stage, the exudate within alveoli (fibrin, inflammatory cells, etc.) undergoes progressive enzymatic digestion to produce a semifluid debris that is either reabsorbed, ingested by macrophages, or coughed up.

- In favorable cases, the normal lung parenchyma is restored to its normal state. However, complete resolution may not occur and complications develop.

Distribution and Texture of Lesions in Pneumonias



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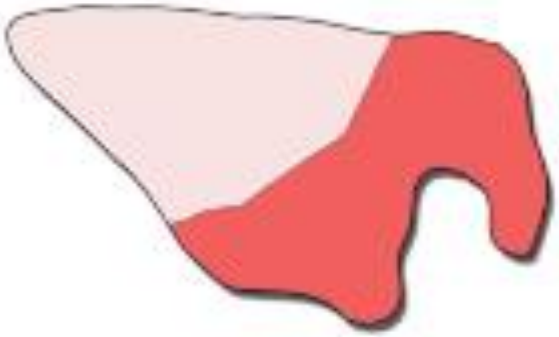
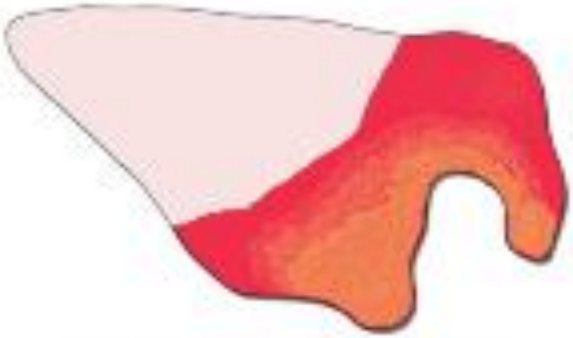
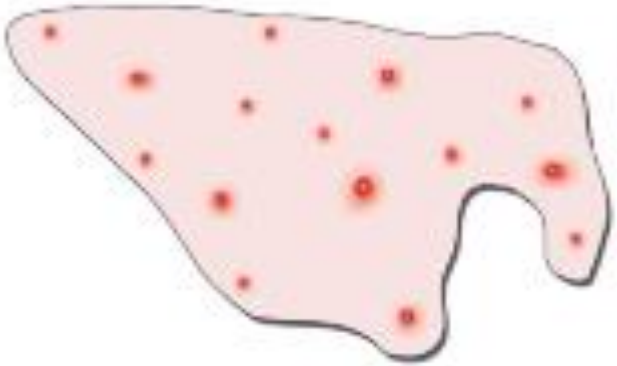


Suppurative Bronchopneumonia
Cranioventral and firm

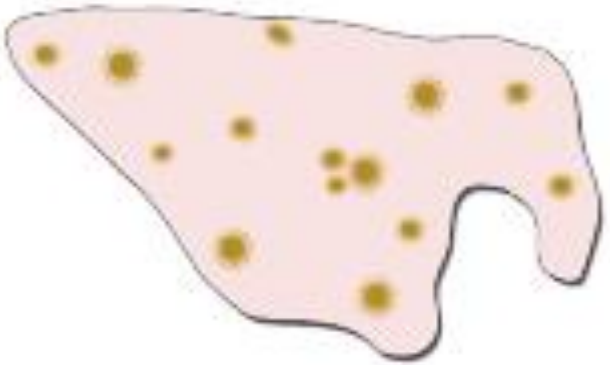


Fibrinous Bronchopneumonia
Cranioventral and hard

Interstitial Pneumonia
Difuse and Elastic



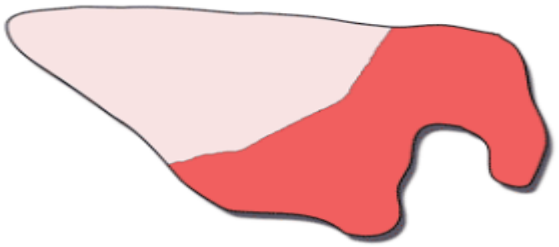
Embolic Pneumonia
Multifocal and nodular



Granulomatous Pneumonia
Multifocal and nodular

❖ Suppurative Bronchopneumonia

- Bronchopneumonia is the most common form of lung inflammation.
- It is particularly common in young calves and pigs and in sheep of any age.
- Bronchopneumonias in animals are primarily bacterial in origin and characterized by intense exudation of inflammatory cells (chiefly neutrophils) into **alveoli.**
- Also, whenever the lesions are lobular and centered around the bronchiolar tree, that is why the term bronchopneumonia is used.

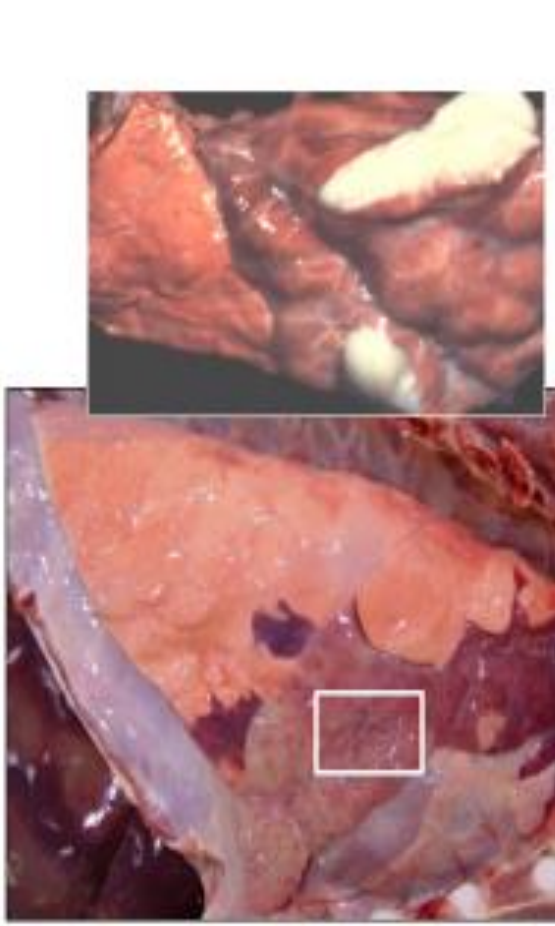


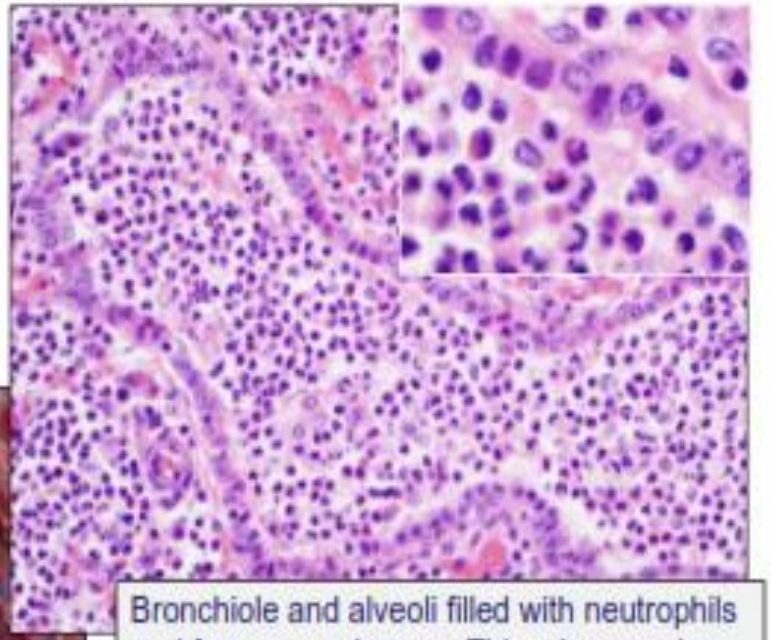
- Many species of pathogenic bacteria may cause bronchopneumonia.
  - In calves, pigs, and sheep, Pasteurella spp (P. multocida and/or P. hemolytica) and Corynebacterium pyogenes are usually found.
  - In horses, Streptococcus spp. Staphylococcus spp. Corynebacterium equi and E. coli are important.
  - In dogs canine distemper virus is considered to be a primary etiologic agent.

- Bacteria will pathogenic potential only when the respiratory environment is suitably altered by other agents (viruses, external environmental factors, etc.). In other words, bacterial agents usually act as secondary invaders.

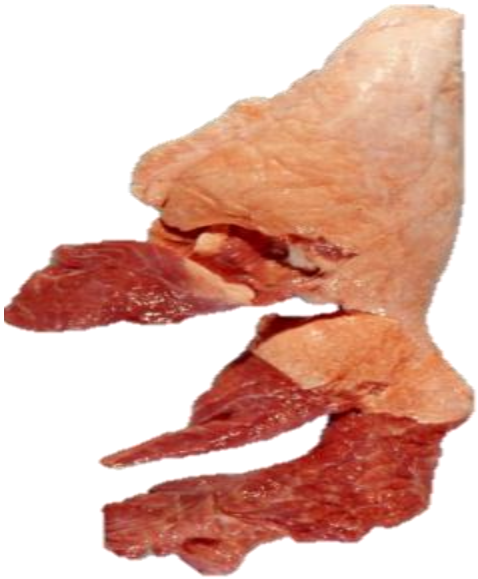
- **Gross lesions:** Affected lung is consolidated and the lobular pattern is accentuated. **Color** varies from red (acute, hyperaemia) to grey (chronic inflammation, atelectasis, fibrosis).
  - Typically, purulent/pus exudate can be expressed from airways; in chronic bronchopneumonia the exudate takes a mucoid appearance.
- **Microscopically:** Large number of polymorphonuclear leukocytes in bronchoalveolar space in acute cases and a mixture of PMN,PAM and mucus (goblet cell hyperplasia) in the more chronic cases.

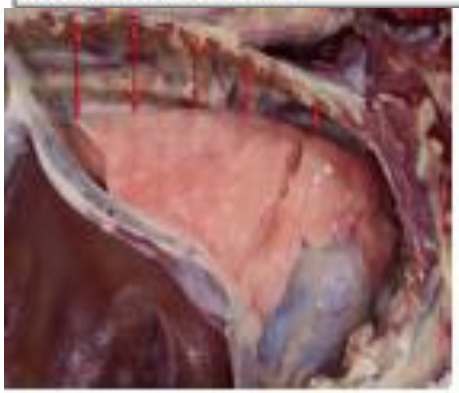
Suppurative Bronchopneumonia / Histopathology





Bronchiole and alveoli filled with neutrophils and few macrophages. This microscopic findings are typical of suppurative bronchopneumonia





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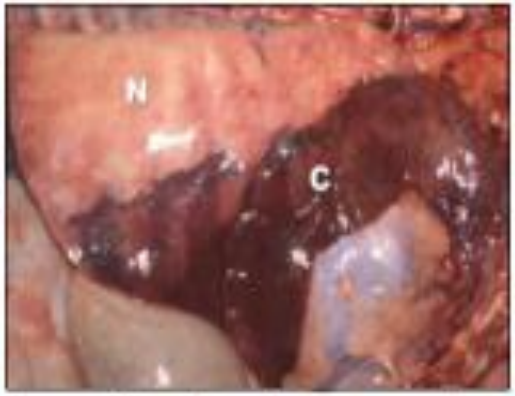
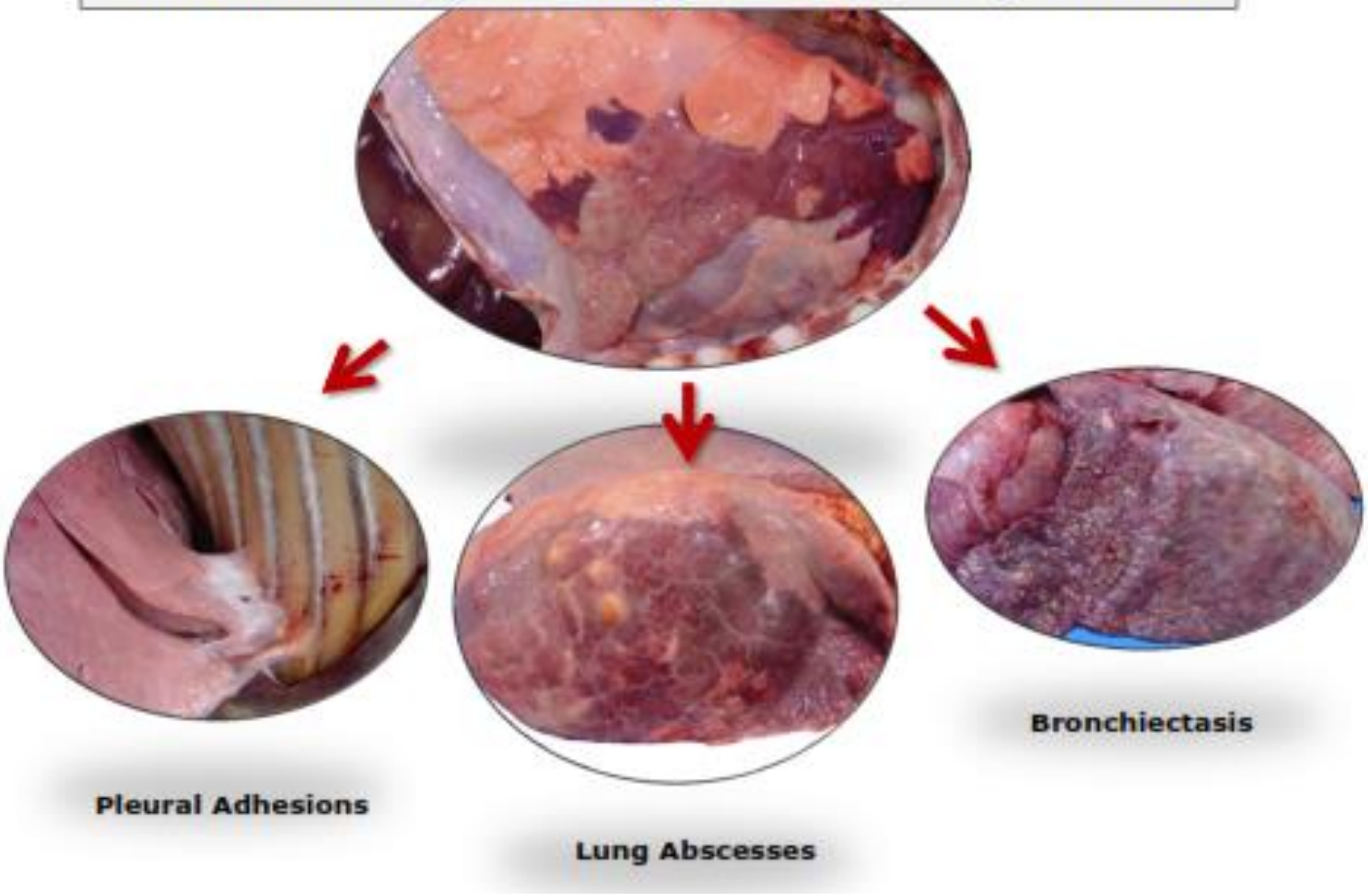


Suppurative Bronchopneumonia
Cranioventral and firm

Most Common Sequels of Suppurative Bronchopneumonia



❖ Fibrinous Bronchopneumonia

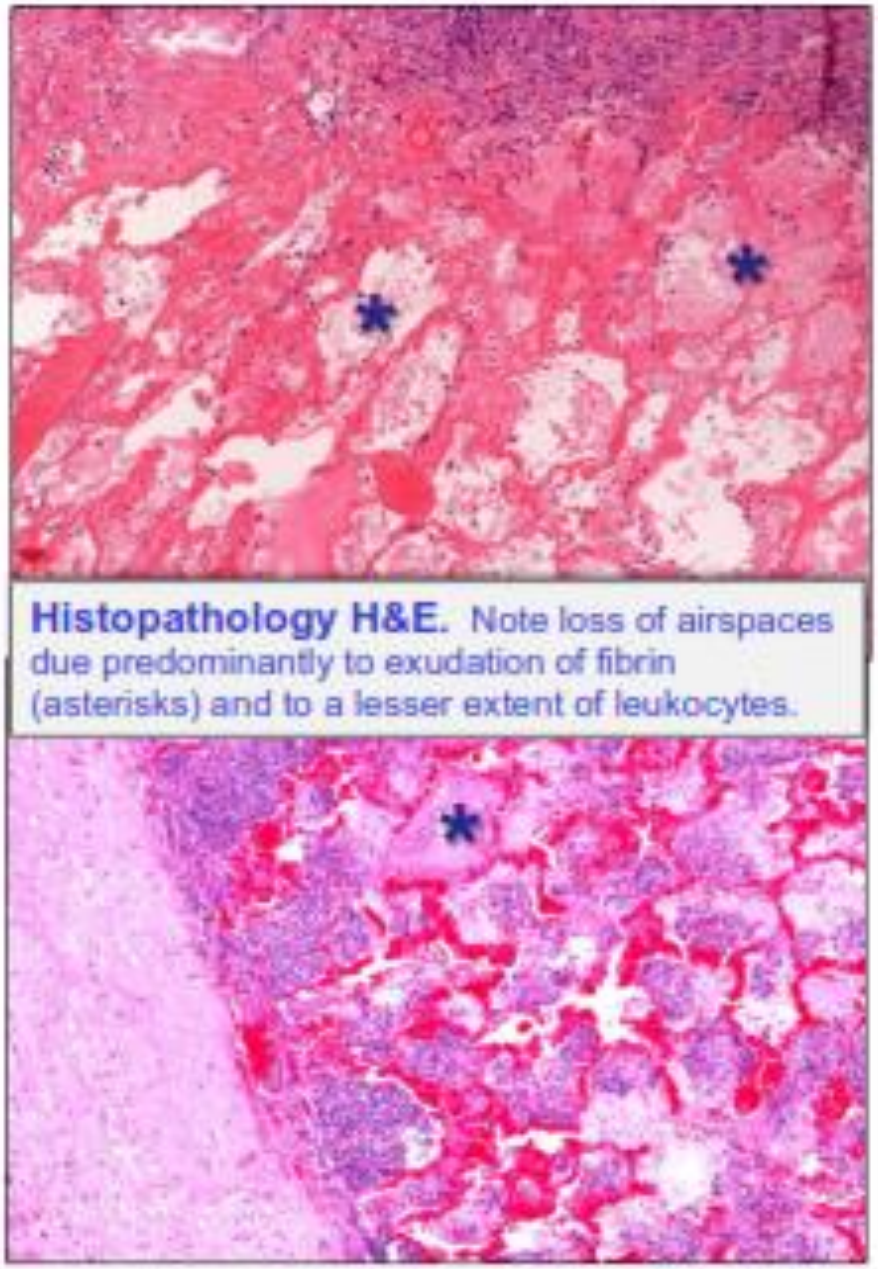
- Fibrinous pneumonia is basically a bronchopneumonia characterized by a marked exudation of fibrin.
- Occasionally, fibrinous pneumonia is hematogenous in the course of septicemic salmonellosis.
- Fibrinous bronchopneumonia is an important disease in cattle, sheep and swine and is usually caused by Pasteurella spp. (P. multocidia and P. hemolytica).
- Examples of disease causing fibrinous bronchopneumonia: Pneumonic Mannheimiosis (shipping fever), Porcine Pleuropneumonia, CBPP.

- **Grossly:**
  - Cranioventral consolidation Lung texure: Hard in consistency,
  - Color of affected lung: Red → yellow → grey (Fibrin on pleura).

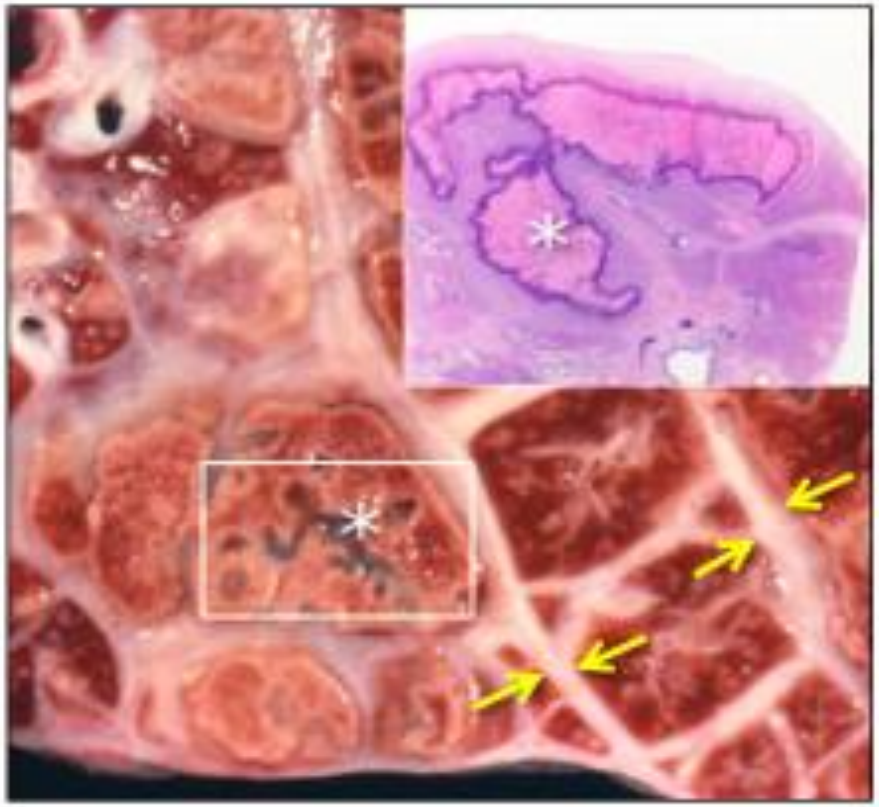
- **Histopathology:** massive exudation of fibrin and polymorphonuclear leukocytes into the bronchoalveolar space and pleural, areas of coagulative necrosis.


More examples of Fibrinous Bronchopneumonia


Fibrinous Bronchopneumonia

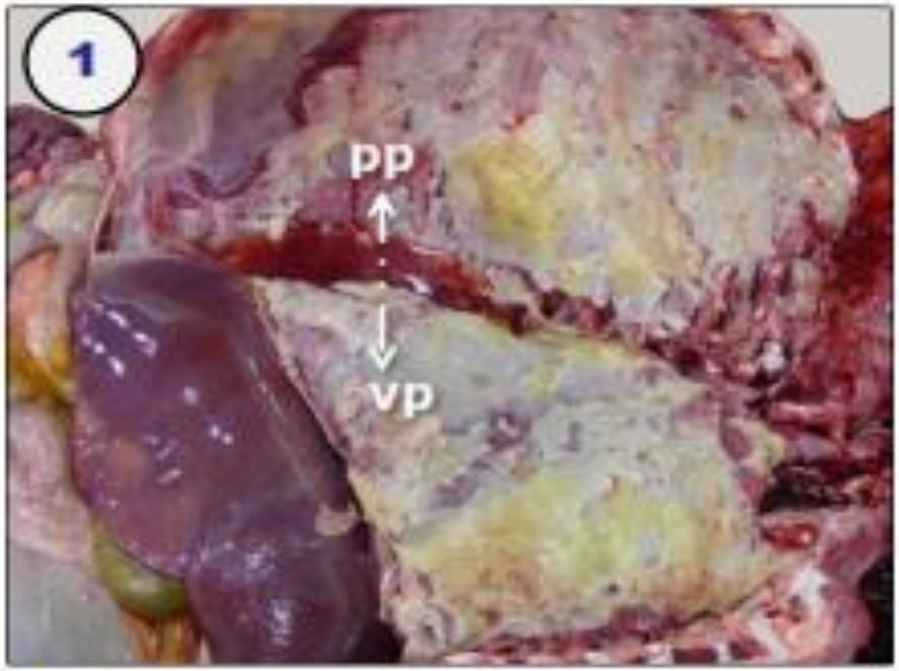


Histopathology H&E. Note loss of airspaces due predominantly to exudation of fibrin (asterisks) and to a lesser extent of leukocytes.



Fibrinous pneumonia, particularly Pneumonic Mannhemiosis (Shipping Fever) and Porcine Pleuropneumonia also cause coagulative necrosis (asterisk) and notable dissention of the interlobular spate

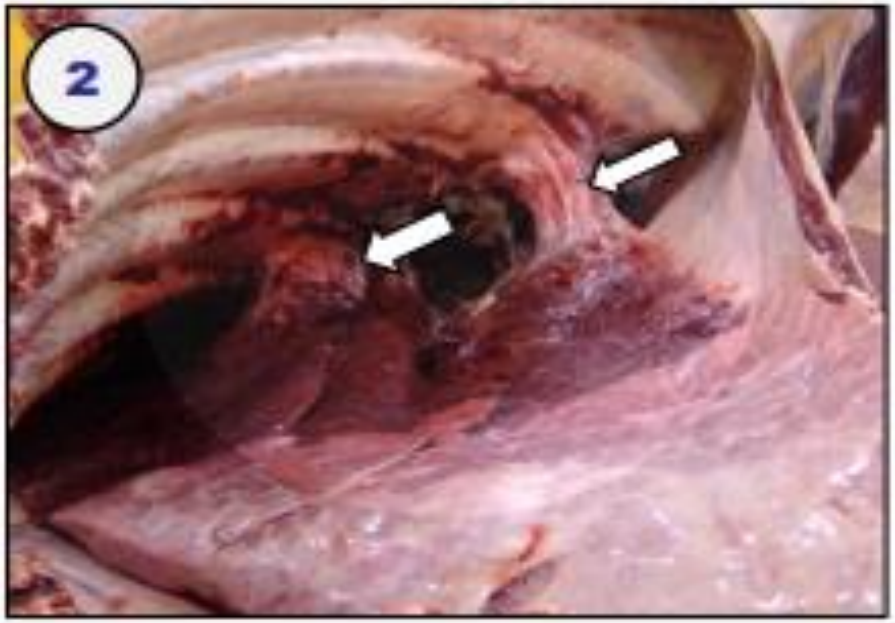
Pleural Adhesions

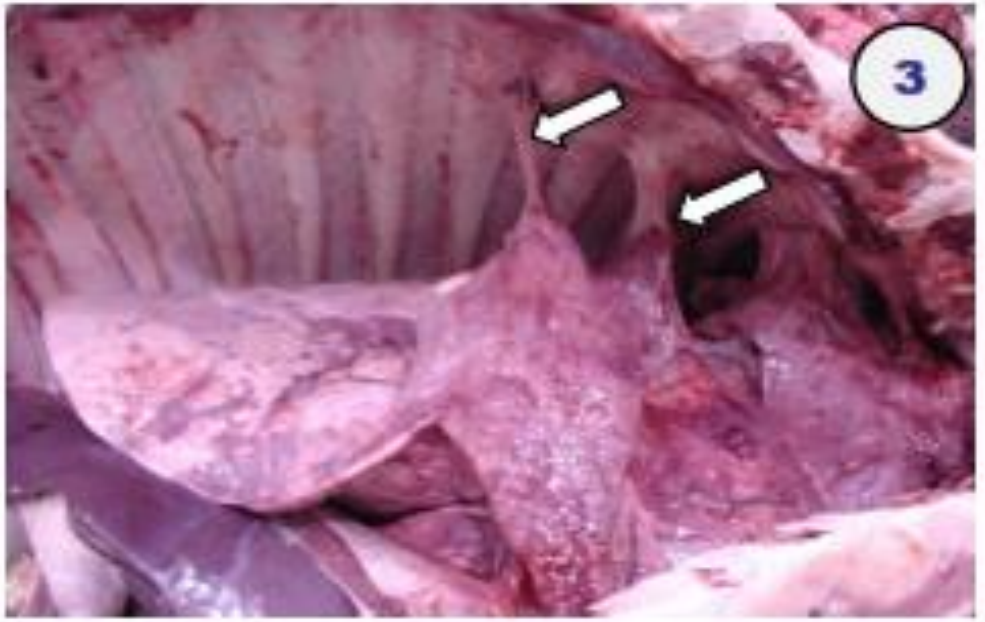


Pleural Adhesions are also important sequel to fibrinous bronchopneumonia.

1- Visceral pleura (vp) and parietal pleura (pp) were already attached (acute case).

2 and 3- Fibrous adhesions between visceral and parietal pleura (arrows).





❖ Interstitial Pneumonia (Primary Atypical Pneumonia)

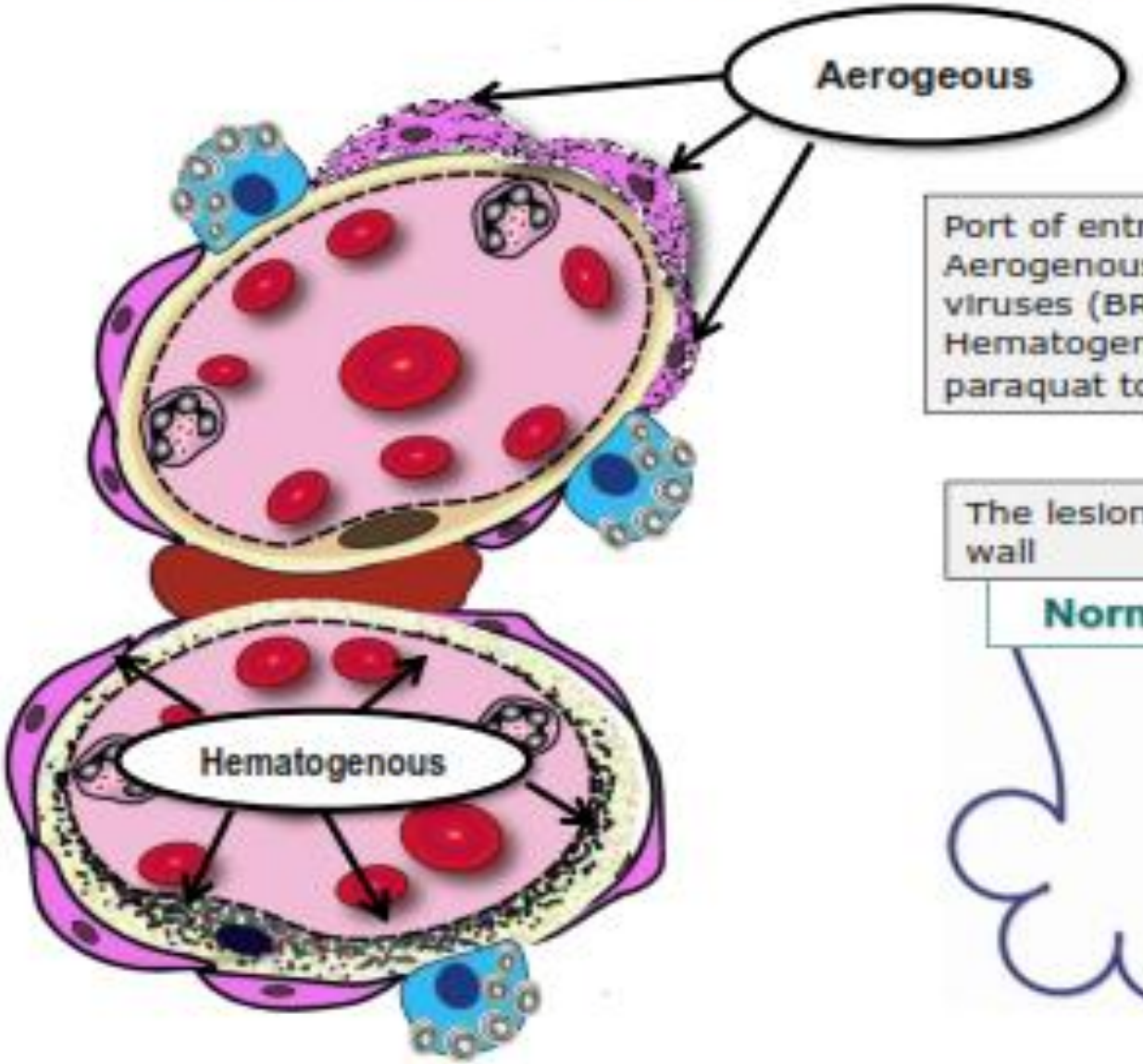
- Interstitial pneumonia **(pneumonitis)** is characterized by inflammation of the alveolar walls with an absence or minimum exudation of inflammatory cells (neutrophils, etc.) into alveolar lumens.

- The causes of IP are numerous and include viral infections (feline pneumonitis, canine distemper, equine viral rhinopneumonitis, etc.), chemical toxicosis (turpentine, kerosene, etc.), mycoplasma infection (enzootic pig pneumonia, etc.), and some allergic reactions.

- In addition, interstitial pneumonia is common in septicemic diseases and/or those characterized by sustained or intermittent bacteremia **(**colibacillosis, salmonellosis, erysipelas, leptospirosis, etc.).

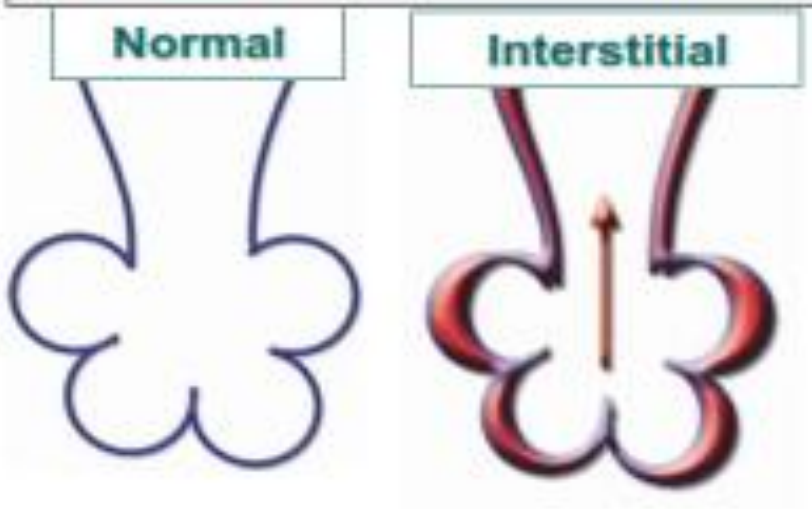
- A diagnosis of interstitial pneumonia depends on the detection of any one of a combination of the following:
  - Thickening of the alveolar walls by exudates **(fibrin, etc.)**, and by an infiltration of inflammatory cells **(macrophages, plasma cells, and lymphocytes).**
  - Proliferation of alveolar epithelial cells.
  - Formation of hyaline membranes in the alveoli and covering alveolar ducts.

Ports of Entry for Interstitial Pneumonia



Port of entry could be either Aerogenous such as with respiratory viruses (BRSV, IBR), Distemper) or Hematogenous such as in herbicide paraquat toxicity or sepsis (ARDS).

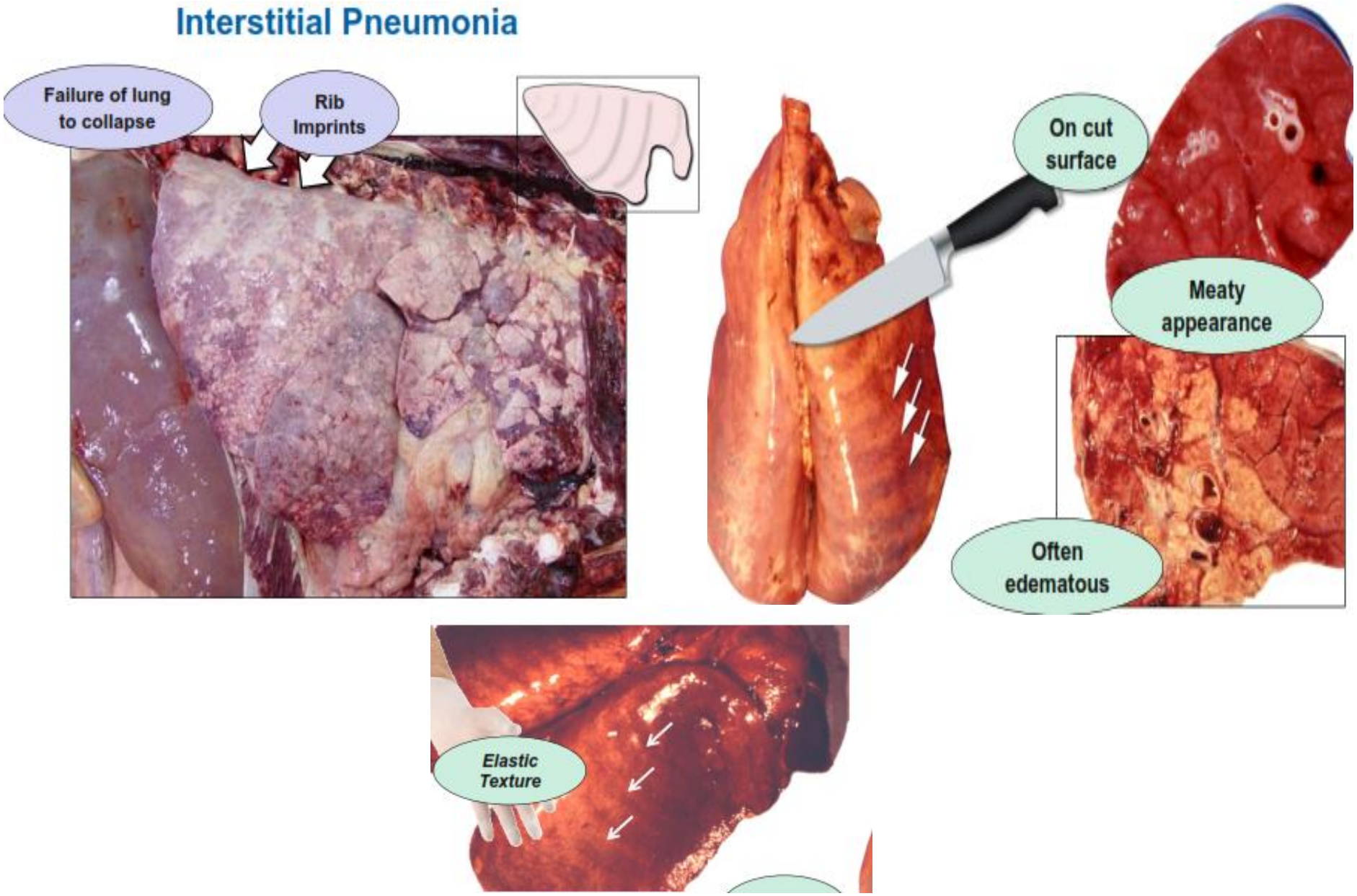
The lesion is centered in the alveolar wall



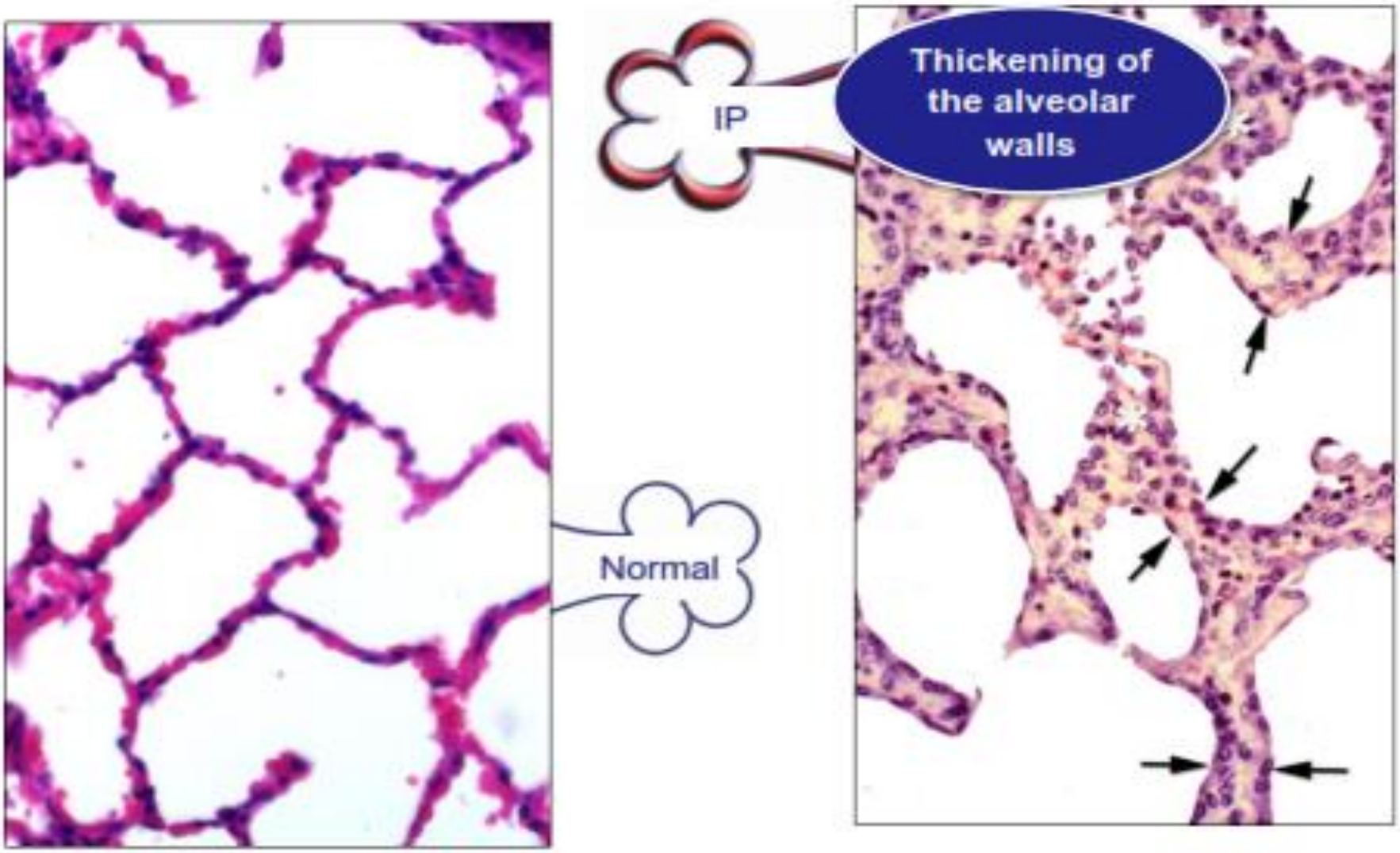
- **Gross lesions:**
  - The lungs fail to collapse when the thorax is opened;
  - occasional costal imprints are visible on the pleural surface. Meaty appearance on the cut surface.
  - The color depends on blood: tissue ratio and type of exudate or fibrous scarring.
- It is difficult to diagnose grossly generally requiring histopathologic confirmation.

- **Histopathology:** The primary lesion is centered in the alveolar wall.
  - Thickening of alveolar walls.
  - Interstitial exudation or proliferation of type II pneumonocyt
  - In chronic interstitial pneumonia there is alveolar fibrosis.

Interstitial Pneumonia



Histopathology / Interstitial Pneumonia



❖ Embolic (Hematogenous) Pneumonia

- Embolic pneumonia refers to foci of lung inflammation initiated by the lodgement of emboli of pathogenic organisms or septic fragments of thrombi in the pulmonary arteries or capillaries.

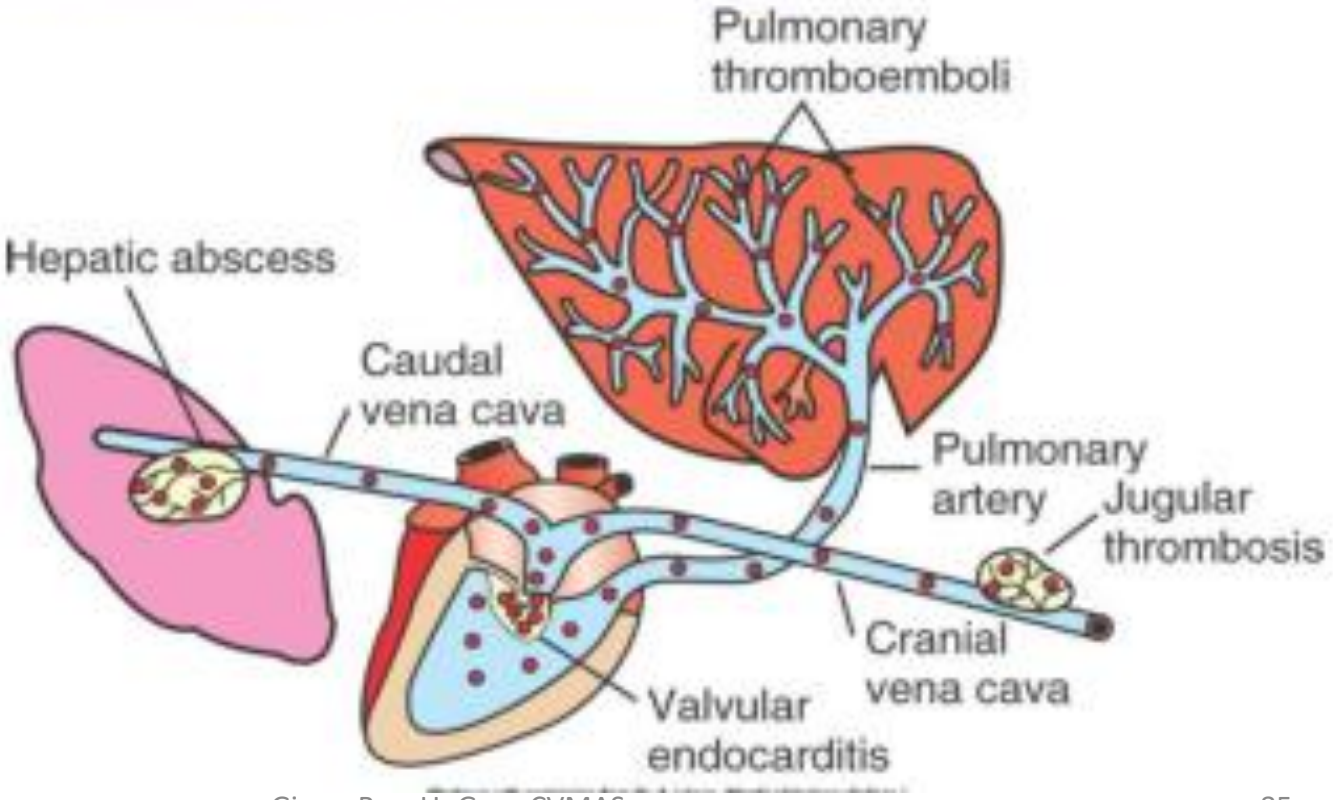
- Most organisms which cause embolic Pneumonia are pyogenic and abscessation is to be expected.

- Endocarditis, ruptured hepatic abscess (vena cava thrombosis in cattle), Omphalophlebitis cause embolic Pneumonia

Embolic Pneumonia

Most common etiologies:

- Vegetative endocarditis (right side of the heart)
- Jugular thrombosis
- Rupture of hepatic abscesses into the vena cava (cattle
- Embolic foreign body (hair, septic emboli, etc).

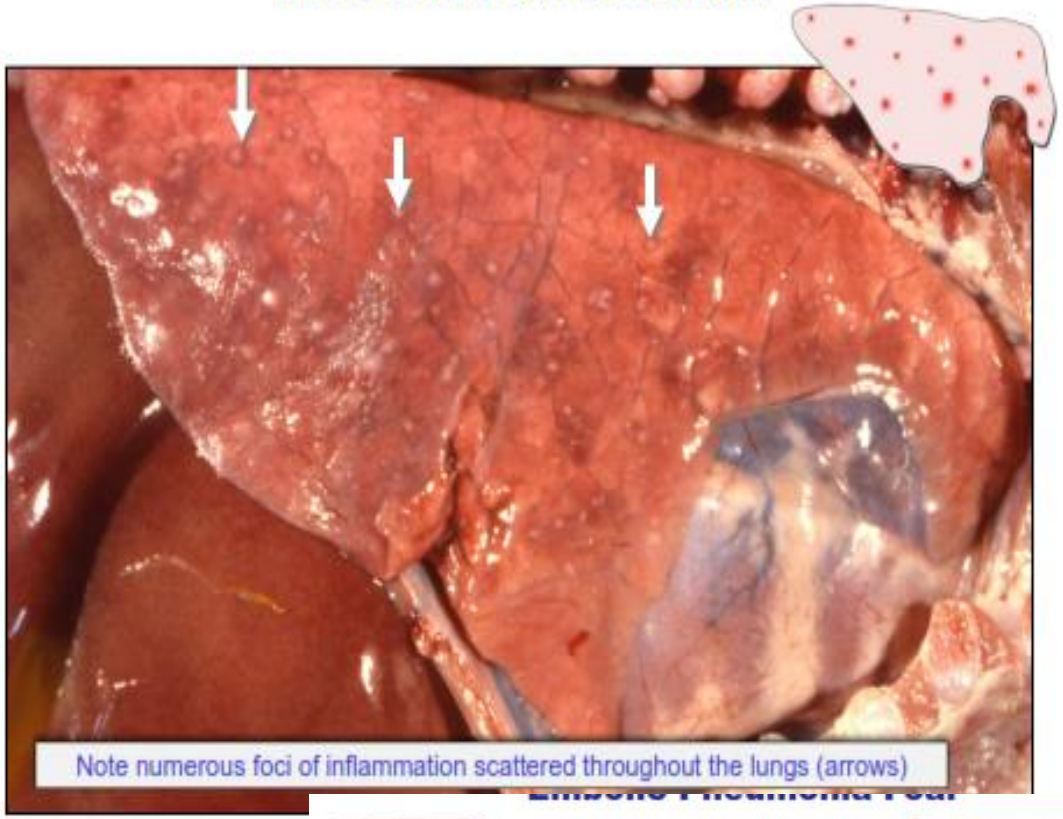


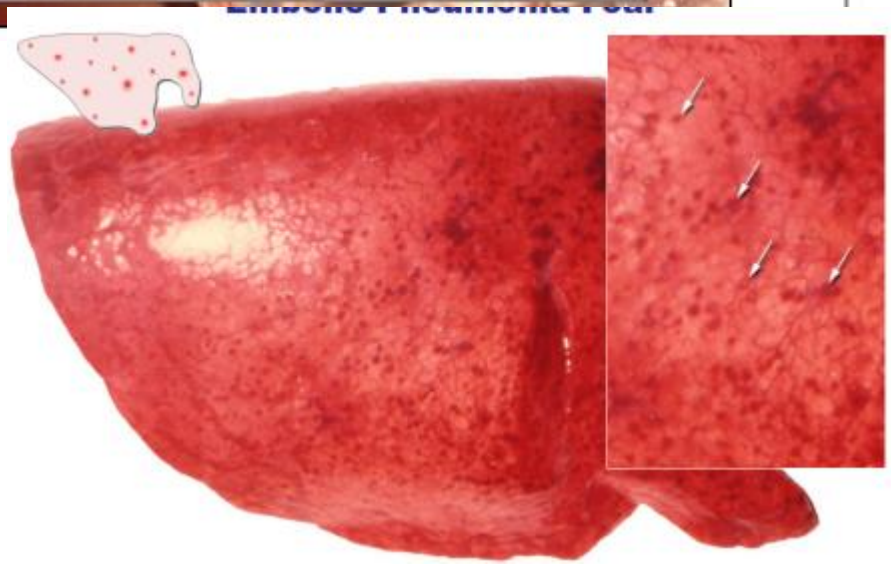
- **Gross lesions:** Variable number of foci, often with a white center and red hemorrhagic margins. Eventually embolic lesions may progress to abscesses.

- **Histopathology:** Septic emboli attached to pulmonary capillaries, pulmonary edema, microabscesses.

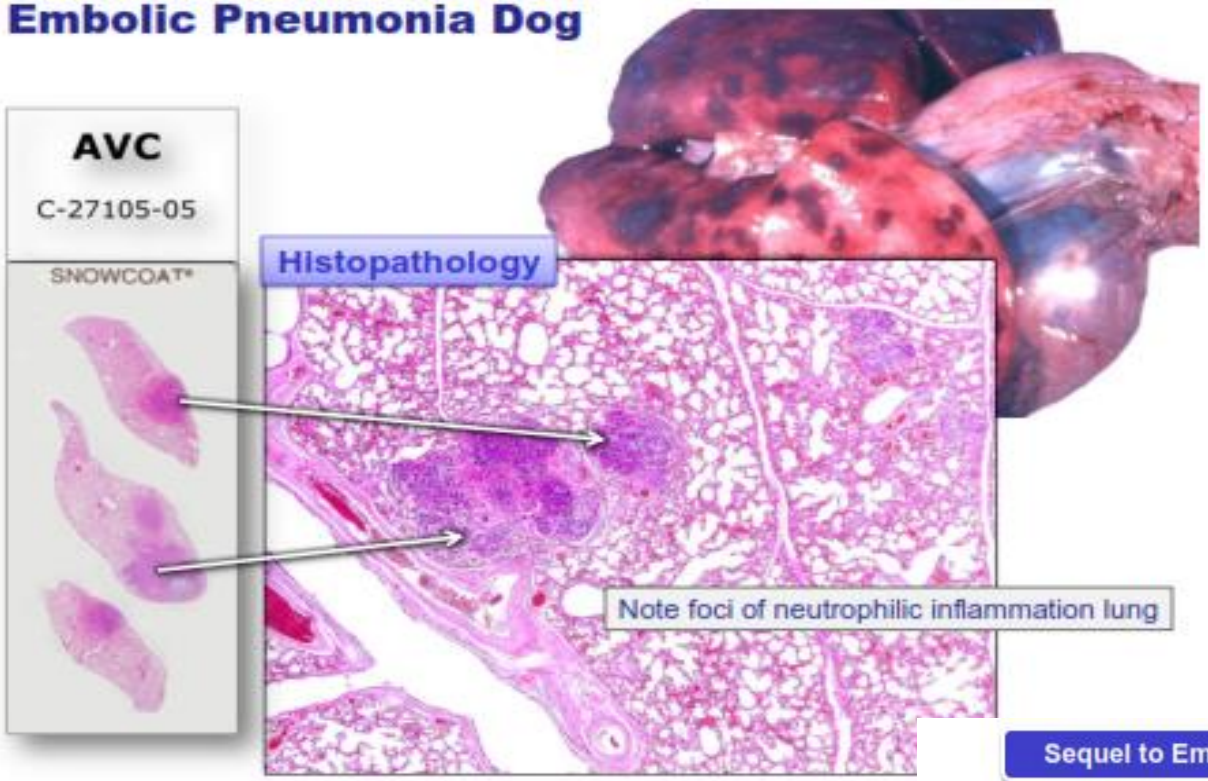
- **Common sequels:** Abscesses in all pulmonary lobes.

Embolic Pneumonia Calf





Embolic Pneumonia Dog



Sequel to Embolic Pneumonia: Pulmonary abscesses

❖ Granulomatous Pneumonia

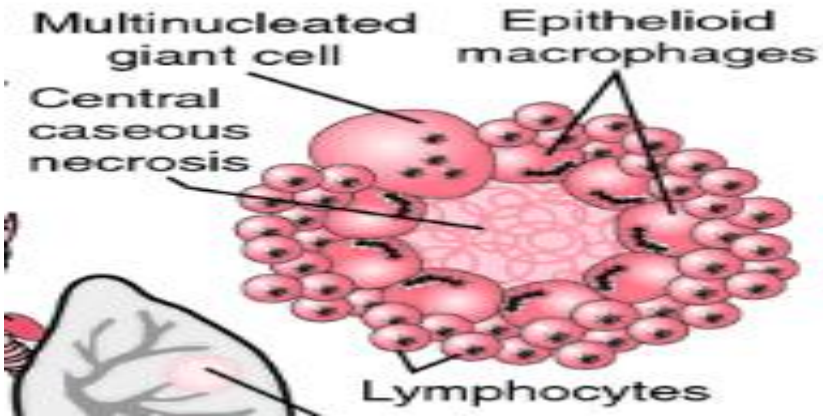
- Refers to a distinctive pattern of chronic lung inflammation evoked by certain etiologic agents (usually higher bacteria and fungi).

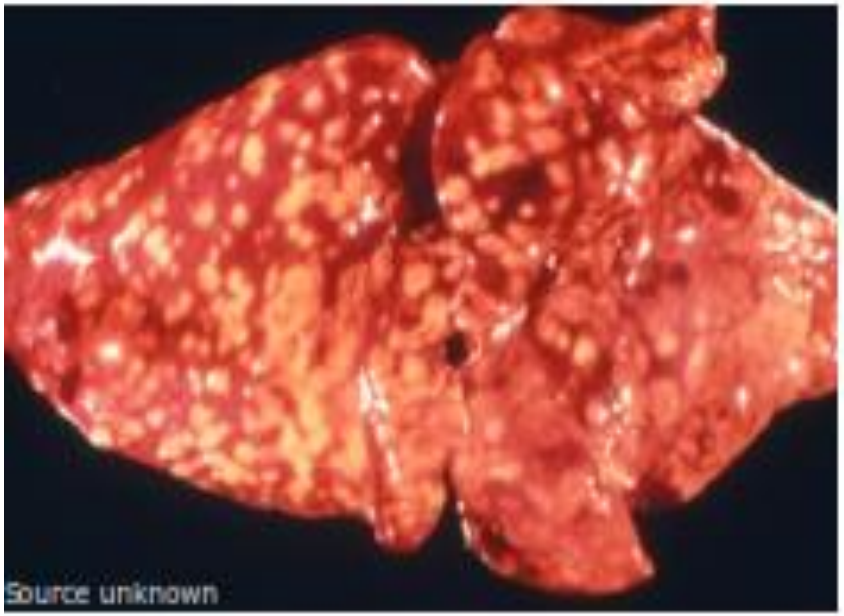
- **Etiology:** Tuberculosis, systemic mycosis, some parasites (*Muellerius capillaris; larva migrans*). Usually caused by microorganisms, parasites (ova, larvae) or foreign material (inhaled food particles) difficult to eliminate by phagocytosis.

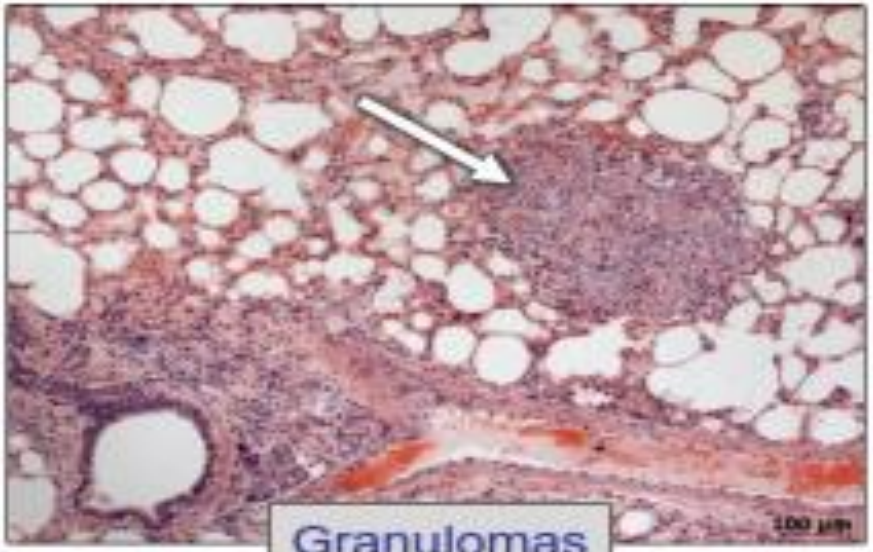
- The typical granuloma consists of collections of modified macrophages (epithelial cells), usually surrounded by a rim of lymphocytes.

- **Gross lesions:** Granulomas in the lung and sometimes in other organs too.
  - Be aware that granulomatous pneumonia can resemble lung cancer and may require histopathological confirmation.

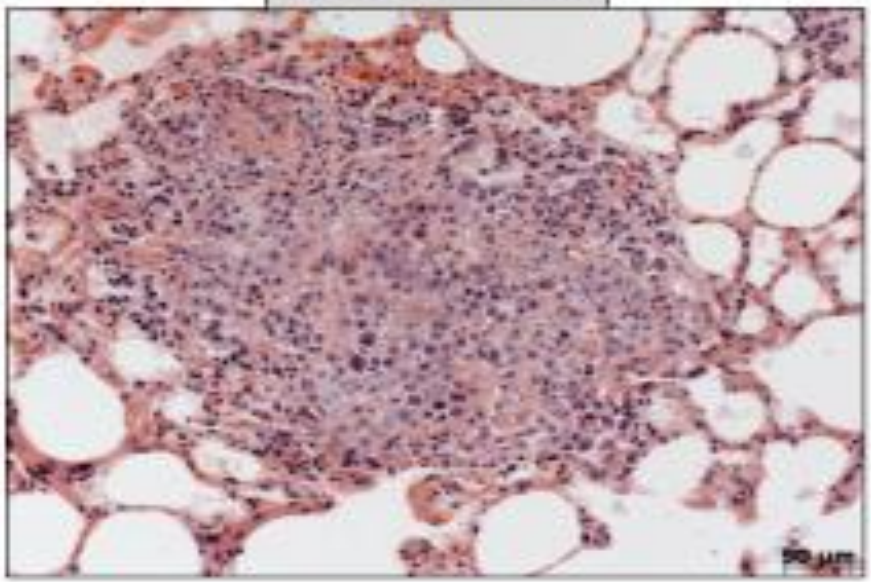
- **Histopathology:** Variable size nodules with a necrotic center infiltrated by macrophages and giant cells and surrounded by connective tissue mixed with lymphocytes and plasma cells.

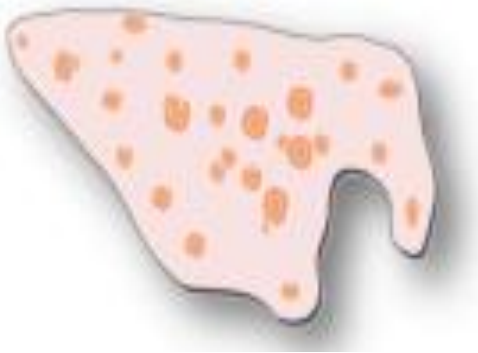






Granulomas





Type of pneumonia	Portal of entry	Lesion distribution	Kind of exudates	Example
Broncho-pneumonia	Aerogenous	Antero-ventral	Suppurative or fibrinous, on surfaces (bronchi, pleura)	Enzootic pneumonia, shipping fever, *Bordetella*
Interstitial pneumonia	Aerogenous or hematogenous	Anteroventral or diffuse	Often can't see them, they are within alveoli	Influenza, PRRS, toxins
Granulomatous pneumonia	Aerogenous or hematogenous	Multifocal	Granulomas, caseous nodules	TB, *Blasto*, *Aspergillus*
Embolic pneumonia	Hematogenous	Multifocal	Purulent foci	Septicemia, *Actinobacillus equuli*

○ Special Forms of Pneumonia

1. Gangrenous Pneumonia

- Is not an independent type of pneumonia, but a complication of other forms in which there is necrosis and invasion of lung tissue by saprophytic and putrefactive bacteria.

- The usual cause of this condition is the introduction into the lungs of materials (medicines, etc.) intended for the gastrointestinal tract (saprophytic and putrefactive bacteria are usually introduced with the foreign material).

- At necropsy, the lungs may be yellowish-green to greenish-black, foul smelling, with extensive cavitations.

- **NOTE:** Gangrenous pneumonia is also referred to as aspiration pneumonia, inhalation pneumonia, foreign body pneumonia, medication pneumonia, and lipid pneumonia..

2. Verminous Pneumonia

- Refers to lung inflammation caused by parasites (larvae and/or adults)

3. Hypostatic Pneumonia

- Refers to lung inflammation that develops subsequent to the accumulation of blood and the inhalation of upper respiratory pathogens in the ventral portions of the lungs.

Specific Diseases Characterized by Lung Lesions

I. Paramyxoviruses

a) Parainfluenza-3

- Parainfluenza type 3 virus induces acute respiratory disease in a wide variety of species including cattle, sheep and goats, and horses. It attacks cells of the conducting airways.
- It can cause pneumonia alone, but is more commonly part of the etiologic complex of enzootic pneumonia in calves or shipping fever in adults.

b) Bovine respiratory syncytial virus

- Bovine respiratory syncytial virus occurs either alone in an outbreak form of pneumonia or in concert with other agents, especially bacteria, in the shipping fever syndrome.
- The virus attacks conducting airway epithelium, most severely that at the broncho-alveolar junction.
- **Histologically** there are frequently syncytial giant cells formed by fusing bronchiolar epithelial cells.

c) Canine distemper

- Canine distemper virus is pantropic, which means that a number of tissues and organs are targeted by the virus.
- The virus infects epithelium in multiple organs, so for the respiratory system, this would include nares, trachea, bronchi, bronchioles, and alveolar cells.
- Consequently, the pneumonia could look like a bronchopneumonia or an interstitial pneumonia. And, if the dog survives the pneumonia, the virus usually goes on the brain.

d) Peste des petits ruminants (PPR)

- This virus is closely related to rinderpest, and causes enteritis and pneumonia in sheep and goats.
- The disease is endemic in much of Africa and the Middle East, but is foreign to this hemisphere.

II. Orthomyxoviruses

- The Orthmyxoviridae include the influenza viruses, which infect several domestic species.

✓ *Equine influenza*

  – Disease usually occurs in young animals that are stressed and/or grouped with older horses. It may occur in outbreak form, with high morbidity but low mortality.

  – The virus infects both ciliated and alveolar cells, so it can look like a bronchopneumonia or an interstitial pneumonia.

III. Maedi

- Maedi is a chronic progressive interstitial pneumonia of sheep which is caused by a viral Maedi occurs principally in sheep **(2-years old or older)**, but it has been reported in goats.
- **Grossly**, the lungs are larger, and 2-4 times as heavy as normal lungs.
  - They do not collapse completely when the chest cavity is open and appear grey to pink in color.
  - Enlargement of the bronchial and mediastinal lymph nodes is a constant feature.

- **Microscopically,** changes are characteristic of a chronic interstitial pneumonia.
  - There is rather uniform thickening of alveolar walls by hyperplastic alveolar lining cells, lymphocytes, and mononuclear cells.
- There is an absence of healing.
- No treatment has been successful.

❑ Jaagsiekte disease is a chronic progressive interstitial pneumonia of sheep, characterized by rather uniform thickening of alveolar walls and by hyperplastic alveolar lining cells. The disease is similar in character to Maedi.

- **Bacterial pneumonias**

1. Bovine Enzootic Pneumonia

- Bovine enzootic pneumonia is composed of a number of etiologies, all of which can be interacting in synergy. It usually starts with a viral infection, maybe PI-3, or BHV-1, or a mycoplasmal infection, *Mycoplasma bovis*.

- After those are set up, then opportunists move in and set up camp - *Pasteurella multocida*, *Arcanobacterium pyogenes*, *or E. coli.* In some cases, *Mannheimia hemolytica* can move in and cause a full-fledged shipping pneumonia.

2. Pneumonic Pasteurellosis

- The members of the *Pasteurella genus* (*p. multocida and Mannheimia haemolytica*) are widely distributed among a variety of animal species.

- They are carried subclinically, usually in the upper respiratory tract, and migrate down the bronchial tree to cause disease when the opportunity presents itself.

3. Tuberculosis

- Bovine tuberculosis, caused by *Mycobacterium bovis*, *is* the principal respiratory tuberculous disease seen in animals.
- The tuberculous pulmonary process starts at the bronchiolo-alveolar junction and develops into a tuberculous granuloma, or tubercle.
- This tubercle is the result of an ongoing battle between the tubercle bacilli and the cell mediated immune response.

- **Parasitic diseases of the lungs**
- Lesions due to parasites in the lung come in two categories.
  - There are those parasites who live specifically in lung,
  - then there are others who merely do damage as they migrate through to somewhere else.

Host	Parasite
Cattle	Dictyocaulus viviparus
Cat	Aelurostrongylus abstrusus
Dog	Filaroides milski and F. osleri
Horse	Dictyocaulus arnfieldi
Sheep	Dictyocaulus filaria
Sheep	Protostrongylus rufescens
Sheep	Muellerius capillaris
Cattle	Dictyocaulus viviparus
Swine	Metastrongylus apri, M. salmi, and M. pudendotectus

Important Lung Worms

- **Calcification Of The Lungs**
  - Calcification of the lungs occurs rather frequently in the dog. It usually occurs as a consequence of vitamin D toxicosis in young dogs, whereas in older dogs, it is usually associated with chronic renal diseases and hyperparathyroidism **(metastatic calcification).**
  - **Grossly**, the lungs are gritty, firm to hard, and they do not collapse when the thoracic cavity is opened.
  - **Microscopically,** calcium concretions are found in alveolar walls.

- **Melanosis of The Lungs**
  - Melanosis refers to a deposition of melanin pigments in various organs and tissues:
  - the lung is a common site of involvement.
  - Affected animals are in a state of normal health.

- **Lung Neoplasia**
- Most neoplasms encountered in the lungs are metastatic growths (lungs are especially prone to the lodgement of tumor emboli).
- However, any component of lung tissue can give rise to primary neoplasms.
- Primary lung neoplasms are rather infrequent; but the incidence is much higher in dogs than in any other species.
- The common tumour is lymphocytoma

Pathology of Thoracic Pleura

- Pathologic involvement of the pleura is usually a secondary complication of some underlying disease.

1. Pleuritis

- **Pleuritis** (inflammation of the pleura) is the most common condition encountered and it is usually secondary to pneumonia.

- When copious exudation into the pleural sacs accompanies the inflammation, the lesion is commonly designated as **"pleurisy with effusion."**

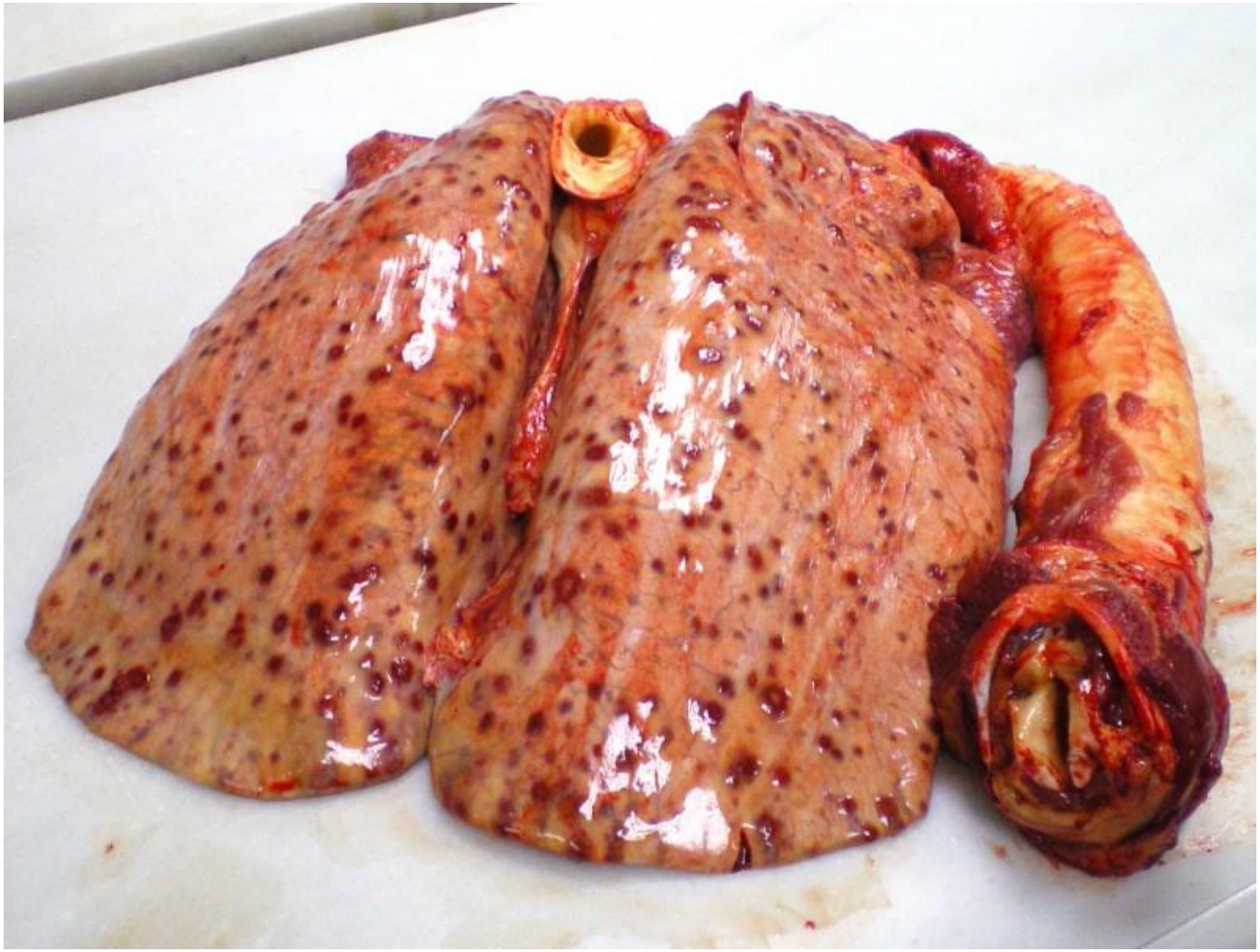
- primary pleuritis is associated with bovine blackleg, Glasser's disease, sporadic bovine encephalomyelitis, etc.

- The exudate is voluminous, the diaphragm is depressed, and the lungs are compressed and displaced to the caudodorsal portion of the thorax.

- Pasteurella multocida, Escherichia coli, and Sterptococci are present in most cases.

2. Abnormal contents

- Noninflammatory pleural collections **(fluid, blood, chyle, air)** may be observed. Hydrothorax, hemothorax, chylothorax, and pneumothorax.



➢ Air Sacculitis

- Inflammation of the air sacs is called air sacculitis
- Mainly caused by Mycoplasma gallisepticum, Escherichia coli
- Grossly: Pin point pale foci seen in early change
  - Cloudy –Mild inflammation
  - Thick – Moderate
  - Thick with whitish cheesy exudate – severe
  - Thick with yellowish cheesy material indicate complicated infection


QUESTIONS