RESPIRATORY SYSTEM L-3

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PNEUMONIA

- The pulmonary inflammatory response varies according to the nature of the causative agent, their distribution and their persistence.
- Pneumonia can be classified on
  - **Temporal basis as acute, subacute, or chronic**
  - **Aetiologic basis,** verminous, viral, bacterial, fungal etc.
  - **Morphologic basis**
    - Morphologically, there are two approaches.
    - **One approach** — type of inflammation.
      - **exudative** — catarrhal, fibrinous, suppurative, haemorrhagic, or necrotizing
      - **proliferative** — alveolar type II cells, fibroblasts, macrophages, and possibly additional elements.
    - **Second,** — morphologic
      bronchopneumonia, interstitial pneumonia, lobar pneumonia

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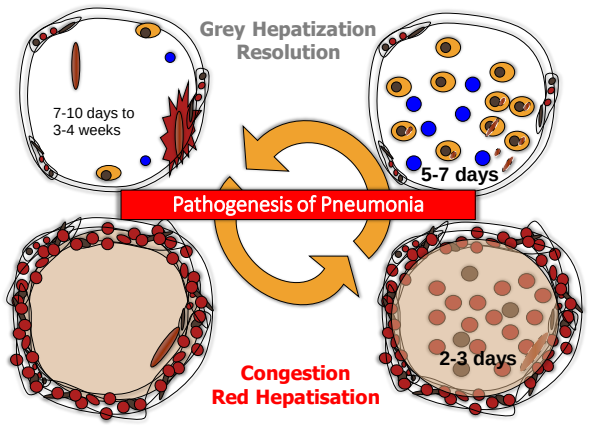
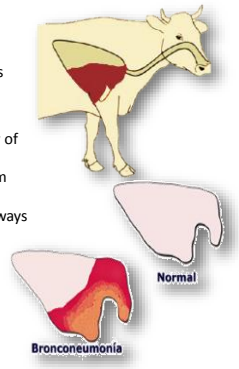


BRONCHOPNEUMONIA

- **Origin** – broncho-alveolar junction
- **Aerogenous** — cranioventral regions of the lungs
- The **bronchiolar-alveolar** junctions – greatest vulnerability
- There are three main reasons for the vulnerability of this region.
  - site of deposition of **small particles** (0.5-3.0 um diameter)
  - not protected by **mucous blanket** of larger airways nor an effective alveolar **macrophage system**.
  - "**funnel**" or "bottleneck," – trap the exudates
- Increased exposure — crowding of animals



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BRONCHOPNEUMONIA

- Many species of bacteria are involved
  - In **sheep and *cattle -- Pasteurella spp. & Corynebacterium pyogenes***
  - In **horses,** ***Streptococcus spp. and C. equi.***
- Lack of specific **immunity** to the organisms involved.
- Lack of **non-specific defense** mechanisms
- Most outbreaks are in **young,** especially after stress — shipping.
- **Mixing of animals** with different microbial floras
- **Predisposing causes –** debility, immunodeficiency, preexisting cardiopulmonary disease, and prolonged anesthesia or illness.

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BRONCHOPNEUMONIA

- Typical gross appearance is of **irregular consolidation in cranioventral region**
  - The **cranial and middle lobes –** in species having well-defined lobation (cattle).
  - **Consolidated lung** — dark red, gray-pink, gray,
  - Multiple, small, evenly spaced, **gray-white, bulging foci** separated by narrow, deep red zones**.**
  - **Pleuritis** may be present**.**
  - **Cut surface –** appears moist — mucopurulent or purulent material in small airways and foam may be in large airways.
  - Cut surface of fibrinous inflammation, — has a dull, dry appearance

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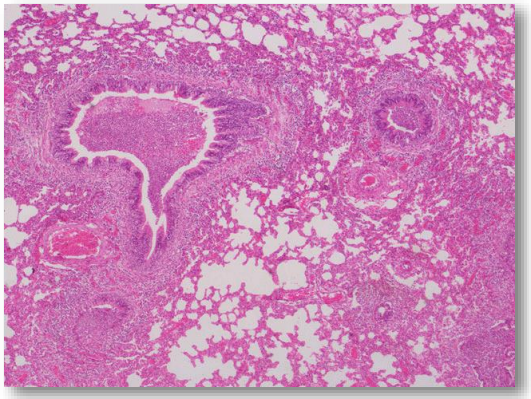

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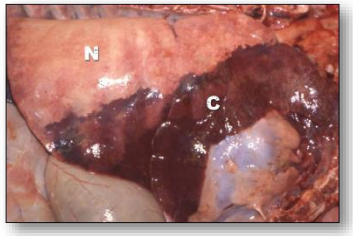
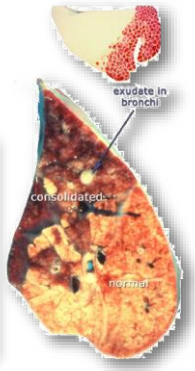
BRONCHOPNEUMONIA (Purulent)

- Firm Texture
- Cranioventral Consolidation
- Purulent exudate in bronchioles





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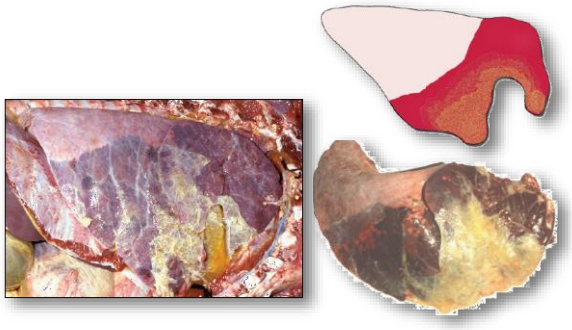
BRONCHOPNEUMONIA (Fibrinous)



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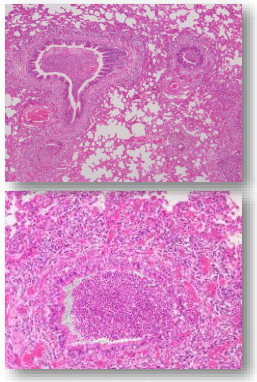
BRONCHOPNEUMONIA

Histologically

- In early bronchopneumonia, **bronchioles and immediately adjacent alveoli** are filled with neutrophils, and sometimes admixture of various amounts of cell debris, mucus, fibrin, and macrophages
- Bronchiolar epithelium — necrotic or hyperplastic
- Bronchi often show similar but usually less severe changes.
- Alveolar atelectatasis — edema or serofibrinous exudate, erythrocytes, macrophages,
- Congestion



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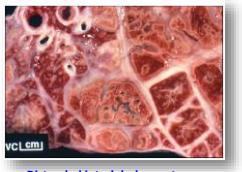
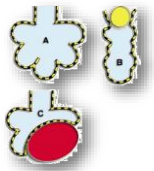
BRONCHOPNEUMONIA

Histologically

- Oedematous or serofibrinous fluid — may be in interstitial sites
  - The **red stage of consolidation — 2 or 3 days**
  - **gray appearance — within 5 to 7 days.**
    - Proliferation of alveolar type II cells can also occur during this period unless there is severe purulent or fibrinonecrotic inflammation.
  - Can begin to **resolve — in 7-10 days** — lung return to **normal 3-4 weeks**
- Severe bronchopneumonia — **death — by combination of hypoxaemia and toxaemia**



- Distended interlobular septa
- Thrombosis
- Coagulative necrosis



- Atelactasis: Atele (incomplete), Ectasia (dilatation)

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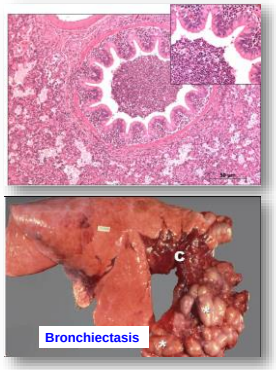
BRONCHOPNEUMONIA

Histologically

- **Complete resolution** can occur but require integrity of alveolar basement membranes, readily cleared exudate, and rapid killing of the infectious agent.
- **Atelectasis** is both a preliminary and a sequel to bronchopneumonia.
- Bronchopneumonia may become **chronic**.
- The lesions of chronic bronchopneumonia are those of chronic suppuration with fibrosis.
- Alveolar parenchyma is mainly atelectatic and fibrotic.



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LOBAR PNEUMONIA

- Lobar pneumonia — entire pulmonary lobes, or major portions of lobes
- Lobar pneumonias are **rapidly merging, fulminating bronchopneumonias**
- Lobar pneumonias have **close relationship to bronchopneumonias**
- Result of **overwhelming** spread of the inflammatory process
- caused by **virulent organism**
  - *Pasteurella haemolytica in cattle*
  - *Mycoplasma mycoides — in cattle and goats*
- **Infectious lobar pneumonias –** cranioventral lung.

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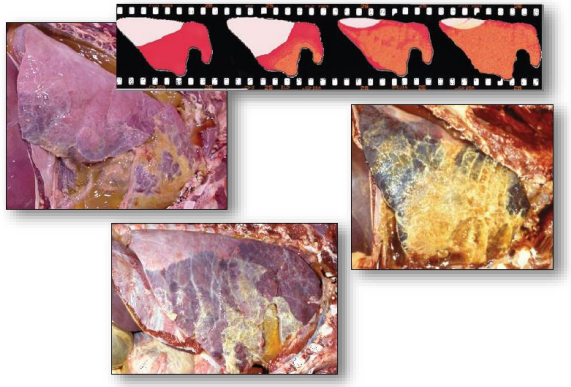

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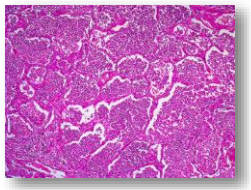
LOBAR PNEUMONIA

- **Aspiration pneumonia** — lateral zones of the caudal lobe of one side or the dorsal zones of both sides.
- Lobar pneumonias are **hemorrhagic, fibrinous, fibrinopurulent or necrotizing and sometimes gangrenous**
- **Gross appearance** — reddish black through deep red to reddish brown or gra**y**
- Prominent **distension of interlobular septa by serofibrinous exudate,**
- Development of **irregular, discrete zones of necrosis with swollen pale borders**

Aspiration pneumonia



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LOBAR PNEUMONIA

- The cut surface in early cases exudes bloody fluid and; later, fibrinous lobar pneumonias, becomes **grayish brown**; finely granular, dry, and friable.
- The interlobular septa, perivascular, peribronchial, and subpleural sheaths become widely distended by serofibrinous or fibrinous exudate within the loose connective tissue and especially in the lymphatic's

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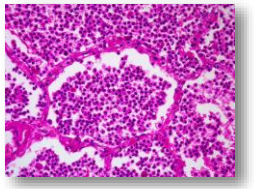
LOBAR PNEUMONIA

- **Small airways — purulent exudate or more fibrinous exudate**
- A feature common to most lobar pneumonias is massive **proliferation of bacteria**
- They are especially prominent within developing necrotic foci and tend to be concentrated close to the leukocytic boundary zones.
- The **complications of lobar pneumonia** are obviously more frequent and serious than those of the **less severe bronchopneumonias.**
- **Death is frequent**, usually with accompanying **pleuritis** and sometimes with **pericarditis**



Fibrinous Pleuritis

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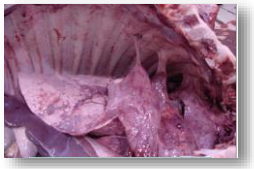
LOBAR PNEUMONIA

- If the animal survives, **resolution with some degree of scarring occur**
- **Peritonitis** may arise by haematogenous spread of the infection or direct extension from the pleura through the diaphragmatic lymphatics.
- Additional complications include toxaemic degeneration of parenchymatous organs, **endocarditis**, fibrinous polyarthritis, meningitis and hemolytic icterus.



Pleural adhesions

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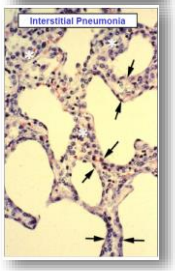
INTERSTITIAL PNEUMONIA

- Diffuse or patchy damage to **alveolar septa** is the essential feature of interstitial pneumonia
- Chronic inflammation — predominantly a **proliferative response** involving alveolar walls and supporting stroma
- Absence of lesions around small airways differentiates interstitial pneumonia from bronchopneumonia.
- **Grossly,**
  - **Whole lungs --- dorsocaudal regions**
- The alveolar septal damage is caused by
  - blood-borne insult in most instances
  - The inhale irritants – toxic gases or fumes – 100% oxygen
  - The pneumoconioses — inorganic dusts.



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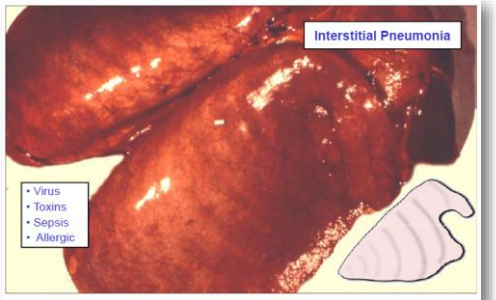

Acute, severe, diffuse interstitial pneumonia / Horse.

Note rib imprints on lung surfaces which result from lack of deflation at the time when the negative pressure is removed by opening the thorax.

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INTERSTITIAL PNEUMONIA

Histologically

- Range from acute to chronic.
- Predominantly within the alveolar walls and the interstitial tissues
- Alveolar type I cells eventually suffer most damage because of their poor reparative capacity and inability to regenerate.
- Flooding of alveoli with serofibrinous exudate, and congestion-and oedema of alveolar walls
- Replacement of necrotic type I epithelium by proliferation of type II cells
- Complete resolution
- Proliferation of alveolar type II cells marks the shift from the exudative to the proliferative stage of interstitial pneumonia.
- Fibrosis is a critical feature of the proliferative phase
- Interstitial fibrosis occurs more rapidly when there is considerable interstitial edema or serofibrinous exudation.

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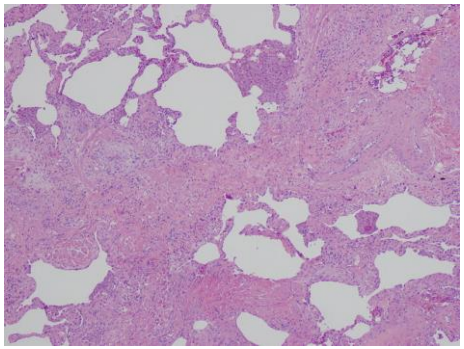

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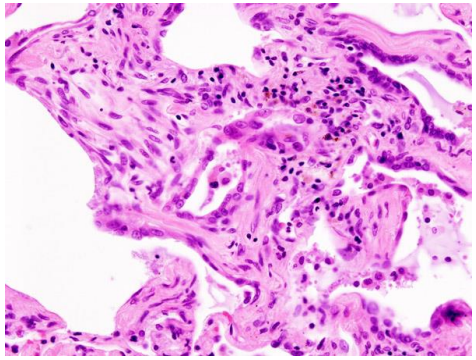

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INTERSTITIAL PNEUMONIA

Common Viral Infections Causing Interstitial Pneumonia

- **Cattle**
  - Bovine Respiratory Syncytial Virus
  - Parainfluenza-3 virus.
- **Small Ruminants**
  - Ovine Adenovirus,
  - Respiratory Syncytial Virus,
  - Lymphoid Interstitial Pneumonia (Maedi),
  - Caprine Arthritis Encephalitis (CAE).
- **Horses**
  - Equine Influenza,
  - Equine Virus Rhinopneumonitis (EVR),
  - Adenovirus,
  - Equine Viral Arteritis.

BRONCHO-INTERSTITIAL PNEUMONIA

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