Respiratory System Pathology

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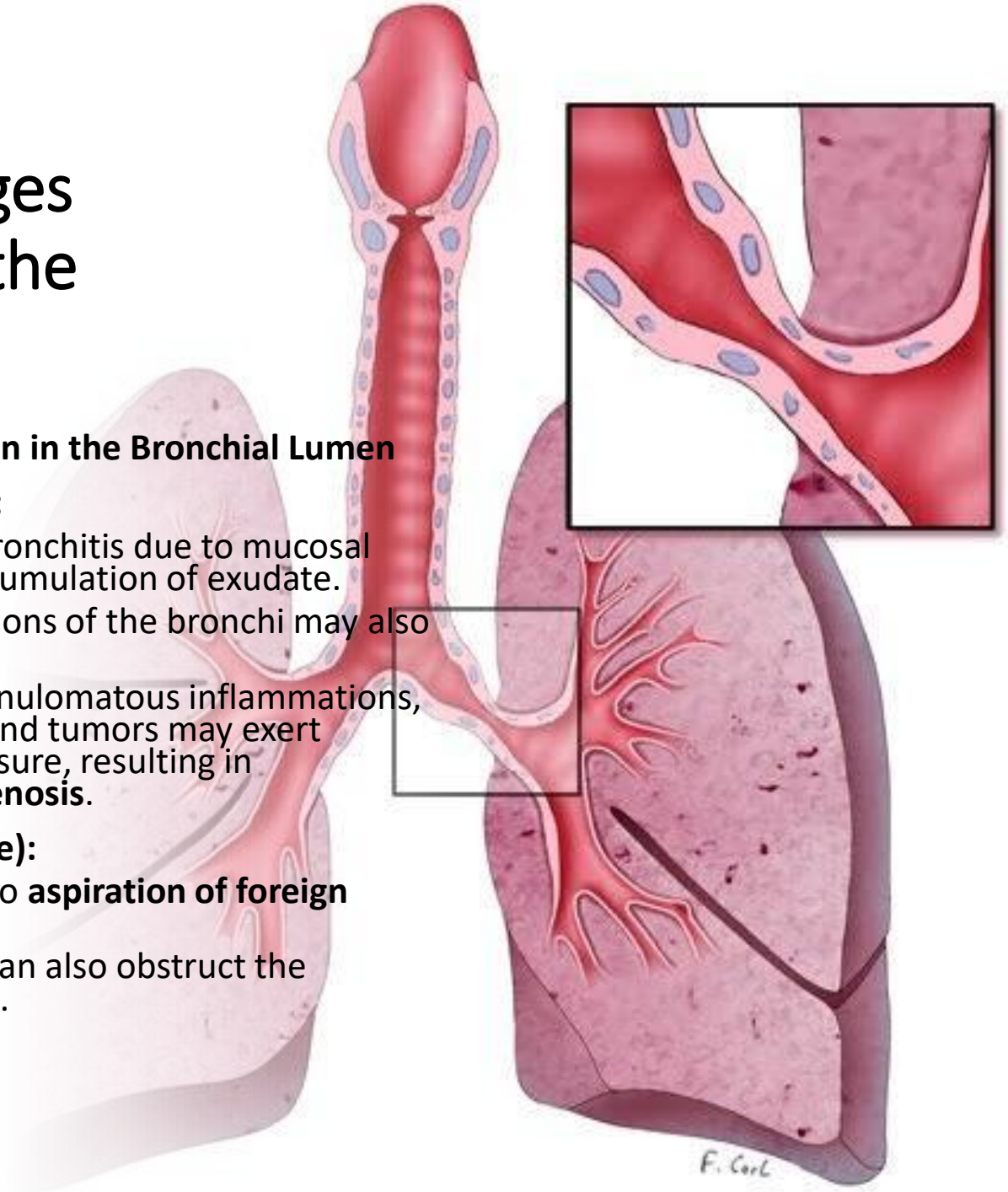
Bronchi and Bronchioles

Structural (Form) Changes Observed in the Bronchi

Stenosis and Obstruction in the Bronchial Lumen

- **Stenosis (narrowing):**
  - Occurs during bronchitis due to mucosal swelling and accumulation of exudate.
  - Spastic contractions of the bronchi may also contribute.
  - Surrounding granulomatous inflammations, peribronchitis, and tumors may exert continuous pressure, resulting in **compression stenosis**.
- **Obstruction (blockage):**
  - May occur due to **aspiration of foreign bodies**.
  - **Lung parasites** can also obstruct the bronchial lumen.



Structural (Form) Changes Observed in the Bronchi

BRONCHIECTASİS

Localized and permanent dilation of one or more bronchi.

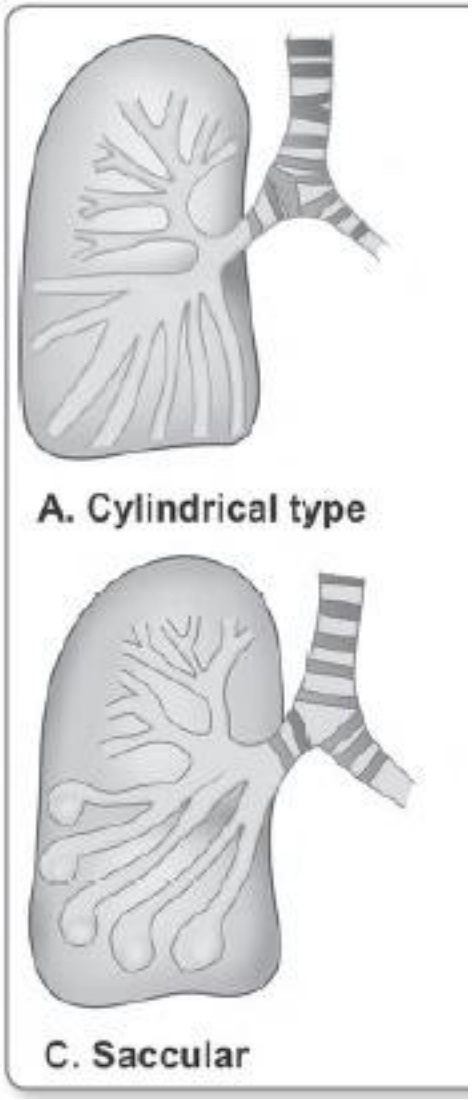
Causes:

- **Acquired:** Chronic inflammations (e.g, tuberculosis, mucosal infections, abscesses)
- Secondary to aspiration
- Anomalies such as *immotile cilia syndrome* → leads to exudate accumulation
- Traction by scar tissue in surrounding tissues
- **Congenital:** Very rare; due to congenital malformations.

BRONCHIECTASIS

Types:

- **Saccular:** Localized, pouch-like dilation; usually *follows focal necrotizing bronchitis*
- **Cylindrical:** Uniform dilation along the entire length of the bronchus; commonly seen in cattle *after chronic suppurative bronchitis*



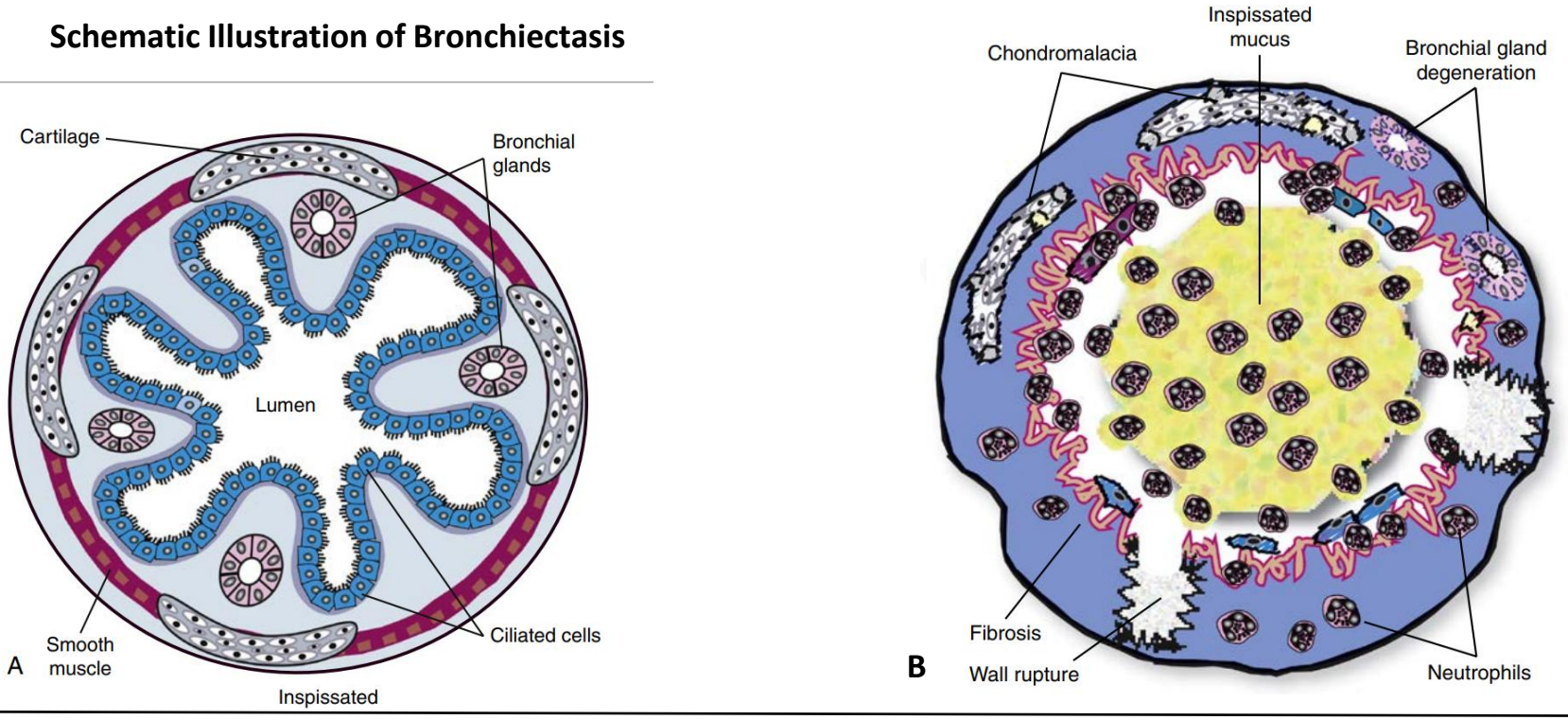
BRONCHIECTASIS

Pathogenesis

- Exudate accumulates in the bronchial lumen → neutrophil enzymes and free radicals *weaken the bronchial wall*
- **Atelectasis** in alveoli → **traction** on the bronchial wall leads to dilation
- **Impaired mucociliary clearance** → increased mucus and exudate → the process perpetuates

BRONCHIECTASIS

Schematic Illustration of Bronchiectasis



- Appearance of the Mucosa, Submucosa, Bronchial Glands, and Cartilage in a Normal Bronchus (A)

- Bronchiectasis: The affected bronchus is dilated and has lost the normal mucosal folds projecting into the lumen.
- Inflammation is characterized by mucosal loss, destruction of the bronchial wall, fibrosis, and atrophy of the cartilage and bronchial glands. (B)

Zachary, J. F., & McGavin, M. D. (Eds.). (2012). *Pathologic Basis of Veterinary Disease5: Pathologic Basis of Veterinary Disease*. Elsevier Health Sciences.

BRONCHIECTASIS

Gross Findings

- Dilated bronchi in the cranioventral regions.
- Filled with yellow-green pus; the parenchyma is atelectatic or fibrotic.
- Advanced cases: cystic appearance and honeycomb pattern.

BRONCHIECTASIS

Histopathological Findings

- Granulation tissue and fibrosis in the bronchial wall.
- Mucus, necrotic debris, inflammatory cells, and blood within the lumen.
- Mucosal ulceration, metaplasia/hyperplasia.
- Destruction of cartilage and glands.

BRONCHIECTASIS

Species-Specific Conditions Associated with Bronchiectasis

Cattle: Common following chronic bronchopneumonia.

Horse: Observed in the bronchiolitis-emphysema complex.

Dog: Seen in colisepticemia and immotile cilia syndrome.

Pig, Sheep, Goat: Associated with pulmonary parasites.

BRONCHIECTASIS

Outcomes / Complications

- The condition is chronic and does not resolve.
- Complications include: bronchopneumonia, bronchopleural fistula, septic thrombosis, hemorrhage, metastatic abscesses, and secondary amyloidosis.

Immotile Cilia Syndrome (Kartagener's Syndrome)

- Occurs in **humans, dogs, and rats.**
- **Ciliary defects** lead to *bronchiectasis, sinusitis, and infertility*.
- In dogs, approximately 50% exhibit the complete triad; sperm show impaired motility.
- Hereditary, autosomal recessive.

Oligospermia and azoospermia occur due to *defective cilia in the epididymis and vas deferens*.

Additionally, defective cilia lead to **rhinitis, bronchopneumonia, bronchiectasis,** and **hydrocephalus.**

This syndrome has been reported in English Pointers, Springer Spaniels, and Old English Sheepdogs.

It is considered an autosomal recessive disorder.

Bronchitis

- Bronchi and bronchioles are affected in conjunction with both upper respiratory tract inflammations and pulmonary inflammations.
- **Route of Infection:**
  - Primarily via the aerogenous route from the upper respiratory tract (descendens).
  - Less commonly by ascending spread (ascendens), particularly in verminous and granulomatous pneumonias.
- **Diseases Associated with Bronchitis:**
  - Tuberculosis → bronchitis accompanied by bronchopneumonia.
  - Pseudotuberculosis → pulmonary abscesses rupture into the bronchi → caseous bronchitis.
- **Other causes:** irritant gases, allergens, foreign bodies.
- **Predisposing factors: cold weather, environmental factors, weakened immunity.**
- **Course:** acute or chronic.
- Acute bronchitis is usually observed in conjunction with upper respiratory tract lesions or pneumonia.

Acute Bronchitis

- **Catarrhal, purulent, mucopurulent, fibrinous, ulcerative, fibrinonecrotic (diphtheritic), and occasionally granulomatous.**

Lesion characteristics:

- Vary depending on the underlying cause.
- Hypersensitivity → eosinophilic infiltration
- Viral infections → inclusion bodies
- In most cases, lesions are nonspecific and reflect the severity and duration of the condition.

Histopathological Findings

•**Epithelium:** Loss of cilia, degeneration, and necrosis → detachment from the basement membrane and desquamation into the lumen.

Goblet cells: Swollen and filled with mucus.

Other glandular cells: Affected in a similar manner.

Lamina propria: Edema, hyperemia, and mild neutrophilic infiltration.

Progression: Leukocytes migrate through the epithelium and accumulate in the lumen.

•**Repair:**

- In transient inflammation → repair occurs through the proliferation of basal and intermediate cells.
- In extensive damage → intact secretory cells also participate in regeneration through proliferation.

Catarrhal Bronchitis
It is the simplest form of inflammation.
Gross Findings

- Mucous exudate in the bronchial lumen
- Hyperemia and edema in the mucosa → swelling

Pathogenesis

•Acute, mild irritation leads to increased secretion by goblet cells, serous cells, and seromucous glands.

Species Variation

•The number of secretory cells and gland density varies among species, resulting in differences in the quantity and composition of secretions.

Histopathological Findings

•Ciliated epithelial cells are the most sensitive.

•The initial damage is observed in these cells.

Ulcerative and Fibrinonecrotic Bronchitis

Ulcerative Bronchitis

- Occurs in severe viral or bacterial infections.
- Extensive destruction of the epithelial layer → exposure of the lamina propria.
- Often develops as a sequel to chronic purulent bronchitis.

Fibrinonecrotic Bronchitis

- Thick, yellow membrane → exudate firmly adherent to the mucosa
- The reaction is severe → affects the larynx, trachea, and cranioventral regions of the lungs
- **Clinical sign:** expulsion of a bronchial cast-like mass through coughing
- **Mostly lethal**

Causative Agents:

- BRSV, IBR (Infectious Bovine Rhinotracheitis), rinderpest
- Mycotic bronchitis may also act as a primary cause.

- **Necrotic (Putrid) Bronchitis**
  - Characterized by necrosis of the mucosa.
  - **Causes:** severe parasitic invasion, aspirated foreign bodies, bronchiectasis.
  - Develops through the involvement of various microorganisms.
  - The bronchial mucosa appears greenish-brown and emits a foul odor.
- **Allergic (Asthmatic/Allergic) Bronchitis**
  - Clinically resembles asthma.May resolve if the underlying cause is eliminated.
- **Pathogenesis and Resolution**
  - Ciliated epithelium undergoes degeneration, detachment from the basement membrane, and exfoliation.
  - Subsequently, repair occurs through exudative inflammation followed by epithelial regeneration.

- **Inflammation of Large Bronchi – Healing**
  - **In most cases, healing occurs without scarring.**
  - Rarely, destruction of the bronchial wall, mild fibrosis, or granulation tissue polyps may be observed.
- **Small Bronchi and Bronchioles**
  - Their inflammation often **leads to bronchopneumonia**.
  - It is of greater significance because they are located within the lung parenchyma.
- **Characteristics of Large Bronchi**
  - Inflammation is usually limited and of lesser importance.
  - Due to their wide lumen, **exudate can be expelled via the cough reflex.**
  - Epithelium: pseudostratified, ciliated, supported by secretory cells.
  - The peribronchial connective tissue is abundant, limiting the spread of inflammation.
- **Difference from Small Bronchioles**
  - In small bronchi and bronchioles, the peribronchial connective tissue is sparse, allowing wider spread of inflammation.

Chronic Bronchitis

- Occurs **as a result of ongoing epithelial injury**.
- **Causes**: bacterial, parasitic, or allergic in origin.
- The relative importance of each cause varies among species.

Chronic Bronchitis

- **Chronic Catarrhal / Mucopurulent Bronchitis in Dogs**
  Mucus hypersecretion leads to persistent coughing.
  Commonly observed in small breed, obese dogs.
- **Postmortem findings:** excessive mucus or mucopurulent exudate.
  The bronchial mucosa appears thickened, hyperemic, and edematous.
- **Microscopic findings:** goblet cell hyperplasia, squamous metaplasia, infiltration of lymphocytes and plasma cells.
- **Complications:** bronchopneumonia (in ~25% of cases), alveolar atelectasis, bronchiectasis.
  In advanced cases, pulmonary hypertension may develop, resulting in cor pulmonale.

Chronic Bronchitis

Infectious Agents

- The most important pathogen in dogs is ***Bordetella bronchiseptica***.
- Following acute infection, normal defense mechanisms are impaired, leading to chronicity.
- **(IN HUMANS)**
- Commonly observed in cigarette smokers.
- Excessive mucus secretion results in persistent coughing.
- Chronic obstructive bronchitis may develop.

Chronic Bronchitis

Chronic Suppurative Bronchitis in Cattle

- Most commonly occurs as a sequel to bronchopneumonia.
- Often accompanied by **bronchiectasis**.
- **Bacterial agents:** *Actinomyces (Corynebacterium) pyogenes*, *Pasteurella* spp.

Allergic Bronchitis (Cats & Dogs)

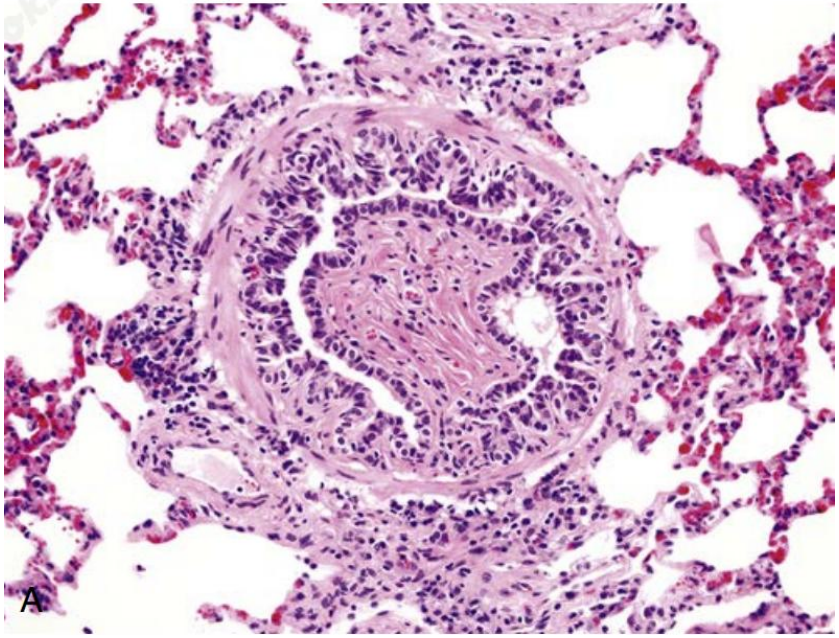
- **Clinical signs:** coughing, wheezing, dyspnea, and eosinophilia in blood or lavage fluid.
  Responds to medications such as sympathomimetics and corticosteroids.
- **Microscopy:** eosinophilic infiltration in the lamina propria and goblet cell hyperplasia.
  In chronic cases, eosinophils are the predominant inflammatory cells.

Acute Bronchiolitis

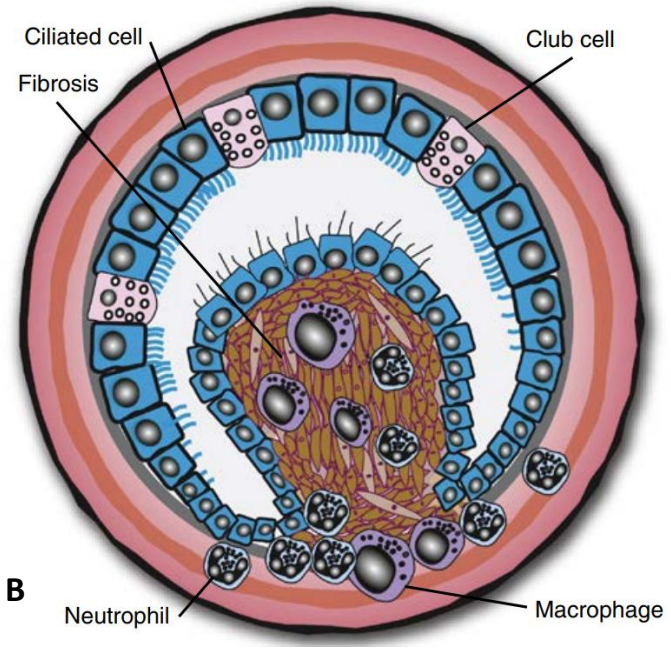
- **Initial response:** epithelial necrosis → desquamation of cells into the lumen → exposure of the basement membrane.
- **Regeneration:** renewal of ciliated and secretory cells through proliferation of Clara cells.
- **Severe cases:** fibrinous exudate → fibroblast infiltration → *bronchiolitis fibrosa obliterans* (organizing bronchiolitis).

- **Immun response**:
  - Neutrophils predominate in the initial phase, followed by mononuclear cells.
  - Eosinophils are prominent in allergic or parasitic conditions.
  - Peribronchiolar lymphoid hyperplasia is particularly associated with Mycoplasma infections.

Bronchiolitis Obliterans



Chronic inflammation of the bronchiolar wall characterized by granulation tissue formation, resulting in a nodular mass (center) that protrudes into the bronchiolar lumen, is lined by bronchiolar epithelium, and is firmly attached to the airway. H&E. (A)



The organized exudate, composed of connective tissue, macrophages, lymphocytes, and neutrophils, is adherent to the bronchiolar wall and covered by respiratory epithelial cells. (B)

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Chronic Bronchiolitis

- **The main finding** is hyperplasia and metaplasia of epithelial cells.
- **Squamous metaplasia:** Caused by continuous exposure to irritant gases such as SO_2, NO_2, and O_3.
- **Goblet Cell Metaplasia:**
  - Transformation of secretory Clara cells into mucus-producing goblet cells.
  - **Result:** sticky mucus → impaired mucociliary clearance → airway obstruction.Klinik sonuçlar:
- **Chronic Obstructive Pulmonary Disease (COPD):**
- Emphysema and atelectasis.
- *Heaves* in horses (chronic bronchiolitis-emphysema complex) is a typical example.

Heaves (Chronic Bronchiolitis-Emphysema Complex in Horses)

- Known among horse breeders as **heaves** or **broken wind**, the condition is now scientifically classified as **chronic obstructive pulmonary disease (COPD)**.
- *The most prominent finding is* **generalized chronic bronchiolitis**.
- **Emphysema**—characterized by airway dilation and tissue destruction—is less commonly observed.
- In some cases, alveoli may remain distended with air.

- *Emphysema is most often associated with bronchiolitis and typically localized in the cranial lobes*.

Heaves (Chronic Bronchiolitis-Emphysema Complex in Horses)

Gross Findings

In most cases, no gross lesions are observed in the lungs.

However, **emphysema** may develop in severe cases.

Histopathological Findings

- **Goblet cell metaplasia and increased mucus secretion.**
  In type I hypersensitivity reactions (anaphylaxis), under the influence of histamine, prostaglandins, and leukotrienes:
- There is an increase in **goblet cell numbers**.
- **Eosinophils** become the predominant cell population in bronchiolitis and are present at varying densities.
- Excessive mucus production occurs.
  - As mucus production increases, the number of eosinophils decreases (inverse relationship).
  - There is a marked increase in the number of mast cells around the bronchioles.

Heaves (Chronic Bronchiolitis-Emphysema Complex in Horses)

Etiology and Pathogenesis

- The exact cause is not fully understood.
  - Allergic, infectious, and toxic factors may play a role.
- The clinical onset following exposure to **moldy hay, bedding, persistent dust, or aerosols** suggests an **allergic response.**
  - **Example agents:** *Microspora faeni*, *Aspergillus fumigatus*, and hay dust.
- **Experimental findings:**
  - Pneumotoxins in the blood, particularly **3-methylindole**, cause damage to the bronchiolar epithelium in horses.

LUNGS

- **1. Postmortem Changes**
- **Postmortem hypostasis:** Accumulation of blood in vessels on the dependent side of the body, which may be confused with hyperemia and edema.
- **Differential Features Between Cruor (Postmortem Clot) and Thrombosis**
- **Findings of Aspiration and Autolysis**
- **Postmortem Emphysema:** Accumulation of putrefactive gases within the alveoli.

2. Kongenital Anomalies

•**Pulmonary Agenesis:** Complete failure of lung development; incompatible with life.

•**Accessory Lungs:** Presence of supernumerary lobes appearing as masses within the thoracic or abdominal cavity.

•**Bronchial Hypoplasia:** Underdevelopment of the bronchi; cartilage and muscular layers may be absent.

•**Congenital Lobar Emphysema:** Characterized by airway collapse and overinflation of the affected lobe.

•**Pulmonary Hypoplasia:** Commonly associated with diaphragmatic hernia.

•**Congenital Alveolar Dysplasia (in dogs):** Reduced number and developmental irregularity of alveoli.

•**Congenital Melanosis:** Pigmented foci of no functional significance, typically observed incidentally during slaughter, especially in pigs and cattle.

Immotile Cilia Syndrome (Ciliary Dyskinesia / Primary Ciliary Dyskinesia)

- It is a disorder associated with **recurrent chronic pneumonia and infertility in dogs**.
- Due to microtubular defects in ciliated epithelial cells—particularly within the respiratory epithelium and spermatozoa—cilia exhibit impaired motility.
- This defect compromises mucociliary clearance.
- As a result, chronic rhinitis, sinusitis, bronchitis, bronchiectasis, and pneumonia may develop.

Abnormalities in Lung Inflation

- There must be a balance between **air** and **capillary blood flow** in the lungs (ventilation-perfusion ratio).
- These two components must be in close contact with the alveolar membrane.
- **If this balance is disrupted:**
  - **Inadequate ventilation → Atelectasis (lung collapse)**
  - **Overinflation → Emphysema may develop.**

Atelectasis (Atelectasia pulmonum)

- **Atelectasis** is the complete or partial collapse of the lungs, meaning the alveoli are unable to expand with air.

Atelectasis (Atelectasia pulmonum)

- In **the fetal period**, the lungs contain no air; this condition is referred to as **fetal atelectasis**.
- Fetal lungs appear partially inflated due to the presence of **fetal lung fluid** within the bronchoalveolar spaces.
- This fluid normally reaches the oropharynx via the trachea and bronchial pathways and mixes with the amniotic fluid.

Atelectasis (Atelectasia pulmonum)

- **After birth**, the fluid is rapidly absorbed through the lymphatic vessels, replaced by air, and the alveoli expand.
- If **respiratory movements do not begin** in cases of *premature birth*, the lung tissue remains completely uninflated, a condition termed **diffuse fetal atelectasis**.

Postnatal Atelectasis

- Postnatal atelectasis is classified into two groups: **congenital (neonatal)** and **acquired**.

Congenital (Neonatal) Atelectasis:

- This refers to the failure of the lungs to adequately expand with air during the first breaths in newborns.
    - **Causes**
        - Airway obstruction
        - Aspiration of amniotic fluid at birth (meconium aspiration syndrome)
        - Hypoxia-induced damage to the respiratory center in the brainstem
        - Malformations of the central nervous system
        - Cerebral trauma during birth
        - Laryngeal dysfunctions
        - Congenital anomalies of the lungs and associated structures

Acquired Atelectasis

It is the **loss of air** in lung regions that were previously aerated.

- **Other names:** Alveolar collapse, postnatal atelectasis

Congenital Atelectasis

- It is mostly observed in a **focal (localized)** pattern. The animal breathes, but the pulmonary parenchyma does not become adequately aerated.
- Affected lung regions are **small**, **firm in consistency**, **lack crepitation**, and **sink in water**.
- **Macroscopically**, they appear **dark blue**, with **dilation of alveolar capillaries**.
- **Microscopically**, **interalveolar septa are engorged with dilated vessels**, and the **alveolar spaces appear narrowed**.
- Some alveoli may contain **flattened epithelial cells aspirated due to hypoxia** or **granular material of amniotic origin**.
- Even if the first breath occurs, if air does not remain within the lungs and **escapes**, **atelectasis may still develop**.
- **Congenital atelectasis due to surfactant deficiency** is known in neonates as **neonatal hyaline membrane disease** (*acute respiratory distress syndrome*).

Hyaline Membrane Disease (Neonatal Respiratory Distress Syndrome)

- It typically occurs in **premature neonates born to diabetic or alcoholic mothers**.
- A similar condition is observed especially in **foals**, and more rarely in **lambs, puppies, and piglets**.
- Foals and piglets often produce a **barking-like sound** while attempting to breathe; hence, the condition is colloquially referred to as **“barkers.”**
- Most surviving foals exhibit **hypoxia-induced brain damage**.
- These animals display **abnormal behaviors** and lack **normal fear responses**, leading to their description as **“wanderers.”**
- The disease is most commonly diagnosed during **necropsy**.

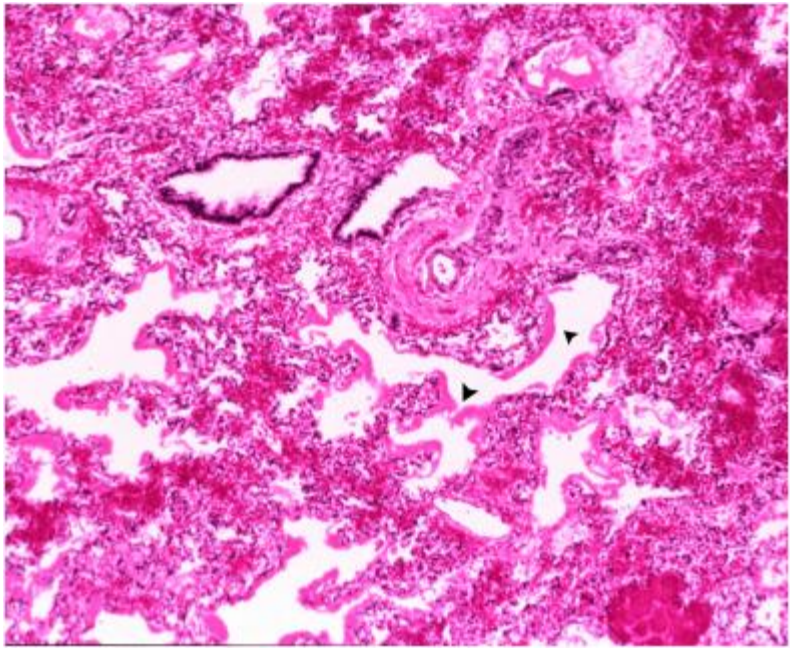
Hyaline Membrane Disease (Neonatal Respiratory Distress Syndrome)

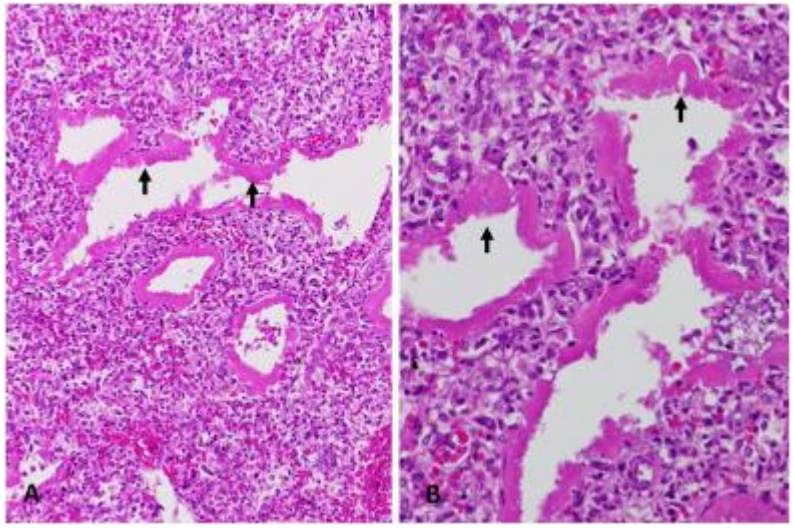
Gross Findings:

- Extensive areas of the lungs resemble fetal lung in appearance; they are heavy, meaty in consistency, airless, and have a bluish discoloration.
- There is generalized edema throughout the body.

Histopathological Findings

- **Interalveolar septa appear closely apposed**, while the **bronchioles are dilated**.
- Many **terminal and respiratory bronchioles** are lined with **thick, homogeneous, eosinophilic hyaline membranes**.
- The rapid formation of these membranes indicates that the animal **never actually inhaled**.





https://doi.org/10.1016/j.acpath.2024.100115

Surfactant Deficiency – Underlying Disorder

- The primary issue is the **inability to properly regulate surface tension within the alveoli**.
- Immaturity of **type II pneumocytes**, **insufficient synthesis** of surfactant, or **metabolic abnormalities** may lead to reduced surfactant activity.
- In piglets, the delayed maturation of type II cells is frequently attributed to **fetal hypothyroidism** and possibly to **hypoadrenocorticism**.
- **Additional factors that exacerbate the condition in the presence of surfactant deficiency include:**
    - Fetal Asphyxia
    - Aspiration of amniotic fluid
    - Reduced arteriolar blood flow in the lungs
    - Inhibition of surfactant activity by fibrinogen and serum components present in the edema fluid

Acquired Atelectasis – Collapse

- It occurs in adults and may arise from various causes.
- The most common form is **obstructive (resorptive) atelectasis**.

Obstructive Atelectasis

- It occurs due to obstruction of the bronchi or bronchiolar lumens by **exudate**, **foreign bodies**, **parasites**, or **tumors**.
- The severity depends on whether the obstruction is complete and on the presence of **collateral ventilation**.
- In **dogs and cats**, collateral ventilation is well developed; therefore, **lobar or segmental bronchial obstruction** is typically required for atelectasis to occur.

Acquired Atelectasis – Collapse

In **cattle and sheep**, **collateral ventilation is poorly developed**; therefore, even a **minor obstruction** of a bronchus or bronchiole may be sufficient to cause atelectasis.

- **Horses exhibit an intermediate pattern** between dogs and cattle.
- Following obstruction, the air distal to the blockage is **resorbed within 1–2 days**.
    - Sequence of Resorption : **O_2 → CO_2 → N_2**.
- Clinically
    - In **lambs and calves**, it is commonly observed in the **anterior lobes** during **enzootic pneumonia**, often accompanied by **peribronchitis nodosa**.
    - In **cattle and sheep**, it may also develop in the **diaphragmatic lobes** as a result of **verminous bronchitis**.

Post-Bronchitic Obstructive Atelectasis

- **Local anoxia develops in the alveolar wall.**
- **Increased permeability** of alveolar capillaries allows **plasma fluid to leak into the alveolar spaces**, resulting in **pulmonary edema**.
- In affected areas, **tissue consistency increases**, while the **volume appears normal or slightly enlarged**.
- On cut surface, **fluid oozes from the tissue**.

Microscopically, a **frothy mixture of edema fluid and gas** is observed within the alveolar spaces.

- **Alveolar epithelial cells appear swollen**.
- **Emphysema may develop around these regions**.
- These areas are frequently **complicated by bronchopneumonia**.

Compression Atelectasis (Pressure Atelectasis)

- **Causes:** External compression of the lungs by **fluid, gas, or masses**, such as **pneumothorax, hydrothorax, hemothorax, chylothorax, empyema, intrathoracic tumors**, or **meteorism**.
- **Macroscopic findings:**
  - The affected portion of the lung appears **pale**, **collapsed**, and **firm in consistency**.
  - On cut surface, the tissue is **gray-red**, and the **pleura is thickened and folded**.
  - If there is **no pleural fibrosis or adhesion**, the **entire lung may collapse**.
- **Microscopic findings:**
  - **Alveolar walls appear flattened and closely apposed**.
  - **Bronchial and bronchiolar walls** are also approximated.
  - **Lumens are empty**.
- **Complication:** Surrounding areas may exhibit **emphysema** and **edema**.

Hypostatic Atelectasis

- **Causes:**
  - **Prolonged recumbency** (particularly in large animals).
  - May develop **following extended anesthesia**.
- **Pathogenesis:**
  - Air–blood balance is disrupted, and respiration becomes difficult.

    Drainage is insufficient, and surfactant activity decreases.

    Lung tissue remains in its altered state for a long time.
- **Results:**
  - Mild inflammation and fibrosis develop later.
  - In sheep, sharply demarcated, band-like or lobular atelectasis may be observed in the cranioventral regions during slaughter.
  - It is mostly due to obstruction of the bronchi and bronchioles by exudate.
  - In some cases, no obstruction is detected, and the cause remains unexplained.

Massive Atelectasis (Massive Lung Collapse)

- **Causes:**
  - Mostly develops following pneumothorax.
  - May be observed in cats and dogs that remain on mechanical ventilation in intensive care for extended periods, receiving 80–100% oxygen.
- **Pathogenesis:**
  - Oxygen is completely resorbed into the tissues.
  - No gas is present in the lungs upon postmortem examination.
- **Macroscopic findings:**
  - The lungs are completely contracted.
  - They are dark red in color and firm in consistency.
  - Blood oozes from the cut surface.
- **General Features:**
  - Fetal atelectasis is diffuse, whereas obstructive atelectasis presents a lobular pattern.
  - Other types exhibit variable features between these two forms.
  - In all forms of atelectasis, the lungs become predisposed to secondary infections.

Consequences of Atelectasis

- **Short-term atelectasis is** generally **completely reversible.**
- **In long-standing cases, affected show induration** and **collapse.**
- **Complications:**lung regions
  - **Edema**
  - **Carnification** (replacement of lung tissue by fibrous connective tissue)
  - Development of **pneumonia**
- **In cases of circulatory disturbance, cor pulmonale** (right-sided heart failure) may occur.
- **If a sufficient amount of functional lung tissue is** affected**, death** may result.
- **If the animal survives,** most of the resulting changes may be **reversible.**

Summary of Atelectasis (Pulmonary Collapse)

- **Atelectasis** is the complete or partial collapse of the lungs, meaning the alveoli are unable to expand with air.

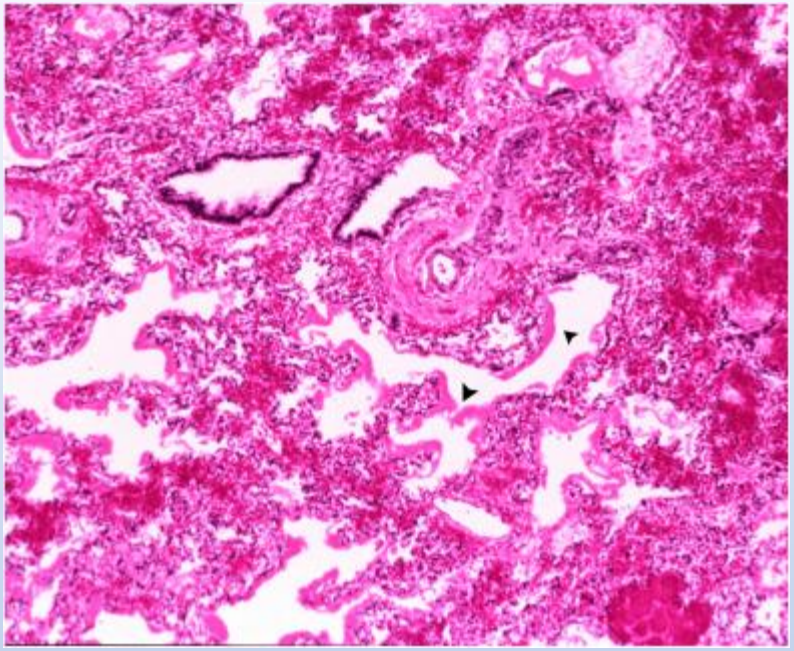
Summary of Atelectasis (Pulmonary Collapse)

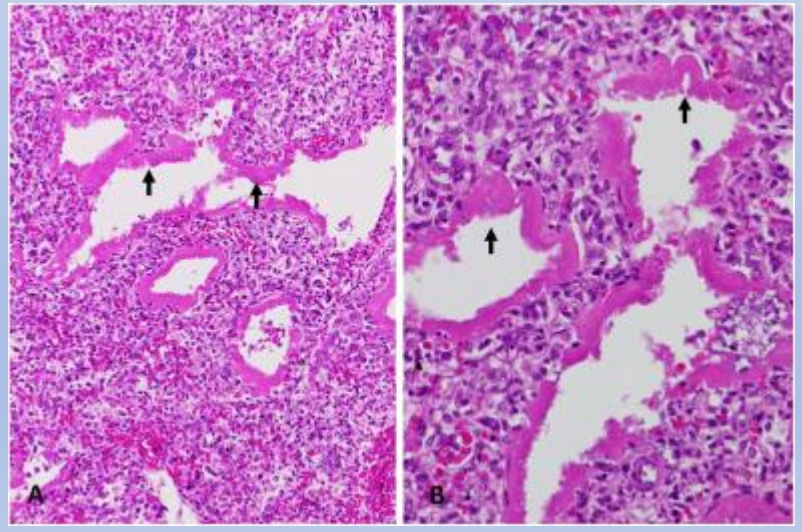
A. Fetal (Congenital) Atelectasis

- Occurs before the first breath; lungs contain **fetal lung fluid** instead of air.
- In premature neonates or those with **brainstem hypoxia**, alveoli remain unexpanded.
- Lungs are **dark, firm, airless**, and **sink in water**.
- Microscopically: alveoli collapsed, capillaries dilated, epithelial cells flattened.

Special Form:

- **Hyaline Membrane Disease (Neonatal Respiratory Distress Syndrome)**
  - Due to **surfactant deficiency** (immature type II pneumocytes).
  - Common in **premature foals, piglets, lambs, and human infants**.
  - Lungs are heavy, meaty, bluish, and airless.
  - **Hyaline membranes** line the terminal bronchioles and alveoli.





https://doi.org/10.1016/j.acpath.2024.100115

Summary of Atelectasis (Pulmonary Collapse)

B. Acquired (Postnatal) Atelectasis

- Occurs after birth — in previously aerated lungs.

Main Types:

- **Obstructive (Resorptive) Atelectasis**
  - Caused by **bronchial obstruction** (exudate, parasites, tumors, foreign material).
  - Air distal to the blockage is absorbed → collapse.
  - Common in **enzootic pneumonia** (lambs, calves) or **verminous bronchitis**.
  - Affected area: firm, airless, reddish-gray.
  - Microscopy: edema fluid + gas bubbles, swollen epithelium, sometimes surrounded by emphysema.
- **Compression (Pressure) Atelectasis**
  - Due to **external pressure**: pleural fluid, gas, tumors, or abdominal distension.
  - Lungs appear **pale, collapsed, firm**, with **thickened pleura**.
  - Microscopically: flattened alveolar walls; empty lumens.
  - Neighboring areas may show **emphysema or edema**.
- **Hypostatic Atelectasis**
  - From **prolonged recumbency** or **anesthesia**, especially in large animals.
  - Poor ventilation and drainage → mild inflammation and fibrosis.
  - Seen as sharply demarcated, band-like collapse areas (often cranioventral).
- **Massive Atelectasis**
  - Usually after **pneumothorax** or prolonged **mechanical ventilation** (80–100% O_2).
  - Lungs completely airless, dark red, and firm; blood oozes on section.

Summary of Atelectasis (Pulmonary Collapse)

Consequences

- **Reversible** if short-term.
- Chronic cases → **induration, fibrosis, pneumonia**, and occasionally **cor pulmonale** (right-sided heart failure).
- Severe collapse → death from respiratory failure.

Emphysema (Emphysema pulmonum)

- Emphysema is the **overdistension of the lungs with air**.
- It primarily arises from the **abnormal and permanent dilation of airspaces** located at the terminal ends of the bronchioles.
- During this process, **structural alterations also occur in the alveolar walls**.
- Emphysema is classified based on its **location**, **extent**, and **duration**.
- According to its ***anatomical location***, it is divided into two types:
  - **Alveolar (vesicular) emphysema**
  - **Interstitial emphysema**

Types of Emphysema

- **Alveolar emphysema:** Air accumulates within the alveoli.
- **Interstitial emphysema:** Air is located in interlobular, subpleural, and other interstitial regions of the lung. In general, the term **"emphysema"** refers to **alveolar emphysema**.

Emphysema by Duration

- **Acute emphysema:** Temporary and **reversible** in nature.
- **Chronic emphysema:** Characterized by **structural alterations in lung tissue**; it is **irreversible** and **destructive**.

- The increased air may be localized to one or several regions of the lung, which is referred to as **partial emphysema**, or it may involve the entire lung, in which case it is termed **diffuse** or **universal emphysema**. Based on these criteria, emphysema in the lung is classified as:
- **I. Alveolar (Vesicular) Emphysema**
- **a. Acute Partial Alveolar Emphysema**
- **aa. Acute Diffuse Alveolar Emphysema**
- **b. Chronic Partial Alveolar Emphysema**
- **bb. Chronic Diffuse Alveolar Emphysema**
- **II. Interstitial Empyhsema**

a. Acute Partial Alveolar Emphysema

- **Obstructive emphysema** develops due to **temporary narrowing (stenosis) or blockage** of small bronchi and bronchioles by **mucus or inflammatory exudate** (as seen in bronchitis or bronchiolitis).
- With forceful inspiration, the obstruction may be overcome, allowing air to reach the distal alveoli.
- However, due to the weaker pressure during expiration, the air cannot be fully expelled.
- This leads to **air trapping**, **overdistension**, and **septal ruptures** in bronchioles and alveoli.
  In some cases, **complete obstruction** of bronchi and bronchioles results in **focal atelectasis**.

Acute Diffuse Alveolar Emphysema

- It is fundamentally caused by **airway (valvular) stenosis**.
- It typically occurs in **fatal cases of asphyxiation** and in **anaphylactic shock** (due to bronchial spasm).
- It may also develop in **bronchiolar asthma**, **aspiration of gastric contents**, and **laryngeal stenosis** (caused by foreign bodies, edema, spasm, etc.).
- Lungs with **widespread emphysema** do not collapse; they appear **pale pink or whitish-yellow**, and the **cut surfaces are dry and have a doughy, inflated consistency**.

b. Chronic Partial Alveolar Emphysema

- It is usually **obstructive emphysema** resulting from **valvular stenosis**.
- Due to **loss of parenchyma**, the condition is **irreversible**.
  The coexistence of **pale emphysematous areas** and **dark atelectatic regions** gives a characteristic **"chessboard" appearance**.
- **Bullous emphysema** frequently develops at the **lung margins** and **subpleural regions**.
- These **bullae** are **larger than 1 cm**, and appear **grayish-white** due to **pressure-induced anemia**.
- In bronchiolar and pulmonary inflammation, **collateral emphysema** may be observed in alveoli surrounding the inflamed areas.
- In **cattle, sheep, and pigs**, it typically occurs in the **diaphragmatic lobes** during **pulmonary strongylosis**.

bb. Chronic Diffuse Alveolar Emphysema

- It is particularly observed in **heaves (recurrent airway obstruction)** in **horses**.
- It may also develop following **resection of a lung lobe** or when a lobe becomes **non-functional**.
- **Compensatory emphysema** occurs as the **remaining lung tissue expands** under alveolar pressure to fill the **thoracic cavity**.
- This type does **not involve destructive emphysema** or **associated complications**.

Senile emphysema

- This type is common in **humans** and is characterized by **diffuse emphysema**.
- It results from the **partial loss of alveoli**, **alteration of alveolar architecture**, and **dilation of alveolar ducts and respiratory bronchioles** within the pulmonary parenchyma.
- It is accompanied by **cartilage and muscle atrophy in the bronchial wall**, **reduction of elastic fibers**, and **perivascular and bronchiolar fibrosis**.
- At necropsy, the lungs appear **voluminous and pale**, and they do not fully collapse.
- Lungs with diffuse emphysema **fill the thoracic cavity**, and **rib imprints** are often visible on their surfaces.
- The **heart may be completely surrounded by the enlarged lungs**.

II. Interstital empyhsema

- It occurs when air enters the **interlobular and subpleural connective tissue**.
- Small **vesicles** are seen in these regions, while larger accumulations form **bullae**.
- Air may reach the lymph nodes, mediastinum, and even the subcutaneous tissue via lymphatic and vascular spaces.
- *It is most commonly observed in cattle, developing as a result of overdistension and rupture of alveoli.*
- **Causes:** Acute or chronic obstructive emphysema, pulmonary strongylosis, chronic interstitial pneumonia, severe coughing, rib fractures, and foreign bodies.

Microscopic examination: Alveoli and alveolar ducts are **dilated and stretched**; the lumens are **filled with air and appear empty**.

- Interalveolar septa are thinned, capillaries are compressed, and do not contain erythrocytes.
- Alveolar epithelial cells appear slender and elongated, resembling endothelial cells.
- When septa rupture, adjacent alveoli coalesce, resulting in the formation of **bullous emphysema.**
- The edges of the ruptures are irregular, and small bulb-like structures are observed in these areas.
- Artifact-related tears, on the other hand, are smooth and regular.
- **In interstitial emphysema**, air accumulates within the septal tissue, leading to destruction of alveolar structures.
- In chronic emphysema, structural changes are also present in addition to these findings.

- In domestic animals, the structure of the lungs varies among species, and therefore, the types of emphysema also differ accordingly.
- **Alveolar emphysema** is observed in **all domestic animal species**.
- **Interstitial emphysema**, however, develops **only in cattle**, as collateral ventilation is absent in this species, allowing pressure-induced rupture and the passage of air into the interstitium.

Summary of Emphysema (Pulmonary Emphysema)

- **Definition:**
  Overdistension of the lungs with air, caused by abnormal and permanent **dilation** of airspaces at the ends of bronchioles, with destruction of alveolar walls.

Summary of Emphysema (Pulmonary Emphysema)

Classification

A. Based on Location

- **Alveolar (Vesicular) Emphysema:** Air accumulates within alveoli.
- **Interstitial Emphysema:** Air escapes into interlobular, subpleural, or mediastinal connective tissue.

In general, “emphysema” refers to alveolar emphysema.

Summary of Emphysema (Pulmonary Emphysema)

Classification

B. Based on Duration

- **Acute:** Temporary and reversible.
- **Chronic:** Irreversible and destructive, with permanent structural damage.

C. Based on Extent

- **Partial (Localized):** Involves one or more regions.
- **Diffuse (Generalized):** Involves the entire lung.

Summary of Emphysema (Pulmonary Emphysema)

Types

Alveolar Emphysema

- **Acute Partial:**

  Due to temporary obstruction by mucus or exudate → air trapping → alveolar distension and rupture.

  In severe cases, complete obstruction causes focal atelectasis.

- **Acute Diffuse:**

  Caused by airway stenosis (asphyxia, anaphylaxis, asthma, aspiration, laryngeal edema).

  Lungs are pale, dry, fail to collapse, and have a doughy consistency.

Summary of Emphysema (Pulmonary Emphysema)

Types

Alveolar Emphysema

- **Chronic Partial:**

  Often due to valvular stenosis.

  Irreversible loss of parenchyma → mixed pale (emphysematous) and dark (atelectatic) areas = “chessboard” pattern.

  Bullous emphysema develops subpleurally (bullae >1 cm).

  May accompany bronchitis, pneumonia, or parasitic infections (e.g., strongylosis).

- **Chronic Diffuse:**

  Common in horses with **heaves (recurrent airway obstruction)** or after lobectomy.

  The remaining lung expands compensatorily (non-destructive).

Senile Emphysema:
Seen in aged humans; diffuse loss of alveoli, elastic fibers, and bronchiolar support → lungs enlarged, pale, with rib impressions.

Summary of Emphysema (Pulmonary Emphysema)

Interstitial Emphysema

- Air penetrates interlobular and subpleural connective tissue → **vesicles or bullae.**
- May extend to mediastinum, lymph nodes, or subcutis.
- Seen mainly in **cattle** (lack of collateral ventilation).
- Causes: chronic bronchitis, strongylosis, interstitial pneumonia, coughing, rib fractures, foreign bodies.

Summary of Emphysema (Pulmonary Emphysema)

Gross and Microscopic Findings

- **Gross:** Lungs pale, spongy, fail to collapse; in diaphragmatic lobes diaphragm appears flat.
- **Microscopic:**
  - Alveoli and ducts dilated and empty.
  - Septa thin; capillaries compressed, without RBCs.
  - Epithelial cells elongated.
  - Septal rupture → coalescent alveoli → bullae formation.
  - Interstitial air accumulation damages septa.
  - Chronic cases show fibrosis and structural remodeling.

Results

- The outcome of emphysema depends on its extent. If widespread, death may occur due to **hypoxia** or **anoxia**.
- **Bullous** and **interstitial emphysema** may result in pneumothorax.
- When circulation is affected, may develop **cor pulmonale**
- Emphysema is an extremely important **primary disease** in humans.
- In human emphysema, there is **abnormal and permanent enlargement of airspaces extending to the terminal bronchioles**, accompanied by **destruction of the alveolar walls** (**alveolar emphysema**).
- It must be distinguished from **simple enlargement of airspaces without destruction**, which may be **congenital** (e.g., in **Down syndrome**) or **age-related** (referred to as **senile lung**, sometimes mistakenly called **senile emphysema**).
- The **pathogenesis of emphysema in humans remains controversial**.

Pasture or Fog Emphysema (Fog Fever, Acute Bovine Pulmonary Emphysema and Edema)

•In Turkey, the condition is known as **pasture/fog emphysema**; in the **United Kingdom**, it is referred to as **fog fever**; and in the **United States**, it is called **toxic atypical interstitial pneumonia (AIP)**.
•The disease is **pasture-associated** and typically occurs in **adult cattle** during **early autumn (August–September)** after consuming **fresh regrown green forage** following harvest.

•**L-tryptophan** present in the pasture plays a key role in the pathogenesis.
•**L-tryptophan** is metabolized in the **rumen** to **3-methylindole**, which then enters the bloodstream and reaches the lungs.
•In the lungs, **non-ciliated bronchiolar epithelial cells (Clara cells)** convert this compound into **pneumotoxic metabolites**

- The initial lesion is **severe necrosis of bronchiolar epithelial cells and type I pneumocytes**.
- Subsequently, structural changes occur in the **alveolar walls and lumens**.
- The condition has been experimentally reproduced in **cattle, sheep, and goats** by administration of **DL-tryptophan**.
- It is thought that, in addition to intoxication, an **allergic reaction** may also play a role in the pathogenesis

- **Clinical Findings:** Within two weeks following a change in pasture, affected animals exhibit respiratory distress, including expiratory dyspnea, mouth breathing, and emphysema extending from the lungs to the back.

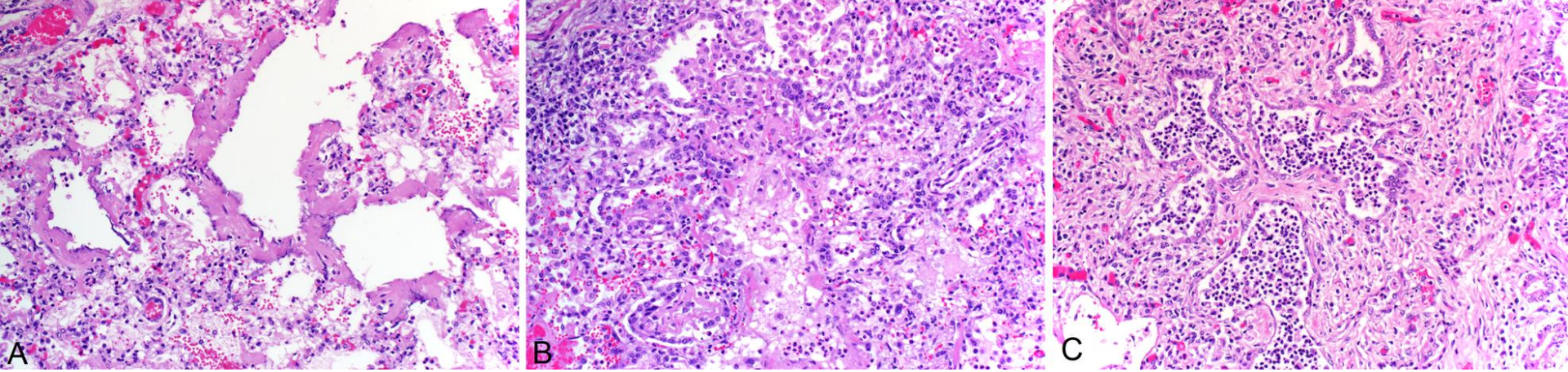
Prognosis:

- In acute cases, **morbidity ranges from 10% to 60%**, and **lethality from 25% to 50%**.
- In severe cases, **death occurs 2–4 days after the onset of symptoms**.
- **Mild cases generally recover**.

Gross Findings

- Large amounts of **frothy edema fluid** are present in the trachea and bronchi.
- The mucosa beneath the edema fluid shows **congestion**, along with **petechial** and **ecchymotic hemorrhages**.
- The lungs exhibit **severe interstitial emphysema**, usually accompanied by *bullae and pulmonary edema*.
- Subcutaneous emphysema may also be observed.
- The lungs are enlarged and appear **purplish-brown** due to parenchymal congestion and edema, and they do not collapse.
- Lobes are homogeneously affected, and the cut surfaces are moist.
- Lesions show a **sublobular or lobular distribution** and typically display **diffuse involvement in the dorsocaudal region**.

Haydock, L. A. J., Fenton, R. K., Sergejewich, L., Squires, E. J., & Caswell, J. L. (2022). Acute interstitial pneumonia and the biology of 3-methylindole in feedlot cattle. *Animal Health Research Reviews*, *23*(1), 72–81. doi:10.1017/S1466252322000020



- **Acute Fatal Cases:**
  - Edema and **eosinophilic hyaline membranes** in the alveoli.
  - Alveolar walls are thickened and edematous, with abundant eosinophils.
  - Mild hyperplasia of type II pneumocytes is present.
- **Subacute Cases:**
  - Marked **hyperplasia of type II alveolar epithelial cells** (epithelialization, fetalization, adenomatosis).
  - The hyperplasia is **persistent.**
- **Recovery Phase:**
  - Eosinophilic and mononuclear cell infiltration is observed in peribronchiolar regions.
  - **Residual fibrosis** may be present.

Haydock, L. A. J., Fenton, R. K., Sergejewich, L., Squires, E. J., & Caswell, J. L. (2022). Acute interstitial pneumonia and the biology of 3-methylindole in feedlot cattle. *Animal Health Research Reviews*, *23*(1), 72–81. doi:10.1017/S1466252322000020

Circulatory Disturbances

Ischemia – Anemia

- **Causes:** General: Blood loss, trace element deficiencies, systemic anemias, localized: Pneumothorax, hydrothorax, tumor, abscess, embolism, emphysema, fibrosis

•**Gross Findings:** The lung appears pale, whitish-pink, and reduced in size.

Hyperemia – Congestion

•**Active hyperemia:** Acute infections, gas/irritant exposure, exudate drainage in pleuritis, high altitude, trauma, shunts, valvular stenosis.

•**Passive (acute) hyperemia:** Left-sided heart failure; the lung is heavy, dark blue, with bloody, frothy fluid on cut surface, and edema is common.

- **Histopathological Findings:** Alveoli contain eosinophilic edema fluid, erythrocytes/macrophages; capillaries are dilated, septa are thickened, and lymphatic vessels are enlarged.

•**Passive (chronic) hyperemia:** Mitral stenosis/insufficiency. The lung is firm, dry, and does not collapse. Fibrosis and sclerosis develop. Heart failure cells (hemosiderin-laden macrophages) are typical. Cor pulmonale may be observed.

Hemorrhage

- **Perrhexis:** Trauma (rib fracture, stab wound, gunshot, aneurysm rupture).
- **Diapedesis:** Toxicosis, infection, hypoxia, asphyxia, brain trauma.

- **Gross Findings:** Petechiae, ecchymoses, diffuse hemorrhages; dark red areas and subpleural foci.
- **Histopathological Findings:** Erythrocytes, hemosiderin-laden macrophages.
- **Special type:** Exercise-induced hemorrhage (horses, racing).Epistaxis occurs; seen in over 75% of cases by endoscopy, with subpleural foci in the dorsocaudal region macroscopically.

Pulmonary Edema

1. Definition and Basic Mechanism

- Pulmonary edema is the leakage of the fluid component of blood into the **alveoli** and/or **interstitial tissue**.
- The **Starling equilibrium** is the determining factor:
  - Capillary–interstitial hydrostatic/osmotic pressure gradient,
  - Capillary surface area and permeability,
  - Alveolar epithelial permeability,
  - Lymphatic drainage capacity,
  - Surface tension (surfactant).
- Under normal conditions, the alveoli do not fill with fluid because:
  - The **alveolar epithelium is less permeable** than the endothelium.
  - **Interstitial pressure is lower** than intra-alveolar pressure, and fluid is **removed via lymphatic drainage**.

Pathogenesis and Classification

Pulmonary edema occurs through **two main mechanisms**:

A. Hemodynamic Edema (Transudate)

•**Reason:** hydrostatic pressure↑ or osmotic pressure ↓.

•**Characteristic:** low protein content, clerarer fluid.

•**Main Causes:**

- Left-sided or bilateral heart failure (↑ left atrial pressure→ Cardiogenic edema).
- Mitral stenosis / insufficiency
- Hypoalbuminemia (liver diseases, nephrotic syndrome, protein-losing enteropathies)
- Impaired lymphatic drainage (tumor invasion)
- Hypervolemia (excessive fluid transfusion)

B. Permeability Edema (Exudate)

•**Reason:** Capillary endothelial and type I pneumocyte injury, inflammatory mediators.

•**Characteristic:** High protein content, darker eosinophilic fluid.

•**Main Causes:**

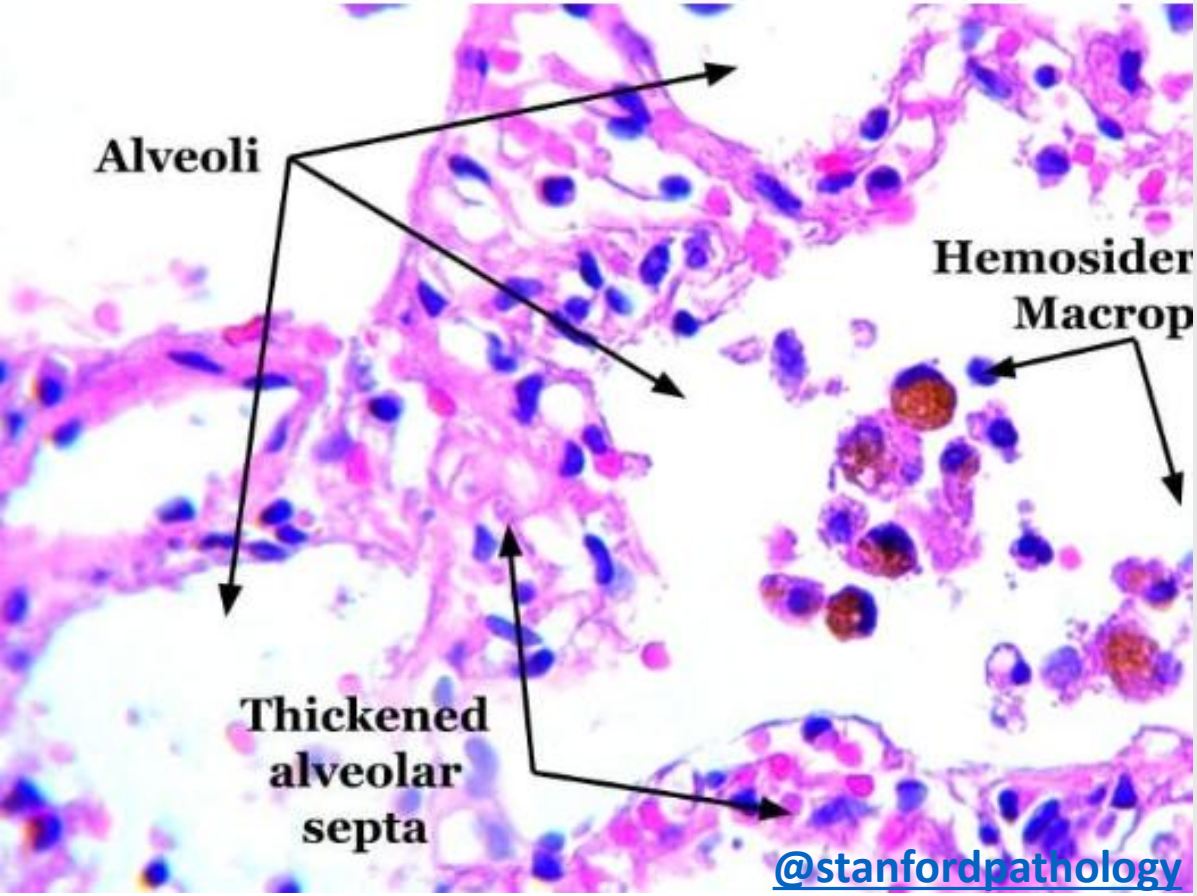
- **Infections**: pneumotropic viruses (influenza, BRSV)
- **Toxins and gases:** NO_2, SO_2, H_2S, 3-methylindole, phosgene, α-naphthylthiourea
- **Hypoxic–inflammatory causes:** septicemia, toxemia, ARDS, anaphylaxis, type I hypersensitivity
- **Mechanical/chemical:** inhaled corrosive gases, high oxygen concentration (80–100% O_2)
- **Metabolic/other:** pancreatitis, shock-like conditions, surfactant deficiency (neonatal hyaline membrane disease)

Special Conditions

- **Cardiogenic edema:** Due to heart failure; may be reversible with treatment.
- **ARDS (Adult Respiratory Distress Syndrome):** Diffuse alveolar damage, hyaline membranes, neutrophil aggregation, cytokines, and free radicals.
- **Neurogenic edema:** In brain trauma, damage to the vasomotor center leads to increased catecholamines and altered permeability.

Gross Findings

- **Acute Edema**
  - The lung is heavy, reddish or pale, moist, and has a doughy consistency
  - .Finger impressions remain, and frothy fluid (fluid + air) is seen on the cut surface.
  - In cattle and pigs: the interlobular septa are edematous, and the lobular architecture is prominent.
- **Chronic Edema**
  - Protein-rich, turbid, and viscous fluid.
  - Accompanied by congestion and fibrosis.
- **Severe Edema**
  - Abundant foam in the trachea and bronchi.
  - Foam may also be present at the nostrils.



https://www.instagram.com/p/CWONt2HpD_A/

Histopathological Findings

- **Hemodynamic Edema**
  - Alveoli are filled with homogeneous, pale eosinophilic fluid.
  - The fluid is typically low in protein and may disappear in formalin.
- **Permeability Edema**
  - The edema fluid is darker eosinophilic and has high protein content.
  - Alveolar epithelial and endothelial damage is prominent.
  - Hyaline membrane formation is observed in acute cases.
- **Chronic Cardiogenic Edema**
  - Hemosiderin-laden macrophages **("heart failure cells")**
  - Fibrosis and muscular hypertrophy in capillaries
  - Sclerosis in alveolar septa

Hyaline Membranes

Definition

- It is a **specific form of pulmonary edema**.
- Microscopically, it appears as **mucopolysaccharide–protein accumulations** lining the walls of **alveoli, alveolar ducts, and bronchioles**.
- It interferes with **gas exchange**, leading to **severe diffusion impairment**.

Pathogenesis

- Capillary permeability disturbance
- Incomplete lung development (prematurity)
- Insufficient surfactant production
- Plasma proteins leaking into alveoli mix with surfactant → formation of hyaline membranes

Hyaline Membranes

Observed in the following conditions:

- **Allergic Causes**
  - In cattle, along with **pasture emphysema** and **acute edema**
  - **Verminous pneumonias**
  - **Inhaled gases**
  - **Uremic pneumonia** in dogs

Neonatal Respiratory Distress Syndrome (NRDS):

- In human infants → idiopathic respiratory distress syndrome
- Similarly observed in foals, lambs, piglets, kittens, and puppies
- Type II alveolar cells are scarce, and surfactant (phospholipid-lecithin) levels are insufficient → alveoli are underdeveloped
- In piglets, a recessive genetic form is known as Barker syndrome
- Hypoparathyroidism has also been reported in some cases

- **Thrombosis, Embolism, and Infarction**
  - Parasites such as *Dirofilaria immitis* and *Angiostrongylus vasorum*, endocrinopathies such as hyperadrenocorticism and hypothyroidism, glomerulopathies, and hypercoagulable states are causes of pulmonary arterial thrombosis and thromboembolism in dogs.
  - When the pulmonary artery or major vessels are occluded, **hemorrhagic infarction** develops, characterized by coagulative necrosis due to hypoxia.
  - **Septic emboli**, consisting of fragments detached from an infected mural thrombus (within a chamber or vessel), become trapped in the pulmonary circulation. These emboli typically originate from right-sided bacterial endocarditis in all species, from abscesses extending into the vena cava in cattle, and from septic arthritis or omphalitis in farm animals.

Thrombosis and Thromboembolism

•Causes in Dogs:

- Parasites → *Dirofilaria immitis*, *Angiostrongylus vasorum*.
- Endocrinopathies → Hyperadrenocorticism, hypothyroidism
- Glomerulopathies
- Hypercoagulable states
- Result**: Pulmonary arterial thrombosis** and **thromboembolism.**

Infarction

- **Obstruction of the pulmonary artery or major vessels → hemorrhagic infarction**
- Mechanism: hypoxia → coagulative necrosis

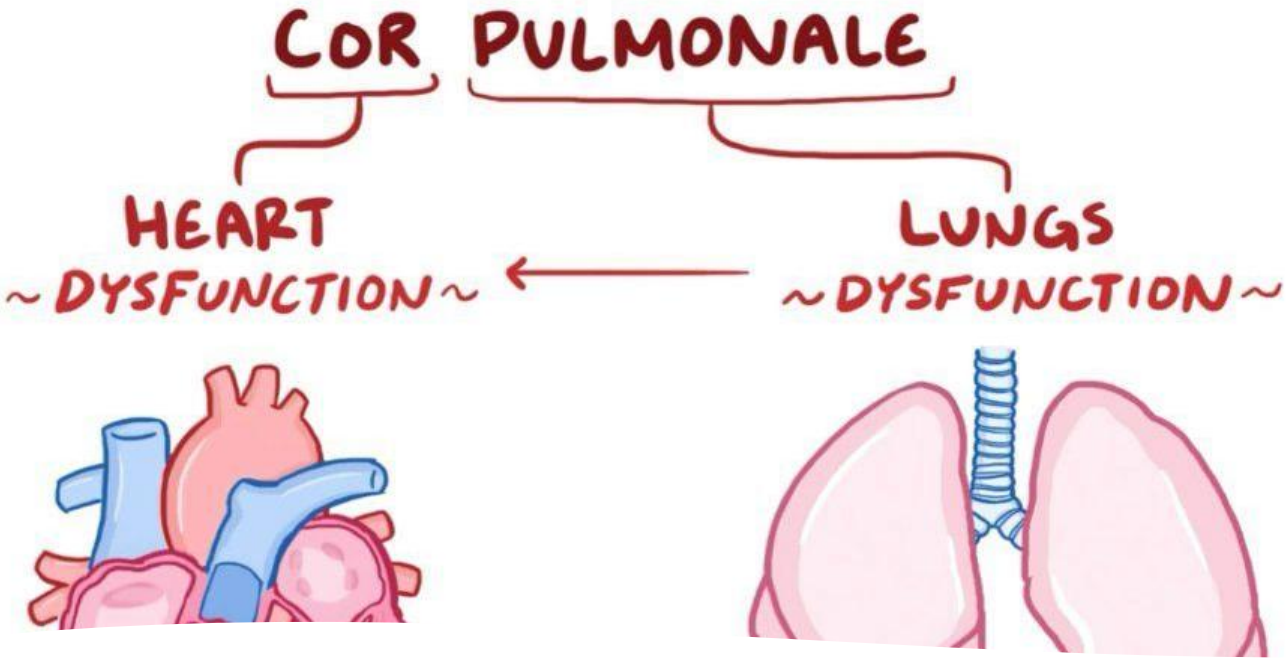
Septic Emboli

Source: Fragments detached from an **infected mural thrombus**

They become **trapped in the pulmonary circulation**

Causes by species:

- In all species → **bacterial endocarditis of the right heart**
- In cattle → **abscesses extending into the vena cava**
- In farm animals → **septic arthritis**, **omphalitis**



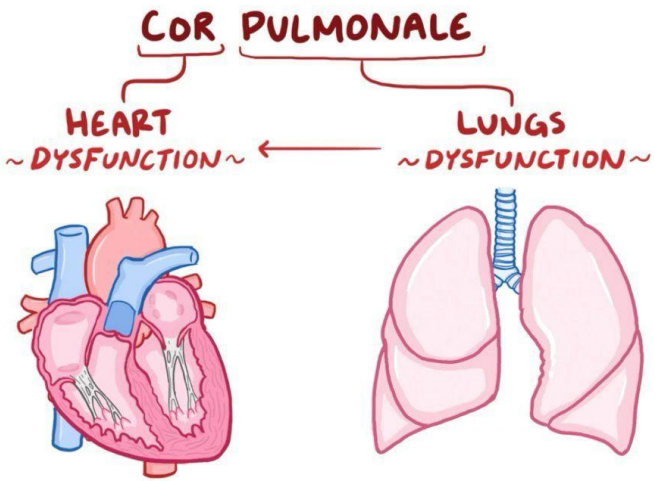
Cor Pulmonale

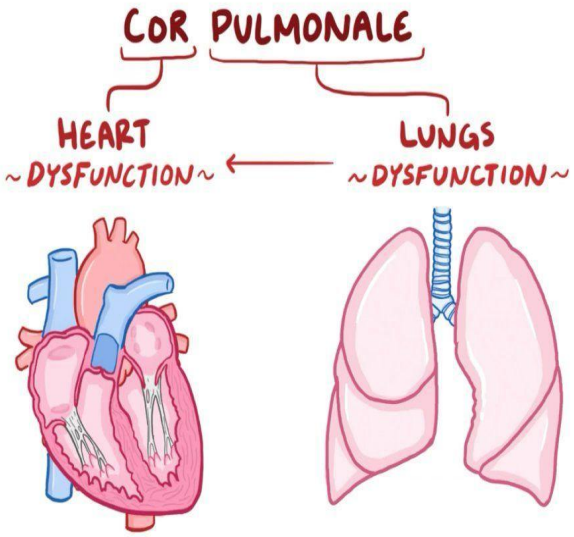
Definition

- **Pulmonary hypertension** secondary to diseases of the lung parenchyma and vascular system results in **right ventricular hypertrophy and/or dilation**.
- In other words, the changes in the right heart are **secondary** (i.e., not due to primary heart disease).

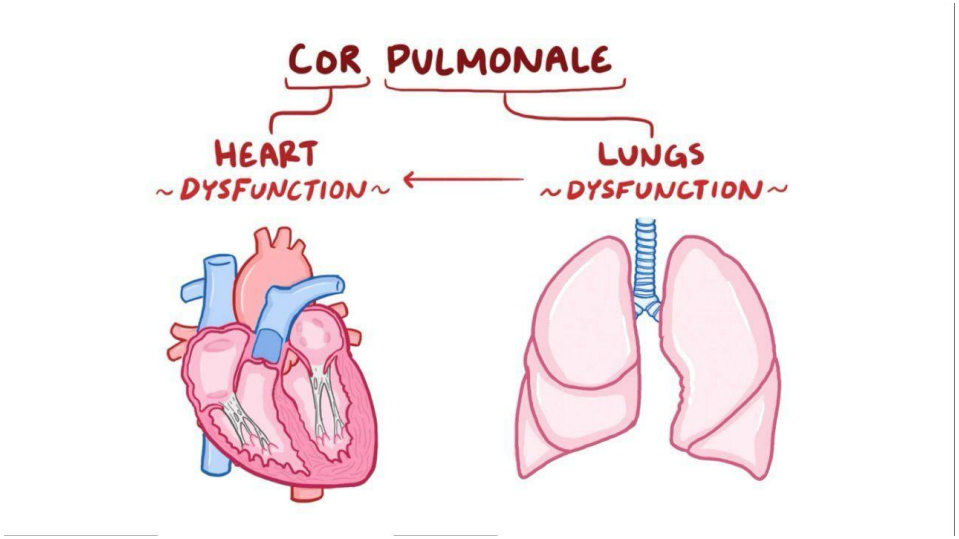
Causes

- Chronic lung diseases (e.g., bronchitis, emphysema, fibrosis, interstitial pneumonia)
- Vascular disorders
- In some cases, **cardiac abnormalities** (e.g., right-to-left shunt, mitral stenosis) may also lead to **secondary pulmonary hypertension** and are thus included in the **cor pulmonale** category.





- **Classification**
- **1. Acute Cor Pulmonale**
- Usually develops after **massive pulmonary embolism**.
- Characterized by **sudden dilation of the right ventricle**.
- **2. Chronic Cor Pulmonale**
- More common form.
- Develops due to diseases that cause **chronic pulmonary hypertension**.
- There is sufficient time for **right heart hypertrophy** to occur.
- ◆ **Compensatory/Concentric Hypertrophic Cor Pulmonale**
- Chronically hypertrophied form of the right heart.
- ◆ **Eccentric Cor Pulmonale**
- Hypertrophied heart that undergoes **progressive dilation** over time.
- In this case, **right-sided heart failure** develops.



- **General Circulatory Disorders:**
- Edema, passive hepatic hyperemia, circulatory failure
- **Pulmonary Hypertension:**
- Increased pressure in the pulmonary circulation → increased workload on the right heart → hypertrophy

Specific Conditions

- **Brisket Disease (High Altitude Disease):**
- **Occurs in cattle, and less commonly in horses and sheep.**
- **Seen in animals grazing at altitudes above 2500 meters.**
- **Due to hypoxia, animals develop subcutaneous edema along with chronic cor pulmonale.**