Pathology: High-Yield Summary (Lec 1)

1. Atelectasis (Lung Collapse)

Key Concept: Shunting of poorly oxygenated blood from pulmonary arteries into veins leads to hypoxia.

Type	Mechanism	Key Clinical Association	Reversibility
Resorption	Airway obstruction prevents air from reaching alveoli.	**Mucus plugs (Post-Op)**, foreign bodies, tumors.	Yes
Compression	Mechanical pressure from the pleural cavity.	**Pleural effusion**, Pneumothorax, Basal atelectasis (elevated diaphragm).	Yes
Contraction	Local or diffuse fibrosis prevents expansion.	**Fibrosis/Scarring** (Chronic conditions).	**No (Permanent)**

⚠️ Common Student Mistakes:

- **The Mediastinal Shift:** In **Resorption** atelectasis, the mediastinum shifts **TOWARD** the collapsed lung. In **Compression** (especially tension pneumothorax), it shifts **AWAY**.
- **Post-Op Scenario:** If a patient has difficulty breathing 2 days after surgery and it improves after coughing up sputum, it is **Resorption** atelectasis (due to a mucus plug), not ARDS.

2. ARDS

Key Concept: Sudden onset of hypoxemia and bilateral pulmonary edema in the absence of heart failure.

- **Clinical Definition:** .defined as respiratory failure (Failure of the respiratory function-> gas exchange either the oxygenation or the elimination of CO2) occurring within 1 week of a known clinical insult with bilateral opacities on chest imaging, NOT fully explained by effusions, atelectasis, cardiac failure, or fluid overload.

- **Pathology: Diffuse Alveolar Damage (DAD)**.
- **The "Hyaline Membrane":** Formed by fibrin-rich edema fluid mixed with necrotic epithelial cell debris (Type I pneumocytes).
- **The Main Player: Neutrophils**. They release proteases and ROS that damage both the endothelium and epithelium.

⚠️ Common Student Mistakes:

- **Stage Recognition:** * *Acute Stage:* Hyaline membranes + Edema.
    - *Organizing Stage:* **Type II Pneumocyte proliferation** (the "stem cells" of the lung trying to repair) and fibrosis.

3. Obstructive vs. Restrictive Diseases

Key Concept: Obstruction = Flow problem (Out). Restriction = Volume problem (In).

- **Obstructive (COPD, Asthma):** Difficulty getting air **OUT**.
    - **FEV1/FVC Ratio:** Decreased (< 0.7).
    - **TLC:** Increased (Hyperinflation/Air trapping).
- **Restrictive (Fibrosis, ARDS, Chest wall deformities):** Difficulty getting air **IN**.
    - **FEV1/FVC Ratio: Normal or Increased** (because both FEV1 and FVC decrease proportionately).
    - **TLC:** Decreased.

4. Emphysema (The "Pink Puffer")

Key Concept: Permanent enlargement of airspaces distal to terminal bronchioles WITHOUT significant fibrosis.

- **Mechanism:** Imbalance between **Proteases (Elastase)** and **Antiproteases (α1-Antitrypsin)**.
- **Centriacinar (Centrilobular):** * Most common.
    - Associated with **Smoking**.
    - Location: Upper lobes (apical segments).
- **Panacinar (Panlobular):**
    - Associated with **α1-Antitrypsin deficiency**.
    - Location: Lower zones of the lung.

⚠️ Common Student Mistakes:

- **Fibrosis:** Students often think Emphysema involves fibrosis because it's "chronic." It does **NOT** involve significant fibrosis (unlike restrictive diseases).
- **The Alveolar Wall:** The primary defect is the **destruction of alveolar walls**, which reduces elastic recoil and causes airway collapse during expiration.

5. Chronic Bronchitis (The "Blue Bloater")

Key Concept: Clinical diagnosis—Productive cough for 3 months in 2 consecutive years.

- **Pathology:** Hypertrophy of mucus glands.
- **Reid Index:** Thickness of the gland layer / Thickness of the wall (between epithelium and cartilage).

6. Asthma

- **Type:** Type I Hypersensitivity (IgE mediated).
- **Morphology (The "Triad"):**
    1. Mucus plugs with epithelium.
    2. Eosinophil protein (Galectin-10).
    3. Thickened basement membrane + Smooth muscle hypertrophy.
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