RESPIRATORY SYSTEM

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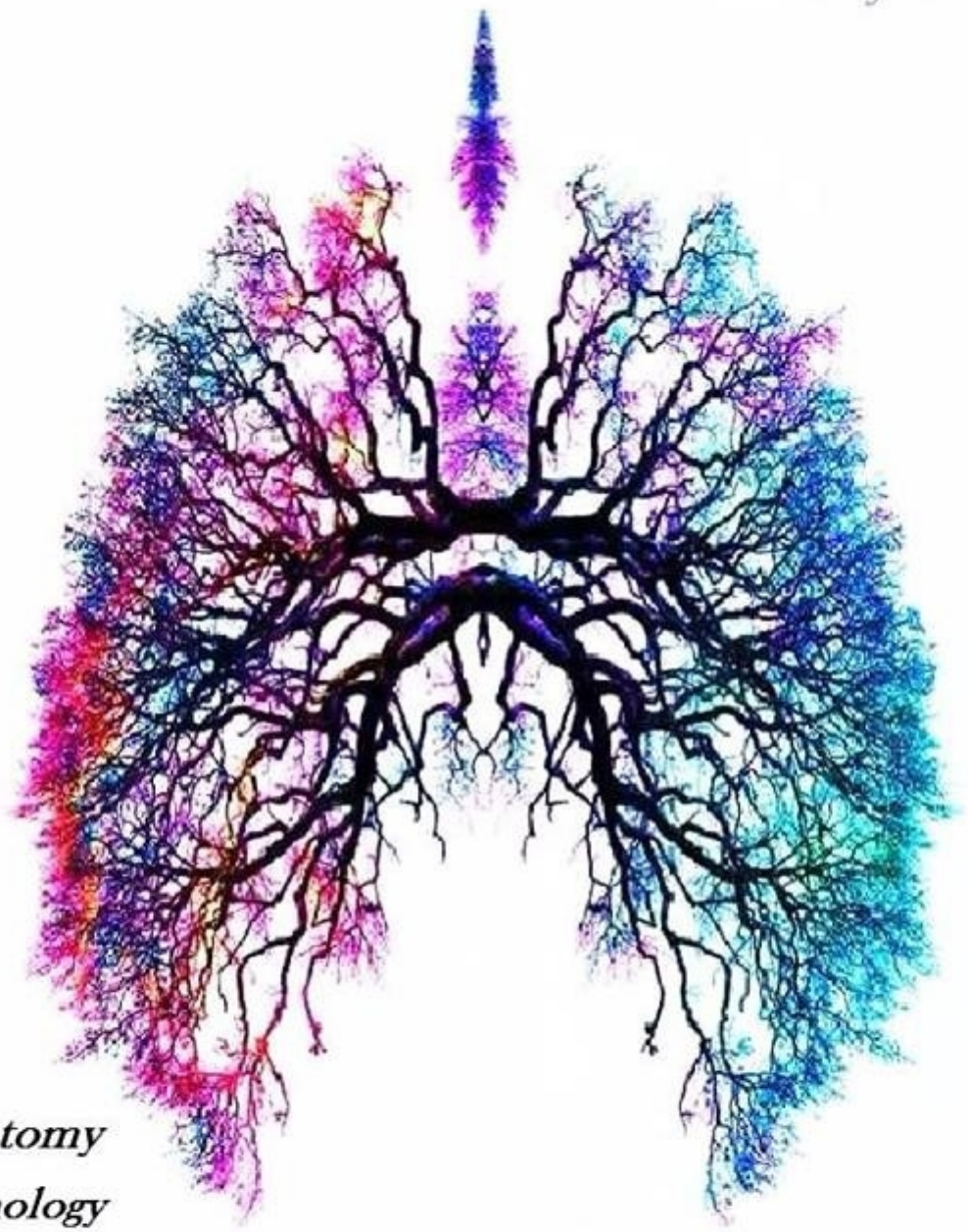

- ○ Anatomy
- ● Pathology
- ○ Physiology
- ○ Pharmacology
- ○ Microbiology
- ○ PBL

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Lecture # 1

Sheet ☐

Slide ■

Other ☐

Diseases of the Respiratory system

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I. ATELECTASIS (COLLAPSE)

- Is loss of lung volume caused by *inadequate expansion of air spaces.*
- It results in shunting of inadequately oxygenated blood from pulmonary arteries into veins, thus giving rise to hypoxia.

- **On the basis of the underlying mechanism , atelectasis is classified into three forms:**

1. Resorption atelectasis
2. Compression atelectasis
3. Contraction atelectasis

1. Resorption atelectasis. :

- Occurs when an **obstruction** prevents air from reaching distal airways.
- The air already present gradually becomes absorbed, and alveolar collapse follows.
- Depending on the level of airway obstruction, an entire lung, a complete lobe, or one or more segments may be involved.

1. The most common cause of resorption collapse is obstruction of a bronchus by mucus or mucopurulent plug

a. Postoperatively

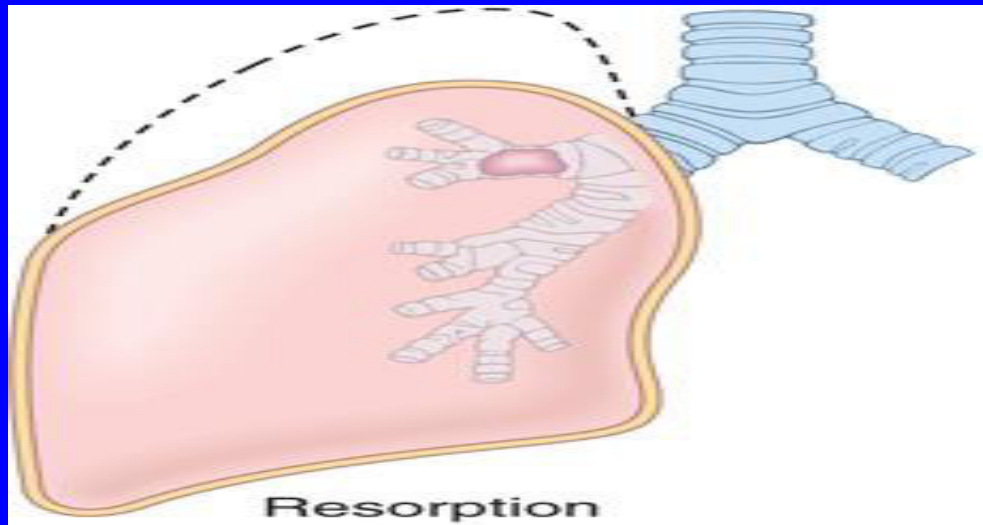
b. Complicate bronchial asthma, bronchiectasis, chronic bronchitis,

2. Obstruction by:

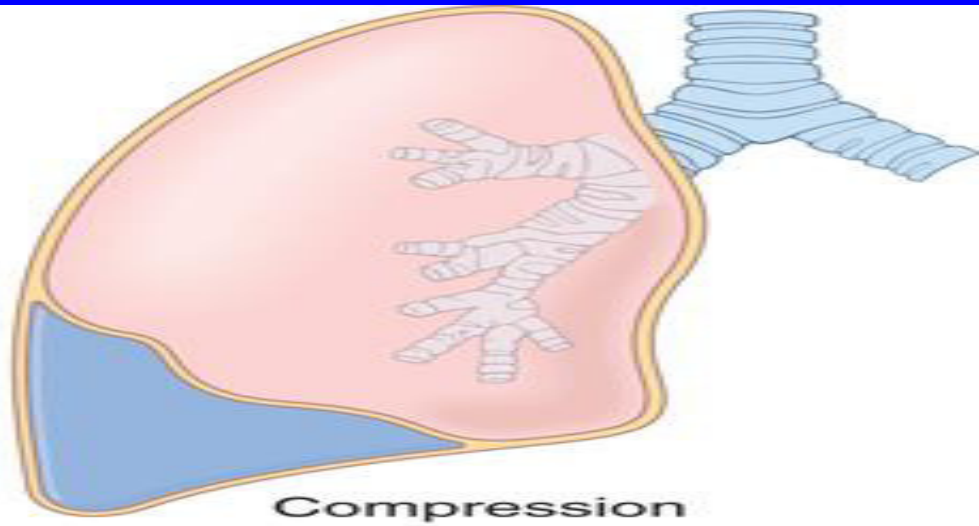
a. Tumor, or

b. Foreign body aspiration, particularly in children.

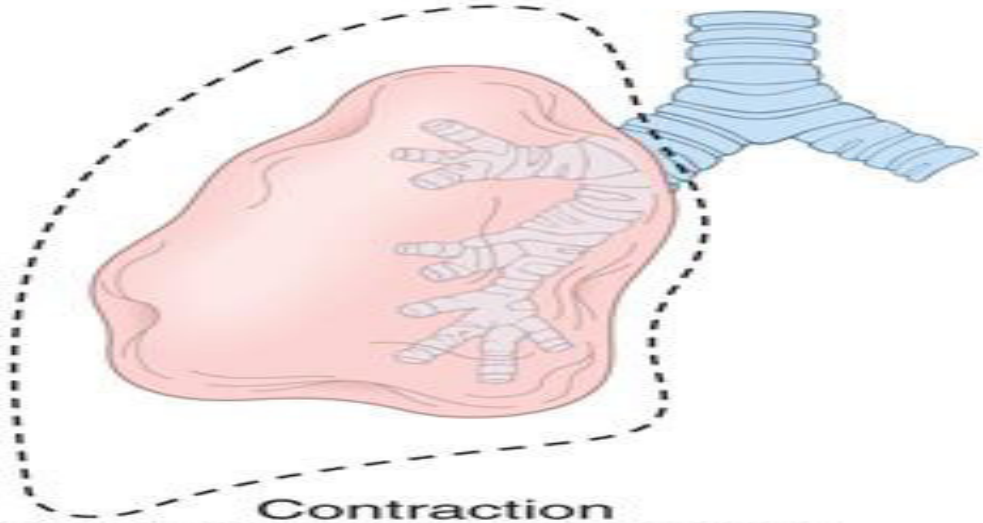
Types of atelectasis



Resorption



Compression



Contraction

2. ***Compression atelectasis***.

- Sometimes called *passive* or *relaxation atelectasis*)
- Is usually associated with accumulation of fluid, blood, or air within the pleural cavity, which mechanically collapses the adjacent lung.

- **Causes:**

a. Pleural effusion, caused most commonly by congestive heart failure (CHF).

b. Leakage of air into the pleural cavity (pneumothorax)

c. Hemothorax

d. Basal atelectasis resulting from the elevated position of the diaphragm commonly occurs in

a. Bedridden patients,

b. In patients with ascites, and

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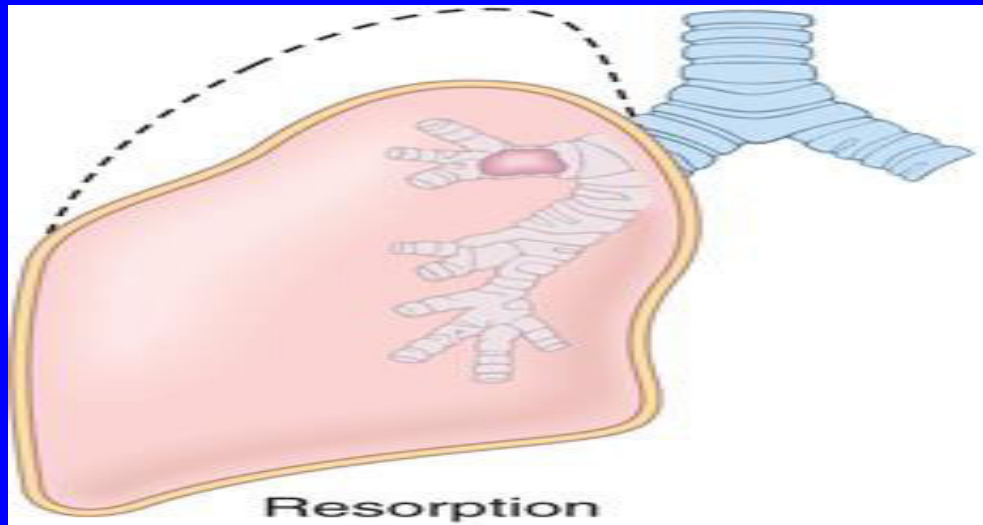
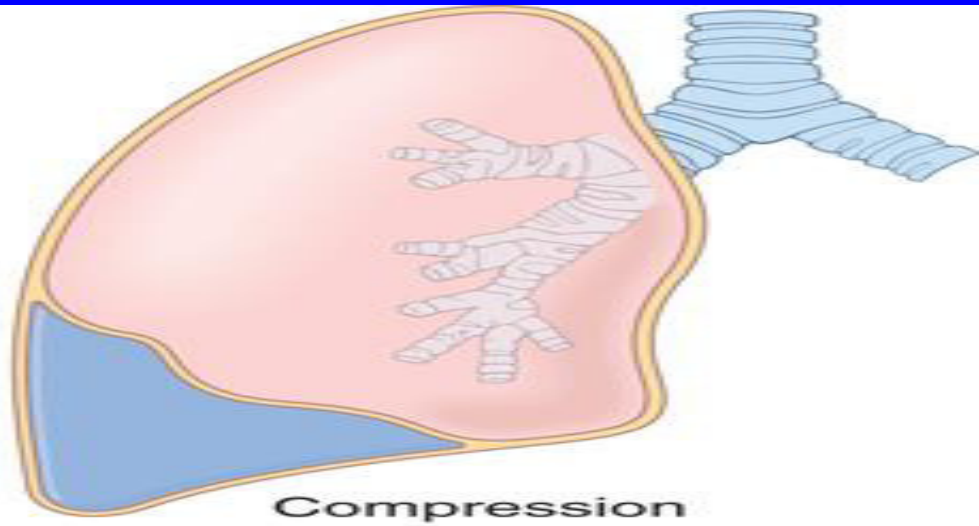
Types of atelectasis



Resorption



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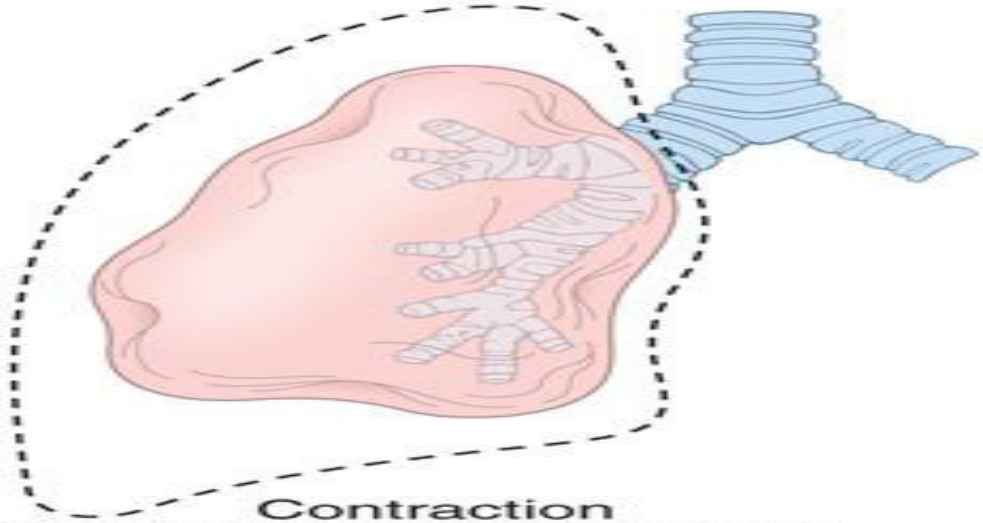

Contraction

C.- Contraction atelectasis.

- Called *cicatrization* atelectasis
- Occurs when either local or generalized fibrotic changes in the lung or pleura or prevents expansion of air spaces

Note

- Atelectasis (except when caused by contraction) is potentially reversible and should be treated promptly to prevent hypoxemia and superimposed infection of the collapsed lung.

II. ACUTE LUNG INJURY

- The term includes a spectrum of bilateral pulmonary damage (endothelial and epithelial), which can be initiated by numerous conditions.
- Clinically, acute lung injury manifests as:

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1. Acute onset of dyspnea,
2. Decreased arterial oxygen pressure (hypoxemia),
3. Development of bilateral pulmonary infiltrates on the chest radiograph,
4. Absence of clinical evidence of primary left-sided heart failure

- The pulmonary infiltrates in acute lung injury are caused by damage to the alveolar capillary membrane, rather than by left-sided heart failure , such accumulations constitute an example of *noncardiogenic pulmonary edema*.

- **Note**-

- Acute lung injury can progress to the more severe *acute respiratory distress syndrome*

Acute Respiratory Distress Syndrome
ARDS

- Is a clinical syndrome caused by diffuse alveolar capillary and epithelial damage.
- The usual course is characterized by:

A. Rapid onset of life-threatening respiratory insufficiency,

b. And severe arterial hypoxemia that is refractory to oxygen therapy and may progress to multisystem organ failure.

Note: The histologic manifestation of ARDS in the lungs is known as *diffuse alveolar damage* (DAD).

ARDS

- Occurs in a multitude of clinical settings
- And is associated with either

a. Direct injury to the lung or

b. Indirect injury in the setting of a systemic process

Direct Lung Injury

I. Common Causes

1. Pneumonia
2. Aspiration of gastric contents

II. Uncommon Causes

1. Pulmonary contusion

- Indirect causes

I. common causes

1. Sepsis

2. Severe trauma with shock

II. Uncommon causes

Acute pancreatitis

Note:

- Respiratory distress syndrome of the newborn is pathogenetically distinct; it is caused by a primary deficiency of surfactant

PATHOGENESIS

- The alveolar-capillary membrane is formed by two separate barriers: the microvascular endothelium and the alveolar epithelium.
- In ARDS, the integrity of this barrier is compromised by either endothelial or epithelial injury, or, more commonly, both.

The acute consequences of damage to the alveolar capillary membrane include:

1. Increased vascular permeability and alveolar flooding
2. Loss of diffusion capacity,
3. Widespread surfactant abnormalities caused by damage to type II pneumocytes

Suggested mechanism:

- In ARDS, **lung injury is caused by an imbalance of pro-inflammatory and anti-inflammatory mediators.**

As early as 30 minutes after an acute insult,

a. Increased synthesis of interleukin 8 (IL-8), a potent neutrophil chemotactic and activating agent, by pulmonary macrophages.

b. Release of this and IL-1 and tumor necrosis factor (TNF), leads to endothelial activation

C. Activated neutrophils release a variety of oxidants, proteases, leukotrienes that cause damage to the alveolar epithelium and endothelium.

d- Combined assault on the endothelium and epithelium increases vascular leakiness and loss of surfactant that render the alveolar unit unable to expand.

- The destructive forces by neutrophils can be counteracted by endogenous antiproteases, antioxidants, and anti-inflammatory cytokines (e.g., IL-10)

- In the end, it is the balance between the destructive and protective factors that determines the degree of tissue injury and clinical severity of ARDS

Note:

- Neutrophils are thought to have an important role in the pathogenesis of ARDS

MORPHOLOGY

In the **acute phase of ARDS**

Gross,

1. The lungs are red, firm
2. Airless, and heavy.

Microscopic examination reveals:

1. Capillary congestion,
2. Necrosis of alveolar epithelial cells,
3. Interstitial and intra-alveolar edema and hemorrhage,
4. Increased numbers of neutrophils within the vascular space, the interstitium, and the alveoli.

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5. The most characteristic finding is the presence of **hyaline membranes,** alveolar ducts

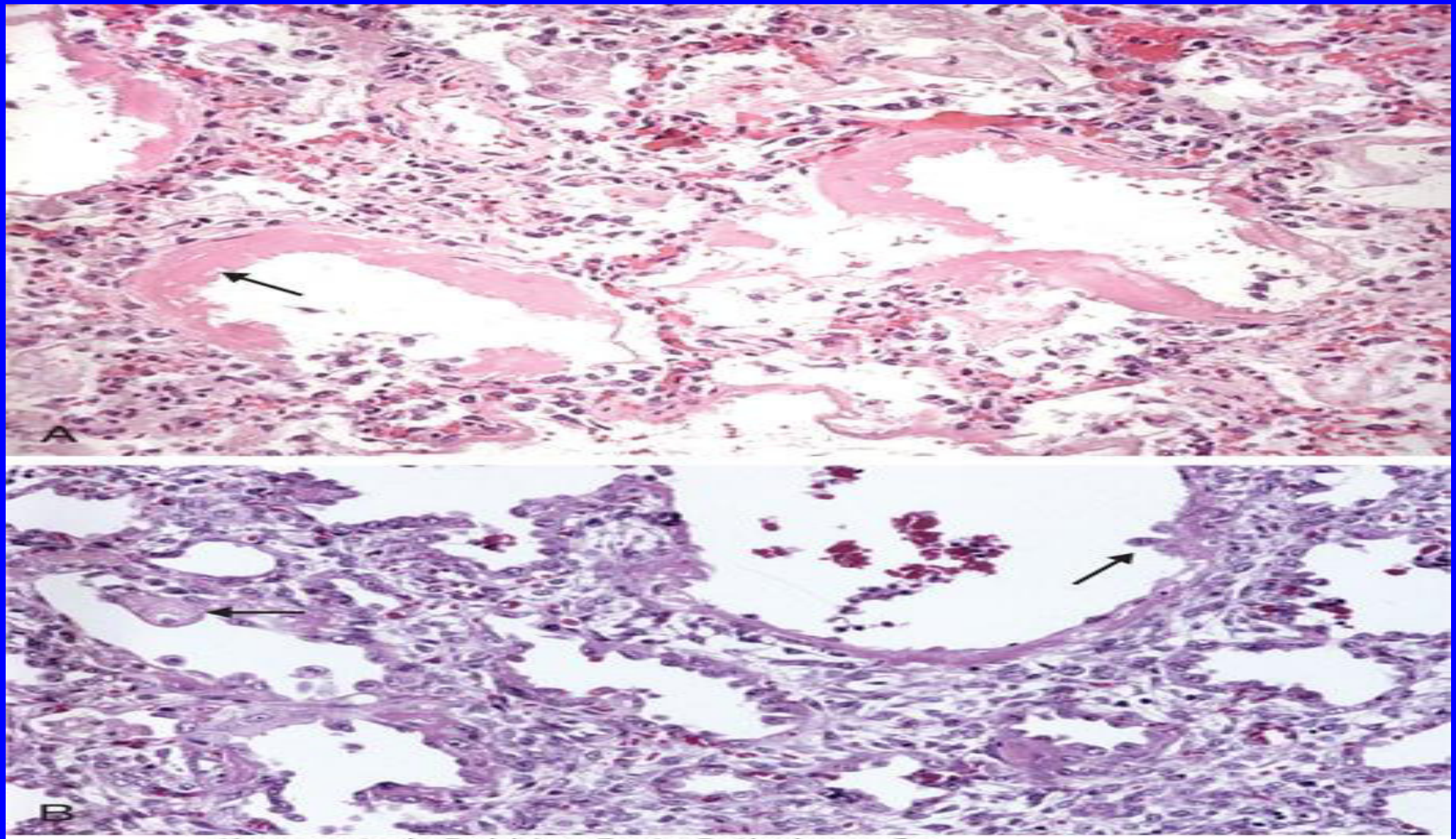
- Such membranes consist of

a. fibrin-rich edema fluid

b. Remnants of necrotic epithelial cells.

- Overall, the picture is similar to that seen in respiratory distress syndrome in the newborn .

ARDS



In the organizing stage,

- Vigorous proliferation of type II pneumocytes occurs in an attempt to regenerate the alveolar lining.

Resolution is unusual-

a. More commonly, there is organization of the fibrin exudates, with resultant intra-alveolar fibrosis.

b. Marked thickening of the alveolar septa ensues, caused by proliferation of interstitial cells and deposition of collagen..

Clinical Features

- Approximately 85% of patients develop the clinical syndrome of acute lung injury or ARDS within 72 hours of the initiating insult.
- With improvements in supportive therapy, the mortality rate ARDS cases occurring yearly has decreased from 60% to 40% in the last decade.

Predictors of poor prognosis include

- Advanced age
- Underlying bacteremia (sepsis
- The development of multisystem (especially cardiac, renal, or hepatic) failure.

- If the patient survives the acute stage, diffuse interstitial fibrosis may occur, with continued compromise of respiratory function.
- However, in most patients who survive the acute insult and are spared the chronic sequelae, normal respiratory function returns within 6 to 12 months