

THE RESPIRATORY SYSTEM

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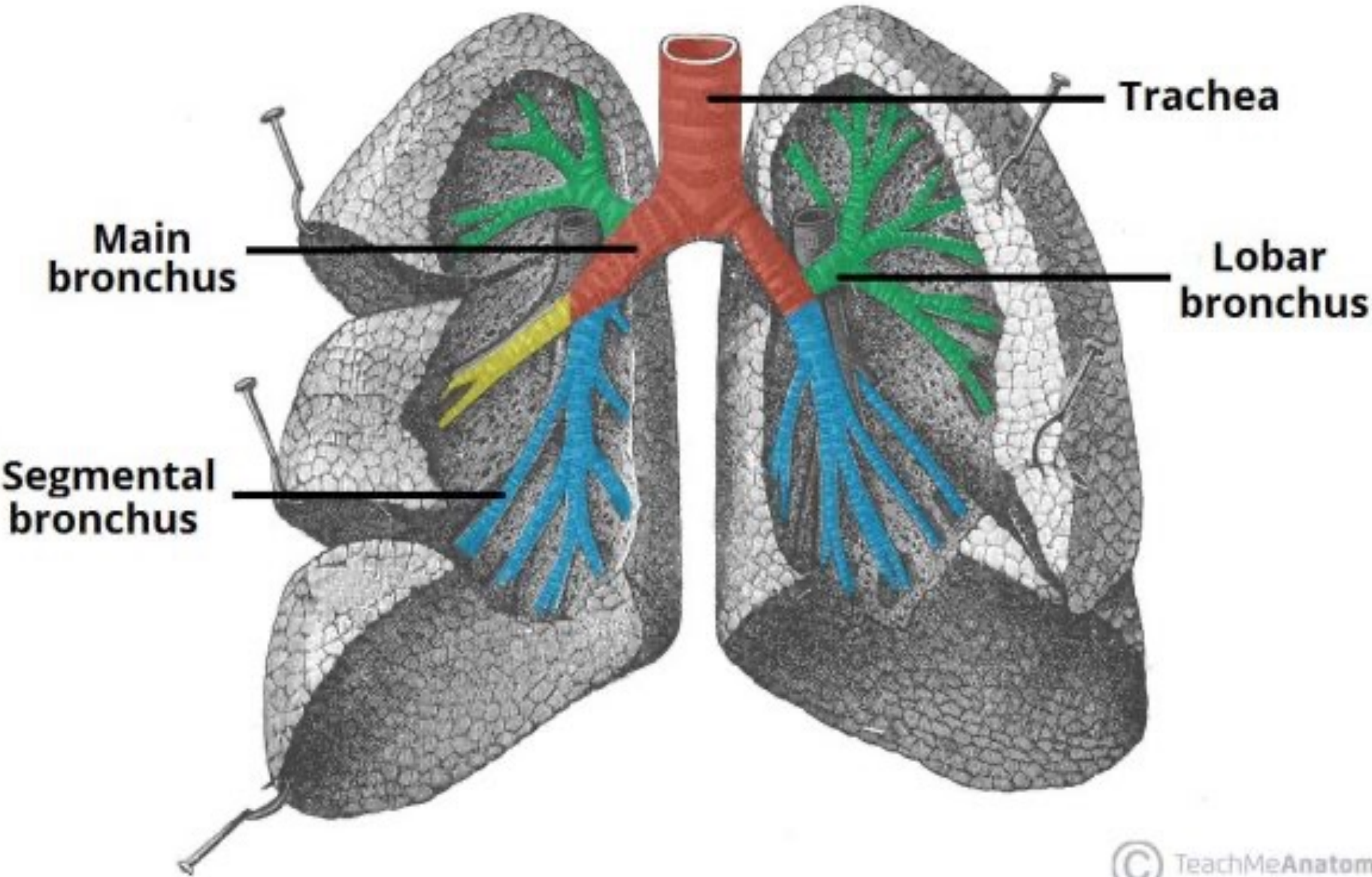
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OBJECTIVES:

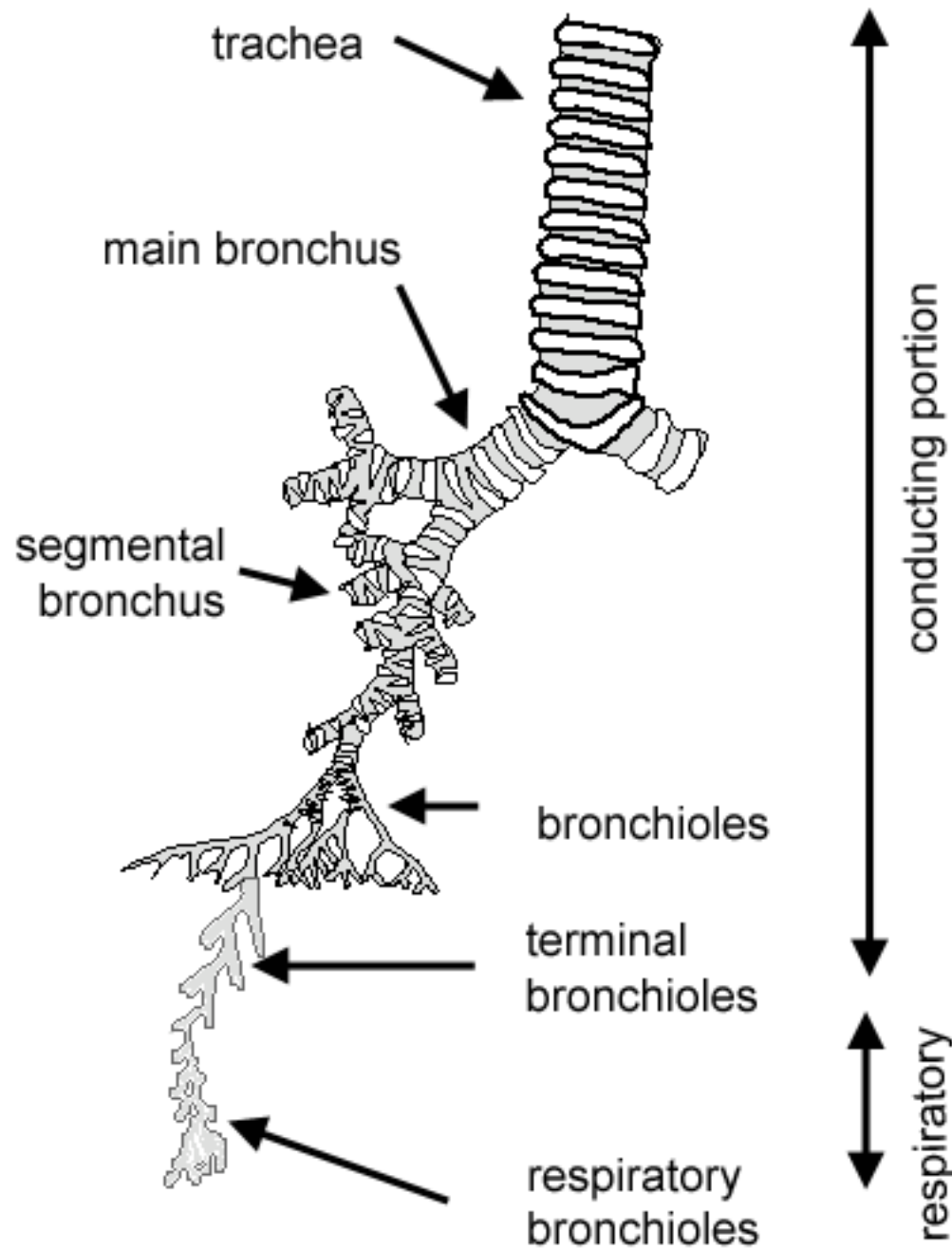
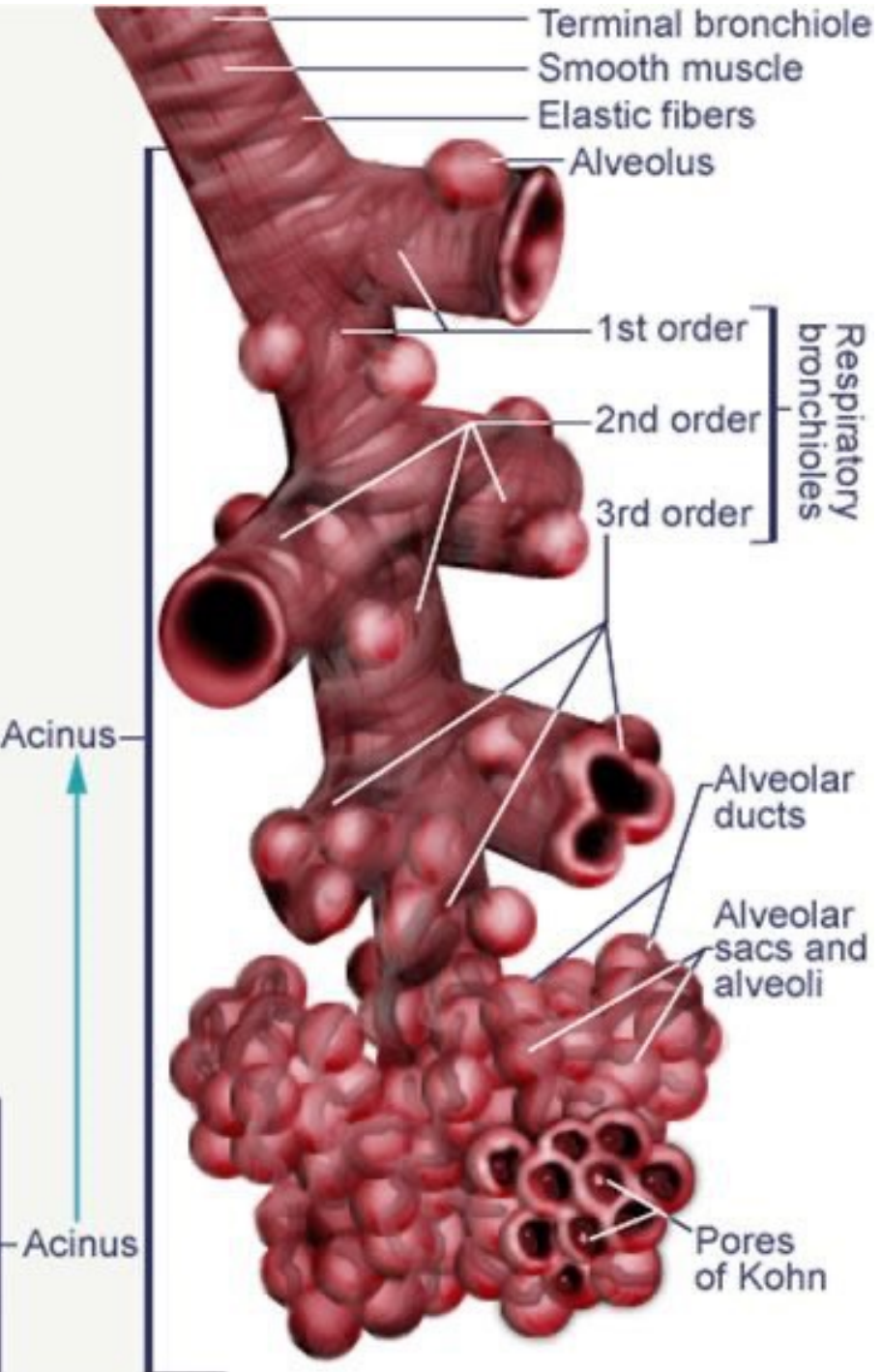
- **Function and anatomy**
- **Atelectasis (Collapse)**
- **Acute respiratory distress syndrome (ARDS)**
- **Restrictive vs. Obstructive lung diseases**

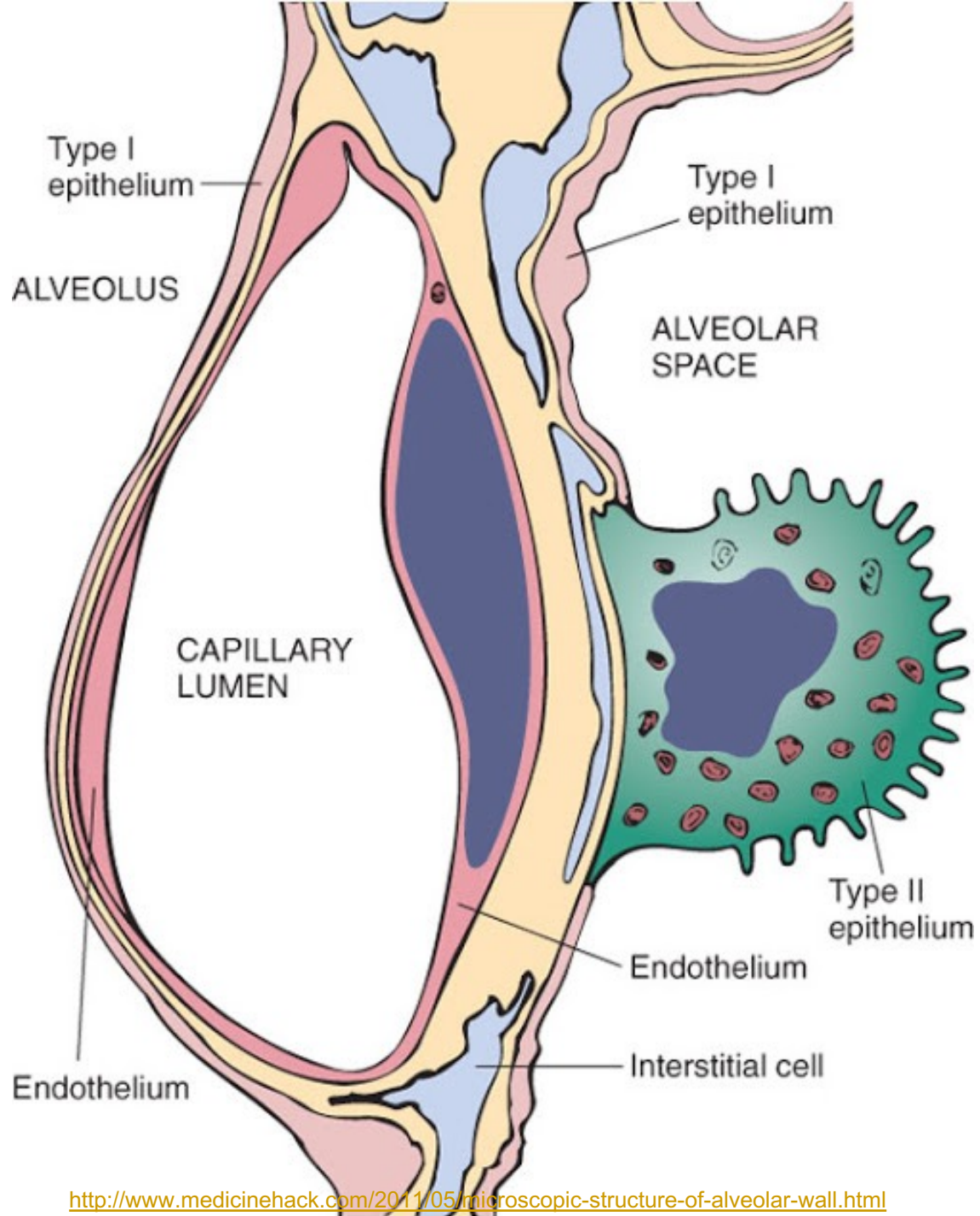
FUNCTION AND ANATOMY:

The major function of the lung is to replenish oxygen and remove carbon dioxide from blood.

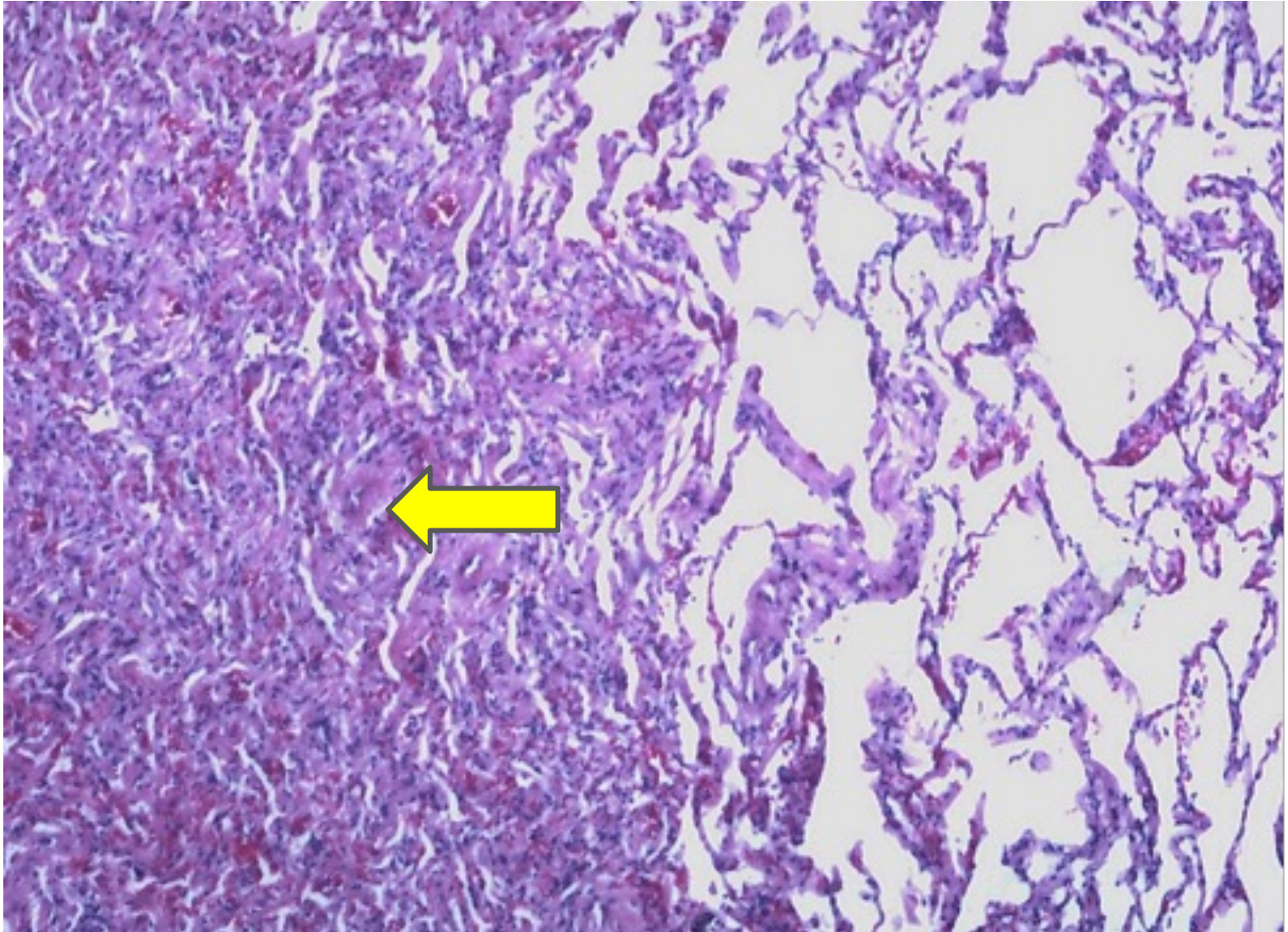


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ATELECTASIS (COLLAPSE)

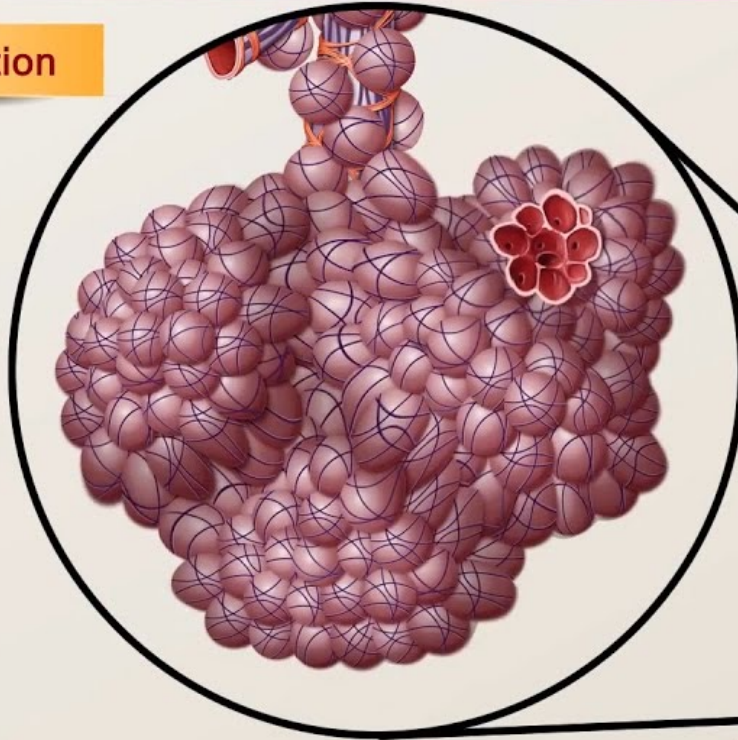


Atelectasis

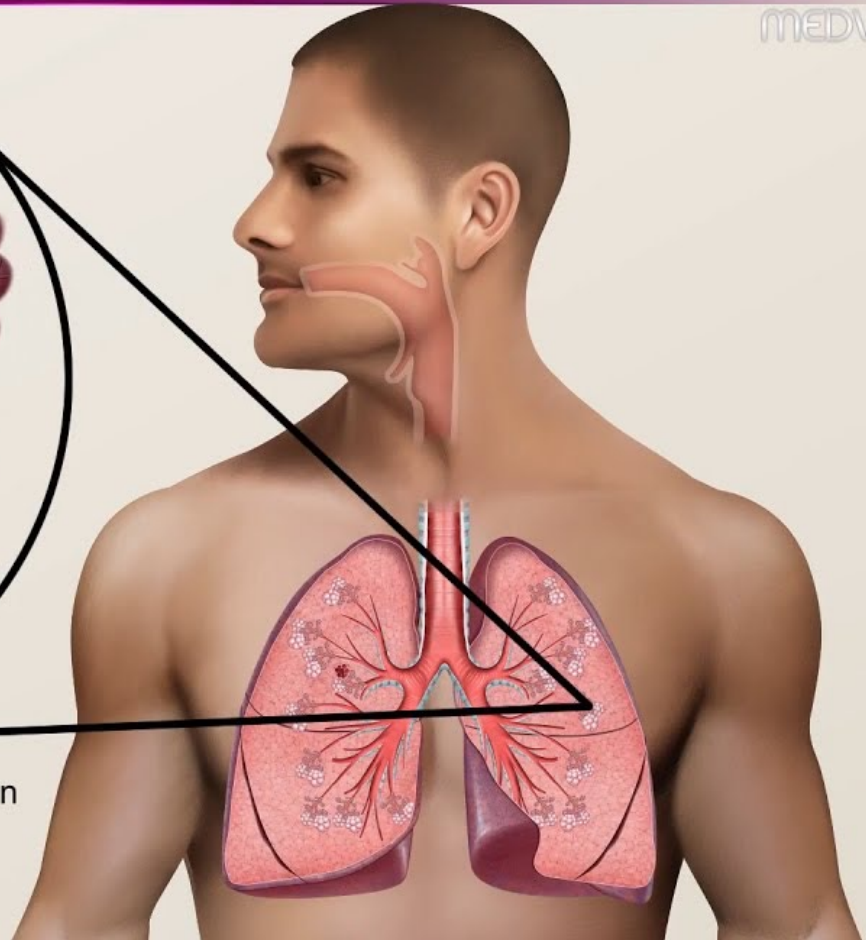


MEDVIZZ

Definition



- ◆ Loss of lung volume caused by inadequate expansion of the airspaces (collapse)

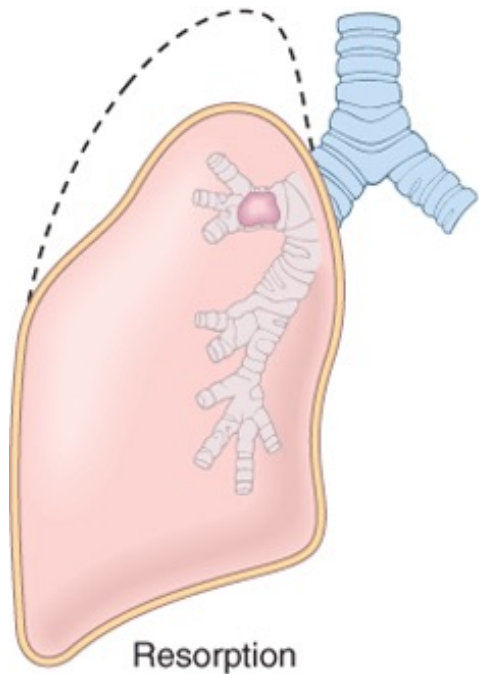


ATELECTASIS (COLLAPSE)

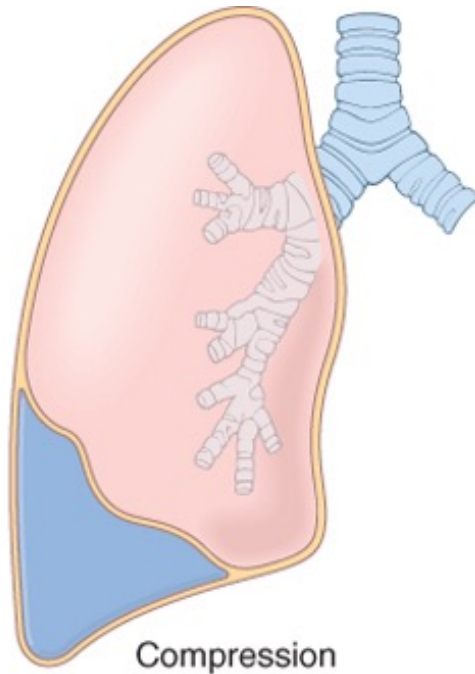
- is loss of lung volume caused by inadequate expansion of air spaces.
- It results in shunting of inadequately oxygenated blood from pulmonary arteries into pulmonary veins → resulting in ventilation perfusion imbalance and hypoxia.
- The collapsed airway are at risk of infection

THREE TYPES OF ACQUIRED ATELECTASIS:

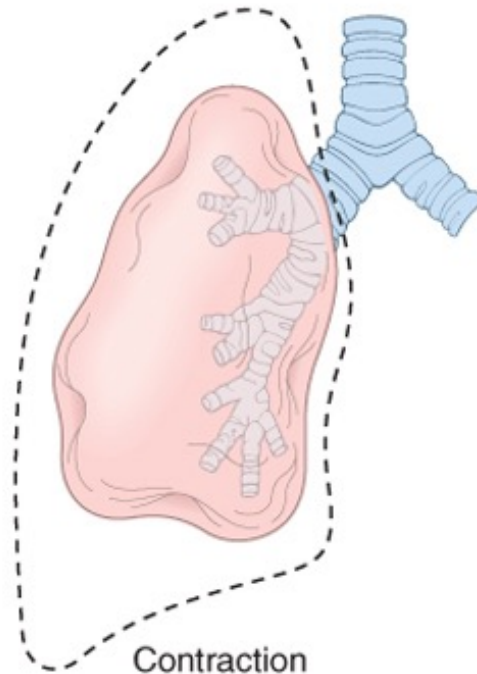
- **Resorption atelectasis**
- **Compression atelectasis**
- **Contraction atelectasis (cicatrization atelectasis)**



Resorption



Compression



Contraction

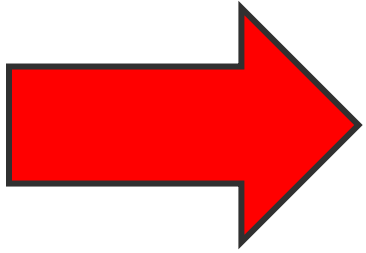
1.RESORPTION ATELECTASIS

- Due to total obstruction of an airway (bronchi, segmental bronchi or terminal bronchi) preventing air from reaching distal airways.
- The air already present in the distal airways gradually resorbed resulting in alveolar collapse.

RESORPTION ATELECTASIS, CAUSED BY:

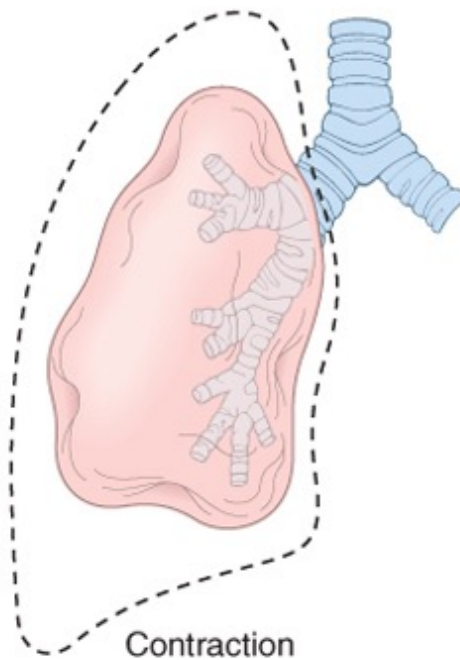
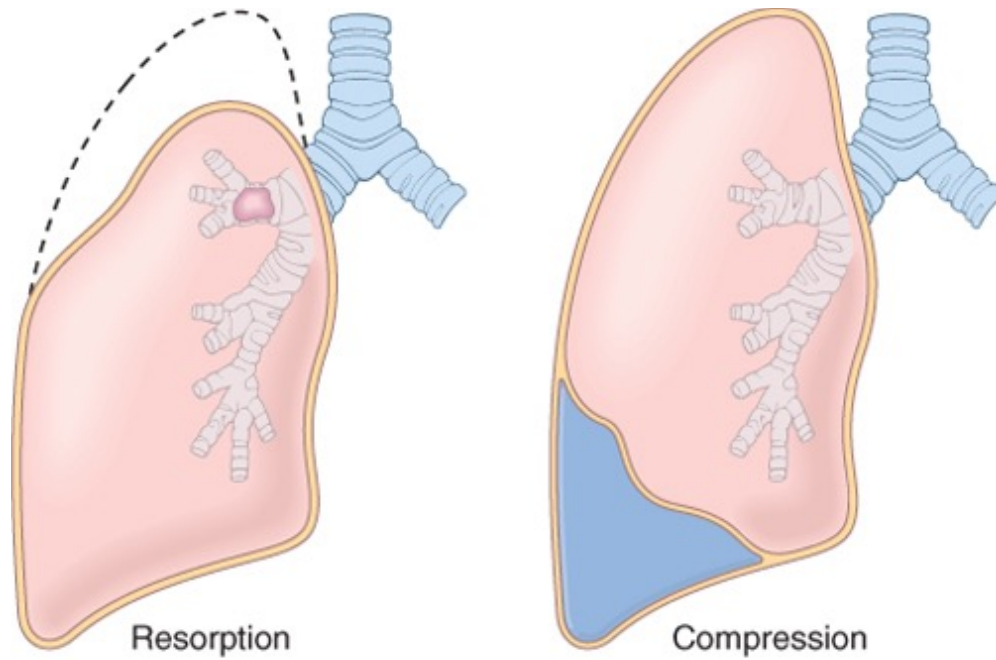
- The most common cause is Obstruction of a bronchus by:
 - ✓ Intrabronchial mucous or mucopurulent plugs in post operative patients.
 - ✓ Foreign body aspiration, especially in children
 - ✓ Obstructive lung disease: bronchial asthma, bronchiectasis, chronic bronchitis
 - ✓ Intrabronchial tumors.

Reversibility?



or

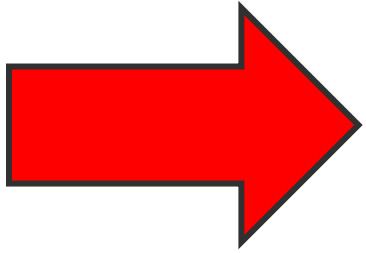




2. COMPRESSION ATELECTASIS

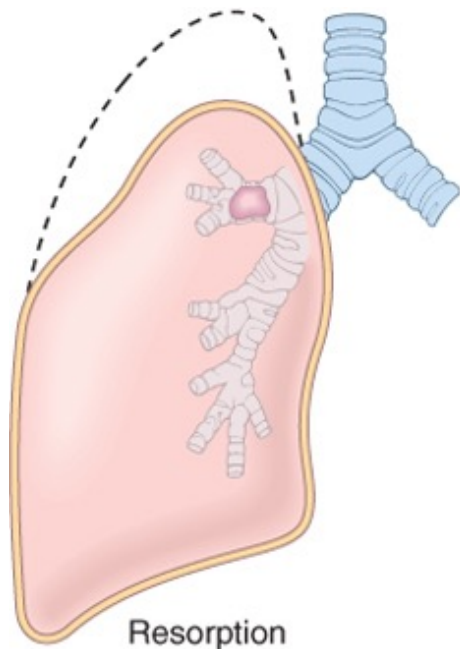
- caused by the accumulation of significant amount of fluid (blood, exudate or transudate), air (pneumothorax) or tumor within pleural cavity, which mechanically collapse adjacent lung (small airways and alveoli)
- E.x:
 - a. Pleural effusion: in Congestive Heart Failure
 - b. Pneumothorax: air in the pleural cavity due to RTA

Reversibility?

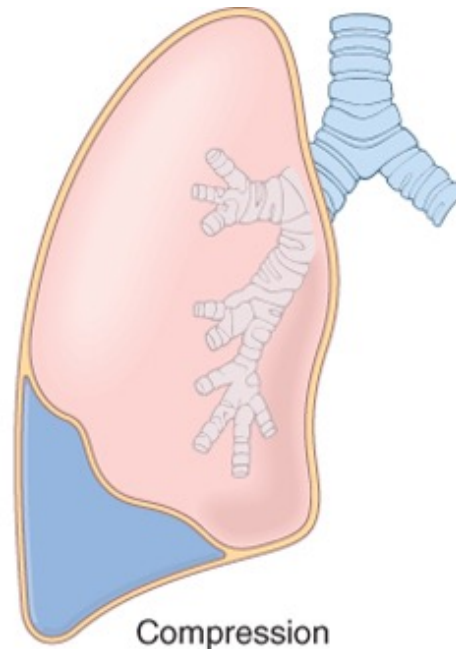


or

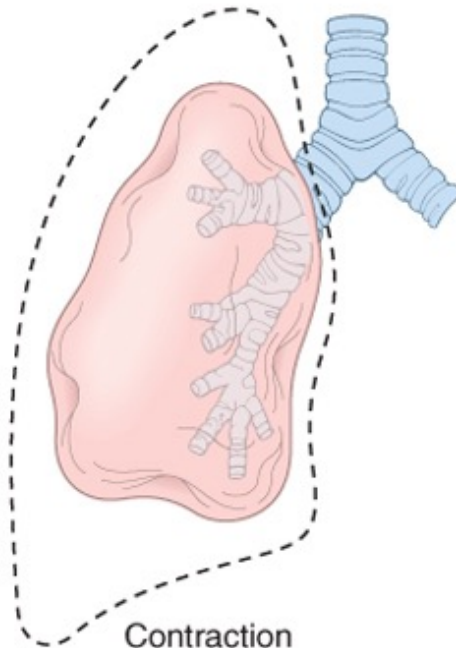




Resorption



Compression



Contraction

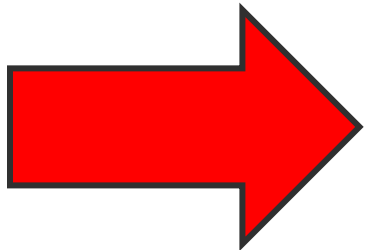
3. CONTRACTION ATELECTASIS (CICATRIZATION ATELECTASIS)

- Occurs due to local or generalized fibrosis/scarring of the lung or pleura that prevents full expansion of the lung

Reversibility?



or



ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

- **ARDS defined as** respiratory failure 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.
- considered to be the severe end of a spectrum of acute lung injury
- The histologic manifestation of this disease is called **diffuse alveolar damage (DAD)**

ARDS

- graded based on the severity of the changes in arterial blood oxygenation into mild, moderate and severe
- Severe ARDS characterized by rapid onset of life-threatening **respiratory insufficiency, Cyanosis , Severe arterial hypoxemia that becomes refractory to oxygen therapy and may progress to multisystem organ failure**

Table 15.2 Conditions Associated With Development of Acute Respiratory Distress Syndrome

Infection
Sepsis ^a Diffuse pulmonary infections ^a Viral, <i>Mycoplasma</i> , and <i>Pneumocystis</i> pneumonia; miliary tuberculosis Gastric aspiration ^a
Physical/Injury
Mechanical trauma including head injuries ^a Pulmonary contusions Near-drowning Fractures with fat embolism Burns Ionizing radiation
Inhaled Irritants
Oxygen toxicity Smoke Irritant gases and chemicals
Chemical Injury
Heroin or methadone overdose Acetylsalicylic acid Barbiturate overdose Paraquat
Hematologic Conditions
Transfusion-associated lung injury (TRALI) Disseminated intravascular coagulation
Pancreatitis
Uremia
Cardiopulmonary Bypass
Hypersensitivity Reactions
Organic solvents Drugs

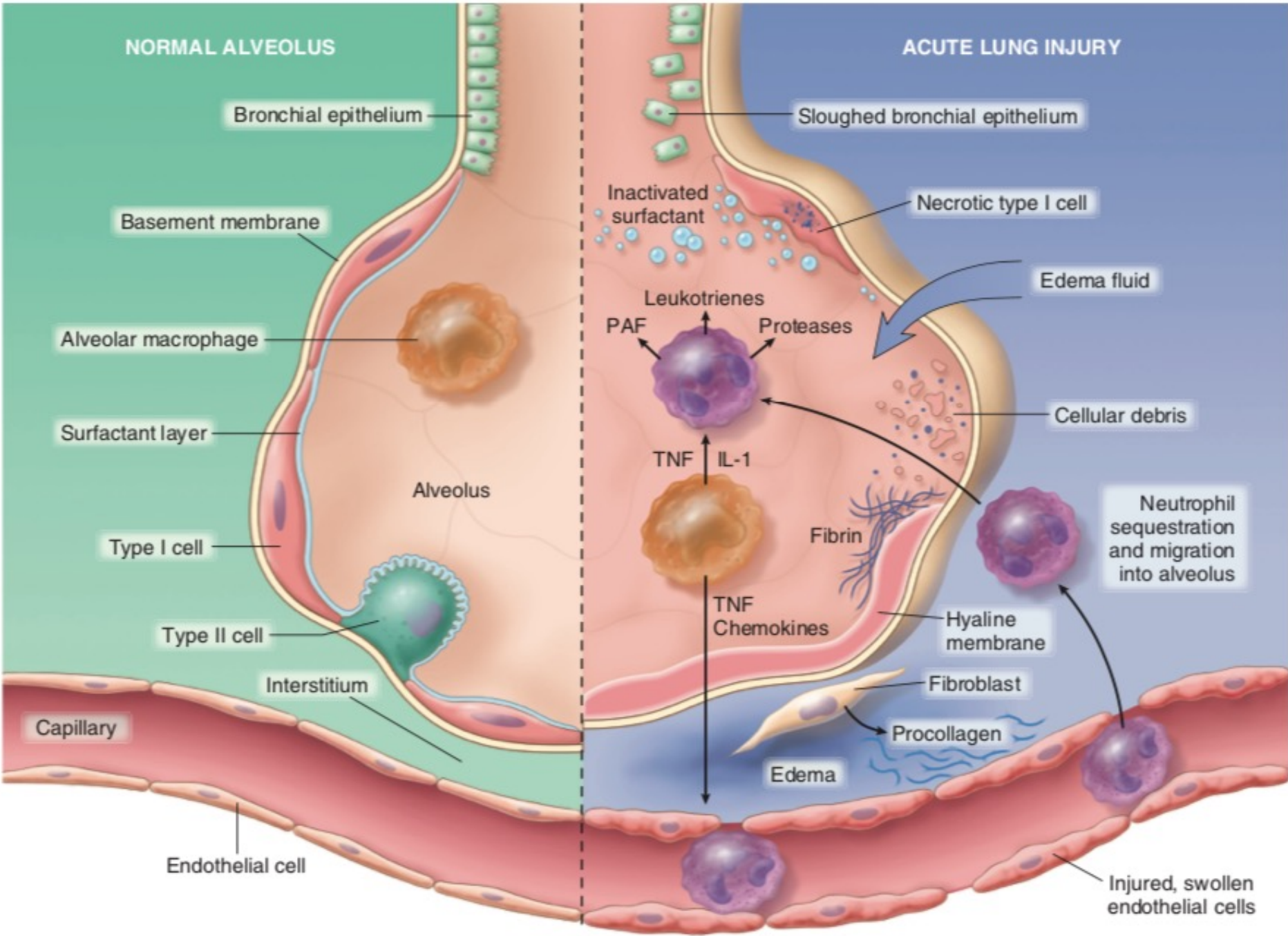
^aMore than 50% of cases of acute respiratory distress syndrome are associated with these four conditions.

ARDS **should not** be confused with respiratory distress syndrome of the newborn; the latter is caused by a deficiency of surfactant caused by prematurity.

**Just a
reminder...**



PATHOGENESIS



PATHOGENESIS:

- the integrity of the alveolar-capillary membrane is compromised by endothelial and epithelial injury.
- As early as 30 minutes after an acute insult, there is increased synthesis and release of IL-8, IL-1 and TNF by pulmonary macrophages.
- leading to endothelial activation and sequestration
- activation & chemotaxis of neutrophils in pulmonary capillaries.

PATHOGENESIS/CONT.

- Activated neutrophils release reactive oxygen species & proteases that damage the alveolar epithelium and endothelium causing vascular leakiness and loss of surfactant that render the alveolar unit unable to expand.
- the destructive forces are counteracted by endogenous anti-proteases and anti-oxidants

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

HISTOLOGY:

- In the acute phase of ARDS :
 - The most characteristic finding is the presence of **hyaline membranes**
 - consists of fibrin-rich edema fluid admixed with remnants of necrotic epithelial cells

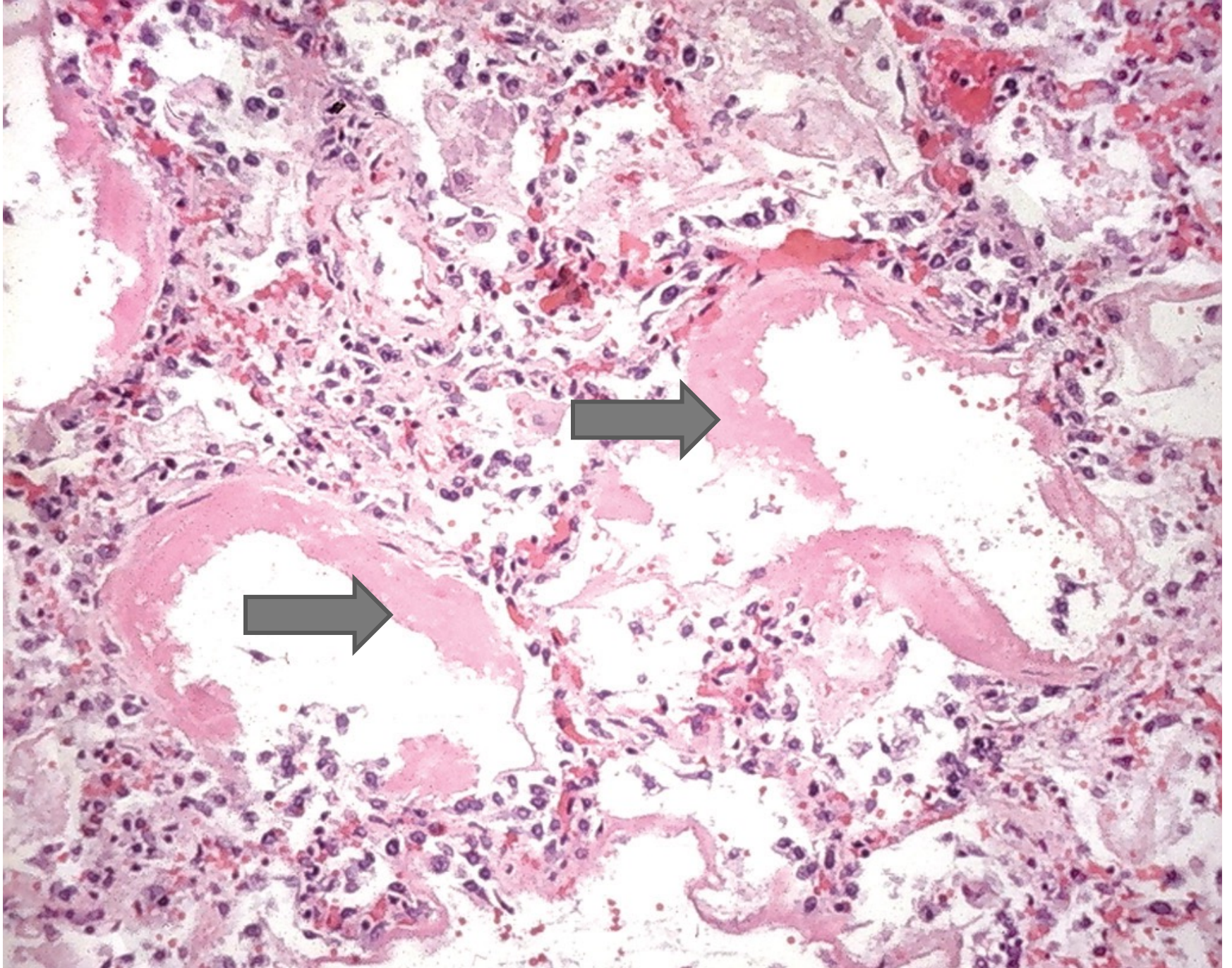


FIGURE 13.3A, ROBBINS BASIC PATHOLOGY, 10TH EDITION

HISTOLOGY:

In the organizing stage:

- **proliferation of type II pneumocytes**
- **intraalveolar fibrosis** due to organization of the fibrin-rich exudates.
- **Marked thickening of the alveolar septa due to proliferation of interstitial cells and collagen deposition.**

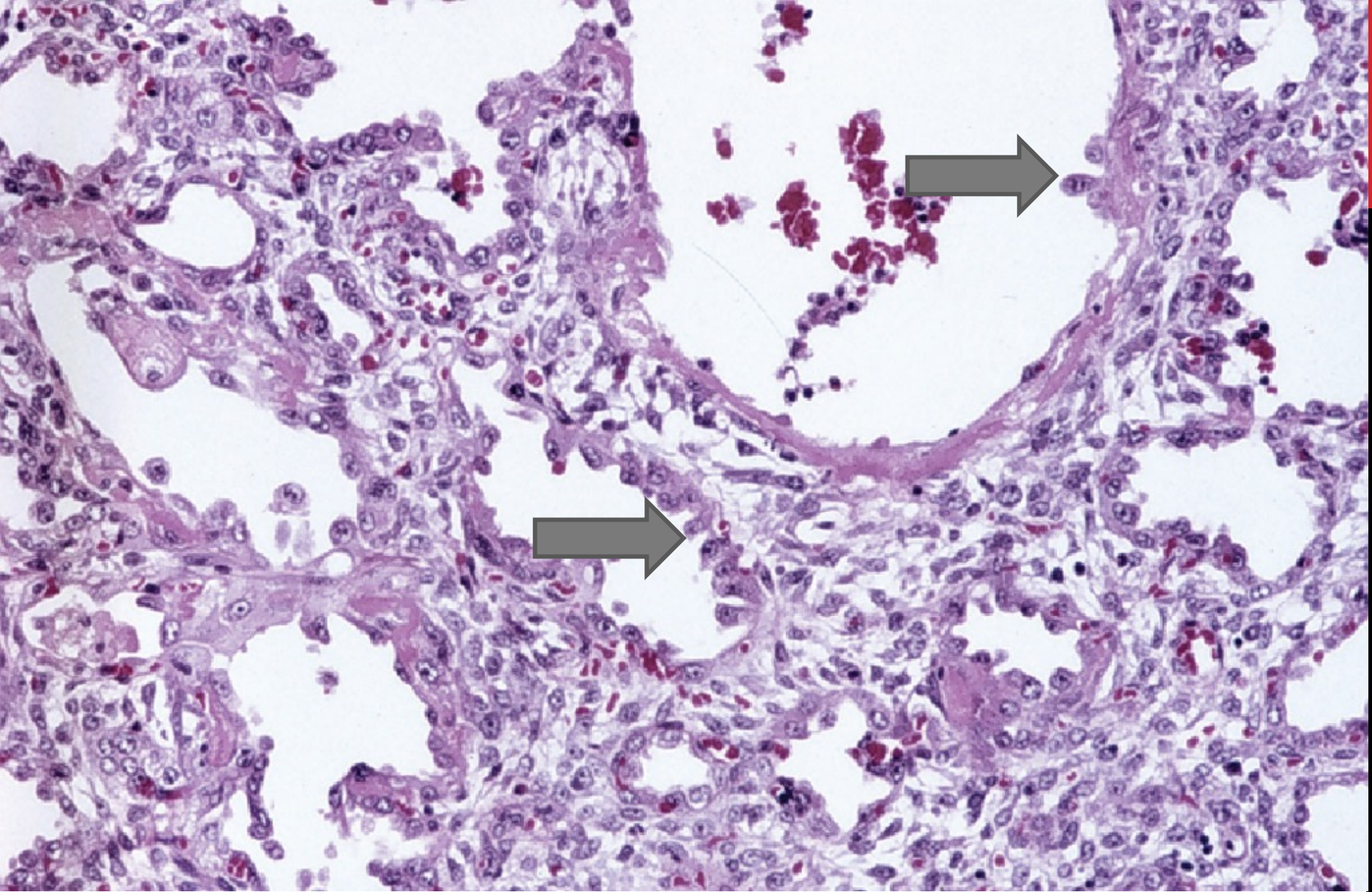


FIGURE 13.3B, ROBBINS BASIC PATHOLOGY, 10TH EDITION

CLINICAL FEATURES

- Patients are hospitalized for one of the predisposing conditions
- Profound dyspnea and *tachypnea* followed by increasing cyanosis and hypoxemia, respiratory failure, and the appearance of *diffuse bilateral infiltrates* on radiographic examination.
- Hypoxemia may be refractory to oxygen therapy due to ventilation-perfusion mismatch, and respiratory acidosis can develop.

OUTCOME:

- The overall hospital mortality rate is 38.5%.
- The majority of deaths are attributable to sepsis, multiorgan failure, or severe lung injury.
- Most patients who survive the acute insult recover normal respiratory function within 6 to 12 months, but the rest have lung damage resulting in interstitial fibrosis and chronic pulmonary disease.

PREDICTORS OF POOR PROGNOSIS

- 1. advanced age**
- 2. bacteremia (sepsis)**
- 3. development of multiorgan failure**



OUTCOME



DIFFUSE PULMONARY DISEASES can be classified into two
Categories:

1- **OBSTRUCTIVE AIRWAY DISEASES**: characterized by an increase in resistance to airflow caused by partial or complete obstruction at any level

2- **RESTRICTIVE DISEASES**: characterized by reduced expansion of lung parenchyma and decreased total lung capacity.

Restrictive defects occur in two general conditions:

1. chest wall disorders in the presence of normal lungs:

- severe obesity, diseases of the pleura, and neuromuscular disorders that affect the respiratory muscles

2. acute or chronic interstitial lung diseases:

- The classic **acute** restrictive disease is **ARDS**.
- **Chronic restrictive diseases** include the pneumoconioses, interstitial fibrosis of unknown etiology, and sarcoidosis.



A 58-year-old man with ischemic heart disease undergoes coronary artery bypass graft surgery under general anesthesia. Two days postoperatively, he experiences increasing respiratory difficulty with decreasing arterial oxygen saturation. On physical examination, his heart rate is regular at 78/min, respirations are 25/min, and blood pressure is 135/85 mmHg. The hemoglobin concentration has remained unchanged, at 13.7 g/dL, since surgery. After he coughs up a large amount of mucoid sputum, his condition improves. Which of the following types of atelectasis does he most likely have?

A) Compression

B) Contraction

C) Resorption

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THANK YOU!