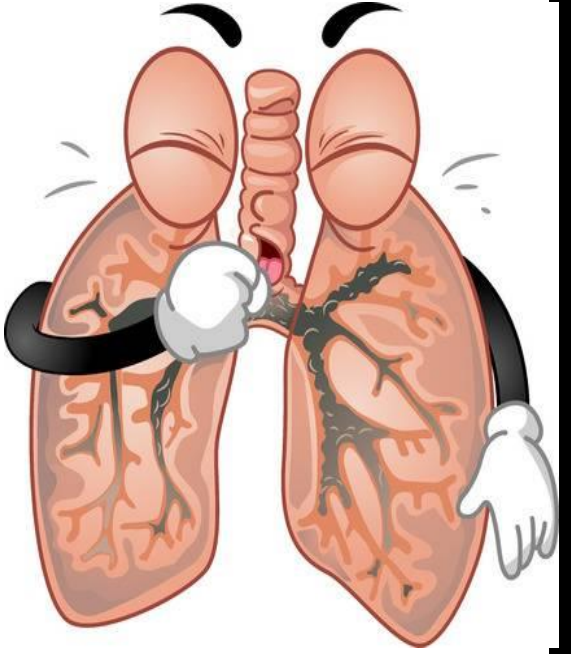

THE RESPIRATORY SYSTEM

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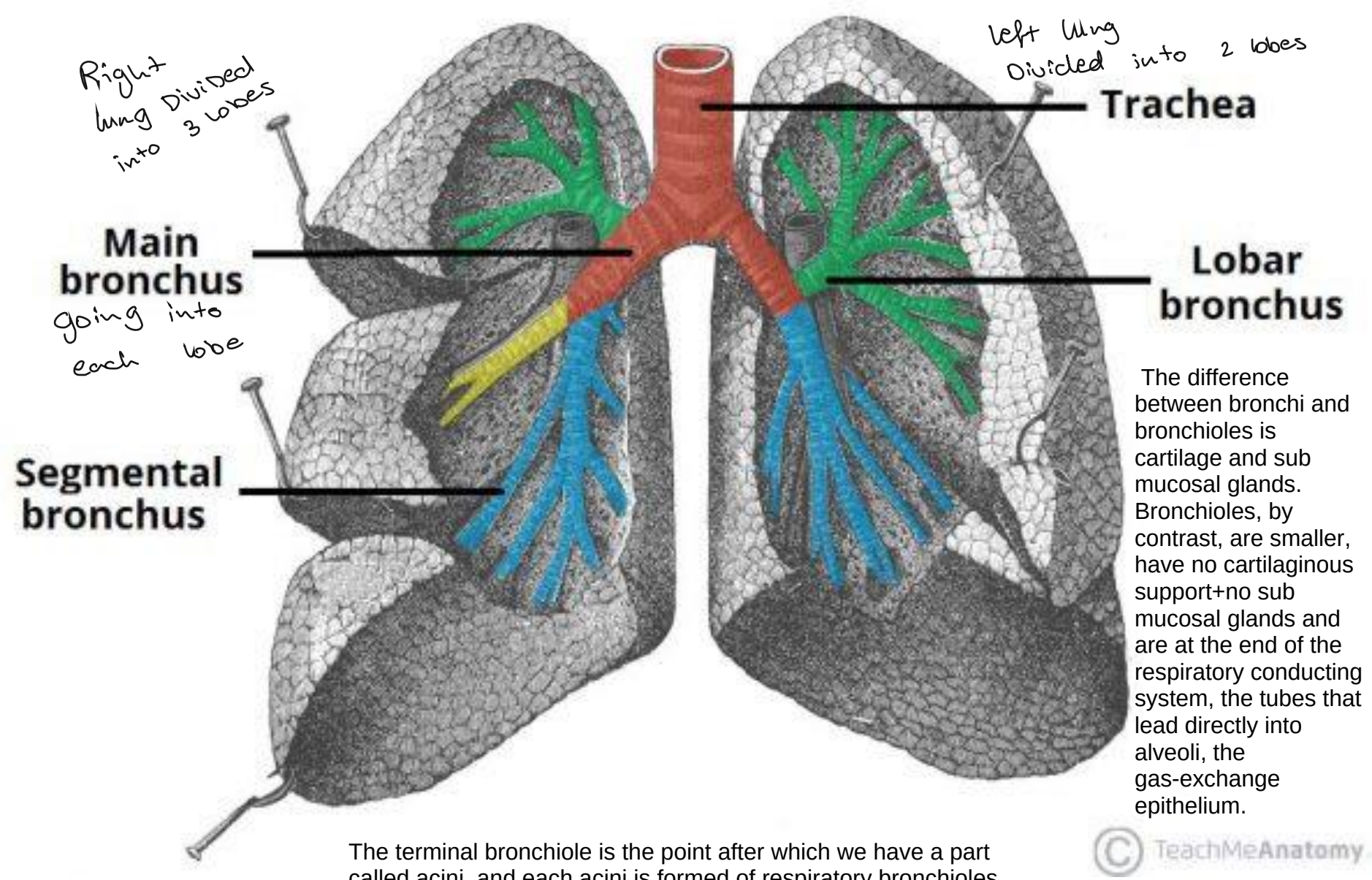
https://www.123rf.com/photo_38644498_stock-illustration-mascot-illustration-of-the-lungs-coughing-violently.html

OBJECTIVES:

- Function and anatomy
- Microscopic structure of the alveolar wall
- 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.

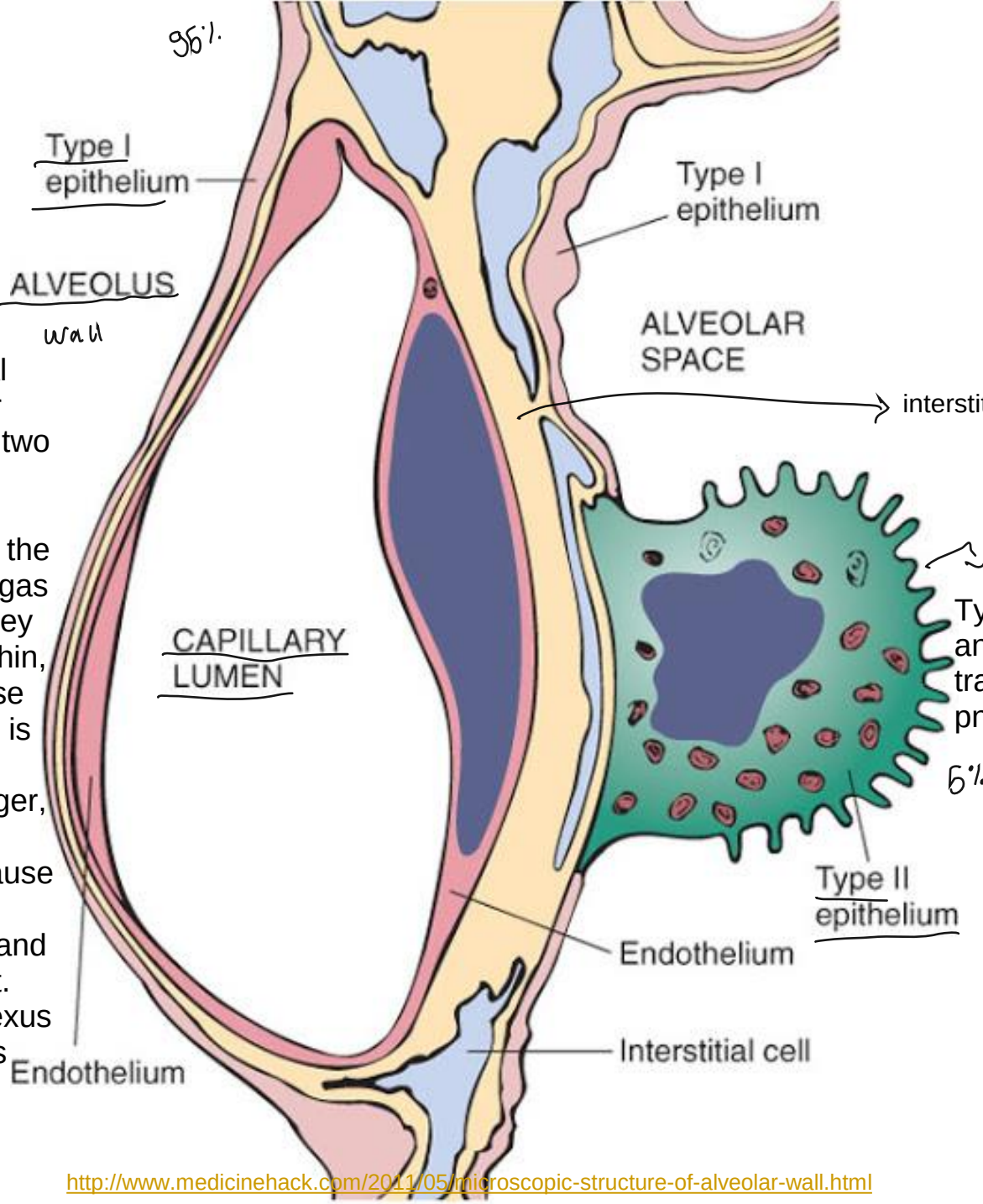


The difference between bronchi and bronchioles is cartilage and sub mucosal glands. Bronchioles, by contrast, are smaller, have no cartilaginous support+no sub mucosal glands and are at the end of the respiratory conducting system, the tubes that lead directly into alveoli, the gas-exchange epithelium.

The terminal bronchiole is the point after which we have a part called acini, and each acini is formed of respiratory bronchioles and alveolar duct and alveolar sac, this is the blind end of the lung, the walls in acini are entirely made of alveoli and these walls alow the ultimate gas exchange.

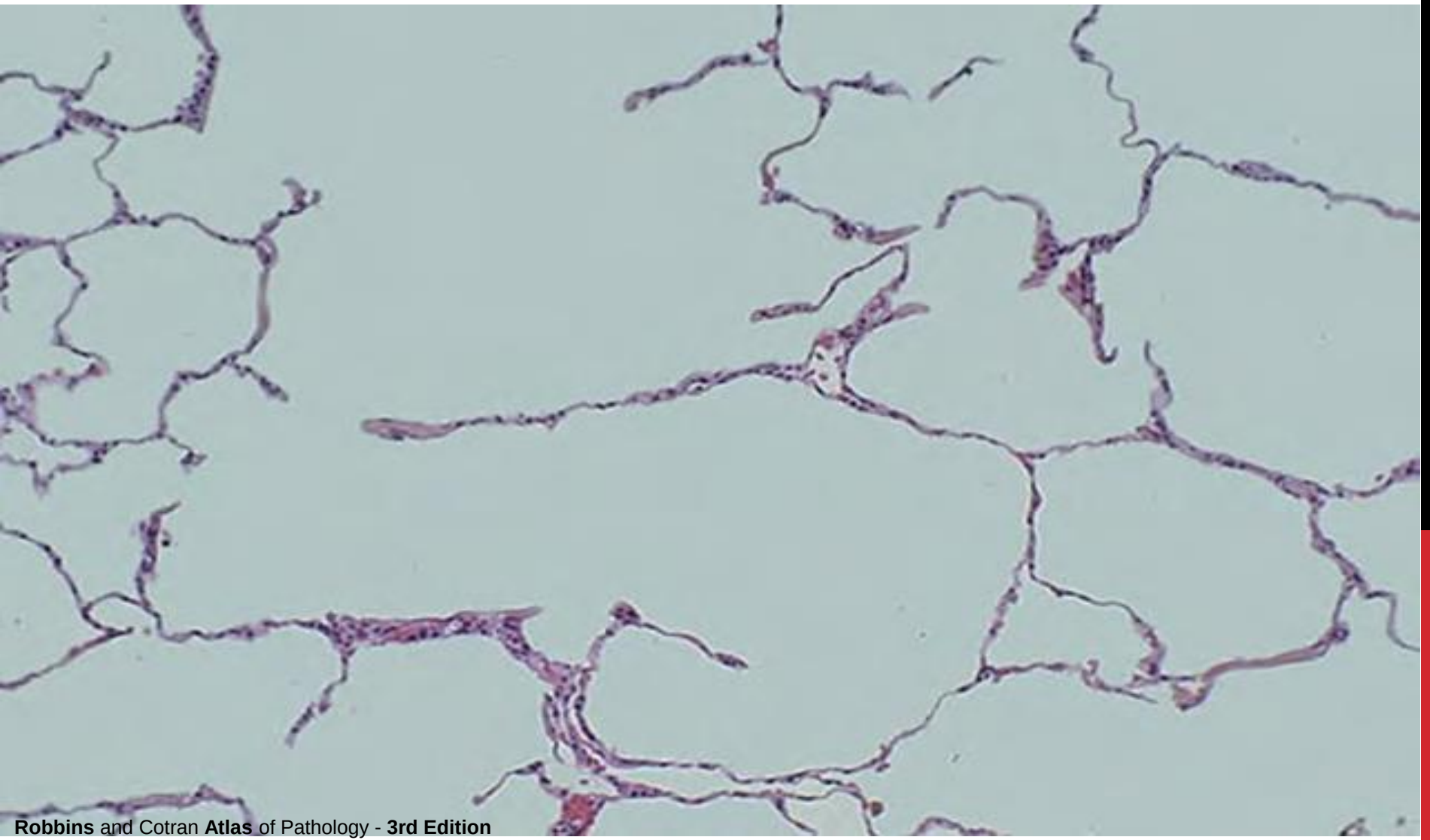
By TeachMeSeries Ltd (2019)https://teachmeanatomy.info/thorax/organs/tracheobronchial-tree/

The surface epithelial cells of the alveoli, or pneumocytes, are of two types. The type I pneumocytes(The majority) form part of the barrier across which gas exchange occurs. They can be identified as thin, squamous cells whose most obvious feature is their nuclei. Type II pneumocytes are larger, They appear foamier than type I cells because they contain phospholipid bodies and pulmonary surfactant. Capillaries form a plexus around each alveolus



http://www.medicinehack.com/2011/05/microscopic-structure-of-alveolar-wall.html

ATELECTASIS



Robbins and Cotran **Atlas** of Pathology - **3rd Edition**

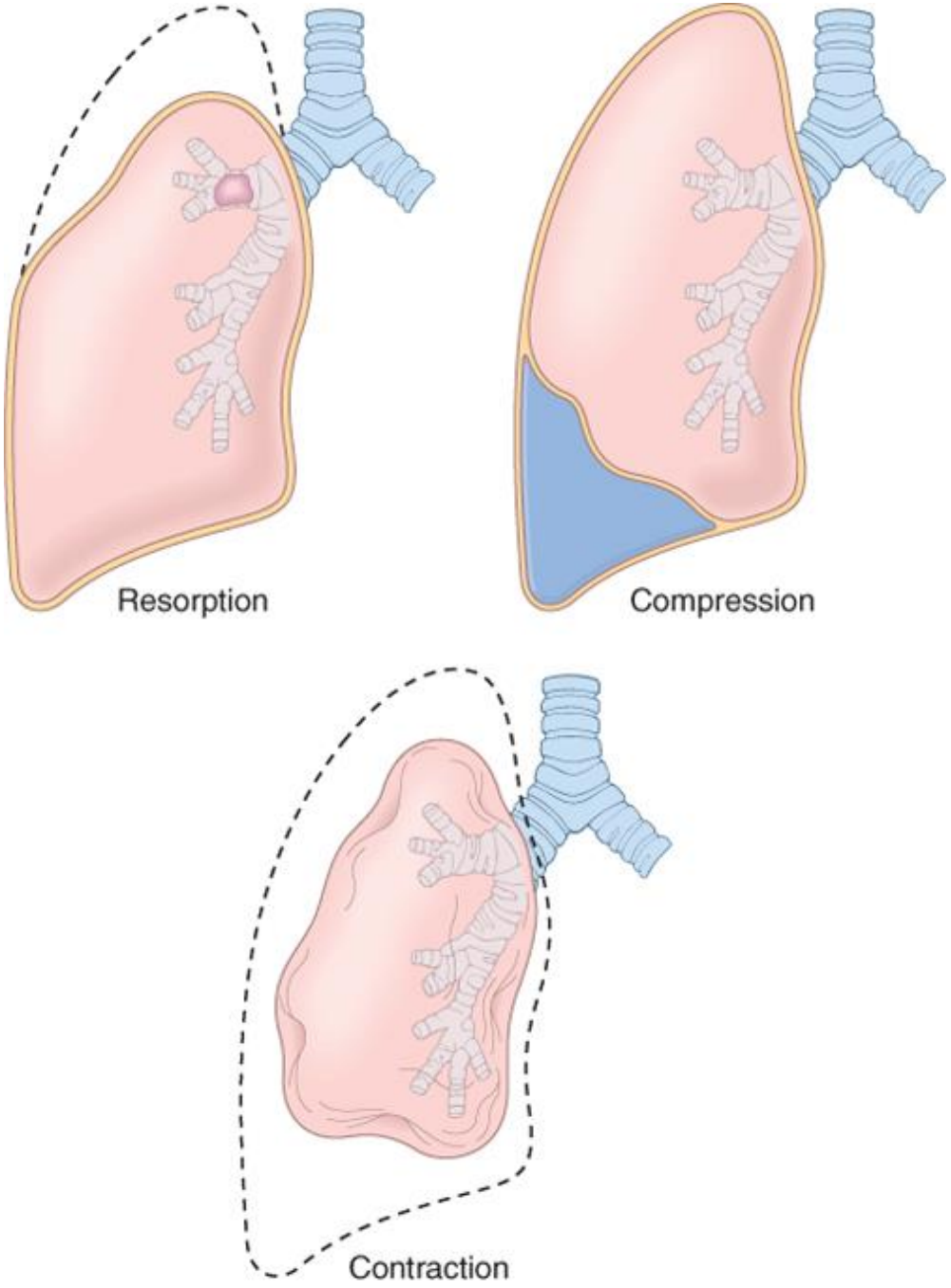
ATELECTASIS

- is loss of lung volume caused by inadequate expansion of air spaces. (The lung Collapsed, we lost a space for gas exchange) And that's why There is an
- It results in shunting of inadequately oxygenated blood from pulmonary arteries into veins, thus giving rise to a ventilation perfusion imbalance and hypoxia.

It is a condition in which one or more areas of the lung receive oxygen but no blood flow, or they receive blood flow but no oxygen due to some diseases and disorders.

THREE TYPES:

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



1.RESORPTION ATELECTASIS

Reversible and lungs can go back to it's normal size (No scarring, everything is possible)

- Due to total obstruction of a bronchus preventing air from reaching distal airways.
- The air already present gradually becomes absorbed, and alveolar collapse follows.

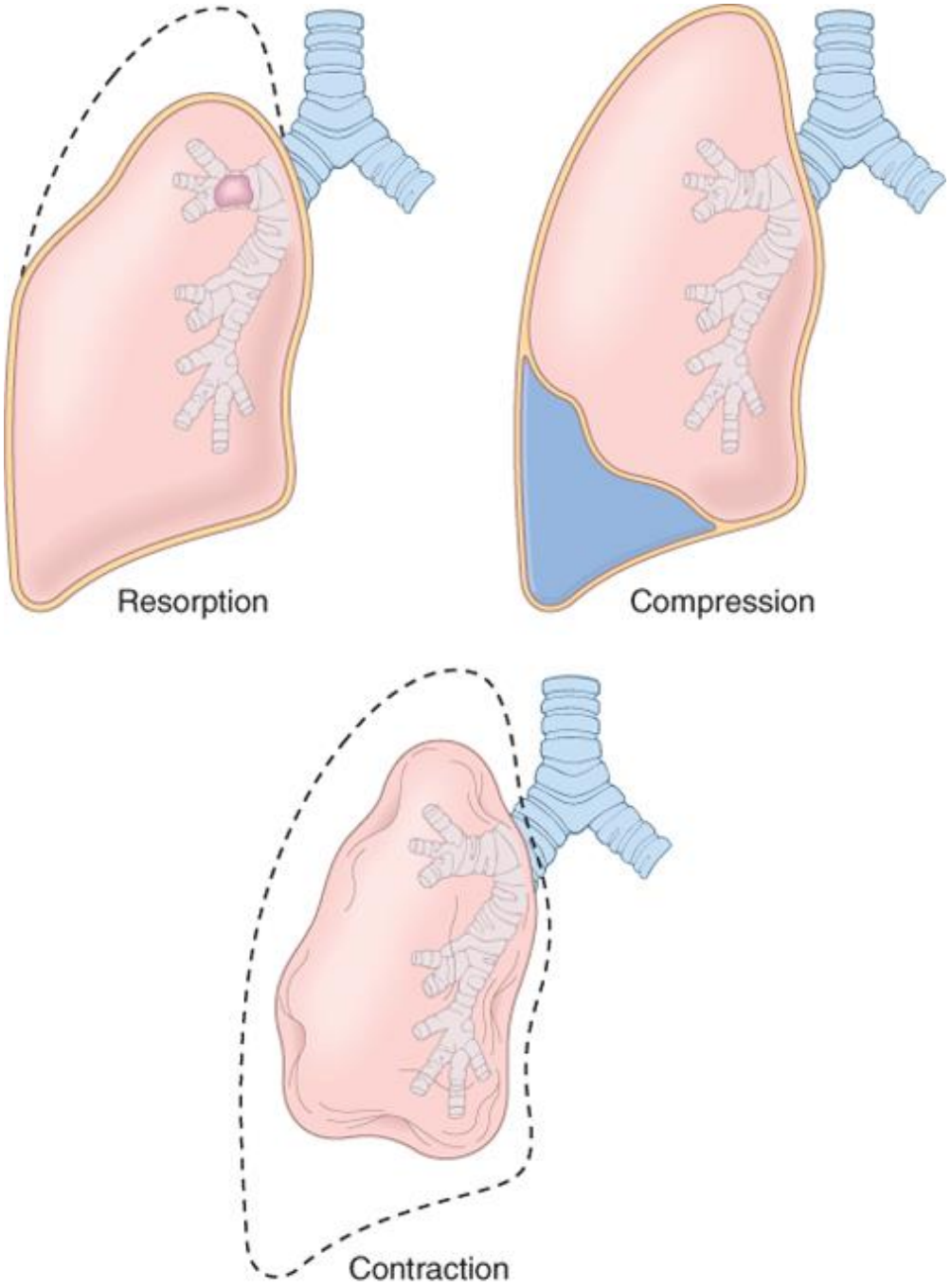
RESORPTION ATELECTASIS, CAUSED BY:

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

Chronic bronchitis is one type of COPD (chronic obstructive pulmonary disease). The inflamed bronchial tubes produce a lot of mucus. This leads to coughing and difficulty breathing

Bronchiectasis is a disease in which there is permanent enlargement of parts of the airways of the lung

Bronchial asthma is a chronic inflammatory disorder of the airways associated with airway hyper responsiveness that leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing particularly at night or in the early morning.



The problem is with the pleural cavity and it's reversible

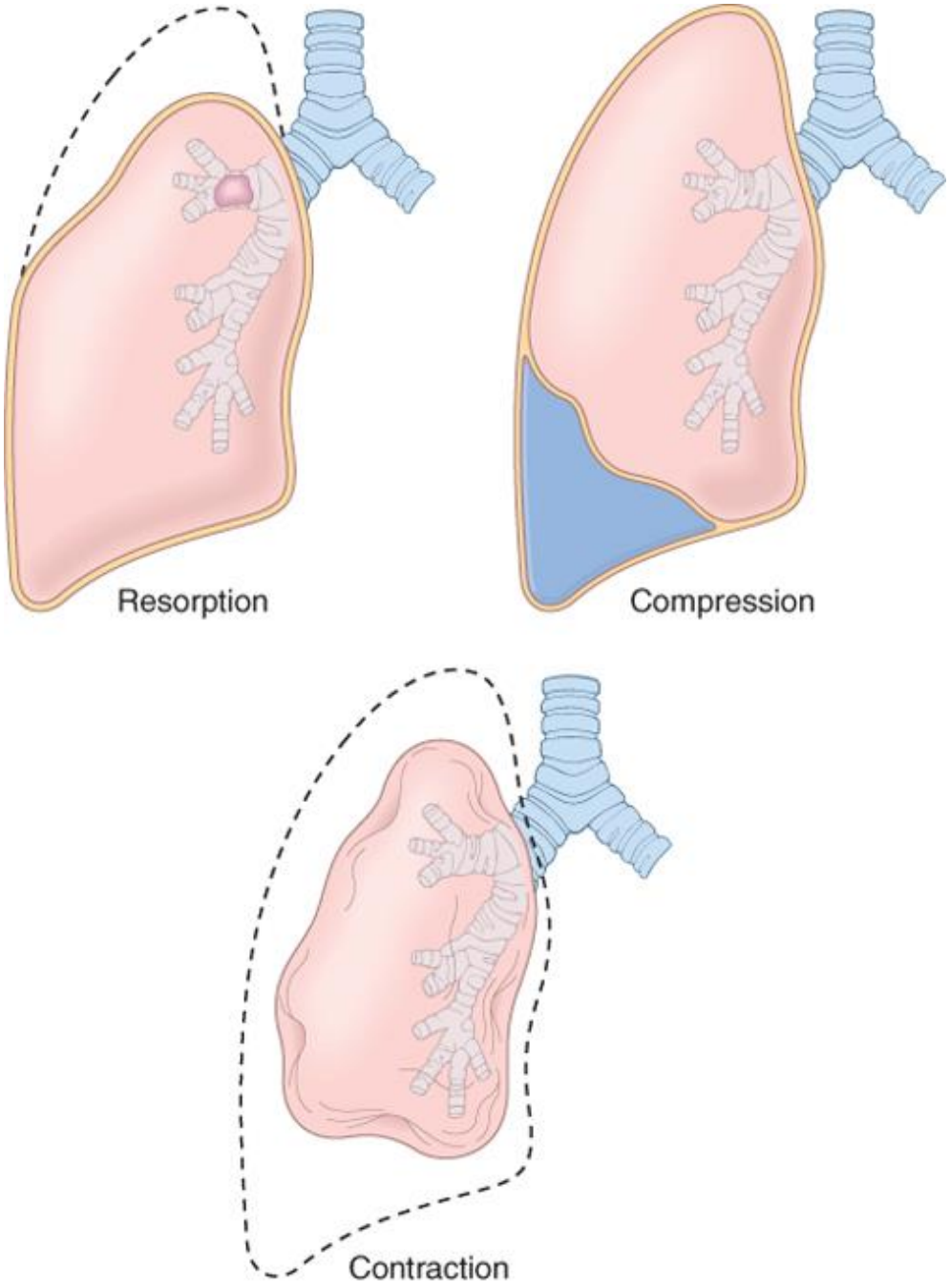
2. COMPRESSION ATELECTASIS

Common cause: stab wound

- caused by accumulation of fluid, blood, or air within pleural cavity, which mechanically collapse adjacent lung.

a. Pleural effusion like in Congestive Heart Failure
b. Pneumothorax: air in the pleural cavity
c. Infection can cause compression atelectasis

pneumothorax occurs when air leaks into the space between your lung and chest wall. This air pushes on the outside of your lung and makes it collapse.



3. CONTRACTION ATELECTASIS (CICATRIZATION ATELECTASIS)

↳ scar formation

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

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

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

Diagnosis in ARDS is by exclusion

Graded according to the arterial blood gases

- The epidemiology and definition are evolving.

- Formerly considered to be the severe end of a spectrum of acute lung injury

Opacity: air space filling, It appears as white spots on the X-Ray covering areas of the lung

Causes Dyspnea

- **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.

Respiratory failure is a condition in which your blood doesn't have enough oxygen or has too much carbon dioxide. Sometimes you can have both problems. When you breathe, your lungs take in oxygen. The oxygen passes into your blood, which carries it to your organs.

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS):

- graded based on the severity of the changes in arterial blood oxygenation.

- extensive bilateral injury to alveoli known histologically as **diffuse alveolar damage (DAD)**

Diffuse alveolar damage (DAD) represents a global injury to the gas-exchange surfaces that is caused by disruption of the blood-air barrier leading to exudative edema and fibrosis, and resulting in severely impaired blood and tissue oxygenation

SEVERE ARDS:

- **characterized by rapid onset of life-threatening:**

  a. **respiratory insufficiency**.

  b. **Cyanosis**

  c. **Severe arterial hypoxemia** that becomes refractory to oxygen therapy and may progress to multisystem organ failure.

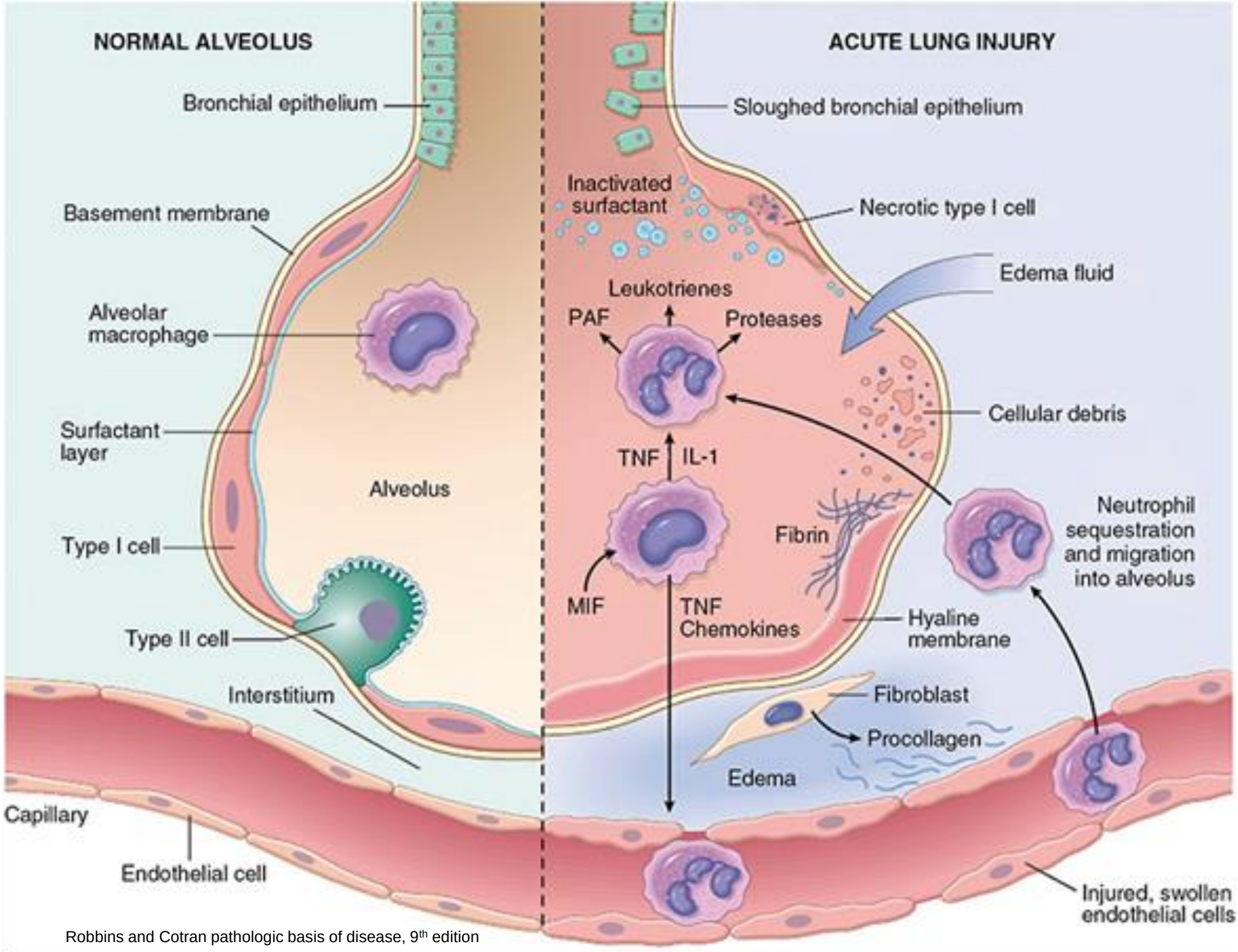
Hypoxemia is defined as a condition where arterial oxygen tension (Pao2) is below normal (normal Pao2 = 80–100mmHg). Hypoxia is defined as the failure of oxygenation at the tissue level. ... Hypoxia and hypoxemia may or may not occur together. Generally, the presence of hypoxemia suggests hypoxia (Extra information for better understanding)

ARDS; TRIGGERS:

- pneumonia (35%–45%)
- sepsis (30%–35)
- Aspiration
- trauma (including brain injury, abdominal surgery, and multiple fractures)
- pancreatitis
- transfusion reactions.

Pneumonia is an infection that inflames the air sacs in one or both lungs. The air sacs may fill with fluid or pus (purulent material), causing cough with phlegm or pus, fever, chills, and difficulty breathing. A variety of organisms, including bacteria, viruses and fungi, can cause pneumonia (extra information)

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



Robbins and Cotran pathologic basis of disease, 9th edition

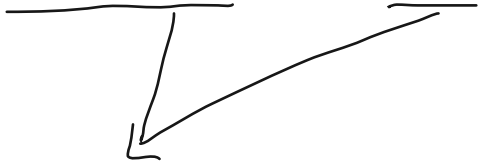
PATHOGENESIS:

Patient has pneumonia (An example) which triggers injury

- the integrity of the alveolar-capillary membrane is compromised by endothelial and epithelial injury.

The injured cells start to release certain cytokines that will aid in inflammatory reaction and will activate pulmonary macrophages

- 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.



They act in chemotaxis and they also Attract leukocytes (mainly neutrophils) from the circulation into the tissues

- leading to endothelial activation and sequestration and activation & chemotaxsis 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

Neutrophils will produce more chemical mediators and will released more cytokines, some of them will result in formation of reactive oxygen species and proteases that will cause damage/necrosis and lysis of the epithelium lining, more fluid (filled with fibrils+ cell debris) will accumulate in the interstitum and the alveolar spaces and then the fluid will start to line up in the alveolar wall to form (hyaline membranes)

- **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.**

  → Anti-oxidants and anti-proteases

HISTOLOGY:

- In the acute phase of ARDS :

  - The most characteristic finding is the presence of **hyaline membranes**—> Made of fibril rich edema fluid and the cellular debris

  - consists of fibrin-rich edema fluid admixed with remnants of necrotic epithelial cells (similar to respiratory distress syndrome of the newborn)

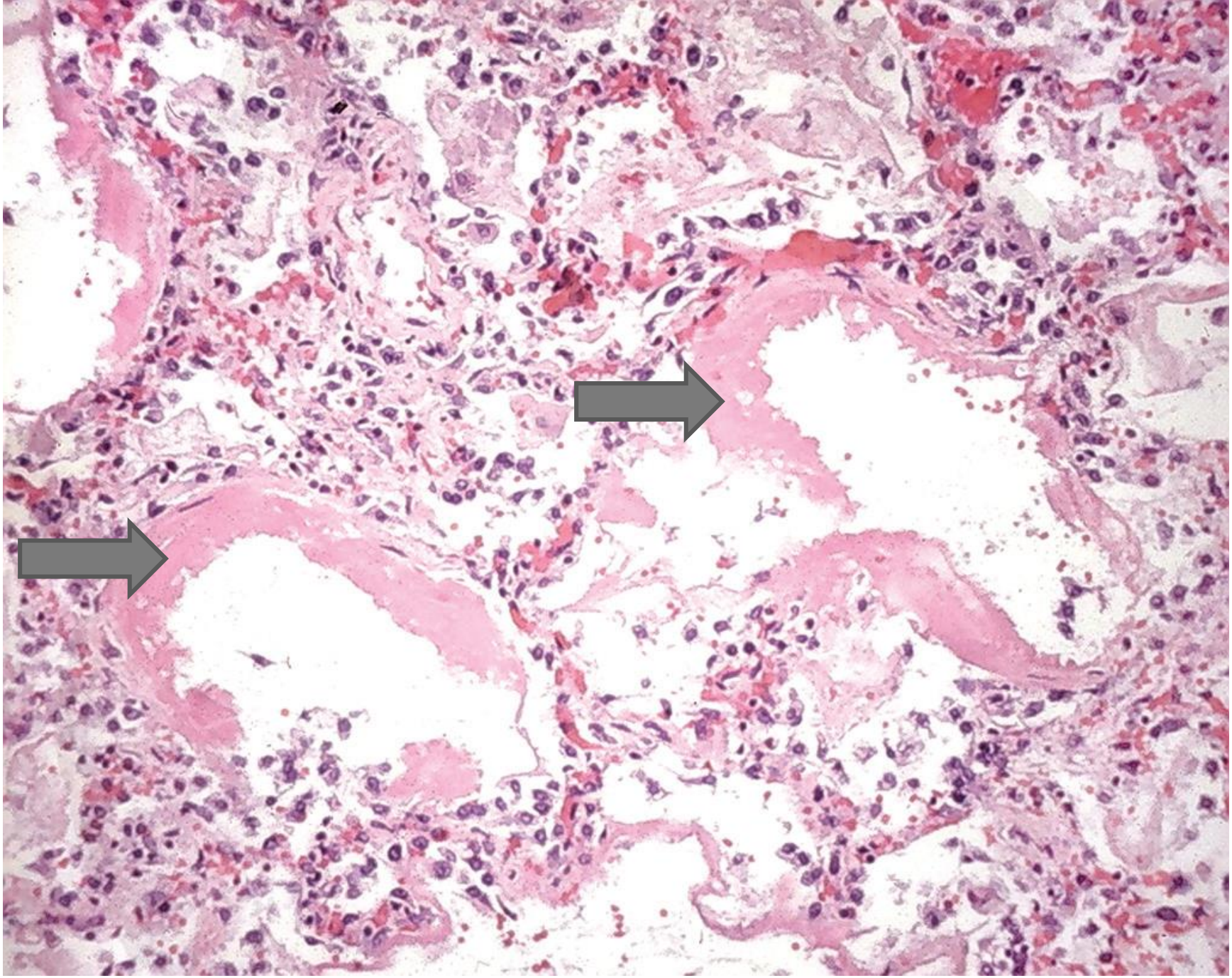


FIGURE 13.3A,ROBBINS BASIC PATHOLOGY, 10TH EDITION

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.

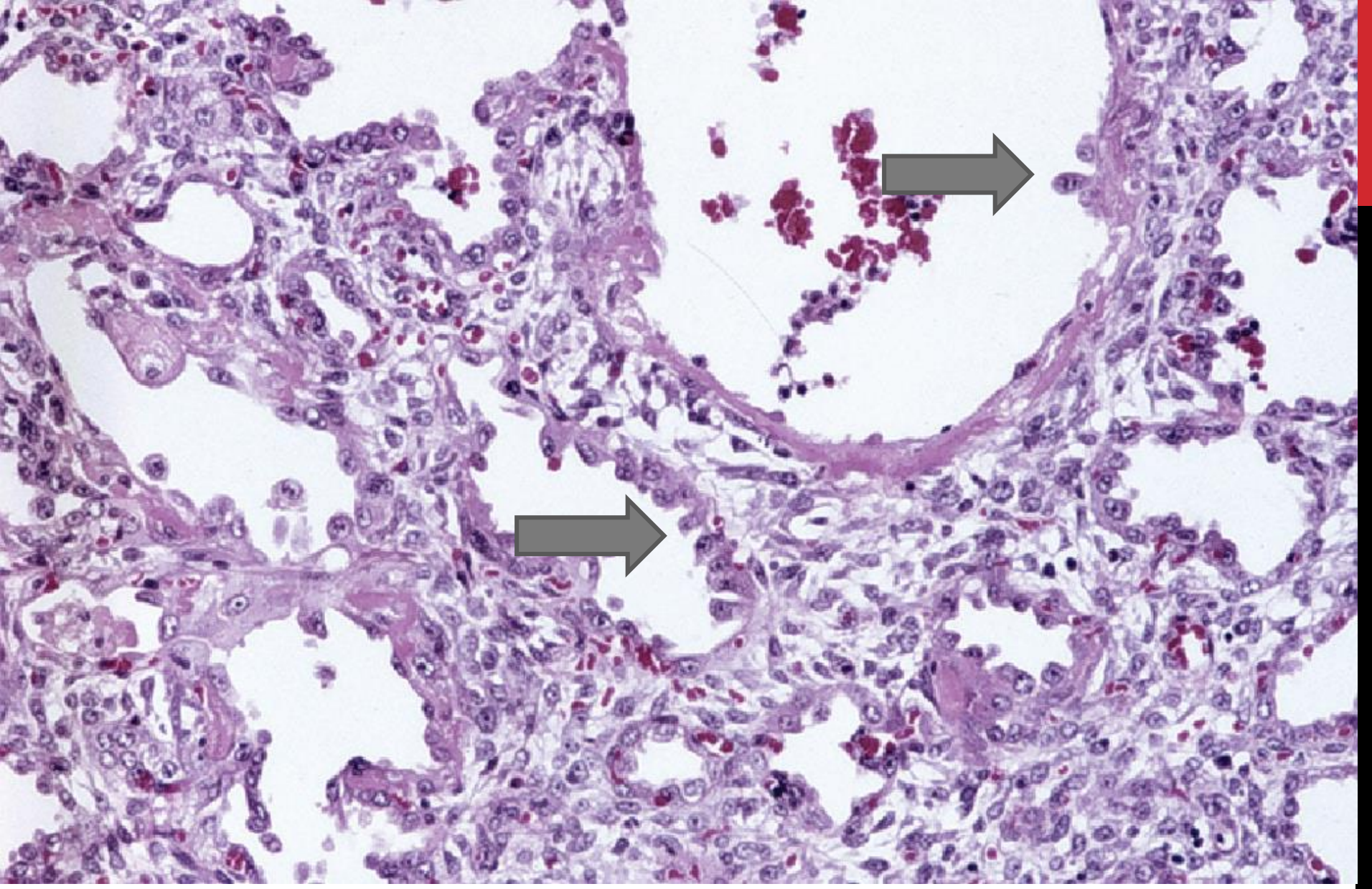


FIGURE 13.3B,ROBBINS BASIC PATHOLOGY, 10TH EDITION

CLINICAL FEATURES

- Patients are hospitalized for one of the predisposing conditions

abnormally rapid breathing

Shortness of breath

- 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

OUTCOME:

- The overall hospital mortality rate is 38.5%.

- Most patients who survive the acute insult recover normal respiratory function within 6 to 12 months, but the rest develop diffuse interstitial fibrosis leading to chronic respiratory insufficiency

↓

Chronic respiratory failure is a condition that results in the inability to effectively exchange carbon dioxide and oxygen, and induces chronically low oxygen levels or chronically high carbon dioxide levels.

PREDICTORS OF POOR PROGNOSIS

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

OBSTRUCTIVE VS. RESTRICTIVE

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 disea**ses include the **pneumoconioses, interstitial fibrosis of unknown etiology, and sarcoidosis**.

a group of interstitial lung diseases caused by the inhalation of certain dusts and the lung tissue's reaction to the dust.

is an inflammatory disease that affects the lung and lymph nodes

THANK YOU!