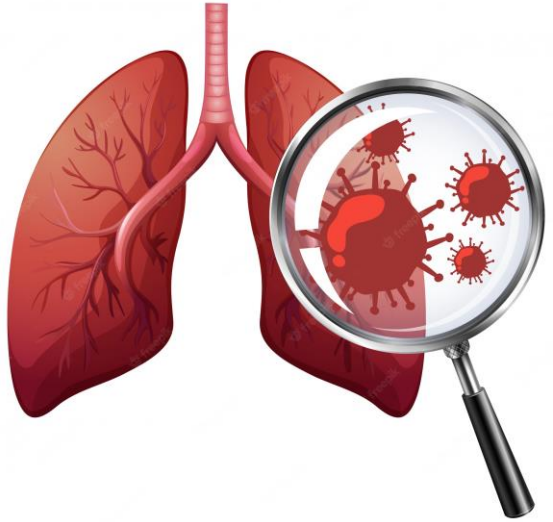


RSM – 1

Atelectasis and Pulmonary vascular Diseases

Dr. Eman Kreishan, M.D.

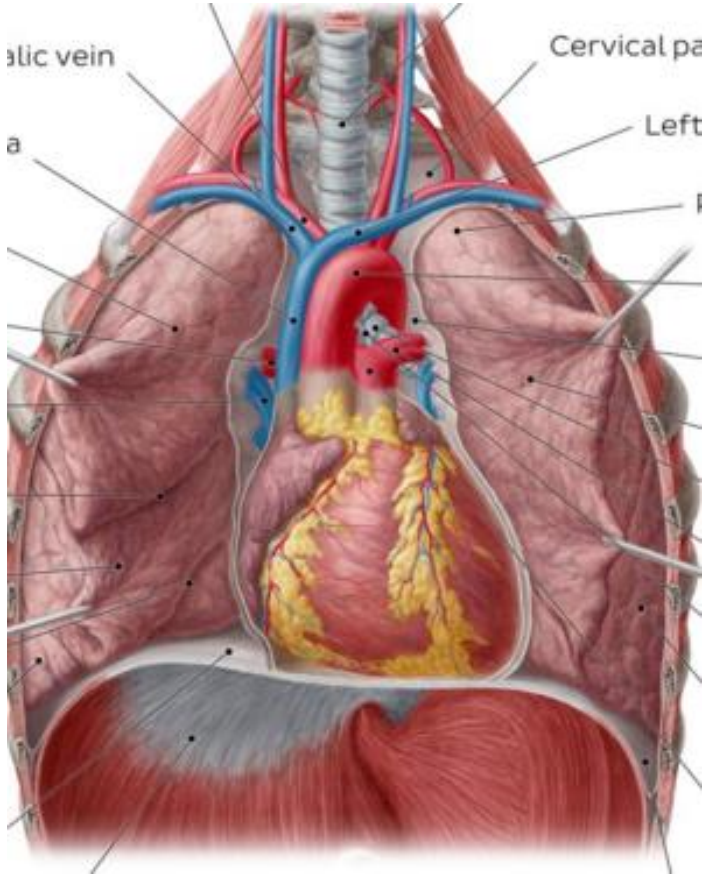
11-10-2022

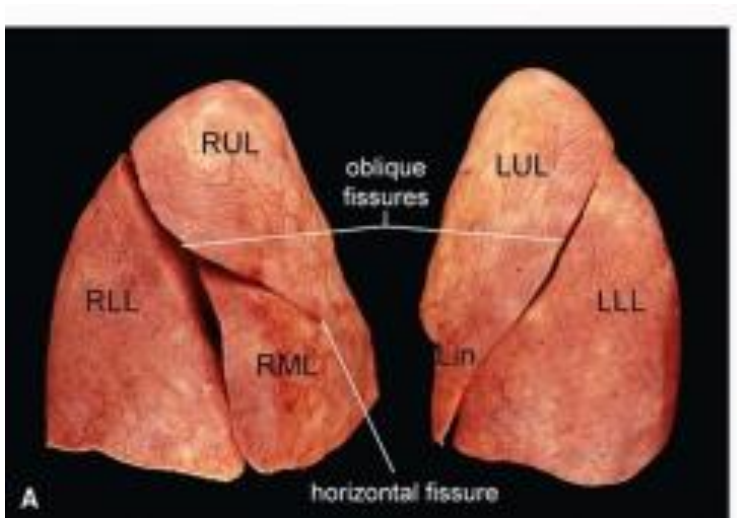
NOTES

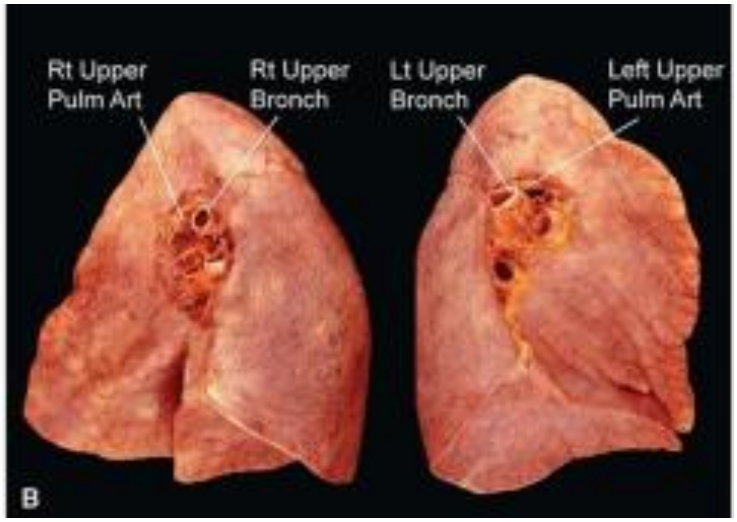
Office hours: Sunday and Tuesday, 12-2 pm.

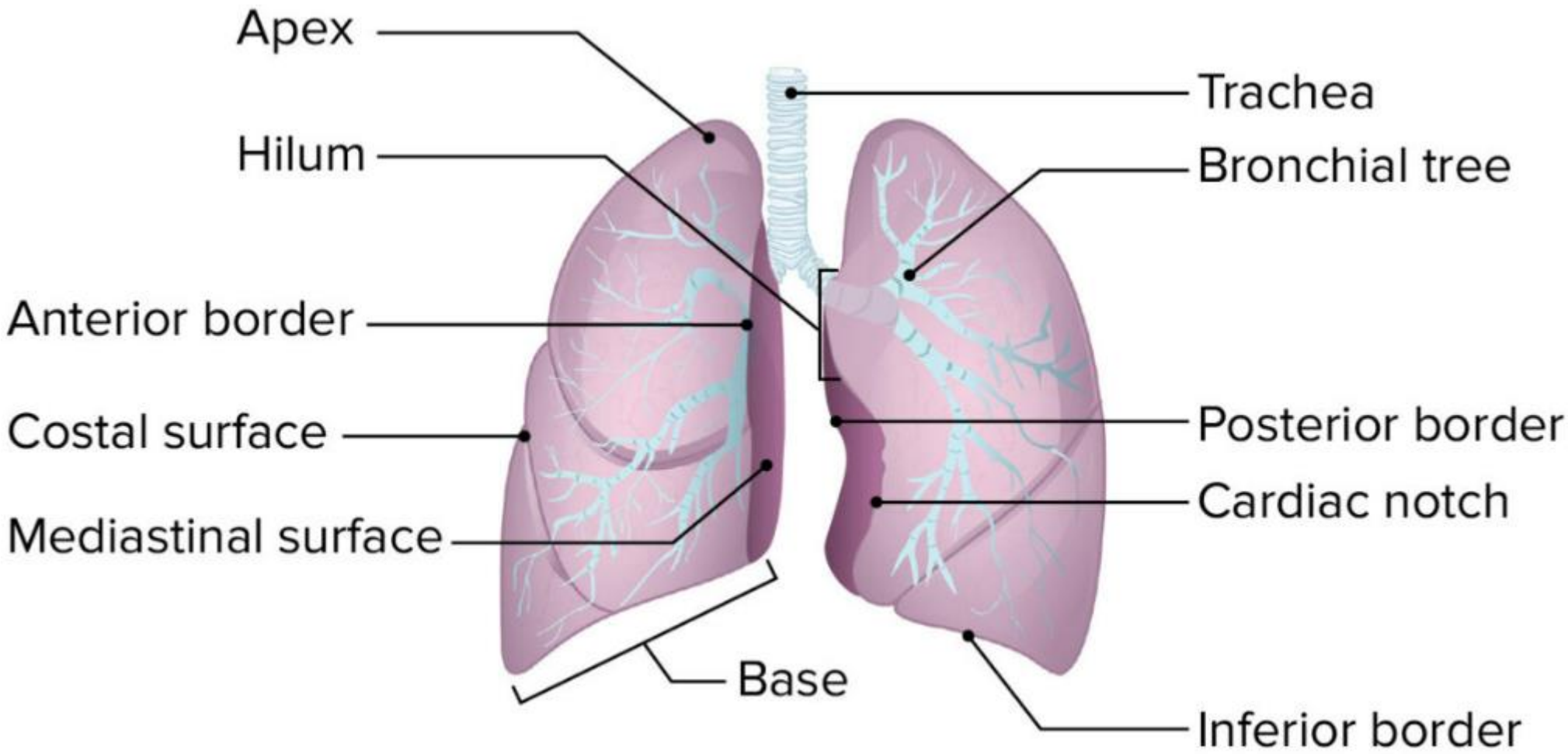
My office at second floor.

Introduction.. Anatomy

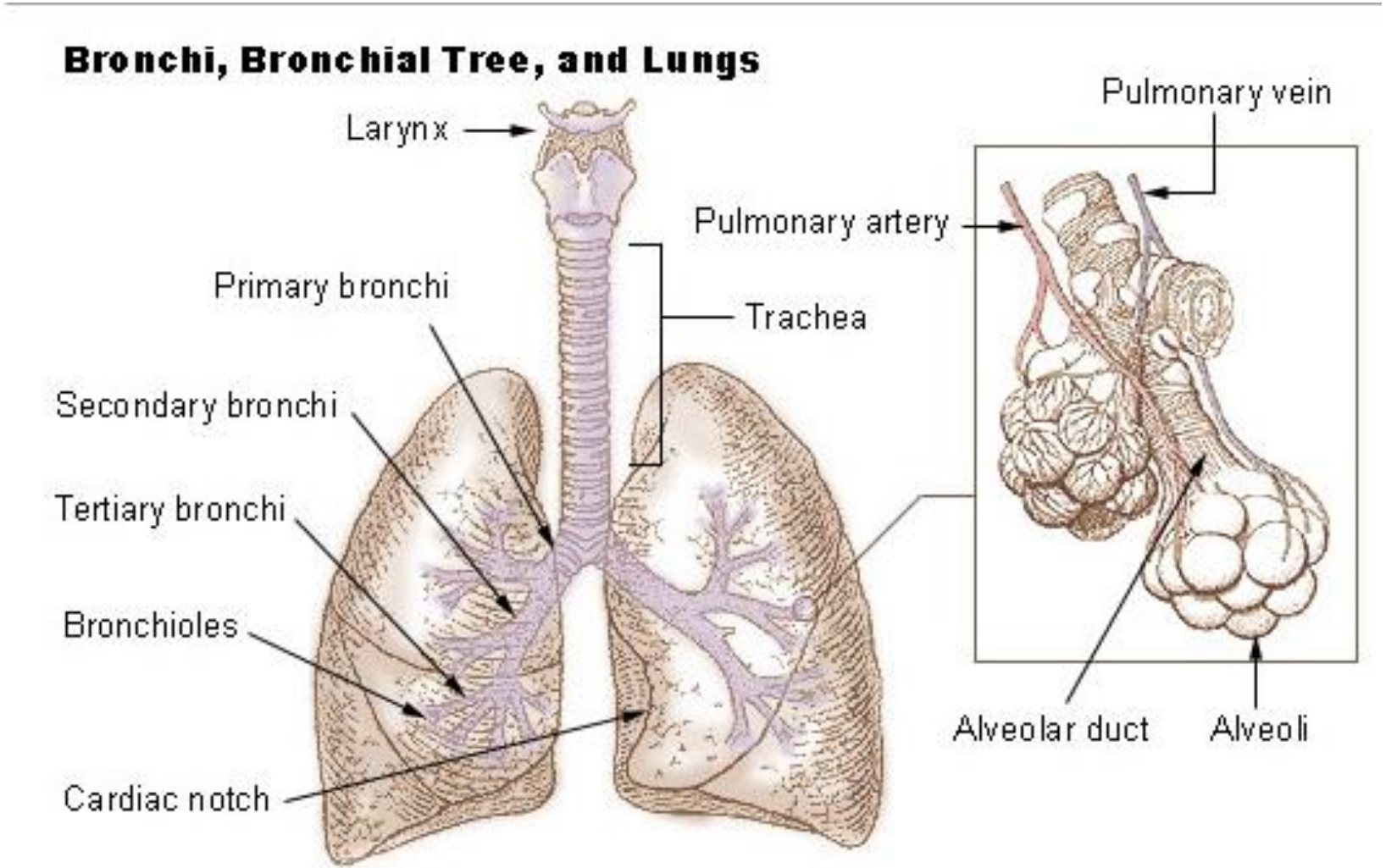




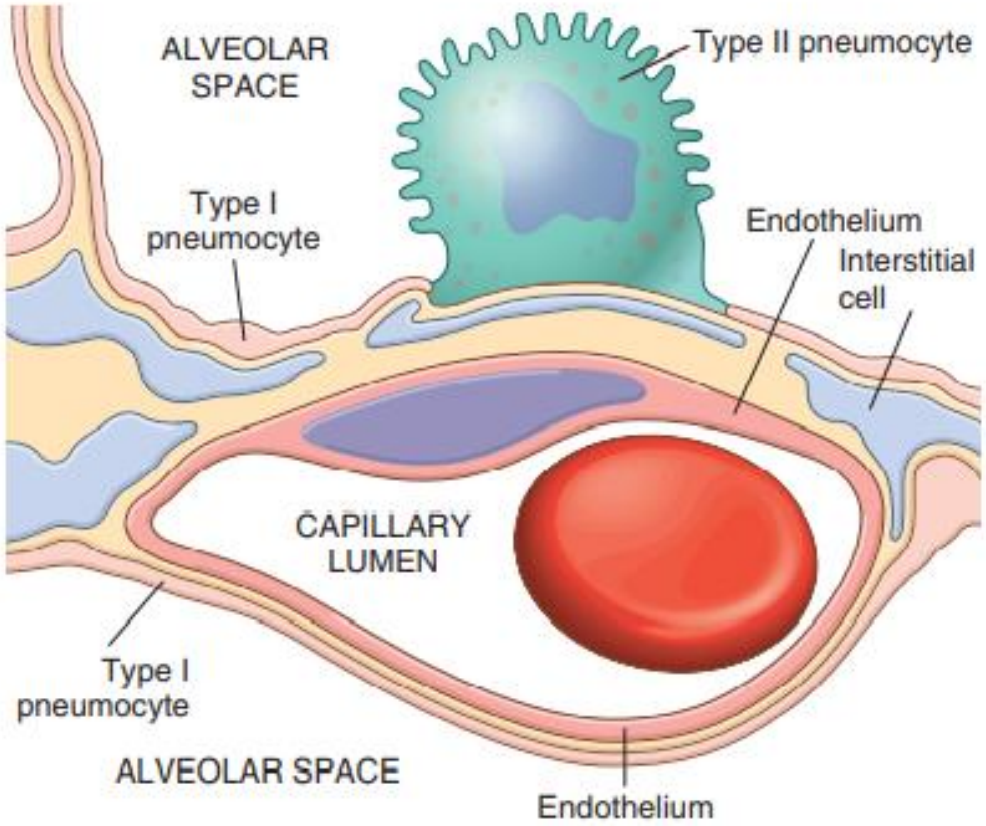


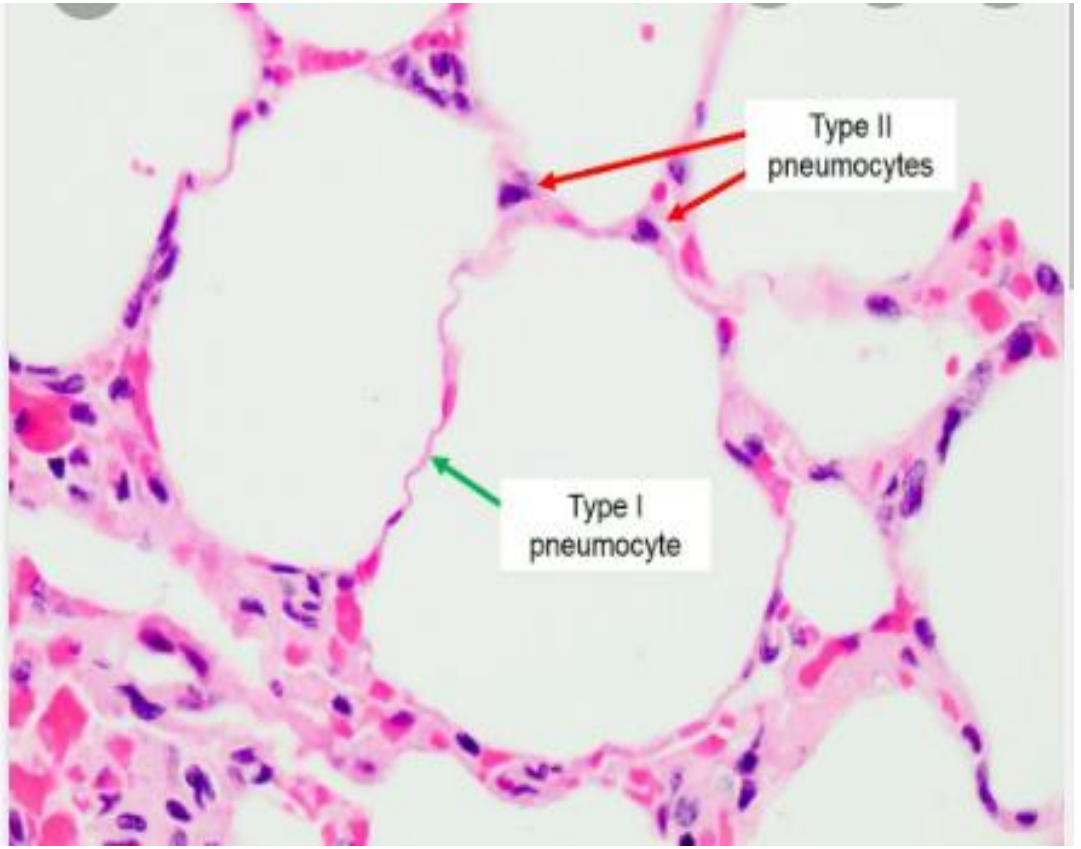

Apex
Hilum
Anterior border
Costal surface
Mediastinal surface
Base
Trachea
Bronchial tree
Posterior border
Cardiac notch
Inferior border

All the respiratory passages from the trachea to the respiratory bronchioles are called the tracheobronchial tree

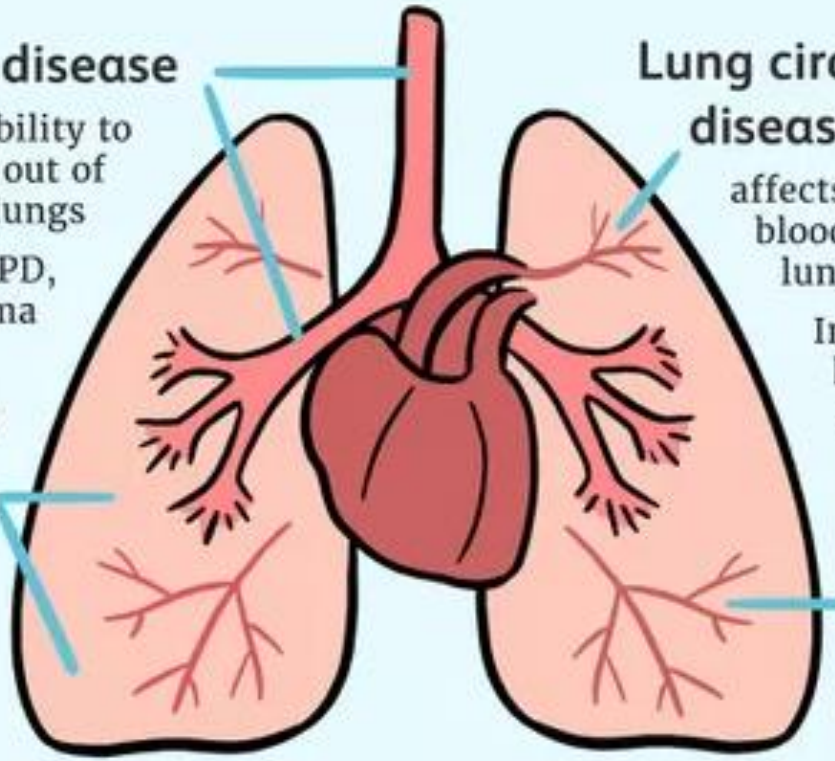


HISTOLOGY


ALVEOLAR SPACE
Type II pneumocyte
Type I pneumocyte
Endothelium
Interstitial cell
CAPILLARY LUMEN
Type I pneumocyte
ALVEOLAR SPACE
Endothelium


Type II pneumocytes
Type I pneumocyte

Types of Lung Disease



Airway disease

affects the body's ability to move air in and out of the lungs

Includes: asthma, COPD, bronchitis, emphysema

Lung tissue disease

damaged tissue from scarring or injury

Includes: pulmonary fibrosis, sarcoidosis

Lung circulation disease

affects the circulation of blood to and from the lungs

Includes: pulmonary hypertension, pulmonary edema

RESPIRATORY MODULE PATHOLOGY LECTURES

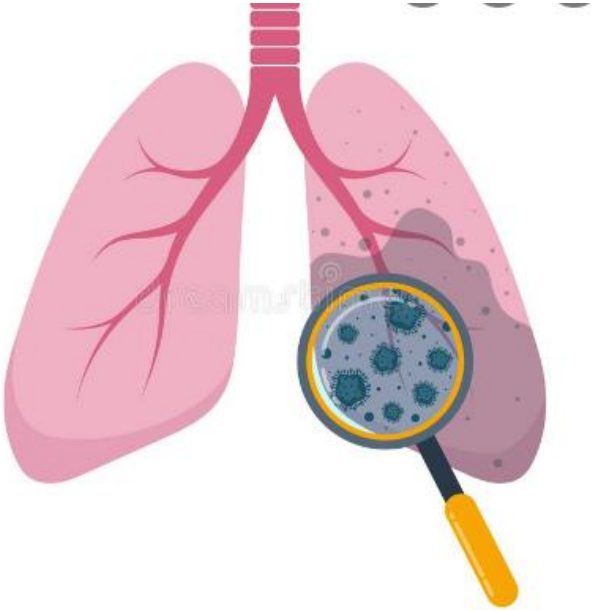
Atelectasis and Pulmonary Diseases of Vascular Origin.

Obstructive Lung (Airway) Diseases-2.

Chronic Interstitial (Restrictive, Infiltrative) Lung Diseases.

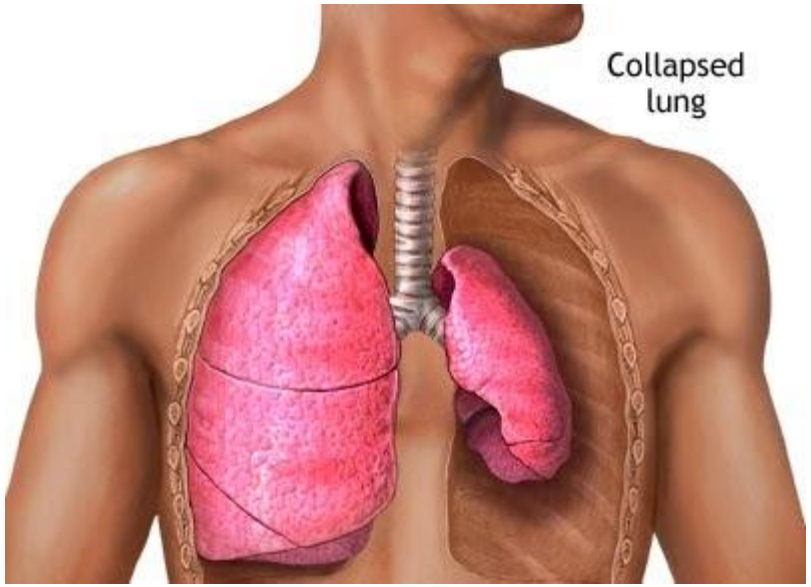
Pulmonary Infections-2.

Lung Tumors -2.



ATELECTASIS

Collapse and 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 a ventilation perfusion imbalance and hypoxia.



ATELECTASIS IS CLASSIFIED INTO THREE FORMS:

1. Resorption atelectasis:

occurs when an obstruction prevents air from reaching distal airways.

Any air present gradually becomes absorbed, and alveolar collapse follows.

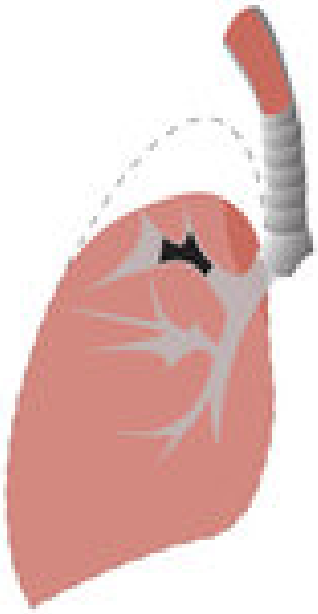
The most common causes are:

intrabronchial mucous or mucopurulent plugs.

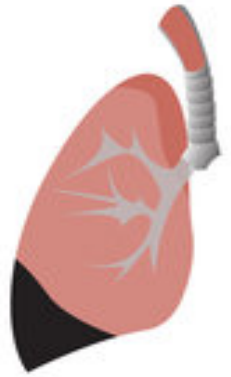
foreign body aspiration.

bronchial asthma and bronchiectasis.

chronic bronchitis and intrabronchial tumor.



Resorption atelectasis

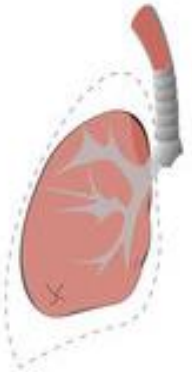


Compression atelectasis

2. Compression atelectasis:

associated with accumulation of fluid, blood, or air within the pleural cavity.

Usually associated with congestive heart failure.



Contraction atelectasis

3. Contraction atelectasis:

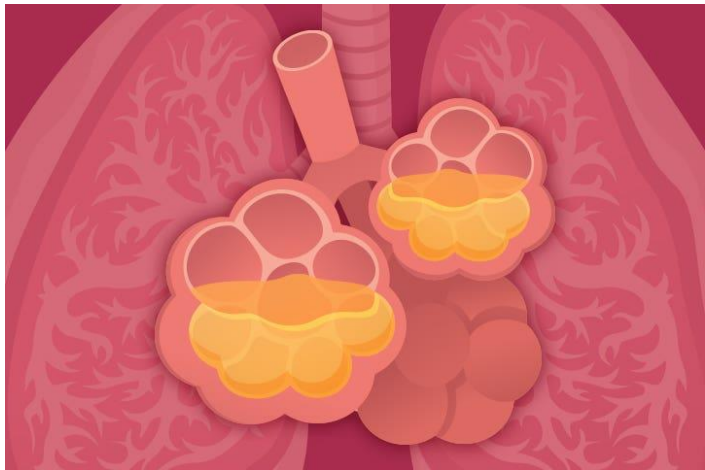
occurs when local or diffuse fibrosis affecting the lung

Acute respiratory distress syndrome

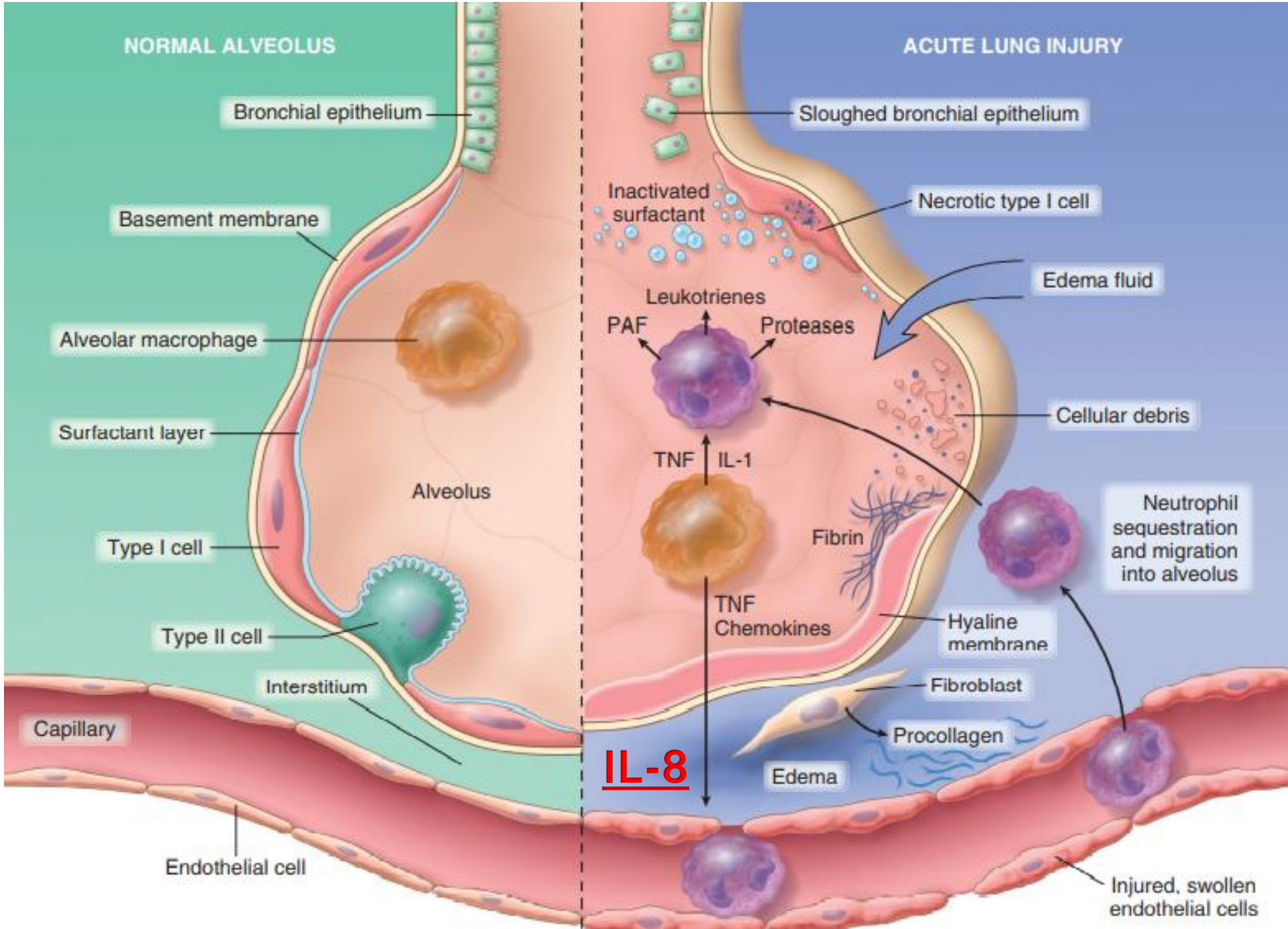
Respiratory failure occurring within 1 week of a known clinical insult with bilateral opacities on chest imaging, not fully explained by effusions, atelectasis, cardiac failure, or fluid overload.

Causes are diverse; the shared feature is that all lead to extensive bilateral injury to alveoli.

Severe ARDS is characterized by rapid onset of life threatening respiratory insufficiency, cyanosis, and severe arterial hypoxemia that is refractory to oxygen therapy.



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



PATHOGENESIS

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

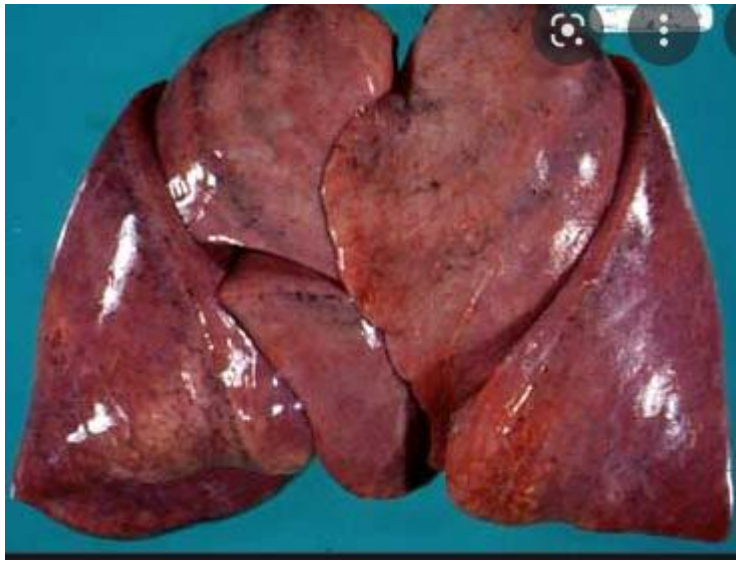
There is increased synthesis of interleukin 8 (IL-8), a potent neutrophil chemotactic and activating agent.

Release of IL-8, IL-1 and tumor necrosis factor (TNF), leads to endothelial activation and sequestration and activation of neutrophils in pulmonary capillaries

Activated neutrophils release a variety of products (e.g., reactive oxygen species, proteases) that damage the alveolar epithelium and endothelium.

The assault on the endothelium and epithelium causes vascular leak and loss of surfactant that render the alveolar unit unable to expand.

MORPHOLOGY



Acute phase:

Gross: the lungs are dark red, firm, airless, and heavy.

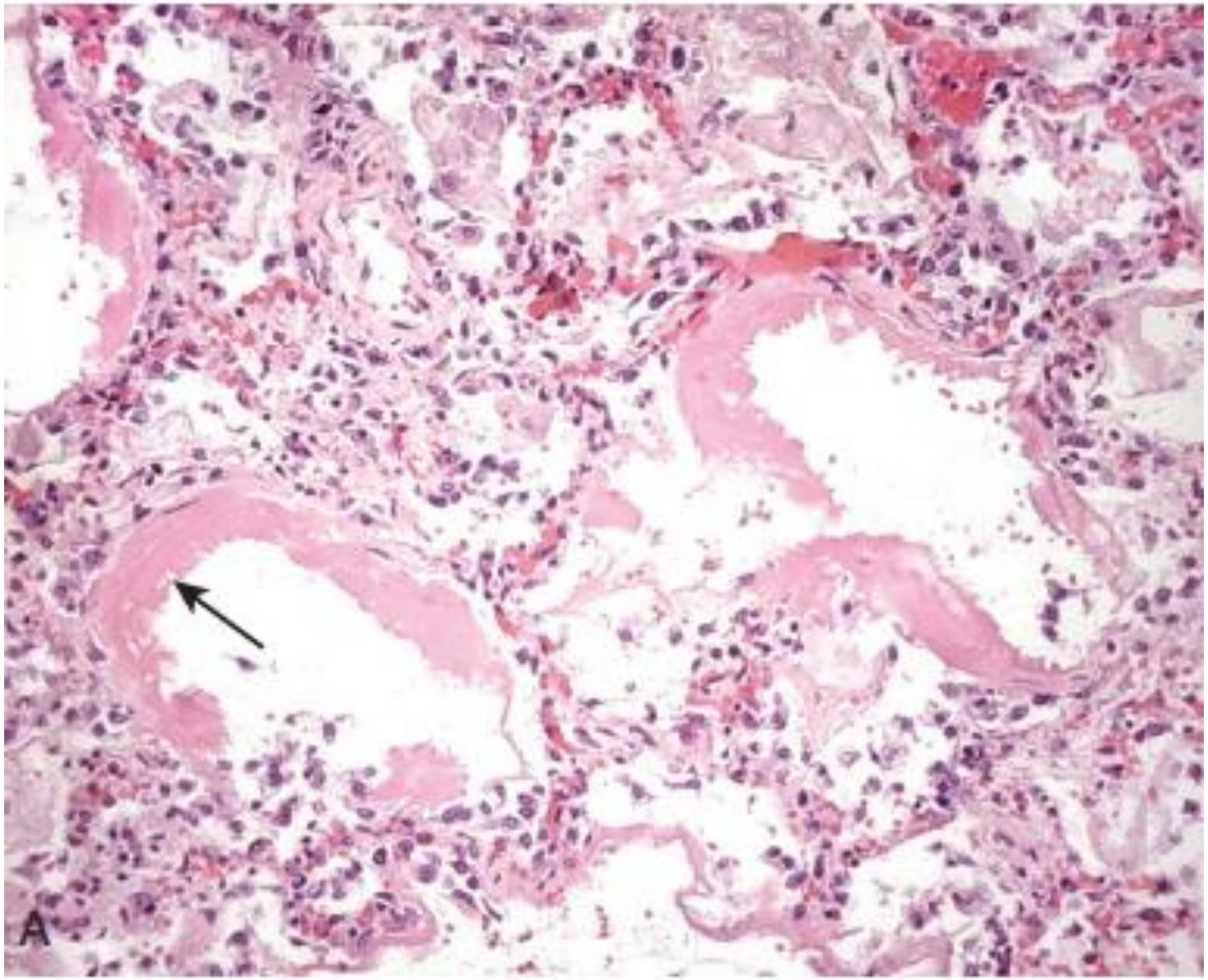
Microscopic examination :

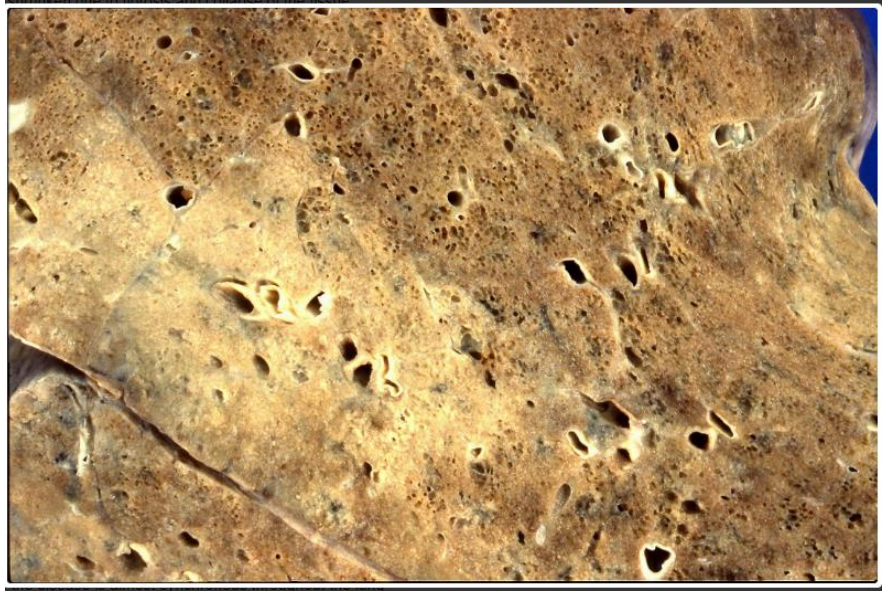
capillary congestion.

necrosis of alveolar epithelial cells.

interstitial and intraalveolar edema and hemorrhage .

❖The most characteristic finding is the presence of hyaline membranes (fibrin-rich edema fluid admixed with remnants of necrotic epithelial cells).





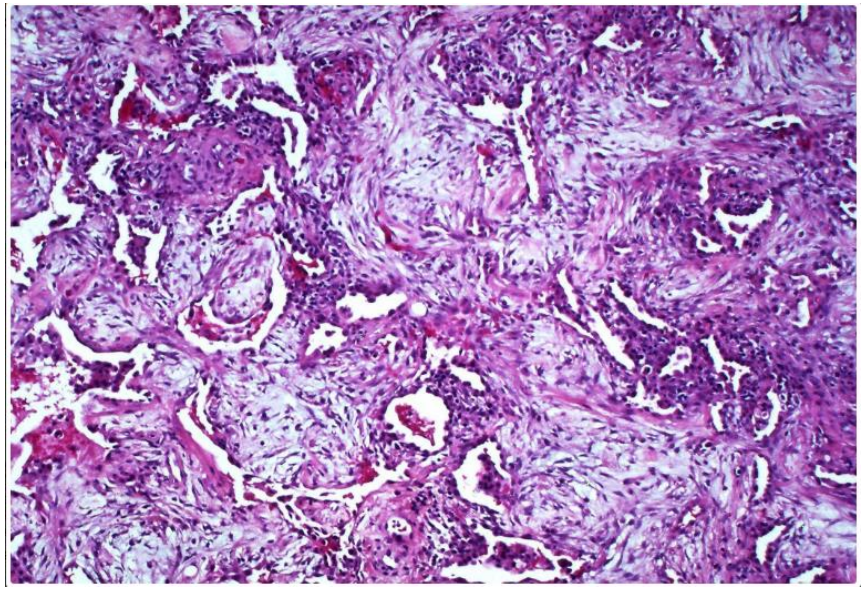
2. organizing stage:

type II pneumocytes proliferate.

fibrin-rich exudates organize into intraalveolar fibrosis.

Marked thickening of the alveolar septa

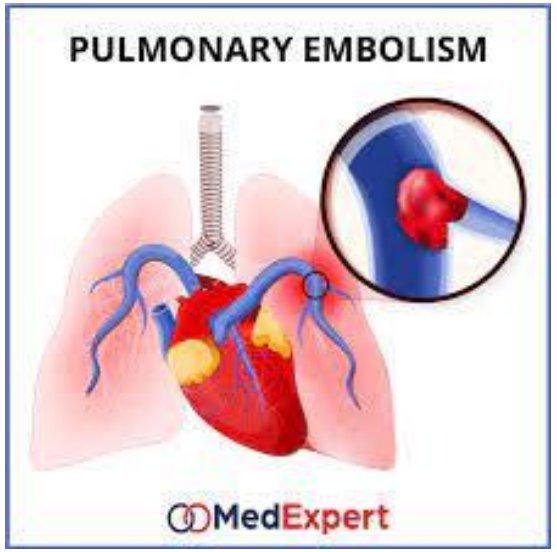
due to proliferation of interstitial cells and deposition of collagen.



PULMONARY DISEASES OF VASCULAR ORIGIN

1. PULMONARY EMBOLISM, HEMORRHAGE, AND INFARCTION:

More than 95% of all pulmonary emboli arise from thrombi within the large deep veins of the legs.



The risk factors are :

(1) prolonged bed rest .

(2) surgery, especially orthopedic surgery on the knee or hip.

(3) severe trauma (including burns or multiple fractures).

(4) congestive heart failure.

(5) in women, the period around parturition or the use of oral contraception pills with high estrogen content

THERE ARE TWO IMPORTANT CONSEQUENCES OF PULMONARY ARTERIAL OCCLUSION:

(1) an increase in pulmonary artery pressure from blockage of flow and vasospasm caused by neurogenic mechanisms and/or release of mediators (e.g., thromboxane A2, serotonin).

(2) ischemia of the downstream pulmonary parenchyma.

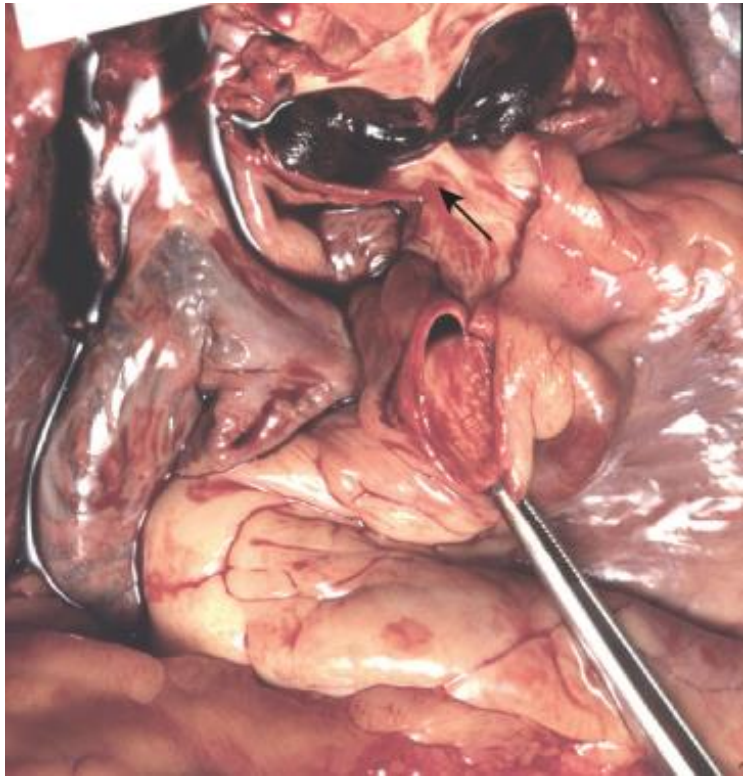
Thus, occlusion of:

- major vessel results increase in pulmonary artery pressure, diminished cardiac output, right sided heart failure (acute cor pulmonale).
- smaller vessels are occluded, the result is less catastrophic and may even be clinically silent

MORPHOLOGY :

The consequences of pulmonary embolism, as noted, depend on the size of the embolic mass and the general state of the circulation:

A large embolus may embed in the main pulmonary artery or at the bifurcation as a saddle embolus.



no time for morphologic alterations in the lung

patient die

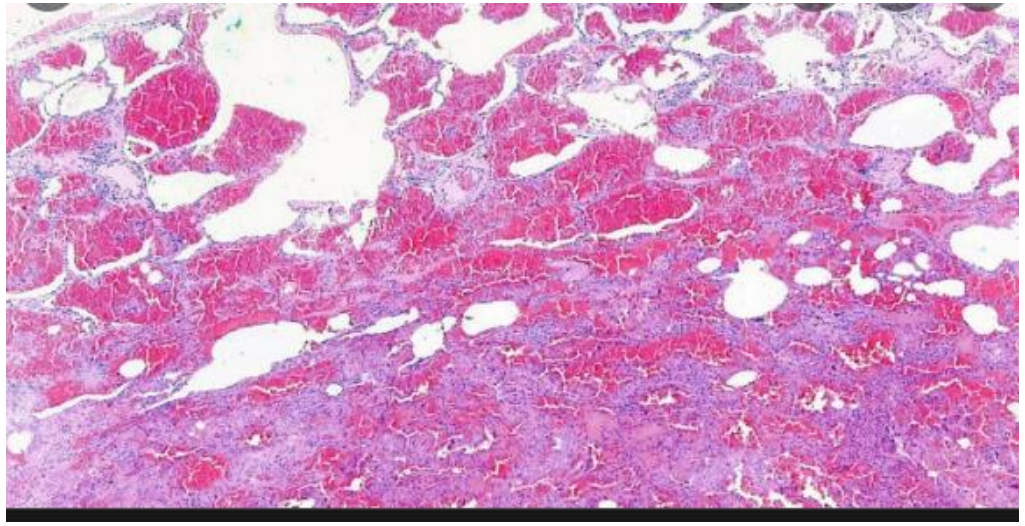
Smaller emboli become impacted in medium-sized and small-sized pulmonary arteries.

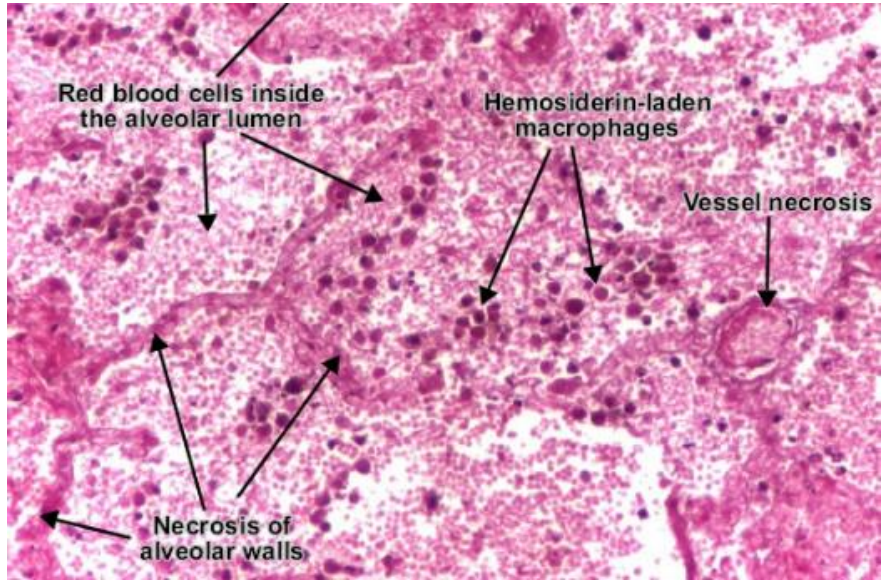
With adequate circulation and bronchial arterial flow:

alveolar hemorrhage may occur as a result of ischemic damage to the endothelial cells.

With compromised cardiovascular status:

as may occur with congestive heart failure, infarction results.





CLINICAL FEATURES

1. Most (60% to 80%) are clinically silent because they are small; the bronchial circulation sustains the viability of the affected lung parenchyma, and the embolic mass is rapidly removed by fibrinolytic activity.

2. In 5% of cases, death, acute right-sided heart failure, or cardiovascular collapse.

3. Obstruction of small to medium pulmonary branches (10% to 15% of cases) causes pulmonary infarction.

4. less than 3% of cases, recurrent "showers" of emboli lead to pulmonary hypertension, chronic right-sided heart failure.

HOW I CAN MODIFY THE RISK FACTORS???

patients who have experienced one pulmonary embolism have a 30% chance of developing a second.

Prophylactic therapy may include:

anticoagulation.

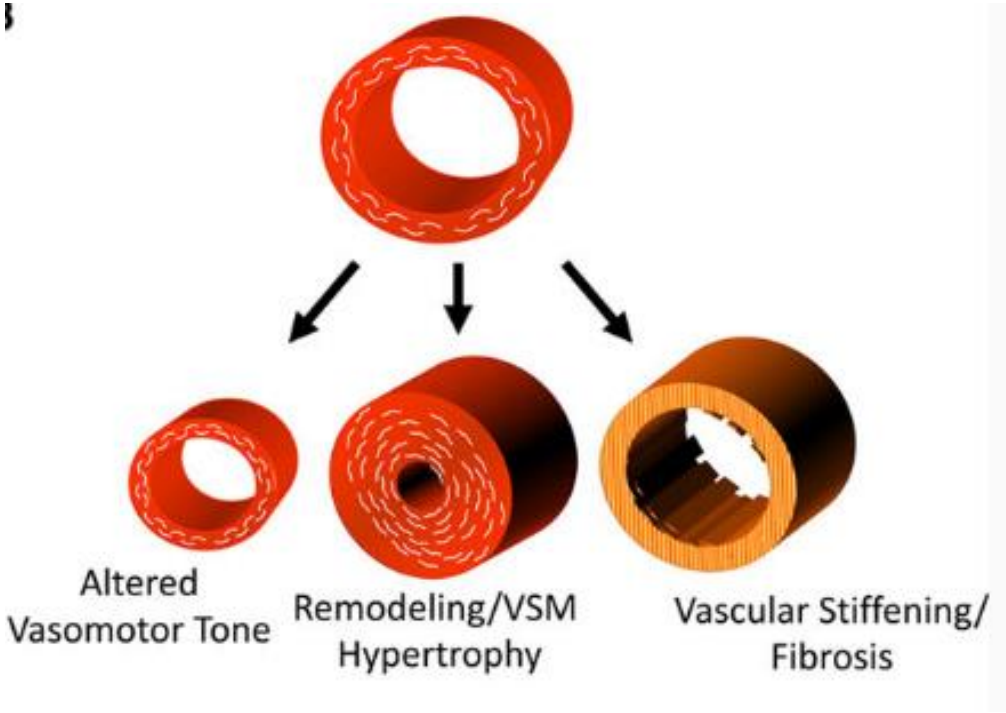
early ambulation for postoperative patients.

application of elastic stockings.

leg exercises for bedridden patients.

PULMONARY HYPERTENSION

Defined as pressures of 25 mm Hg or more at rest, may be caused by a decrease in the cross-sectional area of the pulmonary vascular bed or, less commonly, by increased pulmonary vascular blood flow.



WHO has classified pulmonary hypertension into the following five groups:

*systemic sclerosis

*Obstructive sleep apnea

*idiopathic pulmonary arterial hypertension

Mitral stenosis

reducing the functional cross-sectional area

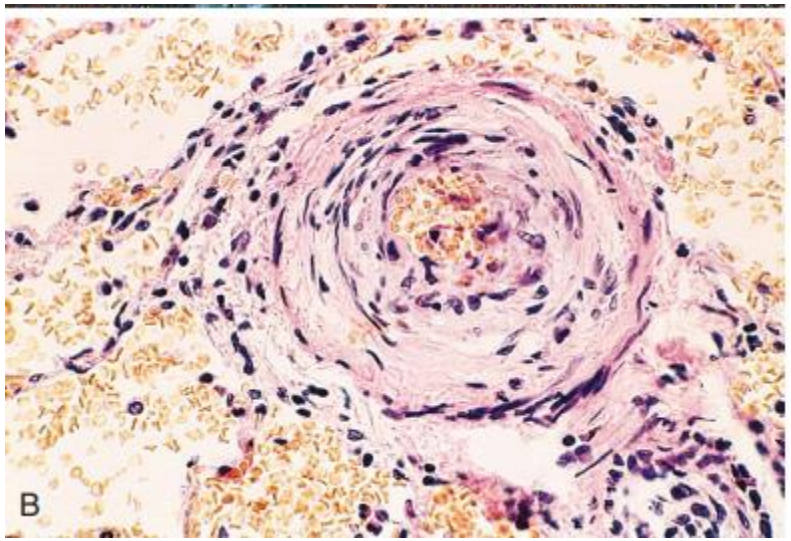
obliterate alveolar capillaries, increasing pulmonary resistance to blood flow

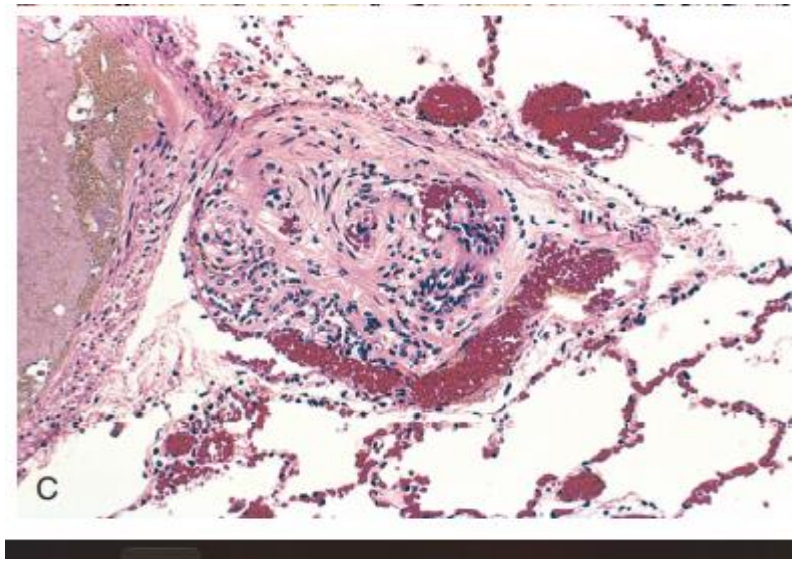
MORPHOLOGY

- All forms of pulmonary hypertension are associated with:
  - ✓ medial hypertrophy of the pulmonary muscular and elastic arteries.
  - ✓ pulmonary arterial atherosclerosis.

right ventricular hypertrophy.

plexiform lesion: tuft of capillary formations is present, producing a network, or web, that dilated thin-walled, small arteries





CLINICAL FEATURES

The presenting features are usually:

dyspnea and fatigue.

Anginal chest pain.

Over time:

respiratory distress and cyanosis.

right ventricular hypertrophy.

Death from decompensated cor pulmonale, often with superimposed thromboembolism and pneumonia.