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Doctor 2020 - wateen - medicine - MU

pathology sheet

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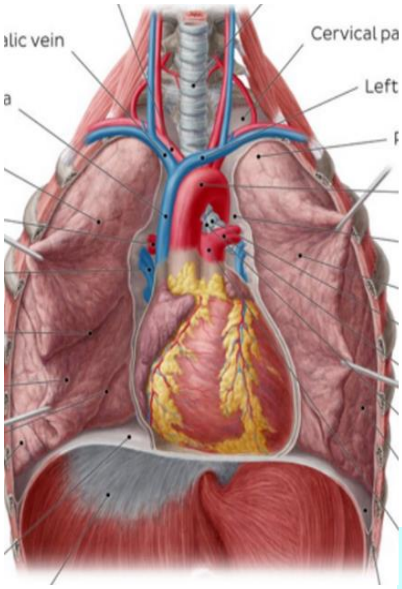
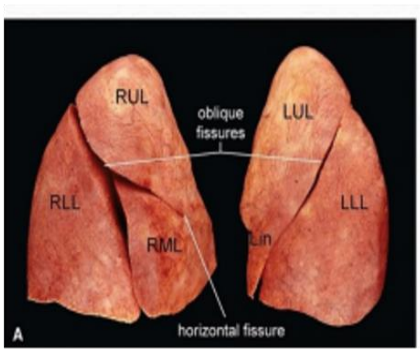
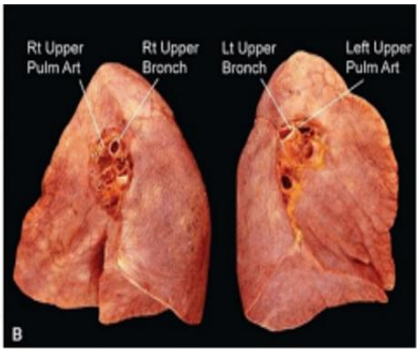
Atelectasis and Pulmonary vascular Diseases

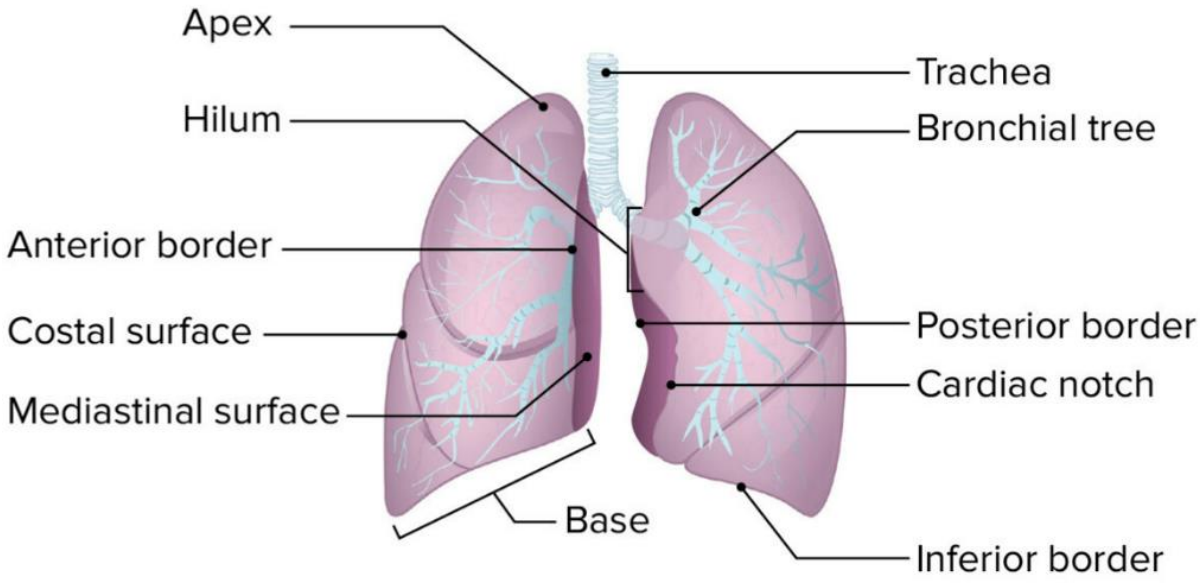
Anatomy of the lung:



Anterior view **posterior view**



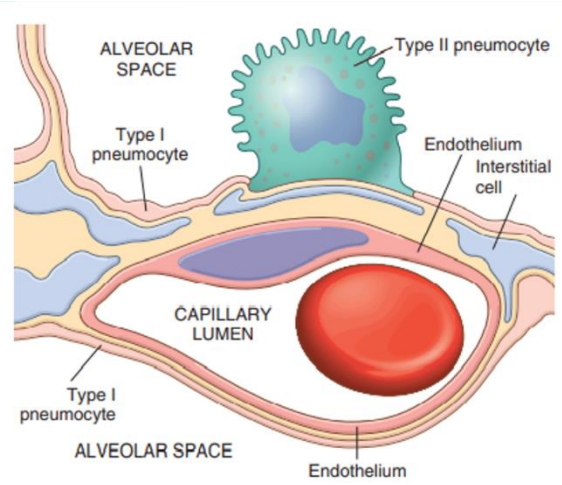


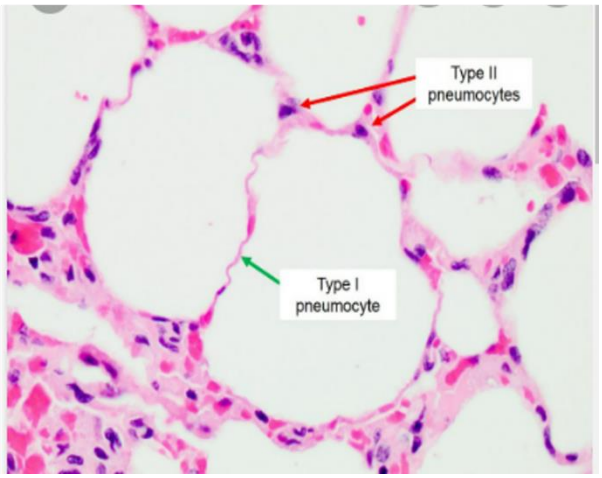


Alveoli → spaces have air

- **Contents of septa :**
  - **Lining → type I pneumocyte**
  - **Associated cells → type II pneumocyte**

Histology of the lung:





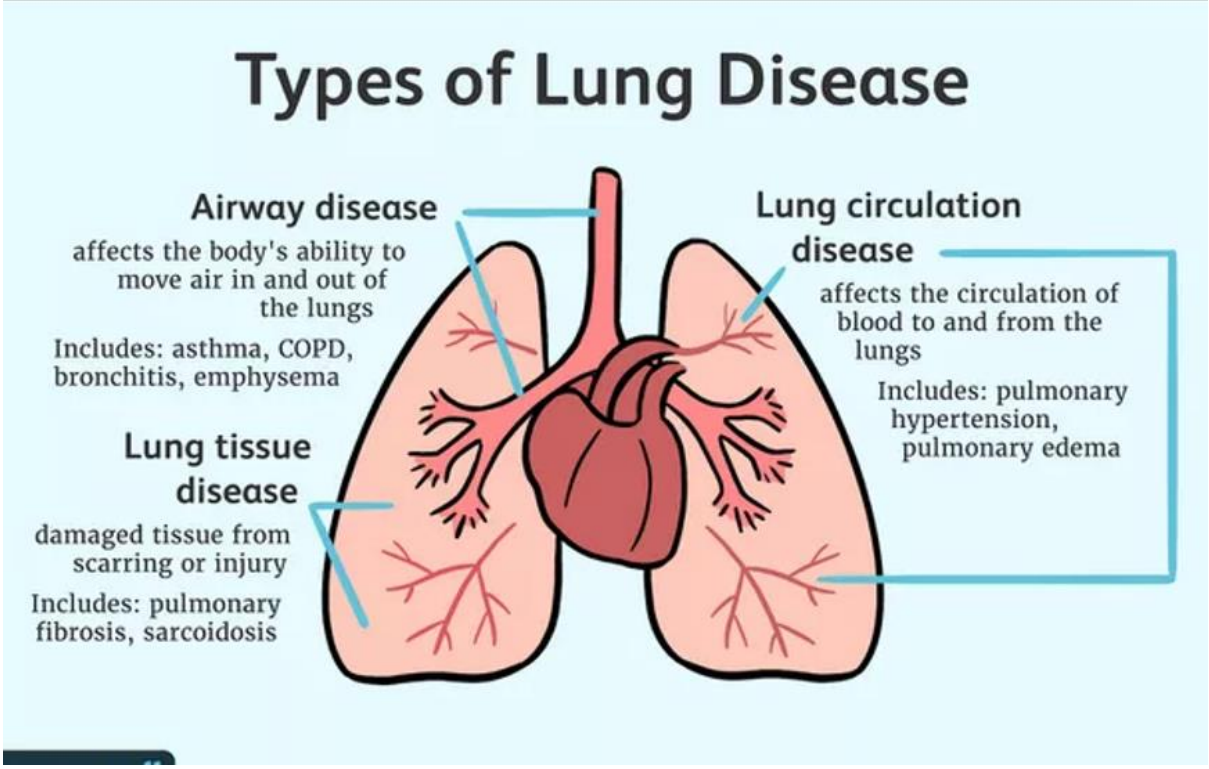
In the barrier, the surfactant

prevents collapse,

maintains surface tension

Thin septa

All the respiratory passages from the trachea to the respiratory bronchioles are called the tracheobronchial tree (functional unit of lung)



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.

- vein (oxygenated)

- artery (deoxygenated)

- When gas exchange between the capillary and the alveoli .. But the artery is not loaded in O_2, the area where there is obstruction (there is no air) so gas exchange does not occur.

- the lung (shrink + collapse) so its size decreases.

ATELECTASIS IS CLASSIFIED INTO THREE FORMS:

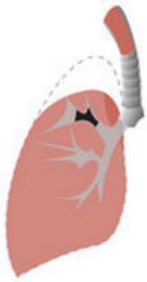
1. Resorption atelectasis: (at level of bronchi)

✓occurs when an obstruction prevents air from reaching distal airways.

(and the rest air → will be resorbed)

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

✓The most common causes are:



Resorption atelectasis

❖intrabronchial mucous or mucopurulent plugs.

❖foreign body aspiration.

❖bronchial asthma and bronchiectasis.

❖chronic bronchitis and intrabronchial tumor

- Due to an obstruction, the air (in the blockage area) will be exchanged without air, so that is a collapse.

- If an elderly patient has a drunkenness, most likely be tumor

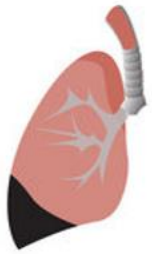
- If a small patient is a foreign body aspiration

2. Compression atelectasis:

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

-Usually associated with congestive heart failure.

- The normal in the pleura region without fluid, but in this case, it is in fluid, like blood or air..., so the lung does not expand due to the presence of fluid

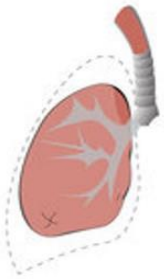


Compression atelectasis

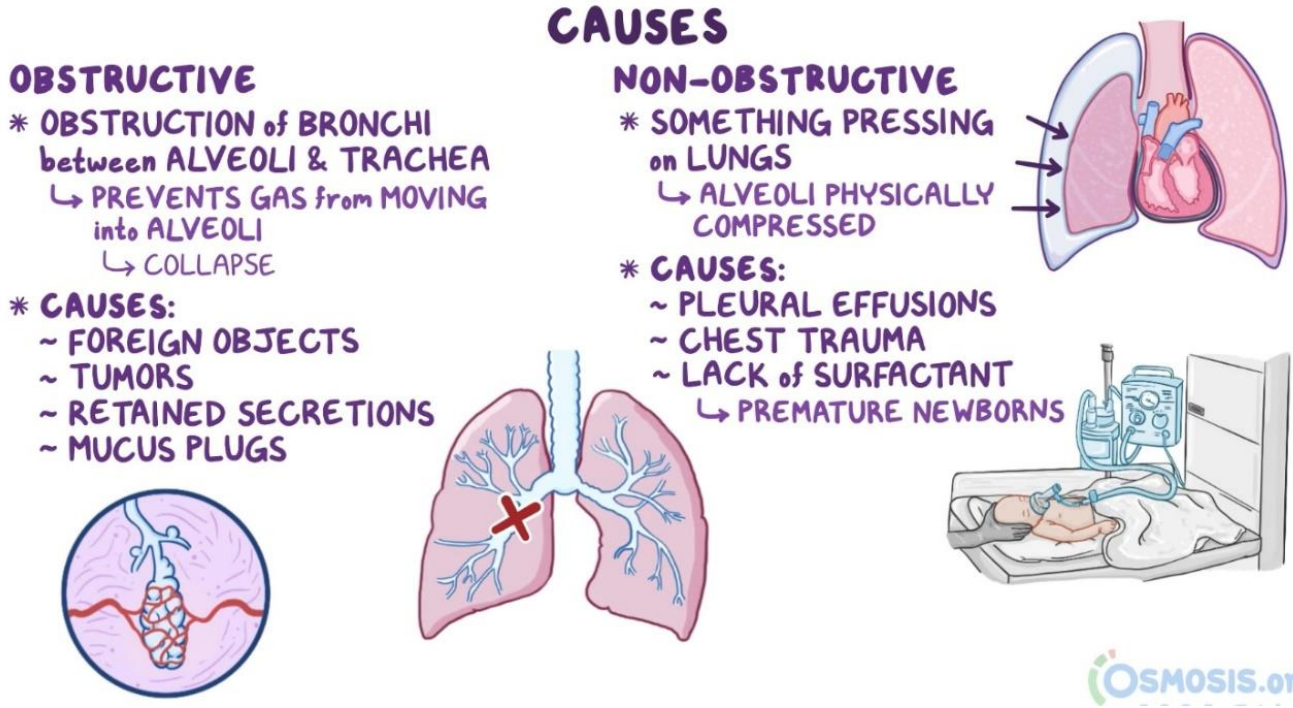
3. Contraction atelectasis: (at the level of the lung)

-occurs when local or diffuse fibrosis affecting the lung

(normal alveoli & bronchi , but disorder in lung → don't expand)



Contraction atelectasis

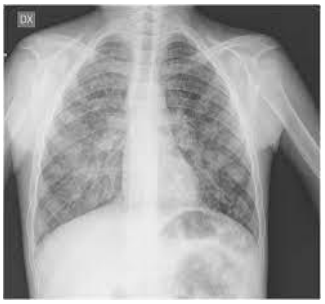


Acute respiratory distress syndrome (ARDS)

-Respiratory failure occurring within 1 week of a known clinical insult with <u>bilateral opacities</u> on chest imaging (due to decrease in the ratio of gas) , not fully explained by effusions, atelectasis, cardiac failure, or fluid overload.

- Inside the alveoli, instead of being in the air, it becomes an exudate. When the air enters, it cannot find a place in the alveoli

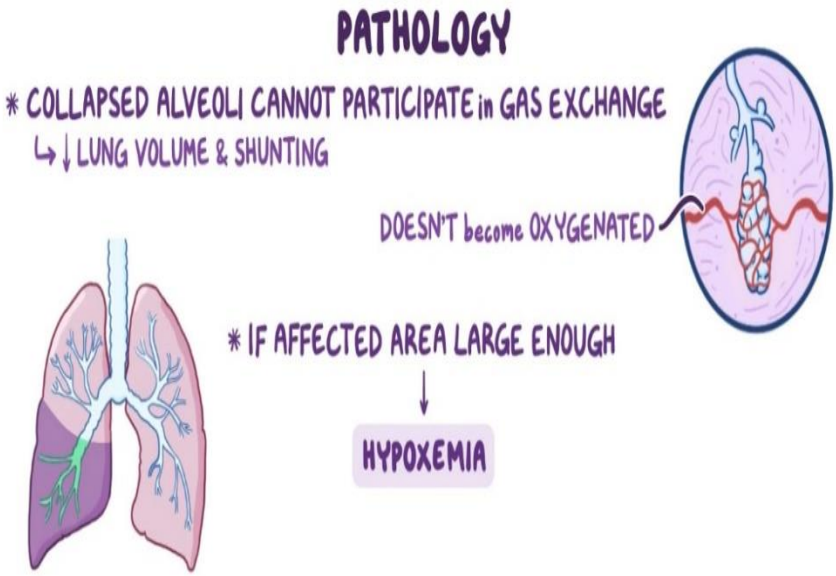
→ hypoxia occurs.



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

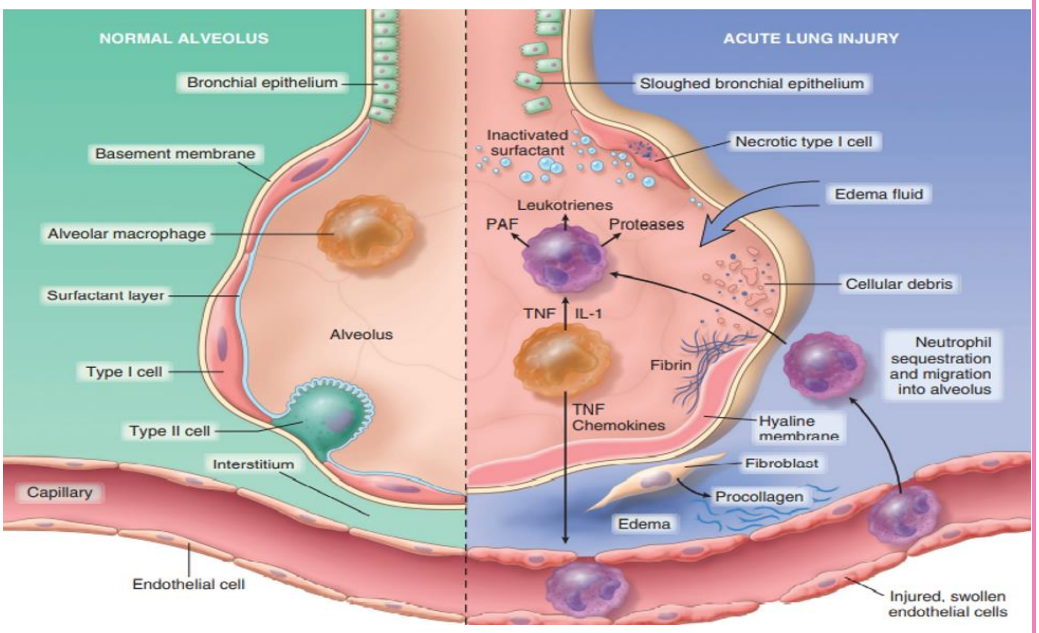
-Severe ARDS is characterized by rapid onset of life-threatening respiratory insufficiency, cyanosis, and severe arterial hypoxemia that is refractory to oxygen therapy.

- If we give the patient oxygen therapy, he does not benefit and the same problem remains



- DAD → Occurs due to the damage caused by neutrophils

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.

- In the capillary _ alveoli surface

It releases IL-8 that attracts the first immune cell (neutrophils)

that produce (proteases and reactive oxygen species)

-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.

In area will happen:

1- swelling

2- expand space between endothelium of blood vessels and destroy the alveolar epithelium

MORPHOLOGY

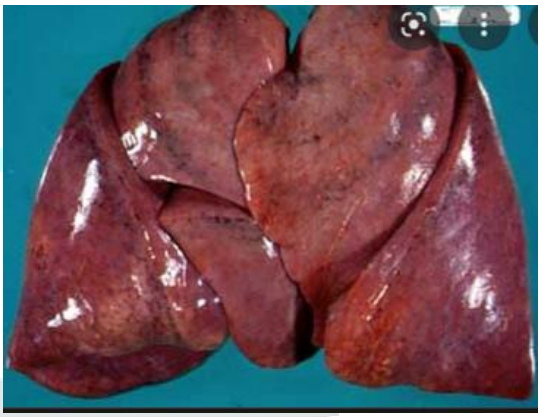
1- acute phase:

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

- Why is the lung dark red, with the destruction done to the endothelium of blood vessels, all fluids will leak from the vessels into the lungs

-Why is the lung heavy due to Exudate

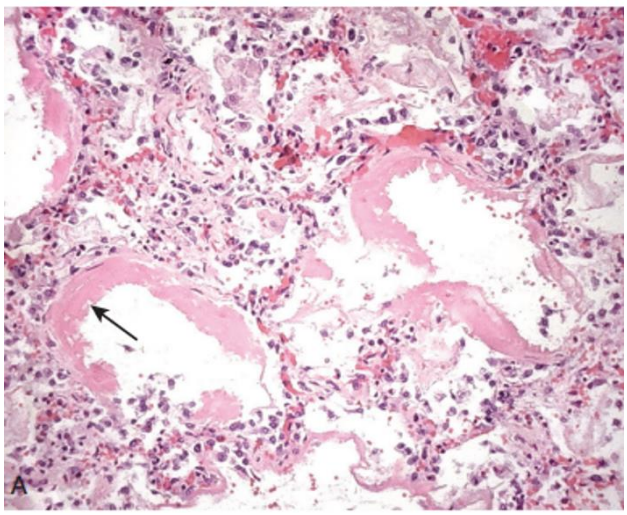
-Microscopic examination:



- **capillary congestion.**
- **necrosis of alveolar epithelial cells.**
- **interstitial and interaalveolar edema and hemorrhage.**

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

- Hyaline cartilage and congestion and thick wall



2. organizing stage:

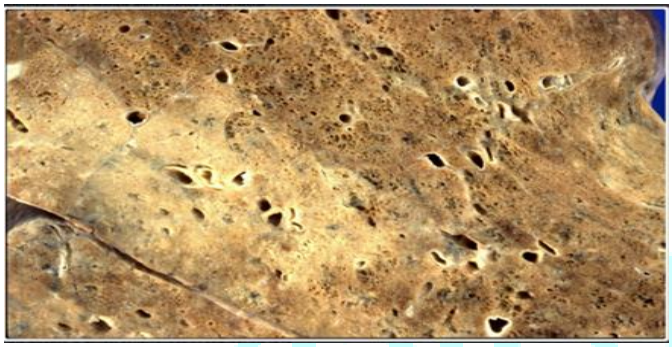
-type II pneumocytes proliferate.

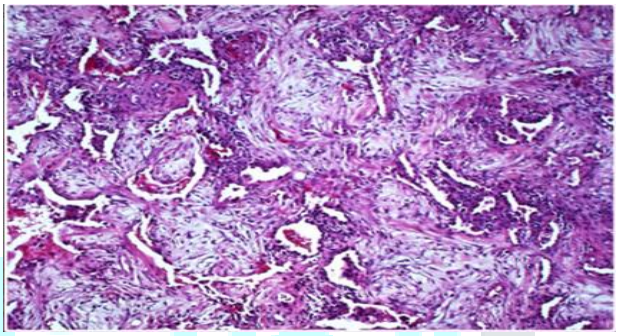
-fibrin-rich exudates organize into intraalveolar fibrosis.

-Marked thickening of the alveolar septa due to proliferation of interstitial cells and deposition of collagen.

- The normal is in a small opening + thin wall

- But with the presence of fibers, alveoli will get sugary and collapse as a result of collagen deposition





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.

- Most cases of pulmonary embolism (PE) are caused by deep vein thrombosis (DVT).

-<u>The risk factors are:</u>

(1) prolonged bed rest.

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

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

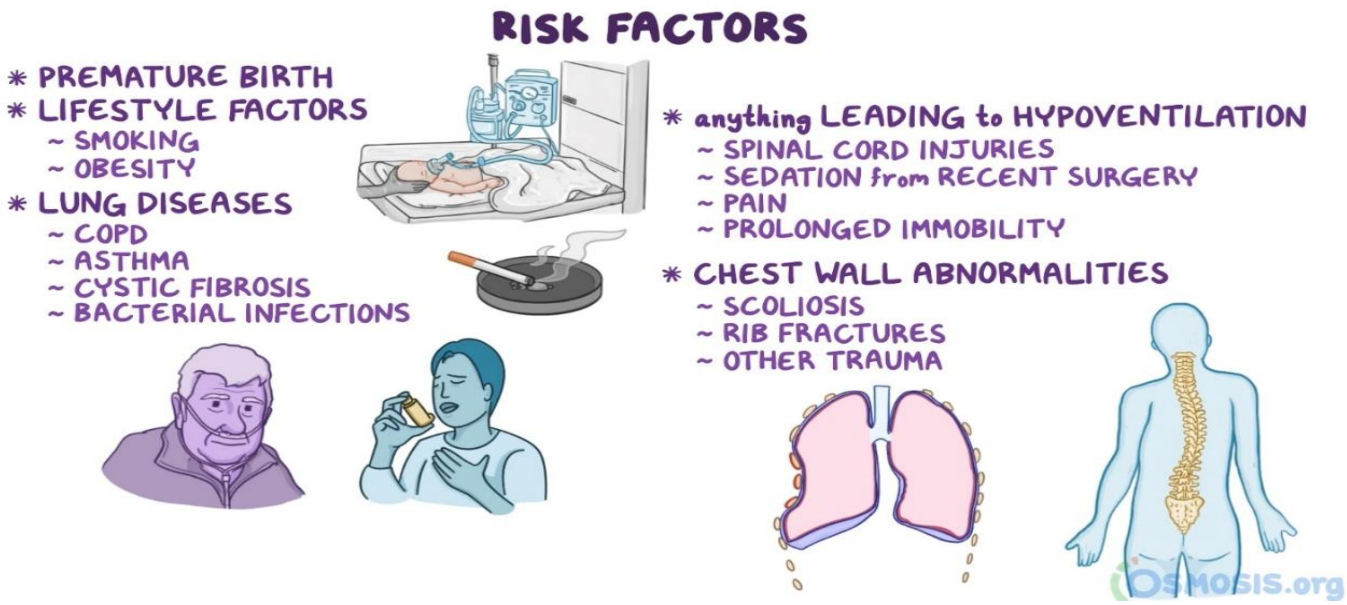
(4) congestive heart failure.

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

- Estrogen increases the coagulant factor so clot occurs.

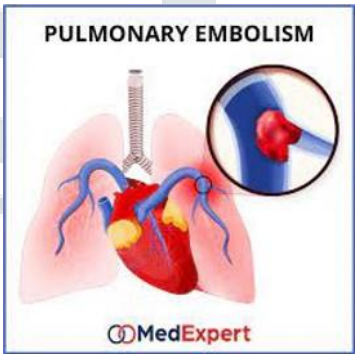
* embolus → systemic

* thrombus → locally



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).

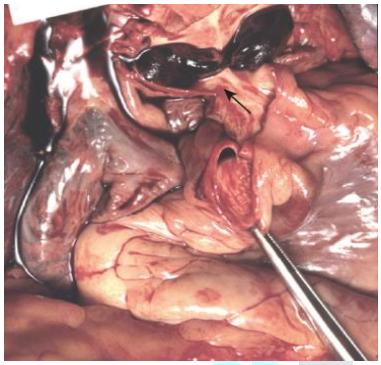
-smaller vessels are occluded, the result is less catastrophic and may even be clinically silent

MORPHOLOGY :

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

-The danger in embolism depends on its size

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



- If it is as big as saddle embolism, the patient dies immediately because it obstruction the pulmonary artery

NO TIME for morphologic alteration in lung. Patient die

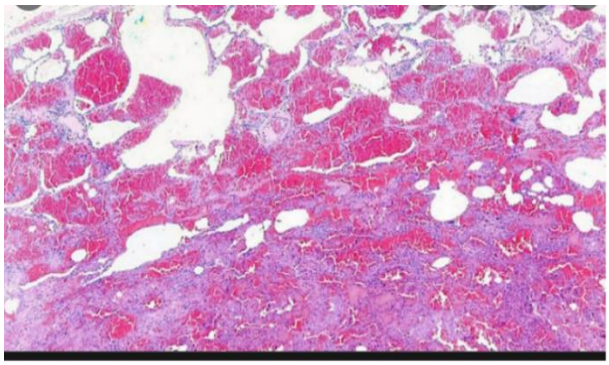
-Smaller emboli become impacted in medium-sized and small-sized pulmonary arteries.

-With adequate circulation and bronchial arterial flow:

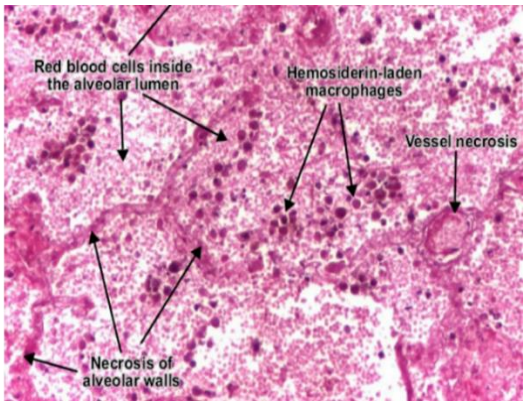
alveolar hemorrhage may occur as a result of ischemic damage to the endothelial cells

-With compromised cardiovascular status:

as may occur with congestive heart failure, infarction results.



Alveolar hemorrhage → Smaller embolus with good circulation



Infarction → smaller embolus with compromised Cardiovascular status

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.

- 30% of those who have PE are at risk of returning due to DVT

-Prophylactic therapy may include:

✓ anticoagulation.

✓early ambulation for postoperative patients.

- The patient must move after the operation until he becomes stasis

✓ application of elastic stockings.

- The blood in the vessels (in the foot) is moving against gravity, so the upward pumping is weak, the elastic stockings help the blood to go up

✓ leg exercises for bedridden patients.

- If a patient does not move on the bed, we move him or turn him

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.

-When pressure increases on blood vessels, walls become hypertrophy and becomes thick due to increased pressure

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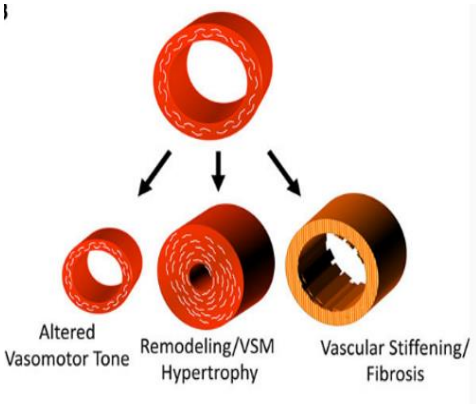

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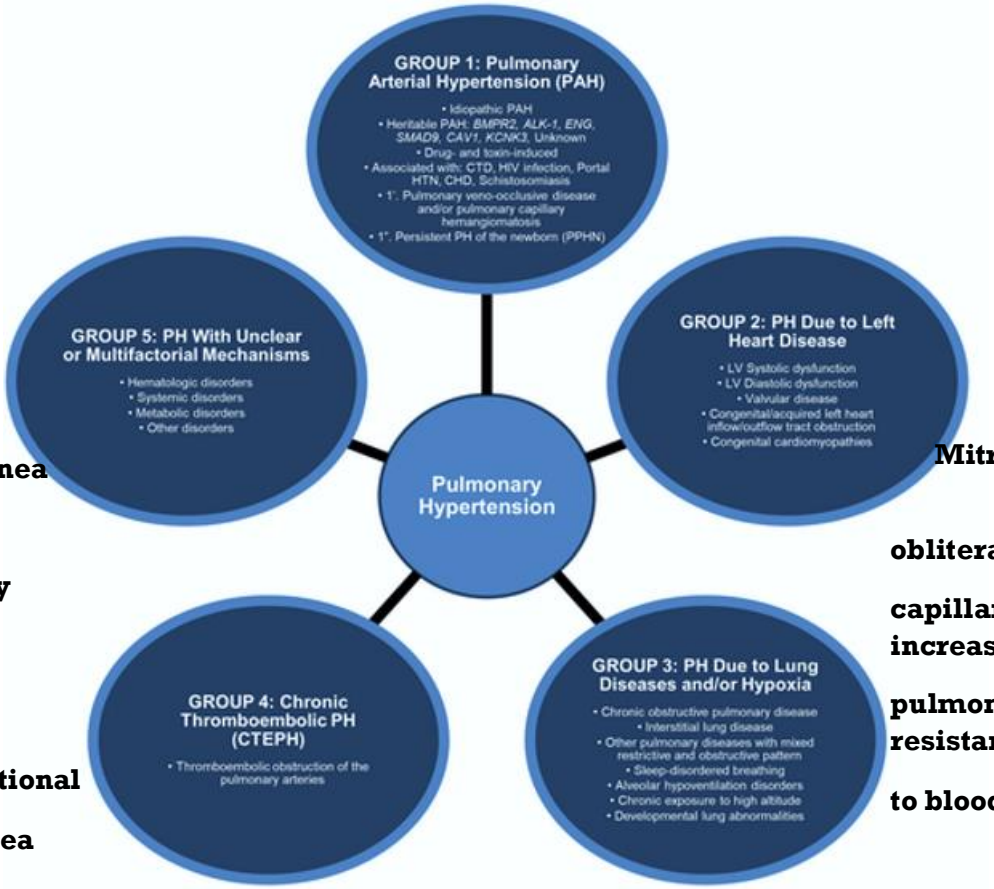


***systemic sclerosis**

***Obstructive sleep apnea**

***idiopathic pulmonary arterial hypertension**

reducing the functional cross-sectional area

Mitral stenosis

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.**

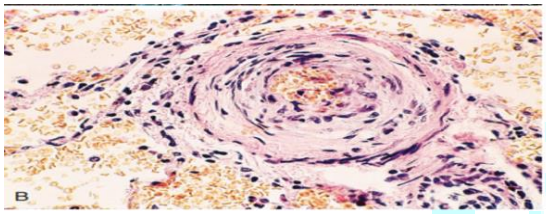
- It becomes hypertrophy although it is originally thin

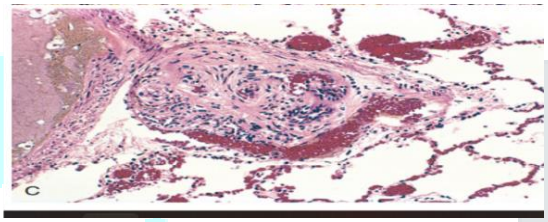
✓**pulmonary arterial atherosclerosis.**

✓**right ventricular hypertrophy.**

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

- Group of capillaries intertwined with each other





CLINICAL FEATURES

-The presenting features are usually:

- nonspecific symptoms

✓**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.**