

**Lecture one**  
**lung pathology**  
**4<sup>th</sup> year MBBS**

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# Expectation at end of lecture

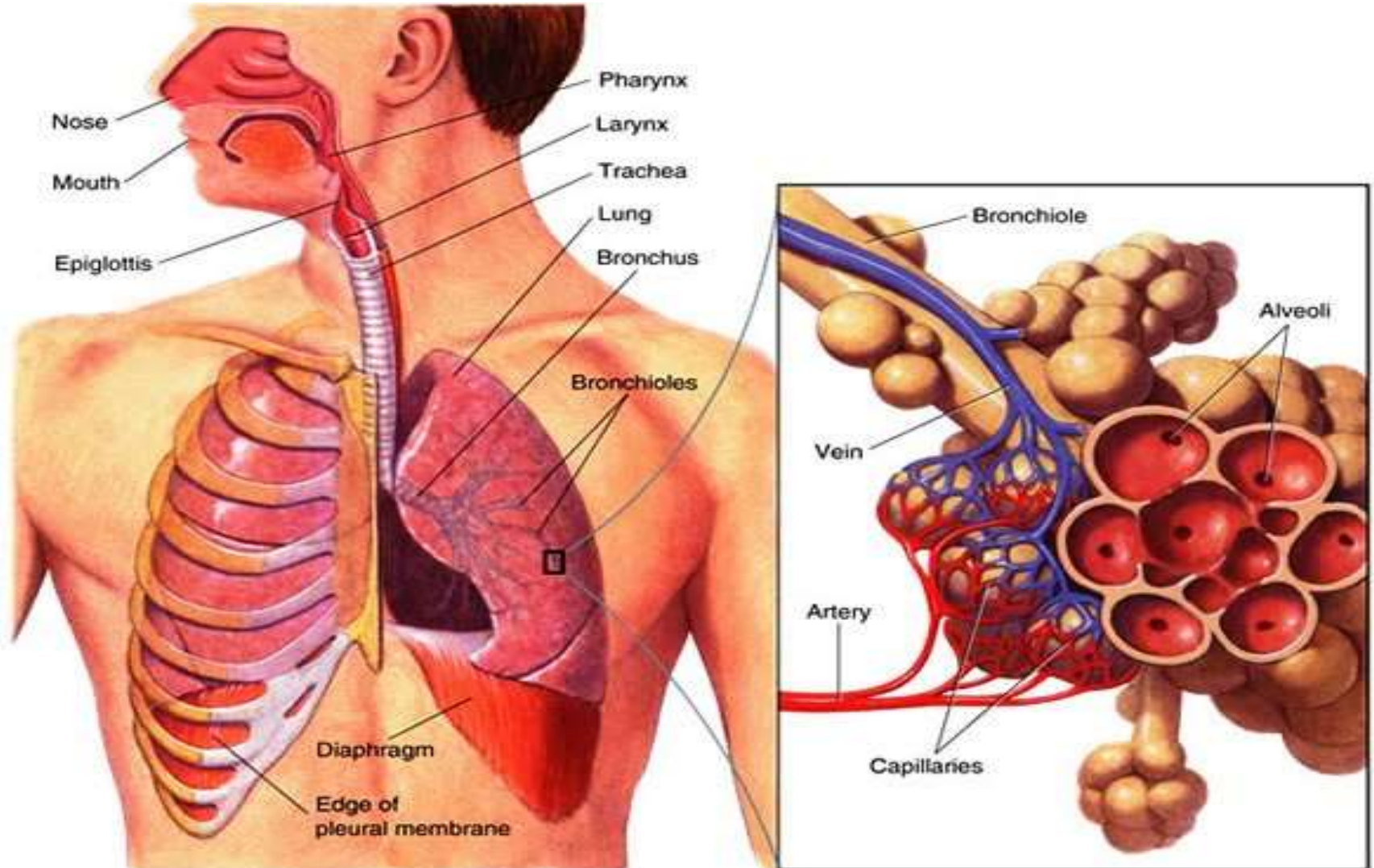
- Brief review of anatomy and physiology related to lungs
- Atelectasis
- Pulmonary edema
  - Cardiogenic pulmonary edema
  - Non cardiogenic pulmonary edema
- Important questions

- **REVIEW OF ANATOMY AND  
PHYSIOLOGY RELATED TO  
LUNGS**

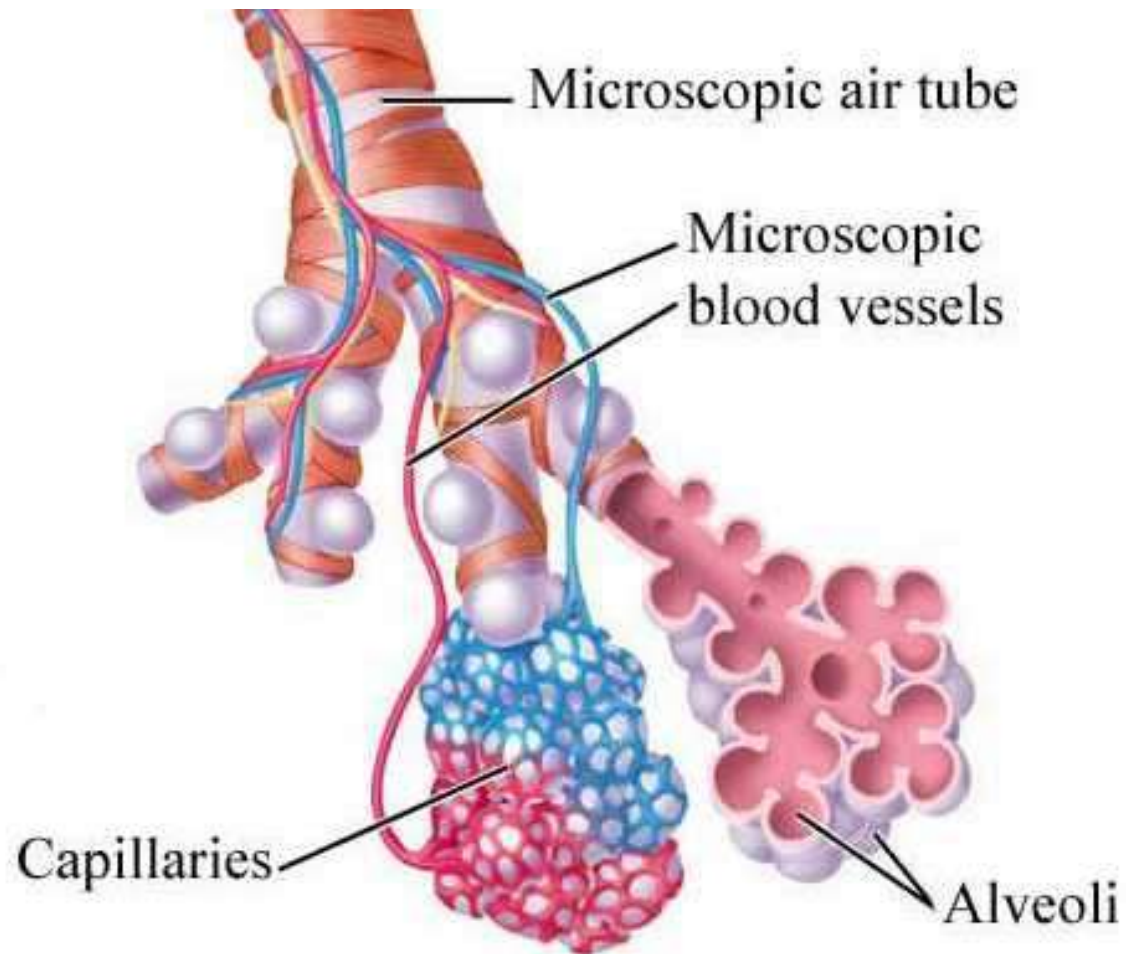
# Lungs

- The major function of the lung is to excrete carbon dioxide from blood and replenish oxygen.
  - Trachea
  - Bronchi & Bronchiole
  - Acinus
  - Alveoli

# Respiratory system review of anatomy

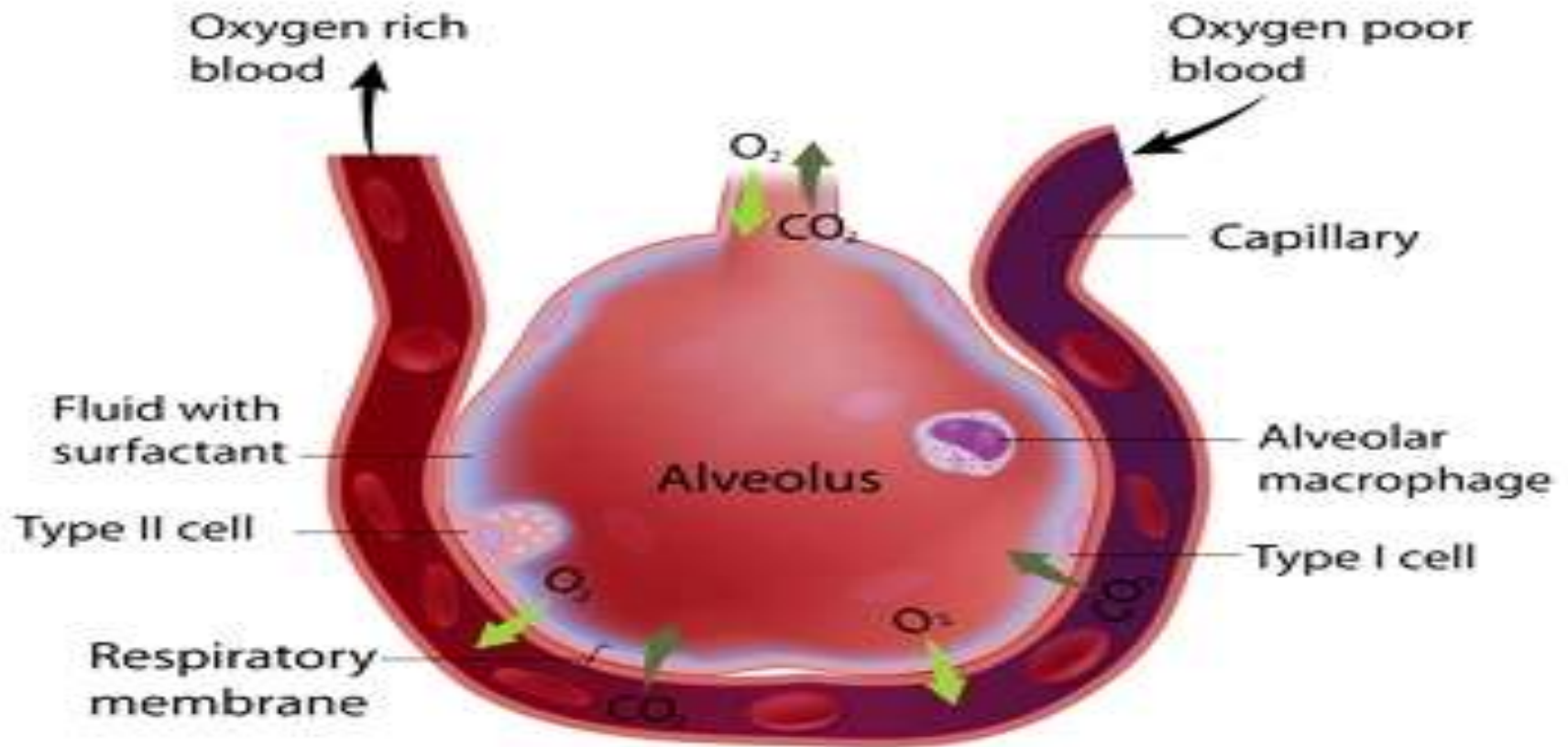


# Alveoli and capillaries junction



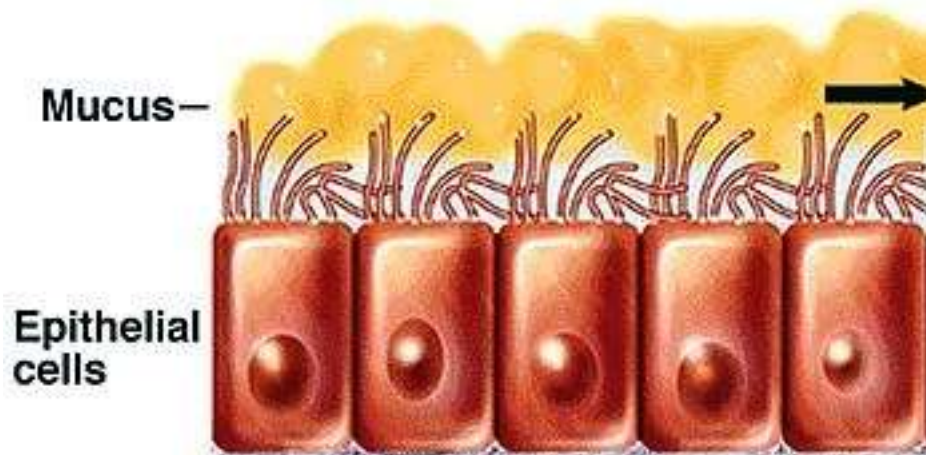
# Alveoli and capillaries junction with surfactants

## Structure of an Alveolus



# Alveoli and capillaries junction

- Muco ciliary apparatus
  - Cilia beat in rythmic fashion from alveoli to proximal trachea
  - Remove mucous and debris containing bacteria
  - Mucous is then swallowed or coughed out of body



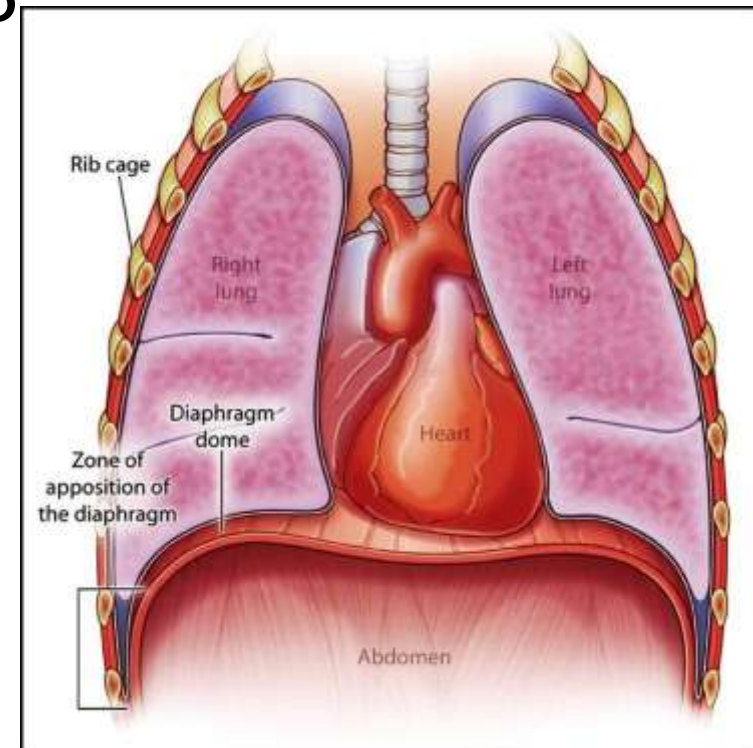


# Bronchial muscles

- Smooth muscle contract in response to emotion and parasympathetic activity  
....bronchoconstriction
- Smooth muscles relax in response to sympathetic activity

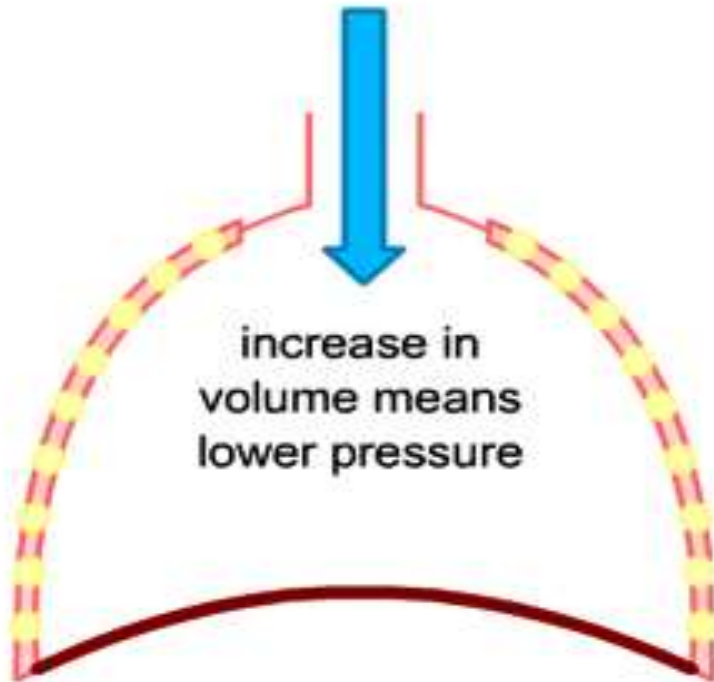
# DIAPHRAGM

- Muscular layer separating lung from abdomen
- Major Muscle of inspiration
- Nerve supplies C3,C4 and C 5



# DIAPHRAGM

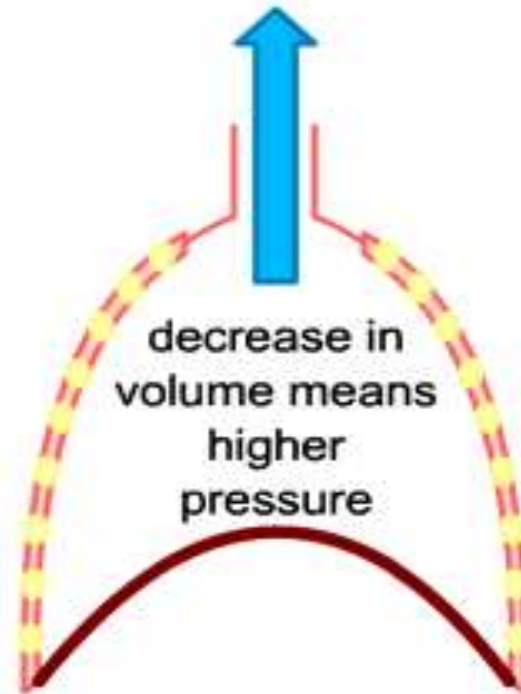
breathing in



increase in  
volume means  
lower pressure

ribs move up and out  
diaphragm flattens  
volume of chest increases

breathing out



decrease in  
volume means  
higher  
pressure

ribs fall  
diaphragm moves up  
volume of chest decreases

# Common developmental defects in lung

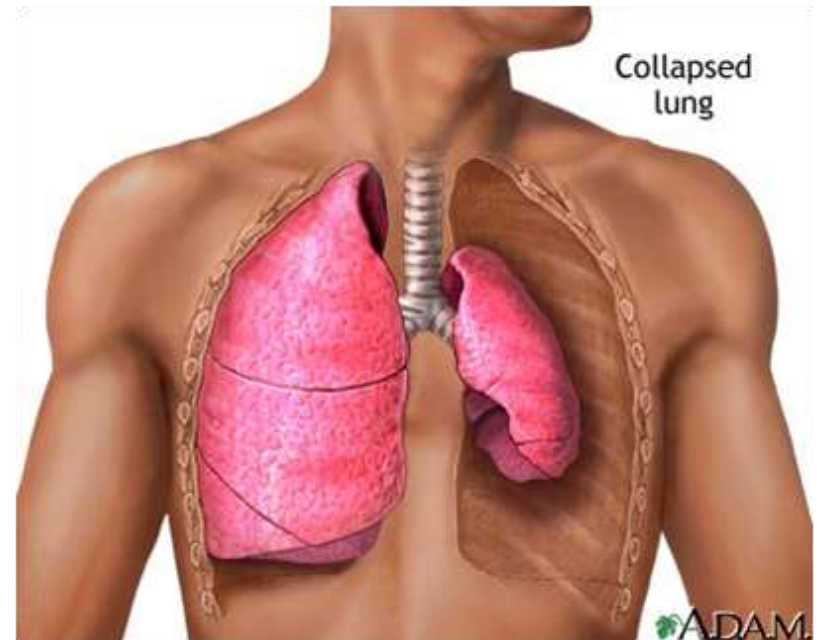
- Agenesis or hypoplasia of both lungs, one lung, or single lobes
  - Tracheal and bronchial anomalies (atresia, stenosis, tracheoesophageal fistula)
  - Vascular anomalies
  - Congenital lobar overinflation (emphysema)
  - Foregut cysts
  - Congenital pulmonary airway malformation

- Now pathology starts

- **ATELECTASIS**

# ATELECTASIS

- an area of collapsed or nonexpanded lung.
- predispose for infection due to decreased mucociliary clearance.



# ATELECTASIS

## 1. Obstruction/resorption atelectasis

- collapse of lung due to resorption of air distal to an obstruction;
- aspiration of a foreign body, chronic obstructive pulmonary disease (COPD), and postoperative atelectasis.

# Atelectasis

## 2. **Compression atelectasis**

- atelectasis due to fluid, air, blood, or tumor in the pleural space.

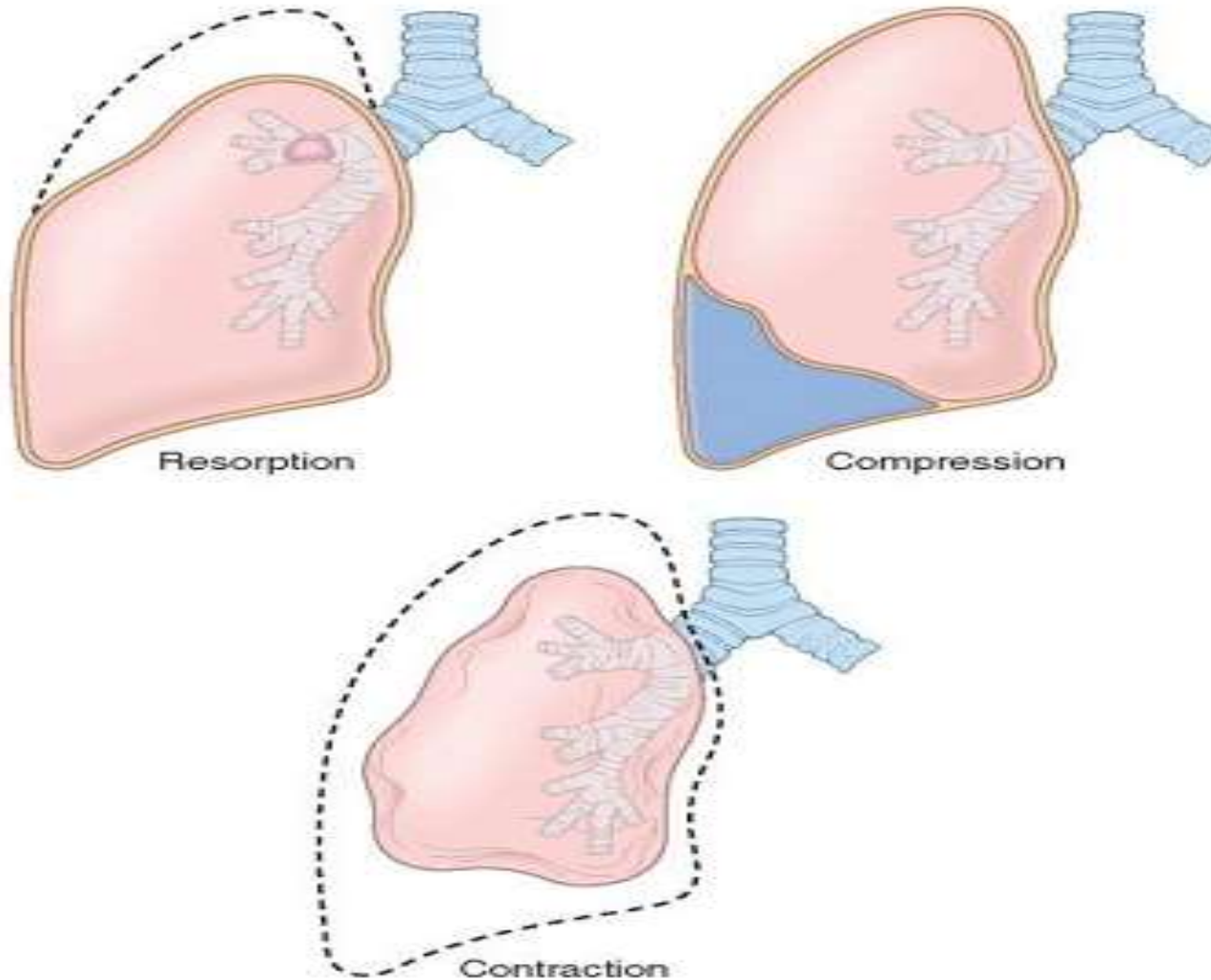
## 3. **Contraction (scar) atelectasis**

- is due to fibrosis and scarring of the lung.

## 4. **Patchy atelectasis** is due to a lack of surfactant, as occurs in hyaline membrane disease of newborn or acute (adult) respiratory distress syndrome (ARDS).



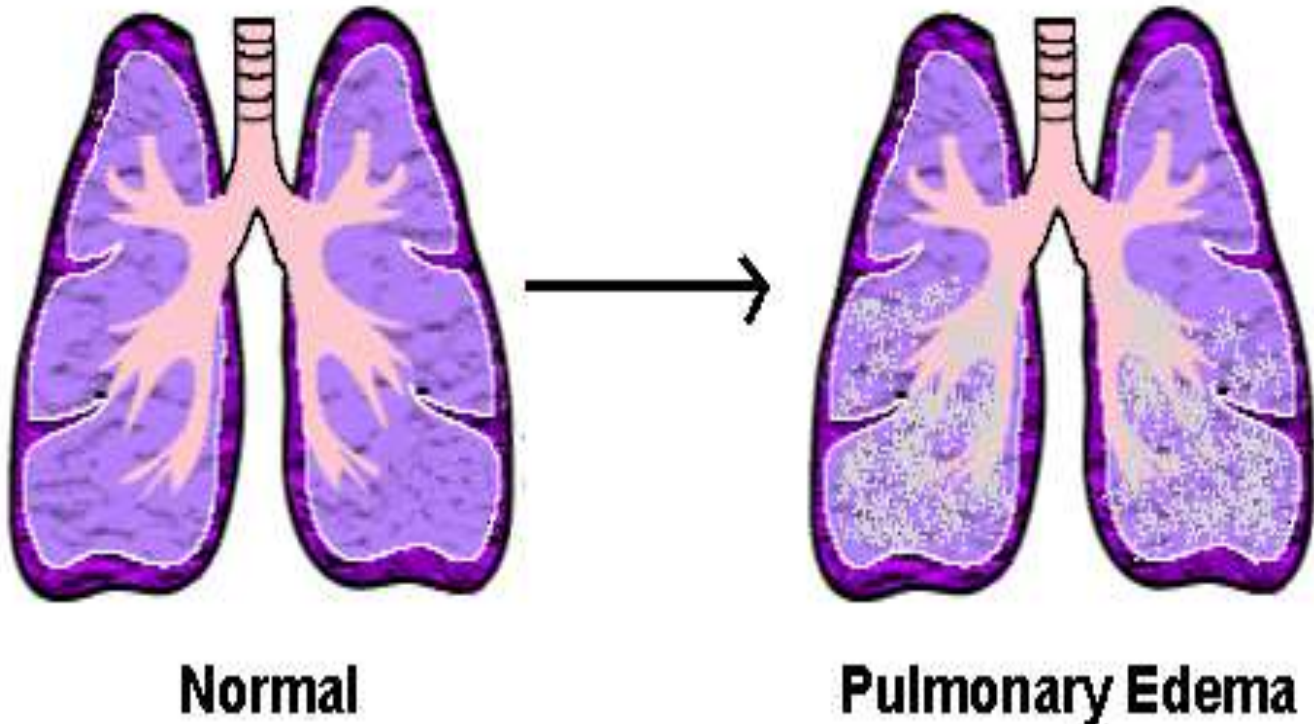
# Atelectasis



- Pulmonary edema

# Pulmonary edema

- It is collection of fluid in lung tissue.



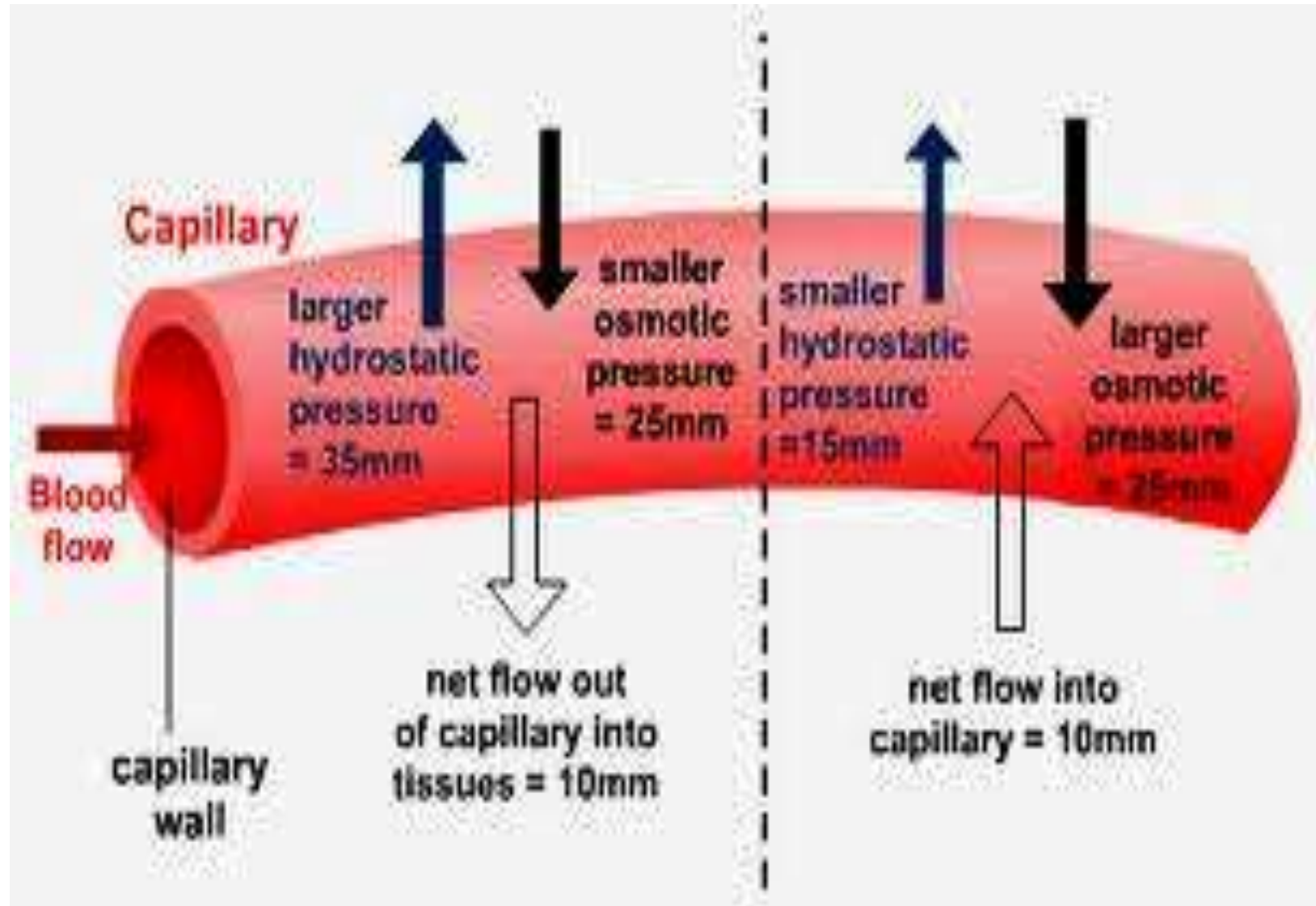
# Pulmonary edema

- It is collection of fluid in lung tissue.

## ❖ Causes

- ❖ cardiogenic pulmonary edema
- ❖ Non cardiogenic pulmonary edema

# Causes OF pulmonary edema



# Pulmonary Edema types

## cardiogenic

- **Increased hydrostatic pressure**
  - **RAISED PAP AND LVEDP > 18**
  - (increased pulmonary venous pressure)
- Left-sided heart failure (common)
- Volume overload
- Pulmonary vein obstruction

# Pulmonary edema types

- **EDEMA DUE TO MICROVASCULAR INJURY (ALVEOLAR INJURY)**
- **Infections:** pneumonia, septicemia Inhaled gases: oxygen, smoke
- **Liquid aspiration:** gastric contents, near-drowning
- **Drugs and chemicals:** chemotherapeutic agents (bleomycin), other medications (amphotericin B), heroin, kerosene, paraquat Shock, trauma  
Radiation Transfusion related

# **Pulmonary edema types undetermined origin**

- **EDEMA OF UNDETERMINED ORIGIN**
- High altitude
- Neurogenic (central nervous system trauma)

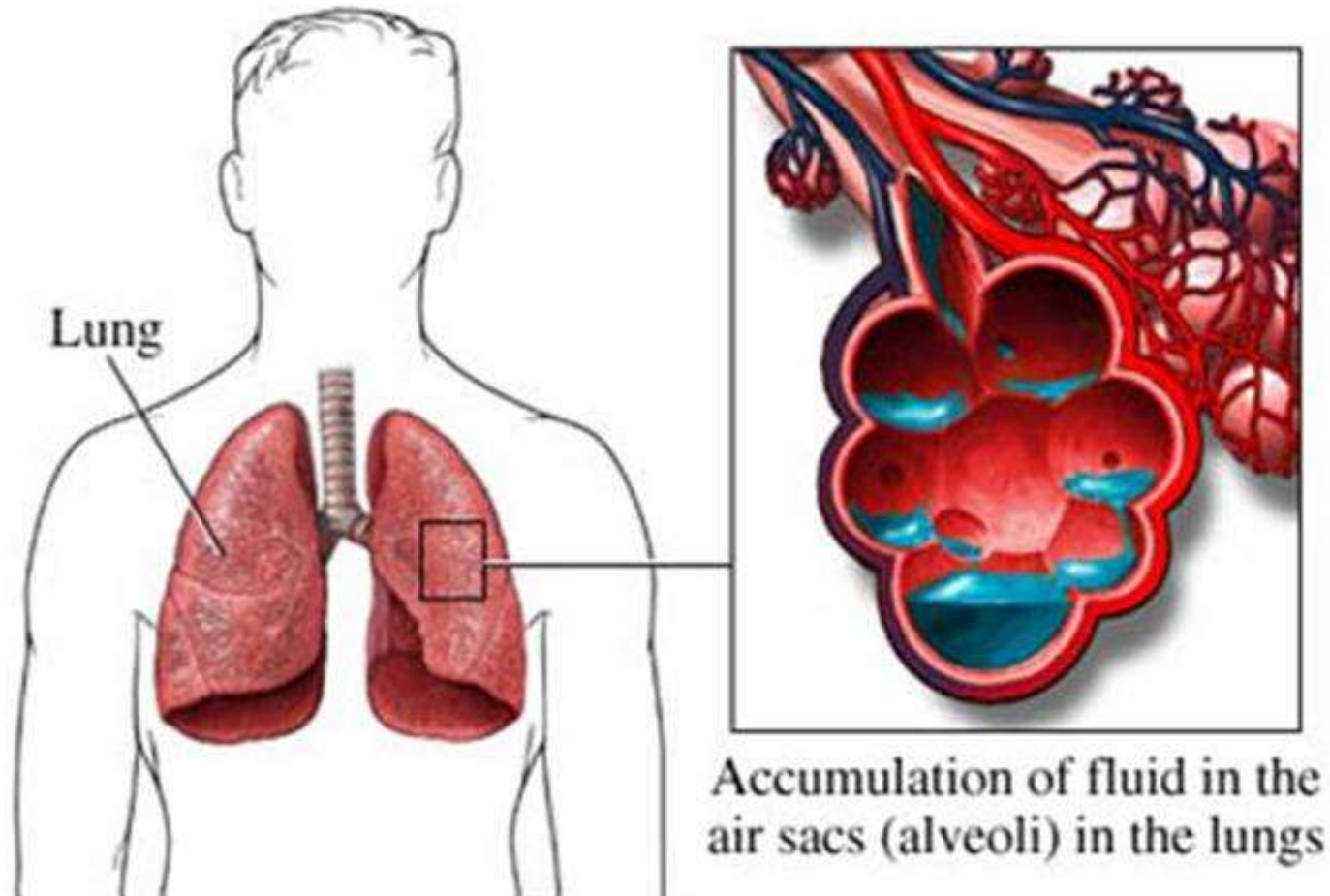


- **Cardiogenic pulmonary edema**

# Cardiogenic pulmonary edema

- Due to left ventricular failure due to IHD and fluid overload
- Pulmonary artery pressure are high  $> 18\text{mmHg}$
- Initially basal edema then goes upward..
- Heart failure cell(macrophages filled with hemosiderin)
- Fibrosis and thickening of alveolar walls and septa in long standing cases...
- Brown induration
- Engorged capillaries

# Carcinogenic pulmonary edema



- **NON CARDIOGENIC  
PULMONARY EDEMA**  
–**ARDS and ALI**

**abrupt onset of significant hypoxemia and diffuse pulmonary infiltrates in the absence of cardiac failure.**

# NON CARDIOGENIC PULMONARY EDEMA

- Accumulation of fluid inside lung parenchyma due to damage to alveolar walls.
- Fluid from alveolar enter into peri-alveolar tissue and from blood into alveoli..

# **Acute Respiratory Distress Syndrome**

- **INFECTION**

- Sepsis
- Pneumonia ...Viral, Mycoplasma, and Pneumocystis pneumonia; miliary tuberculosis
- Gastric aspiration

- **PHYSICAL/INJURY**

- Mechanical trauma, including head injuries
- Pulmonary contusions Near-drowning
- Fractures with fat embolism
- Burns Ionizing radiation

# **Acute Respiratory Distress Syndrome**

- **INHALED IRRITANTS**

- Oxygen toxicity Smoke Irritant gases and chemicals

- **CHEMICAL INJURY**

- Heroin or methadone overdose Acetylsalicylic acid  
Barbiturate overdose Paraquat

- **HEMATOLOGIC CONDITIONS**

- Multiple transfusions
  - Disseminated intravascular coagulation

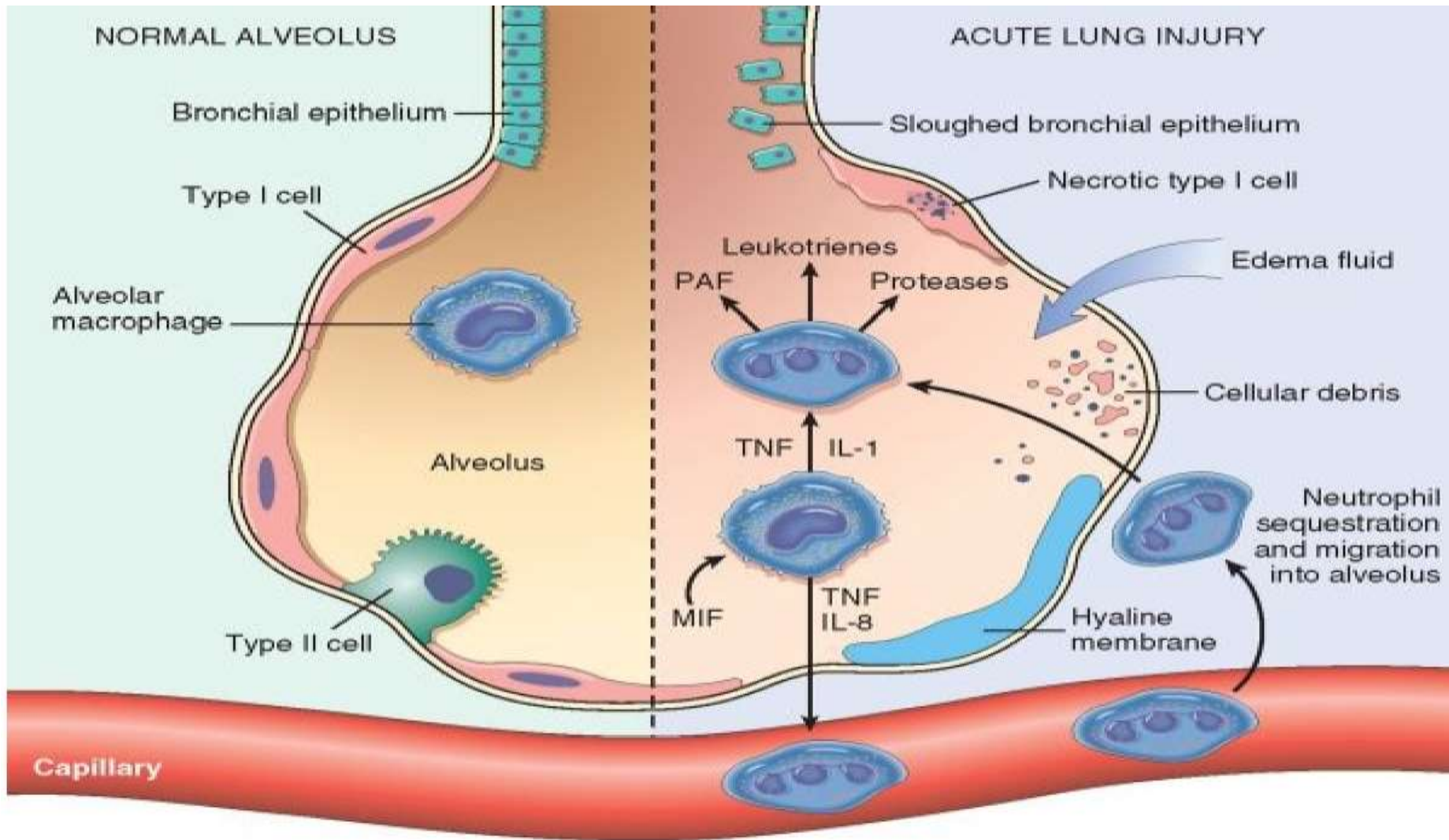
- **PANCREATITIS UREMIA CARDIOPULMONARY BYPASS HYPERSENSITIVITY REACTIONS .....**

# Acute Respiratory Distress Syndrome

- **Pathogenesis**

- Damage occurs to **alveolar capillary membrane**
- increased vascular permeability and alveolar flooding, loss of diffusion capacity, and widespread **surfactant abnormalities** caused by damage to type II pneumocytes.
- Endothelial injury also triggers the formation of microthrombi that add the insult of ischemic injury
- **Hyaline membranes** result from inspissation of protein rich edema fluid that entraps debris of dead alveolar epithelial cells





**FIGURE 15-4** The normal alveolus (*left side*) compared with the injured alveolus in the early phase of acute lung injury and acute respiratory distress syndrome. Pro-inflammatory cytokines such as interleukin 8 (IL-8), interleukin 1 (IL-1), and tumor necrosis factor (TNF) (released by macrophages), cause neutrophils to adhere to pulmonary capillaries and extravasate into the alveolar space, where they undergo activation. Activated neutrophils

# Acute respiratory distress syndrome mediator

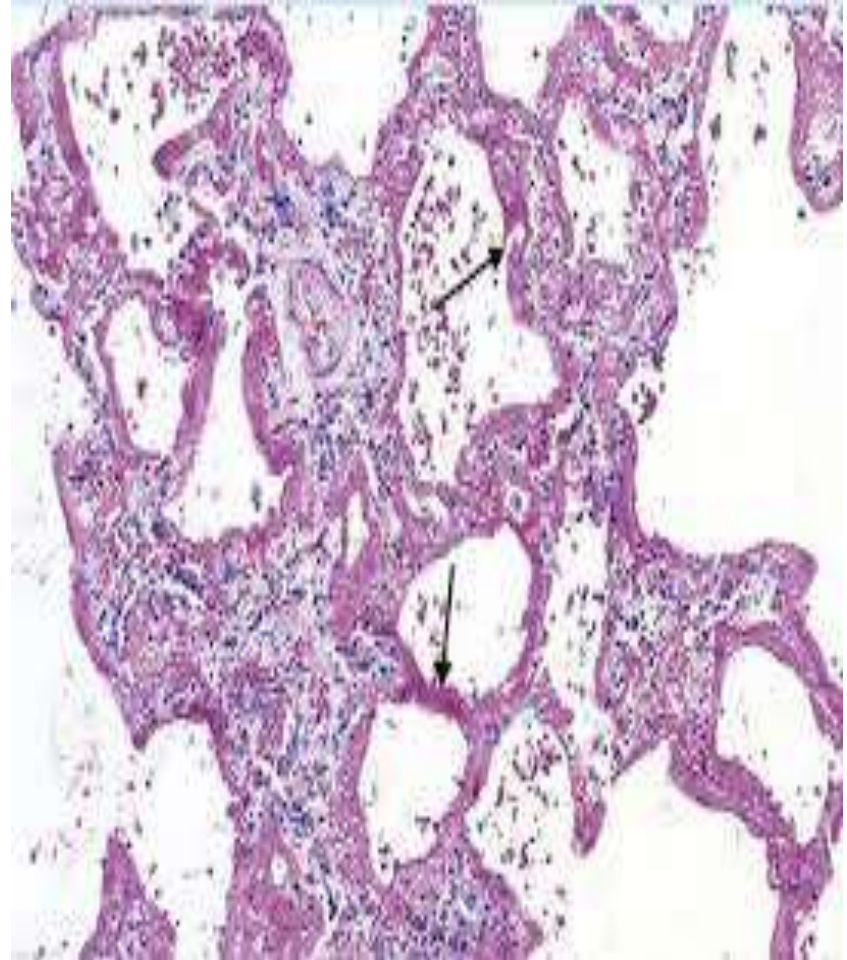
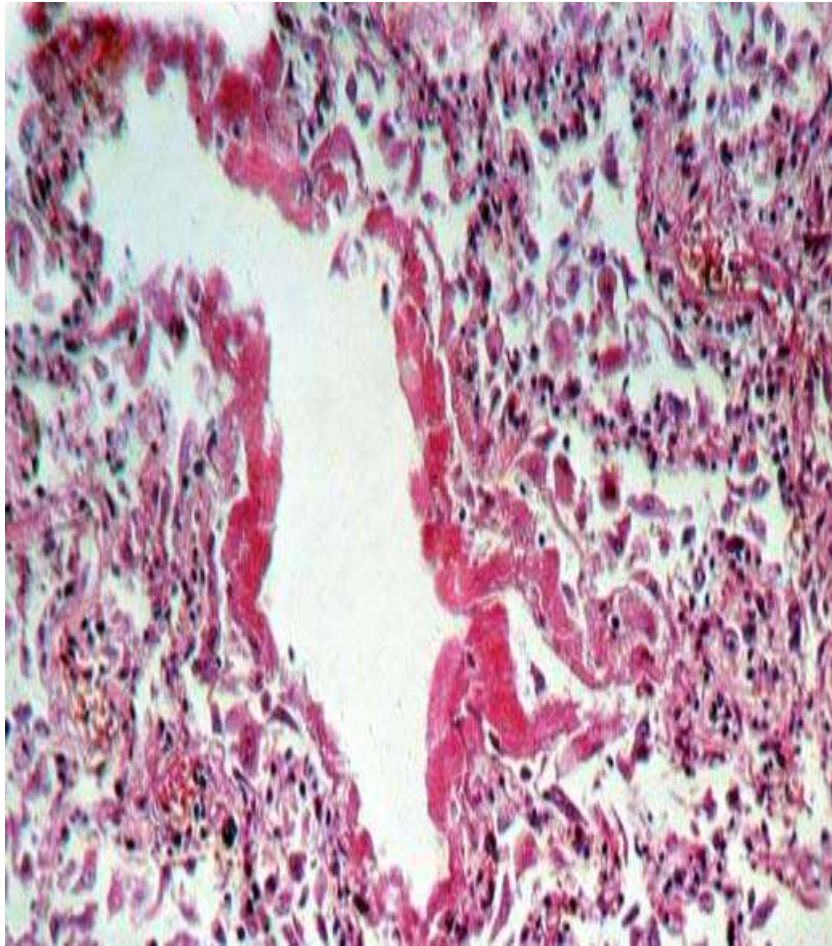
- **Proinflammatory**
  - Interleukin -1 & 8
  - Platelet activating factor PAF
  - *nuclear factor*  $\kappa B$  (NF- $\kappa B$ ),
  - TNF
  - Tissue factor
  - Protein C decreased
  - Thrombin
  - Neutrophils
    - Neutrophilic accumulation in lung
    - Increased oxidative damage
- **Anti-inflammatory**
  - Interleukin 10 ,antioxidants and antiproteases

# Acute respiratory distress system

## **Morphology.**

- **Proliferative stage /Acute stage**
  - the lungs are heavy, firm, red, and boggy.
  - They exhibit congestion, interstitial and intra-alveolar edema, inflammation, fibrin deposition, and diffuse alveolar damage.
  - The alveolar walls become lined with waxy hyaline membranes

# Acute Respiratory Distress Syndrome hyaline membrane



# Acute respiratory distress syndrome morphology and staging

- **In the organizing stage,**
  - type II pneumocytes undergo proliferation, and there is a granulation tissue response in the alveolar walls and in the alveolar spaces.
- **Fibrotic stage**
  - fibrotic thickening of the alveolar septa ensues, caused by proliferation of interstitial cells and deposition of collagen

# Acute respiratory distress syndrome

## Clinical presentation

- Dyspnea shortness of breath ,hypoxia and acute respiratory failure and right heart failure
- **Outcome** most resolve by the end of 3<sup>rd</sup> week while few undergo fibrotic stage and long term dependency on ventilator.
- high mortality unless supportive care with lung protective strategies.

# Question

- What is the function of nose?
- What are the functions of mucocilliary apparatus?
- What are the constituent of **alveolar capillary membrane**?
- What is **ALI and ARDS**?
- What are the causes of cardiogenic pulmonary edema?
- What are the causes of non cardiogenic pulmonary edema?

# Reference

- Robin basic pathology 9<sup>th</sup> edition
- ARDS group network website
- Kaplan pathology



**GET UP  
AS  
LECTURE HAS ENDED**

