SNS COLLEGE OF ALLIED HEALTH SCIENCES

SNS Kalvi Nagar, Coimbatore - 35

Affiliated to Dr MGR Medical University, Chennai

DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE TECHNOLOGY

COURSE NAME: PATHOLOGY II

II YEAR

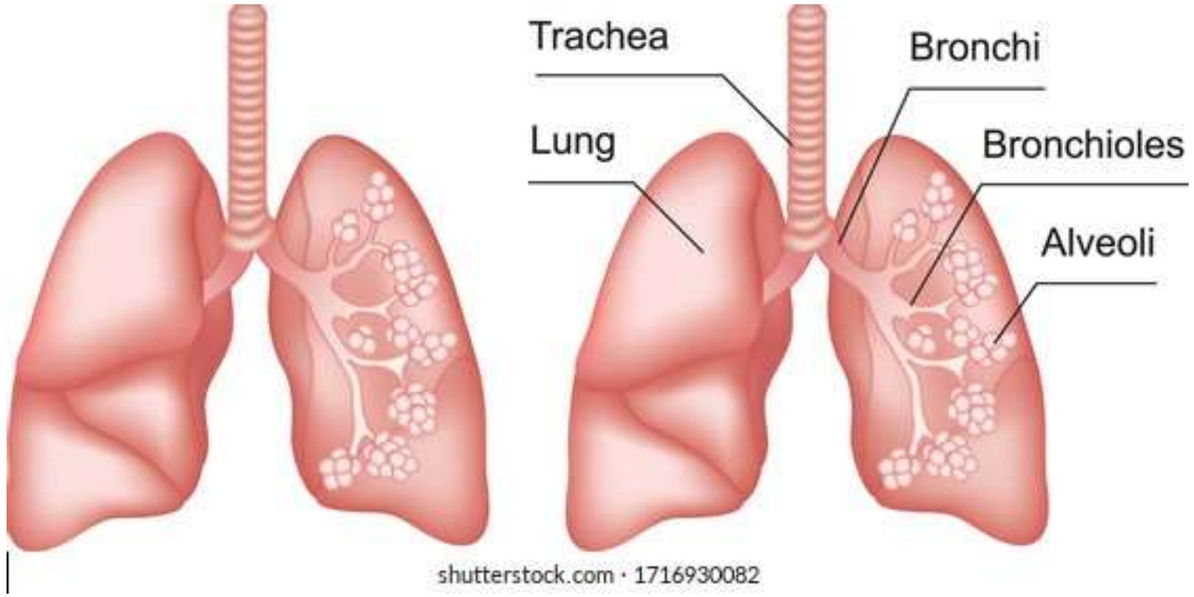
UNIT II : PATHOLOGY OF LUNG

TOPIC 1 : ARDS

Introduction

- During life, the right and left lungs are soft and spongy and very elastic.

- In the child, they are pink, but with age, they become dark and mottled because of the inhalation of dust particles.

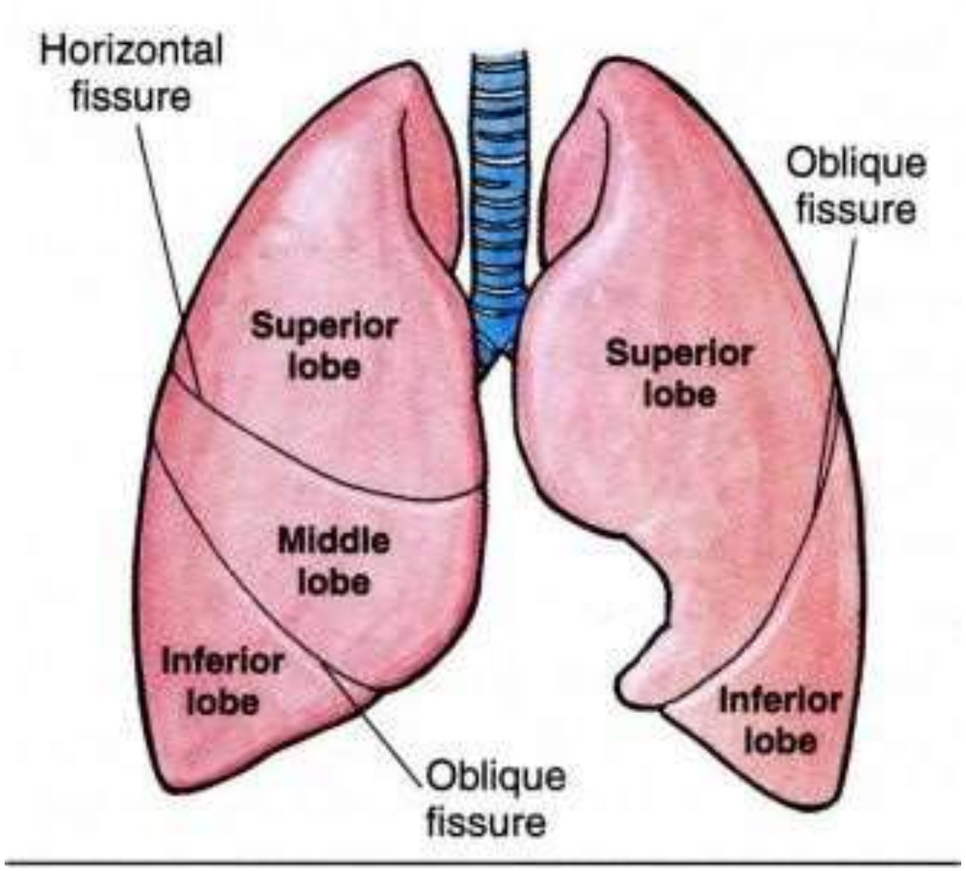


Anatomy of Lung

RIGHT LUNG:

- The normal *adult right lung* weighs 375 to 550 gm (average 450 gm)

- It divided by two fissures (oblique and horizontal) into three lobes—the upper, middle and lower lobes.



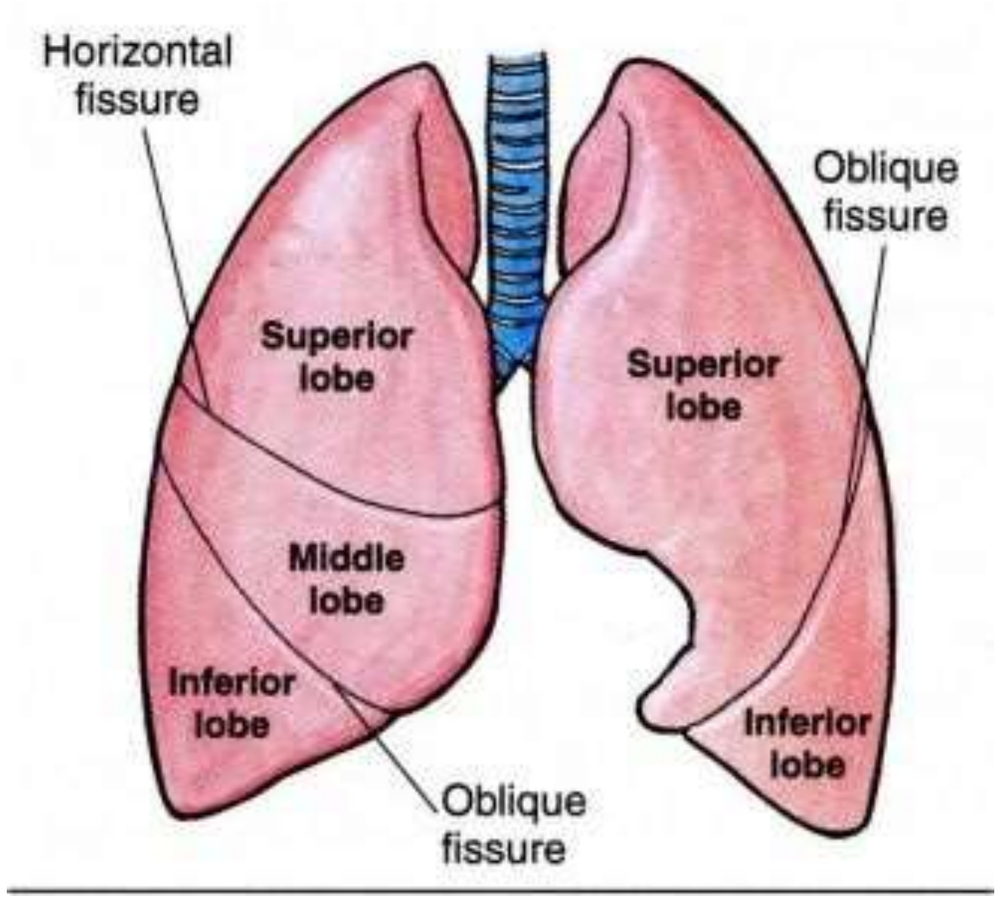
Anatomy of Lung

LEFT LUNG:

- The weight of the normal *adult left lung* is 325 to 450 gm (average 400 gm)

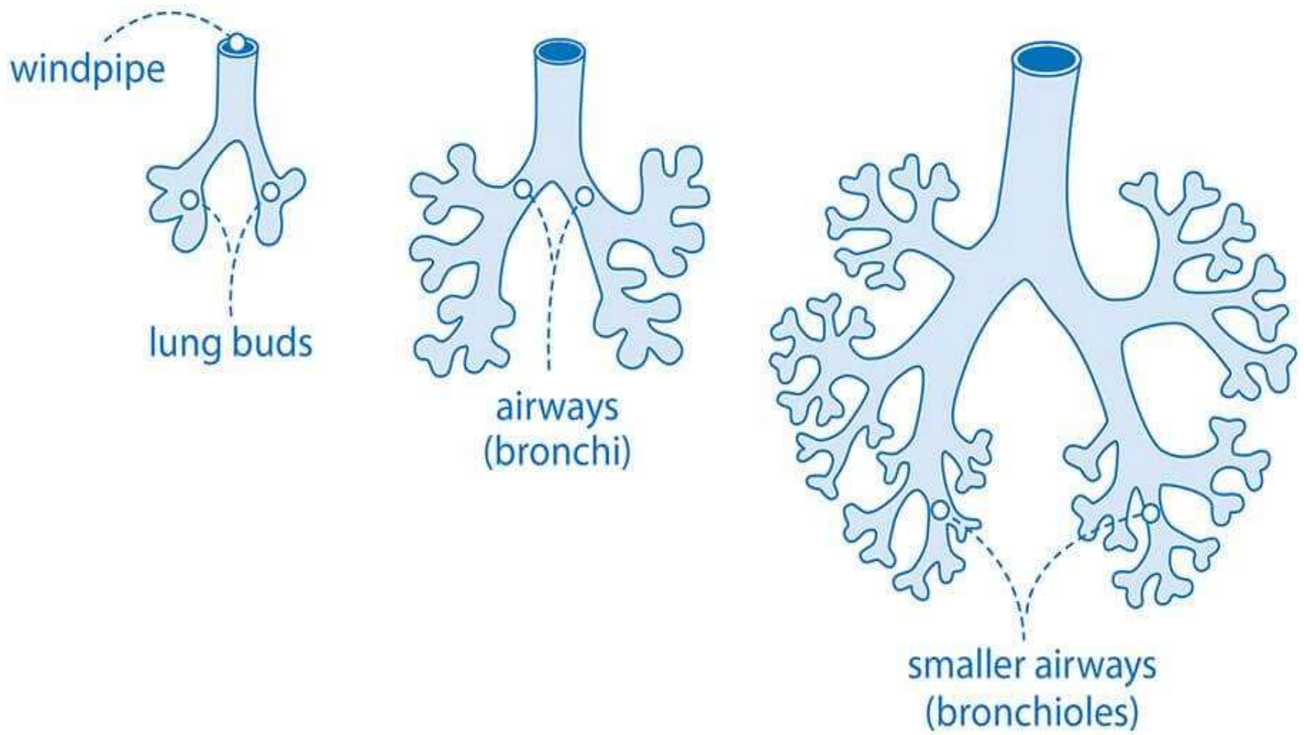
- It has one fissure (oblique) dividing it into two lobes—the upper and lower lobes

- The middle lobe is represented by the lingula



Airway of Lung

- The airways of the lungs arise from the trachea
- It divided into right and left main bronchi
- It divide and subdivide further, eventually terminating into the alveolar sacs

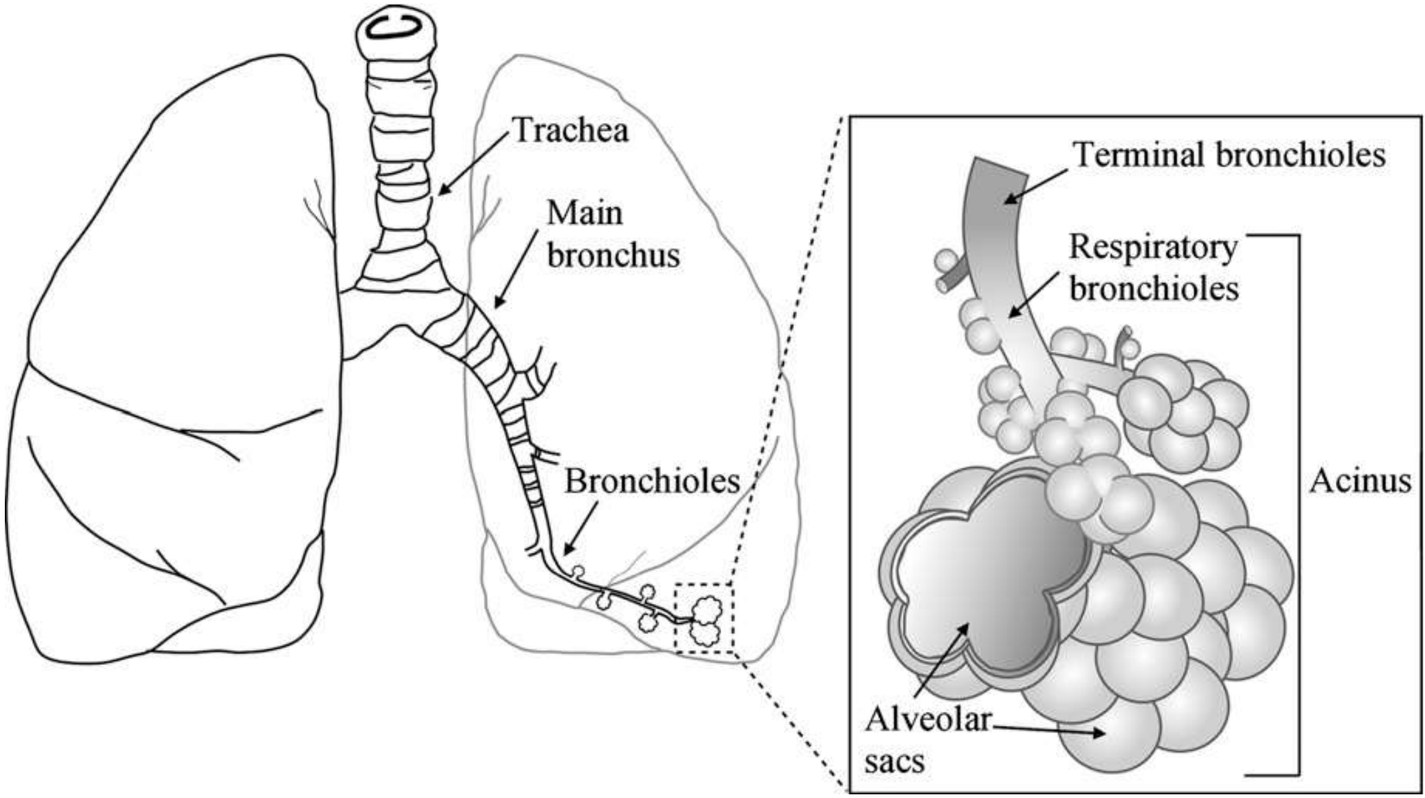


Airway of Lung

The part of the lung tissue distal to a terminal bronchiole is called an ***acinus.***

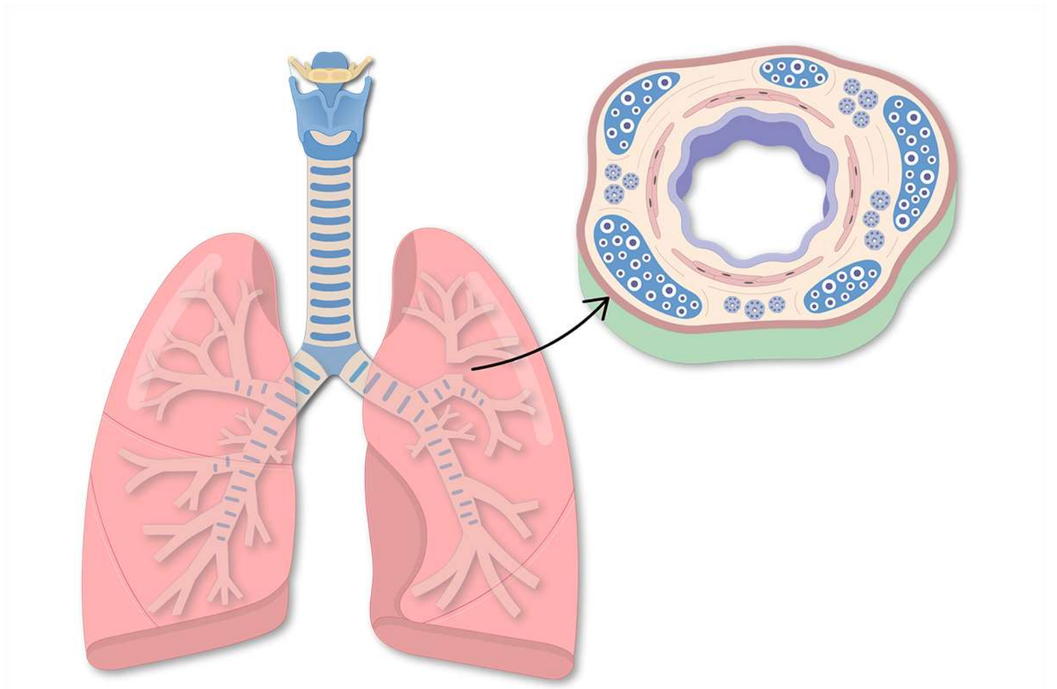
An acinus consists of 3 parts:

1. **respiratory bronchioles**
2. **Several alveolar ducts.**
3. **Alveolar sacs**



Pathology of Lung

- Paediatric lung disease (congenital and acquired)
- Pulmonary vascular disease (PVD)
- Pulmonary infections
- Chronic obstructive pulmonary disease (COPD)
- Chronic restrictive pulmonary disease
- Tumours of lungs



Paediatric lung disease

There are more number of congenital abnormalities of lungs. Some of them are,

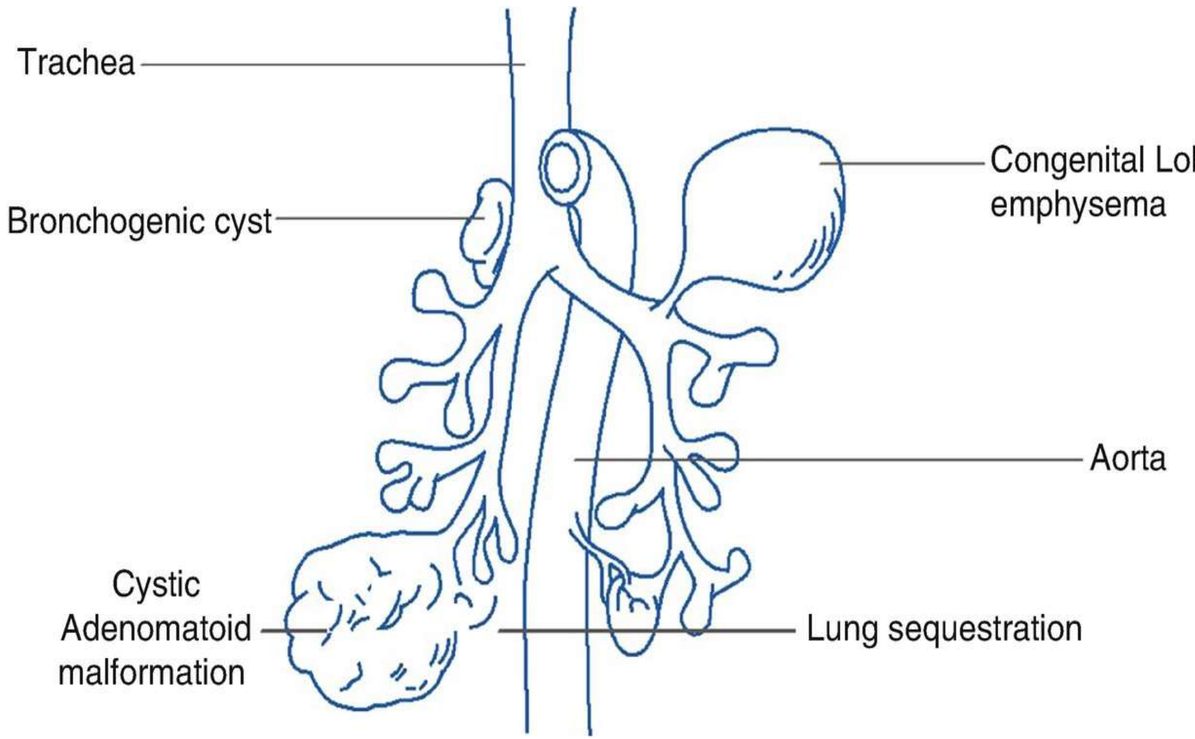
- **Congenital cysts**
- **Broncho-pulmonary Sequestration**
- **Acute Respiratory Distress Syndrome (Hyaline membrane disease)**
- **Bronchopulmonary Dysplasia**
- **Atelectasis and Collapse**
- **Sudden Infant Death Syndrome**

Congenital Cysts

- Developmental defects involving deficiency of bronchial or bronchiolar cartilage, elastic tissue and muscle result in **congenital cystic disease of lungs.**

- These cysts may contain air or may get infected and become abscesses.

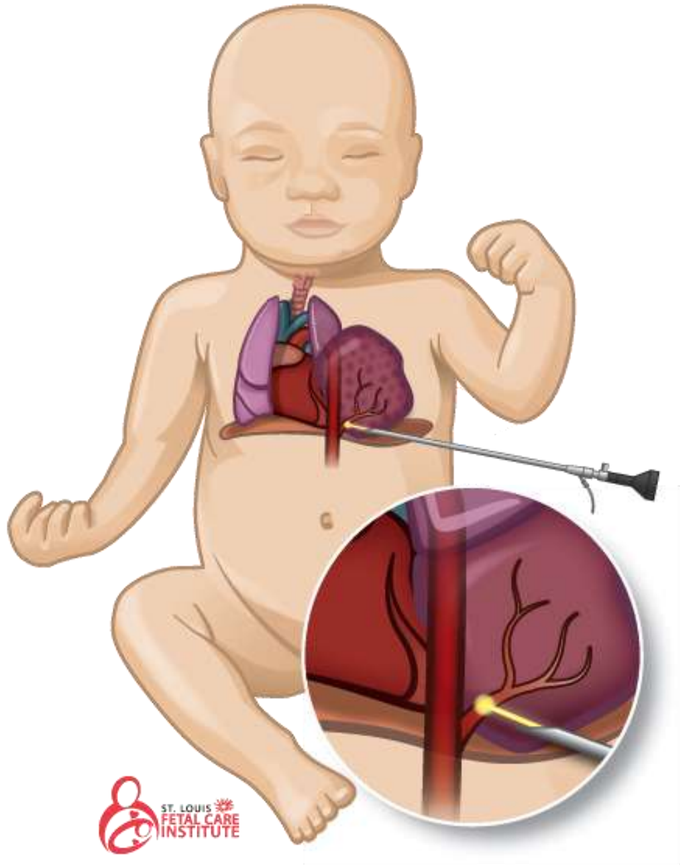
- Cysts may rupture into bronchi producing haemoptysis, or into the pleural cavity producing pneumothorax.



Bronchopulmonary Sequestration

- Sequestration is the presence of lobes or segments of lung tissue which are not connected to the airway system.

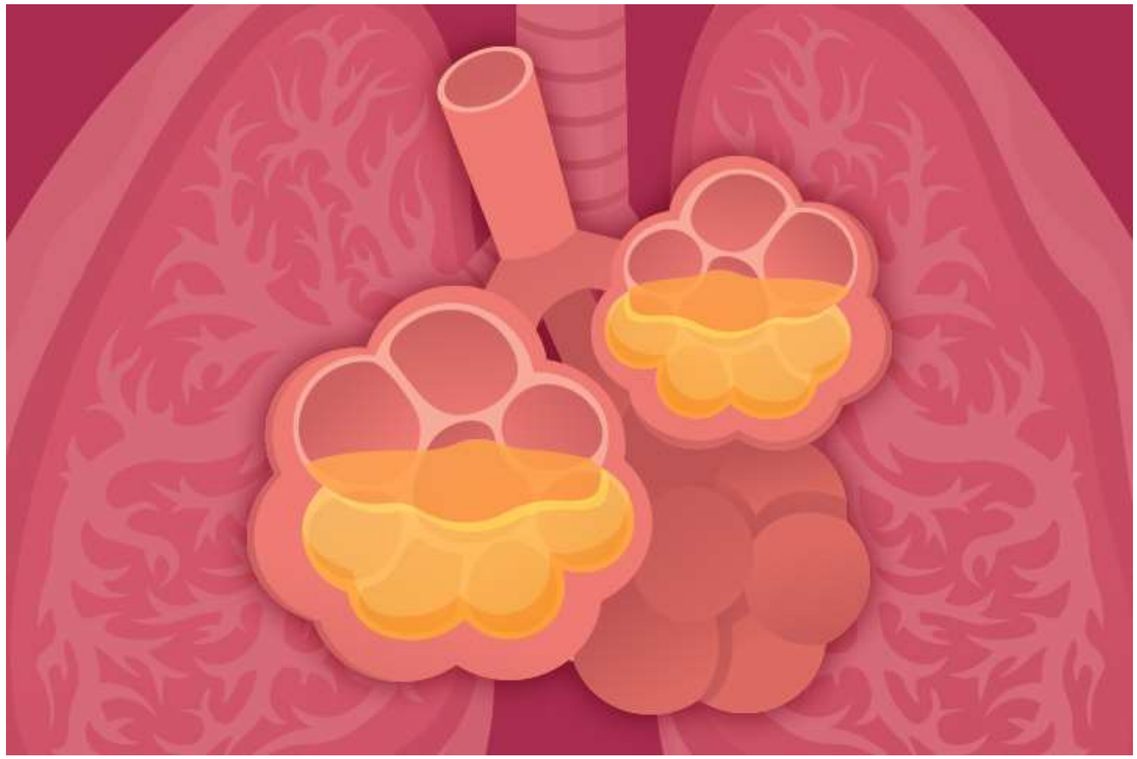
- Sequestration may be Intralobar or Extralobar.



ARDS (Acute Respiratory Distress Syndrome)

ARDS

- Acute Respiratory Distress Syndrome (ARDS) is a life threatening lung condition that prevents enough oxygen from getting into blood

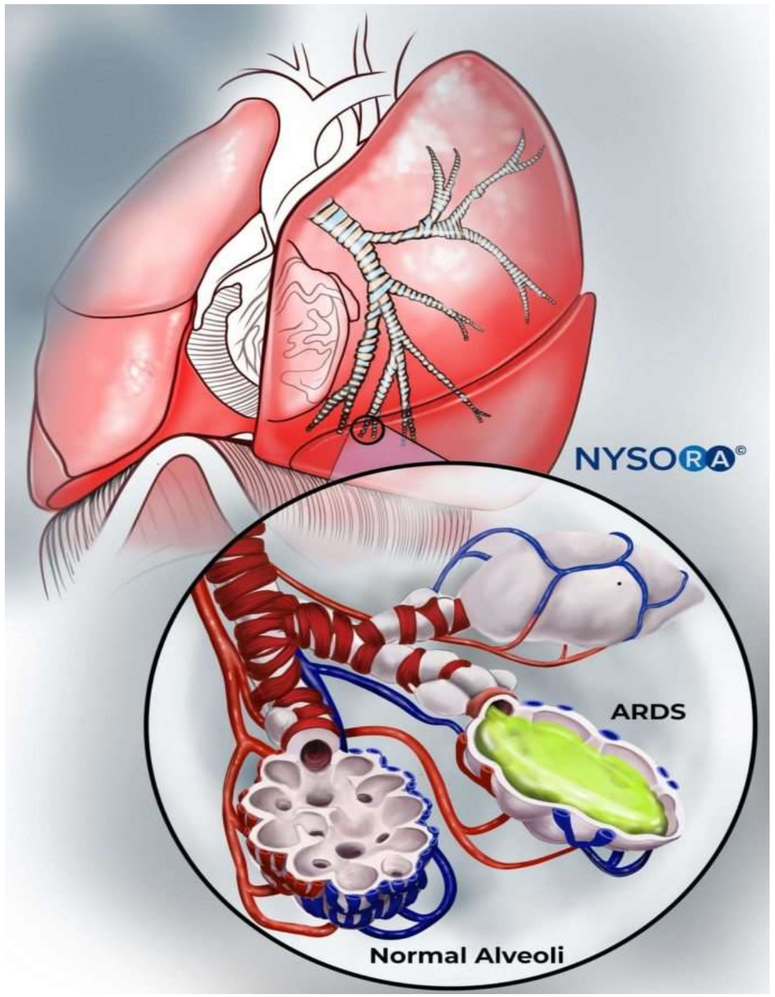


- Other names of ARDS

1. Adult hyaline-membrane disease
2. Adult respiratory insufficiency syndrome
3. High output respiratory failure
4. Congestive atelectasis
5. Haemorrhagic lung syndrome
6. Stiff-lung syndrome
7. Shock lung
8. White lung

Definition

Acute respiratory distress syndrome (ARDS) is a sudden and progressive form of acute respiratory failure in which the ***alveolar capillary membrane becomes damaged and more permeable*** to intravascular fluid resulting in severe dyspnoea, hypoxemia and diffuse pulmonary infiltrates.



Clinical Features

- **Neonatal ARDS** occurring in new born infants begins with dyspnoea within a few hours after birth.

- Tachypnoea

- Hypoxia and

- Cyanosis in severe cases death may occur within a few hours.

Clinical Features

Adult ARDS is known by other ways like,

- Shock-lung syndrome
- Diffuse alveolar damage (DAD)
- Acute alveolar injury
- Traumatic wet lungs
- Post-traumatic respiratory insufficiency

Neonatal Etiology

- Preterm infants
- Infants born to diabetic mothers
- Delivery by caesarean section
- Infants born to mothers with previous premature infants
- Excessive sedation of the mother causing depression in respiration of the infant
- Birth asphyxia from various causes such as coils of umbilical cord around the neck

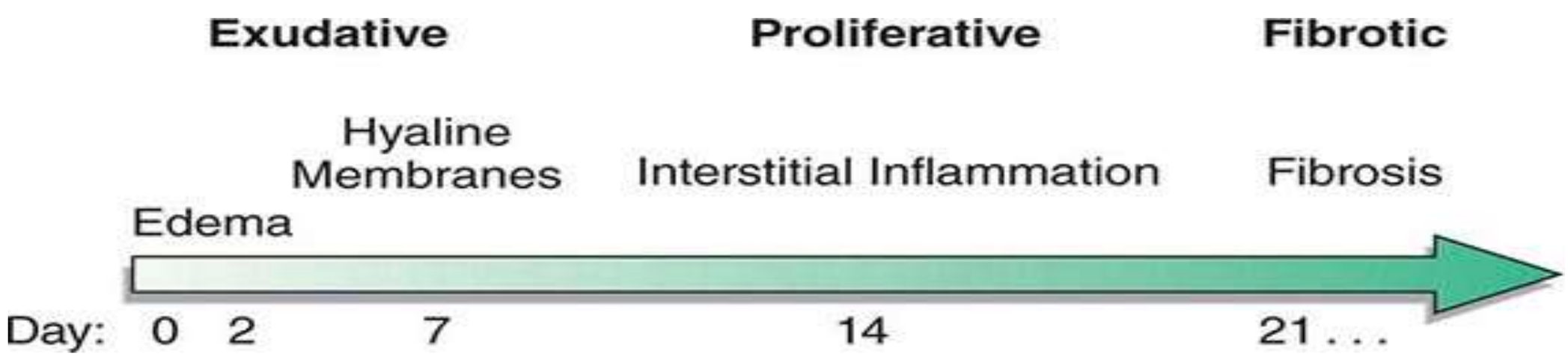
Adult Etiology

- Shock due to sepsis, trauma, burns
- Diffuse pulmonary infections, chiefly viral pneumonia
- Pancreatitis
- Oxygen toxicity
- Inhalation of toxins and irritants e.g. smoke, war gases, nitrogen dioxide, metal fumes etc.
- Narcotic overdose
- Drugs e.g. salicylates, colchicine
- Aspiration pneumonitis
- Fat embolism
- Radiation.

Pathophysiology

3 phases

1) **Exudative phase** - **Hyaline Membrane** - 0 to 7 day
2) **Proliferative phase** - **Interstitial Inflammation** - 7 to 21 day
3) **Fibrotic phase** - **Fibrosis** - after 21 days

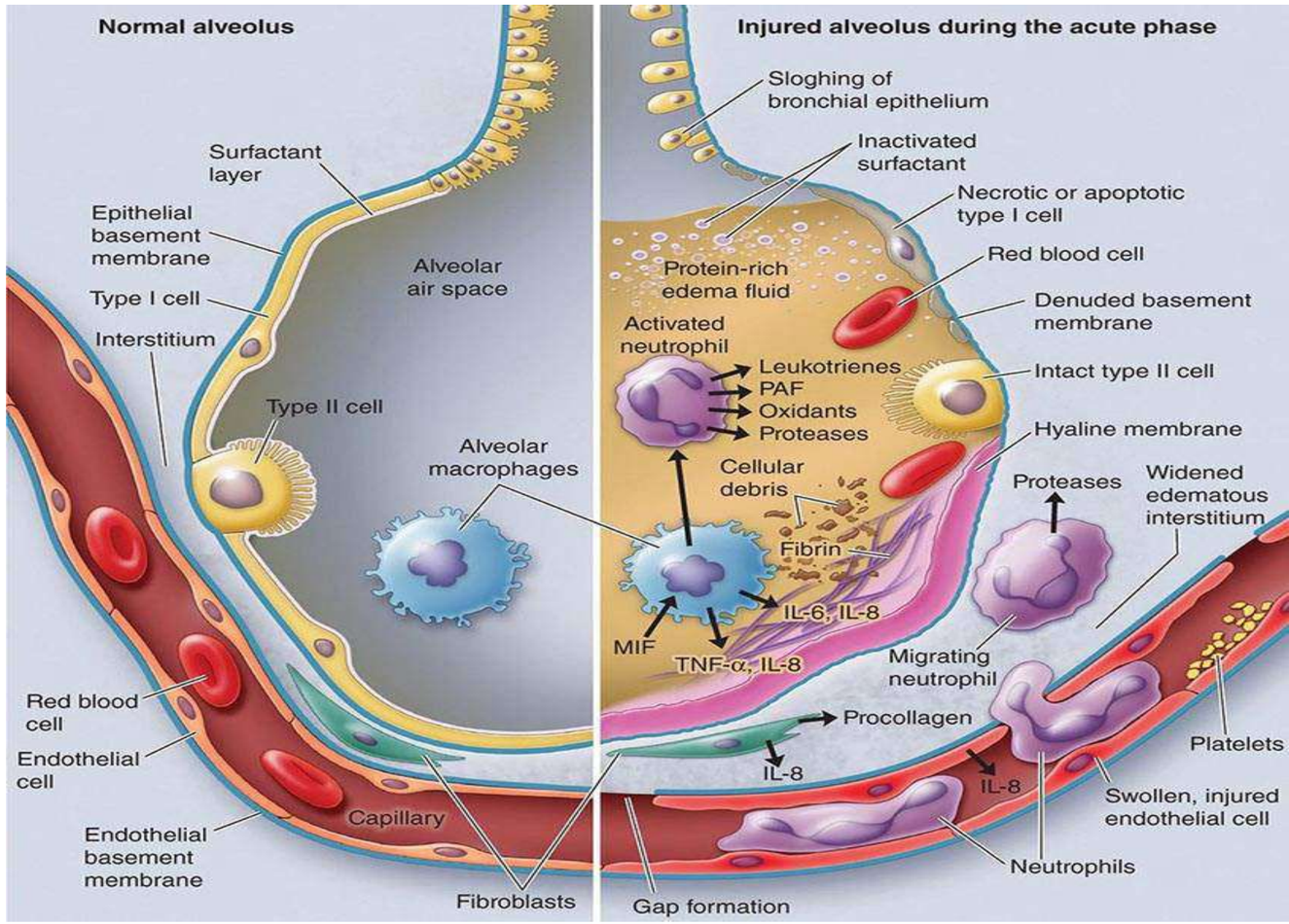


Exudate Phase

- Direct or indirect injury to the alveolus causes **alveolar macrophages** to release **pro-inflammatory cytokines**

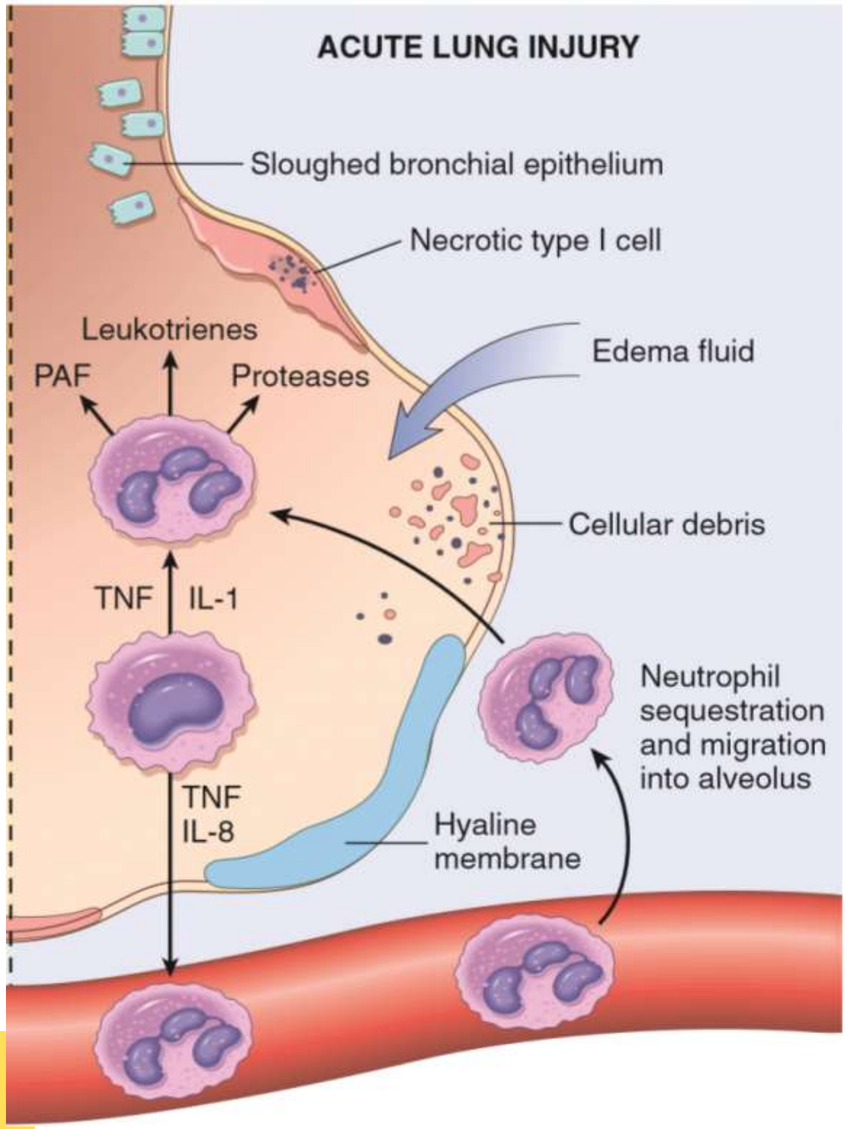
- Cytokines attract neutrophils into the alveolus and interstitum, where they damage the **alveolar-capillary membrane (ACM).**

- ACM integrity is lost, interstitial and alveolus fills with **proteinaceous fluid**, surfactant can no longer support alveolus **(loss of surfactant)**



Exudate Phase

- Neutrophilic Infiltrate
- Alveolar Haemorrhage
- Proteinaceous Pulmonary Oedema
- Cytokines (TNF, IL1,8)
  - » ↑ Inflammation
  - » ↑ Oxidative Stress and Protease Activity
  - » ↓ Surfactant Activity
- Elastase - induced capillary and alveolar damage
  - » ↑ Alveolar flooding
  - » ↓ Fluid clearance



Exudate Phase

Increased vascular permeability

↓

Type 1 – pneumocytes necrosis

↓

Intra-alveolar oedema

↓

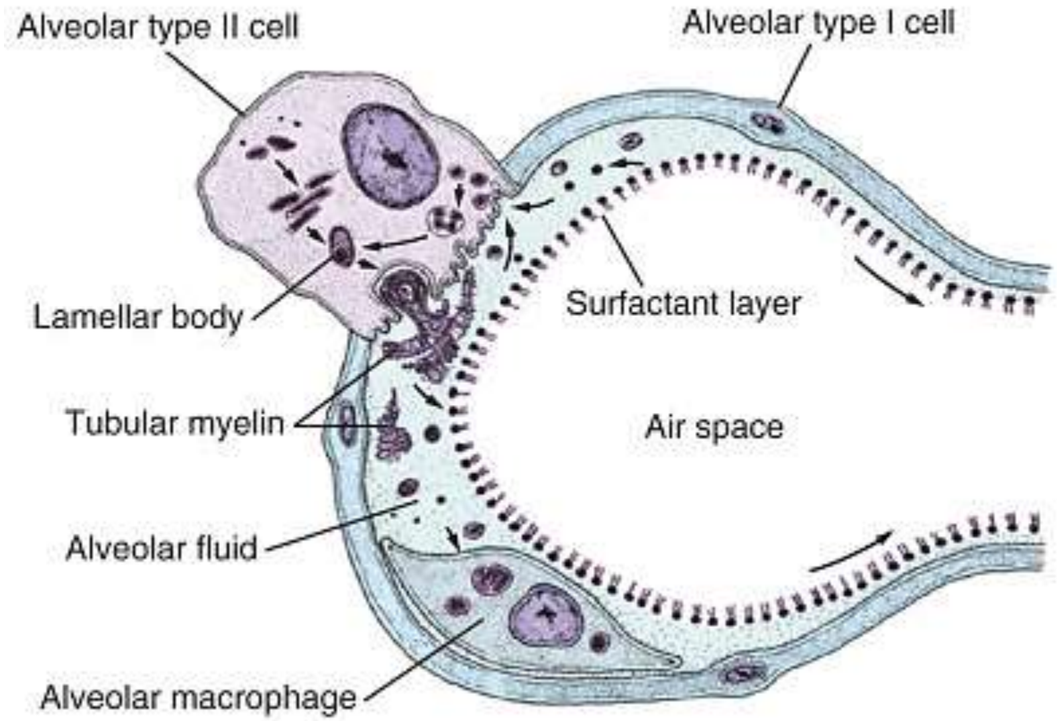
Fibrin deposition → Formation of hyaline membranes → Loss of surfactant

↓

Collapse → **‘Stiff lung’**

Proliferative Phase

- Lasts around 7-21 days
- **Initiation of lung repair occurs**
- Organisation of alveolar exudates shift from PMN to lymphocyte rich infiltrate
- Type II Pneumocyte synthesize surfactant
- Proliferate differentiate into Type I cells
- Re-line alveolar walls

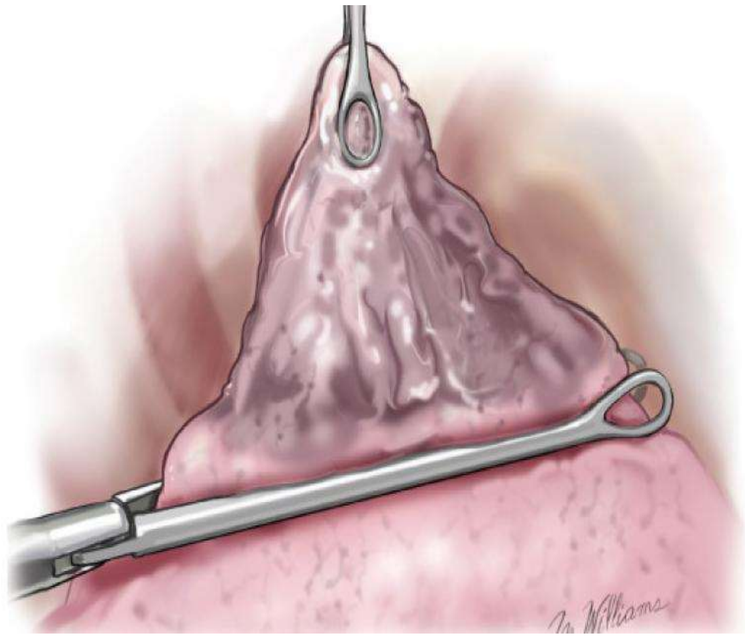


Fibrotic Phase

- Extensive alveolar duct and interstitial fibrosis.

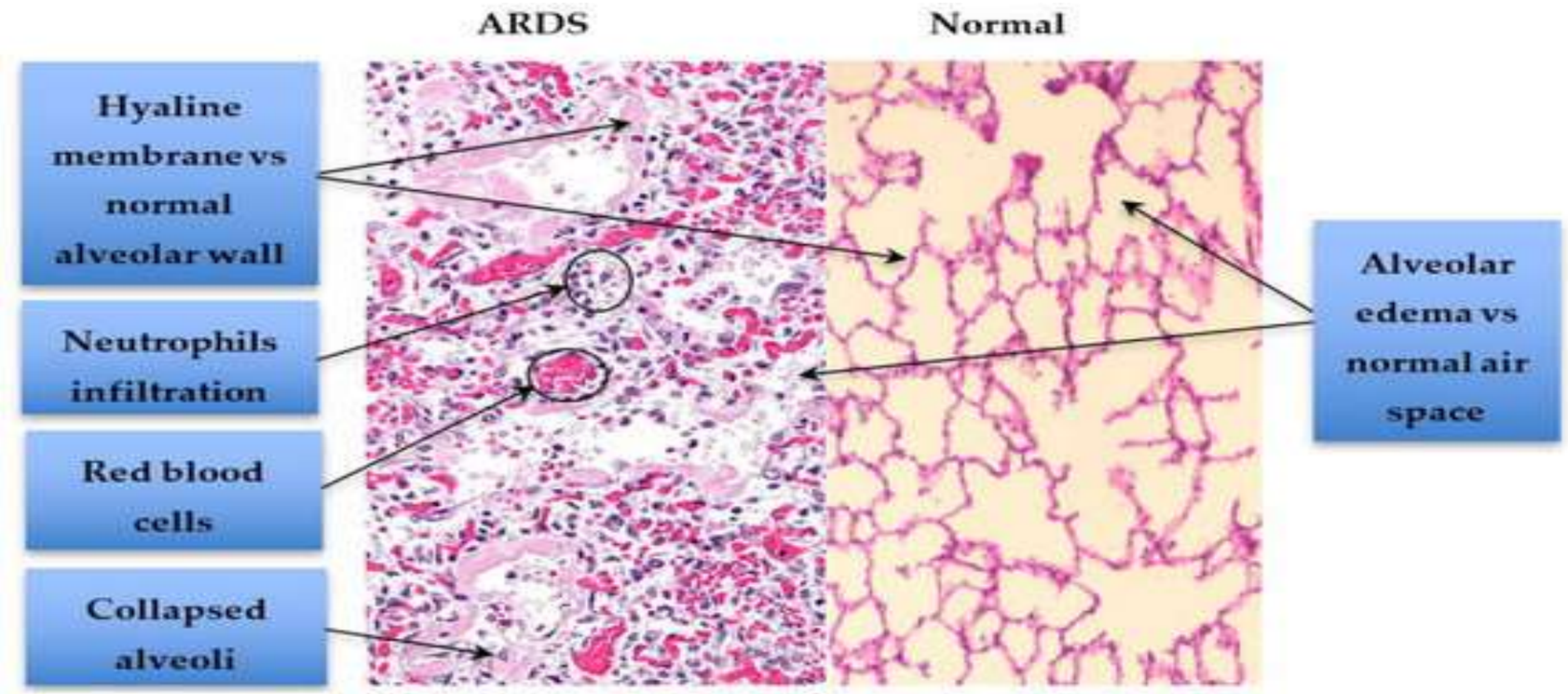
- Emphysema like changes with bullae formation.

- Fibrosis may result into progressive vascular occlusion and pulmonary hypertension.



Morphology

- Collapsed alveoli
- Necrosis of alveolar epithelial cells
- Interstitial and intra-alveolar oedema



Consequences of ARDS

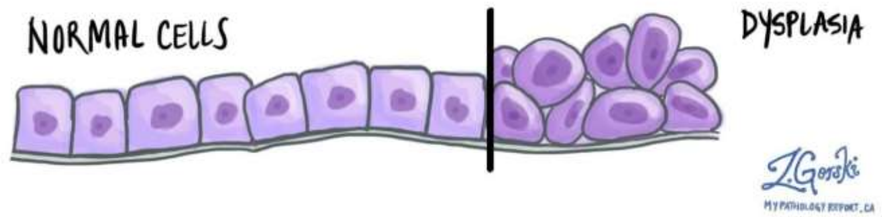
Death

- High mortality rate
- Stiff lung in adult ARDS fails to respond to oxygen therapy

Resolution

- Milder cases of neonatal ARDS recover with adequate oxygen therapy
- The hyaline membrane is liquefied by the neutrophils and macrophages

Other Sequele - bronchopulmonary dysplasia



Diagnosis

- Mainly clinical-no specific diagnostic tests
- Laboratory tests
- Diagnostic imaging
- Hemodynamic monitoring
- Bronchoscopy

Lab Tests

- **ABG analysis**

- To exclude cardiogenic pulmonary edema – **Echocardiogram**

- **Hematological**- either leukocytosis or leucopenia

Treatment

- Treat the underlying cause.
- Conservative fluid management.
- Non invasive ventilation.
- Mechanical Ventilation