DISEASES OF THE RESPIRATPORY SYSTEM

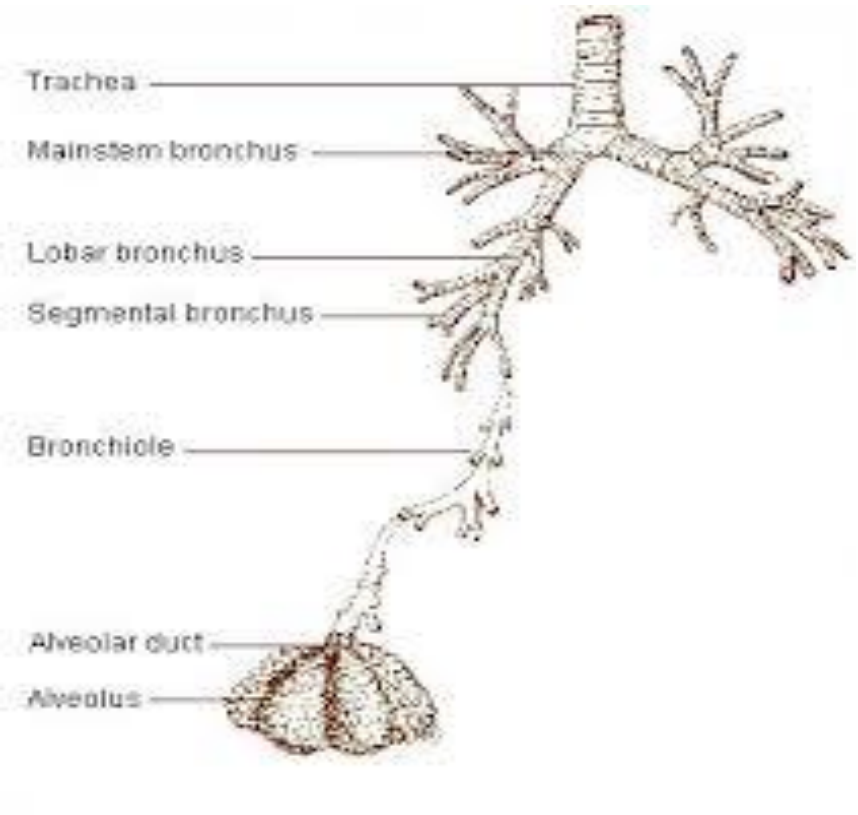
LECTURE 1

DR HEYAM AWAD

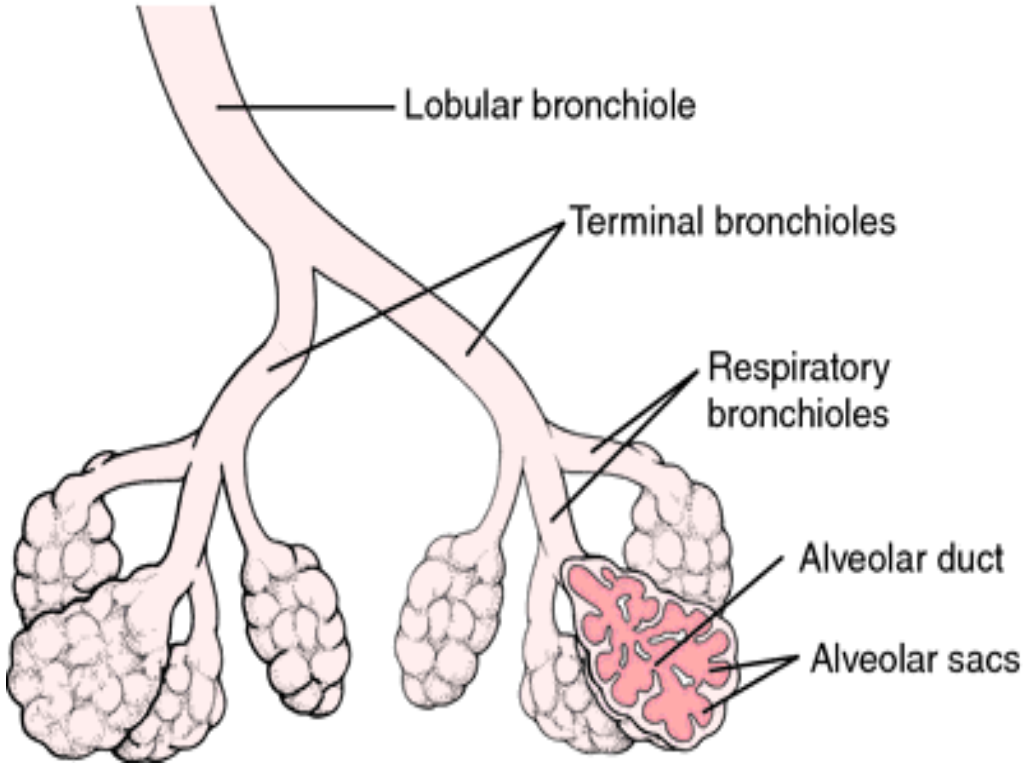
FRCPATH

- **SRTUCTURE AND FUNCTION OF THE RESPIRATOY SYSTEM.**
- **ATELECTASIS.**
- **ADULT RESPIRATORY DISTRESS SYNDROME.**

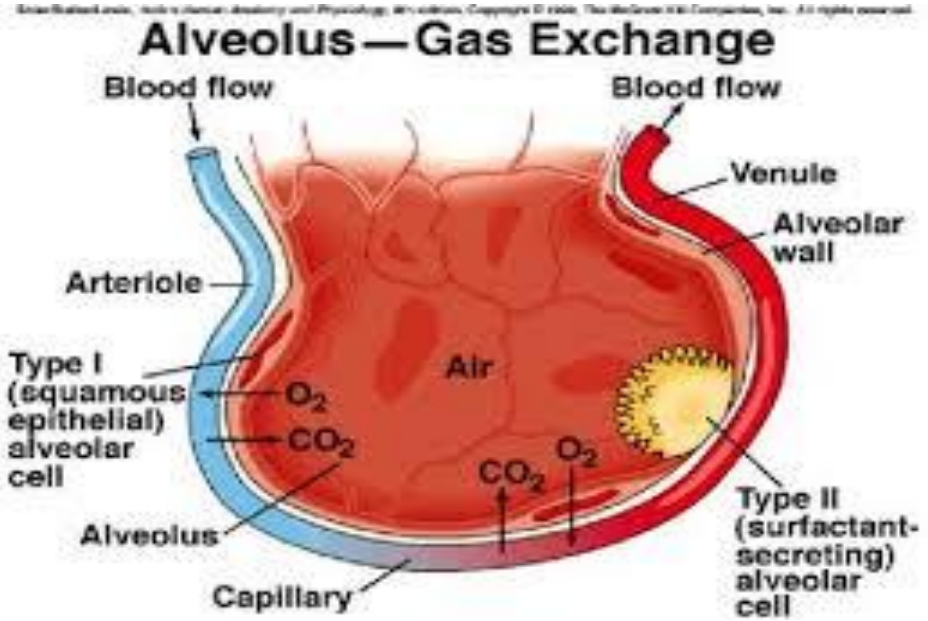
STRUCTURE OF THE RESPIRATORY SYSTEM



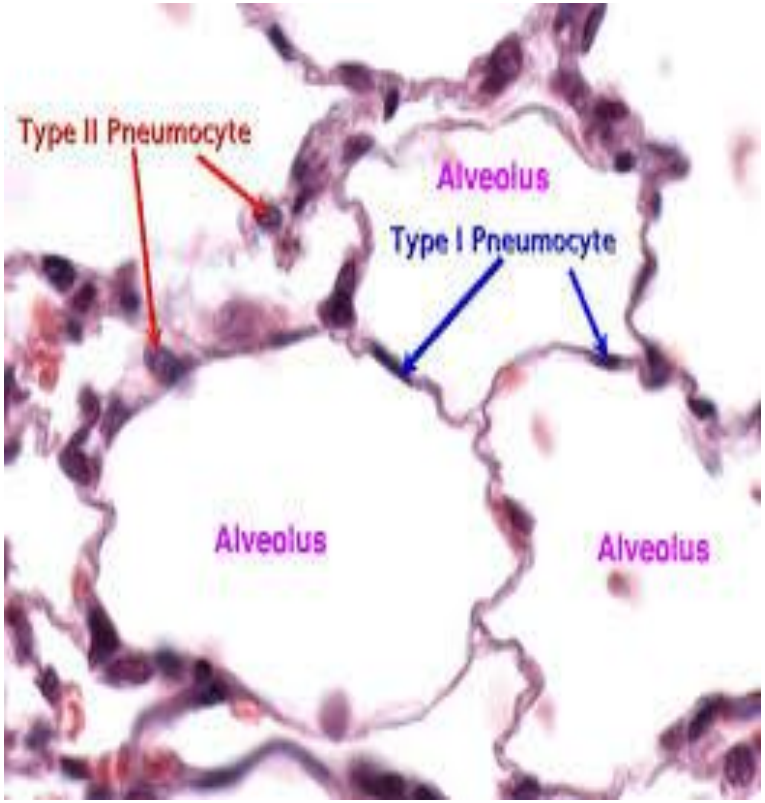
STRUCTURE OF THE RESPIRATORY SYSTEM



ALVEOLI


Alveolus—Gas Exchange
Blood flow
Blood flow
Venule
Alveolar wall
Arteriole
Type I (squamous epithelial) alveolar cell
Air
O_2
CO_2
CO_2
O_2
Alveolus
Capillary
Type II (surfactant-secreting) alveolar cell

TYPE 1 & 2 PNEUMOCYTES



ALVEOLI: LARGE SURFACE AREA

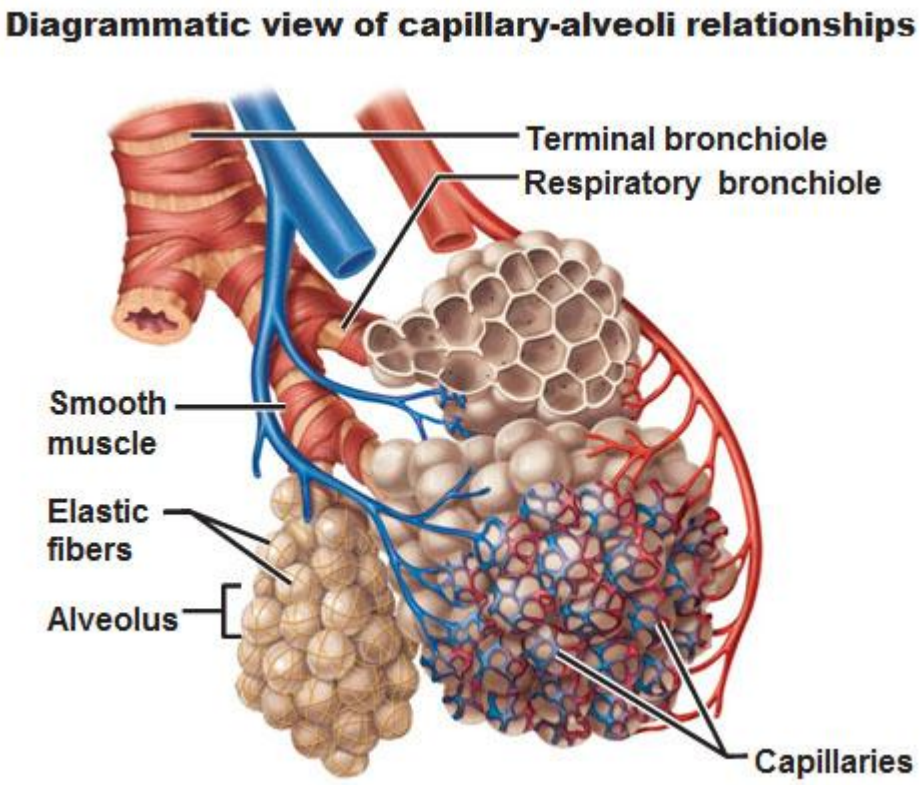
Lungs

A pair of lungs contains about ***300 million alveoli.***

This subdivides the volume of the lungs and creates a total alveolar surface area of about 1000 ft.^{2} (like a room 33 ft. x 30 ft.).

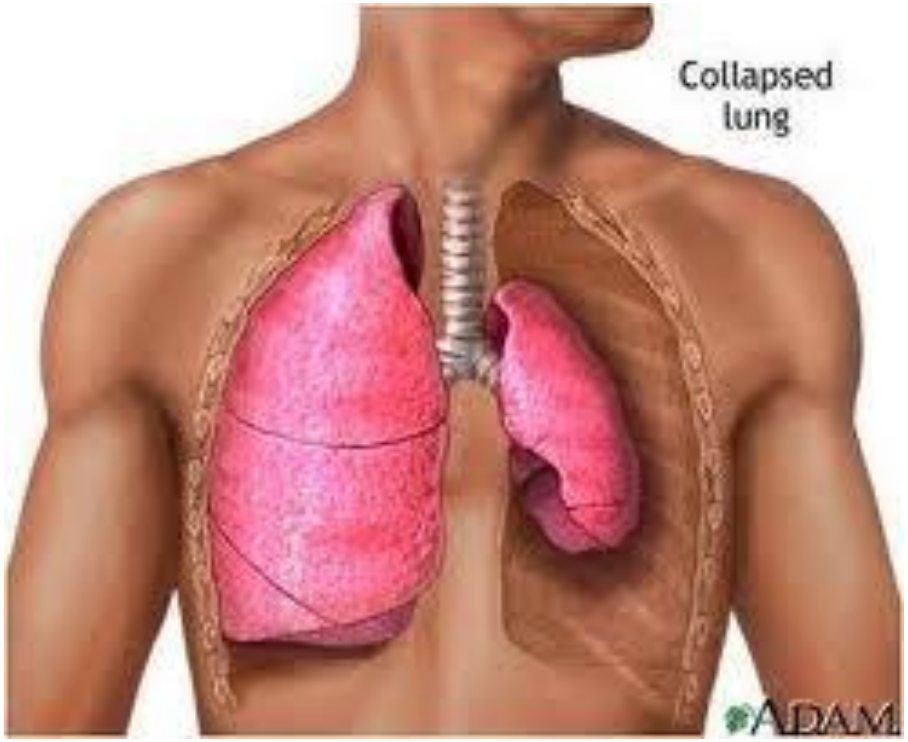
The advantage to having this is that it allows for a very ***large surface area for gas exchange.***

ALVEOLI: RICH BLOOD SUPPLY

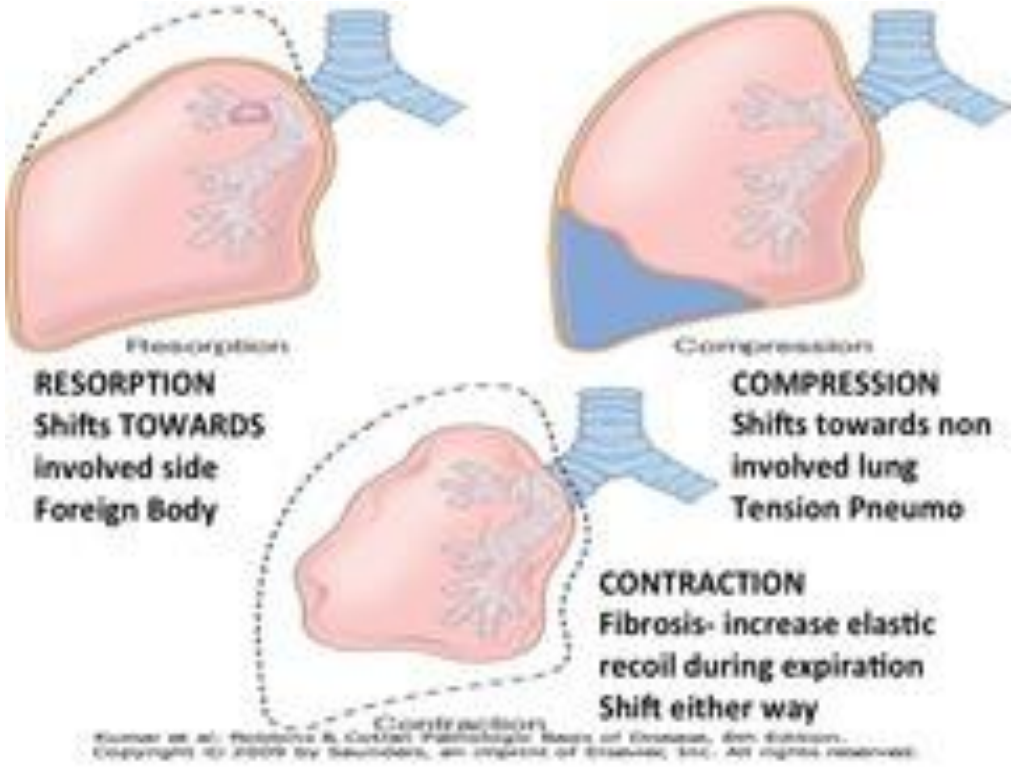


ALVEOLI: THIN MEMBRANES

ATELECTASIS = LUNG COLLAPSE



TYPES OF ATELECTASIS



RESORPTION ATELECTASIS

- ***OBSTRUCTION BY:***

  *MUCOUS OR MUCOPURULENT PLUG (POST-OP, ASTHMA, BRONCHIECTASIS OR CHRONIC BRONCHITIS)

  *TUMOUR.

  *FOEIGN BODY .

COMPRESSION ATELECTASIS

ACCUMOLATION OF :

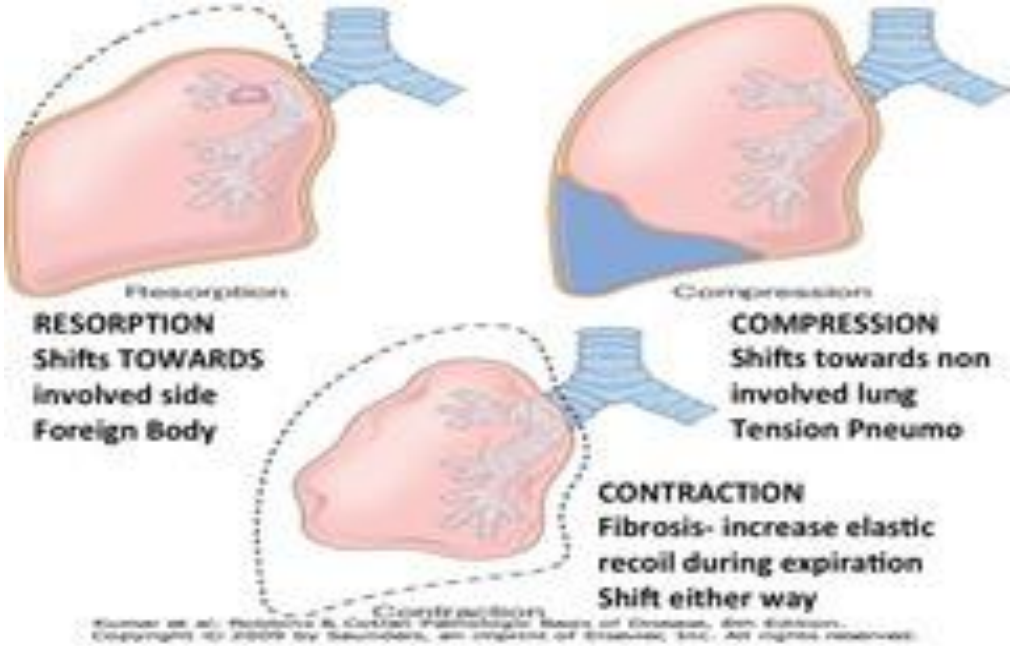
- *FLUID* (PLEURAL EFFUSION)
- BLOOD (HAEMOTHORAX)
- AIR (PNEUMOTHORAX)

ALL WITHIN THE PLEURAL CAVITY.

CONTRACTION ATELECTASIS

- LOCAL OR GENERALISED FIBROSIS.

- ATELECTASIS…….IS IT REVERSIBLE???????

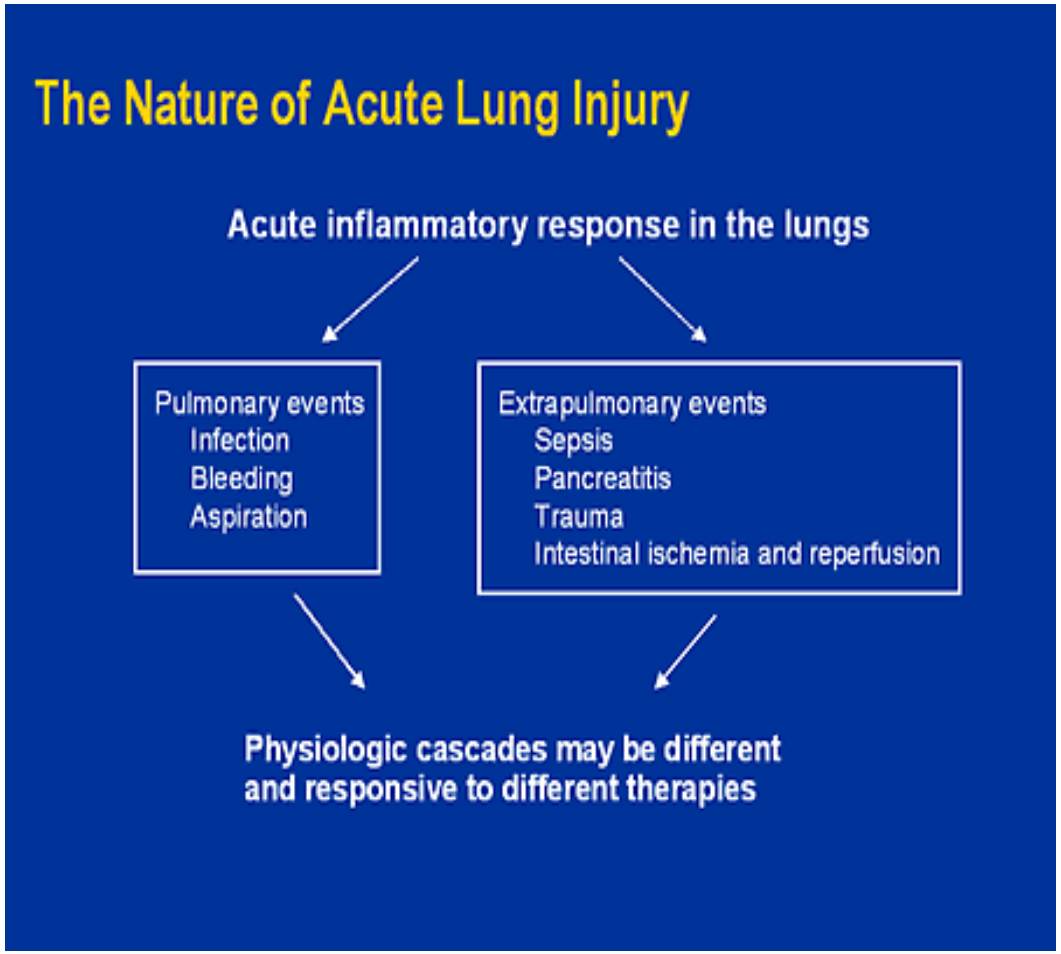


ADULT RESPIRATORY DISTRESS SYNDROME

ACUTE LUNG INJURY

- BILATERAL PULMONARY DAMAGE.
- ENDOTHELIAL AND EPITHELIAL DAMAGE.
- DUE TO DIRECT OR INDIRECT LUNG INJURY.
- ACUTE DYSPNEA + HYPOXEMIA + BILATERAL PULMONARY INFILTRATES WITHOUT PRIMARY LEFT SIDED HEART FAILURE.
- CAN PROGRESS TO ACUTE RESPIRATORY DISTRESS SYNDROME.

The Nature of Acute Lung Injury



MAIN CAUSES OF ACUTE LUNG INJURY

DIRECT LUNG DAMAGE :

- PNEUMONIA.
- ASPIRATION.

INDIRECT LUNG INJURY :

- SEPSIS.
- SEVERE TRAUMA WITH SHOCK.

ACUTE RESPIRATORY DISTRESS SYNDROME: CLINICAL FEATUERES.

- LIFE THREATENING RESPIRATORY INSUFFICIENCY.
- CYANOSIS.
- HYPOXEMIA REFRACTORY TO OXYGEN THERAPY.
- MAY PROGRESS TO MULTISYSTEM ORGAN FAILURE.

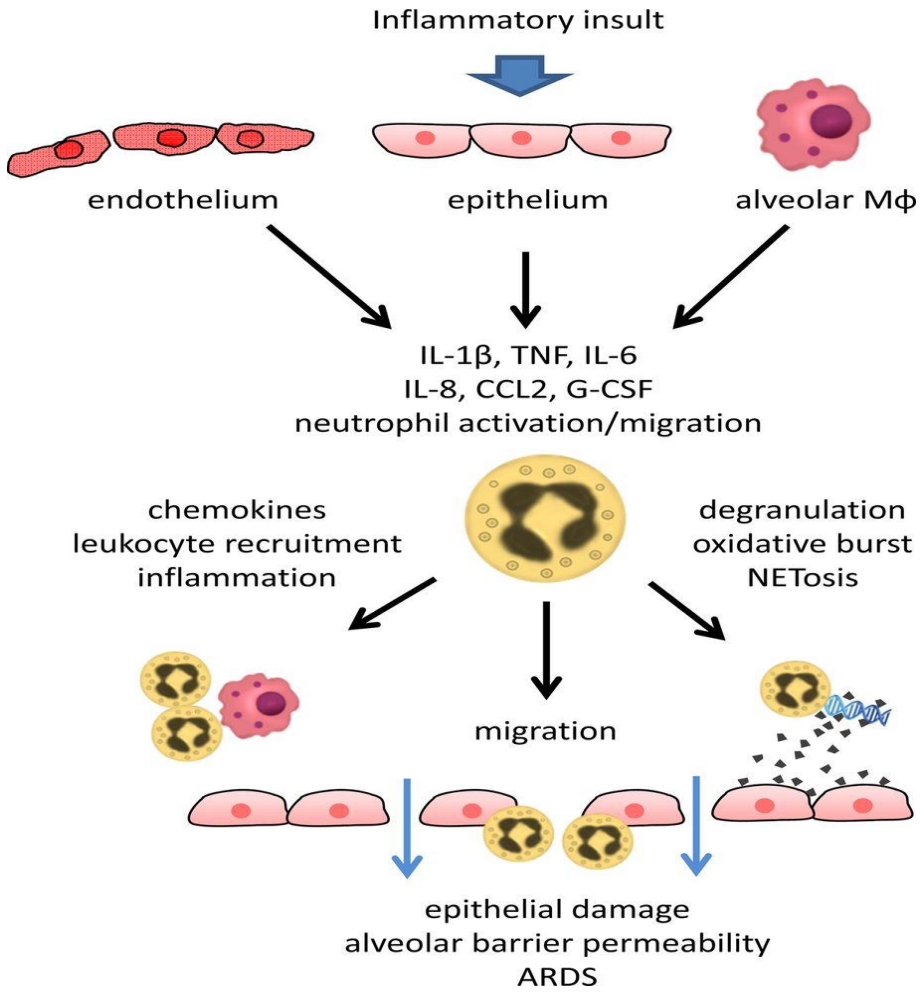
PATHOGENESIS

*IMBALANCE BETWEEN PRO AND ANTI-INFLAMMATORY MEDIATORS.

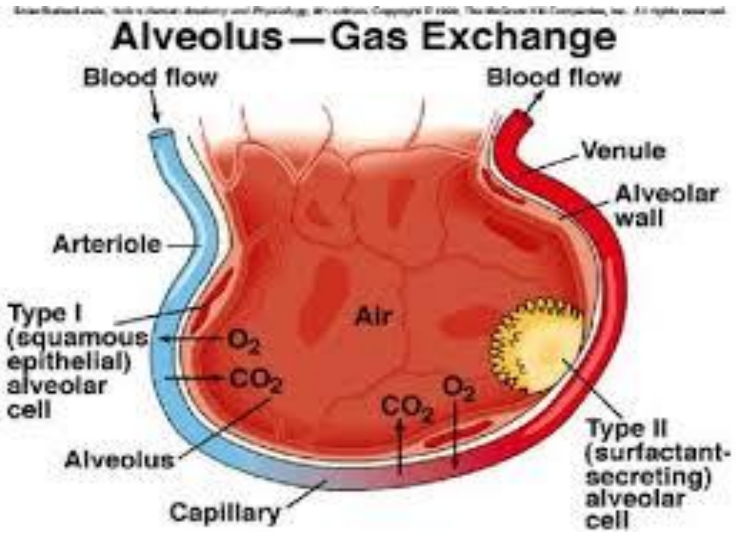
*WITHIN HALF AN HOUR OF THE INSULT: IL8.

*IL8: SYNTHESISED BY ALVEOLAR MACROPHAGES. IT CAUSES NEUTROPHIL CHEMOTAXIX AND ACTIVATION.

ARDS: PATHOGENESIS



BARRIER INTEGRETY LOST OR COMPROMISED



- IL8, IL1 &TNF CAUSE NEUTROPHIL AND ENDOTHELIAL ACTIVATION.

- ACTIVATED NUTROPHILS PRODUCE OXIDANTS, PROTEASES, PAF, LEUKOTRIENS.

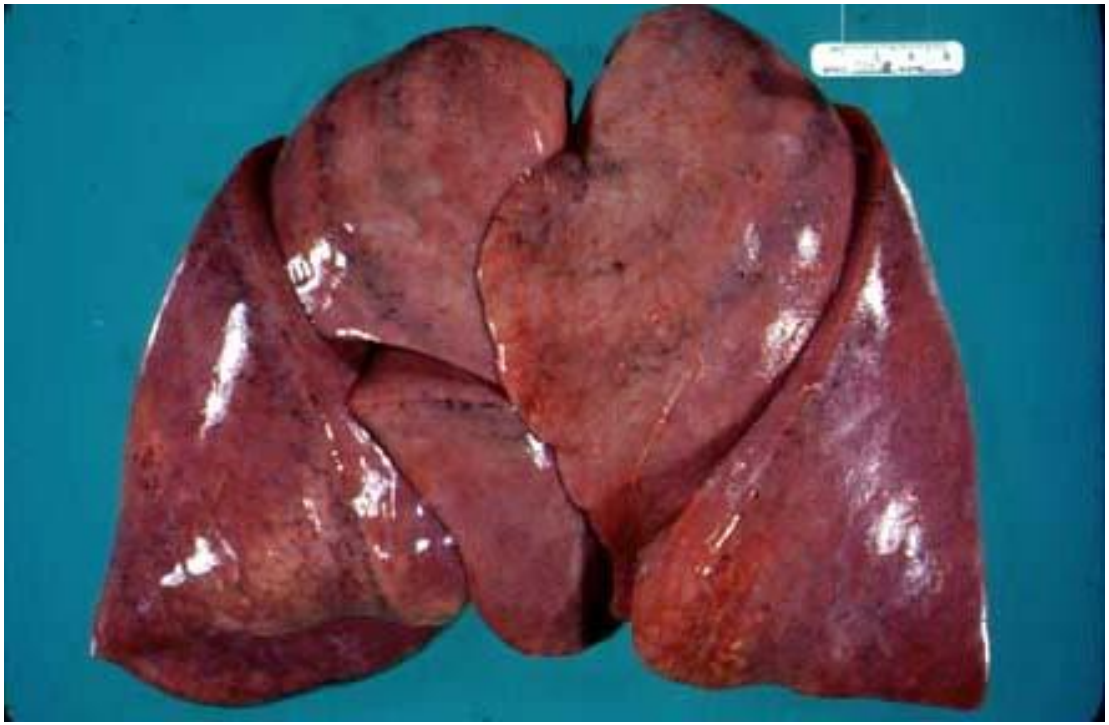
- THESE CAUSE EPITHELIAL AND ENDOTHELIAL DAMAGE .
- CAUSES IMPAIRED BARRIER AND SURFACTANT LOSS.

- ANTIPROTEASES, ANTIOXIDANTS, IL10 ….UPREGULATED BY CYTOKINES. THESE HAVE ANTIINFLAMMATORY EFFECT.

- ***THE BALANCE DETERMINES DEGREE OF INJURY.***

MORPHOLGY OF ARDS

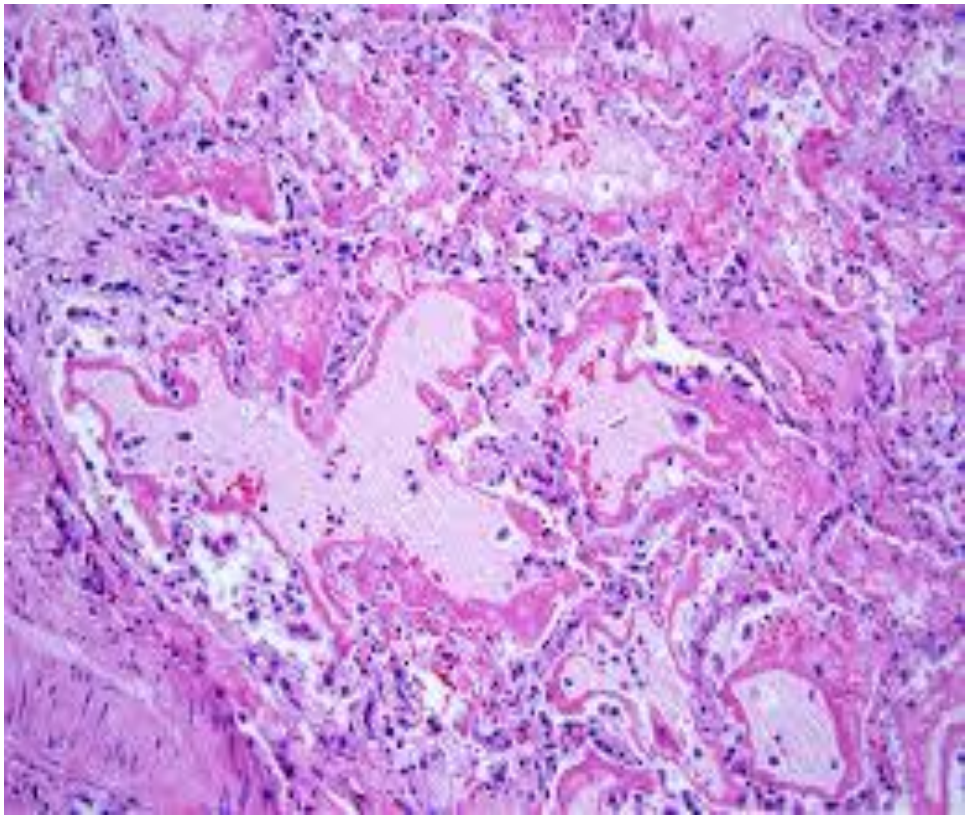
- LUNGS: RED, FIRM, AIRLESS AND HEAVY.



MORPHOLOGY OF ARDS

- CAPILLARY CONGESTION.
- NECROSIS OF ALVEOLAR EPITHELIAL CELLS.
- INTERSTITIAL EDMA.
- INTRA-ALVEOLAR EDEMA.
- HEMORRHAGE.
- NEUTROPHILS IN CAPILLARIES.
- *HYALINE MEMBRANES*.

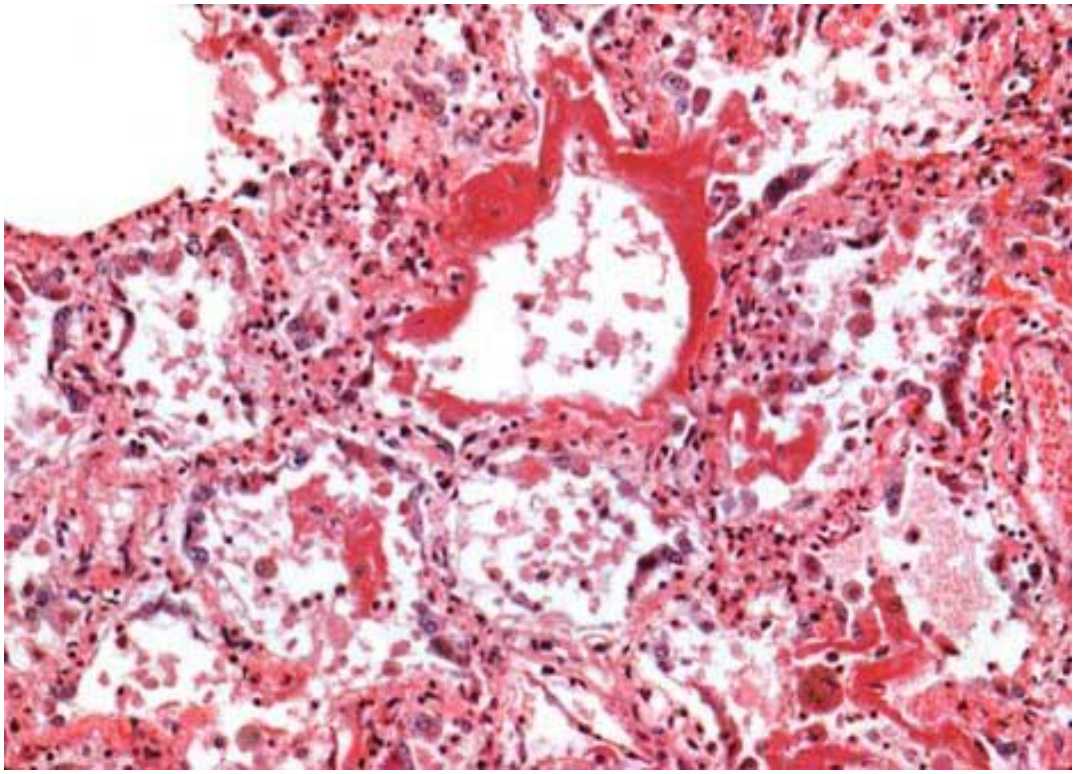
ARDS



HYALINE MEMBRANES

- LINE THE ALVEOLI.
- CONSIST OF: EDEMA RICH IN FIBRIN AND NECROTIC EPITHELIAL CELLS.

HYALINE MEMBRANES



HISTOLOGY OF ARDS

ORGANIZING STAGE:

- PROLIFERATION OF TYPE 2 PNEUMOCYTES.
- INTRA-ALVEOLAR FIBROSIS.
- THICKENING OF ALVEOLAR SEPTA DUE TO PROLIFERATION OF INTERSTITIAL CELLS AND DEPOSITION OF COLLAGEN.

CLINICAL FEATURES

- 80% DEVELOP ARDS WITHIN 72 HOURS OF THE INSULT.
- MORTALITY DECREASED FROM 60% TO 40% IN USA IN THE LAST DECADE.
- POOR PROGNOSIS:

*OLD AGE.

*SEPSIS.

*MULTISYSTEM FAILURE.

OUTCOME

- SURVIVORS END WITH DIFFUSE INTERSTITIAL FIBROSIS.
- THIS CAUSES COMPROMISE OF RESPIRATORY FUNCTION.
- SURVIVALS WHO DON’T HAVE CHRONIC CONSEQUENCES RETAIN NORMAL RESPIRATORY FUNCTION WITHIN 6-12 MONTHS.