# ACUTE RESPIRATORY RESPONSE SYNDROME(ARDS)

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# Outline

- Case discussion
- Definitions
- Pathophysiology
- Aetiology
- Treatment

# Case Discussion

- 25 year old female with facial burns. Coughing out soot stained sputum & dyspneic.
- Respiratory rate 30/ minute. Bilateral coarse crepts. $SpO_2$ 90% on $FiO_2$ 0.8
- CXR
- ABG PH 7.31

  $PO_2$ 58mmHg ($FiO_2$ 0.8)

  $PCO_2$ 26mmHg

  What is the diagnosis?

# ARDS

- First described in 1967
- Acute respiratory distress, refractory cyanosis, reduced lung compliance, diffuse infiltrates on CXR
- Initially, Adult Respiratory Distress Syndrome

# ARDS

- Incidence 75 per 100,000 population
- Mortality 40%
- Risk factors for death –

  Elderly, organ failure,

  chronic liver disease,

  failure to improve during first week of therapy

# ARDS: The New Definition

- The ARDS Definition Task Force. Acute Respiratory Distress Syndrome. The Berlin Definition. JAMA online May 21, 2012.

# What is ARDS?

- "acute diffuse, inflammatory lung injury, leading to
- increased pulmonary vascular permeability, increased lung weight,
- loss of aerated lung tissue…[with] hypoxemia and bilateral radiographic opacities, associated with
- increased venous admixture, increased physiological dead space, and decreased lung compliance."

# Definitions 1994

- Acute onset – Within 7 days of trigger event
- Severity of hypoxemia

  $Pa_{O_2}/FiO_2 < 300$
- Bilateral opacities in lung on CXR/CT
- Not due to Cardiac failure/ fluid overload

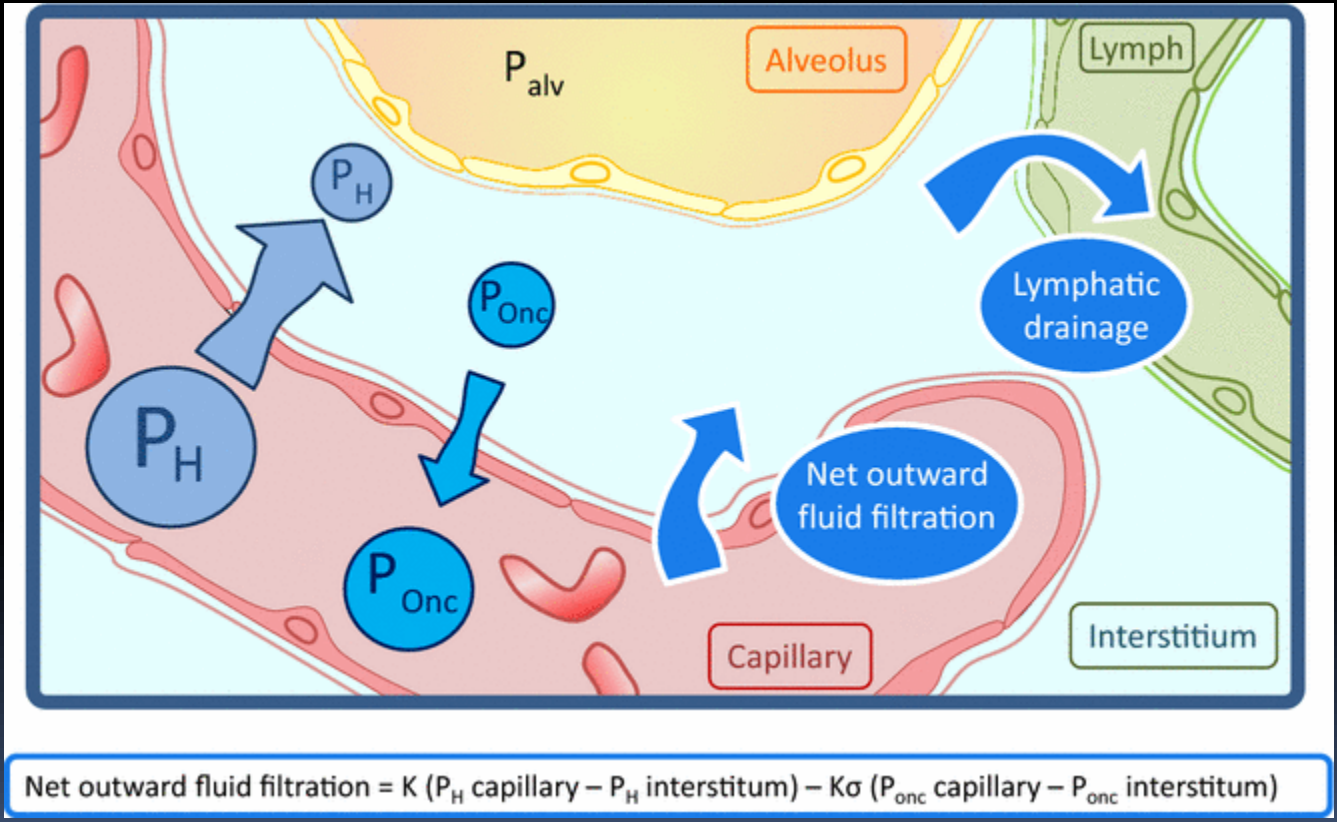
# Pathophysiology

- Increased permeability of alveolar- capillary membrane

  Disruption of the alveolar epithelium Diffuse alveolar damage

  Capillary injury
- Damage caused by neutrophils, cytokines, immune complexes, toxins, mechanical ventialtion

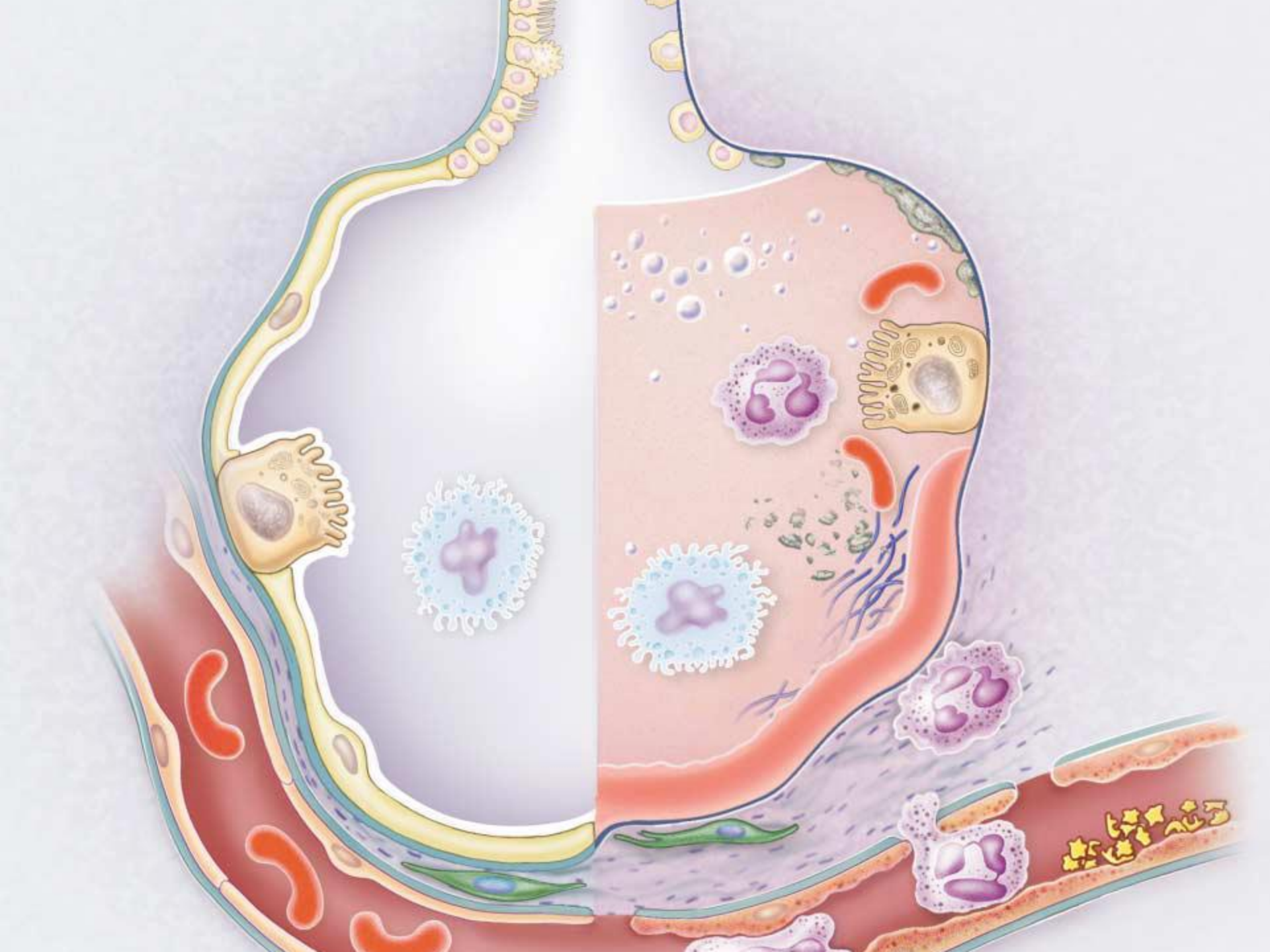
# Starlings Forces -Lung



# Pathophysiology - II

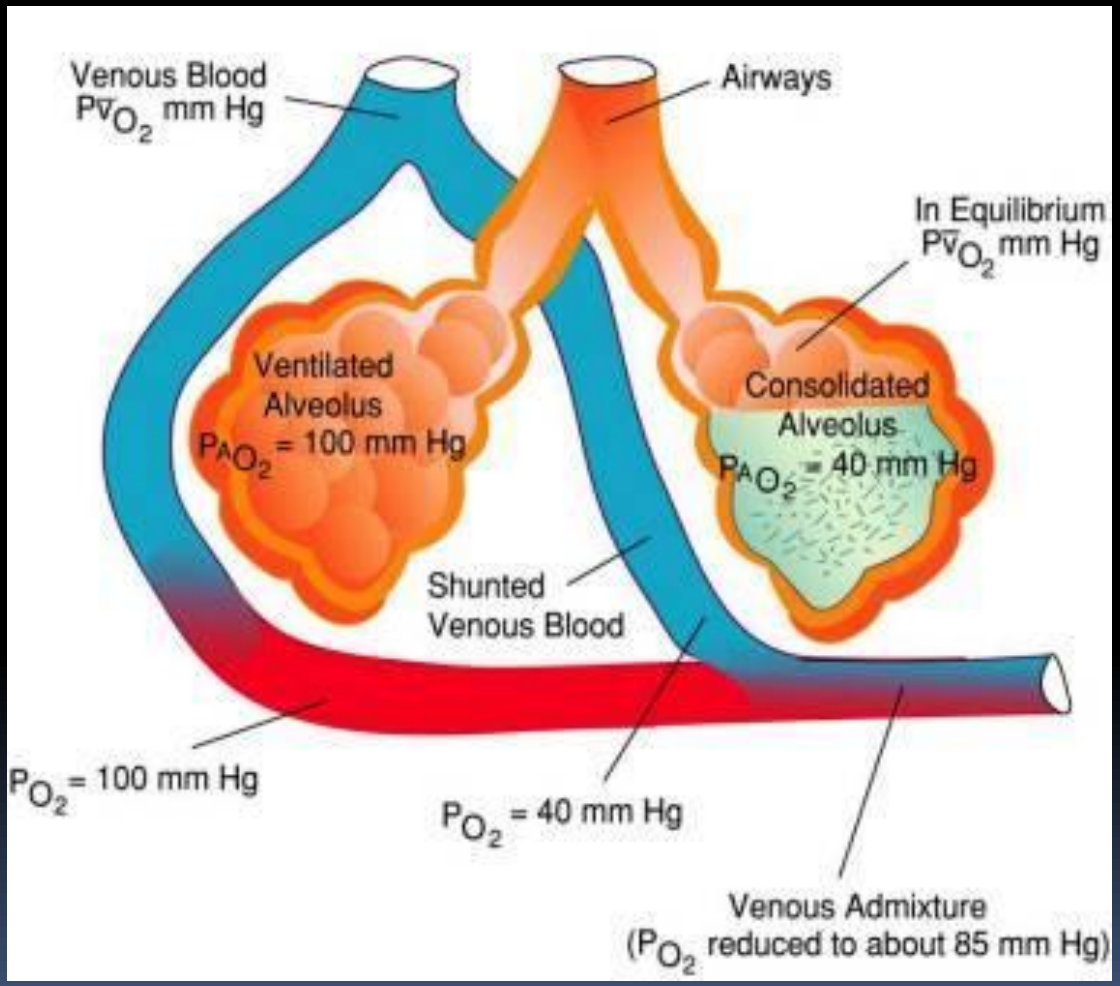
- Normal capillary hydrostatic pressures
- Damage to type II pneumocytes

  Reduced surfactant, impaired removal of edema fluid, fibrosis
- Leakage of neutrophils and protein rich edema fluid into alveoli
- Alveolar filling, atelectasis, consolidation



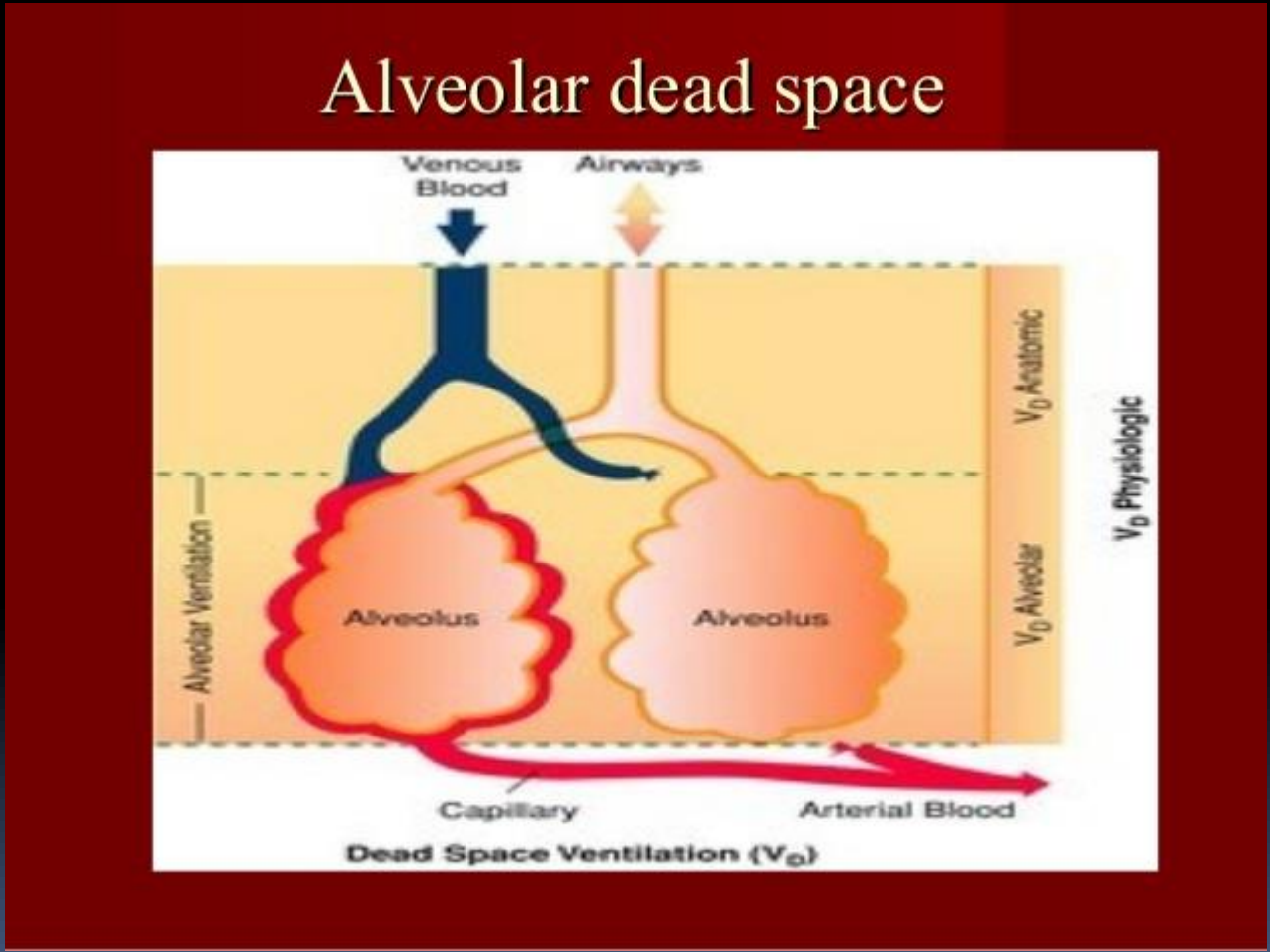
# SHUNT OF PULMONARY ARTERIAL BLOOD

# Intrapulmonary Shunt



# DEAD SPACE

# Alveolar Dead Speace



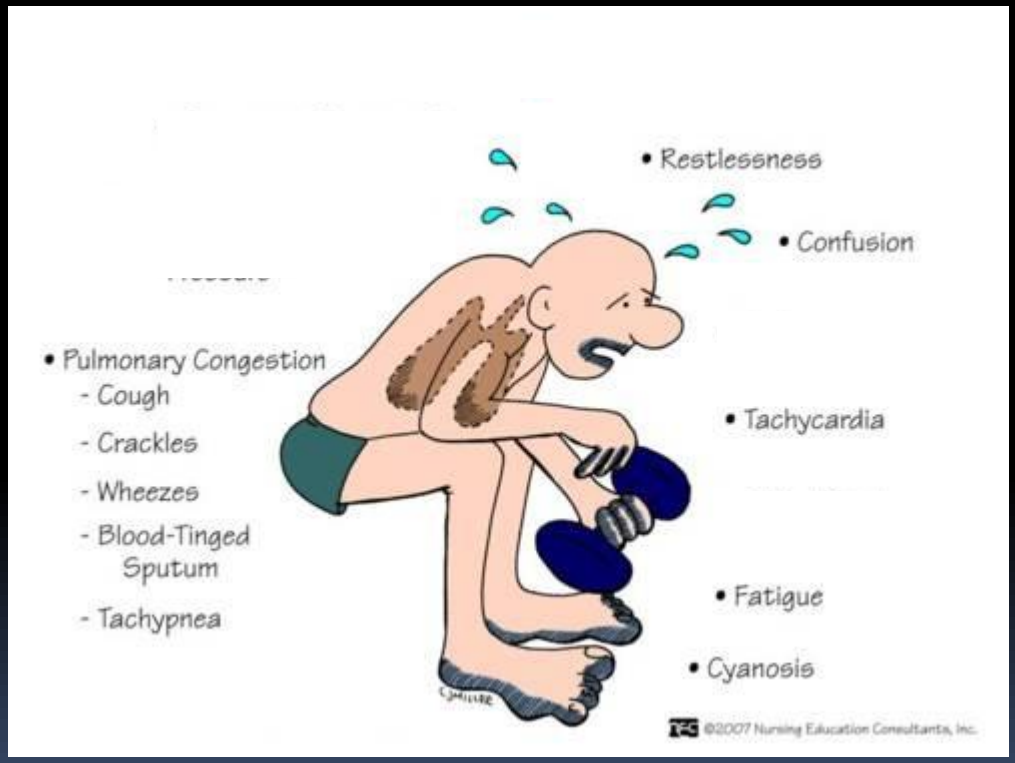
# COMPLIANCE

# Compliance of Lung

# Pathophysiology - III

- Reduced compliance of lung
- Increased work of breathing & dyspnea

# Increased Work of Breathing



# Pathophysiology - IV

- Initial phase

  Exudative,

  Refractory hypoxia

# Pathophysiology - V

- Sequalae

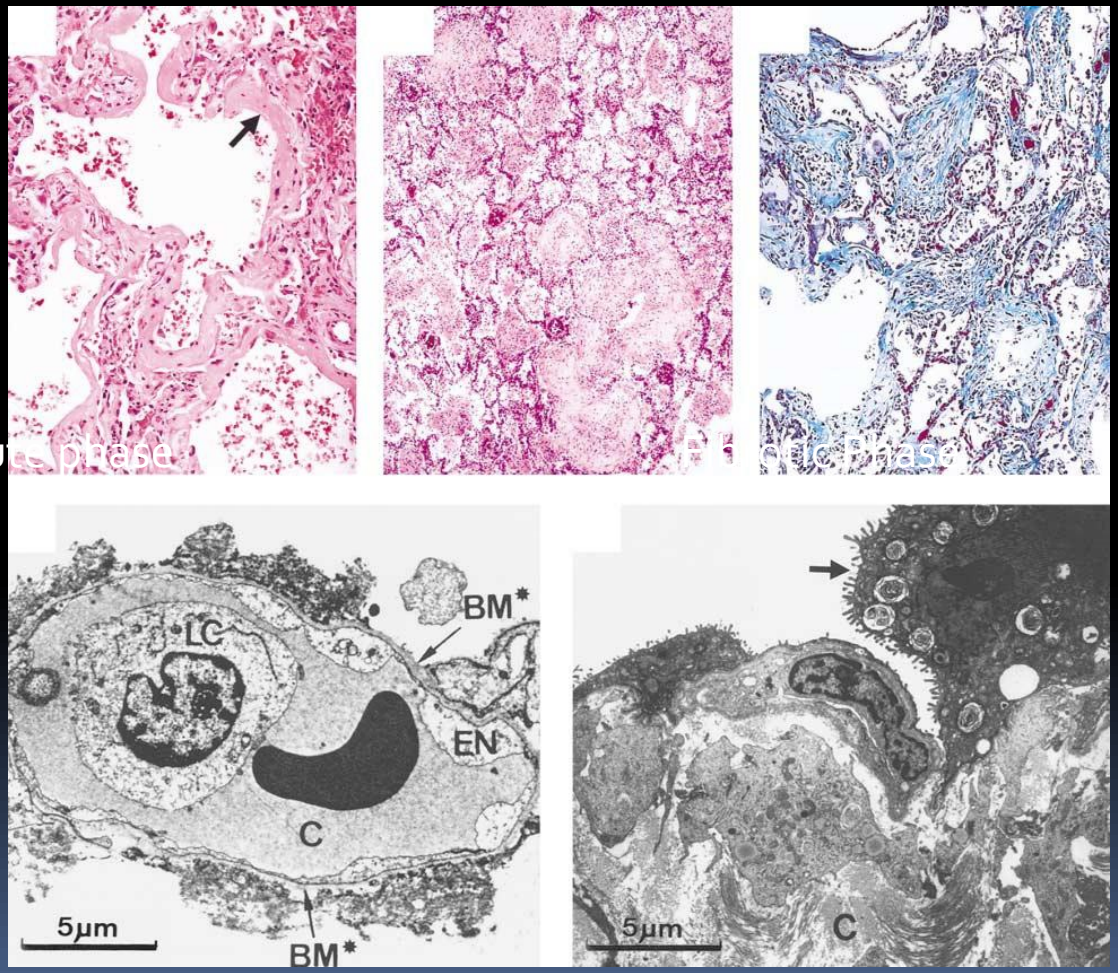
  a) Complete resolution

  b) Partial resolution with progressive fibrosis

  c) Progressive hypoxemia, multiple organ failure & death

# Resolution With Fibrosis

# Histological Changes in ARDS



# Aetiology

| Direct | Indirect |
| --- | --- |
| Inhalational injury<br>Severe infection<br>Drowning<br>Chest injury/ pulmonary contusion<br>Aspiration<br>Pneumonia | Sepsis<br>Transfusion of blood products<br>Drugs<br>Acute pancreatitis<br>Cardiopulmonary bypass |

# Investigations

- Diagnosis of ARDS
- Identify aetiology

# Treatment

- Specific therapy

  Identify & treat sepsis early

  Protective lung ventilation

  - Low tidal volumes
  - Optimal PEEP
  - Limiting airway pressures
  - Lung recruitment

# Treatment

- Ventilatory support to reverse hypoxia & hypercarbia
- General Intensive Care

  Nutrition

  Peptic ulcer prophylaxis

  DVT prophylaxis

Corticosteroids, surfactant, prostacyclin, – found to be of no value!!!!

# Summary

- ALI/ARDS causes acute severe hypoxia
- Underlying aetiology important
- Increased alveolar-capillary membrane permeability & non cardiogenic pulmonary edema
- Supportive treatment

# References


- The Acute Respiratory Distress Syndrome. L.B.Ware et.al New England Journal of Medicine (2004) 342:18;1335
- **Acute Respiratory Distress Syndrome**The Berlin Definition

  *JAMA.* 2012;307(23):2526-2533. doi:10.1001/jama.2012.5