ARDS

**Definition**
- acute hypoxic respiratory failure with PF ratio of less than 300 with bilateral pulmonary infiltrates and PaOP<18mmHg or no evidence of elevated left atrial pressure

**Aetiology**
- ALI: acute hypoxic respiratory failure with PF ratio of less than 200 with bilateral pulmonary infiltrates and PaOP<18mmHg or no evidence of elevated left atrial pressure

**Symptoms & Signs**
- First described in 1967 as acute onset of tachypnoea, hypoxia and loss of compliance after a variety of stimuli
- Chest X-ray shows appearances consistent with pulmonary oedema
- CT initially shows dependent increase in lung density initially and then more uniform inflammatory change

**Investigation**
- Early treatment of triggers of ARDS is mainstay
  - General: non-invasive ventilation where possible
    - ARDSNet study showed VT of 6ml/kg is superior to 12mls/kg (patients ventilated with AC to avoid excessive spontaneous VT)
  - Mechanical ventilation (ventilation)
    - Overstretch: normal lung is fully inflated at 30cmH2O.
    - Maximum Pplat of 30-35cmH2O is recommended to avoid overstretch; however, transpulmonary pressure may be lower than expected for a given Pplat in patients with high or low chest wall compliance
    - VT that produces unacceptably high Pplat during mechanical ventilation produces the same volutrauma during a spontaneous or supported mode of ventilation and should be avoided
  - Transpulmonary pressure can be measured by an oesophageal balloon or volume-pressure curves can be used to determine overinflation but VT limitation at 6ml/kg is most practical approach
  - Adequate PEEP: improves PaO2 by increasing functional residual capacity & recruiting alveoli but may decrease CO by impairing venous return
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  - CPAP 30-40cmH2O for 30-40s in apneic patient followed by return to controlled ventilation may lead to marked improvement in oxygenation
  - Recruitment manoeuvres: CPAP 30-40cmH2O for 30-40s in apneic patient followed by return to controlled ventilation may lead to marked improvement in oxygenation

**Prognosis**
- Mortality rates for ALI and ARDS are approximately 30%
- Respiratory function generally returns to normal in 6-12 months; some patients have persistent severe restrictive lung disease
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**Treatment**
- General: non-invasive ventilation where possible
  - ARDSNet study showed VT of 6ml/kg is superior to 12mls/kg (patients ventilated with AC to avoid excessive spontaneous VT)
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**Pathophysiology**
- ARDS involves diffuse alveolar damage with pulmonary oedema due to damage of the alveolocapillary barrier, inflammatory infiltrate & surfactant dysfunction
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**Risk Factors**
- Direct: clinical risk factors can be classified as direct and indirect
  - Most common risk factors are sepsis, pneumonia & aspiration of gastric contents
  - Multiple risk factors, acidosis, chronic alcohol abuse & chronic lung disease substantially increase risk

**Additional Measures**
- Prone posture: does not improve survival in RCT
  - May be used as rescue therapy in life-threatening hypoxia

**Manipulation of Pulmonary Circulation**
- Inhaled nitric oxide: only 40-70% of ARDS patients respond to inhaled NO
  - Improves oxygenation in short term but not survival
  - May provide temporary rescue
  - Inhaled prostacyclin: as effective as NO

**Pharmacological Therapy**
- APC in sepsis
- Surfactant: data are lacking and there are problems with distribution to alveoli
- Glucocorticoids: may be used to reduce fibrosis in alveoli based on a small study
- Ketoconazole: antifungal drug that also inhibits thromboxane synthetase and 5-lipoxygenase
- No good evidence to support use

**Carbon Dioxide Target**
- Low VT strategies will increase CO2 unless rate is increased
  - Increased rate may subject the lung to repeated tidal stretch & dynamic hyperinflation
  - Hypercapnia may cause pulmonary HTN and induce arrhythmias
  - No evidence regarding hypercapnia vs normocapnia