# ARDs

## definition

first described in 1967 as acute onset of tachypnoea, hypoxia and loss of compliance after a variety of stimuli

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

## pathophysiology

| Feature | Cause(s) |
|---|---|
| Hypoxaemia | True shunt (perfusion of non-ventilated airspaces)[28] |
| | Impaired hypoxic pulmonary vasoconstriction |
| | $\dot{V}/\dot{Q}$ mismatch is a minor component |
| ↑ Dependent densities (CT) (Collapse/consolidation) | Surfactant dysfunction → alveolar instability |
| | Exaggeration of normal compression of dependent lung due to ↑ weight (↑ lung water, inflammation) |
| ↑ Elastance (↓ Compliance) | Surfactant dysfunction (↑ specific elastance) |
| | ↓ lung volume ('baby lung') |
| | ↑ chest wall elastance |
| | Fibrosing alveolitis (late) |
| ↑ Minute volume requirement | ↑ Alveolar dead space ($V_{Dphys}/V_T$ often 0.4–0.7) |
| | ↑ $\dot{V}_{CO2}$ |
| ↑ Work of breathing | ↑ elastance |
| | ↑ minute volume requirement |
| Pulmonary hypertension | Pulmonary vasoconstriction ($T_xA_2$, endothelin) |
| | Pulmonary microvascular thrombosis |
| | Fibrosing alveolitis |
| | PEEP |

## aetiology

ARDs involves diffuse alveolar damage with pulmonary oedema due to damage of the alveolocapillary barrier, inflammatory infiltrate & surfactant dysfunction

- general
  - 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
- direct
  - pneumonia
  - aspiration of gastric contents
  - lung contusion
  - fat embolism
  - near drowning
  - inhalational injury
  - reperfusion injury
- indirect
  - non pulmonary sepsis
  - multiple trauma
  - massive transfusion
  - pancreatitis
  - cardiopulmonary bypass

## symptoms & signs

1. hypoxaemia
   - shunt
   - impaired hypoxic vasoconstriction
2. increased dependent densities on CT
   - surfactant dysfunction
   - exaggeration of normal compression of dependent lung due to increased weight of lung
3. decreased compliance
   - surfactant dysfunction
   - fibrosing alveolitis (late)
4. increased minute volume requirement
   - increased alveolar dead space
5. increased work of breathing
   - decreased compliance
6. pulmonary hypertension
   - pulmonary vasoconstriction
   - pulmonary microvascular thrombosis
   - fibrosing alveolitis
   - PEEP

## prognosis

- mortality rates for ALI and ARDs are approximately 30%
- respiratory function generally returns to normal in 6-12 months; some patients to have persistent severe restrictive lung disease

## treatment

### general

- early treatment of triggers of ARDs is mainstay

### mechanical ventilation

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)

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
- PEEP improves PaO2 by increasing functional residual capacity & recruiting alveoli but may decrease CO by impairing venous return
- in ARDs patients recruitment of collapsed alveoli occurs along entire volume-pressure curve
- best PEEP strategy is not defined

recruitment maneovres
- CPAP 30-40cmH2O for 30-40s in apneic patient followed by return to controlled ventilation may lead to marked improvement in oxygenation

mode of ventilation
- assist control is used in ARDsNET & prevents volutrauma
- PC and PRVC allow lower PPk, but probably similar Pplat
- other modes including high frequency have been used
- no clear evidence of superiority of one mode over another

target oxygenation:
- cognitive impairment is associated with SaO2 <90% therefore SaO2>90% and PaO2>60 is a reasonable target

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 arrhthymias
- no evidence regarding hypercapnia vs normocapnia

### additional measures

Prone posture:
- does not improve survival in RCT
- may be used as rescue therapy in life-threatening hypoxia

Manipulation of pulmonary circulation:
1. 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
2. inhaled prostacylcin
   - as effective as iNO

Pharmacological therapy:
1. APC in sepsis
2. surfactant
   - data are lacking and there are problems with distribution to alveoli
3. glucocorticoids
   - may be used to reduce fibrosing alveolitis based on a small study
   - definitive data are lacking
4. ketoconazole
   - antifungal drug that also inhibits thromboxane synthetase and 5-lipoxygenase
   - no good evidence to support use

## investigation

- chest X-ray shows appearances consistent with pulmonary oedema
- CT initially shows dependent increase in lung density initially and then more uniform inflammatory change