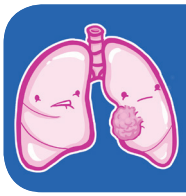

NOTES
ACUTE RESPIRATORY DISEASE

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Acute respiratory disorders induced by changes in atmospheric pressure/direct communication between atmosphere, vasculature/pulmonary conditions, diseases (e.g. pulmonary trauma, pneumonia, sepsis, severe burns)
- Impaired alveolar gas exchange → hypoxemia
- Can lead to potentially fatal conditions

SIGNS & SYMPTOMS

- *Hypoxemia:* dyspnea, tachypnea, chest pain

DIAGNOSIS

DIAGNOSTIC IMAGING
- Medical imaging

OTHER DIAGNOSTICS
- Clinical presentation, history
- Arterial blood gases

TREATMENT

OTHER INTERVENTIONS
- Oxygen therapy
- Mechanical ventilation

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

osms.it/ards

PATHOLOGY & CAUSES

- Acute lung condition
- Widespread diffuse inflammation → increased vascular permeability, loss of pulmonary tissue
- Triggered by pulmonary conditions, diseases (e.g. pulmonary trauma, pneumonia, sepsis)

PATHOLOGY
- Refractory hypoxemia, reduced pulmonary compliance, pulmonary edema without cardiovascular cause (noncardiogenic pulmonary edema)
- Alveolar barrier cells damaged → alveolar sacs flooded → impairs air exchange
  - Pro-inflammatory cytokines released: tumor necrosis factor (TNF), interleukins
  - Interleukins (IL-1, IL-6, IL-8) → neutrophil activation → toxic substances (reactive oxygen species) released → alveolar and capillary damage → oncotic gradient lost → no fluid resorption → fluid in interstitium
- Damaged Type II pneumocytes → surfactant layer malfunction

- Acute inflammatory response → abnormal extravascular fibrin deposition
  - Increased activity of extrinsic coagulation pathway
  - Impaired fibrinolysis

CAUSES

- Systemic infections/septic shock
- Acute lung injury
  - Compromises ability to regulate gas exchange → lungs fill up with fluid in interstitium, alveoli
- Gastric contents aspiration
- Severe pneumonia
- Serious burns
- Mechanical (e.g. near drowning)
- Inflammatory (e.g. pancreatitis)

SIGNS & SYMPTOMS

- Usually begin within first few hours, 1–2 days
- Dyspnea, tachypnea, tachycardia, diaphoresis, low blood oxygenation → cyanosis, diffuse crackles on lung auscultation

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-rays

- Bilateral alveolar infiltrate, pulmonary edema with no cardiovascular cause

CT scan

- Bilateral airspace opacities

Ultrasound

- Subpleural consolidations, pleural line irregularities, no lung gliding

LAB RESULTS

- Respiratory alkalosis → respiratory acidosis

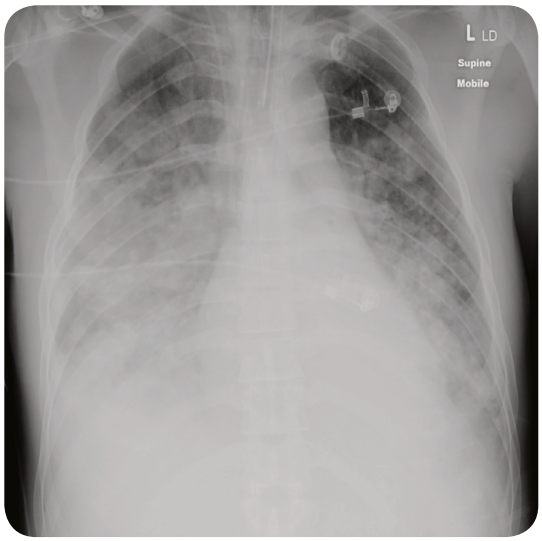

Figure 123.1 A chest radiograph demonstrating diffuse, bilateral, coalescent opacities resembling ground glass.

OTHER DIAGNOSTICS

2012 Berlin definition

- Acute pulmonary injury within week of clinical consultation
- Bilateral opacities on chest X-ray/CT scan unexplained by other pulmonary pathologies (e.g. pleural effusion, lung collapse)
- Respiratory failure without heart failure (noncardiogenic)
- Minimum positive end expiratory pressure (PEEP) of 5cmH_20
- Reduced oxygen in arteries, reduced partial pressure arterial oxygen/fraction of intake of oxygen (PaO_2/FiO_2) ratio
  - *Mild*: 201–300mmHg
  - *Moderate*: 101–200mmHg
  - *Serious*: < 100mmHg

TREATMENT

MEDICATIONS

- Antibiotic therapy
  - After microbiological culture, determines appropriate course of antibiotics
- Diuretics
  - Manage fluid output

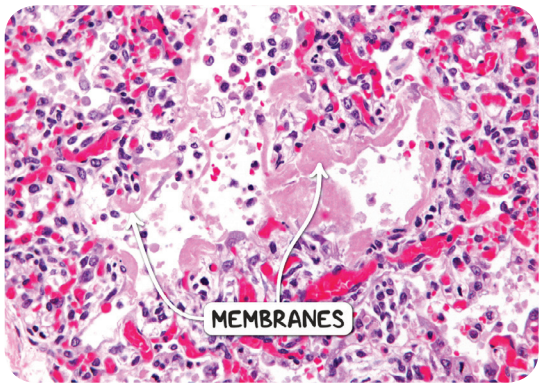

Figure 123.2 The histological appearance of diffuse alveolar damage, the pathological correlate of ARDS. There is a diffuse inflammatory cell infiltrate and pink, hyaline membranes in the alveolar spaces.

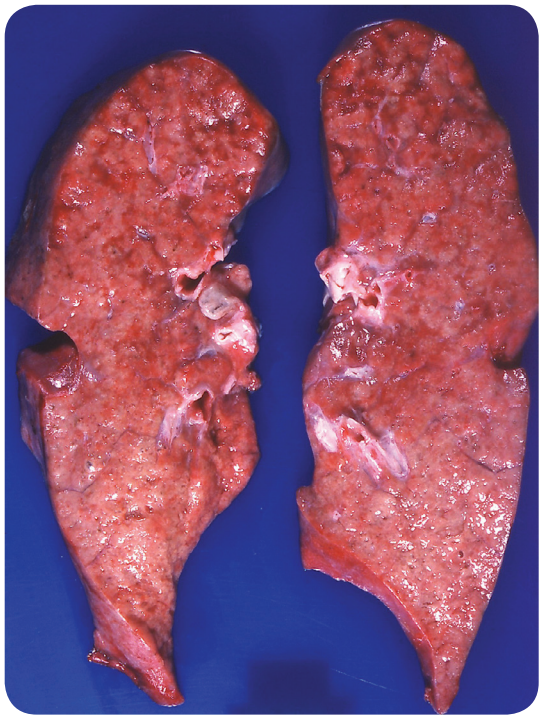

Figure 123.3 The gross pathological appearance of ARDS. There is a diffuse, vaguely nodular infiltrate, most easily visible at the apices.

OTHER INTERVENTIONS

Mechanical ventilation

- Maintain gas exchange to meet metabolic demands
- Endotracheal intubation/tracheostomy (prolonged intubations)
- Monitor parameters
  - *PEEP*: keep alveoli from collapsing, improve oxygenation
  - *Mean airway pressure*: recruit alveoli to open
  - *Plateau pressure*: monitor alveoli for overdistension
- Extracorporeal membrane oxygenation (ECMO)
  - Removes blood from body, artificially removes CO_2, oxygenates red blood cells

ALTITUDE SICKNESS

osms.it/altitude-sickness

PATHOLOGY & CAUSES

- Reaction to exposure to low oxygen concentrations when traveling to high altitude
  - AKA high altitude illness (HAI), acute mountain sickness (AMS)
- Partial pressure of oxygen of inspired air calculated by PiO_2 (mmHg) = FiO_2 (%) x [Pb (mmHg) - 47mmHg]
  - *FiO_2:* fraction of inspired oxygen, not affected by altitude, remains unchanged in 21%
  - *Pb:* barometric pressure
  - *47mmHg:* vapor pressure of water at 37°C/98.6°F
- In high altitudes, ↓ Pb → ↓ PiO_2
- Partial pressure of alveolar oxygen (PAO_2)
  - Pressure in alveolar space after equilibration with blood
- PAO_2 lower than PiO_2
  - Air enters lungs, humidified by upper airway, partial pressure of water vapor reduces partial pressure of oxygen
  - Continual uptake of oxygen from alveoli by pulmonary capillaries
  - Continual diffusion of CO_2 from capillaries into alveoli
- ↓ PiO_2 → ↓ PAO_2, ↓ PaO_2 → hypoxemia
- HAI starts at 1.5km/5,000ft, symptoms noticeable above 2.4km/8,000ft

Adaptive mechanisms

- Hypoxemia → hyperventilation → ↑ expiration of CO_2 by lungs → ↓ PCO_2 → ↑ pH (respiratory alkalosis)
- ↓ PCO_2 , ↑ pH inhibit central, peripheral chemoreceptors, decrease ventilation rate
- Within several days ↑ HCO_3^-, ↓ H^+ kidney excretion → ↓ pH → stimulation of respiratory center to further increase ventilation
- ↑ erythropoietin production → ↑ red blood cell production
- ↑ 2,3 BPG synthesis → ↓ hemoglobin affinity for O_2 → ↑ release of oxygen to tissues

Measures to avoid HAI

- *Acclimatization:* ascending slowly to high altitudes, to adjust to decreasing oxygen levels
- *Preventative medications:* acetazolamide (diuretic); increases bicarbonate kidney excretion

RISK FACTORS

- History of HAI episodes
- Prior exercise/alcohol consumption
- Rapid ascent to high altitude
- Comorbidities that affect breathing (e.g. asthma)

COMPLICATIONS

- Can lead to potentially fatal conditions
  - High altitude cerebral edema (HACE), high altitude pulmonary edema (HAPE)

SIGNS & SYMPTOMS

- Usually appear within 6–12 hours of ascent
- Headache, dizziness, fatigue, nausea, vomiting, loss of appetite, sleep disturbance
- Often improves with time if person does not ascend to higher altitude
- HACE
  - Excessive fatigue, confusion, neurologic deficits (e.g. ataxia, altered mental state)
- HAPE
  - Dry cough, dyspnea

DIAGNOSIS

LAB RESULTS

- Arterial blood gases
  - ↓ PaO_2, ↑ $PaCO_2$, respiratory alkalosis

OTHER DIAGNOSTICS

- Clinical presentation, history of living at low altitude, recent ascent at high altitude

TREATMENT

MEDICATIONS

- Symptom relief
  - E.g. analgesics for headache, antiemetics for nausea
- Carbonic anhydrase inhibitors (e.g. acetazolamide)
  - Increase HCO_3^- excretion; treat respiratory alkalosis

OTHER INTERVENIONS

- Rest
- Descent
- Symptom relief
  - E.g. oxygen to improve breathing
- HACE, HAPE
  - Medical emergencies; require immediate descent/oxygen administration

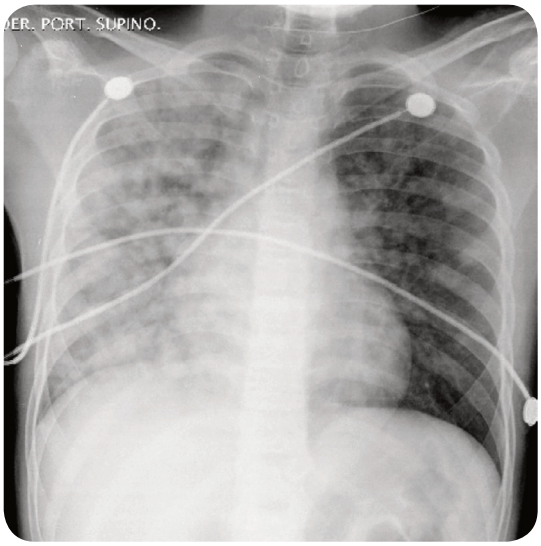

Figure 123.4 A chest radiograph demonstrating acute pulmonary edema in an individual who ascended to 2700m.

DECOMPRESSION SICKNESS (DCS)

osms.it/decompression_sickness

PATHOLOGY & CAUSES

- Gas embolism, occurs when individuals experience sudden decreases in atmospheric pressure
  - AKA diver's disease
- Air breathed at relatively high pressure (e.g. diver descends from water surface) → inspired gases compressed to higher pressure of surrounding water → ↑ partial pressure of oxygen, nitrogen → ↑ oxygen, nitrogen dissolved in blood, loaded in body tissues
  - *Henry's law:* at constant temperature, amount of gas dissolved in liquid directly proportional to partial pressure
- If oxygen, nitrogen quantities high enough → oxygen toxicity/nitrogen narcosis, respectively

- Pressure drops too rapidly (e.g. ascent to water surface) → sum of gas tensions in tissue exceeds ambient pressure → liberation of free gas from tissues due to excess dissolved gases → gas bubbles → vessels blocked, tissues compressed, clotting cascade, inflammation
- Occurs in scuba, deep sea divers, underwater construction workers; during rapid ascent of an unpressurized aircraft
- Caisson disease (chronic decompression sickness)
  - Tunnel workers, moving from caisson to atmospheric pressure

RISK FACTORS

- Right-to-left shunt (e.g. patent foramen ovale/atrial/ventricular septal defect)
- Air travel after diving
- More common in individuals who are biologically male

SIGNS & SYMPTOMS

- Usually develop within one hour of surfacing
- Excessive fatigue, headache
- Depend upon size, location of gas bubbles

Type I DCS

- Skeletal muscles, joints
  - Painful condition, AKA "the bends"; arching of back, posture reminiscent of Grecian bend
- Skin
  - Itching, rash

Type II DCS (more severe)

- Nervous system
  - Paresthesia, amnesia, weakness, paralysis
- Lungs
  - Edema, hemorrhage, atelectasis, emphysema → respiratory distress, AKA "the chokes"; cough, chest pain, dyspnea
- Can progress to permanent injuries/fatal damage

DIAGNOSIS

OTHER DIAGNOSTICS

- Clinical presentation, history of exposure to sudden decreases in atmospheric pressure
- Confirmed if symptoms relieved after recompression

TREATMENT

OTHER INTERVENTIONS

- Hyperbaric oxygen therapy in recompression chamber
  - Under high pressure gas bubbles forced back into solution; slow decompression permits gradual gas elimination via lungs, prevents obstructive bubbles reforming

KEY COMPONENTS OF ACUTE RESPIRATORY DISEASE

	PATHOLOGY/ CAUSES	SIGNS & SYMPTOMS	TREATMENT
ARDS	Inflammation → lung injury → ↑ fluid in alveoli and interstitium →↑ PAP, ↓ compliance, ↓ gas exchange → hypoxemia	Rapidly progressing respiratory distress: dyspnea, tachypnea, tachycardia, use of accessory respiratory muscles, diaphoresis, cyanosis, respiratory crackles	Mechanical ventilation, fluid management
ALTITUDE SICKNESS	Decreased PiO_2 → hypoxemia Severe cases → HACE, HAPE	Within 6-12 hours of ascent: headache, dizziness, fatigue, nausea, sleep disturbance Severe: excess fatigue, neurological (confusion, deficit), pulmonary (dry cough, dyspnea)	Rest, descent, O_2, acetazolamide Severe: rapid descent
DECOMPRESSION SICKNESS	Liberation of free gases in tissues; bubbles block vessels, compress tissue, activate clotting, inflammation Etiology: rapid ascent	Type I DCS: muscle/joint pain, itching Type II DCS: neurologic (paresthesia, amnesia, weakness), pulmonary (edema, hemorrhage, atelectasis, emphysema)	Hyperbaric O_2 therapy