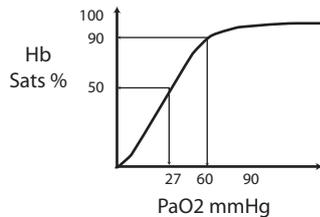


Hypoxaemia is defined as a PO₂ in arterial blood below normal physiological levels.

Different textbooks use different values although a value of less than 60mmHg is useful as this represents the beginning of the steep part of the Hb-O₂ curve and corresponds to a saturation of approximately 90%.

Hb-O₂
Dissociation Curve



Physiological causes of acute hypoxaemia are

- Decreased alveolar oxygen
- Decreased ventilation
- Shunt
- V/Q mismatch
- Diffusion limitation
- Consumption of O₂

$$PAO_2 = FiO_2(P_{atm} - 47) - PCO_2/0.8$$

$$Vent = RR(\text{tidal volume} - \text{dead space})$$

$$V/Q = 0$$

$$V/Q > 0 \text{ but not } 1$$

Ficks law

$$\text{Increased extraction ratio } 100(1 - MV O_2 \text{ cont}/Art O_2 \text{ Cont})$$

Acid base changes

- initially - respiratory compensation
- later - at cellular level

When PaO₂ drops below 60mmHg

- Peripheral chemoreceptors in the carotid bodies and aortic arch sense decreased paO₂
- Central respiratory centre in the medulla rapidly increases ventilation (within 10 mins)
- As a result there may be an improvement in paO₂ (cause dependent)
- pCO₂ decreases due to increased ventilation leading to respiratory alkalosis (pH > 7.45)

At a tissue mitochondrial level

- When the partial pressure reaching the mitochondria drops below 5mmHg
- Oxidative phosphorylation is impaired
- Anaerobic pathways of energy production utilised producing lactate and hydrogen ions
- Cause an increased anion gap metabolic acidosis due to lactate (pH < 7.35)

Ventilation increase augmented to compensate for the metabolic acidosis and CO₂ drops further

HCO₃ levels drop as it buffers the acidosis, renal compensation to retain more HCO₃

Increased hydrogen ion levels shift the HbO₂ curve right enabling improved O₂ delivery at the tissues

Eventually the brain becomes hypoxaemic and respiratory drive is depressed, thereby removing respiratory compensation and resulting in increasing acidosis, failure of the Na.K.ATPase pumps in most cells, cell lysis and death.