FRCA Podcast

Episode 1 - Smoke Inhalation Injury

Injury to the respiratory tract

Thermal	Upper respiratory tract (above glottis) Swelling and obstruction.
Physical	Bronchi and bronchioles Particulate matter blocking airways (V/Q mismatch).
Chemical	Bronchioles and alveoli Acids, alkalis and organic compounds (aldehydes, sulphur dioxide) - products of combustion. Damage to epithelium. Impaired gas transfer (V/Q mismatch). Nitric oxide (inhaled) vasodilation (V/Q mismatch).

Systemic toxicity

Carbon monoxide	250 times more affinity than oxygen for haemoglobin Replaces oxygen at binding sites (oxygen carrying capacity). Shifts oxygen dissociation curve to the left (oxygen release). Normal levels: < 2% for non-smokers, 12% for smokers. > 30% is considered as carbon monoxide poisoning. Organ dysfunction due to hypoxia (heart, brain). Pulse oximetry reads 99%. Blood gas co-oximetry gives true value. Treatment with 100% oxygen reduces the half-life of carbon monoxide from several hours to 90 minutes. Competitive, reversible inhibition of Cytochrome C oxidase (terminal enzyme of the mitochondrial electron transport chain).

Cyanide	Non-competitive inhibitor of Cytochrome C oxidase. Functionally irreversible (high affinity) without treatment. Signs of extreme tissue hypoxia (organ dysfunction, lactate) but with a paradoxically *reduced* arterio-venous oxygen gradient. Paired arterial and mixed venous blood gases resemble each other (PaO_2 and PvO_2, SaO_2 and SvO_2). Consider things in terms of blood oxygen content. Venous blood will appear "arterialised". Absolute values may not be physiological given the clinical context (supplemental oxygen, presence of carbon monoxide) but the relative values will indicate a failure to utilise oxygen (in contrast to a problem of oxygen delivery). Management with Hydroxycobalamin 5 g. Binding to cyanide to form cyanocobalamin.

Normal physiological arterial and mixed venous oxygen gradients

	Saturation	Oxygen Content
Arterial	SaO_2 99%	CaO_2 = 20 ml/dL
Mixed venous	SvO_2 70%	CvO_2 = 15 ml/dL

Note: central venous saturations are often slightly higher, i.e. SvO_2 75% (sampled from CVC).

Summary

Thus, smoke inhalation injury comes down to five pathophysiological mechanisms: upper airway obstruction (swelling), lower respiratory tract ventilation (particulate matter), alveolar gas transfer and pulmonary perfusion (V/Q mismatch), oxygen delivery (carbon monoxide) and oxygen utilisation (cyanide).