Describe the physiological consequences of breathing 100% oxygen at sea level.

(1):
\[ \text{PaO}_2 = (\text{P ATM} - \text{SVP of H}_2\text{O}) \times \text{FiO}_2 - (\text{PA CO}_2 \times \text{RQ}) \]
\[ \text{PaO}_2 = 100\text{mmHg at FiO}_2 \text{ 21\%} \]

(2):
\[ \text{cO}_2 = (\text{PO}_2 \times \text{SatO}_2 \times [\text{Hb}] \times 1.34) + (0.023 \times \text{PO}_2) \]

(3):
\[ \text{SaO}_2 97.5\% \text{ at PA O}_2 100\text{mmHg} \]
\[ \text{SaO}_2 98.8\% \text{ at PA O}_2 150\text{mmHg} \]
(probably best to quickly sketch out this graph, but I couldn’t be bothered)

Therefore, owing to equation (2) and the haemoglobin dissociation curve, at \( \text{PaO}_2 > 100\text{mmHg} \) there is:
- Only a minimal increase in haemoglobin saturation
- Only a minimal increase in dissolved oxygen concentration

**Oxygen stores**

Functional residual capacity = lung volume with no active insp. or exp. effort
= volume when equilibrium between chest wall recoil and lung elastic recoil

If FiO\(_2\) increased -> more oxygen in FRC (not exhaled with tidal volume)

⇒ Able to tolerate a period of hyperventilation

**Absorption atelectasis**

Nitrogen is an inert gas, comprising ~78% of room air
As it is biologically inert, it is in equilibrium throughout body tissues

- There is no concentration gradient to cause absorption from the lungs

If FiO\(_2\) increased
- \( \text{P}_1\text{O}_2 \) of O\(_2\) increases
- \( \text{P}_\text{aN}_2 \) decreases as a result
- Moves out of alveoli down its concentration gradient
  - Total alveolar pressure falls -> alveolar collapse

Clinically significant when FiO\(_2\) > 50%

**Hypoxic vasoconstriction**
In under ventilated lungs portions, there is adaptive pulmonary vasoconstriction, reducing V/Q mismatching.

If \( F_iO_2 \) increased:

- Under ventilated lung portions have a higher \( P_aO_2 \)
  - V/Q mismatching \( \rightarrow \) venous admixture, \( \downarrow \) pulmonary vein \( P_AO_2 \)

**Haldane effect**

Deoxygenated haemoglobin has a higher affinity for \( CO_2 \) than oxygenated Hb.

If \( SaO_2 \) is artificially raised, lower \( CO_2 \) carrying capacity \( \rightarrow \) build-up of \( CO_2 \) in tissues

**Oxygen radicals**

A radical is a molecule with an unpaired electron outside of an electron shell.

- Highly reactive, cause tissue damage

Important reactive oxygen species (ROS): Superoxide radical \( (O_2^-)\); peroxide radical \( (O_2^{2-})\); hydroxide radical \( (\cdot OH)\)

ROS can usually be eliminated by cellular antioxidant defence mechanisms.

When \( F_iO_2 > 50\% \)

- Defense mechanisms overwhelmed
- Parenchymal injury
  - Tracheobronchitis
  - Pulmonary capillary endothelial damage \( \rightarrow \) pulmonary oedema + fibrosis

Examiners Commets

The question related to physiological changes occurring when \( FiO2=1 \). Many candidates focused on the toxic effects of oxygen, which were often incorrect (CNS symptoms will not occur at one atmosphere). Candidates simply lacked knowledge, those that did have some understanding failed to provide adequate detail (ie. it was occasionally mentioned oxygen stores are increased but not the mechanism by which or extent to which stores are increased). In addition, it was expected that candidates would outline and describe the mechanism behind the changes in \( PaO2\) in arterial and mixed venous blood, shift in \( CO2 \) ventilation, hypoxic pulmonary vasoconstriction as well as pulmonary toxic effects.

6 (60\%) of candidates passed