Q1 Describe the physiological consequences of breathing 100% oxygen at sea level. (May 2011)

CHANGES TO BODY OXYGEN LEVELS

PIO2 = 760mmHg (atmospheric pressure) x 1.0 = 760mmHg

Inspired air is warmed and moistened. At body temperature (37), saturated water vapour pressure is 47mmHg.
Hence PO2 = (760-47) x 1.0 = 713mmHg

Alveolar PO2 is influenced by oxygen consumption and alveolar ventilation.
PAO2 = PIO2 – (PCO2/0.8) + F
If PACO2 = 40, then PAO2 = 713 – 50 + 2 = 665mmHg

Due to physiological shunt, arterial PO2 will be approx. 660

The amount of oxygen delivered to the capillary bed depends on the cardiac output, arterial oxygen saturation and haemoglobin.

CaO2 = (1.34 x Hb x sats) + (0.003 x PaO2)
= (1.34 x 15 x 100%) +(0.003 x 660) = 22.1ml oxygen/100ml blood

At FiO2 0.21 and other parameters unchanged, CaO2 = 21.15ml/100ml blood. Hence breathing 100% oxygen does not increase the oxygen content of blood significantly.

CHANGES TO OXYGEN STORES

FRC (volume ~2.4L) will fill with oxygen, increasing stores from 500ml to 2.4L

Blood stores of oxygen will increase marginally (1105ml/hour from 1050ml/hr)

ADVERSE EFFECTS

- TOXICITY → pulmonary toxicity can occur with diminished vital capacity by 500-800ml, probably due to absorption atelectasis. CNS toxicity will not occur at one atmosphere.
- ABSOPRTION ATELECTASIS → occurs when an airway is blocked by mucous or similar; high partial pressure of oxygen within the alveolus causes it to flow down its concentration gradient into the blood and the alveolus collapses → V/Q mismatch
- LOSS OF HYPOXIC PULONARY VASOCONSTRICTION → can lead to worsening V/Q mismatching
- IN PREMATURE INFANTS → retrolental fibroplasia due to vasoconstriction of retrorbital vessels, leading to blindness