Q19 Describe the types of dead space in the respiratory system (50% marks). Explain the consequences of increased dead space on gas exchange (50% mark) (March 2010)

Dead space – that component of the tidal volume which does not take part in gas exchange.

TYPES OF DEAD SPACE

- Apparatus → dead space associated with synthetic breathing apparatus such as ETT and ventilator tubing

- Anatomical → that volume of the conducting airways which does not take part in gas exchange (usually 2ml/kg or often estimated at 150ml). Anatomical DS can be measured using the modified Fowler's method (inspiration of a normal tidal volume breath of 100% oxygen, then expiration against a spirometer and rapid gas analyser – the initial part of exhalation will be 100% oxygen which filled the conducting airways. As alveolar gas is expired, the % of measured CO2 (or N2) will rise and is graphed. A line is drawn such that Va = Vb and that line corresponds to the dead space volume.

- Alveolar → that volume of gas reaching the alveoli which does not take part in gas exchange due to inadequate perfusion of those alveoli (this value may be significant in PE, low cardiac output state, or in the patient on PPV or who is hypotensive, in whom alveolar pressure in West's Zone 1 is greater than pulmonary arterial pressure, compressing the vessels to produce dead space)

- Physiological → the entire component of the tidal volume which does not eliminate CO2 (ie, anatomical + alveolar; in fit healthy people the physiological and anatomical values will be very similar). Physiological dead space can be measured using Bohr's equation $VD/VT = (PACO_2-PECO_2) / PACO_2$ which utilizes the measurable parameters of tidal volume, PaCO2 and the pCO2 of mixed expired gas.

CONSEQUENCES OF INCREASED DEAD SPACE

- Increase in PaCO2 as blood passing by unventilated alveoli does not have the chance to eliminate CO2
- The rise in PaCO2 activates central chemoreceptors, triggering a rise in minute ventilation to compensate (increase in resp rate +/- V_T)
- This results in an increase in work of breathing
- High PaCO2 → respiratory acidosis, sympathetic stimulation
- Eventually hypercapnoea will cause hypoxaemia (as per the alveolar gas equation, PAO2 = PIO2 –(PCO2/R) + F if the PCO2 becomes dramatically high, this will reduce the PAO2 and cause hypoxaemia.
- High PaCO2 + hypoxia → cardiac arrhythmias, sedation.