**Q21 Describe the physiological consequences of a progressive rise in blood carbon dioxide levels (Sept 2012)**

Normal $PaCO_2$ = 40mmHg
$PaCO_2$ will be a balance between $CO_2$ input (production, via aerobic metabolism, and inhalation, if rebreathing) and $CO_2$ excretion

Effects of a rise in $PaCO_2$:

RESPIRATORY

- ↑$PaCO_2$ stimulates the central chemoreceptors, which lie on the ventral surface of the medulla and respond to changes in $PaCO_2$ sensed as a change in the pH of the surrounding ECF
- $PaCO_2$ is the most important minute-to-minute controller of respiration; for every 1mmHg change in $PaCO_2$, minute ventilation changes by 2-3L/min. this effect is enhanced in the presence of hypoxia.
- Peripheral chemoreceptors in the carotid and aortic bodies also sense $PaCO_2$, and contribute to the early phase of the response
- ↑$PaCO_2$ will cause acidosis, which is also sensed by the carotid body chemoreceptors, and contributes further to the rise in minute ventilation
- ↑$PaCO_2$ → ↑$PACO_2$, which results in ↓$PAO_2$ (as per the alveolar gas equation) → hypoxia sensed by peripheral chemoreceptors causing a further rise in ventilation
- Shift of the oxygen/Hb dissociation curve to the right, increasing oxygen offloading at the tissues
- ↑PVR → V/Q mismatch

CNS

- $PaCO_2$ is the most important controller of CBF (a linear relationship exists between the two)
- ↑ CBF→ ↑ ICP
- ↑ $PaCO_2$ → ↓pH → cellular dysfunction (clinically this is often seen as a reduction in LOC)

ANS

- Catecholamine release due to hypercapnoea and hypoxia

CVS

- ↑$PaCO_2$ causes ↓ heart rate / contractility, however this may be overridden by catecholamine release
- ↑ afterload due to ↑PVR due to alveolar hypoxia

RENAL

- ↑$PaCO_2$ (severe) can cause anuria
- Renal retention of $HCO_3^-$ for buffering