Second 2008
VIVA 2

What is normal cerebral blood flow?

This viva explored the candidates' knowledge in relation to the following points
Cerebral Blood Flow, it's measurement and factors that alter it
Cerebral Blood Flow and acute changes in altitude
Morphine, typical dose, CNS effects and metabolites

Examination feedback: There was good understanding of cerebral blood flow, and most candidates used figures to good effect during the viva. Areas of weakness were an understanding of the methods of measuring CBF and differential flow to the white and gray matter. Candidates also were less confident with the different CNS effects of morphine. Adapting factual knowledge to define what would happen in states of altered physiology was also handled poorly.

"Describe cerebral blood flow"

is 750ml minute (15% CO)
O2 consumption is 3ml/100g which given a weight of 1400g is about 50ml (20%)
it is determined by the Cerebral Perfusion Pressure / Cerebrovascular Resistance
CPP is calculated by MAP - CVP, or in pathological states MAP - ICP (starling resistor model)

"How is CBF measured?"

measurement is via the Fick principle using the Kety-Schmidt method
fick principle states that the amount taken up by the organ/[arterial]-[venous] = flow
K-S used NO (highly inert and diffusable) and measured uptake/artial-venous conc diff

"What factors alter CBF?"

intrinsically it demonstrates autoregulation
primarily myogenic stretch factors
local metabolic control is more specific to regional distribution rather than total flow
extrinsically
hypoxia results in increased flows when it drops below 50mmHg
raised CO2 shows a near linear increase in blood flow 20mmHg to 80mmHg

"What happens to Cerebral Blood flow at altitude?"

as the pCO2 drops there is decreased blood flow, however when the O2 drops below 50mmHg then flow will increase

"Please discuss the pharmacology of Morphine"

naturally occuring phenanthrene derivative from the papver somniferum plant
available as liquid for injection or oral formulations
action is via GPCR mu opiod receptors modulating pain responses, in particular slow pain
side effects include dysphoria or euphoria, confusion, depressed resp drive, decreased gut motility

A bioavailability is 30% orally due to high first pass metab
D Vd 3-4l/kg, protien binding 30%, low lipid solubility rel to other opiates, pKa 8 (25% ionised)
M metabolised hepatically via gluconoridation, active metabolite M6G, may accumulate in CRF
E half life 2-4 hours, excretion in urine and faeces

"How does morphine affect cerebral blood flow"

evidence is mixed
ultimately it is dependent on how the morphine affects the CPP (MAP-CVP or ICP) and CVR
it is possible that there is a blunting of autoregulation, which if associated with a drop in CPP may lead to ischaemia
this may be important in patients with intracranial pathology