Q11 Describe the control of cerebral blood flow (March 2009)

- At rest the brain receives ~ 750ml of blood flow, approximately 15% of total cardiac output.
- Its oxygen consumption is 3-3.5 ml O2 /min/100g, or ~50ml of oxygen/minute (assuming weight of brain 1300-1500g). This is 20% of total body oxygen consumption.
- Cerebral blood flow (CBF) = cerebral perfusion pressure (CPP) / cerebrovascular resistance (CVR). CPP = MAP - CVP, however, when ICP rises above CVP, a starling resistor model is set up, and then CPP = MAP - ICP.

MAIN FACTORS CONTROLLING CBF:

- Autoregulation → the cerebral circulation demonstrates autoregulation, normally within the range of MAP 50-150mmHg (this can alter in certain situations such as chronic hypertension). The mechanism of autoregulation is not entirely understood, however it is believed to be mainly due to pressure autoregulation (the myogenic stretch theory). Metabolic autoregulation is also present (adenosine, NO, pH) but is more significant in regards to regional blood flow.

- Arterial PCO2 → demonstrates an almost linear relationship with CBF between 20mmHg-80mmHg

- Arterial PO2 → CBF increases when PaO2 drops to 50mmHg. Above this, oxygen saturation has little effect.

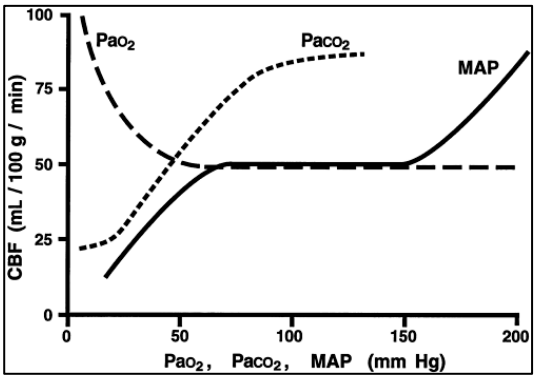

Diagram http://web.squ.edu.om/med-Lib/MED_CD/E_CDs/anesthesia/site/content/v02/020608r00.HTM

OTHER FACTORS THAT CONTRIBUTE TO CBF:

- Neurohumeral control → plays only a small role in the control of CBF
- Intracranial pressure →
  - As per the Monroe-Kelly hypothesis, an increase in one of the substances within the rigid cranial vault must be compensated for by a reduction in one of the other substances in order for ICP to remain constant.
  - An increase in ICP due to a blockage in the removal of CSF or a space occupying lesion may result in compression of cranial venous sinuses, reducing venous blood content
  - Once compensatory mechanisms are exhausted, high ICP results in reduction in CBF and ischaemia.
- Raised cerebral venous pressure → as the movement of blood from the high pressure arteries to low pressure veins is influenced by downstream hydrostatic pressure, an increase in venous pressure (eg, neck ties for an ETT too tight, cerebral sinus thrombosis) will affect cerebral blood flow
- Vascular integrity → cerebral vasculitis can reduce CBF due to reduction in vascular caliber and/or compliance
- Blood viscosity → a increase in viscosity increases resistance to flow as per Hagen-Poiseuelle
- Temperature → $CMRO_2$ falls by ~6% for every 1°C fall in body temperature, accompanied by a similar reduction in blood flow
- Drugs → various anaesthetic agents reduce CMRO2 and thus CBF (thiopentone, propofol)