Q18 Describe the physiology of intracranial pressure and the physiological mechanisms that limit a rise in intracranial pressure (March 2010)

- ICP → pressure within the cranium, determined by total volume of substance within the skull [three compartments: brain tissue (80-85%), cerebrospinal fluid (7-10%) and cerebral blood volume (5-8%)]. Normal ICP <15mmHg
- Monroe-Kellie hypothesis → states that the skull is a rigid box, and for ICP to remain normal, an increase in any one of the volumes must be matched by a decrease in another
- The relationship between volume and pressure in the brain can be represented on an elastance curve (change in pressure/change in volume), which is the inverse of compliance.
- ICP is directly related to intrathoracic pressure and has a normal respiratory swing
- It is increased with coughing, straining and PEEP

CSF

- Specialised extracellular fluid located in the ventricles, the spinal cord central canal and the subarachnoid space that bathes the brain and the spinal cord
- Produced in the choroid plexus and endothelial cells lining the brain capillaries at a rate of 20-30ml/hr (500ml/day)
- Only 150ml present at any one time, ie, constant turnover with excess reabsorbed by arachnoid villi
- Any impairment of CSF reabsorption (eg, meningitis, encephalitis, trauma) can increase ICP

CEREBRAL BLOOD FLOW

- At rest the brain receives around 750ml of flow, which equates to approximately 15% of total cardiac output.
- **Cerebral blood flow (CBF) = cerebral perfusion pressure (CPP) / cerebrovascular resistance (CVR).** The CPP is normally MAP - CVP, however if raised ICP develops, a starling resistor model is set up in which ICP>CVP and therefore CPP = MAP - ICP

RAISED ICP

- Caused by an increase in volume of any of the three components of the brain (CSF, brain tissue, blood)
- Simple compensatory mechanisms - initially CSF is displaced and this acts as a physical buffer to pressure changes
- Venous sinuses are then compressed, displacing blood into the jugular veins, reducing the amount of venous blood present
- Eventually the rise in ICP will exceed CPP and CBF will fall, causing ischaemia and the Cushing reflex (hypertension, bradycardia, abnormal respiratory pattern) → Cerebral ischemia at the medullary vasomotor center induces initial activation of the SNS. Such activation will lead to an increase in heart rate, blood pressure, and myocardial contractility in an effort to improve cerebral perfusion. As a result of the high vascular tone, reflex bradycardia mediated by baroreceptors will ensue.