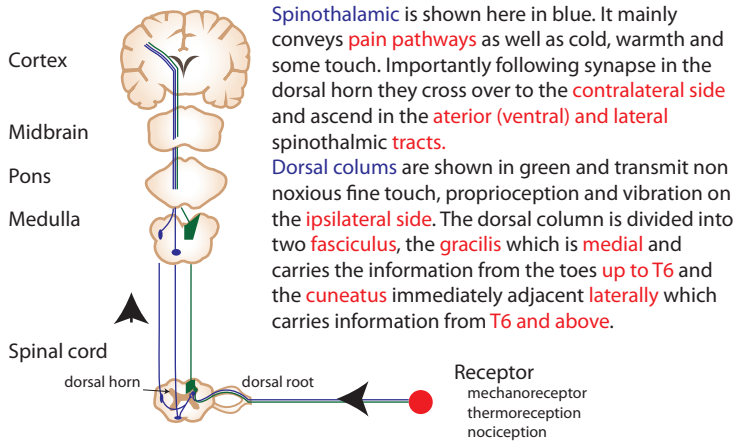
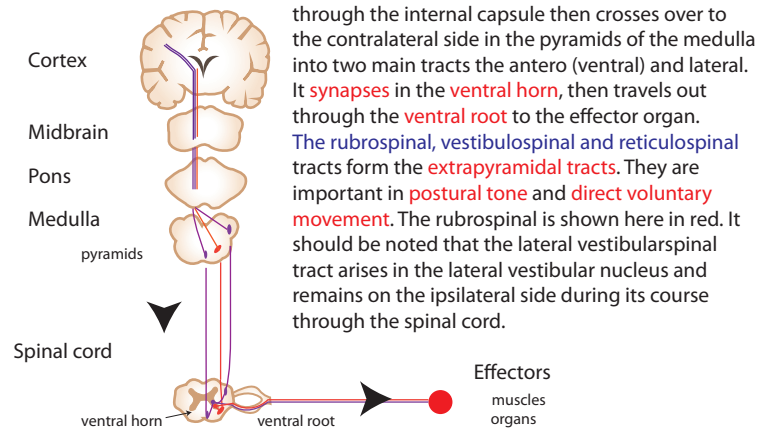


NERVOUS SYSTEM PHYSIOLOGY 2

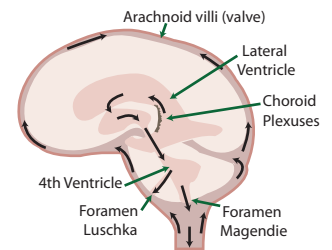
Ascending sensory pathways The afferent nerve fibres enter the CNS via the **spinal cord dorsal roots**, the trigeminal and the vagus nerve roots. After entering the spinal cord they **ascend** within the spinal cord to their **final destination** which is usually designated by the **suffix**. There are **six in total**, only two are shown here the others being spino-reticular, -mesencephalic, -limbic, and -cervicothalamic.



Descending motor pathways from the cerebral cortex excite the spinal cord neurons both directly and indirectly. There are **six descending motor pathways**, two from the cerebral cortex (corticospinal and corticobulbar), and four from the brainstem (the rubrospinal, vestibulospinal, tectospinal and reticulospinal). The **corticospinal tract** (aka **pyramidal tract**) is the **most important** motor pathway and is shown here in purple. It originates in the **motor cortex**, travels through the internal capsule then crosses over to the **contralateral side** in the pyramids of the medulla into two main tracts the **anterior (ventral) and lateral**. It **synapses in the ventral horn**, then travels out through the **ventral root** to the effector organ.

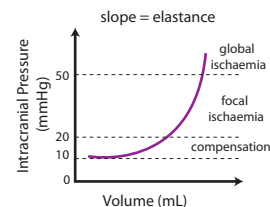


Cerebrospinal fluid is a specialised extracellular fluid located in the **ventricles**, the **spinal cord central canal** and the **subarachnoid space** that bathes the brain and the spinal cord. It has a total **volume of 150mL** in a male adult and is **formed** in two different places, the **choroid plexus (70%)** and the **endothelial cells lining the brain capillaries (30%)**. The formation occurs by both passive filtration and active transport. This occurs at a rate of around **500mL/day**, which equates to a **turnover of 3-4 times per day**. Its formation is **dependent** on the **cerebral perfusion pressure**, with the arterial pressure being most significant (production is unchanged with a modest increase in ICP). Under most conditions it is assumed that the formation of CSF is constant. CSF is **absorbed** in the **arachnoid villi** and granulations and these **act as valves** with CSF escaping to the venous system but not backflowing, as the **pressure increases the valves open wider**. Whilst it is **isotonic with plasma** their **compositions differ**, CSF having minimal large components due to filtration, with **negligible albumin, globulin and fibrinogen**. It has **increased Cl⁻**, and **Na⁺**. CSF also has increased CO₂ with a **pCO₂ of 50**, leading to a **pH of 7.32**. CSF has less of the other major cations with **less K⁺, Ca⁺⁺ and Mg⁺⁺**. It has a number of important **functions**. Firstly it provides **mechanical protection** for the brain by making it buoyant and reducing its effective weight from 1400g to 50g. This prevents the brain from injury due to changes in position or due to acceleration and deceleration. The second function is maintaining a **constant ionic environment** and preventing sudden fluxes in ion composition. The CSF is also very important in **acid base regulation**, this is especially the case in terms of primary respiratory disorders. CO₂, which diffuses across the membrane, changes to H⁺ and HCO₃⁻ and acts on the central chemoreceptors to adjust the respiratory rate. The CSF provides **intracerebral transport for important substances** such as amino acids and sugars. Finally it protects the brain by physically **buffering increases in ICP** by translocating to the extracranial space. CSF flow is determined by the **pressure in the lateral ventricle** (normally 15cm H₂O) and **cilia direct** flow towards the fourth ventricle. It exits the brain via two foramina, the Foramen of **Luschka** and Foramen **Magendie**.



Cerebral blood flow At rest the brain receives around **750ml of flow** which equates to approximately **15% of total CO**. It has a high myocardial consumption of **3-3.5 ml O₂/min/100g** which when calculated using the weight of the brain (1400g) results in around 50 ml of oxygen which is **20% total consumption** (remembering that total oxygen consumption is around 250ml/min). As always flow = pressure/resistance. In this setting it is the **cerebral blood flow (CBF) = cerebral perfusion pressure (CPP) / cerebrovascular resistance (CVR)**. The CPP is normally MAP - CVP, however in pathological states **where there is raised ICP a Starling resistor model is set up**. In this setting ICP > CVP and therefore the CPP = MAP - ICP. Extrinsic nerve and hormonal control have little influence on CBF. The cerebral circulation demonstrates a very **well controlled autoregulation** normally within the range of **50-150mmHg**. It should be noted that in chronic hypertension this may be reset. The mechanism of autoregulation has not been fully elucidated, however it is believed to be primarily due to **myogenic stretch factors**. Local **metabolic factors** are significant also, but primarily from a **regional cerebral flow** perspective and may include adenosine, NO, H⁺. The exceptions to this are arterial **PCO₂** which demonstrates an **almost linear relationship with CBF from 20mmHg-80mmHg**, and to a lesser extent O₂ which when to oxygen content drops significantly (associated with a **PaO₂ of less than 50mmHg** which is on the steep part of the dissociation curve) there is an increase in CBF. Above this point oxygen saturations have little influence.

Intracranial pressure The intracranial space contains **three compartments: brain tissue (80-85%), cerebrospinal fluid (7-10%) and cerebral blood volume (5-8%)**. Intracranial pressure is determined by the total volume of each because the **cranial vault is not distensible**. The normal intracranial pressure is between 5-15mmHg. The **Monroe-Kellie hypothesis** states that for ICP to remain normal, an increase in any one of the volumes must be matched by a decrease in another. Brain tissue volume comprises mainly of ECF and ICF, CSF volume is determined primarily by the ration of production to absorption, and CBV is the sum of the arterial and venous blood volumes. 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. Initially **CSF is displaced** and this acts as a **physical buffer** to pressure changes but eventually there is a sharp rise in pressure with ongoing increases in volume.



Sleep is an essential, readily **reversible physiological state** characterised by **unconsciousness, reduced muscle tone, analgesia, amnesia, respiratory and autonomic disturbances and dreaming**. There are two main states of sleep, **non rapid eye movement (NREM) sleep** and **REM sleep**. NREM sleep is also known as **slow wave sleep**. It is a deep restful sleep and is characterised by **decreases** in peripheral **vascular tone, blood pressure, respiratory rate, and metabolic rate**. Slow wave sleep is brought about by the **inhibition of the midline pontine and medullary nuclei (raphe nuclei)**. NREM sleep is divided into **four stages**. During progression through the stages the **EEG becomes more synchronised and slows** and the person becomes more difficult to rouse (stage 4 is the most difficult). **REM sleep (paradoxical or stage 5)** is distinct from slow wave sleep because the **brain becomes quite active, skeletal muscle contractions** occur and the EEG resembles **rapid low-voltage, irregular (desynchronised) activity** which is **similar to alert humans**. REM sleep **lasts for 5-30 minutes** and occurs at approximately **90 min intervals**. This period of sleep is characterised by decreased muscle tone, irregular heart rate and respiration and **increased brain metabolism by up to 20%**. **Natural sleep and anaesthesia** share many similarities, but they are clearly distinct. **Both** are characterised by **unconsciousness, analgesia, amnesia, and atonia (in REM sleep)**. The **EEG in sleep** is characterised by **predictable rhythmic variations** generated endogenously. The effects of **anaesthetics** on the **EEG are agent and dose dependent**, but can resemble the patterns observed during different stages of sleep. Natural **sleep** is characterised by **autonomic variability**, whereas most **anaesthetics** produce **autonomic stability** even in the face of painful stimuli. Finally in contrast to anaesthesia natural **sleep** is characterised by **easy arousability and spontaneous movements**.