Head injuries—pathophysiology

The brain is the most sensitive organ in the body to hypoxia and ischaemia. Therefore it is essential to maintain an adequate supply of well oxygenated blood to the injured brain.

Autoregulation maintains a constant supply of blood to the brain between a mean blood pressure (BP) of 50 and 160 mmHg. However, this mechanism is usually **impaired following head injury**. The **cerebral perfusion pressure** (CPP) is the force driving blood through the brain and is normally over 70 mmHg. It is related to the BP and intra-cranial pressure (ICP) by:

CPP = BP – ICP.

A developing **intra-cranial mass lesion** will initially be compensated for by displacement of venous blood and CSF, so the ICP will not rise. When this compensatory mechanism has been exhausted the ICP will rise and the CPP fall. The **Cushing reflex** then comes into play, increasing the BP to maintain cerebral blood flow. The pulse rate also falls due to a vagal reflex. When this compensatory reflex fails progressive cerebral ischaemia will occur leading to cerebral infarction and brain death. A vicious circle becomes established with hypoxia, hypotension and cell breakdown products, which worsen cerebral oedema, contributing to the deterioration.

Brain herniation

Three types of herniation can occur when a mass lesion develops intra-cranially.

- **Sub-falcine herniation**. One hemisphere is displaced beneath the falx, which is seen as midline shift on a CT scan. This can obstruct the foramen of Monro anteriorly, causing unilateral ventricular dilatation and compress the posterior cerebral artery against the falx posteriorly, causing a posterior cerebral infarct.
- **Trans-tentorial herniation**. The uncus of the medial temporal lobe herniates through the tentorial notch. This compresses the occulomotor nerve (dilatated pupil), and the midbrain.
- **Tonsilar herniation**. The cerebellar tonsils herniate through the foreman magnum causing brainstem compression (coning). This is the ultimate cause of brain death.

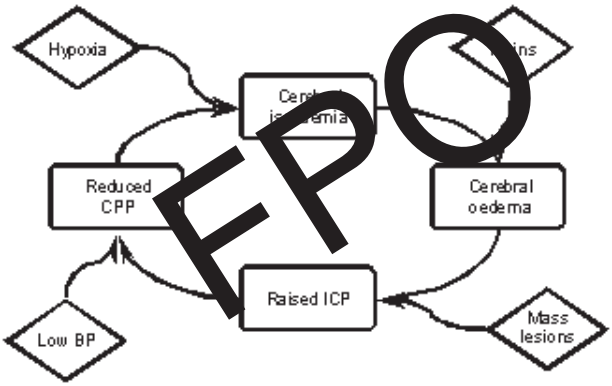

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Fig. 4.1