RAISED INTRACRANIAL PRESSURE/CEREBRAL BLOOD FLOW CONTROL

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The components within the skull are brain, blood and cerebrospinal fluid (CSF). increase in the volume of one component must be accompanied by an equal reduction in the volume of the others in order to maintain the same intracranial pressure (ICP).

The initial compensatory mechanism for an increase in the volume of brain matter or blood is the extrusion of CSF into the spinal sac. When this mechanism is exhausted, further volume increases result in a sudden large increase in ICP. Raised ICP eventually impedes cerebral perfusion which if unchecked initiates a 'vicious cycle' of hypoperfusion, ischemia and cerebral oedema.

Further brain swelling causes:

• Herniation of the temporal lobe through the tentorium and of the cerebellar tonsils through the foramen magnum.

• Brainstem torsion with reduced cerebral blood flow (CBF) and sudden obstruction of CSF flow with acute hydrocephalus

CLINICAL SIGNS AND SYMPTOMS OF RAISED ICP

• Headache, worse after waking, which is exacerbated by lying down.

• Nausea and vomiting.

• Papilledema.

• Decreased conscious level.

• Hypertension and bradycardia.

• Abnormal respiratory pattern. (Irregular, bradypnea, apnea)

CAUSES OF RAISED ICP

• Traumatic brain injury

• Space-occupying lesions (e.g., tumor, abscess)

• Hemorrhage

• Venous thrombosis

• Infection (e.g., meningitis, encephalitis, abscess)

• Hydrocephalus

• Metabolic encephalopathy (e.g., hypoxia, hypercapnia, hypoglycemia, hepatic)

• Status epilepticus

AGGRAVATING FACTORS

• Venous obstruction (e.g., from poor neck positioning, tube ties, etc.)

• Raised intrathoracic pressure.

• Fiber-optic bronchoscopy increases the ICP significantly but transiently. This is not completely abolished by sedation, analgesia or muscle relaxation.

AMELIORATING FACTORS

• **A head-up tilt of 30°** gives maximal benefit from venous drainage, whilst minimizing the reduction in cerebral arterial pressure due to the hydrostatic pressure difference between the heart and brain level.

CEREBRAL PERFUSION PRESSURE (CPP) CPP is defined as the difference between the systemic MAP and the ICP. In health, it is typically 70–85 mmHg. In patients with traumatic brain injury, guidelines typically advise maintaining a CPP of 50–70 mmHg

CEREBRAL BLOOD FLOW Normal cerebral blood flow (CBF) is approximately 50 mL min−1 per 100 g of brain tissue. Of the three intracranial components described by the Monroe Kellie doctrine, blood is the only one that can be manipulated nonsurgical. The principles of managing CBF are therefore fundamental to the initial management of the patient with raised ICP. Autoregulation of cerebral blood flow is maintained between a mean arterial pressure (MAP) of 50 and 150 mmHg; however, autoregulation can be impaired in brain injury and with the use of various medications, including volatile anesthetic agents.

REGULATING FACTORS

• Cerebral metabolic rate for oxygen (CMRO2) – CBF increases proportionally with increasing CMRO2. This is increased by pyrexia, pain, agitation and seizures.

• PaO2 – there is little change in CBF above a PaO2 of 6.7 kPa. Below this, CBF increases.

• PaCO2 – there is a near-linear relationship between CBF and PaCO2 between 2.7 kPa (maximal vasoconstriction) and 10.6 kPa (maximal vasodilation). Hyperventilation reduces CBF only temporarily, with a return to normal values after approximately 5 hours.

• Other (e.g., autonomic innervation, local factors).

PHARMACOLOGICAL AGENTS THAT AFFECT CBF

- **Inhalational agents**:

Isoflurane has an indirect vasoconstricting effect secondary to a reduction in CMRO2 and a direct vasodilating effect.

Isoflurane provides cerebral protection and ischemic changes do not develop until CBF is reduced to 8–10 mL min−1 100g–1. At >1.5 MAC or in the damaged brain the vasodilating effect predominates.

Sevoflurane has similar effects to isoflurane. Autoregulation of cerebral blood flow is preserved up to 1.5 MAC

Desflurane produces complete abolition of autoregulation at 1.5 MAC and is a more potent cerebral vasodilator than isoflurane or sevoflurane at equivalent MAC dose. It is frequently used in neuroanaesthetic practice at a low dose in combination with remifentanil.

Nitrous oxide causes significant global increase in CBF by direct vasodilatation

- **Hypnotics**

Propofol reduces ICP, but may also reduce CPP due to a fall in MAP. In patients with intracerebral tumors, there is less cerebral swelling after opening the dura than when volatiles are used. Propofol without narcotic does not prevent the rise in ICP on intubation.

Barbiturates and midazolam produce a dose dependent reduction in CMRO2 and cerebral blood flow.

- **Muscle Relaxants**

Suxamethonium may cause a small, transient increase in ICP. However, it is commonly used in rapid sequence induction for patients with traumatic brain injury.

Non-depolarizing muscle relaxants do not have any effect on ICP. Opioids In the absence of controlled ventilation, opioids will increase ICP secondary to hypercapnia from respiratory depression. In addition, some studies suggest that opioids may increase CBF, though the mechanism is poorly understood.

- **Osmotic agents**

The initial effect of a bolus of **mannitol i**s hemodynamic, augmenting intravascular volume and MAP and CPP. With intact autoregulation, cerebral vasoconstriction and a decreased metabolic rate reduce ICP. In addition, plasma expansion reduces blood viscosity, improving cerebral microvascular perfusion. The osmotic effect occurs after approximately 15 minutes, and removes water from the brain tissue down an osmotic gradient. This osmotic effect relies on the presence of an intact blood–brain barrier and both the hemodynamic and osmotic

effects are less effective in the damaged brain. Mannitol subsequently induces an osmotic diuresis, which may reduce MAP and CPP.

Hypertonic saline is an alternative to mannitol and is becoming more frequently used in clinical practice. A number of trials suggest that hypertonic saline is more effective than mannitol in reducing ICP, and presents an advantage in that it does not induce an osmotic diuresis