Intracranial pressure, cerebral blood flow and brain oedema

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Abstract

The normal autoregulatory systems that keep the intracranial pressure (ICP) within a tight physiological range will be outlined, along with the various mechanisms that culminate in pathological decompensation and raised ICP. Intracranial hypertension is a common neurosurgical problem that can be due to many causes including intracranial mass lesions and associated oedema, disorders of cerebrospinal fluid circulation or more diffuse intracranial pathologies. In this article we will review the three main types of cerebral oedema: cytotoxic, vasogenic and transependymal, their different mechanisms of formation and treatment. The signs and symptoms of intracranial hypertension will be described, together with indications for ICP monitoring and measurement methods. A detailed stepwise approach to managing raised ICP, including medical and surgical strategies, will be provided and the evidence of these treatments discussed. In the future, further appropriately powered, prospective, randomized controlled trials will be required to better guide treatment of this condition.

Keywords autoregulation; cerebral blood flow; cerebral oedema; intracranial pressure

Intracranial pressure

The normal intracranial contents are: brain (1400 ml), cerebral blood volume (150 ml) and cerebrospinal fluid (CSF) (80 ml). These components are contained within the fixed volume of the rigid skull and exert an even distribution of pressure termed the intracranial pressure (ICP). The normal physiological range of ICP varies with age (mmHg): adult: 10–15; children: 3–7; term infants 2–6. ICP can also be measured in cmH_2O, where 1 mmHg (torr) is equal to 1.36 cmH_2O. Raised ICP is a clinical feature of many neurological illnesses and requires urgent diagnosis and treatment.

The Monro–Kellie doctrine states that the sum of the three intracranial component volumes is constant. Any increase in a compartmental volume, for example from a brain tumour, spontaneous haematoma or traumatic brain injury (TBI), must be offset by a decrease in the volume of the other components otherwise the ICP will rise. Autoregulatory compensatory mechanisms can buffer increases in volume to ensure the ICP is kept within normal limits: 1) displacement of CSF into the spinal intradural space; 2) changes in vascular resistance resulting in increased drainage of cerebral veins and venous sinuses into systemic venous system. Once these mechanisms become exhausted, decompensation occurs and the ICP will rise (Figure 1, red asterisk). Decompensation occurs earlier with a rapid increase in volume, for example from a spontaneous haematoma, or malignant tumour in comparison with a slow growing benign lesion. As ICP rises, a vicious cycle of further brain swelling and secondary brain injury occurs. This is a life-threatening condition that can result in compromised brain circulation causing cerebral ischaemia. Once the ICP equals the arterial pressure, arterial blood will be unable to enter the brain resulting in massive infarction. A localized mass with associated swelling can result in brain shift and herniation.

Cerebral blood flow and autoregulation

The brain requires a constant cerebral blood flow (CBF), and receives 12% of the cardiac output. Of all body tissues it is the least tolerant of ischaemia. CBF interruption for 5 seconds will cause loss of consciousness and ischaemia of longer than 3 minutes will result in irreversible brain damage. Specific areas of brain have varying requirements depending on their metabolic activity (e.g. white matter 20 ml/g/minute to grey matter 100 ml/g/minute). The following formulae link CBF to cerebral vascular resistance (CVR) and cerebral perfusion pressure (CPP), and CPP to the mean arterial pressure (MAP) and ICP:

$$CBF = CPP/CVR$$

$$CPP = MAP - ICP$$

Under normal physiological conditions cerebral autoregulation maintains a constant CBF of approximately 50 ml/minute/100 g of brain over a wide range of CPP (50–140 mmHg) by altering the vascular resistance (Figure 2). Autoregulation is impaired after TBI and the curve shifts to the right. Once autoregulatory mechanisms are completely exhausted the CBF correlates closely with CPP. Neurointensive care management of these patients includes ICP monitoring and allows for targeted CPP management using intravenous fluids and inotropes. Evidence supports CPP of over 60 mmHg[1] but systemic morbidity over 70 mmHg.[2]

Dynamic imaging techniques are available to provide an insight into either blood flow through vessels, or to assess cerebral perfusion.

- Transcranial Doppler (TCD) is an ultrasound-based non-invasive technique that can be performed at the bedside test. It measures the velocity of blood flow in a specific artery in real time. Areas where the skull is thinnest are used as insonation windows (e.g. temporal area for middle cerebral artery). The technique is widely used in patients in the diagnosis and management of vasospasm and its clinical correlate of neurological ischaemic deficit following subarachnoid haemorrhage. Following TBI, TCD may guide individual specific CPP targets, and test autoregulatory reserve. The technique is highly operator dependent, and long-term recordings are limited by the requirement for a fixed accurate probe.
- CT perfusion has been used as an adjunct in acute stroke to image the ischaemic penumbra. MRI perfusion has been

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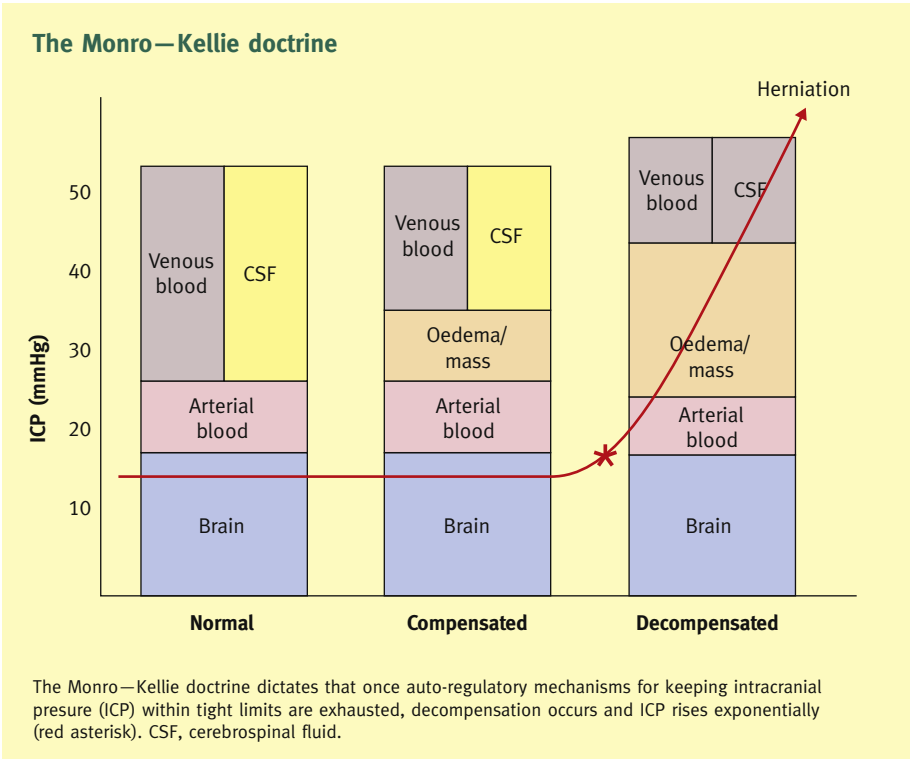
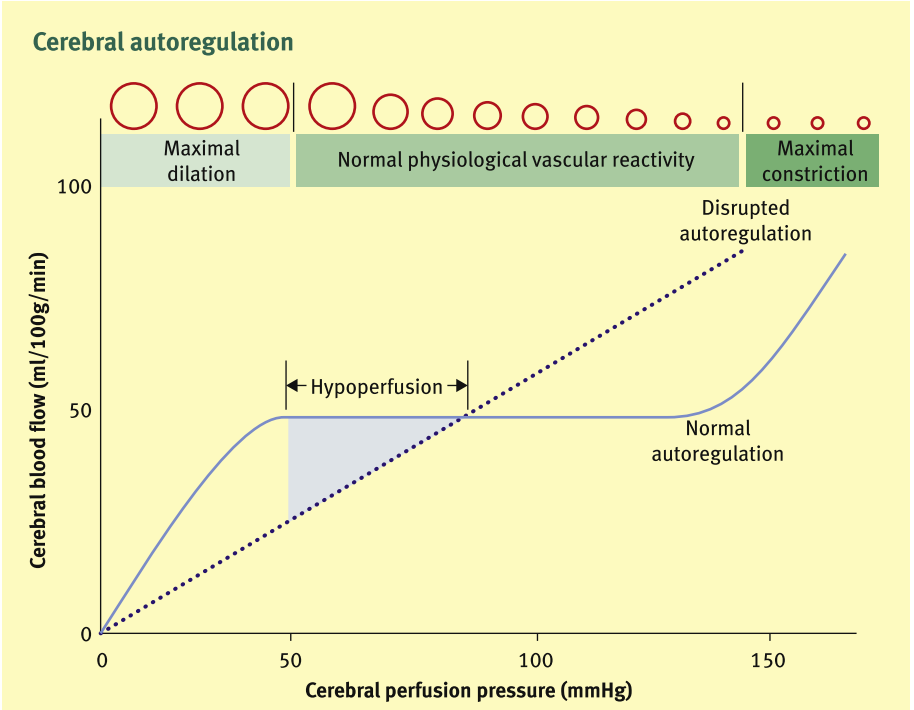


The Monro—Kellie doctrine dictates that once auto-regulatory mechanisms for keeping intracranial presure (ICP) within tight limits are exhausted, decompensation occurs and ICP rises exponentially (red asterisk). CSF, cerebrospinal fluid.

Figure 1



Cerebral autoregulation maintains a constant cerebral blood flow (CBF) over a wide range of cerebral perfusion pressure (CPP) from 50 to 140 mmHg (solid line) by altering vascular resistance (represented by circles). Following brain injury autoregulation may be completely lost resulting in a linear relationship between CPP and CBF (dashed line).

Figure 2