Intracranial pressure and cerebral haemodynamics

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Abstract

Intracranial pressure (ICP) refers to the pressure within the skull, which is determined by the volumes of the intracranial contents; blood, brain and cerebrospinal fluid. Monro–Kellie homeostasis stipulates that a change in the total intracranial volume is accompanied by a change in the ICP, which is more precisely described by the intracranial pressure–volume relationship. Maintenance of a relatively constant ICP is essential for maintenance of the cerebral perfusion pressure, which in turn determines global cerebral blood flow. Although the physiological process of autoregulation ensures that cerebral blood flow is tightly maintained over a range of cerebral perfusion pressures, large increases in the ICP can result in severely impaired autoregulation, meaning that cerebral blood flow may be compromised. In this review article we provide an overview of the physiological determinants of the ICP and cerebral blood flow. We go on to illustrate how pathological states can compromise physiological compensatory mechanisms in order to potentially dangerous disturbances of the ICP and cerebral blood flow.

Keywords Cerebral blood flow; cerebral perfusion pressure; intracranial pressure; Monro–Kellie doctrine

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Learning objectives

After reading this article you should:

- know the mathematical relationship between cerebral perfusion pressure, intracranial pressure, and mean arterial pressure, and therefore appreciate why increases in the intracranial pressure can be pathological
- understand the profile of the relationship between intracranial volume and intracranial pressure
- understand the concept of Monro–Kellie homeostasis, which states that the intracranial pressure will remain constant if there is no change in the total intracranial volume – which comprises the volumes of brain, cerebrospinal fluid and blood
- have an appreciation of the principle of autoregulation – which maintains cerebral blood flow despite variations in arterial pressure

Intracranial pressure

The term intracranial pressure (ICP) refers to the pressure of the contents contained within the skull. The normal ICP varies cyclically with respiration and the cardiac cycle, and there may also be transient changes in ICP with posture, coughing and straining. Measured in the supine position, the normal range of ICP in adults is 7–15 mmHg.[1] Interestingly, there is no established consensus on a normal range of ICPs measured over a prolonged period of time in freely moving humans.

When the ICP is persistently raised (>15 mmHg) intracranial hypertension develops and the cerebral perfusion pressure – which is the pressure gradient causing cerebral blood flow (CBF) to the brain – will be reduced, leading to focal and then global ischaemia. In addition to this, lesions producing a raised ICP may cause localized displacement of brain tissues across structures in the skull resulting in herniation. Some examples of herniation syndromes are:

- transtentorial (or uncal) herniation, which involves a shift of the uncus of the temporal lobe downwards through the tentorium resulting in compression of important structures such as the posterior cerebral artery, the third cranial nerve and corticospinal fibres
- subfalcine herniation is characterized by displacement of the brain (typically the cingulate gyrus) beneath the free edge of the falx cerebri
- tonsillar herniation, which is a potentially fatal complication of raised ICP and involves herniation of the cerebellar tonsils through the foramen magnum resulting in compression of the brainstem respiratory centres.

Intracranial components and the Monro–Kellie doctrine

The adult skull can be considered as a bony box of fixed volume whose contents are: brain, blood and cerebrospinal fluid (CSF). The Monro–Kellie doctrine stipulates that the sum of the volumes of the three components is constant and therefore that an increase in the volume of any one component needs to be accompanied by a reduction in the volume of at least one of the remaining two components.[2,3] A failure of this homoeostatic mechanism in certain pathological states may result in potentially dangerous increases in intracerebral volume and therefore intracranial pressure. Before considering ICP and cerebral blood flow in further detail, we will first outline the properties of the three intracranial components.

Brain

Brain parenchyma has a mass of approximately 1.4 kg and consists of neurons, glial cells and extracellular fluid. There are three different types of neurons. Afferent neurons transmit information from sensory organs to the central nervous system (CNS), whilst

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efferent neurons transmit information from the CNS to the periphery. Interneurons facilitate communication between afferent and efferent neurons. There are also three types of glial cells, including astrocytes, oligodendrocytes and microglia.

The barrier between the blood and the interstitial fluid of the brain (i.e. the blood—brain barrier (BBB)) consists of tight junctions between capillary endothelial cells and facilitates the maintenance of an appropriate environment for neuronal activity. Pathological increases in the volume of brain tissue can result from tumours or cerebral oedema for example.

Cerebrospinal fluid (CSF)

CSF occupies the space between the arachnoid membrane and the pia mater. CSF is produced by the choroid plexus of the ventricular system and has a number of important functions for the brain including providing buoyancy, mechanical protection, and chemical stability. Importantly, the presence of CSF is thought to reduce the effective weight of the brain to around 25 g, allowing the brain to maintain its density without significant compression of its blood supply.[4] A long postulated, and increasing recently recognized role of CSF is to provide a waste clearance pathway for the CNS that has a somewhat similar role to the lymphatic system found in other organs. This pathway is known as the Glymphatic system and consists of a para-arterial influx route for CSF to enter the brain parenchyma, coupled to a clearance mechanism for interstitial fluid.[5]

CSF is produced at a rate of approximately 500 ml/day (approximately 20 ml/hour), meaning that the entire CSF volume of 150 ml is replaced more than three times daily. Approximately 25 ml of the total CSF volume is present within at ventricular system of the brain. Although it had traditionally been believed that CSF resorption takes place at arachnoid granulations, recent evidence highlights that exchange between the CSF and interstitial fluid compartments is possible across the pia mater.

Cerebral blood flow

The brain receives arterial blood from the internal carotid and vertebral arteries, and its venous drainage is to cerebral veins, venous sinuses and the internal jugular veins. It is interesting to note that the cerebral blood flow is large in comparison to the volume of blood in the cranium at any point in time. Cerebral blood flow is approximately 700 ml/minute, corresponding to 15% of the cardiac output, whilst the intracranial blood volume is only 150 ml.[6]

Based on the simplification of blood being incompressible and uniformly viscous, factors determining cerebral blood flow can be revealed by consideration of the Hagen—Poiseuille equation, as follows:

$$CBF = \frac{CPP.\ \pi.\ R^4}{8.\eta.\ L}$$

Here *CBF* represents cerebral blood flow, *CPP* denotes cerebral perfusion pressure, *R* denotes blood vessel radius and η represents the viscosity of the blood, with *L* indicating the vessel length. We may assume that blood viscosity and vessel length remain constant, leaving the major determinants of cerebral blood flow to be the cerebral perfusion pressure and the vessel radius. The complex, non-linear relationship between CPP and CBF will be discussed in detail below. Changes in the vessel radius have a fourth power effect meaning that halving the vessel radius may theoretically result in a 16-fold reduction in cerebral blood flow.

The above description overlooks the fact that cerebral blood flow is cyclical and driven by the phases of the cardiac cycle. Nevertheless, there are physiological mechanisms which prevent large variations in ICP and cerebral blood flow during the cardiac cycle.

Cardiac systole is associated with an expansion of the blood volume within elastic cerebral arteries and this is accompanied by CSF displacement through the foramen magnum and an increase in venous outflow, hereby maintaining Monro—Kellie homeostasis and ICP. In contrast, during diastole CSF re-enters the cranial compartment and venous outflow decreases. Furthermore, arterial elasticity acts to dampen the arterial pulse pressure wave and this aids with the maintenance of a relatively stable blood flow (Windkessel effect).

The concept of Monro—Kellie homeostasis can be better understood by considering how changes in intracranial pressure can be caused by changes in the volumes of one or more of the intracranial components. Classically, the relationship between intracranial volume and pressure is described as having three parts, as follows.

- A flat part where the ICP remains low despite changes in volume due to effective Monro—Kellie homeostasis. In this portion of the curve the intracranial contents are said to have high compliance as the gradient of the pressure —volume curve is low (compliance = 1/gradient = dV/dP).
- A steep portion where compensatory mechanisms are no longer sufficient and the compliance progressively decreases i.e. increasing dV/dP.
- A plateau phase, indicating a terminal disturbance in cerebrovascular responses where the ICP begins to equilibrate with mean arterial pressure (MAP) and the cerebral perfusion pressure (CPP) is dangerously low.

The principle of Monro—Kellie homeostasis also explains why acute hydrocephalus is a neurosurgical emergency. The underlying pathology in this condition produces a state where the rate of CSF production is greater than the rate of CSF resorption. Given that the maximal rate of CSF production is 20 ml/hour it is possible for the intracranial volume to rapidly increase in acute hydrocephalus, resulting in a potentially fatal increase in the ICP.

Autoregulation

The maintenance of cerebral blood flow is critical since the brain depends on the oxidative metabolism of glucose for its principal energy source and is therefore highly intolerant of both hypoxia and hypoglycaemia. The physiological mechanism which allows the brain to maintain a relatively stable blood flow in spite of large changes in arterial blood pressure is known as autoregulation.[6]

The principle of autoregulation is best understood by considering the relationship between cerebral perfusion pressure (CPP) and cerebral blood flow (CBF). The cerebral perfusion pressure is mathematically given by the difference between the mean arterial pressure and the ICP:[6]

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