Intracranial pressure and cerebral blood flow

Tomas Partington

Andrew Farmery

Abstract

Intracranial pressure (ICP) is determined by the volumes of brain, blood and cerebrospinal fluid within the skull, which is of course of fixed volume. The Monro–Kellie hypothesis states that an increase in volume of one of these components must be compensated for by a reduction in volume of one or both of the others. If this compensation is insufficient, then potentially fatal increases in ICP can occur. Maintenance of relatively constant ICP is essential for normal perfusion of the brain. Cerebral blood flow is regulated both globally, in order to prevent hypo- or hyperperfusion resulting from changes in systemic arterial blood pressure, and locally, to meet the dynamic oxygen and substrate demands of different brain regions. Monitoring of ICP and the cerebral blood supply is possible through a variety of invasive and non-invasive techniques, and these techniques are already established in anaesthesia and intensive care medicine.

Keywords astrocyte; autoregulation; brain; cerebral blood flow; cerebrospinal fluid; intracranial pressure; Monro–Kellie

Royal College of Anaesthetists CPD matrix: 1A01

Learning objectives

After studying this article the reader should understand the:

- components that determine ICP and the Monro–Kellie hypothesis
- concept and importance of cerebral blood flow autoregulation
- controls of cerebral blood flow, including activity-flow coupling and partial pressures of O_2 and CO_2
- techniques for monitoring of ICP, cerebral haemodynamics and cerebral oxygenation, and their applications to clinical management

Intracranial pressure

The Monro–Kellie hypothesis states that "if the skull is intact, then the sum of the volumes of the brain, cerebrospinal fluid (CSF) and intracranial blood volume is constant". This dictates that an increase in volume of one intracranial element must occur at the expense of the volume of one or more of the others. A change in intracranial pressure (ICP) may also result from this volume change, and ICP is variable both in health and disease. In order to understand the causes and compensatory mechanisms for changes in ICP, one must first consider the components present within the skull, and their individual properties.

Brain

The brain parenchyma has a mass of approximately 1400 g, and consists of neurons and glial cells, as well as extracellular fluid. The blood–brain barrier (BBB) consists of tight junctions between capillary endothelial cells, and separates blood from the interstitial fluid of the brain in order to provide an appropriate environment for neuronal activity. Causes of pathological increases in brain tissue include tumours, cytotoxic oedema (due to cell membrane failure) and vasogenic oedema (due to BBB disruption).

Tomas Partington *BA (Oxon) is a Senior Scholar at Wadham College, Oxford, UK. Conflicts of interest: none declared.*

Andrew Farmery *BSc MA (Oxon) MD (Lond) FRCA is a Fellow and Tutor in Physiology at Wadham College, Oxford, UK. Conflicts of interest: none declared.*

Cerebrospinal fluid

CSF occupies the space between the arachnoid membrane and the pia mater. Its functions include maintaining a chemically stable environment, supporting transport of metabolites and neurotransmitters and supplying glucose. The brain can be said to float in the CSF, a consequence of which is an effective reduction in its weight of 97%. The effects of linear and shearing mechanical forces on the brain are also reduced by protective displacement of CSF.

The subarachnoid space, and therefore CSF, also extends along the length of the spinal cord, exiting the cranial cavity via the foramen magnum. This communication provides the potential for some displacement of CSF into the extracranial compartment.

CSF is produced by the choroid plexus at a rate of approximately 500 ml per day, which means that the entire CSF volume (~150 ml) is replaced more than three times over 24 h. The bulk flow theory of CSF absorption occurring solely at arachnoid granulations, due to a pressure gradient, has now been generally replaced by a model of more widespread absorption. Interstitial fluid produced by capillaries in the perivascular space exchanges with CSF across the pia mater. Plasma proteins and other molecules in the CSF are actively taken up by the capillaries, and thus reach the venous outflow.

Blood

Arterial blood supply to the brain is via the internal carotid and vertebral arteries, which enter the skull via the carotid canals and foramen magnum, respectively. Blood drains via the cerebral veins, venous sinuses and internal jugular veins, exiting the cranial cavity through the jugular foramina. Normal intracranial blood volume is around 150 ml, two thirds of which is in the venous system.

Cerebral blood flow (CBF) is high, due to the brain's energy demands. At an average of 50 ml/100 g/min (higher in grey matter than white matter), global CBF is around 700 ml/min, accounting for more than 15% of total cardiac output.

Pathologically increased cerebral blood volume commonly results from haemorrhage (extradural, subdural, intracranial or subarachnoid).

Normal and abnormal ICP

Normal ICP in an adult is 5–13 mmHg, with minor cyclical variations due to arterial pulse pressure and respiration. Other causes for normal variation include posture, coughing and

straining. Sustained ICP greater than 15 mmHg is referred to as "intracranial hypertension". ICP higher than 20 mmHg results in areas of focal ischaemia, whereas global ischaemia is present above 50 mmHg.

As well as reducing global cerebral perfusion pressure, the "mass effect" of lesions producing elevated ICP can cause localised displacement of brain tissue across fixed structures within the skull, known as herniation. Uncal (transtentorial) herniation involves a shift of the innermost temporal lobe down through the tentorium, and can result in cranial nerve III palsy, posterior cerebral artery compression, and hemiparesis. Tonsillar herniation (or "coning") is a potentially fatal consequence of raised ICP, in which the cerebellar tonsils herniate down through the foramen magnum, compressing vital respiratory centres in the brainstem.

Monro–Kellie homeostasis

The complex compensatory mechanism for the transient increase in intracranial blood volume during cardiac systole has been termed "Monro–Kellie homeostasis". The effect of this mechanism is to minimise the change in ICP throughout the cardiac cycle, and to reduce systolic/diastolic blood pressure difference experienced by the cerebral microvasculature.

Figure 1 illustrates a model of the intra- and extra-cranial compartments of blood and CSF, and their communications. (Thin lines = compliant structure, Thick lines = non-compliant structure).

The model can be used to describe the fluid movements occurring during the phases of the cardiac cycle that represent Monro–Kellie homeostasis:

Systole:

- expansion of cerebral arteries causes a pressure wave transmitted through the CSF
- CSF is displaced down through the foramen magnum
- venous outflow increases

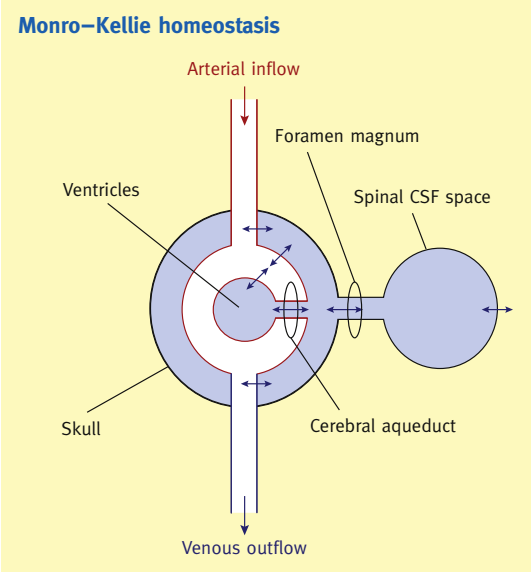


Figure 1 Monro–Kellie homeostasis.

Diastole:

- CSF is displaced back up through the foramen magnum
- venous outflow decreases

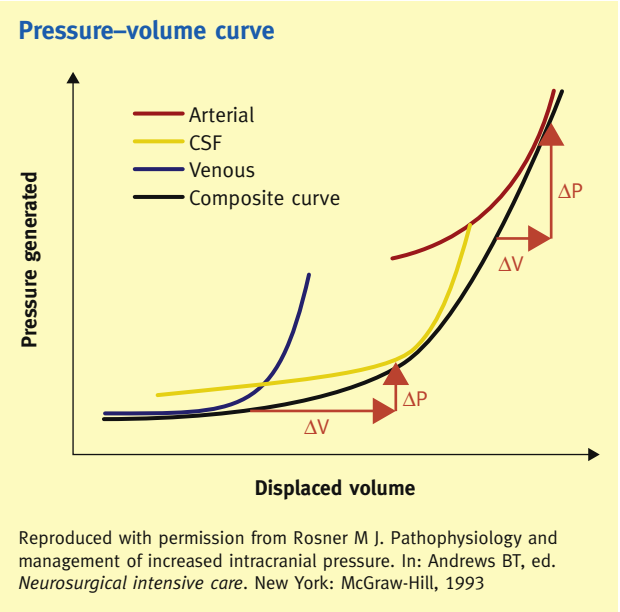
The elastic arterial walls also act to dampen the arterial pulse pressure wave and maintain constant capillary flow throughout the cardiac cycle (Windkessel effect).

Failure of Monro–Kellie homeostasis has been implicated in a range of cerebral disorders, including vascular/Alzheimer's dementia and late-onset depression, amongst others.

An increase in the volume of brain, blood or CSF, whether physiological or pathological, is initially "buffered" by a reduction in volume of venous blood and/or CSF within the skull in order to prevent or reduce a resultant increase in ICP. These components are the first to be displaced because their compartments (i.e. venous drainage system and subarachnoid space) are relatively *compliant*, meaning that venous blood and CSF can be displaced easily, in comparison to brain tissue. Significant volume displacement can be achieved with little change in ICP when compliance (dV/dP) is high, allowing effective compensation for small increases in volume of intracranial elements. However, when these compensatory mechanisms are insufficient, or if the growth of one intracranial component is too fast for them to be effective (slow changes are generally better tolerated than fast ones), then the effective compliance of the intracranial cavity is reduced, and ICP increases as a result (Figure 2).

Cerebral blood flow and autoregulation

The brain depends on oxidative phosphorylation of glucose as its principal source of energy, and is therefore highly intolerant of hypoxia and hypoglycaemia. For this reason, it is critical to



Reproduced with permission from Rosner M J. Pathophysiology and management of increased intracranial pressure. In: Andrews BT, ed. *Neurosurgical intensive care*. New York: McGraw-Hill, 1993

Figure 2 Initial volume displacements are possible with little increase in ICP, due to the compliance of the venous blood and CSF compartments. However, as more volume is displaced (e.g. as tumour size increases) the force (=pressure × area) required to displace a given volume from the cranial cavity becomes greater, therefore a given volume change results in a much more significant change in ICP. Slope of curves = *elastance* (=1/compliance).