

Raised intracranial pressure (ICP) is a common problem in neurosurgical and neurological practice. It can arise as a consequence of intracranial mass lesions, disorders of cerebrospinal fluid (CSF) circulation, and more diffuse intracranial pathological processes. Its development may be acute or chronic. There are well established methods for the measurement, continuous monitoring, and treatment of raised ICP. Evidence from prospective randomised controlled clinical trials that monitoring and treatment of raised ICP per se improves outcome is currently lacking for many conditions.

PATHOPHYSIOLOGY

The normal range of ICP varies with age (table 1) though values in the paediatric population are not well established. Thresholds for initiating treatment for intracranial hypertension vary according to aetiology and within single conditions there is debate about the appropriate upper limit of normal. For example, various authors have suggested thresholds of 15, 20, and 25 mm Hg for the initiation of treatment for raised ICP in patients with head injury.

Table 2 lists some common causes of raised ICP.

Volume–pressure relations

The relation between volume and pressure within the cranium is non-linear (fig 1). The Monro-Kellie hypothesis states that the sum of the intracranial volumes of blood, brain, CSF, and other components (for example, tumour, haematoma) is constant. The skull is considered as an enclosed and inelastic container. An increase in the volume of any one of the intracranial contents must be offset by a decrease in one or more of the others or be associated with a rise in ICP. Intracranial blood (especially in the venous compartment) and CSF are the two components whose volume can adapt most easily to accommodate an increase in the volume of intracranial contents. Once these compensatory mechanisms are exhausted, further increases in volume result in large rises in ICP. Compliance (the change in volume for a given change in pressure) provides an index of compensatory reserve, with low values suggesting a diminished reserve.

ICP waveform

Although continuous ventricular pressure monitoring in humans had been reported earlier, Lundberg first classified ventricular pressure fluctuations in humans in 1960 (fig 2).

- ▶ *A waves or plateau waves*—These comprise a steep rise in ICP from near normal values to 50 mm Hg or more, persisting for 5–20 minutes and then falling sharply. These waves are always pathological and indicate greatly reduced compliance. They are frequently accompanied by neurological deterioration.
- ▶ *B waves*—These rhythmic oscillations occur every 1–2 minutes. ICP rises in a crescendo manner to levels 20–30 mm Hg higher than baseline and then falls abruptly. These waves were originally always associated with Cheyne-Stokes respiration. However, they also occur in ventilated patients and are probably related to changes in cerebrovascular tone and cerebral blood volume. B waves are also indicative of failing intracranial compensation.
- ▶ *C waves*—These oscillations occur with a frequency of 4–8 per minute and are of smaller amplitude than B waves. They are synchronous with spontaneous Traub-Hering-Meyer type variations in blood pressure and are probably of limited pathological significance.

ICP pulse amplitude increased linearly with ICP. On occasion the pulse pressure may rise before a rise in mean ICP, indicating impaired compliance and giving advance warning of a rise in baseline ICP.

ICP and brain shifts

The cranial vault is divided into compartments by the dural reflections of the falx cerebri and tentorium cerebelli. Raised ICP frequently results in pressure gradients between compartments and a shift of brain structures. Many of the clinical counterparts of raised ICP are the consequence of such shifts rather than the absolute level of ICP. Patients with temporal lobe haematomas can

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Table 1 Normal intracranial pressure values

Age group	Normal range (mm Hg)
Adults	<10–15
Children	3–7
Term infants	1.5–6

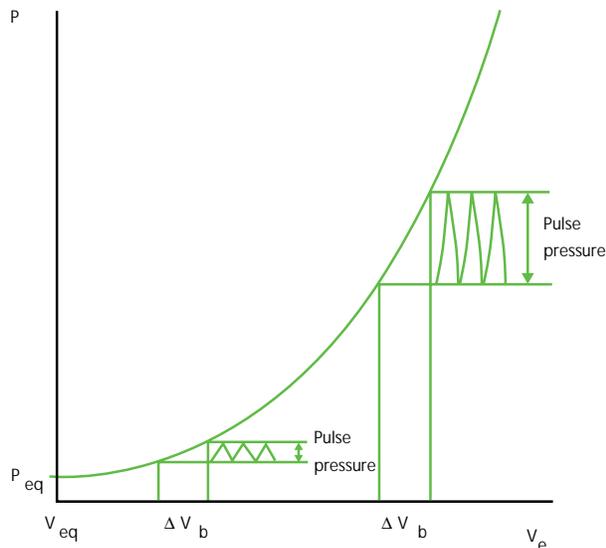


Figure 1 The volume–pressure curve.

undergo lateral transtentorial herniation without a rise in ICP, and it is important not to place uncritical reliance on ICP levels in the management of such patients. Three types of intracranial herniation are generally recognised: transtentorial (either lateral or central), tonsillar, and subfalcine (fig 3).

Interaction with blood pressure and cerebral blood flow

ICP and arterial blood pressure interact to affect cerebral blood flow, particularly in circumstances where cerebrovascular autoregulation has been impaired (such as following head injury). Cerebral perfusion pressure (CPP) is defined as: mean arterial pressure – ICP.

Under normal circumstances, cerebral blood flow is maintained constant over a range of cerebral perfusion pressures by cerebrovascular autoregulation. If autoregulation

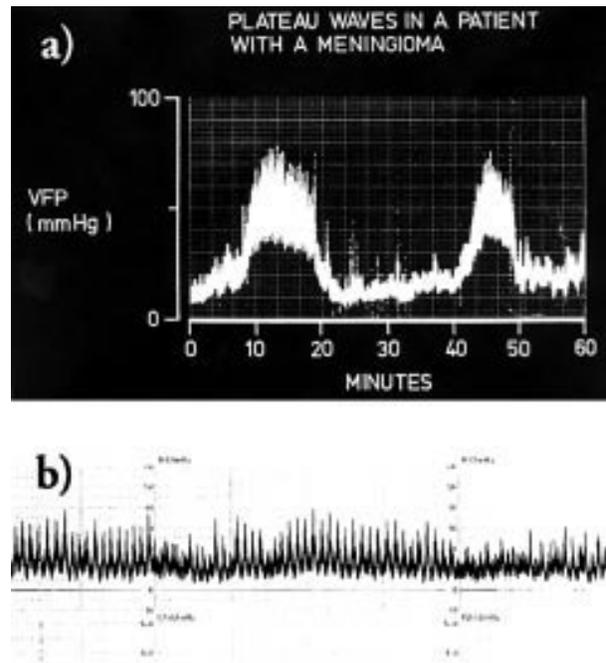


Figure 2 Types of intracranial pressure wave: a) plateau or A waves; b) B waves.

is impaired, changes in blood pressure or ICP can have direct effects on cerebral blood flow. Even if autoregulation is intact, changes in ICP and blood pressure may alter blood volume as a result of dilatation or constriction of cerebral blood vessels. This in turn will have an influence on ICP (fig 4).

ASSESSMENT

Clinical correlates of raised ICP

The combination of headache, papilloedema, and vomiting is generally considered indicative of raised ICP, although there is no consistent relation between the severity of symptoms and the degree of hypertension. Pressure headaches are often described as throbbing or bursting and are exacerbated by any factors that further increase ICP such as coughing, sneezing, recumbency or exertion. Classically the headache of raised ICP is worse in the morning. This has been attributed to a rise in ICP during the night as a consequence of recumbency, a rise in Pco₂ during sleep caused by respiratory depression, and probably a decrease in CSF absorption. Papilloedema is a reliable

Table 2 Examples of causes of raised intracranial pressure

Pathological process	Examples
Localised mass lesions	Traumatic haematomas (extradural, subdural, intracerebral) Neoplasms (glioma, meningioma, metastasis) Abscess Focal oedema secondary to trauma, infarction, tumour
Disturbance of CSF circulation	Obstructive hydrocephalus Communicating hydrocephalus
Obstruction to major venous sinuses	Depressed fractures overlying major venous sinuses Cerebral venous thrombosis
Diffuse brain oedema or swelling	Encephalitis, meningitis, diffuse head injury, subarachnoid haemorrhage, Reye's syndrome, lead encephalopathy, water intoxication, near drowning
Idiopathic	Benign intracranial hypertension

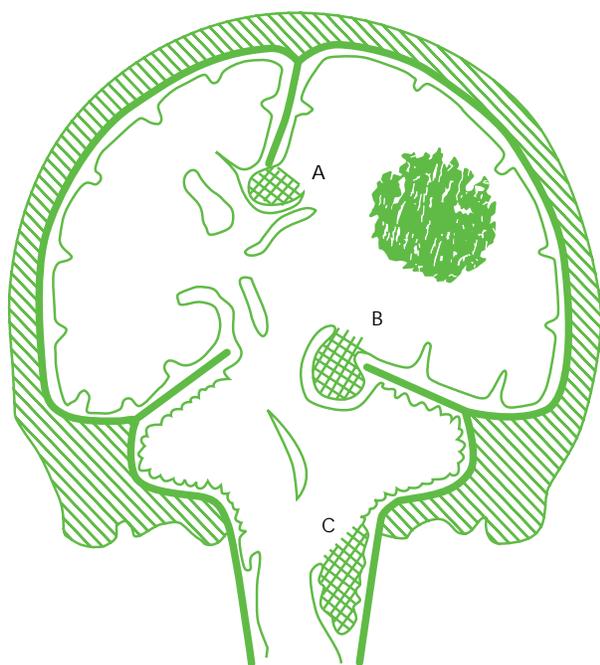


Figure 3 Intracranial herniations. (A) Cingulate herniation. (B) Uncal herniation. Most common clinically observed herniation. Uncus of temporal lobe herniates between rostral brain stem and tentorial edge into the posterior fossa, resulting in a clinical syndrome of progressively impaired consciousness, dilated ipsilateral pupil, and contralateral hemiplegia. (C) Tonsillar herniation. The tonsils of the cerebellum herniate through the foramen magnum into the upper spinal canal, compressing the medulla. Clinically this may result in cardiorespiratory impairment, hypertension, high pulse pressure, Cheynes-Stoke respiration, neurogenic hyperventilation, impaired consciousness, and death. The combination of bradycardia and hypertension is known as Cushing's response, and occurs in about one third of cases of tonsillar herniation.

sign of raised ICP but can require several days of raised pressure to develop. Fundal haemorrhages develop in response to acute and severe rises in ICP (as in subarachnoid haemorrhage and some cases of head injury). Longstanding raised ICP may fail to cause papilloedema if the subarachnoid sleeve around the optic nerve does not communicate with the subarachnoid space. Vomiting tends to be a late feature, usually occurs after waking, and frequently accompanies morning headache.

A progressive deterioration in conscious level (assessed using the Glasgow coma scale (GCS)) usually accompanies rising ICP, and is probably a consequence of caudal displacement of the diencephalon and midbrain. Other signs often seen in association with raised ICP, such as pupillary dilatation, bilateral ptosis, impaired upgaze, extension to pain, and respiratory irregularity, are related to tentorial or tonsillar herniation rather than the absolute level of ICP.

Changes in blood pressure, pulse, and respiratory pattern are usually late signs of raised ICP in clinical practice. These signs are related to brain stem distortion or ischaemia.

ICP monitoring

Indications

ICP monitoring is used either as a guide to treatment (for example, in the management of closed head injury) or as a diagnostic test (for example, in disorders of CSF circulation).

The most common use of continuous ICP monitoring is in the management of severe closed head injury. Bullock and

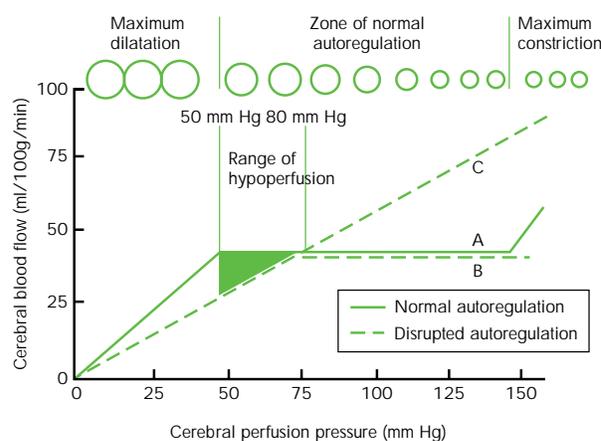


Figure 4 Cerebral autoregulation. Cerebral blood flow is normally maintained constant for perfusion pressures between 50–140 mm Hg (line A). Following injury the capacity for autoregulation may be lost completely (line C) or the threshold for autoregulation may be reset (line B). The circles above the graph represent the degree of precapillary arteriolar dilatation.

colleagues reviewed the published evidence base for the indications for ICP monitoring. They concluded that there were insufficient data to support standard treatment guidelines (no class I evidence). There was, however sufficient class II and III evidence to support the following recommendations:

- ▶ ICP monitoring is appropriate in patients with severe head injury (GCS between 3 and 8 after cardiopulmonary resuscitation) and an abnormal computed tomographic (CT) scan (haematomas, contusions, oedema or compressed basal cisterns)
- ▶ ICP monitoring is appropriate in patients with severe head injury and a normal CT scan if two or more of the following features are noted on admission: age over 40 years, unilateral or bilateral motor posturing, systolic blood pressure < 90 mm Hg
- ▶ ICP monitoring is not routinely indicated in patients with mild or moderate head injury; however, a clinician may choose to monitor ICP in certain conscious patients with traumatic mass lesions.

Other conditions in which ICP monitoring is used as a guide to treatment or for diagnostic reasons are listed in table 3.

Techniques

A variety of different techniques are available for the measurement and monitoring of ICP. Intraventricular fluid filled catheter transducer systems represent the “gold standard” for measuring ICP. This method allows checking for zero and sensitivity drift of the measurement system in vivo. Pressure measurement within the CSF space, provided CSF flow is not blocked, is not subject to the development of intra-compartmental pressure gradients. Access to the CSF space provides a method for ICP treatment via CSF drainage. However, placement of these catheters does involve a ventriculostomy with the attendant small risk of infection, haematoma formation, and seizures.

A variety of catheter tip transducer systems are also available; the Codman and Camino systems in particular are commonly used in the management of head injured patients. Both these systems require a pre-insertion calibration. After insertion these catheters cannot be corrected for zero drift of the catheter.

Sites other than the lateral ventricle can be used for ICP recording; these include the subarachnoid and subdural

Table 3 Indications for intracranial pressure (ICP) monitoring

Condition	Comments
Subarachnoid haemorrhage	With associated hydrocephalus—ventriculostomy allows therapeutic drainage and ICP monitoring ? patients in coma ? as a guide to hypertensive hypervolaemic treatment for delayed ischaemia
Spontaneous intracerebral haemorrhage	There is no good clinical evidence of benefit from monitoring patients in this group
Reye's syndrome	Active treatment of raised ICP decreases mortality Blood ammonia >300 mg/100 ml and deteriorating conscious level usually considered indications for monitoring
Brain tumours	Not indicated routinely, but may be of value in selected patients deemed at high risk of swelling or obstructive hydrocephalus—for example, following posterior fossa surgery
Normal pressure hydrocephalus	An increase in spontaneous nocturnal "B" waves has been reported to be predictive of a good response to CSF diversion
Decompensated hydrocephalus	Can be a valuable diagnostic tool in complex cases
"Benign" intracranial hypertension	Monitoring often undertaken via a lumbar subarachnoid catheter as both a diagnostic test and to monitor response to treatment
Other potential indications	Hypoxic brain swelling after drowning, meningitis, encephalitis, venous sinus thrombosis, and hepatic encephalopathy

spaces as well as brain parenchyma. Such alternative sites may be associated with lower risks of infection and haemorrhage, but often do not provide reliable measurements.

In infants a transduction device placed over the anterior fontanelle provides a non-invasive measure of ICP. Several means of assessing ICP indirectly have also been developed, including measures of the latency of components of the visual evoked response and transcranial Doppler waveform analysis. To date none of these indirect indices of ICP have found a place in routine clinical practice.

MANAGEMENT

Primary management is directed, if possible, at the specific process responsible for the rise in ICP (such as surgical removal of mass lesions, dexamethasone treatment for oedema associated with intracranial tumours, control of hydrocephalus, etc). These aspects of the management of specific conditions will not be reviewed here. Measures for the medical management of raised ICP will be discussed. Much of the clinical work in this area has been in severe head injury (critically reviewed by Bullock and colleagues). Treatments are applied in a sequential manner until control of ICP is obtained, and a number of published protocols exist for the management of ICP in head injured patients.

First tier treatment

General physiologic homeostasis

Significant departures from the normal physiological status can adversely affect ICP and/or cerebral perfusion. Attention is therefore directed at maintaining adequate arterial oxygen tension and the patient being euvolaemic and euosmotic. Pyrexia should be avoided as it increases ICP, being an independent predictor of poor outcome after severe head injury. Seizures contribute to raised ICP and should be managed aggressively using standard anticonvulsant loading regimens.

CSF drainage

When an intraventricular catheter is used to monitor ICP, CSF drainage is an effective method for lowering ICP. This can be accomplished by intermittent drainage for short periods in response to elevations in ICP. The principal risks of ventriculostomy are infection and haemorrhage. Most studies report rates of bacterial colonisation rather than symptomatic infection ranging from 0–19%. The incidence of ventriculostomy associated haematoma is approximately 2%.

Head of bed elevation

Elevation of the head of the bed to 30° improves jugular venous outflow and lowers ICP. In patients who are hypovolaemic, this may be associated with a fall in blood pressure and an overall fall in cerebral perfusion pressure. Care must therefore initially be taken to exclude hypovolaemia. The position of the arterial pressure transducer will also need to be adjusted to ensure reliable measurements of CPP.

Analgesia and sedation

This is usually accomplished using intravenous propofol, etomidate or midazolam for sedation and morphine or alfentanil for analgesia and antitussive effect.

Neuromuscular blockade

Muscle activity may further raise ICP by increasing intrathoracic pressure and obstructing cerebral venous outflow. If this does not respond to analgesia and sedation then neuromuscular blockade is considered. However, the prophylactic use of neuromuscular blockade in patients without proven intracranial hypertension has not been shown to improve outcome. It is associated with an increased risk of complications such as pneumonia and sepsis, and would obscure seizure activity.

Diuretics

The most commonly used agent is mannitol, an intravascular osmotic agent that can draw fluid from both normal and abnormal brain. In addition it increases cardiac preload and CPP, thus decreasing ICP through cerebral autoregulation. Mannitol decreases blood viscosity, resulting in reflex vasoconstriction and decreased cerebrovascular volume. The major problems associated with mannitol administration are hypovolaemia and the induction of a hyperosmotic state. Serum osmolality should not be allowed to rise over 320 mOsm/kg.

Hyperventilation

Hyperventilation lowers ICP by inducing hypocapnoic vasoconstriction mediated through metabolic autoregulation. Unfortunately hyperventilation also induces or exacerbates cerebral ischaemia in a proportion of patients. A further problem is the development of tachyphylaxis as compensation for systemic alkalosis ensues. This diminishes the effect of the established level of hypocapnia and makes weaning more difficult, as a rebound CSF acidosis and vasodilatation result

when eucapnia is restored. The effect of prophylactic ventilation on outcome in patients with head injury has been investigated in a prospective randomised controlled trial that failed to show any benefit from hyperventilation one year after injury, with a possible worsening of outcome in some patient subgroups at three and six months.

Second tier treatments

Barbiturate coma

Barbiturates in high doses are effective in lowering refractory intracranial hypertension, but ineffective or potentially harmful as a first line or prophylactic treatment in patients with head injuries. High dose barbiturate treatment acts by depressing cerebral metabolic activity. This results in a reduction in cerebral blood flow, which is coupled to metabolism, and a fall in ICP. The use of barbiturates in the treatment of refractory intracranial hypertension requires intensive monitoring and is associated with a significant risk of complications, the most common complication being hypotension. This may explain the lack of proven benefit on outcome in head injury. Cerebral electrical activity should ideally be monitored during high dose barbiturate treatment, preferably on a continuous basis, burst suppression activity providing a physiologic end point for dose titration. Withdrawal of treatment should be gradual to avoid rebound intracranial hypertension.

Optimised hyperventilation

This involves the use of more aggressive hyperventilation, with concomitant measurement of jugular venous saturation in an attempt to prevent hyperventilation induced ischaemia. It is based on the hypothesis that there is uncoupling of cerebral blood flow and metabolism after head injury. Relative cerebral hyperaemia occurs and is manifest as a low cerebral arterial venous difference in oxygen. In such patients reducing cerebral blood volume and hence ICP by hyperventilation would not lead to cerebral ischaemia. One major concern with this technique is the extent to which samples taken from one jugular bulb are representative of the oxygen saturation of blood in the contralateral hemisphere or even of variations within the ipsilateral hemisphere. Thus, focal areas of cerebral ischaemia may be produced even though global measures suggest adequate oxygen supply.

Hypothermia

Hypothermia has been investigated in head injury both as a means of controlling ICP and as a possible neuroprotectant strategy. Cooling to 34°C can be effective in lowering refractory intracranial hypertension but is associated with a relatively high rate of complications including pulmonary, infectious, coagulation, and electrolyte problems. There also appears to be a significant rebound in ICP when induced hypothermia is reversed. A recent randomised controlled trial of moderate hypothermia following severe closed head injury, despite imbalance between treatment groups, failed to show any benefit on outcome.

Decompressive craniectomy

This technique has been reported as being beneficial in a number of disorders, including head injury, cerebral infarction, spontaneous intracerebral and subarachnoid haemorrhage, and Reye's syndrome. Such surgery can certainly lower

ICP but may reduce mortality at the expense of unacceptably high levels of morbidity. To date there are no prospective randomised controlled trials that show a convincing beneficial effect on outcome. The technique may, however, turn out to be effective in selected subgroups of patients with refractory intracranial hypertension.

Alternative treatment philosophies

There are alternative approaches to the ICP targeted approach to the management of raised ICP after head injury. In CPP based treatment the aim is to keep CPP above a certain level rather than to treat specific levels of ICP. However, a recent randomised trial has failed to show an improved outcome in head injured patients treated with a CPP based protocol when compared with patients treated with an ICP based protocol. The CPP targeted group had a higher incidence of pulmonary complications.

The Lund protocol focuses on the prevention of vasogenic oedema. This assumes a disruption of the blood-brain barrier following head injury and employs a variety of manipulations to increase the hydrostatic and osmotic forces favouring maintenance of fluid within the intravascular compartment. Uncontrolled trials have yielded results comparable to established management protocols, but there is as yet no controlled clinical trial demonstrating the superiority of this protocol over other methods of ICP management.

Targeted therapy aims to match treatment methods to differing pathophysiological processes and can be seen as a logical approach to the treatment of intracranial hypertension after head injury, which is clearly a heterogeneous multifactorial condition. Continuing advances in intracranial monitoring and imaging technology are likely to make possible the identification of specific pathophysiological profiles in patients. This should allow treatment regimens to be tailored to individual patients, with a better prospect of improved outcome.

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